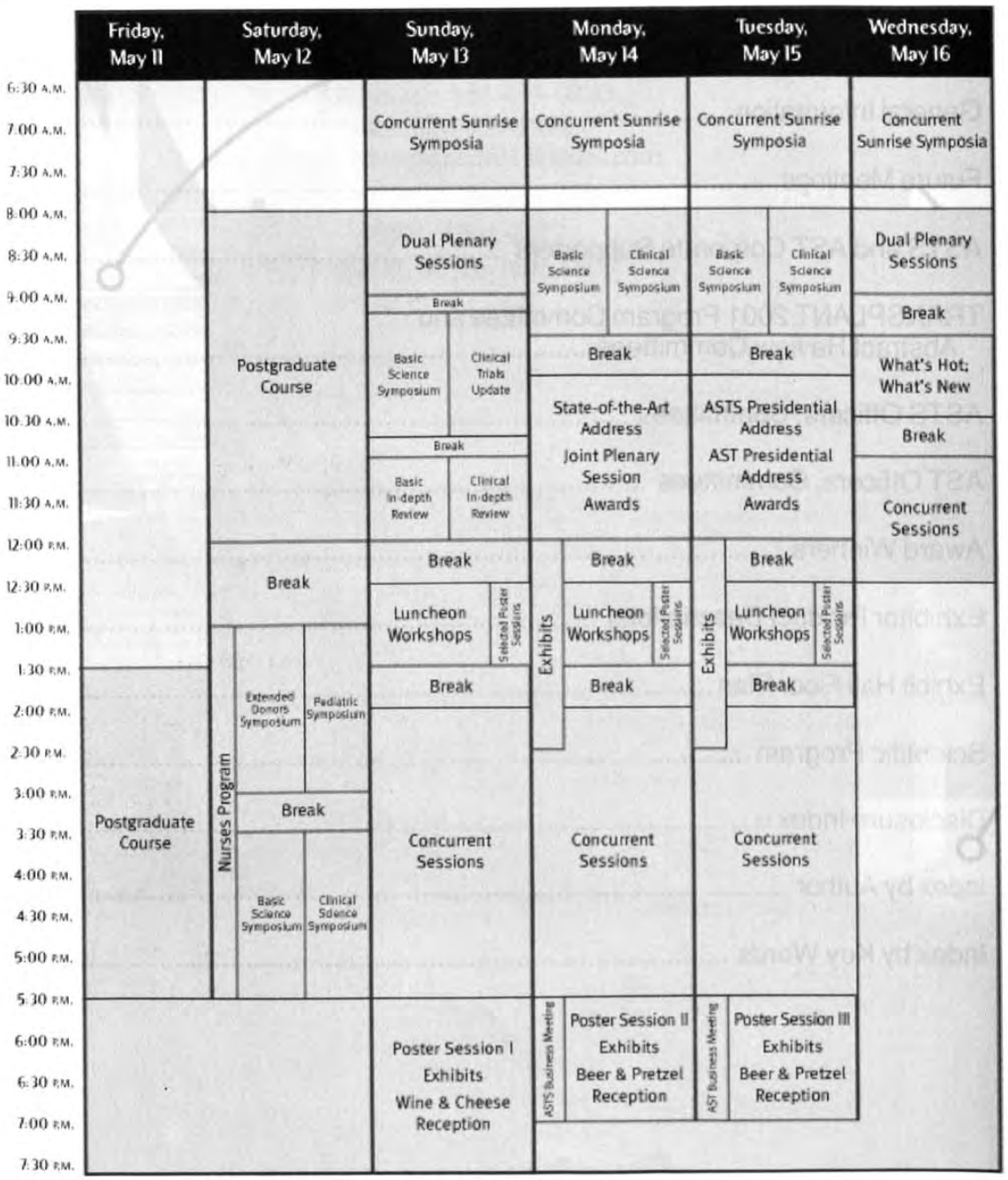
schedule AT A GLANCE



schedule of activities

On Site Registration Hours

Friday, May 11	7:00 A.M. – 7:00 P.M.
Saturday, May 12	7:00 A.M. – 5:30 P.M.
Sunday, May 13	7:00 A.M. – 7:30 P.M.
Monday, May 14	7:00 A.M. – 7:00 P.M.
Tuesday, May 15	7:00 A.M. – 7:00 P.M.
Wednesday, May 16	7:00 A.M. – 12:30 P.M.

Exhibit Hours

Sunday, May 13	5:30 P.M. – 7:30 P.M.
Monday, May 14	11:30 A.M. – 2:30 P.M.
	5:30 P.M. – 7:00 P.M.
Tuesday, May 15	11:30 A.M. – 2:30 P.M.
	5:30 P.M. – 7:00 P.M.

Postgraduate Course

Friday, May 11 — 1:30 P.M. – 5:30 P.M.

Saturday, May 12 8:00 A.M. – 12:00 P.M.

Pre-meeting Symposia

Saturday, May 12

Extended Donors/Allocation Symposium:
Report from the Cadaver Donor Conference

1:00 P.M. – 3:00 P.M.

Pediatrics Symposium:
Transplantation in Adolescents

1:00 P.M. – 3:00 P.M.

Nurses/Coordinators Program

1:00 P.M. – 5:30 P.M.

Basic Science Symposium:
Genomics and Proteomics Overview

3:30 P.M. – 5:30 P.M.

Clinical Science Symposium:
Anti-Microbial Resistance in Transplant Infectious Diseases

3:30 P.M. – 5:30 P.M.

TRANSPLANT 2001 Scientific Sessions

Sunday, May 13	8:00 A.M. – 5:30 P.M.
Monday, May 14	8:00 A.M. – 5:30 P.M.
Tuesday, May 15	8:00 A.M. – 5:30 P.M.
Wednesday, May 16	8:00 A.M. – 12:30 P.M.

Note: Concurrent Sessions will be held in both the Sheraton and Hotel Intercontinental Chicago.

Sunrise Symposia

Sunday, May 13	6:30 A.M. – 7:45 A.M.
Monday, May 14	6:30 A.M. – 7:45 A.M.
Tuesday, May 15	6:30 A.M. – 7:45 A.M.
Wednesday, May 16	6:30 A.M. – 7:45 A.M.

ASTS Business Meeting

Monday, May 14 — 5:45 P.M. – 6:45 P.M.

AST Business Meeting

Tuesday, May 15 — 5:45 P.M. – 6:45 P.M.

Parallel Luncheon Workshops

Limited Attendance

Sunday, May 13	12:30 P.M. – 1:30 P.M.
Monday, May 14	12:30 P.M. – 1:30 P.M.
Tuesday, May 15	12:30 P.M. – 1:30 P.M.

Note: Luncheon workshops will be located in both the Sheraton and the Hotel Intercontinental Chicago.

Poster Sessions (Presenters in Attendance)

Sunday, May 13	5:30 P.M. – 7:30 P.M.
	with wine and cheese reception
Monday, May 14	5:30 P.M. – 7:00 P.M.
	with beer and pretzel reception
Tuesday, May 15	5:30 P.M. – 7:00 P.M.
	with beer and pretzel reception

Welcome to the Joint American Transplant Meeting May 11-16, 2001

How to Use this Program Book

The Program Book is divided into four sections: Association and general information, scientific program, abstracts and indexes. All accepted abstracts are published in this supplement and begin on page 136.

Association and General Information

This section will list the officers and committee members of the American Society of Transplant Surgeons and the American Society of Transplantation, the master schedule of all meeting events, times and schedules of the specific activities, lists of corporate partners, future meetings and exhibitors. Please reference this section for any logistic and/or time questions regarding the meeting in general.

Scientific Program

The 2001 Scientific Program for the Joint Meeting begins on page 47, including:

- Day-at-a-Glance pages with the daily schedules of events for each day
- Pre-meeting Symposia and Mini-Courses
- Sunrise Symposia
- Plenary Sessions
- Concurrent Sessions
- Poster Sessions
- Parallel Luncheon Workshops

Abstracts

All oral abstract presentations listed in the program section have been assigned a chronological program number. This number corresponds with the number of the abstract printed in this supplement. Poster presentations will be held in the River Exhibition Hall. All poster presentations have been assigned a chronological program number for reference when locating your printed abstract. Board numbers for poster presentations are identified with a P before the number. Please refer to the program for the appropriate board number when locating a poster for viewing.

Indexes

Disclosure Index

The disclosure index lists all abstracts by program number where a potential relationship that could affect the objectivity of a presentation has been disclosed. This index may be found on page 477.

Author Index

The author index begins on page 479 of this supplement. The author index lists all abstract authors in alphabetical order. Numbers correspond with the abstract and program numbers.

Key Word Index

The key word index, lists key words from each abstract in alphabetical order. The Key Word index begins on page 511.

General Information

Target Audience

This meeting is designed for physicians, surgeons, scientists, nurses, organ procurement personnel, and pharmacists who are interested in the clinical and research aspects of solid organ and tissue transplantation

Overview of TRANSPLANT 2001

- To provide a forum for exchange of new scientific and clinical information relevant to solid organ and tissue transplantation.
- To create an arena for the interchange of ideas regarding care and management of organ and tissue transplant recipients.
- To facilitate discussions of the socioeconomic, ethical and regulatory issues related to solid organ and tissue transplantation.
- A variety of formats have been planned to encourage the exchange of new scientific and clinical information and support an interchange of opinions regarding care and management issues, as well as socioeconomic, ethical and regulatory issues relevant to organ and tissue transplantation.
- Scientific material is presented through symposia, oral abstracts, concurrent workshops, and poster presentations as well as small group sessions designed for in-depth exploration of both clinical and basic science topics.

Educational Objectives

The objective of this activity is to help you understand, examine, and discuss:

- The latest advances in transplantation, both clinical and basic;
- Barriers to successful outcomes;
- Challenges in pediatric transplantation;
- Immunosuppressants inthe various organs;
- The newest technologies in immunology and transplant research;
- Advances in xenotransplantation
- Infections associated in transplantation;
- The latest finding in cell and stem cell research;
- Progress in transplant tolerance;
- The problem of delayed renal allograft function;
- The pros and cons of heart and heart-lung transplantation;
- Current information on immunobiology;
- The latest clinical trial results:
- Future challenges in kidney-pancreas and pancreas;
- Practice guidelines for follow-up and management of transplant recipients;
- The path of curing autoimmune diabetes.

Abstracts

Abstracts selected for TRANSPLANT 2001 are presented in plenary, concurrent and poster sessions that highlight the most outstanding papers from all transplant specialties.
All accepted abstracts are published in this official TRANSPLANT 2001 program book, a supplement to the new joint AST/ASTS journal, **American Journal of Transplantation** (AJT).
All abstracts selected for presentation and the final program are available on-line through the TRANSPLANT 2001 website, www.transplant2001.org.

Registration

All attendees must be registered for the meeting. Registration is located on the lobby level in the built-in registration counters off the main hotel lobby of the Sheraton Chicago Hotel and Towers. Registration hours are listed on page 5 under the schedule of activities. Program distribution is located in the "Coat Check" area on the lobby level, near the Gift Shop. Your meeting bag and program will be available for pick up during the same hours as open registration hours.

Restaurant Reservation Service

A restaurant reservation desk is open in the registration area. Staff will be available for assisting you with dinner reservations during the following hours:

Friday, May 11	1:00 PM – 6:00 PM
Saturday, May 12	11:00 AM– 6:00 PM
Sunday, May 13	11:30 AM – 7:30 PM
Monday, May 14	11:30 AM – 7:00 PM
Tuesday, May 15	11:30 AM – 7:00 PM

Postgraduate Course

Due to a high number of requests for review and update material, the Planning Committee has developed a course that will provide the type of programming that this requires. It is designed for attendees wishing to review some of the basic and important issues related to transplantation. Efforts have been made to cover issues related to specific organs in addition to universal transplant topics. Speakers have been asked to review their topics as well as present the most recent data. They have been instructed to not only present their research but also summarize important recent publications/work in the area they are discussing. The course will begin on Friday May 11 at 1:30 PM and end Saturday, May 12 at 12:00 Noon. The program for this new event can be found on page 47. This event takes place in the Sheraton. There is a separate registration for this and it is not included in the full meeting registration.

Pre-meeting Symposia

On Saturday, May 12, there are several pre-meeting symposia beginning at 1:00 PM. All pre-meeting symposia take place in the Sheraton. These events are included in your registration for the Annual Meeting.

Special Course for Transplant Nurses and Coordinators

A special mini-course for transplant nurses and coordinators is taking place on Saturday, May 12 from 1:00 PM to 6:00 PM. Registration is included with the Annual Meeting Registration. This event takes place in the Sheraton.

Two Hotels

Selected parallel luncheon workshops and concurrent sessions are located in the Hotel Intercontinental Chicago. This hotel is located approximately three blocks from the Sheraton. A 30-minute break before and after the luncheons and between the concurrent sessions has been included in the program to allow for travel between the two properties. Shuttle buses will be running continuously between the Sheraton and Intercontinental between 11:45 AM and 6:00 PM on Sunday - Tuesday.

Parallel Luncheon Workshops

Parallel Luncheon Workshops, designed to be small interactive working sessions, are limited in size. Most of the luncheons are sold out. If you are not registered for one, you should check at the registration desk for available sessions.

Selected Poster Sessions

The posters are an important part of the scientific program of TRANSPLANT 2001. The Planning Committee has selected a number of top posters to be presented in special open sessions as mini-oral presentations. These sessions take place Sunday - Tuesday from 12:30 PM - 1:30 PM. A cash luncheon will be available on the Promenade Ballroom of the Sheraton.

Sunrise Symposia

Starting on Sunday, May 13 through Wednesday, May 16, there are concurrent Sunrise Symposia each morning from 6:30 AM to 7:45 AM. These are single-topic symposia that are open to all registrants for TRANSPLANT 2001. A continental breakfast will be provided each morning. All Sunrise Symposia take place in the Sheraton.

Exhibits

The exhibits are an integral part of the complete educational experience and will feature the latest in technology and research in the field of transplantation medicine. The exhibits are located in the Sheraton Chicago Hotel and Towers and ample opportunity is provided for attendees to visit the exhibits. Detailed information on each of the exhibits and booth location are provided in this program on page 33. Along with your name badge, you have an electronic business card. By presenting this card to exhibitors during show hours, exhibiting companies can easily capture your name and address information for follow up. Exhibit hours are listed on page 5 under the schedule of activities.

Member Services

There are membership booths for both the ASTS and AST located in the Sheraton River Exhibition Hall. Membership, future meeting information, public policy information, and society literature as well as computer demonstrations of the ASTS and AST web sites, are available. Members and non-members are encouraged to stop by to find out about the many benefits of ASTS and AST membership.

Continuing Medical Education Accreditation

The ASTS designates this educational activity for a maximum of 44.5 hours in category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Pharmacists ACPE

Fujisawa Healthcare, Inc. is approved by the American Council on Pharmaceutical Education as a provider of continuing pharmaceutical education. This information is not intended as an endorsement of, or as a recommendation of, any Fujisawa Healthcare, Inc. product mentioned in this material.

Nursing

Amedco is approved as a provider of continuing education by the MN Nurses Association, which is accredited as an approver of continuing education in nursing by the Amercian Nurses Credentialing Center's Commission on Accreditation, Provider P-73.36; 44.5 Contact Hours.

Transplant Coordinators ABTC

The American Board of Transplant Coordinators (AVTC) has approved this educational offering for up to 44.5 Category I Continuing Education Points for Transplant Coordinators (CEPTCs).

ACCME Accreditation Statement

The activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (AACME) by the American Society of Transplant Surgeons (ASTS). The ASTS is accredited by the ACCME to provide continuing medical education for physicians.

Full Disclosure Policy

All faculty participating in the continuing medical education programs sponsored by the American Society of Transplant Surgeons are expected to disclose to the program audience any real or apparent conflict(s) of interest related to the content of their presentation(s).

A disclosure statement listing all faculty who have indicated potential relationships that could affect the objectivity of their presentation can be found in your meeting bag. Disclosures related to abstract presentations can be found on page 477.

ADA Compliance

The Sheraton Chicago Hotel and Towers and Hotel Intercontinental Chicago are fully accessible to the physically challenged.

Instructions for Obtaining Credits

CME

If you would like continuing medical education (CME) credits, simply pickup a CME Certificate form at the CME Information Desk, located on the ballroom level of the Sheraton, or the Registration Desk. At the end of your last day of the meeting just fill out the form and turn in the bottom copy at the CME Information Desk or Registration Desk. The top copy is your CME certificate, which you keep. That's all you need to do, no waiting for the mail! No other CME certificate will be issued.

The CME information desk will be open during educational hours.

Pharmacists (ACPE)

Stop by the CME Information Desk. We have everything you need there.

Nursing

Nurses can receive contact hours for attending Transplant 2001. We have all the details at the CME Information Desk and the Registration Desk.

Transplant Coordinators (ABTC)

Transplant Coordinators can receive contact hours for attending Transplant 2001. We have all the details at the CME Information Desk and the Registration Desk.

International Attendees

For international attendees a "Certificate of Attendance" can be picked up at the CME Information Desk or the Registration Desk.

Evaluation

Your Opinions Are Important To Us!

EVALUATION FORMS are located at the CME Information Desk or the Registration Desk. We take your input seriously and use it to plan and improve the meeting each year. Stop by and pick one up.

Speaker Ready Room

There are Slide Preview/Speaker Ready Rooms for presenters located in Sheraton Chicago Hotel and the Hotel Intercontinental. Be sure to check the program for hotel and room location.

All session rooms, concurrent and plenary, will be set for both slide and/or computer presentations. Speakers are asked to bring their computer presentation formatted to run on a windows-based PC on either CD-rom, zip drive, jazz drive or floppy disk. Speakers who are using computer presentation will be asked to check in their presentation the day BEFORE they are scheduled to present. Please read your acceptance instructions carefully to avoid any problems.

IF YOU ARE PRESENTING IN THE HOTEL INTERCONTINENTAL DO NOT CHECK YOUR SLIDES INTO THE SHERATON.

Sheraton Speaker Ready Room: Parlor C

Friday, May 11	11:00 AM - 6:00 PM
Saturday, May 12	7:00 AM - 6:00 PM
Sunday, May 13	6:00 AM - 6:00 PM
Monday, May 14	6:00 AM - 6:00 PM
Tuesday, May 15	6:00 AM - 6:00 PM
Wednesday, May 16	6:00 AM - 12:30 PM

Intercontinental Hotel Speaker Ready Room: Ohio

Sunday, May 13	7:00 AM - 6:00 PM
Monday, May 14	7:00 AM - 6:00 PM
Tuesday, May 15	7:00 AM - 6:00 PM

Poster Sessions

The Posters Sessions are an important education event of this meeting. We hope you support and attend these scientific presentations. All poster presentations will be held in the River Exhibition Hall on the ground floor of the Sheraton. All posters presentations have been assigned a chronological program number for reference when locating your printed abstract in this supplement. A P# indicates board number location for poster boards in the hall. Please refer to the program for the appropriate board.

Posters may be set up and viewed during the following times:

Sunday May, 13
Set up: 3:00 PM - 5:30 PM
Viewing: 5:30 PM - 7:30 PM
Presenters in attendance: 5:30 PM - 7:30 PM
Dismantle: Immediately after session

Monday May, 14
Set up: 7:00 AM - 8:00 AM
Viewing: 8:00 AM - 7:00 PM
Presenters in attendance: 5:30 PM - 7:00 PM
Dismantle: Immediately after session

Tuesday May, 16
Set up: 7:00 AM - 8:00 AM
Viewing: 8:00 AM - 7:00 PM
Presenters in attendance: 5:30 PM - 7:00 PM
Dismantle: Immediately after session

Poster Session I will be held in conjunction with the opening of the Exhibits and the wine and cheese reception. Presenters will be in attendance during the entire time from 5:30 PM - 7:30 PM. Presenters for Poster Session II and III on Monday and Tuesday should be in attendance from 5:30 PM - 7:00 PM and beer and pretzels will be served. Posters will be open for viewing on Monday and Tuesday from 8:00 AM - 7:00 PM.

Posters are to be removed immediately following the end of the poster session. Any posters left on the board will be removed and can be picked up at the Freeman desk in the back of the hall before Wednesday, May 16th at 12:00 noon. Any posters not picked up by that time will be discarded.

Message Center

Novartis is supporting a Message Center, located in the River Exhibition Promenade, directly outside the Exhibit Hall. Please check with them for fax and telephone numbers or call the Sheraton Chicago Hotel and Towers main number, 312-464-1000 and ask to be connected with the TRANSPLANT 2001 Message Center.

Shuttle Buses

There will be continuous shuttle bus service between the Sheraton, Intercontinental and all TRANSPLANT 2001 hotels. Bus schedules will be posted in the lobbies of all the hotels. Service will begin daily at 6:00 AM each morning.

Press

Press may register for the meeting free of charge as Press. Proper press credentials must be presented with your form. Press registration does not include any luncheon workshops or the postgraduate course. The TRANSPLANT 2001 Press Room is located in Parlor B in the Sheraton Hotel. This office will be open Sunday-Wednesday from 10:00 AM - 5:00 PM daily.

Future Joint American Transplant Meetings

Transplant 2002
May 15 – 19, 2004
Boston, MA

Transplant 2003
May 31 – June 4, 2003
Washington, DC

Transplant 2004
April 27 – May 1, 2002
Washington, DC

Transplant 2005
May 20 – 25, 2005
Seattle, WA

Posters may be set up and viewed during the following times:

Sunday May, 13
Set up: 3:00 PM - 5:30 PM
Viewing: 5:30 PM - 7:30 PM
Presenters in attendance: 5:30 PM - 7:30 PM
Dismantle: Immediately after session

Monday May, 14
Set up: 7:00 AM - 8:00 AM
Viewing: 8:00 AM - 7:00 PM
Presenters in attendance: 5:30 PM - 7:00 PM
Dismantle: Immediately after session

Tuesday May, 16
Set up: 7:00 AM - 8:00 AM
Viewing: 8:00 AM - 7:00 PM
Presenters in attendance: 5:30 PM - 7:00 PM
Dismantle: Immediately after session

Poster Session I will be held in conjunction with the opening of the Exhibits and the wine and cheese reception. Presenters will be in attendance during the entire time from 5:30 PM - 7:30 PM. Presenters for Poster Session II and III on Monday and Tuesday should be in attendance from 5:30 PM - 7:00 PM and beer and pretzels will be served. Posters will be open for viewing on Monday and Tuesday from 8:00 AM - 7:00 PM.

Posters are to be removed immediately following the end of the poster session. Any posters left on the board will be removed and can be picked up at the Freeman desk in the back of the hall before Wednesday, May 16th at 12:00 noon. Any posters not picked up by that time will be discarded.

Message Center

Novartis is supporting a Message Center, located in the River Exhibition Promenade, directly outside the Exhibit Hall. Please check with them for fax and telephone numbers or call the Sheraton Chicago Hotel and Towers main number, 312-464-1000 and ask to be connected with the TRANSPLANT 2001 Message Center.

Shuttle Buses

There will be continuous shuttle bus service between the Sheraton, Intercontinental and all TRANSPLANT 2001 hotels. Bus schedules will be posted in the lobbies of all the hotels. Service will begin daily at 6:00 AM each morning.

Press

Press may register for the meeting free of charge as Press. Proper press credentials must be presented with your form. Press registration does not include any luncheon workshops or the postgraduate course. The TRANSPLANT 2001 Press Room is located in Parlor B in the Sheraton Hotel. This office will be open Sunday-Wednesday from 10:00 AM - 5:00 PM daily.

Future Joint American Transplant Meetings

Transplant 2002
May 15 – 19, 2004
Boston, MA

Transplant 2003
May 31 – June 4, 2003
Washington, DC

Transplant 2004
April 27 – May 1, 2002
Washington, DC

Transplant 2005
May 20 – 25, 2005
Seattle, WA

ASTS and AST Corporate Supporters

The ASTS and AST gratefully acknowledge the support of unrestricted educational grants from the following companies (at time of publication):

American Society of Transplant Surgeons

Founder's Circle

Fujisawa Healthcare, Inc.

Novartis Pharmaceuticals Corporation

Roche Laboratories Inc.

Wyeth-Ayerst Pharmaceuticals

Friend

Tyco Healthcare - Valley Lab

American Society of Tranplantation

Platinum

Fujisawa Healthcare, Inc.

Novartis Pharmaceuticals Corporation

Wyeth-Ayerst Pharmaceuticals

Gold

Roche Laboratories Inc.

Friend

MedImmune, Inc.

Corporate Supported Social Events and/or Symposia

The supporters of the ASTS and AST have planned a number of events for the evenings during the Joint American Transplant Meeting. Information regarding these events will be sent from the individual sponsors or if you are interested in attending, you may stop at their booth in the Exhibit hall.

Saturday, May 12

Sponsored by Novartis Pharmaceutical Inc.

Sunday, May 13

Sponsored by Roche Laboratories Inc.

Monday, May 14

Sponsored by Wyeth-Ayerst Inc.

Tuesday, May 15

Sponsored by Fujisawa Healthcare Inc.

TRANSPLANT 2001 Program Committee

Joint 2001 Executive Planning Committee

Hugh Auchincloss, Jr., MD
Mark L. Barr, MD
Fadi G. Lakkis, MD
J. Richard Thistlethwaite, Jr., MD, PhD

Joint 2001 Planning Committee

David M. Briscoe, MD
Jonathan S. Bromberg, MD, PhD
Kenneth E. Drazan, MD
Donald E. Hricik, MD
Stuart J. Knechtle, MD
Jerzy W. Kupiec-Weglinski, MD, PhD.
Michael R. Lucey, MD
Joren C. Madsen, MD, DPhil
Arthur J. Matas, MD
John P. Roberts, MD
Peter G. Stock, MD, PhD
Angus W. Thomson, PhD. DSc.

Transplant 2001 Abstract Review Committee

Kidney - Acute/Chronic Rejection
Chairs:
Jimmy A. Light
Douglas J. Norman
Enver Akalin
Colm C. Magee
Thomas R. McCune
Thomas G. Peters
David Rush
Susan L. Saidman
Fred P. Sanfilippo
Kim Solez
Randall Sung

Kidney - GVH, Complications, Infections
Chairs:
Daniel C. Brennen
Geraldine G. Miller
Nesli Basgoz
Bryan N. Becker
Roy D. Bloom
Emily A. Blumberg
Jay Alan Fishman
Simin Goral
Susan Keay
Marian G. Michaels
Ram Peddi
Jutta Preiksaitis
Emilio Ramos
Robert H. Rubin
Gary G. Singer

Kidney - Immunosuppression A
Chairs:
Mitchell L. Henry
Flavio Vincente
William J. C. Amend
Robert S. Gaston
Sundaram Hariharan
Harold J. Helderman
Christopher L. Marsh
Todd E. Pesavento
Raymond Pollak
Timothy I. Pruett
James A. Schulak
Ron Shapiro
Alan H. Wilkinson

Kidney - Immunosuppression B
Chairs:
Gabriel M. Danovitch
Mark D. Pescovitz
John J. Curtis
Elmahdi A. Elkhammas
Ronald S. Filo
M. Roy First
Lorenzo Gallon
Thomas A. Gonwa
William H. Marks
Paul W. Nelson
Martha Pavlakis
John D. Pirsch
Charles F. Shield
Stephen J. Tomlanovich

Kidney - Pediatrics, Recurrent Disease
Chairs:
Mark R. Benfield
Matthew R. Weir
Kenneth L. Brayman
David J. Cohen
Richard A. Cohn
Vikas R. Dharnidharka
Robert B. Ettenger
Bertram L. Kasiske
Minnie Sarwal
Asher Schachter
Bradley A. Warady

Kidney - Preservation, Donation/Allocation, Economics/Public Policy, Surgical Techniques, and Other
Chairs:
David J. Conti
Francis L. Delmonico
Stephen T. Bartlett
Nicholas J. Feduska
Carl E. Haisch
Marquis E. Hart
David Hull
Lawrence G. Hunsicker
Richard J. Knight
Jeffrey A. Lowell
Robert M. Merion
Edgar L. Milford
Brian M. Murray
Richard V. Perez
John A. Powelson
Lloyd E. Ratner
James J. Wynn

Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics
Chairs:
Gary Levy
Jorge Reyes
James D. Eason
Jean C. Emond
Thomas M. Fishbein
David R. Grant
Douglas W. Hanto
Thomas G. Heffron
Abhinav Humar
Lynt B. Johnson
Goran B. Klintmalm
John R. Lake
Sue V. McDiarmid
Michael J. Millis
Leendert C. Paul
Jorge Rakela
Richard J. Rohrer
Charles B. Rosen
Hugo R. Rosen
Lewis W. Teperman
Russell H. Wiesner

Liver - Infections, Complications, Recurrent Disease, Surgical Techniques
Chairs:
Alan N. Langnas
Carlos V. Paya
Jeffrey S. Crippin
Sandy Feng
John D. Hughes
Mark W. Johnson
Igal Kam
Ruud A. Krom
Maureen F. Martin
Victor J. Navarro
Kim M. Olthoff

Liver - Preservation, Economics/Public Policy, Donation Allocation, Other
Chairs:
Charles M. Miller
Michael K. Porayko
Gregory T. Everson
Richard B. Freeman
Gregory J. Gores
Eliezer Katz
Emmet B. Keeffe
Paul Martin
Santiago J. Munoz
John J. Poterucha
Christopher R. Shackleton
Mitchell L. Shiffman

Pancreas and Islets - All Topics
Chairs:
Osama Gaber
James Shapiro
Christopher E. Freise
Lillian W. Gaber
Paul F. Gores
Scott A. Gruber
Rainer WG Gruessner
Angelika C. Gruessner
Dixon B. Kaufman
David K. Klassen
Ali Naji
Cristiana Rastellini
Camillo Ricordi
Paul R. Robertson
David Shaffer
Michael E. Shapiro
Robert J. Stratta
David E. R. Sutherland
Harold C. Yang

Heart/Lung - All Topics
Chairs:
Bruce R. Rosengard
David O. Taylor
R. Morton Bolman
David M. Follette
Edward R. Garrity, Jr.
Leo C. Ginns
Marshall Hertz
Maryl R. Johnson
Jon Kobashigawa
Robert B. Love
James E. Loyd
Janet R. Maurer
Si M. Pham
Robert C. Robbins
Larry L. Schulman
Sara J. Shumway
Thoralf Sundt III
Hannah A. Valentine

Bone Marrow - All Topics
Chairs:
Ginny L. Bumgardner
Adriana Zeevi
Donald C. Dafoe
Lisa Florence
Mary S. Leffell
T. Mohanakumar
Susan L. Orloff
Nancy Reinsmoen
Mark D. Stegall
Brian M. Susskind

Immunosuppression, Preclinical Studies
Chairs:
Paul C. Kuo
Stanislaw Stepkowski
Michael M. Abecassis
Giacomo P. Basadonna
Rafik M. Ghobrial
Roslyn B. Mannon
Michael Melter
Randall E. Morris
Barbara Murphy
Bruno Watschinger
Lucille E. Wrenshall

Tolerance
Chairs:
Robert Lechler
Judith M. Thomas
Alan D. Kirk
Christian P. Larsen
Takashi Maki
Soji F. Oluwole
Abdul S. Rao
Terry B. Strom
Manikkam Suthanthiram
Megan Sykes
Ana Maria Waaga

Acute/Chronic Rejection
Chairs:
Wayne W. Hancock
Kenneth A. Newell
William M. Baldwin
Eugenia V. Fedoseyeva
Gregg A. Hadley
Phil F. Halloran
Peter Nickerson
Allen J. Norin
Thomas C. Pearson
James W. Williams

Allorecognition, Antigen Presentation, Co-Stimulation and Other
Chairs:
William J. Burlingham
Ann M. VanBuskirk
Gilles Benichou
Elizabeth H. Field
Peter S. Heeger
Sheri M. Krams
Charles G. Orosz
John P. Vella

Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines
Chairs:
Robert L. Fairchild
Olivia M. Martinez
Keith D. Bishop
Anil Chandraker
Anita Chong
Thomas M. Coffman
Ashwani Khanna
Didier A. Mandelbrot
David Perkins
Jeffrey D. Punch

Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH
Chairs:
Anthony M. Jevnikar
David M. Rothstein
David A. Geller
Xian Chang Li
Jay S. Markowitz
Anthony P. Monaco
Noriko Murase
Philip J. O'Connell
Patricia Thistlethwaite
Kenneth A. West
Xin Xiao Zheng

Tissue Injury/Preservation
Chairs:
Ana J. Coito
Christopher Y. Lu
Pierre-Alain Clavien
Douglas G. Farmer
David K. Imagawa
Milan M. Kinkhabwala
Richard M. Lewis
Hamid Rabb
Daniel Shoskes
Stefan Tullius
Hans-Dieter Volk

Xenotransplantation
Chairs:
Jonathan P. Fryer
Richard N. Pierson
David H. Adams
Amelia M. Bartholomew
David K. C. Cooper
Donald V. Cramer
Duane Davis
Julia I. Greenstein
Joseph R. Leventhal
Jeffrey S. Platt
Daniel R. Salomon
Robert Zhong

2000 – 2001
ASTS Board of Directors

President
Nancy L. Ascher, MD, PhD

President-Elect
Marc I. Lorber, MD

Immediate Past President
Ronald W. Busuttil, MD, PhD

Past President
Joshua Miller, MD

Secretary
Abraham Shaked, MD, PhD (2002)

Treasurer
Richard J. Howard, MD, PhD (2003)

Councilors-at-Large

Anthony M. D'Alessandro, MD (2001)

James A. Schulak, MD (2001)

Arthur J. Matas, MD (2002)

Camillo Ricordi, MD (2002)

David M. Follette, MD (2003)

Goran B. G. Klintmalm, MD, PhD (2003)

Executive Director
Gail D. Durant

Ronald W. Busuttil	1999-2000	Robert J. Corry	1986-1987
Joshua Miller	1998-1999	Anthony Monaco	1985-1986
Ronald M. Ferguson	1997-1998	H.M. Lee	1984-1985
Hans W. Sollinger	1996-1997	Oscar Salvatierra	1983-1984
Nicholas L. Tilney	1995-1996	G. Melville Williams	1982-1983
Mark A. Hardy	1994-1995	Richard L. Simmons	1981-1982
Frank P. Stuart	1993-1994	James Cerilli	1980-1981
Clyde F. Barker	1992-1993	Jermiah G. Turcotte	1979-1980
Arnold G. Diethelm	1991-1992	Frederick K. Merkel	1978-1979
David E. R. Sutherland	1990-1991	John S. Najarian	1977-1978
Barry D. Kahan	1989-1990	Thomas L. Marchioro	1976-1977
J. Wesley Alexander	1988-1989	Folkert O. Belzer	1975-1976
John C. McDonald	1987-1988	Thomas E. Starzl	1974-1975

ASTS Mission Statement

The ASTS and its members are committed to leading the way in the 21st Century in:

1. Fostering and advancing the practice and science of transplantation for the benefit of patients and society.
2. Guiding those who make policy decisions that influence the practice and science of transplantation.
3. Increasing organ donation.
4. Defining and promoting training and the career-long education of transplant surgeons, scientists and physicans.
5. Advancing the professional development and careers of transplant surgeons, scientists and physicians.

2000-2001
ASTS Committee Listing

Term expires at end of annual meeting in year indicated
*Nominations Committee Chair rotates annually to current President

ADVISORY COMMITTEE ON ISSUES

Chairman – Joshua Miller	2003
Benedict Cosimi	2001
William Pfaff	2001
James D. Perkins	2001
Byers W. Shaw, Jr.	2001
Goran Klintmalm	2002
Micahael Abecassis	2003
John McDonald	2003
John Najarian	2003
Robert Corry	2003

BYLAWS

Chairman – Douglas W. Hanto	2001
Mark M. Pescovitz	2001
Wright Pinson	2001
Ron Shapiro	2001
John Colonna	2002
John Brems	2002
John Burke	2002
Albin Gritsch	2003
Randy Bollinger	2003

EDUCATION

Chairman – Charles Miller	2002
Christopher P. Johnson	2001
Christopher Marsh	2001
Jon Ordorico	2001
Ronald M. Pelletier	2001
John F. Valente	2001
Glenn Halff	2002
Eliezer Katz	2002
Milan Kinkhabwala	2002
Lewis Teperman	2002
Craig Smith	2003
Norman Kneteman	2003
Patricia Sheiner	2003
David Reich	2003

*NOMINATIONS

Chairman – Nancy Ascher	2003
Ronald W. Busuttil	2002
Joshua Miller	2001
Anthony D'Alessandro	2001
James A. Schulak	2001
Arthur Matas	2002
Camillo Ricordi	2002

ETHICS

Chairman – Francis Delmonico	2003
Camillo Ricordi	2001
Carlton Young	2001
Donald Dafoe	2002
Scott Gruber	2002
Susan Orloff	2002
John Colonna	2003
Steve Bartlett	2003

INFORMATICS AND DATA MANAGEMENT

Chairman – Robert Merion	2001
Jonathan Paul Fryer	2001
Suzanne T. Ildstad	2001
Henry T. Lau	2001
Arthur J. Matas	2001
Allan M. Roza	2001
James F. Whiting	2001
Chris Shackleton	2002
Jorge Reyes	2003
Lloyd Ratner	2003
Robert Fischer	2003

MEMBERSHIP

Chairman – Douglas Farmer	2003
Richard M. Lewis	2001
Christopher R. Shackleton	2001
R. Patrick Wood	2001
Steve Colquhoun	2002
John Fung	2002
David Laskow	2002
Phil Seu	2002
Curtis Holt	2003
Lewis Teperman	2003

NEWSLETTER

Editor – Michael M. Abecassis	2001
Asst. Editor – Dixon B. Kaufman	2003
Amy L. Friedman	2001
Robert Bruce Love	2001
Bruce G. Sommer	2001
Giacomo Basadonna	2002
Steve Rudich	2002
Donnie Aultman	2003

PROGRAM, PUBLICATIONS & POSTGRADUATE COURSE*

Chairman – J. Richard Thistlethwaite, Jr.	2001
Morton Bolman	2001
Carl E. Haisch	2001
Allan D. Kirk	2001
Raymond Pollak	2001
Jay Markowitz	2002
Mike Millis	2002
Stuart Knechtle	2003
Jonathan S. Bromberg	2003
Peter Stock	2003
Charles Miller	2003
Chris Larsen	2003
John Roberts	2003
Michael M. Abecassis	Advisor
Hugh Auchincloss	Advisor

2000-2001
ASTS Committee Listing (Continued)

SCIENTIFIC STUDIES

Chairman – Paul Kuo	2002
Randall E. Morris	2001
Kenneth Alan Newell	2001
Francis Thomas	2001
Amy Freidman	2002
John Gross	2002
Dilip Kittur	2002
Kim Olthoff	2002
Mark Ghobrial	2003
Michael Shapiro	2003

STANDARDS ON ORGAN PROCUREMENT

Chairman – Jean Emond	2003
Vice Chair – Andy Tzakis	2002
Frederick R. Bentley	2001
James Michael Millis	2001
John M. Rabkin	2001
Eliezeer Katz	2002
Glyn Morgan	2002
Steve Rudich	2002
Richard Lopez	2003
Jeff Reese	2003

THORACIC ORGAN TRANSPLANTATION

Chairman – Robert E. Michler	2001
Sara J. Shumway	2001
David Follette	2002
Bruce Rosengard	2002
Marc Barr	2003
Patricia Thistlethwaite	2003

LOCAL ARRANGEMENTS

Chairman – J. Richard Thistlethwaite, Jr.	2001
Michael M. Abecassis	2001
Alan G. Birtch	2001
Casimir F. Firlit	2001
Dixon Kaufman	2001
Richard M. Lewis	2001
Raymond Pollak	2001
James W. Williams	2001

VANGUARD

Chairman – Kenneth E. Drazan, MD	
Dale Distant	2001
Douglas Farmer	2001
Sandy Feng	2001
Darla Granger	2001
Milan Khinkabwala	2001
Joseph Leventhal	2001
Chris Siegel	2001
Patricia Thistlethwaite	2001

AWARDS COMMITTEE

Chairman – Thomas G. Peters	2003
Vice Chair – Jerzy Kupiec-Weglinski	2003
Allan Kirk	2002
James Wynn	2002
Steve Rudich	2003
Nicholas Stowe	2003
Jim Markman	2003
Paul Grieg	2003
Ron Shapiro	2003
Andre Stieber	2003
Clark Bonham	2003
Timothy Pruett	2003

GOVERNMENT AND SCIENTIFIC LIAISON

Chairman – James Schulak	2003

ACS Board of Governors

Barry D. Kahan

Health Care Finance Administration

Hans Sollinger	2002
Skip Campbell	2003
Bob Gordon	2003
Marlon Levey	2003

National Association of Medical Examiners

Goran B. G. Klintmalm

UNOS

Ronald Ferguson	2001
John Rapkin	2002
Andy Klein	2003

AMERICAN SOCIETY OF TRANSPLANTATION

AST

"Improving Human Life by Advancing the Field of Transplantation"

2000-2001
AST Board of Directors

President
Mohamed H. Sayegh, MD

President-Elect
Laurence A. Turka, MD

Secretary-Treasurer
William E. Harmon, MD

Immediate Past President
John R. Lake, MD

Councilors-at-Large
Gabriel M. Danovitch, MD (2001)
Michael R. Lucey, MD (2001)
E. Steve Woodle, MD (2001)
Mark L. Barr, MD (2002)
Francis L. Delmonico, MD (2002)
Richard N. Fine, MD (2002)
Connie L. Davis, MD (2003)
Jay A. Fishman, MD (2003)
Angus Thomson, PhD DSc, FRCPath (2003)

Executive Director
Susan J. Nelson

Past Presidents

John R. Lake, MD	1999-2000	M. Roy First, MD	1990-1991
John F. Neylan, MD	1998-1999	William E. Braun, MD	1989-1990
J. Harold Helderman, MD	1997-1998	Barry S. Levin, MD	1988-1989
Leslie W. Miller, MD	1996-1997	Lawrence G. Hunsicker, PhD	1987-1988
Douglas J. Norman, MD	1995-1996	Nancy E. Goeken, PhD	1986-1987
Thomas A. Gonwa, MD	1994-1995	Fred Sanfilippo, MD, PhD	1985-1986
Manikkam Suthanthiran, MD	1993-1994	Robert B. Ettenger, MD	1984-1985
Alan R. Hull, MD	1992-1993	Charles B. Carpenter, MD	1983-1984
Ronald H. Kerman, PhD	1991-1992	Ronald D. Guttmann, MD	1982-1983*
		Terry B. Strom, MD	1982-1983**

* *First President*

** *First Past President*

AST Mission Statement

The American Society of Transplantation is an organization of transplant professionals dedicated to research, education, advocacy, and patient care in transplantation science and medicine. Through the work of the AST, the transfer of information from the basic science laboratories to the transplant clinics will ultimately lead to new scientific advances and improvements in patient care.

(Approved by the Board of Directors on March 4, 1998)

2000-2001
AST Committee Listing

Awards Committee

Chair-John Lake	2001
Co-Chair-Mohamed Sayegh	2002
Co-Chair-Laurence Turka	2003
William Bennett	2001
Judith Thomas	2001
Robert Ettenger	2001
Arthur Matas	2001
Peter Heeger	2001
Alan Krensky	2001
Jonathan Bromberg	2001
Robert Rubin	2001
Amir Tejani	2001
T Mohanakumar	2001
Angus Thomson	2001
Maria Rosa Costanzo	2002
Russell Wiesner	2002
Adaani Frost	2002
Marshall Hertz	2002
James Young	2002
Jerzy Kupiec-Weglinski	2003
Abdul Rao	2003
Anthony Jevnikar	2003
Peter Nickerson	2003
Gilles Benichou	2003
Sherry Krams	2003
Richard Mitchell	2003
Nicole Suciu-Foca	2003
Robert Fairchild	2003

Basic Science Committee

Chair-Angus Thomson	2002
Co-Chair-Robert Lechler	2003
Olivia Martinez	2002
Charlie Orosz	2002
Alan Krensky	2002
Wil Burlingham	2002
Eugenia Fedoseyeva	2002
Chris Lu	2002
Jeffrey Platt	2002
Fadi Lakkis	2002
Megan Sykes	2003
Ashwani Khanna	2003
Steve Rose	2003
Judy Thomas	2003
Giles Benichou	2003
Stan Stepkowski	2003
Geraldine Miller	2003
Adriana Zeevi	2003
JW Kupiec-Weglinski	2003
Ana Maria Waaga	2003
David Rothstein	2003
Chaker Adra	2003
Gary Levy	2003
Xin Xiao Zheng	2003

Clinical Practice Committee

Chair-Theodore Steinman	2002
Co-Chair-Wadi Suki	2003
Bryan Becker	2001
Frank Smart	2001
Kim Olthoff	2002
Kenneth Cox	2002
Fuad Shihab	2003
Charles Nolan	2003
John Vella	2003
Steven Alexander	2003

Development Committee

Chair-Amir Tejani	2003
Co-Chair-Sue McDiarmid	2003
Past Chair-Leslie Miller	2001
Ronald Guttmann	2002
John Lake	2003
J. Harold Helderman	2003
Ex Officio-Mohamed Sayegh	
Ex Officio-Laurence Turka	

Education Committee

Chair-Donald Hricik	2002
Co-Chair-Jeffrey Platt	2003
Past Chair-Laurence Turka	2001
Janet Mauer	2002
Harold Helderman	2002
Jay Fishman	2003
Arthur Matas	2002
Keith Bishop	2003
Barbara Murphy	2003
Gabriel Danovtich	2003
Michael Lucey	2003
Floyd Pennington (consultant)	
Norman Skog (consultant)	

Generics Committee

Chair-Steven Takemoto
Co-Chair-Amir Tejani
Roy First
Harold Helderman
Alan Leichtman
Hamid Rabb

Infectious Disease Committee

Chair-Jutta Preiksaitis	2002
Co-Chair-Carlos Paya	2003
Past Chair-Jay Fishman	2001
Susan Keay	2001
Burt Meyers	2001
Carl Perlino	2001
Rosemary Soave	2001
Bernard Kubak	2002
Robin Avery	2002
George Pankey	2002
Connie Davis	2003
Hans Dieter Volk	2003
David Oldach	2003

2000-2001
AST Committee Listing (Continued)

Intrathoracic Organs Committee

Chair-Marshall Hertz	2002
Co-Chair-Joren Madsen	2003
Past Chair-David Taylor	2001
Bruce Rosengard	2001
Edward Garrity	2001
Randall Morris	2001
Frank Smart	2002
Theresa DeMarco	2002
Adaani Frost	2003
Bartley Griffith	2003
Janet Maurer	2003
Guillermo Torre-Amione	2003
Branislav Radovancevic	2003
David DeNofrio	

Kidney Pancreas Committee

Chair-Sundaram Hariharan	2002
Co-Chair-Giacomo Basadonna	2003
Angelo DeMattos	2001
David Conti	2001
Nina Tolkoff-Rubin	2001
Todd Pesavento	2002
Mark Pescovitz	2002
Ginny Bumgardner	2002
Lawrence Chang	2002
John Pirsch	2002
David Klassen	2003
Robert Stratta	2003
Sharon Inokuchi	2003
Martha Pavlakis	2003
Jeffrey Stoff	2003
Ram Peddi	2003
John Odorico	2003
Raja Khauli	2003
Philip O'Connell	2003
Nadey Hakim	2003

Liver & Intra-Abdominal Committee

Chair-Hugo Rosen	2002
Co-Chair-Marwan Abouljoud	2003
Past Chair-Norman Kneteman	2001
David Mulligan	2001
Robert Fontana	2001
Robert Brown,Jr.	2001
Thomas Schiano	2002
Amy Friedman	2002
Kris Kowdley	2002
Rafik Ghobrial	2003
Anthony Post	2003
Vinod Rustigi	2003
Tarek Hassanein	2003
Thomas Fishbein	2003

Membership Committee

Chair-Robert Gish	2002
Co-Chair-David DeNofrio	2003
Past Chair-Venkateswara Rao	2001
Phillip Ruiz	2001
Jorge Rakela	2001
Jon Jones	2001
Mariana Markell	2001
Robert Fisher	2002
Mathew Weir	2002
Ron Shapiro	2002
Maryl Johnson	2002
Sheri Krams	2002
Didier Mandelbrot	2003
Anil Chandraker	2003

Nominating Committee

Chair-John Lake	Past President
Co-Chair-Mohamed Sayegh	President
Laurence Turka	President Elect
Marshall Hertz	2001
Sundaram Hariharan	2001
Hugo Rosen	2001
Steven Webber	2001
Angus Thomson	2001
Amir Tejani	2001
Richard Fine	2001

Organ Donation Committee

Chair-Francis Delmonico	2002
Co-Chair-Edward Alfrey	2003
Thomas Peters	2001
Edward Garrity	2001
Osama Gaber	2001
Brian Pereira	2002
Kareem Abu-Elmagd	2002
Barry Rayburn	2002
Jean Tchervenkov	2002
Roshan Shrestha	2002
William Harmon	2003
Emilio Ramos	2003
Robert Gaston	2003
Todd Pesavento	2003

Patient Care & Education Committee

Chair-Simin Goral	2002
Co-Chair-Thomas McCune	2003
Past Chair-Steven Tomlanovich	2001
Jerry McCauley	2001
Craig Shadur	2001
Carlos Zayas	2001
Mitchell Shiffman	2002
Clyde Yancey	2002
Gabriel Danovtich	2002
Carl Cardella	2003
Roy First	2003
Manual Pascual	2003
James Whiting	2003
Roy Bloom	2003
Michael Germain	2003

2000-2001
AST Committee Listing (Continued)

Pediatric Committee

Chair-Steven Webber	2002
Co-Chair-Ruth McDonald	2003
Past-Chair Amir Tejani	2001
Peter Whitington	2001
Mouin Seikaly	2001
Stuart Kaufman	2001
Phil Rosenthal	2002
Mark Boucek	2002
Jeff Lowell	2002
Minnie Sarwal	2003
Asher Schachter	2003
David Briscoe	2003
Sharon Bartosh	2003
Elias David- Neto	2003
Frederick Kaskel	2003
Stephen Alexander	2003

Practice Guidelines Committee

Chair-Gabriel Danovitch	2002
Co-Chair-John Pirsch	2003
Past Chair-Bertram Kasiske	2001
Brennan McGuire	2001
Peter Whitington	2001
Mandeep Mehra	2001
David Rush	2001
Michael Charlton	2002
John Curtis	2002
Randall Starling	2002
Emilio Ramos	2003
Carl Cardella	2003
William Braun	2003
Sundaram Hariharan	2003
David Roth	2003
Mathew Weir	2003
Chuck Cangro	2003
Richard Formica	2003

Publications Committee

Chair- Robert Rubin	2002
Co-Chair-William Harmon	2003
Past Chair-Mohamed Sayegh	2001
Manikkam Suthanthiran	2001
Amir Tejani	2001
Maria Rosa Costanzo	2001
Hugh Auchincloss	2001
Laurence Turka	2002
John Lake	2003
Mark Barr	2003

Journal Oversight Sub Committee Of the AST Publications Committee

Mohamed Sayegh	2003
John Lake	2003
Mark Barr	2003
William Harmon	2003

Public Policy Committee

Chair-William Harmon	2002
Co-Chair-David Briscoe	2001
David Cohen	2001
Steve Woodle	2003
Jeffrey Crippin	2003
William Bennett	2003
John Lake	2004
Harold Helderman	2004
Francis Delmonico	2004
Peter Whitington	2004
Daniel Brennan	2004
Hector Ventura	2004
Scott Gruber	2004
Hannah Valentine	2004
Ex-Officio-Mohamed Sayegh	
Ex-Officio-Laurence Turka	

Scientific Studies Committee

Chair-Steve Takemoto	2002
Co-Chair Hamid Rabb	2003
Past Chair-Steve Woodle	2001
Greg Gores	2002
Flavio Vincenti	2002
Kenneth Drazen	2002
Bruce Rosengard	2003
Osama Gaber	2003
Lillian Gaber	2003
Gary Levy	2003
Alan Leichtman	2003
Oleh Pankewycz	2003

Training & Manpower Committee

Chair-William Dec	2002
Co-Chair-Venkateswara Rao	2003
Past Chair-Connie Davis	2001
Adaani Frost	2001
David Taylor	2001
Robert Brown, Jr	2001
Bruce Kaplan	2002
Fred Regenstein	2002
Andres Blei	2002
Dale Renlund	2002
Frank Smart	2002
Barry Levin	2002
David Roth	2002
Edward Garitty	2002

Renal Transplant Training Program Certification Subcommittee of the Training & Manpower Committee

Chair-Connie Davis	2002
Co-Chair-Jeffrey Stoff	2003
Douglas Norman	2001
William Couser	2001
Sharon Adler	2001
Flavo Vincenti	2002
John Curtis	2002
Fuad Shihad	2002
Wadi Suki	2003
Donald Hricik	2003

2000-2001
AST Committee Listing (Continued)

Xenotransplantation Committee

Chair-Randall Morris	2002
Co-Chair- David Adams	2003
Past Chair-Jay Fishman	2001
Donald Cramer	2001
Hugh Auchincloss	2001
Claus Hammer	2001
John Fung	2002
Jeffrey Platt	2003

Ad Hoc Women's Health Committee

Chair-Dianne McKay
Co-Chair-Olivia Martinez
Barbara Murphy
Martha Pavlakis
Sheri Krams
Connie Davis
Nina Tolkoff Rubin
Michelle Josephson
Ginny Bumgardner

Ad Hoc Islet Transplant Committee

AST Chair- Hugh Auchincloss
ASTS Chair-Camillo Riccordi
Giacomo Basadonna
Osama Gaber
Bernard Hering
Dixon Kaufman
Martha Pavlakis
James Shapiro
Craig Smith
J. Richard Thistlethwaite, Jr.

Ad Hoc Conflict of Interest Policy Committee

Chair-Jay Fishman
Co-Chair-William Harmon
Mark Barr
Ronald Guttmann
Richard Fine

Ad Hoc Reorganization Committee

Chair- Laurence Turka
Connie Davis
Richard Fine
Michael Lucey

Domestic Young Investigator Awards

Geetha Chalasani
MEMORY BUT NOT NAIVE T CELLS REJECT VASCULARIZED CARDIAC ALLOGRAFTS IN THE ABSENCE OF SECONDARY LYMPHOID ORGANS. (Abstract #1)
Sunday, May 13, 2001, 8:00 AM
Sheraton Ballroom 1-3, Sheraton

Bibo Ke
HEME OXYGENASE-1 GENE TRANSFER PREVENTS FAS/FAS LIGAND-INDUCED APOPTOSIS IN VITRO AND IMPROVES ALLOGRAFT FUNCTION IN VIVO. (Abstract #2)
Sunday, May 13, 2001, 8:15 AM
Sheraton Ballroom 1-3, Sheraton

Nader Najafian
REGULATORY ROLE OF CTLA4 AND CYTOKINES IN PHYSIOLOGIC TERMINATION OF ALLOIMMUNE RESPONSES IN VIVO. (Abstract #3)
Sunday, May 13, 2001, 8:30 AM
Sheraton Ballroom 1-3, Sheraton

J. Stuart Wolf, Jr.
RANDOMIZED CONTROLLED TRIAL OF HAND-ASSISTED LAPAROSCOPIC VERSUS OPEN SURGICAL LIVE DONOR NEPHRECTOMY. (Abstract #6)
Sunday, May 13, 2001, 8:45 AM
Sheraton Chicago Ballroom 4-7, Sheraton

Masayuki Sho
NEW INSIGHTS INTO THE INTERACTION BETWEEN T CELL COSTIMULATION BLOCKADE AND CONVENTIONAL IMMUNOSUPPRESSION IN VIVO. (Abstract #29)
Sunday, May 13, 2001, 2:20 PM
Chicago Ballroom 8, Sheraton

Katsuhito Teranishi
INFUSION OF GAL TYPE 6 OLIGOSACCHARIDES IN BABOONS RESULTS IN TOTAL DEPLETION OF ANTI-PIG ANTIBODIES. (Abstract #65)
Sunday, May 13, 2001, 2:20 PM
Empire Room, Intercontinental

Rolf N. Barth
XENOGENEIC THYMOKIDNEY TRANSPLANTATION IN A PIG-TO-BABOON MODEL: EVIDENCE OF SPECIFIC T CELL UNRESPONSIVENESS. (Abstract #70)
Sunday, May 13, 2001, 3:10 PM
Empire Room, Intercontinental

Vera S. Donnenberg
UPREGULATION OF P-GP PROTECTS GRAFT INFILTRATING T-CELLS FROM APOPTOSIS: A MECHANISM FOR IMMUNOSUPPRESSIVE DRUG RESISTANCE. (Abstract #76)
Sunday, May 13, 2001, 2:40 PM
Exchange Room, Intercontinental

Kerrie L. Faia
PROTEASOME TARGETING IN TRANSPLANTATION. (Abstract #118)
Sunday, May 13, 2001, 4:10 PM
Chicago Ballroom 8, Sheraton

Benjamin D Ehst
ACUTE REJECTION OF A SKIN GRAFT EXPRESSING A DEFINED MINOR ANTIGEN. (Abstract #126)
Sunday, May 13, 2001, 4:00 PM
Chicago Ballroom 9, Sheraton

Liqing Wang
ORGAN-SPECIFIC EFFECTS OF TARGETING NF-κB TO REDUCE ISCHEMIA/REPERFUSION INJURY. (Abstract #153)
Sunday, May 13, 2001, 4:00 PM
Empire Room, Intercontinental

R. Sreekumar
DISTINCTION OF ACUTE CELLULAR REJECTION FROM RECURRENCE OF HCV THROUGH INTRAGRAFT GENE EXPRESSION PATTERNS. (Abstract #172)
Sunday, May 13, 2001, 4:10 PM
Grand Ballroom, Intercontinental

Fulung Luan
SIROLIMUS PREVENTS TUMOR PROGRESSION: mTOR TARGETING FOR THE INHIBITION OF NEOPLASTIC PROGRESSION. (Abstract #428)
Monday, May 14, 2001, 10:30 AM
Sheraton Chicago Ballroom 4-7, Sheraton

Engin Ozkaynak
ICOS/B7RP-1 COSTIMULATION IN ACUTE AND CHRONIC ALLOGRAFT REJECTION. (Abstract #430)
Monday, May 14, 2001, 11:00 AM
Sheraton Chicago Ballroom 4-7, Sheraton

Thiagarjan Ramcharan
LIVING KIDNEY DONATION: LONG-TERM (20-37 YRS) CONSEQUENCES. (Abstract #450)
Monday, May 14, 2001, 2:00 PM
Chicago Ballroom 8/9, Sheraton

Domestic Young Investigator Awards (Continued)

Michael V. Autieri
ENHANCED VASCULAR SMOOTH MUSCLE CELL EXPRESSION OF EARLY GROWTH RESPONSE FACTOR-1 AS A RESULT OF ACUTE CELLULAR REJECTION AND GRAFT VASCULOPATHY IN CARDIAC TRANSPLANT RECIPIENTS. (Abstract #492)
Monday, May 14, 2001, 2:30 PM
Exchange Room, Intercontinental

Michael J. Goldstein
ANALYSIS OF FAILURE IN LIVING DONOR LIVER TRANSPLANTATION (LRT): DIFFERENTIAL OUTCOMES IN CHILDREN AND ADULTS. (Abstract #498)
Monday, May 14, 2001, 2:00 PM
Grand Ballroom, Intercontinental

Richard S. Lee
THE ROLE OF DONOR ANTIGEN AND CD28-B7 T CELL COSTIMULATORY BLOCKADE IN A CLINICALLY RELEVANT LARGE ANIMAL MODEL OF CARDIAC TRANSPLANTATION. (Abstract #508)
Monday, May 14, 2001, 2:10 PM
King Arthur Court Ballroom, Intercontinental

Akira Yamada
ACTIVE REGULATION OF ALLOGRAFT REJECTION THROUGH THE INDIRECT PATHWAY. (Abstract #510)
Monday, May 14, 2001, 2:30 PM
King Arthur Court Ballroom, Intercontinental

J. Lee
EVIDENCE FOR A HUMAN CD4+ T REGULATORY CELL SPECIFIC FOR AN ALLO-PEPTIDE DERIVED FROM DONOR HLA CLASS I. (Abstract #514)
Monday, May 14, 2001, 3:10 PM
King Arthur Court Ballroom, Intercontinental

Wei Gao
INHIBITION OF IκB KINASE (IKK) IN TRANSPLANTATION. (Abstract #531)
Monday, May 14, 2001, 5:00 PM
Chicago Ballroom 10, Sheraton

Dmitry V. Samsonov
INTERACTIONS AMONG RECIPIENT MONOCYTES AND DONOR ENDOTHELIUM IN THE MAINTENANCE OF THE INDIRECT PATHWAY OF ALLORECOGNITION. (Abstract #858)
Tuesday, May 15, 2001, 2:10 PM
Chicago Ballroom 10, Sheraton

Peta J. O'Connell
$CD8\alpha^+$ (*LYMPHOID-RELATED*) AND $CD8\alpha^-$ (*MYELOID*) DENDRITIC CELL SUBSETS DIFFERENTIALLY REGULATE ORGAN ALLOGRAFT SURVIVAL. (Abstract #860)
Tuesday, May 15, 2001, 2:30 PM
Chicago Ballroom 10, Sheraton

Ye Feng
CRITICAL ROLE OF CD103 IN PROMOTING ALLOGRAFT DESTRUCTION BY CD8+ T CELLS. (Abstract #909)
Tuesday, May 15, 2001, 3:10 PM
Empire Room, Intercontinental

P.A. Pappas
THE USE OF SERUM CITRULLINE AS A MARKER OF ACUTE CELLULAR REJECTION IN ISOLATED SMALL BOWEL TRANSPLANTATION. (Abstract #917)
Tuesday, May 15, 2001, 3:00 PM
Exchange Room, Intercontinental

Eugenia V. Fedoseyeva
CROSSREACTIVE TH1 RESPONSES TO DONOR MHC PEPTIDE AND CARDIAC MYOSIN ARE PRESENT DURING CHRONIC REJECTION OF HEART ALLOGRAFTS. (Abstract #948)
Tuesday, May 15, 2001, 4:00 PM
Chicago Ballroom 10, Sheraton

Jennifer Trofe
PTLD IN KIDNEY TRANSPLANTATION: A 32 YEAR EXPERIENCE IN 405 PATIENTS FROM THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY. (Abstract #957)
Tuesday, May 15, 2001, 4:00 PM
Chicago Ballroom 6/7, Sheraton

M.J. Aull
LONG-TERM GRAFT FUNCTION IN RENAL TRANSPLANTATION USING NON-HEART-BEATING DONORS. (Abstract #966)
Tuesday, May 15, 2001, 4:00 PM
Chicago Ballroom 8/9, Sheraton

Linlin Ma
TREATMENT WITH ANTISENSE OLIGODEOXYRIBONUCLEOTIDES TARGETING FAS mRNA ATTENUATES RENAL ISCHEMIA-REPERFUSION INJURY. (Abstract #1020)
Tuesday, May 15, 2001, 4:00 PM
King Arthur Court Ballroom, Intercontinental

Domestic Young Investigator Awards (Continued)

Guilherme Costa
LIVER TRANSPLANTATION OF HIV POSITIVE PATIENTS IN THE ERA OF HIGHLY ACTIVE ANTIRETROVIRAL THERAPY (HAART). (Abstract #1029)
Tuesday, May 15, 2001, 4:00 PM
Renaissance Ballroom, Intercontinental

Shizhong Chen
THE IDENTIFICATION OF A G-PROTEIN-COUPLED RECEPTOR EDG-6 AS A TARGET OF FTY720, A NOVEL TRANSPLANTATION DRUG. (Abstract #1278)
Wednesday, May 16, 2001, 8:15 AM
Sheraton Ballroom 1-3, Sheraton

Marlies E.J. Reinders
CD40 AND ANGIOGENESIS: A LINK BETWEEN ALLOIMMUNE RESPONSES AND NON-IMMUNE MECHANISMS OF ALLOGRAFT REJECTION? (Abstract #1280)
Wednesday, May 16, 2001, 8:45 AM
Sheraton Ballroom 1-3, Sheraton

Xiu-Da Shen
THE CD40 LIGAND AND CD28 T CELL COSTIMULATION PATHWAYS ARE REQUIRED FOR NON-ANTIGENIC WARM ISCHEMIA/REPERFUSION INJURY IN MOUSE LIVER MODEL. (Abstract #1282)
Wednesday, May 16, 2001, 9:15 AM
Sheraton Ballroom 1-3, Sheraton

Anirban Bose
FAILURE TO INDUCE ALLOGRAFT ACCEPTANCE IN PERFORIN-DEFICIENT MICE. (Abstract #1306)
Wednesday, May 16, 2001, 11:00 AM
Chicago Ballroom 8, Sheraton

Daniel A. Falco
IDENTIFICATION OF EBV-SPECIFIC CD8+ T CELLS USING MHC/PEPTIDE TETRAMERS IN PEDIATRIC TRANSPLANT RECIPIENTS. (Abstract #1320)
Wednesday, May 16, 2001, 11:50 AM
Chicago Ballroom 9, Sheraton

R.P. Pelletier
DONOR DOWN-REGULATED T CELL RESPONSES IN TRANSPLANT PATIENTS. (Abstract #1322)
Wednesday, May 16, 2001, 12:10 PM
Chicago Ballroom 9, Sheraton

International Young Investigator Awards

Eric Bedard
SERPIN REGULATION BY A MYXOMA VIRUS ENCODED IMMUNOREGULATORY PROTEIN PREVENTS CHRONIC REJECTION IN RAT RENAL ALLOGRAFTS. (Abstract #436)
Monday, May 14, 2001, 2:40 PM
Chicago Ballroom 10, Sheraton

Peter J.H. Smak Gregoor
A PROSPECTIVE, RANDOMISED STUDY OF WITHDRAWAL OF CYCLOSPORINE OR PREDNISONE IN RENAL TRANSPLANT RECIPIENTS TREATED WITH MYCOPHENOLATE MOFETIL, CYCLOSPORINE, AND PREDNISONE: 18 MONTHS FOLLOW-UP DATA. (Abstract #441)
Monday, May 14, 2001, 2:00 PM
Chicago Ballroom 6/7, Sheraton

Philipp v Breitenbuch
RAPAMYCIN INHIBITS TUMOR GROWTH AND METASTASIS IN MICE BY ANTIANGIOGENESIS. (Abstract #459)
Monday, May 14, 2001, 2:00 PM,
Sheraton Ballroom 1-3, Sheraton

Richard N. Saunders
RAPAMYCIN INCREASES GLOMERULAR PROFIBROTIC GENE EXPRESSION AFTER CYCLOSPORIN DOSE REDUCTION IN PATIENTS WITH CHRONIC ALLOGRAFT NEPHROPATHY. (Abstract #477)
Monday, May 14, 2001, 3:20 PM
Sheraton Ballroom 4/5, Sheraton

Carla C. Baan
BLOCKADE OF THE IL-2Rα-CHAIN AFFECTS THE DEATH SIGNALS OF GRAFT INFILTRATING LYMPHOCYTES AFTER CLINICAL HEART TRANSPLANTATION. (Abstract #489)
Monday, May 14, 2001, 2:00 PM
Exchange Room, Intercontinental

Hendrik Jan Ankersmit
AUGMENTED T CELL APOPTOSIS IN VIVO VIA CD95 AND TNF R1 AND ACTIVATION INDUCED T-CELL DEATH BY ANTI-THYMOCYTE ANTIBODY TREATMENT OF STABLE CARDIAC TRANSPLANT RECIPIENTS. (Abstract #491)
Monday, May 14, 2001, 2:20 PM
Exchange Room, Intercontinental

Natalia Shulzhenko
CD27 INTRAGRAFT GENE EXPRESSION IN REJECTING AND NON REJECTING CARDIAC ALLOGRAFT RECIPIENTS. (Abstract #496)
Monday, May 14, 2001, 3:10 PM
Exchange Room, Intercontinental

Acar Tuzuner
QUANTITATIVE (TAQMAN™) PCR FOR BK VIRUS AND CIDOFOVIR THERAPY: ROLE IN MANAGEMENT OF BKV INDUCED RENAL ALLOGRAFT DYSFUNCTION. (Abstract #537)
Monday, May 14, 2001, 4:30 PM
Chicago Ballroom 6/7, Sheraton

Florian Kern
ANALYSIS OF CD8 AND CD4 T-CELL REACTIVITY TO CYTOMEGALOVIRUS - MEASURING REACTIVITY AT THE TOTAL PROTEIN AND EPITOPE LEVEL. (Abstract #1006)
Tuesday, May 15, 2001, 4:40 PM
Exchange Room, Intercontinental

Michitaka Ozaki
ADENOVIRALLY OVER-EXPRESSED REDOX FACTOR-1(REF-1) PROTECTS AGAINST POST-ISCHEMIC LIVER INJURY BY REDUCING OXIDATIVE STRESS AND NF-kB DNA BINDING ACTIVITY. (Abstract #1279)
Wednesday, May 16, 2001, 8:00 AM,
Sheraton Ballroom 1-3, Sheraton

Nina Babel
EVIDENCE FOR GENETIC SUSCEPTIBILITY TOWARDS DEVELOPMENT OF POST TRANSPLANT LYMPHOPROLIFERATIVE DISORDER (PTLD) IN SOLID ORGAN TRANSPLANT PATIENTS. (Abstract #1283)
Wednesday, May 16, 2001, 8:30AM
Sheraton Chicago Ballroom 4-7, Sheraton

Torsten Boehler
FTY720 MEDIATES REVERSIBLE REDUCTION OF LYMPHOCYTE COUNTS IN HUMAN RENAL ALLOGRAFT RECIPIENTS – EVIDENCE FOR ALTERED LYMPHOCYTE TRAFFICKING FOR THE MECHANISM OF ACTION OF FTY720. (Abstract #1334)
Wednesday, May 16, 2001, 11:00 AM
Sheraton Ballroom 4/5, Sheraton

AST 2001 Grants and Awards

The following AST grants and awards will be announced and presented on Monday, May 15th at 11:30 AM following the Joint Plenary Session in Sheraton Chicago Ballroom 4-7, Sheraton Hotel

Fujisawa Career Basic Science Award
Fujisawa Career Clinical Science Award
Novartis Established Investigator Basic Science Award
Novartis Established Investigator Clinical Science Award
Wyeth-Ayerst Basic Science Young Investigator Award
Wyeth-Ayerst Clinical Science Young Investigator Award

AST Basic Science Fellowship Grant
AST Council's Fellowship Grant
AST/Novartis Fellowship Grant
AST/Sangstat Fellowship Grant

AST Clinical Fellowship Grants
AST/Fujisawa Fellowship Grant
AST President's Fellowship Grant

AST Faculty Grants
AST Council's Faculty Grant
AST Faculty Grant
AST/Roche Faculty Grant

AST Special Fellowship Grants
AST/JDFI Fellowship Grant

AST Special Faculty Grants
AST Basic Scientist Faculty Grant
Women's & Minority Faculty Grant

ASTS 2001 Awards

The following ASTS awards will be announced and presented on Tuesday, May 15th at 11:10 AM following the ASTS and AST Presidential Addresses in Sheraton Chicago Ballroom 4-7, Sheraton Hotel

Fujisawa, USA Faculty Development Award
Folkert Belzer, MD Research Fellowship of the ASTS and National Kidney Foundation
Roche Laboratories Scientist Scholarship
Roche Presidential Travel Award
ASTS-Novartis Fellowship in Transplantation
ASTS Mid Level Faculty Research Award
ASTS Thoracic Surgery Fellowship
ASTS Collaborative Scientist Research Award

ASTS & AST SPECIAL ACHIEVEMENT AWARDS

The following AST awards will be announced and presented on Tuesday, May 15th at 11:40 AM in the Sheraton Chicago Ballroom 4-7, Sheraton Hotel

ASTS Roche Pioneer Award
AST Roche Distinguished Achievement Award

TRANSPLANT 2001
Exhibitor Product Descriptions

ABBA International

261 Park Lane
King of Prussia, PA 19406 Booth #307

International market research and consulting firm specializing in the healthcare market

Abbott Laboratories – Hospital Products Division

200 Abbott Park Road, D-97J, AP30
Abbott Park, IL 60064-6154 Booth #410

Hextend® (6% Hetastarch in Lactated Electrolyte Injection) is a synthetic colloidal solution, pharmacologically classified as a plasma volume expander, and is intended to support oncotic pressure as well as provide electrolytes. Hextend® is a safe, economical alternative to albumin, and is available in 500 ml containers for regular or pressure infusion.

Abbott Laboratories International

200 Abbott Park Road, Dept. 304/AP30
Abbott Park, IL 60064-3537 Booth #917

You are invited to visit the Abbott Laboratories exhibit where representatives will be on hand to answer your questions regarding Gengraf (cyclosporine capsules, USP).

American Biosystems, Inc.

1020 West County Road F
St. Paul, MN 55126 Booth #417

The ABI Vest® Airway Clearance System is a portable device which provides airway clearance therapy using high-frequency chest wall oscillation. Used as an alternative to conventional CPT for more than 11 years, the Vest has been prescribed by more than 1,300 physicians and is effective in most patient populations requiring airway clearance.

American Liver Foundation

75 Maiden Lane, Suite 603
New York, NY 10038 Booth #414

The *American Liver Foundation* is a nonprofit, national voluntary health organization dedicated to the prevention, treatment, and cure of hepatitis and other liver diseases through research, education, and advocacy on behalf of those affected by or at risk of liver disease

Athena Diagnostics, Inc.

Four Biotech Park
377 Plantation Street
Worcester, MA 01605 Booth #915

Athena Diagnostics introduces the PKDx Molecular Diagnostic Testing Services, the first molecular diagnostic assay to help in the diagnosis of Autosomal Dominant Polycystic Kidney Disease. The PKDx Molecular Diagnostic Testing Service is appropriate to a wide range of patients including live related donors.

Automated Medical Products Corporation

PO Box 2508
Edison, NJ 08818 Booth #612 & 614

The leader in mechanical retraction systems for many types of surgery. We develop, manufacture, and internationally distribute our **Iron Intern®** line of mechanical arms and accessories.

Aventis Pharmaceuticals

399 Interpace Parkway
Parsippany, NJ 07054 Booth #522 & 524

You are cordially invited to visit the Aventis Pharmaceuticals booth, which will feature Synercid® I.V. (quinupristin-dalfopristin for Injection.) Information on educational materials and other resources will be available.

Axcan Scandipharm, Inc.

22 Iverness CenterParkway, #310
Birmingham, AL 35242 Booth #906

Broadening the scope of research in liver disease and Marketers of URSO® 250.

Axis Clinical Software, Inc.

5201 SW Westgate Drive, Suite 216
Portland, OR 97221-2425 Booth #816

PATS for Windows is a sophisticated clinical outcomes information system designed to empower every clinical user. *PATS* can accommodate all organs and is optimized for speed in longitudinal analysis and reporting of user defined clinical and financial data. *PATS* facilitates UNOS UNet™ reporting, clinical research, recipient tracking, accreditation, risk stratification and national database participation.

Baptist Health

904 Autumn Road, Suite 500
Little Rock, AR 72211 Booth #311

Baptist Health is the largest healthcare system in Arkansas, consisting of five owned hospitals and eight affiliate hospitals. Baptist Health also owns over 30 primary care clinics throughout Arkansas.

Barr Laboratories

2 Quaker Road
Pomona, NY 10970 Booth #516

Barr Laboratories is a specialty pharmaceutical company engaged in the development, manufacture and marketing of generic and proprietary pharmaceuticals, and distributor of ViaSpan (Belzer's UW) — the organ preservation solution.

Cincinnati Transplant Institute

1615 Monte Diablo Avenue
San Mateo, CA 94401 Booth #615 & 617

The Cincinnati Transplant Institute is a transplant service company dedicated to developing therapeutic and diagnostic solutions to transplant patients, caregivers, and centers as well as industry and government affairs surrounding transplant. Areas of focus include clinical research, education, laboratory testing, and practice management.

Circe Biomedical, Inc.

99 Hayden Avenue
Lexington, MA 02421 Booth #416

The company's lead product, the HepatAssist® Liver Support System, is an investigational system designed to bridge acute liver failure patients to recovery, or transplantation. The HepatAssist System is an extracorporeal system intended to temporarily provide essential liver functions. It has progressed further in controlled clinical trials than any other liver support system being evaluated today. The system combines Circe's core technologies of isolation, purification, and cryopreservation of primary mammalian cells, and fabrication of membrane materials, with pore sizes permitting the selective passage of biomolecules. These technologies permit the integration of isolated cells, and membranes into biomedical systems. Following plasmapheresis, patient plasma is circulated through the HepatAssist circuit and bioreactor. There, it passes through hollow fiber membranes and interacts with the surrounding live porcine hepatocytes. Patients are currently being enrolled in phase II/III multi-center, randomized and controlled, pivotal trials for fulminant hepatic failure, and primary graft non-function, at 19 sites in the USA and Europe.

Clinimetrics Research Associates, Inc.

5285 Hellyer Avenue
San Jose, CA 95138 Booth #814

Founded in 1988, Clinimetrics Research Associates is a full service contract research organization (CRO) that offers a complete array of integrated research services including clinical trial management, project management, data management and biostatistics as well as compliance auditing services. Clinimetrics serves an international clientele of pharmaceutical and biotechnology companies.

Coalition on Donation

1100 Boulders Parkway, Suite 700
Richmond, VA 23225-8770 Booth #514

The Coalition on Donation is a not-for-profit alliance of national organizations and local coalitions across the United States that have joined forces to educate the public about organ and tissue donation, correcting misconceptions about donation and creating a greater willingness to donate. The Coalition directs the National Campaign for Organ and Tissue Donation, a public education effort that has received more than $270 million in donated media advertising since 1994.

Cubist Pharmaceuticals

24 Emily Street
Cambridge, MA 02139 Booth #712-714

Cubist Pharmaceuticals is focused on becoming a global leader in the research, development and commercialization of novel antimicrobial drugs to combat serious and life-threatening bacterial and fungal infections. Cubist is evaluating the safety and efficacy of daptomycin in the EDGE™ (*Evaluation of Daptomycin in Gram-positive Entities*) clinical trial program. Currently, the Company is enrolling patients in three international Phase III trials in skin & soft tissue, pneumonia and urinary tract infections.

Cylex Inc.

8980 Old Annapolis Road, Suite I
Columbia, MD 21045 Booth #415

Cylex Inc. has developed and patented *in vitro* CMI™ technology for the rapid assessment of immune function in whole blood. The principle of the test depends upon the activation of intracellular ATP in lymphocytes in response to foreign stimulation. This activation is accomplished following incubation with allogeneically mismatched cells, mitogens, or antigens for 24 hours or less. After incubation, CD4+ or CD8+ cells are selected using magnetic particles, washed, and lysed to release intracellular ATP. The ATP is then detected using luciferin/luciferase to produce a chemiluminescent signal. Pre-clinical data demonstrates that the phytohemagglutinin (PHA)-stimulated response in lymphocytes is dramatically suppressed in transplant recipients receiving immunosuppressive therapy relative to their untreated counterparts.
The potential implications of this technology for the management of the transplant patient include monitoring the bioactivity of immunosuppressive drugs, identifying early rejection events, and assessing the susceptibility to infections. Donor compatibility may also be determined in a mixed lymphocyte format.
In vitro CMI™ technology has applications beyond transplantation to monitoring the pathogenesis of disease in HIV patients, including their response to therapy. The technology has value in assessing immune function in patients with other infectious diseases (like HCV), cancer, and autoimmune conditions. The efficacy of vaccines and new therapeutic drugs can also be determined *in vitro* using this methodology. Compared to existing research methodologies, *in vitro* CMI™ technology offers a rapid method for the assessment of cell-mediated immune function.

Designs For Vision, Inc.

760 Koehler Avenue
Ronkon Koma, NY 11779 Booth #812

Custom fit surgical telescopes (loupes) w/true magnification powers of 2x5, 3x5, 4x5 and 6.0x. Gallelian (std fld) and Prismatic (exp fld) models. Surgical fiberoptic illumination w/ surgeons choice of 3 fiberoptic light source, including xenon with frame mounted or headset options.

Dialysis & Transplantation

7628 Densmore Avenue
Van Nuys, CA 91406 Booth #421

Dialysis & Transplantation is celebrating its 28th year of distinguished service to the renal care community. Among nephrology journals, *D&T* has the largest multidisciplinary audience in the U.S.A. and is read in more than 120 foreign countries. *D&T* provides peer-reviewed articles that have specific clinical application on topics of most interest to renal care practitioners.

Dr. Franz Köhler Chemie GmbH

Neue Bergstrasse 3-7
Alsbach-Haehnlein, D-64665
Germany Booth #716

Dr. Franz Köhler Chemie GmbH is an independent, family owned business with representations on all five continents. DFKC has an establishment of over 40 years as high quality pharmaceutical manufacturer of various entities. DFKC provides products in the field of electrolytes, contrast media, antidotes, anaesthetics compounds as well as organ protective and cardioplegic solutions. Particularly the latter ones provide protection of ischemic organs from donor to recipient and their use have extended the possibilities to grant new opportunity to otherwise fatally ill patients. Although the companies primary businesses are cardiac surgery and perfusion therapy, antidotes as well provide tools to effectively save life in case of e.g. industrial disasters, whilst the available ionic- as well as nonionic contrast media are equally efficient diagnostics.

Elsevier Science

655 Avenue of the Amercias
New York, NY 10010 Booth #809

TRANSPLANTATION PROCEEDINGS, published by ELSEVIER SCIENCE, is an official publication of The Transplantation Society and more than 20 other transplant associations. *THE JOURNAL OF HEART AND LUNG TRANSPLANTATION* is the official journal of The International Society for Heart and Lung Transplantation. All manuscripts undergo extensive peer-review by recognized authorities in the field. Stop by our booth for **FREE** sample copies and a special meeting discount!

Eon Labs

227-15 N. Conduit Avenue
Laurelton, NY 11413 Booth #412

Almost 9 out of 10 patients surveyed prefer Eon Labs' AB Rated Cyclosporine Soft Gelatin Capsules, USP (Modified) over the other generic. Eon's Cyclosporine is available in 25mg and 100 mg softgel capsules...the dosage that's closer to the brand name drug to which transplant patients are accustomed. Patients also appreciate the user-friendly packaging that can be opened without scissors.

Fujisawa Healthcare, Inc.

Three Parkway North
Deerfield, IL 60015 Booth #707

Fujisawa Healthcare, Inc. will have representatives available to discuss Prograf® (tacrolimus), indicated for the prophylaxis of organ rejection in patients receiving allogeneic liver and kidney transplants. Prograf® is now also available in 0.5mg capsules.

HemoTherapies, Inc.

11975 El Camino Real, #104
San Diego, CA 92130 Booth #520

The HemoTherapies Unit is the only FDA-cleared Liver Dialysis™ technology for acute hepatic encephalopathy due to acute-on-chronic or fulminant hepatic failure, or severe drug overdose. This toxin removal device effectively removes drugs and toxins up to 5,000 daltons that are dialyzable and bound to charcoal (e.g. acetaminophen, tricyclics, barbiturates).

Hickman-Kenyon Systems

11011 Q Street, Suite 105B
Omaha, NE 68137 Booth #613

Hickman-Kenyon Systems began developing patient tracking software in 1993, and is the industry leader in organ transplant tracking systems. OTTR Patient Tracking Software provides a flexible, long-term, patient-centered approach to coordinating the activities and communications of specialized areas of medicine. OTTR Software runs on Windows NT and Windows 95, 98 and 2000, and can interface with existing systems to receive laboratory results, textual reports, and ADT data automatically.
OTTR is highly configurable and HKS personnel work closely with physicians and health care professionals to satisfy the specific needs of each program.

IMPRA, Inc., a subsidiary of C.R. Bard, Inc.

1625 W. 3rd St.
PO Box 1740
Tempe, AZ 85280 Booth #309

We design ePTFE vascular and endovascular grafts, knitted and woven polyester grafts, patches and fabrics, felts and pledgets, carotid shunts, vascular probes, tapes, pouches, and accessories to meet the unique needs of clinicians and patients for peripheral, abdominal thoracic, and AV access procedures. Vascular and endovascular workshops, educational library, and "Stick With Us" workshop complement our offerings.

International Liver Transplantation Society

17000 Commerce Parkway, Suite C
Mount Laurel, NJ 08054 Entrance Foyer

International Liver Transplantation Society is a nonprofit, multi-disciplinary society promoting education and research to raise the standard of care for liver transplantation patients. Stop by the ILTS booth for a complimentary issue *Liver Transplantation* and membership information.

Israel Penn International Transplant Tumor Registry

University of Cincinnati
231 Albert Sabin Way, ML 0558
Cincinnati, OH 45267-0558 Booth #618

The Israel Penn International Transplant Tumor Registry (IPITTR) was established in 1967 by Dr. Israel Penn and includes data on 13,000 transplant recipients witH cancer. It remains the largest repository of data on transplant malignancies, and provides a valuable resource to the international transplant community for scientific studies and patient consultations.

The Liposome Company, Inc.

One Research Way
Princeton, NJ 08540 Booth #525

The Lipososme Co., Inc. a subsidiary of Elan Corporation, plc, markets ABELCET® (Amphotericin B Lipid Complex Injection), indicated for treatment of invasive fungal infections in patients refractory to or intolerant of conventional amphotericin B.

Lippincott Williams & Wilkins

530 Walnut Street
Philadelphia, PA 19106 Booth #813

Lippincott Williams & Wilkins is a global publisher of medical, nursing and allied health information resources in books, journals, newsletters, and electronic media formats. Please stop by booth # 813 to review one of the many titles that we have available on display.

Medical Insight Research

25 Burlington Mall Road
Burlington, MA 01803 Booth #719

Medical Insight Research is a market research firm specializing in the health care and pharmaceutical industry. We perform economic and market research in these areas.

Medical Media Systems (MMS)

66 Benning Street
West Lebanon, NH 03784 Booth #815

Medical Media Systems (MMS), an advanced medical imaging company, provides accurate 3D visualization and calculation tools of patient specific anatomy. Our 3D modeling service and software package, Preview 2.0 enables precise calculation of relevant distances, diameters, and volumes. Transplant surgeons use our 3D models for complex treatment planning and post-operative analysis on a case-by-case basis. An ISO certified company, MMS is FDA approved in all clinical applications.

MedImmune, Inc.

35 W. Watkins Mill Road
Gaithersburg, MD 20878 Booth #621

MedImmune, Inc. is a fully integrated biotechnology company focused on developing and marketing products that address medical needs in areas such as infectious disease, transplantation medicine, autoimmune disorders and cancer. Headquartered in Gaithersburg, Maryland, MedImmune has manufacturing facilities in Frederick, Maryland and Nijmegen, The Netherlands.

Medsite, Inc.

15355 Vantage Pkwy. West, Suite 195
Houston, TX 77032 Booth #807

Interactive Grand Rounds in Immunology (www.ImmunologyEd.com) is an educational site featuring original case studies in the management of transplant patients. Written by experts in transplant medicine, cases are designated for category 1 CME credit, and can be accessed and completed free of charge. No registration or subscription is required.

Munksgaard International Publishers

35 Noore Sogade, PO Box 2148
Copenhagen DK 1016K
Denmark Booth #513

Munksgaard International Publishers is proud to launch the *American Journal of Transplantation*, the official journal of the AST and the ASTS. Visit Munksgaard's booth number 513 and pick up a free sample copy. Other transplantation and immunology titles on display are: *Clinical Transplantation, Xenotransplantation, Pediatric Transplantation, Transplant Infectious Disease, Immunological Reviews, Reviews in Immunogenetics, Liver, European Journal of Haematology, Traffic*, and other related titles. Book on display: *Pediatric Solid Organ Transplantation.*

Munksgaard/Blackwell Science

350 Main Street
Malden, MA 02149 Booth #515

Blackwell Science and Munksgaard International proudly publish leading journals and books in Transplantation. Please visit our booth for complimentary copies of our journals *Aritificial Organs, Clinical Transplantation, Transplant Infectious Disease, and Xenotransplantation.* Also on display our newest title *Therapeutic Immunology,* Austen, et al., and *Hematopoietic Cell Transplantation,* Thomas et al., *Transplantation,* Ginns et al. and *Immunosupression in Transplantation,* Ginns et al.

National Foundation for Transplants

1102 Brookfield, Suite 200
Memphis, TN 38119 Booth #315

NFT assists solid organ and bone marrow transplant patients nationwide in raising funds for the transplant-related expenses and medications that are not covered by insurance.

National Kidney Foundation

30 East 33rd Street
New York City, NY 10016 Booth #313

The National Kidney Foundation (NKF) is the leading voluntary health agency dedicated to preventing kidney and urinary tract diseases, improving the health of individuals and families affected by these diseases and increasing the availability of organs and tissue for transplantation. It conducts programs in research, public and professional education, patient and community services, advocacy and organ and tissue donation.

National Kidney Foundation Singapore

81 Kim Keat Road
Singapore 328836 Booth #519-521

The National Kidney Foundation Singapore (NKFS) together with our world-renowned partners, the American Society of Nephrology (ASN) and the American Nephrology Nurses Association (ANNA), is proud to present NephroAsia 2001 in Singapore from June 14-14, 2001.
A first in Asia – NephroAsia 2001 – Asia's premier nephrology meeting is expected to attract over 2,000 international delegates. This milestone event brings together state-of-the-art nephrology forums and cutting – edge scientific advances in renal healthcare.

Novartis Pharmaceuticals

59 Route 10, Bldg. 701/223
East Hanover, NJ 07936 Booth #607

Novartis Pharmaceuticals Corporation is again a proud supporterof the AST and ASTS Scientific Meeting. As the leading contributor to the advancement of transplant science and medicine for almost 20 years, we continue to build on the proven clinical success of Neoral® (cyclosporine, USP [Modified]), Simulect® (basillximab) and Sandimmune® (cyclosporine, USP). Our focus is to develop meaningful therapeutic advances that improve recipient quality of life and enhance the long-term success of transplantation. In partnership with the transplant community, and with over 300 scientists and clinicians dedicated to transplant research, we have created the broadest pipeline in the field with three compounds in clinical trials, and more than a dozen projects in pre-clinical development. Beyond these efforts, Novartis collaborates with over 30 leading research institutions and partners.

Organ Recovery Systems, Inc.

Organ Recover Systems, Inc.
2570 E. Devon Avenue
Des Plaines, IL 60018
847-824-2600 Booth #408

Organ Recover Systems develops medical devices and chemical solutions for the improved recovery, transport, preservation and assessment of organs, tissues and cells for transplant. Our goal is to produce technology which will improve the quality of traditional donor organs and enable the use of expanded criteria and non-heart-beating donor organs for transplant.

Our Organ Perfusion Services Group offers organ perfusion as an outsourced service to transplant centers and OPO's with a focus on treating expanded criteria kidneys for transplant. Our initial customers include Johns Hopkins Hospital, the University of Maryland Hospital and the Transplant Resource Center of Maryland.

Our Charleston Research Center is a leading laboratory for the development of advanced cell and tissue preservation technology. Our research & development efforts also include thrombolytic repair, ischemic damage repair, tolerance induction and immuno modification of isolated organs ex-vivo.

Organon Teknika

100 Akzo Avenue
Durham, NC 27712 Booth #718

Organon Teknika features our new diagnostic assay, NucliSens® CMV pp67, for active CMV infection in transplant and immunocompromised patients. It is an integral part of our entire line of NucliSens® products, which is based on NASBA amplification technology.

Ortho Biotech

700 Route 202
Raritan, NJ 08869 Booth #907

Ortho Biotech, an emerging leader in Critical Care, markets PROCRIT® (Epoetin alfa) for the treatment of anemia and SPORANOX® (itraconazole) Injection for systemic fungal infections.

Pfizer Scientific Exhibits Charles E. Edmiston, Jr., PhD, Exhibitor

235 East 42nd Street, 219/3/50
New York, NY 10017 Booth #425

Educational exhibit by Charles E. Edmiston, Jr., PhD. and Christopher P. Johnson, MD., FACS. Presenting epidemiology, pathogenesis and prevention of bacterial and fungal infections in the transplant patient.

PHI Enterprises, Inc.

12932 Garden Grove Blvd., Suite E
Garden Grove, CA 92843 Booth # 913

PHI Enterprises, Inc. markets healthcare products to professionals.

Professional Postgraduate Services

400 Plaza Drive, 3rd Floor
Secaucus, NJ 07094 Booth #619

Visit our booth to receive a copy of the *Invitation and Call for Abstracts* for the *2nd International Congress on Immunosuppression* to be held December 6 – 8, 2001 in San Diego, California, USA. Building on the success of the first Congress held in Orlando in 1997, the 2nd Congress will feature the popular controversies sessions and abstract and poster presentations on the latest research on immunosuppression in basic science, heart & lung, kidney, liver, pancreas, and small bowel transplantation. The Congress is sponsored by Professional Postgraduate Services and is supported in part by an unrestricted educational grant from the Fujisawa Pharmaceutical Company, Ltd.

Putnam Associates, Inc.

25 Burlington Mall Road, 6th Floor
Burlington, MA 01803 Booth #620

Putnam Associates, Inc. is an economic research and strategy consulting firm specializing in the healthcare, pharmaceutical and biotechnology industries.

Roche Laboratories, Inc.

340 Kingsland Street Booth #407
Nutley, NJ 07110

Roche Laboratories, Inc. is proud to be a part of Transplant 2001. We invite you to visit our exhibit where our representatives will enthusiastically discuss any questions you may have related to our pharmaceutical products. We are also interested in hearing about any issues of interest to you related to the health care industry in general. We look forward to meeting you on the exhibit floor.

ROTRF (Roche Organ Transplantation Research Foundation)

PostJack 222
Schonblickstrasse 3
Meggen 6045
Switzerland Booth #523

The ROTRF is an independent, registered medical research charity established by the Roche Group in 1998. The mission of the ROTRF is to advance the science of solid organ transplantation by funding research programs that contribute to the knowledge of the clinical and scientific adventure of transplantation.

Researchers with innovative scientific ideas and approaches- also from outside of the direct field of transplantation- are strongly encouraged to apply on our website www.rotrf.org.

Sage Science Press

Sage Science Press
2455 Teller Road
Thousand Oaks, CA 91320 Booth #418

Sage Science Press is dedicated to setting an international standard of excellence in science, technical and medical publishing. Since 1995, Sage Science Press has developed or acquired scholarly research journals in the areas of nursing, clinical pharmacology, neuroscience, mechanical and electrical engineering, robotics, high performance computing, and mathematics.

SangStat, Inc.

6300 Dumbarton Circle
Fremont, CA 94555 Booth #323

SangStat, Inc. is a global biotechnology company building on its foundation in transplantation to discover, develop and market high value therapeutic products in the transplantation, immunology and hematology/oncology areas. Since 1988, SangStat has been dedicated to improving the outcome of organ and bone marrow transplantation through the development and marketing of products to address all phases of transplantation in the worldwide market.

Scanlan International, Inc.

6300 Dumbarton Circle
Fremont, CA 94555 Booth #323

High quality surgical products designed and manufactured by the Scanlan family since 1921. Offering instrumentation designs (in stainless steel and titanium and several single-use products for transplant surgery.

Sidmak Laboratories

Sidmak Laboratories, Inc.
17 West Street
East Hanover, NJ 07936 Booth #423

Sidmak Laboratories is rapidly establishing a significant presence in the managed care market through competitive pricing, high quality research, development, quality assurance, and dedicated world class manufacturing facilities.

Please stop by booth #423 for information on Sidmark Laboratories' products and services. MC Access represents Sidmak Laboratories to the managed care market.

Teleresults Corporation

870 Market Street, Suite 556
San Francisco, CA 94102 Booth #517

TeleResults-Tx multi-organ transplant covers all the phases of transplantation from referral to follow-up. TeleResults interfaces with the hospital ADT and labs; even outside labs. The system is used in daily operation capturing medical history, examination, diagnosis & problems, infections, rejections, graft status, k medications, procedures. Many useful tools such as reminders, task management, data analysis, and grouping of patients by protocols are standard features. Its open database interfaces with commercially available report writers, query tools and statistical packages. It reports to UNOS. The system has built-in communication programs for voice, fax, and Internet web-browser interface to the database.

The RxFiles Corporation

342 Ramiami Trail South
Nokomis, FL 34275 Booth #518

The Intelligent Dosing System™ for Immunosuppressants.

The Transplantation Society

205 Viger Avenue West, Suite 201
Montreal, Quebec, H2Z 1G2
Canada Booth #717

The Transplantation Society serves as the principal international forum for the advancement of both basic and clinical transplantation science throughout the world. Since its founding 34 years ago, the Society's history has closely paralleled the development of organ transplantation to its current status. The Society currently has almost 3,000 member from 65 countries. Among its present and former members are six Nobel Laureates. In addition, the Society biennially awards the Medawar Prize, which is recognized as the world's highest dedicated award for the most outstanding contributions in the field of transplantation.

Please come to The Transplantation Society booth for further information about the Society and membership to the Society.

Thompson Surgical Instruments, Inc.

10170 E. Cherrybend Road
Traverse City, MI 49684 Booth #715

Please visit our booth #715 and see the Elite II Thompson Retractor System.

TransCom Media

119 Cherry Hill Road
Parsippany, NJ 07054 Booth #616

TransCom Media producers of "Transplant Video Journal" a news program about transplantation topics and several print .publications related to the transplant field.

Transonic Systems Inc.

34 Dutch Mill Road
Ithaca, NY 14850 Booth #512

Intraoperative Bloodflow Monitoring. Measures Arterial and Venous Bloodflow pre and post transplantation. Corrects insufficient Bloodflow while in the Operating Room.

TransWeb

The Northern Brewery, Suite 105
1327 Jones Drive
Ann Arbor, MI 48105 Booth #317

TransWeb: All About Transplantation and Donation is a nonprofit educational web site serving the world transplant community. Based at the University of Michigan Medical Center, TransWeb features news and events, real people's experiences, the top 10 myths about donation, a donation quiz, and a large collection of questions and answers, as well as a reference area with everything from articles to videos. For young people, TransWeb features Give Life: The Transplant Journey, an exciting new multimedia trip through the transplant process, told from the donor's family's point of view. A comprehensive collection of medical illustrations, audio and video segments, a full glossary, subject index, and the ability to search the site make the Transplant Journey (and TransWeb as a whole) a useful educational tool for a broad audience, including not only transplant patients and their families, but also students and teachers, health care professionals, the media, and the general public.

The UCLA Immunogenetics Center

950 Veteran Avenue
Los Angeles, CA 90095-1651 Booth #413

The UCLA Immunogenetics Center provides a range of clinical tests and other services in support of transplant hospitals and the transplant community. Clinical Transplants 2000, the 16th annual publication in the series that we publish, along with sets of colored slides and powerpoint format disc derived from the latest book, are now available. Stop by our booth and have a look at these latest offerings.

United Network for Organ Sharing (UNOS)

1100 Boulders Parkway, Suite 500
Richmond, VA 23225 Booth #912

UNOS is a not-for-profit organization that administers the Organ Procurement and Transplantation Network (OPTN) for the U.S. Department of Health and Human Services. Primary functions of UNOS and the OPTN are to maintain the national transplant waiting list; perform donor to potential recipient organ matching; and to collect and report transplant-related date. UNOS has utilized new and emerging technologies to support these functions by creating applications like UNet℠, the UNOS web site, the UNOS Transplant Patient DataSource℠ and the organ allocation models with the intent of better informing and assisting the transplant community, patients and the public.

University Renal Research and Education Association

315 West Huron, Suite 260
Ann Arbor, MI 48103 Booth #622

URREA is the contractor for the Scientific Registry of the Transplant Recipient we will distribute research information.

ViraCor

11100 Ash Street, Suite 202
Leawood, KS 66211 Booth # 319

Dedicated to the detection, management and control of herpes viruses for the good of the patient.

VitaGen Incorporated

3344 North Torrey Pines Ct., Suite 100
La Jolla, CA 92126 Booth #908

Artificial Liver Assist Device in Clinical Trials.

Waters Instruments, Inc.

2411 7th Street, NW
Rochester, MN 55901 Booth #713

Waters continues to be the world leader in pulsatile preservation of organs with the rm3 renal preservation system.

W.B. Saunders/Mosby/Churchill

927 Levernz
Naperville, IL 60565 Booth #914

W.B. Saunders/Mosby/Churchill will have on display all of their books and journals related to transplantation and the new *Morris Kidney Transplantation* will be available.

Weck Closure Systems

PO Box 12600
One Weck Drive
Research Triangle Park, NC 27709 Booth #624

Weck Closure Systems will highlight its revolutionary new Hem-o-lok® polymer clip, as well as its Horizon™, Hemoclip®, and Atrauclip® ligating clip lines. In addition, Weck will be exhibiting the Dexterity® Pneumo Sleeve® and Dexterity® Protractor™ for use in both open and laparoscopic-assisted surgery.

Wyeth-Ayerst Pharmaceuticals

PO Box 8299
Philadelphia, PA 19101 Booth #507

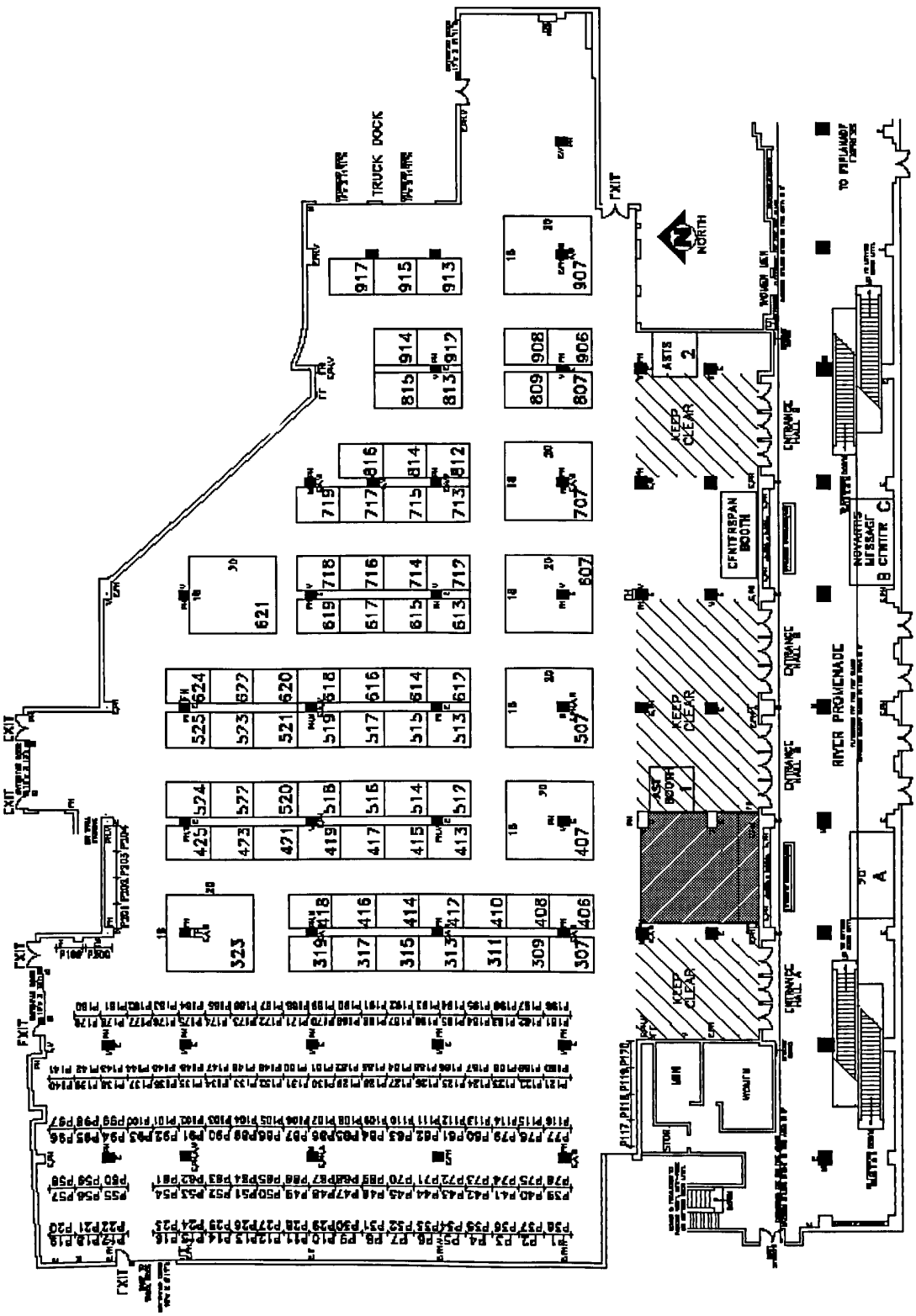
Wyeth-Ayerst Pharmaceuticals welcomes the opportunity to continue their longstanding association with AST and ASTS through participation in your exhibit program. Our professional representatives welcome the opportunity to visit with you and discuss any inquiries you may have concerning our products.

XIX International Congress Society

Juan F. Aranguren 551
P, B. B 1405
Buenos Aires
Argentina Booth #419

XIX International Congress of The Transplantation Society will be held in Buenos Aires (Argentina) in August 18-23, 2002. Important information on the scientific program outline, venue, deadlines, accommodation and travel arrangements for the event will be available at this booth. Welcome.
The Local Organizing Committee
www.transplantation2002.com

Exhibit Floor Plan



TRANSPLANT 2001

The Joint American Transplant Meeting

Postgraduate Course

Friday, May 11, 2001

Transplantation Review and Update

Session I

Chicago Ballroom 6/7, Sheraton
Chairs: Jonathon Bromberg and Gabriel Danovitch

1:30 PM – 2:10 PM **Mechanisms of Allograft Rejection and Strategies for Monitoring Rejection**
Peter Nickerson

2:10 PM – 2:50 PM **Pathology of Allograft Rejection**
Lorraine Racusen

2:50 PM – 3:30 PM **Mechanisms of Current Immunosuppression**
Philip Halloran

3:30 PM - 4:00 PM **Break**

4:00 PM – 4:40 PM **Costimulation Pathways: Basic Science and Potential Clinical Applications**
Laurence Turka

4:40 PM – 5:20 PM **Current Immunosuppressive Regimes in Organ Transplantation**
Gabriel Danovitch

Saturday, May 12, 2001

Postgraduate Course (continued)

Session II

Chicago Ballroom 6/7, Sheraton
Chairs: Peter Stock and Jay Fishman

8:00 AM – 8:40 AM **CMV and Emerging Viruses in Organ Transplant Recipients**
Jay Fishman

8:40 AM – 9:20 AM **Managing Hepatitis B and Hepatits C in Organ Recipients**
Anna Lok

9:20 AM – 10:00 AM **Current Status of Heart and Lung Transplantation**
Mark Barr

10:00 AM – 10:30 AM **Break**

10:30 AM – 11:10 AM **Innovations in Liver Transplantation**
Charles Miller

11:10 AM – 11:50 AM **A New Era for Beta Cell Replacement: Pancreas or Islet Transplantation**
David Sutherland

Pre-Meeting Symposia

1:00 PM – 5:30 PM **Transplant Nurses and Coordinators Special Program**

Chicago Ballroom 9/10, Sheraton
Chairs: Trish Brennan and Cathy Garvey

1:00 PM **Kidney Transplantation in the HIV Positive Patient**
Laurie Carlson

2:00 PM **Liver-Assist Device**
Christopher Freise

3:00 PM **Break**

3:30 PM **Islet Cell Transplant**
Ingrid Larsen

4:30 PM **Living Lober Lung Transplant**
Felicia Schenkel

1:00 PM – 3:00 PM **Extended Donors/Allocation Symposium: Report from the Cadaver Donor Conference**

Chicago Ballroom 6/7, Sheraton
Chairs: Francis Delmonico and Bruce Rosengard

1:00 PM **The True Benefit and Appropriate Sharing of Zero-Mismatched Kidneys**
Edward Alfrey

1:20 PM **Liver Donors: Avoiding Bad Cadaver Donors and Finding the Right Livers to Split**
Jean Emond

1:40 PM **Marginal Donors because of Malignancy or Positive Serology**
Sandy Feng

2:00 PM **Strategies To Increase Donor Lung Utilization**
Edward J. Garrity

2:20 PM **Strategies To Increase Donor Heart Utilization**
John Zariff

1:00 PM – 3:00 PM **Pediatrics Symposium: Transplantation In Adolescents**

Sheraton Ballroom 4/5, Sheraton
Chairs: Amir Tejani and Richard Fine

1:00 PM **Transplantation Outcomes in Teenagers**
Ruth McDonald

1:30 PM **Optimal Immunosuppression in Teenagers**
Deidre Kelly

2:00 PM **Recurrent Disease Post-Transplantation**
Michelle Baum

2:30 PM **Noncompliance and Its Management in Teenagers**
Thomas Nevins

3:00 PM – 3:30 PM **Break**

3:30 PM – 5:30 PM **Two Concurrent Symposia Clinical Science Symposium: Anti-Microbial Resistance In Transplant Infectious Diseases**

Sheraton Ballroom 4/5, Sheraton
Chairs: Jutta Preiksaitis and Susan Keay

3:30 PM **Prevention and Management of Resistant Fungal Infections**
Thomas Walsh

4:00 PM **Prevention and Management of Resistant Bacterial Infections**
Emily Blumberg

4:30 PM **Pathogenesis of Gangiclovir-Resistant CMV**
Micheal Boeckh

5:00 PM **Evading the Immune System: Lessons Learned from Viruses**
Alexandra Lucas

3:30 PM – 5:30 PM **Basic Science Symposium: Genomics and Proteomics Overview**
Chicago Ballroom 6/7, Sheraton
Chairs: Kenneth Drazen and Terry Strom

3:30 PM **Gene-Discovery and Diagnostic Approach to Genomics in Clinical Research**
Minnie Sarwal

4:00 PM **Application of Genome Expression Profiling To Understand the Pathogenesis of EBV and CMV**
Thomas Shenk

4:30 PM **Molecular Classifiation of Tissue-Applications To Diagnosis and Prognosis**
Robert Lipshutz

5:00 PM **Proteomics: New Technologies for Quantitation and Application to Transplant Research**
Ruedi Aebersold

TRANSPLANT 2001
The Joint American Transplant Meeting
Day-at-a-Glance, Sunday, May 13, 2001

6:30 AM - 7:50 AM — **Concurrent Sunrise Symposia**

Page 51 — **Sunrise Symposium I: Allorecognition**
Chicago Ballroom 8-10, Sheraton

Page 51 — **Sunrise Symposium II: Video Session I: Techniques in Living Donor Nephrectomy**
Sheraton Chicago Ballroom 4-7, Sheraton

Page 51 — **Sunrise Symposium III: Bioartificial Organs**
Sheraton Ballroom 1-3, Sheraton

8:00 AM — **Plenary Session I**

Page 51 — **Basic Science**
Sheraton Ballroom 1-3, Sheraton

Page 51 — **Clinical Science**
Sheraton Chicago Ballroom 4-7, Sheraton

9:15 AM - 10:45 AM — **Concurrent Symposia**

Page 51 — **Basic Science Symposium: T Cell Activation**
Sheraton Ballroom 1-3, Sheraton

Page 51 — **Clinical Trials Update: Recent Trials of Immunosuppression**
Sheraton Chicago Ballroom 4-7, Sheraton

11:00 AM - 12:00 PM — **Concurrent Sessions**

Page 52 — **In Depth Reviews: Clinical**
Sheraton Chicago Ballroom 4-7, Sheraton

Page 52 — **In Depth Reviews: Basic Science**
Sheraton Ballroom 1-3, Sheraton

12:30 PM - 1:30 PM — **Parallel Luncheon Workshops**
Page 52 — *Sheraton and Intercontinental*

12:30 PM - 1:30 PM — **Selected Poster Sessions**
Page 52 — *Sheraton*

2:00 PM - 3:30 PM — **Concurrent Sessions**

Page 53 — **Concurrent Session 1: Cytokine Regulation of Alloimmune Responses**
Chicago Ballroom 10, Sheraton

Page 54 — **Concurrent Session 2: Complications in Renal Transplantation**
Chicago Ballroom 6/7, Sheraton

Page 54 — **Concurrent Session 3: Basic Science: Immunosuppression/Tolerance**
Chicago Ballroom 8, Sheraton

Page 55 — **Concurrent Session 4: Basic Science: Rejection I**
Chicago Ballroom 9, Sheraton

Page 55 — **Concurrent Session 5: Kidney Transplantation: Recipient Factors and Outcomes**
Sheraton Ballroom 1-3, Sheraton

Page 56 — **Concurrent Session 6: Kidney Transplantation: Factors Affecting Clinical Outcomes**
Sheraton Ballroom 4/5, Sheraton

Page 56 — **Concurrent Session 7: Xenotransplantation: Preclinical Non-Human Primate**
Empire Room, Intercontinental

Page 57 — **Concurrent Session 8: Lung Transplantation: Bench-to-Bedside**
Exchange Room, Intercontinental

Page 57 — **Concurrent Session 9: Liver Transplantation: Hepatitis C Clinical Outcomes**
Grand Ballroom, Intercontinental

Page 58 — **Concurrent Session 10: Immunosuppression for Pancreas Transplantation**
Renaissance Ballroom, Intercontinental

4:00 PM - 5:30 PM — **Concurrent Sessions**

Page 58 — **Concurrent Session 11: Control of Alloreactive T Cells**
Chicago Ballroom 10, Sheraton

Page 58 — **Concurrent Session 12: Risk Analysis in Renal Transplantation**
Chicago Ballroom 6/7, Sheraton

Page 59 — **Concurrent Session 13: Basic Science: Immunosuppression I**
Chicago Ballroom 8, Sheraton

Page 59 — **Concurrent Session 14: Basic Science: Rejection II**
Chicago Ballroom 9, Sheraton

Page 60 — **Concurrent Session 15: Transplantation: Allocation**
Sheraton Ballroom 1-3, Sheraton

Page 60 — **Concurrent Session 16: Sirolimus in Kidney Transplantation**
Sheraton Ballroom 4/5, Sheraton

Page 61 — **Concurrent Session 17: Mechanisms of Ischemia/Reperfusions Injury I**
Empire Room, Intercontinental

Page 61 — **Concurrent Session 18: Thoracic Organ Donor Shortage Better Management/ Alternative Strategies**
Exchange Room, Intercontinental

Page 62 — **Concurrent Session 19: Liver Transplantation: Hepatitis C II**
Grand Ballroom, Intercontinental

Page 62 — **Concurrent Session 20: Islet Transplantation and Long-Term Results of Pancreas Transplantation**
Renaissance Ballroom, Intercontinental

8:00 AM - 7:30 PM	**Poster Session I**
5:30 PM - 7:30 PM	**Presenters in Attendance**
	Exhibits Open *Wine and Cheese Reception* *River Exhibition Hall*
Page 63	**Kidney - Acute/Chronic Rejection I**
Page 63	**Kidney -GVH, Complications, Infections I**
Page 64	**Kidney - Immunosuppresssion A I**
Page 64	**Kidney - Immunosuppression B I**
Page 65	**Kidney - Pediatrics, Recurrent Disease I**
Page 65	**Kidney - Preservation, Donation/Allocation, Economics/Public Policy, Surgical Techniques, and Other I**
Page 66	**Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics I**
Page 67	**Liver - Infections, Complications, Recurrent Disease, Surgical Techniques I**
Page 68	**Liver - Preservation, Economics/Public Policy, Donation Allocation, Other I**
Page 68	**Pancreas and Islets - All Topics I**
Page 69	**Heart/Lung - All Topics I**
Page 69	**Bone Marrow - All Topics I**
Page 70	**Immunosuppression, Preclinical Studies I**
Page 70	**Tolerance I**
Page 71	**Acute/Chronic Rejection I**
Page 71	**Allorecognition, Antigen Presentation, Co-Stimulation and Other I**
Page 72	**Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines I**
Page 72	**Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH I**
Page 73	**Tissue Injury, Preservation I**
Page 73	**Xenotransplantation**

Sunday, May 13, 2001

Concurrent Sunrise Symposia
6:30 AM - 7:45 AM

Sunrise Symposium I: Allorecognition

Sheraton Ballroom 8-10, Sheraton
Chair: Robert Lechler

6:30 AM Direct recognition: Is it a naive or memory response?
Robert Lechler
6:55 AM Is the indirect pathway responsible for chronic rejection?
Mohamed Sayegh
7:20 AM Direct and indirect pathways and tolerance induction
Kathryn Wood

Sunrise Symposium II: Video Session I: Techniques in Living Donor Nephrectomy

Sheraton Chicago Ballroom 4-7, Sheraton
Chair: Lloyd Ratner

6:30 AM Laparoscopic donor nephrectomy
Lloyd Ratner
6:45 AM Hand-assisted laparoscopic donor nephrectomy
Robert Harland
7:00 AM Anterior retroperitoneal donor nephrectomy
Thomas Peters
7:15 AM Robot-assisted donor nephrectomy
Enrico Benedetti

Sunrise Symposium III: Bioartificial Organs

Sheraton Ballroom 1-3, Sheraton
Chair: Bartley Grifith

6:30 AM Bioartificial kidney
David Hume
6:45 AM Bioartificial pancreas
David Klonoff
7:00 AM Bioartificial liver
Michael Millis
7:15 AM Artificial lung
Bartley Grifith

Plenary Session I: Basic
8:00 AM - 9:00 AM

Sheraton Ballroom 1-3, Sheraton
Chairs: Joshua Miller and Angus Thomson

8:00 AM MEMORY BUT NOT NAIVE T CELLS REJECT VASCULARIZED CARDIAC ALLOGRAFTS IN THE ABSENCE OF SECONDARY LYMPHOID ORGANS. (Abstract #1) *Young Investigator Award*
Geetha Chalasani, Bogumila T. Konieczny, Zhenhua Dai, Fadi G. Lakkis. Atlanta, GA.

8:15 AM HEME OXYGENASE-1 GENE TRANSFER PREVENTS FAS/FAS LIGAND-INDUCED APOPTOSIS IN VITRO AND IMPROVES ALLOGRAFT FUNCTION IN VIVO. (Abstract #2) *Young Investigator Award*
Bibo Ke, Xiu-Da Shen, Judy Melinek, Feng Gao, Thomas Ritter, Hans-Dieter Volk, Roland Buelow, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Berlin, Germany; Fremont, CA.

8:30 AM REGULATORY ROLE OF CTLA4 AND CYTOKINES IN PHYSIOLOGIC TERMINATION OF ALLOIMMUNE RESPONSES IN VIVO. (Abstract #3) *Young Investigator Award*
Nader Najafian, Masayuki Sho, Akira Yamada, Koji Kishimoto, Victor M. Dong, Sigrid E. Sandner, Mohamed H. Sayegh. Boston, MA.

8:45 AM TYPE III IMMUNE IMMUNE REACTIONS IN XENOTRANSPLANTATION. (Abstract #4)
Yoshihiro Miyata, Christine L. Lau, R. Duane Davis, Jeffrey L. Platt, Zoie E. Holzknecht. Rochester, MN; Rochester, MN.

Plenary Session II: Clinical
8:00 AM - 9:00 AM

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: Donald Hricik and Ronald Ferguson

8:00 AM PRELIMINARY RESULTS FROM A HUMAN TOLERANCE TRIAL USING CAMPATH-1H. (Abstract #5)
Allan D. Kirk, S. John Swanson, Roslyn B. Mannon, D. Scott Batty, Wendy Bernstein, Lee Brettman, Chris Chamberlain, Barbara S. DiMercurio, Keith Hunter, Robert Kampen, David Kleiner, Douglas K. Tadaki, David M. Harlan. Bethesda, MD; Washington, DC; Cambridge, MA.

8:15 AM RANDOMIZED CONTROLLED TRIAL OF HAND-ASSISTED LAPAROSCOPIC VERSUS OPEN SURGICAL LIVE DONOR NEPHRECTOMY. (Abstract #6) *Young Investigator Award*
J. Stuart Wolf, Jr., Robert M. Merion, Alan B. Leichtman, Darrell A. Campbell, Jr., John C. Magee, Jeffery D. Punch, Jeremiah G. Turcotte, John W. Konnak. Ann Arbor, MI.

8:30 AM INHIBITION OF CROSSMATCH (CMX) POSITIVITY BETWEEN DONOR-RECIPIENT PAIRS USING INTRAVENOUS GAMMAGLOBULIN (IVIG) WITH SUBSEQUENT TRANSPLANTATION. (Abstract #7)
Stanley C. Jordan, Ashley A. Vo, Suphamai Bunnapradist, Dolly Tyan. L.A., CA.

8:45 AM TEN YEAR EXPERIENCE WITH INTESTINAL TRANSPLANTATION. (Abstract #8)
A. Langnas, S. Chinnakotla, D. Sudan, S. Horslen, B. Shaw, K. Iyer, I. Fox. Omaha, NE.

Concurrent Symposia
9:15 AM - 10:45 AM

Basic Science Symposium: T Cell Activation

Sheraton Ballroom 1-3, Sheraton
Chairs: Judith Thomas and Charles Carpenter

9:15 AM Signaling pathways critical for T cell activation and downregulation
David Rothstein
9:45 AM Signals 1 and 2: Are they distinct signals for T cell activation?
Chris Rudd
10:15 AM New costimulatory pathways
Arlene Sharpe

Clinical Trials Update: Recent Trials of Immunosuppression

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: Connie Davis and Arthur Matas

9:15 AM Anti-CD3, campath 1H, and other antibodies
Stuart Knechtle
9:35 AM TOR and/or calcineurin inhibitors
Marc Lorber
9:55 AM Steroid sparing
Arthur Matas
10:15 AM What's in the pipeline?
Flavio Vincenti
10:35 AM Round Table Discussion of the Recent Clinical Trials

10:45 AM Break

Concurrent Sessions: In Depth Reviews

11:00 AM - 12:00 PM

In Depth Reviews: Clinical

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: Hugh Auchincloss and Stuart Knechtle

11:00 AM **Moving tolerance induction to the clinic**
Jeffrey Bluestone
11:30 AM **Current status of bone marrow transplantation**
Brenda Sandmeier

11:00 AM - 12:00 PM

In Depth Reviews: Basic Science

Sheraton Ballroom 1-3, Sheraton
Chairs: Joren Madsen and Jerzy Kupiec-Weglinski

11:00 AM **Stem cells and the future of transplantation**
John D. Gearheart
11:30 AM **Leukocyte homing**
Ulrich H. von Andrian
12:00 PM **Break**

Parallel Luncheon

12:30 PM - 1:30 PM

Room locations not available at time of publication. Locations will be printed on the tickets and in the mini-program. Be sure to check hotels location.

1. **The extracellular matrix and allograft rejection**
Ana Coito and Mark Stegall
2. **New concepts in ischemial reperfusion injury**
Roland Buelow and Jerzy Kupiec-Weglinski
3. **Managing hepatitis C**
John Lake and Michael Lucy
4. **Reevaluation of the patient on the waiting list**
Leslie Miller and Harold Helderman
5. **Chemokines in rejection**
Wayne Hancock and Robert Fairchild
6. **How to use sirolimus in the clinic**
Alan McDonald and Marc Lorber
7. **The marginal donor**
J. Wesley Alexander and Edward Alfey
8. **Tissue injury as determinant of graft outcome**
Philip Halloran and Nicholas Tilney
9. **Transplantation in infants**
Sue McDiarmid and Thomas Nevins
10. **Obliterative bronchiolitis**
Janet Mauer and Edward Garrity
11. **New approaches in PTLD**
Anne van Buskirk and Lode Swinnen
12. **T cell clonality in alloimmune responses**
Jean Paul Soulillou and Yuan Zhai
13. **MHC peptides / allochimeric molecules**
Mark Ghobrial and Ana Marie Waaga
14. **Complications of renal transplantation: Interactive case presentation**
Connie Davis and John Pirsch
15. **Information systems on organ transplantation**
Robert Merion
16. **Genetically engineered animals for transplantation**
Anthony d'Apici and David Adams
17. **Ethics of relationships to pharmaceuticals**
Mark Siegler and Richard Howard
18. **Complications of heart transplantation**
Mandeep Mehra and Howard Eisen
19. **Mechanism of graft atherosclerosis**
Donald Cramer and Richard Mitchell
20. **Activation of alloreactive CD4 cells**
Andrew Wells and Anna Valujskikh
21. **How old is too old and how sick is too sick for a kidney recipient**
Lawrence Hunsicker and Dianne McKay
22. **Gene polymorphisms**
Ian Hutchinson and Barbara Murphy
23. **Novel approaches to monitor allograft rejection**
Peter Nickerson and Minnie Sarwal

Selected Poster Open Sessions

12:30 PM - 1:30 PM

Selected Posters on Tolerance

Sheraton Ballroom 1-3, Sheraton
Chairs: Hugh Auchincloss and Laurence Turka

12:30 PM **DEFICIENT PRODUCTION OF ANTI-A/B ANTIBODIES AFTER ABO-INCOMPATIBLE INFANT HEART TRANSPLANTATION: CLINICAL NEONATAL B-CELL TOLERANCE? (Abstract #790)**
Lori J. West, Stacey M. Pollock-BarZiv, K. J. Lee, Anne I. Dipchand, John G. Coles, Phillip Ruiz. Toronto, ON, Canada; Miami, FL.

12:35 PM **COSTIMULATORY MOLECULE-EXPRESSING DENDRITIC CELLS POLARIZED BY PROSTAGLANDIN E_2 *PROLONG CARDIAC ALLOGRAFT SURVIVAL IN MICE. (Abstract #1204)***
Mark L. Jordan, Susan M. Specht, Patrick P. Luke, Adrian Morelli, Zhiliang Wang, Angus W. Thomson. Pittsburgh, PA; London, ON, Canada; Pittsburgh, PA.

12:40 PM **INDUCTION OF HEMATOPOIETIC CHIMERISM AND MURINE CARDIAC ALLOGRAFT TOLERANCE WITH A NON-MYELOABLATIVE REGIMEN. (Abstract #354)**
Nozomu Shirasugi, Andrew B. Adams, Megan M. Durham, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

12:45 PM **ANTI-LFA-1 INDUCED ISLET ALLOGRAFT SURVIVAL DOES NOT REQUIRE STAT6. (Abstract #1203)**
Marilyne Coulombe, Mark R. Nicolls, Ronald G. Gill. Denver, CO.

12:50 PM **ALLOCHIMERIC PROTEIN-INDUCED TOLERANCE IS MEDIATED BY POTENT REGULATORY T HELPER 2 CELLS. (Abstract #1254)**
Barton Trawick, Robert Kirken, Min Wang, Neelam Tejpal, Mou-Er Wang, Barry D. Kahan, Stanislaw M. Stepkowski. Houston, TX; Houston, TX.

12:55 PM **PREVENTION OF AUTOIMMUNE DESTRUCTION OF ISLET TRANSPLANTS IN NONOBESE DIABETIC (NOD) MICE. (Abstract #1247)**
Maria Koulmanda, Andi S. Qipo, Neal R. Smith, Hugh Auchincloss, Jr.

1:00 PM **VASCULARIZED ISLET-KIDNEY ALLOGRAFTS CURE SURGICALLY-INDUCED DIABETES AND INDUCE TOLERANCE IN MINIATURE SWINE. (Abstract #830)**
Naoki Kumagai, Rolf N. Barth, John J. O'Neil, John C. LaMattina, Ryu Utsugi, Hiroshi Kitamura, Gordon C. Weir, David H. Sachs, Kazuhiko Yamada. Boston, MA; Boston, MA.

1:05 PM **SPILT TOLERANCE OBSERVED IN LIMB TISSUE ALLOGRAFTS IN SWINE TREATED WITH A 12-DAY COURSE OF CYCLOSPORINE. (Abstract #343)**
David W. Mathes, Mark A. Randolph, Mario G. Solari, Jamal Nazzal, David H. Sachs, W.P. Andrew Lee. Boston, MA; Boston, MA.

Selected Posters on Kidney Transplantation

Sheraton Ballroom 4/5, Sheraton
Chairs: Ronald Ferguson and Arthur Matas

12:30 PM **47 CONSECUTIVE KIDNEY TRANSPLANTS WITHOUT REJECTION-THE IMPORTANCE OF DIURNAL CSA DOSING. (Abstract #191)**
Barry J. Browne, Cynthia Op't Holt, Osemwegie E. Emovon. Mobile, AL.

12:35 PM **ASSOCIATION OF ANTIBODY INDUCTION AND SHORT AND LONG-TERM CAUSE SPECIFIC MORTALITY AFTER RENAL TRANSPLANTATION. (Abstract #625)**
Herwig-Ulf Meier-Kriesche, Friedrich K. Port, Bruce Kaplan. Ann Arbor, MI.

12:40 PM **INTERACTION BETWEEN ACUTE REJECTION AND RECIPIENT AGE ON LONG TERM RENAL ALLOGRAFT SURVIVAL. (Abstract #198)**
Herwig-Ulf Meier-Kriesche, Otto Leiti, Gary S. Friedman, Bruce Kaplan. Ann Arbor, MI; Livingston, NJ.

12:45 PM **GRAFT LOSS AND RECURRENT REJECTIONS IN FLOW CYTOMETRIC CROSS-MATCH POSITIVE PRIMARY RENAL TRANSPLANTS. (Abstract #1052)**
M. Karpinski, D. Rush, J. Jeffery, D. Pochinco, S. Dancea, P. Birk, P. Nickerson. Winnipeg, MB, Canada.

12:50 PM **LUPUS NEPHRITIS IN AFRICAN AMERICAN KIDNEY TRANSPLANT RECIPIENTS CARRIES A POOR PROGNOSIS. (Abstract #653)**
Lee Erbe, Jeremy Kirtz, Jeffery Rogers, Angello Lin, G. Mark Baillie, P.R. Rajagopalan, Kenneth D. Chavin, Prabhakar K. Baliga. Charleston, SC.

12:55 PM **THE EFFECT OF MYCOPHENOLATE MOFETIL ON HEPATITIS B VIRAL LOAD IN STABLE RENAL TRANSPLANT RECIPIENTS WITH CHRONIC HEPATITIS B. (Abstract #203)**
Bart D. Maes, Jos F. van Pelt, Peeters Jacques, Nevens Frederik, Evenepoel Pieter, Kuypers Dirk, Messiaen Thierry, Fevery Johan, Vanrenterghem F. Yves. Leuven, Belgium; Leuven, Belgium.

1:00 PM **RENAL ALLOGRAFT OUTCOMES IN AFRICAN-AMERICAN VERSUS CAUCASIAN TRANSPLANT RECIPIENTS IN THE TACROLIMUS ERA. (Abstract #621)**
Karen L. Hardinger, Robert J. Stratta, M. Francesca Egidi, Rita R. Alloway, M. Hosein Shokouh-Amiri, Lillian W. Gaber, Hani P. Grewal, Marsha R. Honaker, Santiago R. Vera, A. Osama Gaber. Memphis, TN.

1:05 PM **IN VIVO IL-10 PRODUCTION BY PBMC FROM LONG-TERM KIDNEY TRANSPLANT RECIPIENTS WITH EXCELLENT GRAFT FUNCTION. (Abstract #670)**
Hugo Guzman-Rodriguez, Claudia de Leo, Eduardo Mancilla, Ricardo Correa-Rotter, Alfredo Chew-Wong, Josefina Alberu.

1:10 PM **COMPARISON OF OPEN, LAPAROSCOPIC AND HAND-ASSIST LIVE DONOR NEPHRECTOMY. (Abstract #1114.5)**
Ergun Velidedeoglu, Ali Naji, Kenneth L. Brayman, Noel N. Williams, Niraj M. Desai, Luis Campos, Maral Palanjian, Martin Wocjik, Roy D. Bloom, Robert Grossman, Kevin C. Mange, Jo Buyske, Clyde F. Barker, James F. Markman. Philadelphia, PA; Philadelphia, PA.

Concurrent Session 1: Cytokine Regulation of Alloimmune Responses

2:00 PM - 3:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Keith Bishop and Thomas Coffman

2:00 PM **ABSENCE OF BOTH TUMOR NECROSIS FACTOR RECEPTORS P55 AND P75 ON DONOR HEARTS DIMINISHES GRAFT ARTERIAL DISEASE ALTHOUGH P55 OR P75 SINGLE DEFICIENCY DOES NOT. (Abstract #9)**
Jun-ichi Suzuki, Sarah E. Cole, Peter Libby, Richard N. Mitchell. Boston, MA.

2:10 PM **FAILURE TO INDUCE NEONATAL TOLERANCE IN MICE THAT LACK BOTH IL-4 AND IL-13 BUT NOT IN THOSE THAT LACK IL-4 ALONE. (Abstract #10)**
Yoshihiko Inoue, Maylene E. Wagener, Bogumila T. Konieczny, Fadi G. Lakkis. Atlanta, GA.

2:20 PM **THE CONTRIBUTION OF IFN-γ TO ISLET ALLOGRAFT SURVIVAL FOLLOWING ANTI-CD4 THERAPY. (Abstract #11)**
Alexander C. Wiseman, Mark R. Nicolls, Andrew S. Diamond, Josh Beilke, Ron G. Gill. Denver, CO.

2:30 PM **NEUTROPHILS ACCELERATE THE REJECTION OF FULLY MHC-DISPARATE CARDIAC ALLOGRAFTS IN THE ABSENCE OF INTERFERON-γ. (Abstract #12)**
Masayoshi Miura, Qiwei Zhang, Robert L. Fairchild. Cleveland, OH.

2:40 PM **THE ROLE OF TH1 CYTOKINES IN TOLERANCE REVISITED: EFFECT OF T CELL CLONE SIZE. (Abstract #13)**
Koji Kishimoto, Victor M. Dong, Nader Najafian, Terry B. Strom, Laurence A. Turka, Mohamed H. Sayegh. Boston, MA; Boston, MA; Philadelphia, PA.

2:50 PM **CYTOKINE EXPRESSION BY DENDRITIC CELLS DRIVES DIFFERENTIATAION OF VARIOUS T CELL SUBSETS. (Abstract #14)**
Lianfu Wang, Xiaoyan Liang, Shiguang Qian, C. Andrew Bonham, John J. Fung, Lina Lu. Pittsburgh, PA.

3:00 PM **IL-10 ENHANCES CCR5 BUT DOWN-REGULATES CCR7 EXPRESSION BY MYELOID DENDRITIC CELLS: IMPACT ON CHEMOTACTIC AND IN VIVO HOMING RESPONSES. (Abstract #15)**
Takuya Takayama, Nobuyuki Onai, Motohiro Hirano, Kouji Matsushima, Adrian E. Morelli, Hideaki Tahara, Angus W. Thomson. Pittsburgh, PA; Tokyo, Japan.

3:10 PM **SPONTANEOUS DEVELOPMENT OF TGFβ-REGULATED ALLOIMMUNITY BY MURINE RENAL ALLOGRAFT RECIPIENTS. (Abstract #16)**
J. J. Wang, M. E. Wakely, A. A. Bickerstaff, C. G. Orosz. Columbus, OH.

3:20 PM **EXPRESSION OF IL-10 IN THE LIVER ALLOGRAFTS IN A COMPOSITE AUXILIARY PARTIAL LIVER/SMALL BOWEL TRANSPLANTATION MODEL. (Abstract #17)**
Saiho Ko, Yoshiyuki Nakajima, Hiromichi Kanehiro, Hideki Kanokogi, Tetsuhiro Kanamura, Michiyoshi Hisanaga, Yukio Aomatsu, Mitsuo Nagao, Naoya Ikeda, Yasuyuki Urizono, Tsunehiro Kobayashi, Takamune Shibaji, Sanehito Ogawa, Hiroshige Nakano. Kashihara, Nara, Japan.

Concurrent Session 2: Complications in Renal Transplantation

2:00 PM - 3:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: Roy Bloom and Simin Goral

2:00 PM **PHARMACOKINETIC INTERACTION OF CHLORAMPHENICOL WITH CALCINEURIN INHIBITORS. (Abstract #18)**
A. Scott Mathis, Nita Shah, Gary S. Friedman, Jenivieve Villadolid, Kathryn Kalafatas. Livingston, NJ; Piscataway, NJ.

2:10 PM **LOSARTAN DRAMATICALLY REDUCES MASSIVE PROTEINURIA AFTER RENAL TRANSPLANTATION: A MULTICENTRIC AND PROSPECTIVE STUDY. (Abstract #19)**
Beatriz Dominguez-Gil, Pablo Iñigo, Milagros Ortiz, Maria P. Sierra, Fernando Anaya, D. del Castillo, Amado Andres, Jose M. Campistol, Jose M. Morales. Madrid, Madrid, Spain; Barcelona, Barcelona, Spain; Madrid, Madrid, Spain; Cordoba, Spain.

2:20 PM **HETEROGENEITY OF BONE HISTOLOGY IN HYPERCALCEMIC KIDNEY TRANSPLANT RECIPIENTS: DIAGNOSTIC VALUES OF BIOCHEMICAL MARKERS OF BONE REMODELING. (Abstract #20)**
Marie Lafage-Proust, Lionel Rostaing, Jean Cisterne, Pierre Bories, Anne Caillot-Augusseau, Dominique Durand. Toulouse, France; Saint-Etienne, France.

2:30 PM **LYMPHOCELES AFTER KIDNEY TRANSPLANTATION - WHAT'S THE BEST APPROACH FOR TREATMENT? (Abstract #21)**
Brian Grubbs, Dan Zapzalka, David Hunter, Arthur Matas, Abhi Humar. Minneapolis, MN.

2:40 PM **CARDIOVASCULAR (CV) RISK PROFILE IN RENAL ALLOGRAFT RECIPIENTS WITH *DE NOVO* POST-TRANSPLANT DIABETES (PTDM). (Abstract #22)**
Fernando G. Cosio, Todd E. Pesavento, Kwame Osei, Mitchell L. Henry, Ronald M. Ferguson. Columbus, OH; Columbus, OH; Columbus, OH.

2:50 PM **PERIOPERATIVE MI IN RENAL TRANSPLANT RECIPIENTS. (Abstract #23)**
Connie L. Manske, Than Oo, Arthur J. Matas.

3:00 PM **SIROLIMUS-BASED IMMUNOSUPPRESSION FOR IMMUNOPROPHYLAXIS OF ACUTE ALLOGRAFT REJECTION IN PATIENTS WITH CALCINEURIN-INHIBITOR INDUCED HEMOLYTIC UREMIC SYNDROME. (Abstract #24)**
Alan Leichtman, the Sirolimus HUS Compassionate Use Study Investigators. Ann Arbor, MI.

3:10 PM **THE TREATMENT OF HYPERLIPIDEMIA IN RENAL TRANSPLANT RECIPIENTS: DO WE PRACTICE WHAT WE PREACH? (Abstract #25)**
Sandra M. Cockfield, Loreen Wales, Denise Lam. Edmonton, AB, Canada.

3:20 PM **THE USE OF MONO/POLICLONAL ANTIBODIES DOES NOT NEGATIVELY INFLUENCE THE OUTCOME OF LIVER DISEASE AT FIVE YEARS AFTER RENAL TRANSPLANTION IN HEPATITIS C VIRUS POSITIVE PATIENTS. (Abstract #26)**
Beatriz Dominguez-Gil, Nuria Esforzado, Miguel A. Muñoz, Amado Andres, Federico Oppenheimer, Jose L. Rodicio, Jose M. Campistol, Jose M. Morales. Madrid, Madrid, Spain; Barcelona, Barcelona, Spain.

Concurrent Session 3: Basic Science: Immunosuppression/Tolerance

2:00 PM - 3:30 PM

Chicago Ballroom 8, Sheraton
Chairs: Roslyn Mannon and Giacomo Basadonna

2:00 PM **STEALTH, THE 3RD MODEL OF PRIMATE TOLERANCE: EVIDENCE FOR SUSTAINED TH2 CYTOKINE PRODUCTION IN THE LONG TERM (LT) SURVIVORS. (Abstract #27)**
Judith M. Thomas, Andrew Lobashevsky, Cheryl A. Smyth, William Hubbard, Devin Eckhoff, Juan L. Contreras, David Neville, Francis T. Thomas. Birmingham, AL.

2:10 PM **CAN IMMUNOLOGIC UNRESPONSIVENESS IN RHESUS MONKEYS PREDICT A SUCCESSFUL TOLERANCE STRATEGY IN HUMANS? (Abstract #28)**
Stuart J. Knechtle, John H. Fechner, Clifford S. Cho, Kevin G. Brunner, Yinchen Dong, Majed M. Hamawy. Madison, WI.

2:20 PM **NEW INSIGHTS INTO THE INTERACTION BETWEEN T CELL COSTIMULATION BLOCKADE AND CONVENTIONAL IMMUNOSUPPRESSION IN VIVO. (Abstract #29)** *Young Investigator Award*
Masayuki Sho, Nader Najafian, Victor M. Dong, Koji Kishimoto, Akira Yamada, Sigrid E. Sandner, Mohamed H. Sayegh. Boston, MA.

2:30 PM **DENDRITIC CELLS TREATED WITH ANTISENSE OLIGODEOXYRIBONUCLEOTIDES TARGETING CD80 OR CD86 mRNA PROLONG CARDIAC ALLOGRAFT SURVIVAL. (Abstract #30)**
Xiaoyan Liang, Lina Lu, Zongyou Chen, Hong Zhang, Nicholas M. Dean, John J. Fung, Shiguang Qian. Pittsburgh, PA; San Diego, CA.

2:40 PM **EFFECT OF INCREASING ANTI-CD40L (IDEC 131) ANTIBODY DOSE AND CONCOMITANT T-CELL DEPLETION ON PRIMATE ALLOGRAFT SURVIVAL AND HISTOLOGY. (Abstract #31)**
Steffen Pfeiffer, George L. Zorn, III, Agnes M. Azimzadeh, James Atkinson, Robert Newman, Richard N. Pierson, III. Nashville, TN; Nashville, TN; San Diego, CA.

2:40 PM **LONG-TERM METABOLIC STUDIES IN STREPTOZOTIN (STZ) INDUCED PRIMATE RECIPIENTS AFTER TOLERANCE INDUCTION TO ALLOGENIC PANCREATIC ISLET TRANSPLANTS (PIT). (Abstract #32)**
Judith M. Thomas, Juan Conteras, Cheryl Smyth, Devin Eckhoff, Andrew Lobashevsky, Francis T. Thomas. Birmingham, AL.

3:00 PM **SURVIVAL OF PANCREATIC ISLET ALLOGRAFTS IN DIABETIC CYNOMOLOGYS MONKEYS. (Abstract #33)**
Maria Koulmanda, Andi S. Qipo, Neal R. Smith, Tatsuo Kawai, Jack Oneil, Jim Rusche, Dicken Ko, Gordon Weir, Terry Strom, Hugh Auchincloss, Jr. Boston, MA.

3:10 PM **PIRFENIDONE DECREASES TGF-β1 EXPRESSION AND AMELIORATES FIBROSIS IN CHRONIC CYCLOSPORINE NEPHROTOXICITY. (Abstract #34)**
Fuad S. Shihab, William M. Bennett, Hong Yi, Takeshi F. Andoh. Salt Lake City, UT; Portland, OR.

3:20 PM **ATORVASTATIN PREVENTS CHRONIC REJECTION IN RAT CARDIAC ALLOGRAFTS. (Abstract #35)**
Ping Ji, Yale D. Podnos, Ming-Sing Si, Earl Steward, David K. Imagawa. Orange, CA.

Concurrent Session 4: Basic Science: Rejection I

2:00 PM - 3:30 PM

Chicago Ballroom 9, Sheraton
Chairs: Thomas Pearson and Gregg Hadley

2:00 PM **HEME OXYGENASE 1 (H0-1) GENE TRANSFER DELAYED ALLOGRAFT ACUTE REJECTION IN A RAT MODEL. (Abstract #36)**
Christine Chauveau, Cecile Braudeau, Cecile Guillot, Claire Usal, Suhasini Iyer, Jean Paul Soulillou, Marie Cristina Cuturi, Roland Buelow, Ignacio Anegon. Nantes, France; Fremont, CA.

2:10 PM **SELECTIVE BLOCKADE OF CD28-B7 INTERACTION, BUT NOT OF CTLA4-B7, WITH A SCFV-ALPHA 1 ANTITRYPSIN FUSION PROTEIN. (Abstract #37)**
Bernard Vanhove, Geneviève Laflamme, Flora Coulon, Daniel Olive, Roland Buelow, Jérôme Tiollier, Jean-Paul Soulillou. Nantes, France; Lyon, France; Marseille, France.

2:20 PM **BLOCKADE OF THE MEMBRANE LYMPHOTOXIN PATHWAY INHIBITS $CD8^+$ T CELL-MEDIATED REJECTION. (Abstract #38)**
Jun Wang, Zhong Guo, Lingzhong Meng, Qiang Wu, Oliver Kim, John Hart, Gang He, J. Richard Thistlethwaite, Maria-Luisa Alegre, Yang-Xin Fu, Kenneth A. Newell. Chicago, IL.

2:30 PM **INDUCTION OF TH2 RESPONSE TO CARDIAC MYOSIN PREVENTS ACUTE REJECTION OF ALLOGENEIC HEART GRAFTS IN MICE. (Abstract #39)**
Eugenia V. Fedoseyeva, Koji Kishimoto, Hillary Rolls, Victor Dong, Anna Valujskikh, Peter S. Heeger, Mohamed H. Sayegh, Gilles Benichou. Boston, MA; Boston, MA; Cleveland, OH.

2:40 PM **STRATEGY FOR PREVENTION OF VASCULAR REJECTION: INHIBITION OF ECTODOMAIN SHEDDING OF HEPARIN-BINDING EPIDERMAL GROWTH FACTOR-LIKE GROWTH FACTOR BLOCKS MIGRATION AND PROLIFERATION OF VASCULAR SMOOTH MUSCLE CELLS IN RAT AORTIC ALLOGRAFTS. (Abstract #40)**
Ildeok Kim, Shigeki Higashiyama, Masanori Nakamura, Hiroto Egawa, Koichi Tanaka. Kyoto, Japan; Suita, Japan; Osaka, Japan.

2:50 PM **INHIBITION OF DONOR BRAIN DEATH-RELATED INFLAMMATION OF TANSPLANTED KIDNEYS BY RECOMBINANT SOLUBLE P-SELECTIN GLYCOPROTEIN LIGAND (sPSGL). (Abstract #41)**
Martin Gasser, Ana Maria Waaga, Igor Laskowski, Miriam S. Lenhard, Gray D. Shaw, Wayne W. Hancock, Nicholas L. Tilney. Boston, MA; Cambridge, MA; Cambridge, MA.

3:00 PM **ANTI-HLA ANTIBODY-MEDIATED ACTIVATION OF AIRWAY EPITHELIAL CELLS INDUCES THE PRODUCTION OF SEVERAL FIBROGENIC GROWTH FACTORS FOLLOWED BY APOPTOTIC CELL DEATH. (Abstract #42)**
Andres Jaramillo, Leiying Zhang, Elbert P. Trulock, G. Alexander Patterson, T. Mohanakumar. St. Louis, MO.

3:10 PM **ACTIVATION OF T CELLS IN AN INTESTINAL ALLOGRAFT TRIGGERS REJECTION BY INFLUENCING HOST T CELL ACTIVATION AND MIGRATION. (Abstract #43)**
Zheng J. Zhang, Terrance A. Barrett, Levent Kaptanoglu, David Ivancic, Frank P. Stuart, Jonathan P. Fryer. Chicago, IL.

3:20 PM **OVER EXPRESSION OF SMAD2 AND CO-LOCALIZATION WITH TGF-B1 IN CHRONIC REJECTION (CR). (Abstract #44)**
Xiao L. Jiang, Shenglin Ma, Clement Asiedu, Juan L. Contreras, Devin Eckhoff, Francis T. Thomas, Judith M. Thomas. Birmingham, AL.

Concurrent Session 5: Kidney Transplantation: Recipient Factors and Outcomes

2:00 PM - 3:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: Dave Conti and Robert Merion

2:00 PM **RENAL FUNCTION IN THE FIRST YEAR AFTER TRANPLANTATION PREDICTS LONGTERM SURVIVAL. (Abstract #45)**
Christopher P. Johnson, Maureen McBride, Wida S. Cherikh, Christine B. Tolleris, Barbara A. Bresnahan, Sundaram Hariharan. Milwaukee, WI; Richmond, VA; Milwaukee, WI.

2:10 PM **HAVE THE IMPROVEMENTS IN SURVIVAL IN RENAL TRANSPLANTATION EXCEEDED THE IMPROVEMENTS IN SURVIVAL ON DIALYSIS? (Abstract #46)**
Herwig-Ulf Meier-Kriesche, Friedrich K. Port, Akinlolu O. Ojo, Julie A. Arndorfer, Diane M. Cibrik, Bruce Kaplan. Ann Arbor, MI.

2:20 PM **THE IMPACT OF RENAL GRAFT DYSFUNCTION ON GRAFT SURVIVAL IS VARIABLE AND DEPENDENT ON THE TYPE OF INJURY RESPONSIBLE FOR THE DYSFUNCTION. (Abstract #47)**
Viken Douzdjian, Ravi Parasuraman, Atsushi Yoshida, Marwan Abouljoud. Detroit, MI; Detroit, MI.

2:30 PM **PROTEINURIA AFTER RENAL TRANSPLANTATION INCREASES NOT ONLY THE RISK FOR GRAFT FAILURE BUT ALSO THE DEATH RISK. (Abstract #48)**
Joke I. Roodnat, Paul G.H. Mulder, Teun Gelder van, Jacqueline Rischen-Vos, Iza C. van Riemsdijk, Jan N.M. IJzermans, Willem Weimar. Rotterdam; Rotterdam; Rotterdam, The Netherlands.

2:40 PM **FUNCTIONAL RESERVE AND HYPERFILTRATION AFTER CADAVERIC RENAL TRANSPLANTATION. (Abstract #49)**
Elisabetta Bertoni, Alberto Rosati, Maria Zanazzi, Lorenzo Di Maria, Luciano Moscarelli, Marco Gallo, Maurizio Salvadori. Florence, Italy.

2:50 PM **EFFECT OF CALCIUM AND CHOLECALCIFEROL TREATMENT ON BONE MINERAL DENSITY IN RENAL TRANSPLANT RECIPIENTS. (Abstract #50)**
Martin Wissing, Nilufer Broeders, André Schoutens, Bernard Stallenberg, Brigitte Borré, Daniel Abramowicz. Brussels, Belgium; Brussels, Belgium; Brussels, Belgium.

3:00 PM **PRETRANSPLANT DIALYSIS MODALITY IS ASSOCIATED WITH LONG TERM RENAL ALLOGRAFT SURVIVAL AFTER CADAVERIC BUT NOT LIVING DONOR TRANSPLANTION. (Abstract #51)**
Brian J. Gallay, J. Michael Cecka, Richard V. Perez. Sacramento, CA; Los Angeles, CA; Sacramento, CA.

3:10 PM **ELEVATED BODY MASS INDEX (BMI) DOES NOT ADVERSELY EFFECT THE OUTCOME IN RENAL TRANSPLANT PATIENTS. (Abstract #52)**
Stuart Greenstein, Wanda Chin, Richard Schechner, Vivian Tellis.

3:20 PM **OVERWEIGHT IN RENAL TRANSPLANT RECIPIENTS IS ASSOCIATED WITH AN INCREASED RISK OF PROTEINURIA AND HYPERTENSION. (Abstract #53)**
Chew-Wong Alfredo, Ron Oscar, Ricalde Guadalupe, Romo Luis, Parra Ana Laura, Reyes-Acevedo Rafael. Aguascalientes, Mexico.

Concurrent Session 6: Kidney Transplantation: Factors Affecting Clinical Outcomes

2:00 PM - 3:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Bruce Kaplan and Thomas Peters

2:00 PM **IMPROVED RENAL FUNCTION WITH CYCLOSPORINE ELIMINATION IN SIROLIMUS-TREATED RENAL TRANSPLANT RECIPIENTS: ONE-YEAR RESULTS FROM A PHASE II TRIAL. (Abstract #54)**
Donald E. Hricik, for the Rapamune Renal Function Study Group. Cleveland, OH.

2:10 PM **CHRONIC ALLOGRAFT NEPHROPATHY UNIFORMLY AFFECTS HLA NON-IDENTICAL LIVING-RELATED, LIVING-UNRELATED AND CADAVERIC RECIPIENTS. (Abstract #55)**
Nancy R. Krieger, Dennis Heisey, Barbara J. Voss, Brian N. Becker, Yolanda T. Becker, Jon S. Odorico, Anthony M. D'Alessandro, Munci Kalayoglu, John D. Pirsch, Hans W. Sollinger, Stuart J. Knechtle. Madison, WI.

2:20 PM **AN HLA-IDENTICAL LRD KIDNEY TRANSPLANT IS NOT A RISK FACTOR FOR RENAL ALLOGRAFT LOSS IN PATIENTS WITH FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS). (Abstract #56)**
Diane M. Cibrik, Bruce Kaplan, Alan B. Leichtman, Darrell A. Campbell, Herwig-Ulf Meier-Kriesche. Ann Arbor, MI; Ann Arbor, MI.

2:30 PM **IMPACT OF MYCOPHENOLATE MOFETILE (MMF) ON CHRONIC ALLOGRAFT NEPHROPATHY (CAN) IN CHILDREN. (Abstract #57)**
Thomas Henne, Gisela Offner, Kai Latta, Barbara Enke, Christian V. Schnakenburg, Jochen H.H. Ehrich, Juergen Strehlau. Hannover, Germany.

2:40 PM **ANTI-CD20 MONOCLONAL ANTIBODY RESCUE THERAPY FOR REFRACTORY ANTIBODY-MEDIATED REJECTION. (Abstract #58)**
Milagros Samaniego, Henkie Tan, Karen King, Robert Montgomery, Mark Haas, Lloyd Ratner, Andrea Zachary. Baltimore, MD.

2:50 PM **SYNERGISTIC HLA-DR MATCHING EFFECT AMONG SENSITIZED RECIPIENTS WHO RECEIVED ELDERLY DONOR KIDNEYS. (Abstract #59)**
Yong W. Cho, J. Michael Cecka. Los Angeles, CA.

3:00 PM **IMPROVED RESULTS IN HIGH IMMUNOLOGIC RISK RENAL ALLOGRAFT RECIPIENTS USING DACLIZUMAB (ZENAPAX ®) VERSUS TRADITIONAL ANTI-LYMPHOCYTE THERAPY IN MEMBER CENTERS OF THE SOUTH-EASTERN ORGAN PROCUREMENT FOUNDATION. (Abstract #60)**
Francis H. Wright, Leroy R. Thacker, II, Thomas G. Peters. San Antonio, TX; Richmond, VA; Jacksonville, FL.

3:10 PM **LONG-TERM OUTCOME AFTER TREATMENT OF EARLY REFRACTORY ACUTE HUMORAL REJECTION IN KIDNEY TRANSPLANTATION. (Abstract #61)**
Tom Theruvath, Francis Delmonico, Susan Saidman, Nina Tolkoff-Rubin, Winfred Williams, Shamila Mauiyyedi, Marylin Farrell, Bernard Collins, Robert Colvin, A. Benedict Cosimi, Manuel Pascual. Boston, MA; Boston, MA.

3:20 PM **SERUM CREATININE (CR) AT ONE AND SIX MONTHS PREDICTS LONG-TERM ALLOGRAFT SURVIVAL: A SINGLE CENTER ANALYSIS. (Abstract #62)**
Sundaram Hariharan, Yong-Ran Zhu, Allan M. Roza, Mark B. Adams, Christopher P. Johnson. Milwaukee, WI; Milwaukee, WI.

Concurrent Session 7: Xenotransplantation: Preclinical Non-Human Primate

2:00 PM - 3:30 PM

Empire Room, Intercontinental
Chairs: Richard Pierson and Jonathon Fryer

2:00 PM **DIFFERENT IGG SUBCLASSES OF ANTI-GAL MABS DIFFERENTIALLY INDUCE XENOGRAFT REJECTION. (Abstract #63)**
Dengping Yin, LianLi Ma, Jikun Shen, Hui Xu Xu, John Logan, Guerard Byrne, Anita S. Chong. Chicago, IL, United States; Princeton, NJ.

2:10 PM **THE LEVEL OF ANTI-GAL IgM BEFORE TRANSPLANTATION CORRELATES WITH XENOGRAFT SURVIVAL IN hDAF PIG-TO- BABOON HETEROTOPIC HEART TRANSPLANTATION. (Abstract #64)**
Rafael Manez, Fabian Crespo, Alberto Juffe, Alberto Centeno, Eduardo Lopez-Pelaez, Emanuele Cozzi, David J. White. La Coruna, Spain; Cambridge, United Kingdom.

2:20 PM **INFUSION OF GAL TYPE 6 OLIGOSACCHARIDES IN BABOONS RESULTS IN TOTAL DEPLETION OF ANTI-PIG ANTIBODIES. (Abstract #65)** *Young Investigator Award*
Katsuhito Teranishi, Bernd Gollackner, Leo Buhler, Cristoph Knosalla, David H. Sacks, Michel Awwad, David K.C. Cooper. Boston, MA; Charlestown, MA.

2:30 PM **GAS 914, A POLYLYSINE CONTAINING αGAL, REDUCES THE SEVERITY OF ACUTE HUMORAL REJECTION IN hDAF PIG TO PRIMATE HETEROTOPIC HEART XENOTRANSPLANTATION. (Abstract #66)**
Rafael Manez, Alberto Centeno, Eduardo Lopez-Pelaez, Carmen Ruiz de Valbuena, Alberto Juffe, Beverly Holmes, Rudolph Duthaler, Andreas Katopodis. La Coruna, Spain; Cambridge, United Kingdom; Basel, Switzerland.

2:40 PM **THE COMBINED EFFECTS OF PHARMACOLOGIC NEUTRALIZATION OF ANTI-αGAL ANTIBODIES AND COMPLEMENT INHIBITION ON SURVIVAL OF hDAF TRANSGENIC PIG RENAL GRAFTS IN CYNOMOLGUS MONKEYS. (Abstract #67)**
Bernard Hausen, Tuan Lam, Laune Hook, Katrin Boeke, Uwe Christians, Wolfgang Jacobsen, John Higgins, Emanuele Cozzi, Hugh Davies, Rudolf Duthaler, Richard Harrison, Andreas Katopodis, Randall Morris. Stanford, CA; San Francisco, CA; Cambridge, United Kingdom; Basel, Switzerland.

2:50 PM **C1-INHIBITOR (C1-INH) FOR TREATMENT OF ACUTE VASCULAR XENOGRAFT REJECTION (AVR) IN CYNOMOLGUS RECIPIENTS OF PORCINE KIDNEYS. (Abstract #68)**
Jens M. Hecker, Ralf Lorenz, Richard Appiah, Martin Loss, Michael Przemeck, Jan Schmidtko, Arman Jalali, Burkhard Vangerow, Horst Rueckoldt, Juergen Klempnauer, Michael Winkler. Hannover, Germany; Hannover, Germany.

3:00 PM **EVALUATION OF THE EXTRACORPOREAL LIVER PERFUSION SYSTEM USING HUMAN DECAY ACCELERATING FACTOR TRANSGENIC PIG LIVER IN DIRECT CROSS-CIRCULATION WITH BABOON. (Abstract #69)**
Takakazu Matsushita, Iwao Ikai, Ryuta Nishitai, Nagato Katsura, Hiroshi Okabe, Satoshi Yamanokuchi, Koichi Matsuo, Tomohiro Shiotani, Hiroaki Terajima, Yoshio Yamaoka. Kyoto, Japan.

3:10 PM **XENOGENEIC THYMOKIDNEY TRANSPLANTATION IN A PIG-TO-BABOON MODEL: EVIDENCE OF SPECIFIC T CELL UNRESPONSIVENESS. (Abstract #70)** *Young Investigators Award*
Rolf N. Barth, John C. LaMattina, Shin Yamamoto, Naoki Kumagai, Leo Buhler, Hiroshi Kitamura, Michel Awwad, David K.C. Cooper, Megan Sykes, David H. Sachs, Kazuhiko Yamada. Boston, MA; Boston, MA.

3:20 PM **XENOGENEIC PIG ISLETS ESCAPE REJECTION IN IMMUNOSUPPRESSED PRIMATES BUT FAIL TO SUSTAIN NORMOGLYCEMIA. (Abstract #71)**
Martin Wijkstrom, Nicole Kirchhof, Kristin J. Pilon, Raja Kandaswamy, Sue Clemmings, Alison Trexler, Tom Gilmore, David E.R. Sutherland, Bernhard J. Hering. Minneapolis, MN.

Concurrent Session 8: Lung Transplantation: Bench-to-Bedside

2:00 PM - 3:30 PM

Exchange Room, Intercontinental
Chairs: Marshall Hertz and James Allan

2:00 PM **QUANTITATIVE MEASUREMENT OF TNF-α IN BRONCHOALVEOLAR LAVAGE SAMPLES: A POTENTIAL MARKER OF LUNG TRANSPLANT REJECTION. (Abstract #72)**
Tehmina Z. Ali, Sean M. Studer, Jodi L. Layton, Jonathan B. Orens, William M. Baldwin, Barbara A. Wasowska. Baltimore, MD; Baltimore, MD.

2:10 PM **INDIRECT RECOGNITION OF MISMATCHED DONOR HLA CLASS II PEPTIDES IN PEDIATRIC LUNG TRANSPLANT RECIPIENTS WITH BRONCHIOLITIS OBLITERANS SYNDROME. (Abstract #73)**
Kim C. Lu, Eric Mendeloff, Charles B. Huddleston, T. Mohanakumar. St. Louis, MO; St. Louis, MO.

2:20 PM **INTERLEUKIN 6 AND TUMOR NECROSIS FACTOR-α POLYMORPHISMS CORRELATE WITH BRONCHIOLITIS OBLITERANS SYNDROME IN LUNG TRANSPLANT RECIPIENTS. (Abstract #74)**
Kim C. Lu, Rachel L. Lecha, Aviva Aloush, Eric Mendeloff, Elbert P. Trulock, G. A. Patterson, T. Mohanakumar. St. Louis, MO; St. Louis, MO; St. Louis, MO.

2:30 PM **CELL-MEDIATED IMMUNITY TO COLLAGEN V IN LUNG TRANSPLANT RECIPIENTS: CORRELATION WITH COLLAGEN V RELEASE INTO BAL FLUID. (Abstract #75)**
David S. Wilkes, Kathleen M. Heidler, Kazu Yasufuku, Lynn DeVito-Haynes, Ewa Jankowska-Gan, Keith Meyers, Robert Love, William J. Burlingham. Indianapolis, IN; Madison, WI.

2:40 PM **UPREGULATION OF P-GP PROTECTS GRAFT INFILTRATING T-CELLS FROM APOPTOSIS: A MECHANISM FOR IMMUNOSUPPRESSIVE DRUG RESISTANCE. (Abstract #76)** *Young Investigators Award*
Vera S. Donnenberg, Gilbert J. Burckart, John W. Wilson, Adriana Zeevi, Bartley P. Griffith, Aldo Iacono, Albert D. Donnenberg. Pittsburgh, PA.

2:50 PM **LONG TERM RENAL FUNCTION IN PEDIATRIC LUNG TRANSPLANT RECIPIENTS. (Abstract #77)**
S. P. Hmiel, Stuart C. Sweet, Anne M. Beck. St. Louis , MO.

3:00 PM **COMMUNITY ACQUIRED RESPIRATORY VIRUSES IN LUNG TRANSPLANT PATIENTS: INCIDENCE, DIAGNOSTIC METHODS AND OUTCOMES. (Abstract #78)**
Tony N. Hodges, Fernando Torres, Adriana Weinberg, Shaobing Li, Martin R. Zamora. Denver, CO; Denver, CO.

3:10 PM **EMERGENCE OF GANCICLOVIR RESISTANT CYTOMEGALOVIRUS IN LUNG TRANSPLANT RECIPIENTS. (Abstract #79)**
Ashby Jordan, Sangeeta M. Bhorade, Nell S. Lurain, Julie Leischner, Jaime Villanueva, Wickii T. Vigneswaran, Edward R. Garrity. Maywood, IL; Chicago , IL; Maywood, IL.

3:20 PM ***NOCARDIA* INFECTION IN LUNG TRANSPLANT RECIPIENTS. (Abstract #80)**
Shahid Husain, Kenneth R. McCurry, Nina Singh, James H. Dauber, Shimon Kusne. Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA.

Concurrent Session 9: Liver Transplantation: Hepatitis C Clinical Outcomes

2:00 PM - 3:30 PM

Grand Ballroom, Intercontinental
Chairs: Norah Terrault and Kim Olthoff

2:00 PM **THE OUTCOME OF LIVERS FROM HCV+ DONORS. (Abstract #81)**
Ergun Velidedeoglu, Niraj M. Desai, Luis Campos, Kim M. Olthoff, Avi Shaked, Lisa M. Forman, Frederick A. Nunes, Gilian A. Zeldin, Charmaine A. Stewart, Emily Blumberg, John Abrams, Michael R. Lucey, James F. Markmann. Philadelphia, PA; Philadelphia, PA; Philadelphia, PA.

2:10 PM **ORTHOTOPIC LIVER TRANSPLANTATION FOR HEPATITIS C: ANALYSIS OF ALLOGRAFT SURVIVAL USING THE UNOS DATABASE. (Abstract #82)**
Lisa M. Forman, Michael R. Lucey. Philadelphia, PA; Philadelphia, PA.

2:20 PM **IMPACT OF HEPATITIS C ON LIVER TRANSPLANT SURVIVAL OVER TEN YEARS- A REPORT OF THE SEOPF LIVER COMMITTEE. (Abstract #83)**
Jon W. Jones, Members of the SEOPF Liver Committee. Charlotte, NC.

2:30 PM **ADVERSE EFFECTS OF HCV RECURRENCE IN OVER FIVE HUNDRED LIVER TRANSPLANTATIONS. (Abstract #84)**
Rafik M. Ghobrial, Douglas G. Farmer, Randy Stedman, Hasan Yersiz, Charles Lassman, Natale Danino, Eric Collisson, Steve Han, Sammy Saab, Ronald W. Busuttil. Los Angeles, CA; Los Angeles, CA; Los Angeles, CA; Los Angeles, CA.

2:40 PM **HCV VIRAL LOAD AT ONE MONTH AFTER LIVER TRANSPLANTATION IS A PROGNOSTIC FACTOR FOR THE DEVELOPMENT OF RECURRENT HEPATITIS C AND LIVER FAILURE AFTER TRANSPLANTATION. (Abstract #85)**
Francisco Suarez, Alejandra Otero, Manuel Gomez-Gutierrez, Francisco Arnal, Carlos Fernandez-Selles, Jose Luis Vazquez-Iglesias, Rafael Manez. La Coruna, Spain.

2:50 PM **INCREASED MORTALITY RISK IN HEPATITIS C-POSITIVE LIVER TRANSPLANT RECIPIENTS WHO DEVELOP POSTTRANSPLANT DIABETES MELLITUS. (Abstract #86)**
S. Baid, A. B. Cosimi, M. L. Farrell, D. A. Schoenfeld, S. Feng, D. Ko, R. T. Chung, N. Tolkoff-Rubin, M. Pascual. Boston, MA.

3:00 PM **CRYOGLOBULINEMIA PREDICTS POOR OUTCOME FOLLOWING LIVER TRANSPLANTATION IN PATIENTS WITH HEPATITIS C. (Abstract #87)**
Stephen C. Rayhill, Warren N. Schmidt, Adel Bozorgzedeh, Daniel Katz, Michael D. Voight, Douglas R. LaBrecque, Rachel Miller, Mohammed Ibrahim, Patricia A. Kirby, Frank A. Mitros, Youmin Wu. Iowa City, IA; Iowa City, IA; Iowa City, IA.

3:10 PM **EARLY RECURRENCE OF HEPATITIS C AFTER LIVER TRANSPLANTATION WITH DACLIZUMAB INDUCTION. (Abstract #88)**
Gustavo Marino, Vinod K. Rustgi, Carlos E. Marroquin, Jeffrey S. Plotkin, Paul C. Kuo, Amy Lu, Scott Batty, Lynt B. Johnson. Washington, DC; Washington, DC; Washington, DC; Washington, DC.

3:20 PM **PEGYLATED (40 kDa) INTERFERON ALFA-2A (PEGASYS®) IN POST-LIVER TRANSPLANT RECIPIENTS WITH ESTABLISHED RECURRENT HEPATITIS C: A PRELIMINARY REPORT. (Abstract #89)**
Caroline Riely, Peter Ferenci, Markus Peck-Radosavljevic, Wolfgang Vogel, Michael Voigt, Ian M. Marks, Stephen C. Pappas. Memphis, TN; Vienna, Austria; Innsbruck, Austria; Iowa City, IA; Nutley, NJ.

Concurrent Session 10: Immunosuppression for Pancreas Transplantation

2:00 PM - 3:30 PM

Renaissance Ballroom, Intercontinental
Chairs: Osama Gaber and Hans Sollinger

2:00 PM **IMPROVEMENTS FROM STEROID WITHDRAWAL AFTER PANCREAS TRANSPLANTATION: 1-YEAR RESULTS OF A PROSPECTIVE, RANDOMIZED OPEN-LABEL STUDY. (Abstract #90)**
Rainer W.G. Gruessner, David E.R. Sutherland, Elizebeth Parr, Abhinav Humar, Raja Kandaswamy, Angelika C. Gruessner. Minneapolis, MN.

2:10 PM **RAPID CORTICOSTEROID WITHDRAWAL IN SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. (Abstract #91)**
Dixon B. Kaufman, Joseph R. Leventhal, Lorenzo G. Gallon, Michele A. Parker, Frank P. Stuart. Chicago, IL; Chicago, IL.

2:20 PM **A MULTICENTER, OPEN-LABEL, COMPARATIVE TRIAL OF 2 DACLIZUMAB DOSING STRATEGIES VS. NO ANTIBODY INDUCTION IN COMBINATION WITH TACROLIMUS, MYCOPHENOLATE MOFETIL, AND STEROIDS IN SIMULTANEOUS KIDNEY-PANCREAS TRANSPLANTATION: 6 MONTH ANALYSIS. (Abstract #92)**
R. J. Stratta, R. R. Alloway, A. Lo, E. Hodge, The PIVOT Investigators. Memphis, TN; Cincinnati, OH.

2:30 PM **TACROLIMUS (TAC) AND MYCOPHENOLATE MOFETIL (MMF) +/- ANTIBODY INDUCTION IN SIMULTANEOUS PANCREAS KIDNEY (SPK) TRANSPLANTATION: ONE YEAR RESULTS. (Abstract #93)**
G. W. Burke, D. B. Kaufman, D. S. Bruce, D. Sutherland, C. P. Johnson, A. O. Gaber, R. M. Merion, E. Schweitzer, C. L. Marsh, S. A. Gruber, E. Alfrey, J. P. Leone, W. Conception, M. D. Stegall, P. S. Gores, G. Danovitch, P. J. Nunnally, A. K. Henning, W. E. Fitzsimmons. Miami, FL.

2:40 PM **MANAGEMENT OF TACROLIMUS-INDUCED HYPERGLYCEMIA FOLLOWING PANCREAS TRANSPLANTATION. (Abstract #94)**
Benjamin Philosophe, Anne M. Wiland, David K. Klassen, Eugene J. Schweitzer, Alan C. Farney, John O. Colonna, Clarence Foster, Adam M. Frank, Bruce E. Jarrell, Stephen T. Bartlett. Baltimore, MD.

2:50 PM **TACROLIMUS VERSUS CYCLOSPORINE IN PRIMARY SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. PRELIMINARY RESULTS OF A MULTICENTRE TRIAL. (Abstract #95)**
F. Saudek, J. Malaise, R. Margreiter, EUROSPK Study Group. Brussels, Belgium.

3:00 PM **EXPERIENCE WITH RAPAMYCIN IN PANCREAS TRANSPLANTATION. (Abstract #96)**
Jon S. Odorico, John D. Pirsch, Yolanda T. Becker, Bryan N. Becker, Hans W. Sollinger. Madison, WI.

3:10 PM **>10 YEAR FOLLOW-UP AFTER PANCREAS TRANSPLANTATION. (Abstract #97)**
Rainer W.G. Gruessner, David E.R. Sutherland, David L. Dunn, John S. Najarian, Angelika C. Gruessner. Minneapolis, MN.

3:20 PM **PANCREAS AFTER KIDNEY (PAK) VS. SIMULTANEOUS PANCREAS KIDNEY (SPK) TRANSPLANTS: A COMPARISON OF WAITING TIMES, COST, AND QUALITY OF LIFE. (Abstract #98)**
Abhi Humar, Raja Kandaswamy, Thiagarjan Ramcharan, Steven Paraskevas, Rainer W. Gruessner, Angelika Gruessner, David E.R. Sutherland. Minneapolis, MN.

3:30 PM **Break**

Concurrent Session 11: Control of Alloreactive T Cells

4:00 PM - 5:30 PM

Chicago Ballroom 10, Sheraton
Chairs. Olivia Martinez and Ashwani Khanna

4:00 PM **CORRELATION BETWEEN TH1 AUTOIMMUNE RESPONSES TO CARDIAC MYOSIN AND CHRONIC REJECTION OF HEART ALLOGRAFTS. (Abstract #99)**
Victor Dong, Koji Kishimoto, Hillary Rolls, Masayuki Sho, Mohamed H. Sayegh, Gilles Benichou, Eugenia V. Fedoseyeva. Boston, MA; Boston, MA.

4:10 PM **MOUSE CD4+ AND CD8+ T CELLS DISPLAY DIFFERENCES IN ACTIVATION AFTER DIRECT STIMULATION BY ALLOGENEIC VASCULAR ENDOTHELIUM. (Abstract #100)**
Daniel Kreisel, Alexander S. Krupnick, Wilson Y. Szeto, Sicco H. Popma, Alyssa M. Krasinskas, Bruce R. Rosengard. Philadelphia, PA; Philadelphia, PA.

4:20 PM **DIFFERENTIAL LOCALIZATION OF INTERLEUKIN-2- AND -15 RECEPTOR CHAINS IN MEMBRANE RAFTS OF HUMAN T CELLS. (Abstract #101)**
Jens Goebel, Kathy Forrest, Lorri Morford, Thomas L. Roszman. Lexington, KY; Lexington, KY.

4:30 PM **ISLET GRAFT SURVIVAL IS INCREASED IN MICE TREATED WITH AN IL-15 MUTANT/Fc PROTEIN THROUGH THE CONTROL OF CD8+ T-CELL PROLIFERATION. (Abstract #102)**
Sylvie Ferrari Lacraz, Xin Xiao Zheng, Alberto Sanchez-Fueyo, Wlodzimierz Maslinski, Terry B. Strom. Boston, MA.

4:40 PM **T CELL HOMEOSTATIC PROLIFERATION - FUNCTIONAL GAIN AND ROLE OF COSTIMULATION. (Abstract #103)**
Hrefna Gudmundsdottir, Laurence A. Turka.

4:50 PM **CD40: TURNING OFF "OFF SIGNALS" TO REGULATE GENE TRANSCRIPTION. (Abstract #104)**
Michael Melter, Peter H. Lapchak, Soumitro Pal, Christopher Geehan, Debabrata Mukhopadhyay, David M. Briscoe. Boston, MA; Boston, MA; Boston, MA.

5:00 PM **COSTIMULATION PATHWAYS IN THE DIFFERENTIATION OF ALLOREACTIVE CYTOTOXIC T CELLS. (Abstract #105)**
Yuan Zhai, Feng Gao, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA.

5:10 PM **FASL IS REQUIRED FOR COSTIMULATION INDEPENDENT IFN-γ SECRETION AND CYTOTOXICITY. (Abstract #106)**
Joel Trambley, Matthew A. Williams, Angello Lin, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

5:20 PM **RECOMBINANT DIMERIC MHC CLASS I MOLECULES STAIN IN VITRO GENERATED ALLOREACTIVE T-CELLS AND SHOW A CRITICAL ROLE FOR CD8. (Abstract #107)**
Kyoung-Ae Yoo-Ott, Birgit Fricke, Judith Steude, Nicholas Zavazava. Kiel, Germany.

Concurrent Session 12: Risk Analysis in Renal Transplantation

4:00 PM - 5:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs· Ram Peddi and Gary Singer

4:00 PM **A STUDY OF CYTOKINE SECRETION: EFFECT OF GENOTYPIC VARIATIONS IN THE TNF-A, IL-10 AND IFN-G GENES ON THE IN-VITRO CYTOKINE PRODUCTION. RELEVANCE FOR LATE-ONSET PTLD. (Abstract #108)**
Athanasios Vergopoulos, Nina Babel, Ian Hutchinson, Conny Hoeflich, Hans Dieter Volk, Petra Reinke. Berlin, Germany; United Kingdom.

4:10 PM **IMPACT OF DE NOVO PRODUCED ALLOREACTIVE CYTOTOXIC ANTIBODIES AFTER KIDNEY TRANSPLANTATION. (Abstract #109)**
Marco Miozzari, Samuel Henz, Margrith Disler, Daniela Garzoni, Daniel Hertner, Markus Fopp, Rudolf P. Wuthrich. St. Gallen, Switzerland.

4:20 PM **TNFβ GENE POLYMORPHISMS PREDICT URINARY TRACT INFECTIONS FOLLOWING RENAL TRANSPLANTATION. (Abstract #110)**
Pam M. Kimball, Cecil Rhodes. Richmond, VA.

4:30 PM **ROLE OF ANTI-β2 GLYCOPROTEIN 1 ANTIBODIES IN ESRD PATIENTS WITH ANTIPHOSPHOLIPID ANTIBODY SYNDROME. (Abstract #111)**
Smita Vaidya, Todd Y. Cooper, John A. Daller, Kristene K. Gugliuzza. Galveston, TX; Galveston, TX.

4:40 PM **FASTING AND POST-METHIONINE LOAD HOMOCYSTEINE ARE ASSOCIATED WITH PROGRESSION OF CAROTID ATHEROSCLEROSIS IN RENAL TRANSPLANT RECIPIENTS. (Abstract #112)**
Andrew A. House, David Freeman, Anthony M. Jevnikar, Norman Muirhead, David J. Hollomby, Kelly B. Zamke, J. David Spence. London, ON, Canada; London, ON, Canada.

4:50 PM **CLINICAL CHARACTERISTICS OF SIROLIMUS ASSOCIATED PNEUMONITIS IN RENAL TRANSPLANT PATIENTS. (Abstract #113)**
Emmanuel Morelon, Marc Stern, Marie-france Mamzer-Bruneel, Marie-Noelle Péraldi, Henri Kreis. Paris, France; Suresnes, France.

5:00 PM **RENAL TRANSPLANTATION IN HIV+ PATIENTS. (Abstract #114)**
Peter G. Stock, Michelle Roland, Laurie Carlson, Stephen Tomlanovich, Willliam Amend, Tom Coates, Chris Freise, John P. Roberts, Nancy L. Ascher. San Francisco, CA; San Francisco, CA.

5:10 PM **RISK FACTORS FOR PNEUMOCYSTIS CARINII PNEUMONIA IN KIDNEY TRANSPLANT RECIPIENTS. A CASE CONTROL STUDY. (Abstract #115)**
Marcelo V. Radisic, Roberta Lattes, Jennifer Fiore Chapman, María del Carmen Rial, Olga E. Guardia, Fabiana Seu, Domingo H. Casadei.

5:20 PM **THE ASSOCIATION OF WAITING TIME WITH POOR RENAL TRANSPLANT OUTCOME IS UNRELATED TO DIALYSIS MODALITY. (Abstract #116)**
Herwig-Ulf Meier-Kriesche, Friedrich K. Port, Julie A. Arndorfer, Alan B. Leichtman, Diane M. Cibrik, Bruce Kaplan. Ann Arbor, MI.

Concurrent Session 13: Basic Science: Immunosuppression I

4:00 PM - 5:30 PM

Chicago Ballroom 8, Sheraton
Chairs: Michael Abecassis and Michael Melter

4:00 PM **EFFECTS OF THE NOVEL IMMUNOMODULATOR FTY720 ON CIRCULATING B CELLS, NK CELLS, AND T CELLS EXPRESSING THE CHEMOKINE RECEPTORS CCR2, CCR5, CXCR4 AND CXCR3 IN KIDNEY TRANSPLANT PATIENTS. (Abstract #117)**
Leonard M.B. Vaessen, Wendy M. Mol, Jan N.M. IJzermans, Teun van Gelder, Willem Weimar. Rotterdam, The Netherlands.

4:10 PM **PROTEASOME TARGETING IN TRANSPLANTATION. (Abstract #118)** *Young Investigator Award*
Kerrie L. Faia, Wei Gao, Vilmos Csizmadia, Nida Shemmeri, Christine S. Pien, Julian Adams, Peter Elliott, Wayne W. Hancock. Cambridge, MA; Cambridge, MA.

4:20 PM **ANGIOTENSIN II (AII) REGULATION OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) AND ITS RECEPTORS FLT-1 AND FLK-1 IN CHRONIC CYCLOSPORINE NEPHROTOXICITY. (Abstract #119)**
Fuad S. Shihab, William M. Bennett, Hong Yi, Takeshi F. Andoh. Salt Lake City, UT; Portland, OR.

4:30 PM **PICEATANNOL, A SELECTIVE SYK/ZAP BLOCKER, IN COMBINATION WITH SUBTHERAPEUTIC DOSES OF CYCLOSPORIN A PROLONGS ALLOGRAFT SURVIVAL IN RATS. (Abstract #120)**
Gokhan Yagci, Luis Fernandez, Nobuhiro Ishido, Stuart J. Knechtle, Majed M. Hamawy. Madison, WI.

4:40 PM **INHIBITION OF JAK3 ALONE BLOCKS ALLOGRAFT REJECTION AND IS SYNERGISTIC IN COMBINATION WITH CYCLOSPORINE BUT NOT RAPAMYCIN. (Abstract #121)**
Stanislaw M. Stepkowski, Erwin-Cohen Rebecca, Fariba Behbod, Mou-Er Wang, Barry D. Kahan, Robert A. Kirken. Houston, TX; Houston, TX.

4:50 PM **SYNERGISTIC EFFECT OF TACROLIMUS WITH FK778 OR FK779 IN PREVENTION OF ACUTE HEART ALLOGRAFT REJECTION AND IN REVERSAL OF ONGOING ACUTE HEART ALLOGRAFT REJECTION IN THE RAT. (Abstract #122)**
Kupa K. Bilolo, Shijie Qi, Jun Ouyang, Xiang Wang, Dasheng Xu, Pierre Daloze, Ihor Bekersky, William E. Fitzsimmons, Huifang Chen. Montreal, QC, Canada; Deerfield, IL.

5:00 PM **TREATMENT WITH A SHORT COURSE OF LF15-0195 AND CONTINOUS CYCLOSPORINE A INHIBITS ACUTE XENOGRAFT REJECTION IN A RAT-TO-MOUSE CARDIAC TRANSPLANTATION MODEL. (Abstract #123)**
Hao Wang, Karoline A. Hosiawa, Bertha Garcia, Patrick Dutartre, Calvin Stiller, David J. Kelvin, Robert Zhong. London, ON, Canada; London, ON, Canada; London, ON, Canada; London, ON, Canada; London, ON, Canada; Daix, France.

5:10 PM **NEW MECHANISMS OF IMMUNOSUPPRESSION OF SIROLIMUS *IN VIVO*: DIFFERENTIAL SUPPRESSION OF MULTIPLE IMMUNE FUNCTIONS OF T AND B CELLS AND MONOCYTES IN NON-HUMAN PRIMATES. (Abstract #124)**
Camille Dambrin, Tudor Bîrsan, Jochen Klupp, Laurie Hook, Tuan Lam, Uwe Christians, Randall Morris. Stanford, CA; San Francisco, CA.

5:20 PM **MECHANISMS OF INDUCTION OF ALLOGRAFT TOLERANCE IN RAT BY SHORT-TERM TREATMENT WITH LF15-0195. (Abstract #125)**
Elise Chiffoleau, Patrick Dutartre, Claire Usual, Jean-Paul Soulillou, Maria-Cristina Cuturi. Nantes, France; Daix, France.

Concurrent Session 14: Basic Science: Rejection II

4:00 PM - 5:30 PM

Chicago Ballroom 9, Sheraton
Chairs: Kenneth A. Newell and Peter Nickerson

4:00 PM **ACUTE REJECTION OF A SKIN GRAFT EXPRESSING A DEFINED MINOR ANTIGEN. (Abstract #126)** *Young Investigator Award*
Benjamin D. Ehst, Marc K. Jenkins. Minneapolis, MN.

4:10 PM **IMMUNE RESPONSES TO HEAT SHOCK PROTEINS (HSP) WORSEN ALLOGRAFT SURVIVAL AND ENHANCE IFN-γ AND ALLOANTIBODY PRODUCTION. (Abstract #127)**
Milagros Samaniego, Salma Rahimi, Suyi Cao, Lauren Armstrong, Fred Sanfilippo, William Baldwin. Baltimore, MD.

4:20 PM **CD4 FAS-FAS-L CYTOTOXICITY REPRESENTS ONE OF THE EFFECTOR PATHWAYS RESPONSIBLE FOR THE ACUTE REJECTION OF MINOR TRANSPLANTATION ANTIGENS. (Abstract #128)**
Murielle Surquin, Alain Le Moine, Veronique Flamand, Michel Goldman, Daniel Abramowicz. Brussels, Belgium; Brussels, Belgium.

4:30 PM **INFILTRATION OF ALLOGRAFTS BY NATURAL KILLER CELLS PRECEDES T CELLS: CROSS TALK BETWEEN THE INNATE AND ADAPTIVE IMMUNE SYSTEM? (Abstract #129)**
Joyce Popoola, Kathryn J. Wood, Steven H. Sacks, Wilson Wong. London, United Kingdom; Oxford, United Kingdom.

4:40 PM **MECHANISMS OF NITRIC OXIDE MEDIATED CYTOTOXICITY IN CARDIAC ALLOGRAFT REJECTION: PRESERVATION OF ACONITASE, A CRITICAL MITOCHONDRIAL ENZYME, WITH CSA. (Abstract #130)**
Allan M. Roza, Galen M. Pieper, Gail Hilton, Cara L. Olds, Christopher C. Felix, Eugene A. Konorev, Mark B. Adams. Milwaukee, WI; Milwaukee, WI.

4:50 PM **DIVERGING SIGNALING PATHWAYS FOR REGENERATIVE AND PROFIBROTIC EFFECTS OF THROMBIN IN PROXIMAL TUBULAR CELLS (PTEC). (Abstract #131)**
Giuseppe Grandaliano, Paola Pontrelli, Michele Ursi, Loreto Gesualdo, Paolo F. Schena. Bari, Italy.

5:00 PM **LIGATION OF HLA CLASS I INDUCES TYROSINE PHOSPHORYLATION OF $P60^{SRC}$, $P125^{FAK}$ AND PAXILLIN IN HUMAN ENDOTHELIAL CELLS. (Abstract #132)**
Yiping Jin, Elaine F. Reed. Los Angeles, CA.

5:10 PM **INTRAGRAFT GENE EXPRESSION IN ACUTE REJECTION OF KIDNEY ALLOGRAFTS. (Abstract #133)**
Patrick G. Dean, Mark D. Stegall, David I. Schwartz, Anis Khair, Timothy S. Larson, Jorge A. Velosa. Rochester, MN.

5:20 PM **MICROARRAY EXPRESSION ANALYSIS OF ACUTE REJECTION IN HUMAN PANCREAS ALLOGRAFTS. (Abstract #134)**
Lynn M. Jacobson, Roger W. Sands, Jon S. Odorico. Madison, WI.

Concurrent Session 15: Transplantation: Allocation

4:00 PM - 5:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: Richard Knight and Brian Murray

4:00 PM **TRANSPLANTATION WITHOUT A PREOPERATIVE FINAL CROSSMATCH-IT CAN BE DONE IN SELECTED CIRCUMSTANCES. (Abstract #135)**
Arthur J. Matas, Angelika Gruessner, David E.R. Sutherland. Minneapolis, MN.

4:10 PM **THE ADVANTAGES OF SHARING ZERO HLA-MISMATCHED CADAVERIC KIDNEYS. (Abstract #136)**
Mark D. Stegall, Patrick G. Dean, Maureen A. McBride, James J. Wynn.

4:20 PM **ZERO-MISMATCHED KIDNEYS FROM EXPANDED DONORS: IS THE BENEFIT WORTH THE RISK? (Abstract #137)**
Maureen A. McBride, Wida S. Cherikh, James J. Wynn, Kidney and Pancreas Transplantation Committee. Richmond, VA; Augusta, GA.

4:30 PM **WHAT ARE YOUR CHANCES OF GETTING A ZERO-ANTIGEN MISMATCHED KIDNEY? (Abstract #138)**
Harish D. Mahanty, Calvin D. Lou, George J. Chang, John P. Roberts, Lee Ann Baxter-Lowe. San Francisco, CA.

4:40 PM **GRAFT SURVIVAL OF KIDNEYS FROM A /A B DONORS INTO B PATIENTS IS EQUIVALENT TO ABO COMPATIBLE (B AND O → B) TRANSPLANTATION. (Abstract #139)**
Nicolas A. Muruve, Bradley A. Warady, Mark I. Aeder, Daniel Murillo, Paul W. Nelson, Charles F. Shield, III, Christopher F. Bryan. Westwood, KS.

4:50 PM **THE FIRST TWO YEARS OF A REVISED SCHEME FOR ALLOCATING CADAVER KIDNEYS IN THE UK. (Abstract #140)**
R. J. Johnson, S. Armstrong, M. A. Belger, J. D. Briggs, P. J. Morris. Bristol, United Kingdom.

5:00 PM **DONOR/RECIPIENT AGE MATCHING IN RENAL TRANSPLANTATION. (Abstract #141)**
John S. Gill, Dana Miskulin, Brian J.G. Pereira, David N. Landsberg. Boston, MA; Vancouver, BC, Canada.

5:10 PM **KIDNEY ALLOGRAFT SURVIVAL AND HLA CLASS I MATCHING AT THE AMINO ACID TRIPLET LEVEL. (Abstract #142)**
Rene J. Duquesnoy, Steve Takemoto. Pittsburgh, PA; Los Angeles, CA.

5:20 PM **RENAL TRANSPLANTATION FOLLOWING BONE MARROW TRANSPLANTATION: PATIENT OUTCOMES AND NEED FOR IMMUNOSUPPRESSION. (Abstract #143)**
Khaled Hamawi, J. Andrew Bertolatus. Iowa City, IA.

Concurrent Session 16: Sirolimus in Kidney Transplantation

4:00 PM - 5:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Mitchell Henry and Alan Wilkinson

4:00 PM **TWO-YEAR SAFETY AND EFFICACY OF SIROLIMUS IN RENAL TRANSPLANTATION. (Abstract #144)**
Barry D. Kahan, for the Rapamune US and Global Study Groups. Houston, TX.

4:10 PM **OUTCOME OF 300 RENAL TRANSPLANT RECIPIENTS TREATED *DE NOVO* WITH A SIROLIMUS-CYCLOSPORINE REGIMEN AT A SINGLE CENTER. (Abstract #145)**
Barry D. Kahan, Stephen M. Katz, Richard J. Knight. Houston, TX.

4:20 PM **SIROLIMUS AND CARDIOVASCULAR RISK FACTORS IN A CYCLOSPORINE ELIMINATION TRIAL. (Abstract #146)**
Francesco Paola Schena, the Rapamune Renal Function Study Group. Bari, Italy.

4:30 PM **A PROSPECTIVE RANDOMIZED TRIAL OF SIROLIMUS VS. CYCLOSPORINE IN KIDNEY TRANSPLANTATION: IMPACT OF CALCINEURIN INHIBITOR ELIMINATION ON RENAL FUNCTION. (Abstract #147)**
Stuart M. Flechner, David A. Goldfarb, Charles Modlin, Barbara Mastroianni, Kathy Savas, Venkatesh Krishnamurthy, Daniel J. Cook, Andrew C. Novick. Cleveland, OH.

4:40 PM **REMARKABLY LOW RATE OF ACUTE RENAL ALLOGRAFT REJECTION IN AFRICAN AMERICANS RECEIVING SIROLIMUS, LOW-DOSE TACROLIMUS, AND CORTICOSTEROIDS: PRELIMINARY RESULTS OF A PILOT STUDY. (Abstract #148)**
Hany H.S. Anton, Thomas C. Knauss, David Seaman, Christopher Siegel, James A. Schulak, Donald E. Hricik. Cleveland, OH; Cleveland, OH.

4:50 PM **IMPROVED KIDNEY GRAFT FUNCTION IN PATIENTS RECEIVING SIROLIMUS, CYCLOSPORINE AND STEROIDS: THE ROLE OF CYCLOSPORINE BLOOD CONCENTRATION. (Abstract #149)**
Helio Tedesco, Claudia R. Felipe, Paula G. Machado, Riberto Garcia, Marcelo Franco, Jose O. Medina. Sao Paulo, SP, Brazil.

5:00 PM **CONVERSION TO SIROLIMUS: HOW TO DETERMINE THE OPTIMAL WINDOW OF OPPORTUNITY. A SINGLE CENTER EXPERIENCE. (Abstract #150)**
M. F. Egidi, P. A. Cowan, L. W. Gaber, R. J. Stratta, M. H. Shokouh-Amiri, H. P. Grewal, S. H. Vera, M. R. Honaker, A. O. Gaber. Memphis, TN; Memphis, TN.

5:10 PM **SIROLIMUS IS NOT NEPHROTOXIC: EVIDENCE FROM CLINICAL TRIALS. (Abstract #151)**
Tim Mathew, the Sirolimus Clinical Trials Study Groups. Adelaide, Australia.

5:20 PM **SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY PERMITS EARLY CYCLOSPORINE WITHDRAWAL RESULTING IN IMPROVED RENAL FUNCTION: 12-MONTH RESULTS OF THE TRI-CONTINENTAL TRIAL. (Abstract #152)**
Robert W.G. Johnson, Henri Kreis, Rainer Oberbauer, Kerstin Claesson, Josette Eris, Manuel Arias, Leszek Paczek, Alfredo Mota, Francesco Schena, Felix Frey, Ahmed Shoker, Robert Koene, Anders Hartman, the Sirolimus Tri-continental Renal Transplant Study Group. Manchester, United Kingdom.

Concurrent Session 17: Mechanisms of Ischemia/ Reperfusion Injury I

4:00 PM - 5:30 PM

Empire Room, Intercontinental
Chairs: Christopher Lu and Stefan Tullius

4:00 PM **ORGAN-SPECIFIC EFFECTS OF TARGETING NF-κB TO REDUCE ISCHEMIA/REPERFUSION INJURY. (Abstract #153)** *Young Investigator Award*
Liqing Wang, Nida Shemmeri, Kerrie L. Faia, Wei Gao, Vilmos Csizmadia, Wayne W. Hancock. Cambridge, MA.

4:10 PM **LIVER TRANSPLANT PRESERVATION INJURY ACTIVATES THE LPS/TOLL-LIKE RECEPTOR SIGNALING PATHWAY. (Abstract #154)**
George Tsoulfas, Yoshihito Takahashi, Raymond W. Ganster, Gautam Yagnik, Noriko Murase, David A. Geller.

4:20 PM **HEME OXYGENASE-1 GENE TRANSFER INHIBITS INDUCIBLE NITRIC OXIDE SYNTHASE EXPRESSION AND PROTECTS GENETICALLY FAT ZUCKER RAT LIVERS FROM ISCHEMIA/REPERFUSION INJURY. (Abstract #155)**
Ana J. Coito, Xiu-Da Shen, Roland Buelow, Farin Amersi, Carolina Moore, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Fremont, CA.

4:30 PM **CARBON MONOXIDE PROVIDES PROTECTION AGAINST ISCHEMIA/REPERFUSION INJURY IN RAT LIVERS. (Abstract #156)**
Farin F. Amersi, Xiu-Da Shen, Dean Anselmo, Judy Melinek, Ronald W. Busuttil, Roland Buelow, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Fremont, CA.

4:40 PM **HEME OXYGENASE-1 GENE TRANSFER PROTECTS AGAINST ISCHEMIA/REPERFUSION INJURY IN RAT RENAL ISOGRAFT MODEL. (Abstract #157)**
Tom D. Blydt-Hansen, Masamichi Katori, Charles Lassman, Ana J. Coito, Roland Buelow, Robert Ettenger, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Los Angeles, CA; Los Angeles, CA; Freman, CA.

4:50 PM **COLD ISCHEMIA/REPERFUSION (CIR) INJURY IN THE MURINE ORTHOTOPIC LIVER TRANSPLANT (mOLT) MODEL: CRITICAL ROLE OF IL-6 IN RECOVERY. (Abstract #158)**
Xingyi Que, Fotini Debonera, Xavier Aldeguer, Andrew E. Gelman, Gideon A. Zamir, Kim M. Olthoff.

5:00 PM **AN NOVEL INHIBITOR OF RHO-ASSOCIATED PROTEIN KINASE, Y-27632, AMELIORATES HEPATIC ISCHEMIA AND REPERFUSION INJURY. (Abstract #159)**
Keisa Takeda, Maeng Bong Jin, Tsunenori Sakurai, Tsuyoshi Shimamura, Hiroyuki Furukawa, Miri Fujita, Satoru Todo. Sapporo, Hokkaidou, Japan; Sapporo, Hokkaidou, Japan.

5:10 PM **P38 MITOGEN-ACTIVATED PROTEIN KINASE INHIBITOR AS AN ADDITIVE TO UW SOLUTION AMELIORATES REPERFUSION INJURY IN LIVER TRANSPLANTATION. (Abstract #160)**
Daisuke Yoshinari, Izumi Takeyoshi, Mitsunobu Kobayashi, Susumu Ohwada, Yoshihiro Yabata, Koshi Matsumoto, Yasuo Morishita. Maebashi, Gunma, Japan; Kawasaki, Kanagawa, Japan.

5:20 PM **ELEVATED CYCLIC AMP AMELIORATES ISCHEMIA-REPERFUSION INJURY IN RAT CARDIAC ALLOGRAFTS: A NOVEL APPLICATION OF WATER-SOLUBLE FORSKOLIN DERIVATIVE. (Abstract #161)**
Seiichiro Murata, Douglas N. Miniati, Murray Kown, Mark L. Koransky, Robert C. Robbins. Stanford, CA.

Concurrent Session 18: Thoracic Organ Donor Shortage: Better Management / Alternative Strategies

4:00 PM - 5:30 PM

Exchange Room, Intercontinental
Chairs: Robert Love and Robert Robbins

4:00 PM **PRE-OPERATIVE HLA-DR MATCHING AND EXTENDED LUNG PRESERVATION WITH UW SOLUTION COULD SIGNIFICANTLY IMPROVE GRAFT OUTCOME. (Abstract #162)**
Daniel S. Woolley, William J. Burlingham, Lynn D. DeVito-Haynes, Glenn E. Leverson, Wim van der Bij, W. J. de Boer, Bouke G. Hepkema, Keith C. Meyer, Richard D. Cornwell, Robert B. Love. Madison, WI; Groningen, The Netherlands.

4:10 PM **FLOW PRA DETECTED HLA ANTIBODIES CAN IDENTIFY CLINICALLY RELEVANT FLOW CYTOMETRIC POSITIVE CROSSMATCHES AMONG CARDIAC ALLOGRAFT RECIPIENTS. (Abstract #163)**
Piotr Przybylowski, Howard Gebel, Robert Bray, Branislav Radovancevic, O.Howard Frazier, Chris Garcia, Stephanie Rasmussen, Shauna Garner, Barry Kahan, Ronald Kerman. Houston, TX; Shreveport, LA; Atlanta, GA; Houston, TX.

4:20 PM **DONOR CARDIAC TROPONIN T AND PROCALCITONIN ARE INDEPENDENT PREDICTORS OF EARLY GRAFT FAILURE AFTER HEART TRANSPLANTATION. (Abstract #164)**
Evgenij V. Potapov, Frank D. Wagner, Matthias Loebe, Britta Jonitz, Ekaterina A. Ivanitskaia, Julia Stein, Ralf Sodian, Martin Moeckel, Christian Mueller, Roland Hetzer. Berlin, Germany; Berlin, Germany; Berlin, Germany.

4:30 PM **RETROSPECTIVE ASSESSMENT OF CARDIAC CATHETERIZATION AND ANGIOGRAPHY AS A SCREENING TOOL IN CADAVERIC HEART DONORS. (Abstract #165)**
Robert S. D. Higgins, Leah E. Bennett, Abelardo De Anda. Richmond, VA; Richmond, VA.

4:40 PM **THE EFFECTS OF TEMPERATURE ON EARLY CARDIAC GRAFT FUNCTION. (Abstract #166)**
John W. Fehrenbacher, Carlos A. Labarrere, Nicholas L. Finley, Daniel J. Beckman, David A. Hormuth.

4:50 PM **FIRST CLINICAL USE OF THE JARVIK 2000 LEFT VENTRICULAR ASSIST SYSTEM AS A BRIDGE TO TRANSPLANTATION: HEMODYNAMIC AND ECHOCARDIOGRAPHIC ASSESSMENT. (Abstract #167)**
Reynolds M. Delgado, Timothy J. Myers, Branislav Radovancevic, Igor D. Gregoric, Kathy Miller, Tehreen Khan, Romeo Majano, Biswajit Kar, Robert K. Jarvik, O. H. Frazier. Houston, TX; New York, NY.

5:00 PM **HLA ALLOSENSITIZATION DURING VENTRICULAR ASSIST DEVICE SUPPORT CAN BE PREDICTED AND IS RELATED TO DECREASED TRANSPLANT SURVIVAL. (Abstract #168)**
Ganesh S. Kumpati, Daniel J. Cook, Eugene H. Blackstone, Jennifer White, Ashraf S. Abdo, James B. Young, Randall C. Starling, Nicholas G. Smedira, Patrick M. McCarthy. Cleveland, OH; Cleveland, OH; Cleveland, OH; Cleveland, OH.

5:10 PM **NOSOCOMIAL BLOODSTREAM INFECTIONS IN PATIENTS WITH IMPLANTABLE LEFT VENTRICULAR ASSIST DEVICES [LVAD]. (Abstract #169)**
Steven M. Gordon, Steven K. Schmitt, Micah Jacobs, Goormastic Marlene, Nicolas Smedira, Mike Yeager, Janet M. Serkey, Katherine Hoercher, Patrick M. McCarthy.

5:20 PM **SUCCESSFUL TRANSITION OF LVAD PATIENTS FROM HOSPITAL TO HOME: ARE WE APPROACHING THE GOAL OF CHRONIC SUPPORT? (Abstract #170)**
Ashley Sims, Tiffany Buda, Katherine Hoercher, Phyllis Colosimo, Nicholas Smedira, Michael Banbury, Patrick McCarthy.

Concurrent Session 19: Liver Transplantation: Hepatitis C II

4:00 PM - 5:30 PM

Grand Ballroom, Intercontinental
Chairs: Jeffrey Crippin and Richard Rorher

4:00 PM **CHANGES IN HEPATITIS C VIRUS (HCV) POPULATION IN SERUM AND PERIPHERAL BLOOD MONONUCLEAR CELLS IN CHRONICALLY HCV-INFECTED PATIENTS RECEIVING LIVER GRAFT FROM HCV-INFECTED DONORS. (Abstract #171)**
H. Vargas, L.F. Wang, J. Wilkinson, M. Radkowski, T. Laskus, J. Rakela. Scottsdale, AZ; Pittsburgh, PA.

4:10 PM **DISTINCTION OF ACUTE CELLULAR REJECTION FROM RECURRENCE OF HCV THROUGH INTRAGRAFT GENE EXPRESSION PATTERNS. (Abstract #172)** *Young Investigator Award*
R. Sreekumar, R. Wiesner, C. Rosen, M. Charlton. Rochester, MN.

4:20 PM **STEROID-FREE INDUCTION WITH TACROLIMUDS AND DACLIZUMAB IN LIVER TRANSPLANT RECIPIENTS WITH HEPATITIS C – A PRELIMINARY REPORT OF A PROSPECTIVE RANDOMIZED TRIAL. (Abstract #173)**
Tomoaki Kato, Guy Neff, Marzia Montalbano, Olivia M. Hung, Reynel Lavandera, Deborah Weppler, David M. Levi, Jose R. Nery, Seigo Nishida, Antonio D. Pinna, Rajender Reddy, Christopher B. O'Brien, Philip Ruiz, Andreas G. Tzakis. Miami, FL; Miami, FL; Miami, FL.

4:30 PM **INTRAHEPATIC CYTOKINE EXPRESSION ASSOCIATED WITH HEPATITIS C VIRUS (HCV) RECURRENCE POST LIVER TRANSPLANTATION FOR HCV INFECTION. (Abstract #174)**
Amany Zekry, Alex Bishop, Geoffrey W. McCaughan. Sydney, NSW, Australia.

4:40 PM **TRANSMISSION AND EVOLUTION OF VIRAL QUASISPECIES IN HEPATITIS C VIRUS POSITIVE LIVER TRANSPLANT RECEIPIENTS. (Abstract #175)**
J. Rakela, T. Laskus, J. K. Wilkinson, M. Radkowski, H. Vargas, V. Balan, D. D. Douglas, M. E. Harrison, A. Moss, D. C. Mulligan. Scottsdale, AZ; Pittsburgh, PA.

4:50 PM **IS MYCOPHENOLATE BENEFICIAL IN THE TREATMENT OF RECURRENT HEPATITIS C FOLLOWING LIVER TRASNPLANTATION? (Abstract #176)**
Gregory A. Smallwood, Michael E. de Vera, Laurel Davis, Enrique Martinez, Andrei C. Steiber, Thomas G. Heffron. Atlanta, GA; Atlanta, GA; Atlanta, GA.

5:00 PM **ROLE OF MYCOPHENOLATE MOFETIL (MMF) IN LIVER TRANSPLANTATION (LTX) FOR HCV RELATED END STAGE LIVER FAILURE (A PROSPECTIVE RANDOMISED TRIAL). (Abstract #177)**
Ashok B. Jain, Randeep S. Kashyap, John J. Fung. Pittsburgh, PA.

5:10 PM **COMBINATION OF INTERFERON-ALPHA AND RIBAVIRIN FOR RECURRENT HEPATITIS C AFTER ORTHOTOPIC LIVER TRANSPLANTATION—IMPACT ON HISTOPATHOLOGY, VIREMIA AND GRAFT FUNCTION. (Abstract #178)**
Arno Kornberg, Merten Homman, Andrea Tannapfel, Rigo Voigt, Thomas Grube, Johannes Scheele.

5:20 PM **EFFECT OF INTERFERON-α AND RIBAVIRIN ON LIVER HISTOLOGY IN LIVER TRANSPLANT PATIENTS WITH RECURRENT HEPATITIS C. (Abstract #179)**
Aejaz Nasir, Michael E. de Vera, Gregory A. Smallwood, Andrei C. Stieber, Thomas G. Heffron. Atlanta, GA; Atlanta, GA.

Concurrent Session 20: Islet Transplantation and Long-Term Results of Pancreas Transplantation

4:00 PM - 5:30 PM

Renaissance Ballroom, Intercontinental
Chairs: Cristiana Rastellini and Camillo Ricordi

4:00 PM **INSULIN INDEPENDENCE AFTER SINGLE-DONOR ISLET TRANSPLANTATION IN TYPE I DIABETES WITH hOKT3γ-1 (ala-ala), SIROLIMUS, AND TACROLIMUS THERAPY. (Abstract #180)**
Bernhard J. Hering, Raja Kandaswamy, James V. Harmon, Jeffrey Ansite, Sue Clemmings, Tetsuya Sakai, Julianne Zabloski, Kathy Duderstadt, Kathy Jacobsen, Carrie Gibson, Steve Paraskevas, Peter Eckman, Masahiko Nakano, Toshiya Sawada, Hui J. Zhang, David E.R. Sutherland, Jeffrey A. Bluestone. Minneapolis, MN; San Francisco, CA.

4:10 PM **SOLITARY ISLET CELL TRANSPLANTATION FROM A SINGLE DONOR FOR PATIENTS WITH TYPE 1 DIABETES AND HYPOGLYCEMIA UNAWARENESS. (Abstract #181)**
Rodolfo Alejandro, Aileen M. Caulfield, Tatiana Froud, Jacqueline V. Ferreira, Lisa C. Rothenberg, Ismail H. Al-Abdullah, Andres Boker, David A. Baidal, Topaz J. Kirlew, Norma S. Kenyon, Camillo Ricordi.

4:20 PM **EVALUATION OF THE LIVER PRE AND POST PERCUTANEOUS TRANSHEPATIC INTRAPORTAL ISLET CELL TRANSPLANTATION. (Abstract #182)**
Tatiana Froud, Aileen M. Caulfield, Jacqueline V. Ferreira, Andres Boker, David A. Baidal, Lisa C. Rothenberg, Robin Harbach, Yrizarry Jose, Camillo Ricordi, Rodolfo Alejandro. Miami, FL.

4:30 PM **TWO-LAYER (UNIVERSITY OF WISCONSIN SOLUTION/ PERFLUOROCARBON) METHOD OF HUMAN PANCREAS PRESERVATION PRIOR TO ISLET ISOLATION IMPROVED THE YIELD AND VIABILITY. (Abstract #183)**
Shinichi Matsumoto, Theodor H. Rigery, Sabrina A. Qually, Ian Sweet, Yoshikazu Kuroda, R. Brian Stevens. Seattle, WA; Seattle, WA; Seattle, WA; Kobe, Hyogo, Japan.

4:40 PM **PRE AND PERI-OPERATIVE MANAGEMENT INFLUENCES THE CLINICAL OUTCOME OF ISLET TRANSPLANTATION. (Abstract #184)**
Paola Maffi, Federico Bertuzzi, Daniela Guiducci, Carlo Socci, Luca Aldrighetti, Rita Nano, Marco Salvioni, Antonio Secchi, Valerio Di Carlo. Milan, Italy.

4:50 PM **SUSTAINED IMPROVEMENT IN CARDIOMYOPATHY FOLLOWING PANCREAS-KIDNEY TRANSPLANATION: A FOUR YEAR FOLLOW-UP. (Abstract #185)**
Patricia A. Cowan, Donna K. Hathaway, Mona N. Wicks, Robert J. Stratta, M. H. Shokouh-Amiri, A. Osama Gaber. Memphis, TN; Memphis, TN.

5:00 PM **IMPROVEMENT IN HYPERTENSION AFTER SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. (Abstract #186)**
Micheal D. Elliott, Mihai Gheorghiade, Michele A. Parker, Lorenzo G. Gallon, Dixon B. Kaufman.

5:10 PM **HUMAN ISLET TRANSPLANTATION NETWORK FOR THE TREATMENT OF TYPE I DIABETES: FIRST (1999-2000) DATA FROM THE SWISS-FRENCH GRAGIL CONSORTIUM. (Abstract #187)**
José Oberholzer, Pierre-Yves Benhamou, Christian Toso, Laurance Kessler, Alfred Penformis, François Bayle, Charles Thivolet, Xavier Martin, Lionel Badet, Cyrille Colin, Philippe Morel. Geneva, Switzerland; Grenoble, France; Strasbourg, France; Besançon, France; Lyon, France.

Poster Session I

8:00 AM - 7:30 PM
Presenters in Attendance: 5:30 PM - 7:30 PM
Exhibits Open
Wine and Cheese Reception
River Exhibition Hall

♦*Also presented in Selected Poster Session*

Kidney - Acute/Chronic Rejection I

P1 **OUTCOME AFTER TREATMENT OF BIOPSY PROVEN VS. PRESUMPTIVE ACUTE REJECTION (AR) IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #188)**
Abhi Humar, Raja Kandaswamy, Roger Denis, Steven Paraskevas, Kristen Gillingham, Arthur Matas. Minneapolis, MN.

P2 **POOR RENAL ALLOGRAFT FUNCTION AFTER TRANSPLANT (TX) PREDICTS POOR OUTCOME AT 1 YEAR. (Abstract #189)**
Arthur J. Matas, William D. Irish, the DGF Study Group. Minneapolis, MN; Fremont, CA.

P3 **AN ANALYSIS OF TISSUE REPAIR GENES IN NEPHROTOXICITY AND ACUTE REJECTION IN RENAL TRANSPLANT PATIENTS. (Abstract #190)**
Ashwani K. Khanna, Matthew S. Plummer, Barbara Bresnahan, Sundaram Hariharan. Milwaukee, WI; Milwaukee, WI; Milwaukee, WI; Milwaukee, WI.

P4 **47 CONSECUTIVE KIDNEY TRANSPLANTS WITHOUT REJECTION-THE IMPORTANCE OF DIURNAL CSA DOSING. (Abstract #191)♦**
Barry J. Browne, Cynthia Op't Holt, Osemwegie E. Emovon. Mobile, AL.

P5 **SIROLIMUS THERAPY FOR HIGH-RISK RENAL TRANSPLANT PATIENTS. (Abstract #192)**
Barry D. Kahan, the Rapamune Renal Function Study Group. Huston, TX.

P6 **SMADS DEFINE A CHRONIC ALLOGRAFT NEPHROPATHY (CAN) GENOTYPE AND PHENOTYPE: IMPLICATIONS FOR INTRAGRAFT MONITORING IN CAN. (Abstract #193)**
Bryan N. Becker, Lynn M. Jacobson, Gretchen J. Malin, Jacquelyn K. Aschenbrenner, Yolanda T. Becker, Debra A. Hullett. Madison, WI; Madison, WI.

P7 **A MULTICENTRE RANDOMISED CONTROLLED STUDY INVESTIGATING THE EFFECT OF MYCOPHENOLATE MOFETIL (MMF) ON RENAL FUNCTION AFTER CYCLOSPORINE A (CsA) WITHDRAWAL IN RENAL TRANSPLANT PATIENTS WITH A "CREEPING CREATININE": AN INTERIM ANALYSIS OF AN ONGOING STUDY. (Abstract #194)**
Christopher R.K. Dudley, the Mycophenolate Mofetil Creeping Creatinine Study Group. Bristol, United Kingdom.

P8 **DURATION OF DECLARED BRAIN DEATH IN CADAVERIC DONORS. (Abstract #195)**
Darin J. Treleaven, Jacqueline Gough, Christian G. Rabbat, Scott Brimble, David Ludwin, Dianne Arlen, David Russell. Hamilton, ON, Canada.

P9 **A RANDOMIZED AND PROSPECTIVE STUDY COMPARING TREATMENT WITH HIGH-DOSE INTRAVENOUS IMMUNOGLOBULIN WITH MONOCLONAL ANTIBODIES FOR RESCUE OF KIDNEY GRAFTS WITH STEROID-RESISTANT REJECTION. (Abstract #196)**
Domingo H. Casadei, Maria Del Carmen Rial, Gerhard Opelz, Julio C. Goldberg, Jorge A. Argento, Gabriela F. Greco, Olga E. Guardia, Emilio Haas, Eduardo Raimondi. Buenos Aires, Argentina; Heidelberg, Germany; Buenos Aires, Argentina.

P10 **RECIPIENTS OF HIGH-RISK CADAVERIC DONORS THAT SUFFER DELAYED GRAFT FUNCTION (DGF) HAVE WORSE GRAFT SURVIVAL (GS), IMPAIRED RENAL FUNCTION AND MORE ACUTE REJECTION (AR). (Abstract #197)**
Gabriel M. Danovitch, William D. Irish, The DGF Study Group. Los Angeles, CA; Fremont, CA.

P11 **INTERACTION BETWEEN ACUTE REJECTION AND RECIPIENT AGE ON LONG TERM RENAL ALLOGRAFT SURVIVAL. (Abstract #198)♦**
Herwig-Ulf Meier-Kriesche, Otto Leiti, Gary S. Friedman, Bruce Kaplan. Ann Arbor, MI; Livingston, NJ.

Kidney - GVH, Complications, Infections I

P12 **UROLOGIC CANCERS IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #199)**
Christopher Gran, John Hulbert, Ken Roberts, Sid Jain, Arthur Matas, Abhi Humar. Minneapolis, MN; Minneapolis, MN.

P13 **RELATIONSHIPS BETWEEN CYTOMEGALOVIRUS SHEDDING AND EBV VIRAL LOAD AT THE EARLY PHASE OF RENAL TRANSPLANTATION. (Abstract #200)**
Claire Presne, Andre S. Pruna, Philippe Bidet, Veronique Auguste, Gilles Duverlie, Hakim Mazouz, Pierre F. Westeel, Jacques Petit, Albert Fournier, Antoine Garbarg-Chenon. Amiens, France; Paris, France.

P14 **CYCLOSPORINE A USE IS NOT INDEPENDENTLY ASSOCIATED WITH INCREASED PLASMA TOTAL HOMOCYSTEINE LEVELS IN AUSTRIAN OR UNITED STATES CHRONIC RENAL TRANSPLANT RECIPIENTS. (Abstract #201)**
Andrew G. Bostom, Gere Sunder-Plassmann, Manuela Fodinger, Reginald Y. Gohh, Jacob Selhub. Pawtucket, RI; Vienna, Austria; Providence, RI; Boston, MA.

P15 **A COMPARATIVE STUDY OF PROPHYLACTIC ORAL GANCYCLOVIR AND VALACYCLOVIR IN HIGH-RISK KIDNEY TRANSPLANT RECIPIENTS. (Abstract #202)**
Angelito Yango, Abdulrahman Zanabli, Paul Morrisey, Abinash Roy, Reginald Gohh. Providence.

P16 **THE EFFECT OF MYCOPHENOLATE MOFETIL ON HEPATITIS B VIRAL LOAD IN STABLE RENAL TRANSPLANT RECIPIENTS WITH CHRONIC HEPATITIS B. (Abstract #203)♦**
Bart D. Maes, Jos F. van Pelt, Peeters Jacques, Nevens Frederik, Evenepoel Pieter, Kuypers Dirk, Messiaen Thierry, Fevery Johan, Vanrenterghem F. Yves. Leuven, Belgium; Leuven, Belgium.

P17 **INCIDENCE OF POSTTRANSPLANT GLYCEMIC DISORDERS IN RENAL TRANSPLANT RECIPIENTS TREATED WITH LOW AND STANDARD DOSE TACROLIMUS. (Abstract #204)**
Bart D. Maes, Dirk Kuypers, Pieter Evenepoel, Thierry Messiaen, Chantal Mathieu, Willy Coosemans, Jacques Pirenne, Yves Vanrenterghem. Leuven, Belgium; Leuven, Belgium; Leuven, Belgium.

P18 **ERYTHROPOIETIN THERAPY IMPROVES POST-TRANSPLANT ANEMIA AND CHRONIC RENAL TRANSPLANT DYSFUNCTION. (Abstract #205)**
Bryan N. Becker, Yolanda T. Becker, Glen E. Leverson, Dennis M. Heisey. Madison, WI, United States; Madison, WI.

P19 **SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY: AN OVERVIEW OF THE LIPID PROFILE AND CARDIOVASCULAR RISK FACTORS. (Abstract #206)**
Christophe Legendre, Neville Jamieson, José Colon, João Pena, Javier Martinez, Heide Sperschneider, Alfredo Mota, the Sirolimus Tri-continental Renal Transplant Study Group. Paris, France.

P20 **SOLE RELIANCE ON RANDOM / CLINIC BP VALUES MIS-CLASSIFIES HYPERTENSION IN 20% OF RENAL TRANSPLANT RECIPIENTS (RTx) - AN AMBULATORY BP MONITORING (ABPM) ANALYSIS. (Abstract #207)**
S. Jayawardene, R. S. Bakri, H. O'Sullivan, D. J.A. Goldsmith. London, United Kingdom.

P21 **MARKERS OF ACUTE INFLAMMATION ARE RAISED IN RENAL TRANSPLANTATION (RTx) PATIENTS, ESPECIALLY IN THE PRESENCE OF CARDIOVASCULAR DISEASE (CVD). (Abstract #208)**
Rashed S. Bakri, Ben Afzali, Peter J. Lumb, George Chik, Anthony S. Wierzbicki, Martin Crook, David Goldsmith. London, United Kingdom; London, United Kingdom.

P22 **PLASMA TOTAL SIALIC ACID (SA) LEVELS ARE RAISED IN RENAL TRANSPLANT RECIPIENTS (RTx) AND CORRELATE WITH THE PRESENCE OF CARDIOVASCULAR DISEASE (CVD). (Abstract #209)**
Rashed S. Bakri, David J.A. Goldsmith, Ben Afzali, Peter J. Lumb, George Chik, Anthony S. Wierzbicki, Martin Crook. London, United Kingdom; London, United Kingdom.

P23 **WHICH IS THE BETTER METHOD FOR CONTROL OF POST-RENAL TRANSPLANTATION (RTx) HYPERPARATHYROIDISM (HPT) - TOTAL OR SUBTOTAL PARATHYROIDECTOMY (PTX)? (Abstract #210)**
Satishkumar A. Jayawardene, William J. Owen, David J.A. Goldsmith. London, United Kingdom; London, United Kingdom.

P24 **DELAYED FUNCTION OF RENAL ALLOGRAFTS: INCIDENCE, OUTCOME AND RISK FACTORS. (Abstract #211)**
Graeme R. Russ. Adelaide, SA, Australia.

Kidney - Immunosuppression A I

P25 **HUMAN GENE EXPRESSION BY ALLOACTIVATED LYMPHOCYTES (MLR) IN SEVERE COMBINED IMMUNE DEFICIENT MICE (SCID). (Abstract #212) ♦**
Ahmed S. Shoker, Zhao-Rong Lun, Rezvan Choudry.

P26 **EFFECT OF CONCOMITANT TACROLIMUS AND RAPAMYCIN THERAPY ON IMMUNE FUNCTION IN RENAL TRANSPLANT RECIPIENTS. (Abstract #213)**
Ashwani K. Khanna, Matthew S. Plummer, Cathy M. Bromberek, Sundaram Hariharan. Milwaukee, WI; Milwaukee, WI; Milwaukee, WI; Milwaukee, WI.

P27 **USE OF MICROEMULSION FORMULATIONS OF CYCLOSPORINE IS ASSOCIATED WITH AN INCREASED INCIDENCE OF HISTOLOGIC EVIDENCE OF CHRONIC CYCLOSPORINE TOXICITY. (Abstract #214)**
Bernard Benedetto, Robert Madden, Alexander Kurbanov, Michael Germain, George Lipkowitz. Springfield, MA; Springfield, MA.

P28 **TACROLIMUS PHARMACOKINETICS AFTER KIDNEY TRANSPLANTATION. (Abstract #215)**
Felix Braun, Beatrice Peters, Ekkehard Schütz, Thomas Lorf, Ruben Canelo, Michael Oellerich, Burckhardt Ringe. Göttingen, Germany; Göttingen, Germany.

P29 **LATE CORTICOSTEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS ON TRIPLE IMMUNOSUPPRESSIVE THERAPY. RANDOMISED CONTROLLED TRIAL. (Abstract #216)**
Christopher K.T. Farmer, Ian C. Abbs, Rachel M. Hilton, Geoff Koffman, Jane Watkins, Steven H. Sacks. London, United Kingdom.

P30 **WHAT IS THE VALUE OF SHORT SYNACTHEN TESTS IN PREDICTING EASE OF STEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS? RANDOMISED CONTROLLED TRIAL. (Abstract #217)**
Christopher K.T. Farmer, Ian C. Abbs, Rachel M. Hilton, Geoff Koffman, Jane Watkins, Steven H. Sacks. London, United Kingdom.

P31 **SUSTAINED LOW REJECTION RATES WITH TACROLIMUS THERAPY: TWO YEAR FOLLOW-UP OF A LARGE, MULTICENTRE EUROPEAN TRIAL IN RENAL TRANSPLANTATION. (Abstract #218)**
Domingo Del Castillo Caba, the Spanish-Italian Tacrolimus Renal Transplantation Study Group. Cordoba, Spain.

P32 **KINETICS OF EARLY PERIPHERAL BLOOD LYMPHOCYTE RECONSTITUTION FOLLOWING INDUCTION TREATMENT WITH ANTITHYMOCYTE GLOBULIN (THYMOGLOBULIN®) IN KIDNEY TRANSPLANTATION. (Abstract #219)**
E. Renoult, M. Ladrière, M. C. Béné, M. N. Kolopp-Sarda, G. Faure, M. Kessler. Nancy, France.

P33 **USE OF C-2 MONITORING TO OPTIMIZE CYCLOSPORINE MICROEMULSION DOSING IN DE NOVO RENAL TRANSPLANT RECIPIENTS: A SAFETY ANALYSIS. (Abstract #220)**
Edward Cole, Aziz Walele, Daniel Cattran, Stanley Fenton, Catherine O'Grady, Carl Cardella. Toronto, ON, Canada.

P34 **A RANDOMIZED, OPEN-LABEL, PROSPECTIVE STUDY COMPARING TWO DIFFERENT CYCLOSPORIN A AREAS UNDER THE TIME-CONCENTRATION CURVE FOR THE PREVENTION OF REJECTION IN RENAL TRANSPLANTATION. (Abstract #221)**
Zita M.L. Britto, Cristiane F. Alves, Flavio J. Paula, Luis S. Azevedo, Maria Cristina R. Castro, Francine C. Lemos, Eduardo Mazzucchi, Pedro R. Chocair, Joao A. Fonseca, William C. Nahas, Luis E. Ianhez, Elias David Neto. Sao Paulo, Sao Paulo, Brazil.

P35 **SIROLIMUS (SIR) PLUS CYCLOSPORINE (CSA) TO WITHDRAW STEROID (SW) IN LONG TERM KIDNEY TRANSPLANT RECIPIENTS (KTX). (Abstract #222)**
Franco Citterio, Vittorio Alfieri, Paolo Altieri, Pasquale Berloco, Marco Castagneto, Francesco Marchini, Gavina Murgia, Luca Poli, Paolo Rigotti, Giuseppe Segoloni. Rome, Italy; Torino, Italy; Cagliari, Italy; Rome, Italy; Padova, Italy.

Kidney - Immunosuppression B I

P36 **PRELIMINARY RESULTS OF THE USE OF HUMANIZED ANTI-CD154 IN HUMAN RENAL ALLOTRANSPLANTATION. (Abstract #223) ♦**
Allan D. Kirk, Stuart J. Knechtle, Hans W. Sollinger, Flavio G. Vincenti, Scott Stecher, Kari Nadeau. Bethesda, MD; Madison, WI; San Francisco, CA; Cambridge, MA.

P37 **LONG-TERM CYCLOSPORINE IMMUNOSUPPRESSION: IS THERE NEPHROTOXICITY IN KIDNEY TRANSPLANT RECIPIENTS? (Abstract #224)**
Steven Paraskevas, Roger Denny, Thiagarajan Ramcharan, Kristen Gillingham, Raja Kandaswamy, Abhinav Humar, Arthur Matas. Minneapolis, MN.

P38 **CORRELATION BETWEEN CYCLOSPORINE (CsA) PHARMACOKINETIC PARAMETERS AND ACUTE HISTOLOGICAL FINDINGS IN SURVEILLANCE BIOPSIES. (Abstract #225)**
Azemi A. Barama, James Gough, Rachel McKenna, Mauricio Monroy, Serdar Yilmaz, Gerard Murphy, Farshad Sepandj, Halgrimur Benediktsson.

P39 **LOW INCIDENCE OF ADVERSE EVENTS IN A PILOT STUDY IN KIDNEY TRANSPLANTS USING A STEROID FREE, CALCINEURIN SPARING, MYCOPHENOLATE & SIROLIMUS BASED REGIMEN. (Abstract #226)**
C. Marsh, R. Wilburn, L. Wrenshall, R. Stevens, C. Davis. Seattle, WA; Seattle, WA; Seattle, WA.

P40 A PROSPECTIVE MULTICENTER STUDY DEMONSTRATES SAFETY AND EFFICACY OF PERIPHERAL VEIN ADMINISTRATION OF THYMOGLOBULIN FOR INDUCTION IMMUNOSUPPRESSION. (Abstract #227)
Robert Steiner, Douglas Norman, David Cohen. San Diego, CA; OR.

P41 LONG TERM BENEFITS AND SIDE EFFECTS OF CICLOSPORIN (CYA) TO MYCOFENOLATE MOFETIL (MMF) CONVERSION IN RENAL TRANSPLANT PATIENTS. (Abstract #228)
H. François, M. Ammor, R. Djeffal, V. Paradis, F. Kriaa, A. Durrbach, B. Charpentier. Le Kremlin Bicetre, France.

P42 CYCLOSPORIN-A BLOOD CONCENTRATION AT 2 HOURS IS THE BEST PARAMETER TO CALCULATE AREA UNDER THE TIME-CONCENTRATION CURVE (0 TO 4 HOURS). (Abstract #229)
Elias David-Neto, Zita M.L. Britto, Cristiane F. Alves, Francine C. Lemos, William C. Nahas, Luis E. Ianhez. Sao Paulo, Sao Paulo, Brazil.

P43 EFFECT OF LIVING RELATED DONOR BONE MARROW INFUSION ON CHIMERISM IN KIDNEY TRANSPLANT PATIENTS. (Abstract #230)
Gaetano Ciancio, Joshua Miller, Rolando Garcia-Morales, George W. Burke, Camillo Ricordi, Andreas Tzakis, Violet Esquenazi. Miami, FL.

P44 EFFICACY AND SAFETY OF DACLIZUMAB INDUCTION FOR PRIMARY KIDNEY TRANSPLANT RECIPIENTS IN COMBINATION WITH TACROLIMUS, MYCOPHENOLATE MOFETIL AND STEROIDS AS MAINTENANCE IMMUNOSUPPRESSION. (Abstract #231)
Gaetano Ciancio, George W. Burke, Audrey Miller, Kiliana Suzart, Jose Figueiro, Anne Rosen, David Roth, Warren Kupin, Joshua Miller. Miami, FL.

P45 A COMPARISON OF FIBROGENIC GENE mRNA LEVELS IN RENAL TRANSPLANT BIOPSIES TAKEN FROM PATIENTS ON A RANDOMISED TRIAL OF AZATHIOPRIN VERSUS MYCOPHENOLATE MOFETIL. (Abstract #232)
Gareth R. Bicknell, Sunjay Jain, Michael L. Nicholson.

P46 DACLIZUMAB AND MYCOPHENOLATE MOFETIL REDUCE THE NEED FOR CYCLOSPORINE WITHOUT INCREASING RISK FOR ACUTE REJECTION IN RENAL TRANSPLANTATION. (Abstract #233)
Gordon R. Ingle, Asha Moudgil, Ashley Vo, Stanley C. Jordan. Los Angeles, CA.

P47 WHICH PATIENTS BENEFIT FROM CYCLOSPORINE WITHDRAWAL FOLLOWED BY SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY? (Abstract #234)
Henri Kreis, José M. Morales, Peter Morris, Antonio Henriques, Pierre Daloze, Giuseppe Segolini, Uwe Heemann, Eric Nègre, the Sirolimus Tri-continental Renal Transplant Study Group. Paris, France.

Kidney - Pediatrics, Recurrent Disease I

P48 COMPARISON OF POST TRANSPLANT LYMPHO PROLIFERATIVE DISORDERS (PTLD) IN CHILDREN UNDER CYCLOSPORINE AND TACROLIMUS, 766 CONSECUTIVE RECEIPIENTS: 15 YEARS EXPERIENCE. (Abstract #235)
Ashok B. Jain, George Mazariegos, Randeep S. Kashyap, Cataldo Doria, Mike Nalesnik, Jorge Reyes.

P49 ANALYSIS OF HYPERLIPIDEMIA IN CHILDREN WITH KIDNEY TRANSPLANTS. (Abstract #236)
Maria Hardstedt, Kristen Gillingham, Blanche M. Chavers.

P50 SUPERIOR DEATH-CENSORED RENAL ALLOGRAFT SURVIVAL IN OXALOSIS PATIENTS WITH A LIVER TRANSPLANT. (Abstract #237)
Diane M. Cibrik, Bruce Kaplan, Julie A. Arndorfer, Akinlolu Ojo, Alan B. Leichtman, Herwig-Ulf Meier-Kriesche. Ann Arbor, MI.

P51 EFFECTIVENESS OF TACROLIMUS IN PREVENTING THE RECURRENCE OF IgA NEPHROPATHY AFTER RENAL TRANSPLANTATION. (Abstract #238)
Yoshihiko Watanabe, Kazunari Tanabe, Tadahiko Tokumoto, Hiroaki Shimmura, Hiroshi Nihei, Hiroshi Toma. Tokyo; Tokyo, Japan.

P52 CYCLOSPORINE PHARMACOKINETICS UNALTERED BY BASILIXIMAB IN PEDIATRIC RENAL TRANSPLANT RECIPIENTS. (Abstract #239)
J. M. Kovarik, L. Chodoff, A. Korn. East Hanover, NJ.

Kidney - Preservation, Donation/Allocation, Economics/ Public Policy, Surgical Techniques, and Other I

P53 HAND-ASSISTED LAPAROSCOPIC LIVE DONOR NEPHRECTOMY: THE OHIO STATE UNIVERSITY EXPERIENCE. (Abstract #240)
Aamer Ar'Rajab, Ronald P. Pelletier, Mitchell L. Henry, Elmahdi A. Elkhammas, Ginny L. Bumgardner, Elizabeth A. Davies, Ronald M. Ferguson.

P54 WHO BECOMES A NON-DIRECTED KIDNEY DONOR? (Abstract #241)
Cheryl Jacobs, Deborah Roman, Catherine Garvey, Abhi Humar, Arthur Matas. Minneapolis, MN.

P55 KIDNEY CANCERS IN RENAL TRANSPLANT RECIPIENTS. (Abstract #242)
Christopher Gran, John Hulbert, Ken Roberts, Sid Jain, Arthur Matas, Abhi Humar. Minneapolis, MN; Minneapolis, MN.

P56 CV EVENTS AND DEATH ON PROVINCIAL RENAL TRANSPLANT WAITING LIST (RTXWL): THE BC EXPERIENCE. (Abstract #243)
A. Levin, D. Landsberg, L. Siosan, L. Venables, L. Liu, J. Gill, W. Gourlay. Vancouver, BC, Canada; Vancouver, BC, Canada; Vancouver, BC, Canada.

P57 LONG-TERM OUTCOME OF RENAL TRANSPLANTATION IN RECIPIENTS OLDER THAN 65 YEARS. (Abstract #244)
Amado Andres, Juan C. Herrero, Jose M. Morales, Teresa Ortuño, Beatriz Domíguez, Eduardo Hernandez, Manuel Praga. Madrid, Spain.

P58 PROSPECTIVE INCEPTION COHORT ANALYSIS OF OPEN DONOR NEPHRECTOMY WITH HANDOSCOPIC AND LAPAROSCOPIC DONOR NEPHRECTOMY. (Abstract #245)
Amy D. Lu, Lynt B. Johnson, Jeff S. Plotkin, Joseph Buell, James F. Whiting, William H. Marks, Phil Chapman, Kenneth A. Newell, Paul C. Kuo. Washington, DC; Cincinnati, OH; Portland, ME; Seattle, WA; Chicago, IL.

P59 PERCENT IMPROVEMENT IN HEART RATE VARIABILITY IN DIABETIC AND NONDIABETIC KIDNEY AND KIDNEY PANCREAS RECIPIENTS. (Abstract #246)
Ann K. Cashion, Rebecca P. Winsett, Patricia F. Joplin, Robert J. Stratta, Osama Gaber, Donna K. Hathaway. Memphis, TN; Memphis, TN.

P60 ENHANCED CHARACTERIZATION OF PRE-SENSITIZATION STATUS IN KIDNEY TRANSPLANT CANDIDATES USING SENSITIVE TECHNIQUES. (Abstract #247)
Antonina Piazza, Elvira Poggi, Giuseppina Ozzella, Palmina I. Monaco, Simona Servetti, Carlo U. Casciani, Domenico Adorno. Rome, Italy.

P61 QUALITY OF LIFE IN RENAL TRANSPLANT PATIENTS WITH FUNCTIONING GRAFTS; THE STORY UNDERNEATH. (Abstract #248)
Argiris Asderakis, Christopher Brown, Phil Dyer, Robert W.G. Johnson. Cardiff, United Kingdom; Manchester, United Kingdom.

P62 **LAPAROSCOPIC DONOR NEPHRECTOMY: A POSITIVE COMPARISON WITH OPEN PROCEDURES. (Abstract #249)**
Brian M. Gogel, L. Michael Goldstein, Robert C. Schoenvogel, Howard C. Derrick, III, Matthew V. Westmoreland, Michael Seiba, Edmund Q. Sanchez, Shigeru Marubashi, Robert M. Goldstein, Marlon F. Levy, Ernesto P. Molmenti, Carlos G. Fasola, Thomas A. Gonwa, Laura L. Christensen, Goran B. Klintmalm. Dallas, TX.

P63 **FUNCTIONAL PERFORMANCE AND HEALTH-RELATED QUALITY OF LIFE AFTER KIDNEY TRANSPLANTATION ARE ADVERSELY AFFECTED BY DIABETES AND CADAVERIC ORGANS. (Abstract #250)**
David H. VanBuren, William Nylander, Irene Feurer, Theodore Speroff, Robert E. Richie, Simin Goral, Keith Johnson, Harold Helderman, Christina Ynares, Denise VanBuren, Jeannie Hopkins, Sarah Swanson, Jackie Ray, Paul E. Wise, C. Wright Pinson. Nashville, TN; Nashville, TN; Nashville, TN.

P64 **LAPAROSCOPIC LIVE DONOR NEPHRECTOMY (LapNx) RESULTS IN INCREASED UTILIZATION OF LEFT KIDNEY GRAFTS WITH MULTIPLE RENAL ARTERIES - A SURGICAL AND NONSURGICAL OUTCOME ANALYSIS OF 79 CONSECUTIVE CASES. (Abstract #251)**
Christoph Troppmann, K. Wiesmann, B. Wolfe, J. P. McVicar, R. V. Perez. Sacramento, CA.

P65 **INCREASED SENSITIVITY OF FLOW CLASS I PRA BEADS IN DETECTING HLA CLASS I ANTIBODY LEVELS (PRA) COMPARED WITH ANTIGLOBULIN (AHG) IN CANDIDATES FOR CADAVERIC RENAL RETRANSPLANTATION. (Abstract #252)**
Christopher F. Bryan, Scott B. McDonald, Karen A. Baier, Alan M. Luger, Mark I. Aeder, Daniel Murillo, Nicolas A. Muruve, Paul W. Nelson, Charles F. Shield, III, Bradley A. Warady. Westwood, KS.

P66 **OXIDANT STRESS AND ANTIOXIDANT CAPACITY IN URINE OF RENAL TRANSPLANT RECIPIENTS PREDICTS EARLY GRAFT FUNCTION. (Abstract #253)**
Daniel A. Shoskes, Asha R. Shahed, Sun Kim, Hans A. Gritsch, Gabriel Danovitch, Alan Wilkinson. Fort Lauderdale, FL; Torrance, FL; Los Angeles, CA.

P67 **MICROINVASIVE DONOR NEPHRECTOMY (MDN). (Abstract #254)**
Deepak Mital, Weislaw Podlasek, Stephen C. Jensik. Chicago, IL.

P68 **COMPARISON OF CREATININE CLEARANCE AND SERUM CREATININE SIX MONTHS AFTER RENAL TRANSPLANTATION AS PREDICTORS OF LONGTERM GRAFT SURVIVAL. (Abstract #255)**
Colin Geddes, Carl Cardella, Daniel Cattran, Stanley Fenton, Edward Cole. Toronto, ON, Canada; Glasgow, United Kingdom.

P69 **A COMPARISON OF CLINICAL AND HUMANISTIC OUTCOMES IN RENAL TRANSPLANT RECIPIENTS AT ONE AND TEN YEARS. (Abstract #256)**
Elizabeth A. Davies, Lisa R. Raiz, Ronald M. Ferguson. Columbus, OH; Athens, OH.

P70 **THE USE OF MINORS AS LIVE KIDNEY DONORS. (Abstract #257)**
Francis L. Delmonico, William E. Harmon. Boston, MA; Boston, MA.

P71 **BILATERAL PHARMACOKINETIC INTERACTION BETWEEN CYCLOSPORINE A AND ATORVASTATIN IN RENAL TRANSPLANT RECIPIENTS. (Abstract #258)**
Hallvard Holdaas, Anders Aasberg, Anders Hartmann, Ellen Fjeldsaa, Stein Bergan. Oslo, Norway; Oslo, Norway.

P72 **LAPAROSCOPIC DONOR NEPHRECTOMY IN THE PRESENCE OF MULTIPLE RENAL ARTERIES. (Abstract #259)**
Hazem I. Abou El Fettouh, Inderbir S. Gill, Anoop M. Meraney, Brian Herts, Andrew C. Novick, David A. Goldfarb. Cleveland, OH.

P73 **THE FUNCTIONAL WEIGHT - A NEW SCORE FOR EVALUATION OF OLD DONORS. (Abstract #260)**
Heiner H. Wolters, Thorsten Vowinkel, Jens G. Brockmann, Norbert J. Senninger, Karl-Heinz Dietl. Muenster, Germany.

P74 **EXCELLENT SHORT-TERM OUTCOME OF ABO-INCOMPATIBLE RENAL TRANSPLANTATION UNDER TACROLIMUS AND MYCOPHENOLATE MOFETIL IMMUNOSUPPRESSION. (Abstract #261)**
Hiroaki Shimmura, Kazunari Tanabe, Tadahiko Tokumoto, Fusako Toda, Shohei Fuchinoue, Satoshi Teraoka, Hiroshi Toma. Tokyo; Tokyo, Japan.

P75 **PROSPECTIVE NON RANDOMIZED STUDY COMPARING LAPAROSCOPIC DONOR NEPHRECTOMY (LDN), MINIMALLY INVASIVE OPEN DONOR NEPHRECTOMY (MIODN) AND OPEN DONOR NEPHRECTOMY (ODN). (Abstract #262)**
Ignacio T. Castillon-Vela, Arturo Martinez, Evan Vapnek, Vladimir Ayvazyan, Sergey Ayvazyan, Giouziele Nabieva, Robert Naraghi, Roberto Mendez, Rafael G. Mendez, Hamid Shidban.

Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics I

P76 **REDUCTION OF IMMUNOSUPPRESSION FOLLOWING BONE MARROW INFUSIONS IN LIVER TRANSPLANTATION. (Abstract #263)**
A. G. Tzakis, D. Weppler, C. Ricordi, R. Garcia-Morales, R. Cirrocco, N. Kenyon, C. Nery, M. R. Carreno, P. Ruiz, D. Levi, V. Esquenazi, A. D. Pinna, J. Miller. Miami, FL; Miami, FL; Miami, FL; Miami, FL.

P77 **HYPERLIPIDEMIA IN PEDIATRIC LIVER TRANSPLANT PATIENTS ON CYCLOSPORINE-BASED IMMUNOSUPPRESSION. (Abstract #264)**
Hansa M. Gupta, Mohamed Abdolell, Annie H. Fecteau. Toronto, ON, Canada.

P78 **QUESTION OF STEROID WITHDRAWAL UNDER TACROLIMUS FOR PRIMARY BILIARY CIRRHOSIS (PBC), PRIMARY SCLEROSING CHOLAGITIS (PSC) AND AUTOIMMUNE HEPATITIS (AIH) AFTER LIVER TRANSPLANTATION AND LONG-TERM SURVIVAL. (Abstract #265) ♦**
Ashok B. Jain, Randeep S. Kashyap, Santosh Potdar, John J. Fung. Pittsburgh, PA.

P79 **IMMUNOPROPHYLAXIS WITH SIMULECT® (BASILIXIMAB) IN COMBINATION WITH CYCLOSPORINE AND STEROIDS IN LIVER TRANSPLANTATION. (Abstract #266)**
Gerrit Grannas, Rainer Lueck, Thomas Becker, Ernst Kuse, Juergen Klempnauer, Bjoern Nashan. Hannover, Germany.

P80 **TACROLIMUS PHARMACOKINETICS IN THE EARLY PHASE AFTER LIVER TRANSPLANTATION. (Abstract #267)**
Felix Braun, Beatrice Peters, Ekkehard Schütz, Thomas Lorf, Giuliano Ramadori, Michael Oellerich, Burckhardt Ringe. Göttingen, Germany; Göttingen, Germany; Göttingen, Germany.

P81 **OPTIMAL DOSING OF MYCOPHENOLATE MOFETIL (MMF) IS NECESSARY TO DELAY HEPATITIS C RECURRENCE (HEP CR) IN LIVER TRANSPLANT RECIPIENTS (OLT). (Abstract #268)**
Carlos G. Fasola, George J. Netto, Linda W. Jennings, Laura L. Christensen, Ernesto P. Molmenti, Edmund Q. Sanchez, Shigeru Marubashi, Brian M. Gogel, Thomas A. Gonwa, Robert M. Goldstein, Marlon F. Levy, Goran B. Klintmalm. Dallas, TX.

P82 SIROLIMUS IMMUNOSUPPRESSION IN LIVER TRANSPLANTATION: AVOIDING CALCINEURIN INHIBITOR NEURO- AND NEPHROTOXICITY. (Abstract #269)
Daniel H. Kosoy, Glenda Meeberg, David L. Bigam, AM James Shapiro, Winnie Wong, Mang Ma, Klaus Gutfreund, Vincent Bain, Norman M. Kneteman. Edmonton, AB, Canada.

P83 MEASUREMENT OF EPSTEIN-BAR VIRUS (EBV) GENOMIC TITRE IN BLOOD OF PAEDIATRIC LIVER TRANSPLANT RECIPIENTS RECEIVING EITHER CYCLOSPORIN (NEORAL) OR TACROLIMUS (TAC)-BASED IMMUNOSUPPRESSION. (Abstract #270)
David Mutimer, Narinder Kaur, the European Paediatric Tacrolimus vs. Cyclosporin Microemulsion Liver Transplant Study Group. Birmingham, United Kingdom.

P84 AN OPEN LABEL, ACTIVE CONTROLLED STUDY TO EVALUATE THE USE OF MYCOPHENOLATE MOFETIL (CELLCEPT®) IN POST LIVER TRANSPLANT PATIENTS WITH RENAL IMPAIRMENT WHO ARE CURRENTLY ON CYCLOSPORINE / FK506. (Abstract #271)
David J. Reich, Pierre A. Clavian, Leona Kim-Schluger, Ernest E. Hodge, for the Renal Dysfunction after Liver Transplantation Study Group. Philadelphia; Durham; NYC; Nutley, NJ.

P85 DOES EARLY TREATMENT OF HEPATITIS C VIRUS (HCV) WITH ANTI-VIRAL THERAPY ALTER USE OF IMMUNOSUPPRESSIVE DRUGS OR RISK OF ACUTE REJECTION IN LIVER TRANSPLANT (OLT) RECIPIENTS? (Abstract #272)
David J. Heffernan, Mandana Khalili, Kathy Bollinger, Nancy L. Ascher, John P. Roberts, Norah A. Terrault. San Francisco; San Francisco.

P86 APROTININ USE IN PEDIATRIC LIVER TRANSPLANTATION. (Abstract #273)
E. Haase, D. Bigam, A. Senthilselcan, C. Tang, J. Shapiro, N. Kneteman. Edmonton, AB, Canada.

P87 NEW METHOD FOR DETERMINATION OF EBV VIRAL LOAD IN TRANSPLANT RECIPIENTS. (Abstract #274)
David Witte, Pam Groen, John Bucuvalas, Fred Ryckman, Maria Alonso, Thomas Gross. Cincinnati, OH.

Liver - Infections, Complications, Recurrent Disease, Surgical Techniques I

P88 SPLIT-LIVER TRANSPLANTS FOR 2 ADULT RECIPIENTS; AN INITIAL EXPERIENCE. (Abstract #275)
Abhi Humar, Raja Kandaswamy, Timothy Sielaff, Rainer Gruessner, Marci Knaak, Jack Lake, William Payne. Minneapolis, MN.

P89 NEED FOR RIBAVIRIN DOSE ADJUSTMENT IN RELATION TO RENAL FUNCTION FOR TREATMENT OF RECURRENT HCV INFECTION IN POST LIVER TRANSPLANT PATIENTS. (Abstract #276)
Ashok B. Jain, Randeep S. Kashyap, Forrest Dodson, Akhtar Khan, Obaid Shakil, John J. Fung. Pittsburgh, PA.

P90 WHAT IS THE IMPACT OF TRANSARTERIAL CHEMOEMBOLIZATION BEFORE LIVER TRANSPLANTATION FOR HEPATOCELLULAR CARCINOMA? (Abstract #277)
Burckhardt Ringe, Thomas Lorf, Ruben Canelo, Ulrich Hanack, Laszlo Fuezesi, Giuliano Ramadori. Goettingen, Germany; Goettingen, Germany; Goettingen, Germany.

P91 LIVER TRANSPLANTATION FOR CIRRHOSIS FOLLOWING JEJUNOILEAL BYPASS: ANALYSIS OF IMMEDIATE BYPASS REVERSAL. (Abstract #278)
Santosh Potdar, Anthony J. Demetris, S. Forrest Dodson, J. Wallis Marsh, David J. Kramer, John J. Fung, C. Andrew Bonham. Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA.

P92 HEPATITIS C RECURRENCE (HCR) IN LIVER TRANSPLANT RECIPIENTS (OLT): PATTERNS (PR) AND EARLY PREDICTORS (EP) OF GRAFT FIBROSIS (GF). (Abstract #279)
Carlos G. Fasola, George J. Netto, Linda W. Jennings, Ernesto P. Molmenti, Edmund Q. Sanchez, Shigeru Marubashi, Brian M. Gogel, Jeffrey S. Weinstein, Thomas A. Gonwa, Robert M. Goldstein, Marlon F. Levy, Goran B. Klintmalm. Dallas, TX.

P93 ACUTE GASTROINTESTINAL BLEEDING FOLLOWING ORTHOTOPIC LIVER TRANSPLANTATION IN THE SETTING OF LOW DOSE STEROID IMMUNOSUPRESSION. (Abstract #280)
Toby Richards, Ulrike Heiber, Walter Mark, Bridget Gunson, John Buckels, David Mayer, Darius Mirza, Paul McMaster, Daniel Candinas. Birmingham, England, United Kingdom.

P94 BILIARY COMPLICATIONS ASSOCIATED WITH CYTOMEGALOVIRUS (CMV) HEPATITIS IN ORTHOTOPIC LIVER TRANSPLANT (OLT) RECIPIENTS. (Abstract #281)
Manuel I. Rodriguez-Davalos, David D. Douglas, Eric A. Huettl, David M. Kasper, Wyn Harrison, Vijayan Balan, Adyr A. Moss, Mamoud Yousfi, Jorge Rakela, David C. Mulligan. Phoenix, AZ.

P95 APROTONIN EFFECTS BLOOD PRODUCTS USAGE AND REOPERATIVE RATE FOR BLEEDING FOLLOWING ORTHOTOPIC LIVER TRANSPLANTATION. (Abstract #282)
Maher A. Abbas, Peter E. Frasco, Jorge Rakela, M. Edwin Harrison, Adyr A. Moss, David D. Douglas, Vijayan Balan, Joseph G. Hentz, David C. Mulligan. Phoenix, AZ; Phoenix, AZ.

P96 THE PRESENCE OF A CONTEMPORANEOUS LIVER GRAFT IN INTESTINAL TRANSPLANTATION. (Abstract #283)
Debbie Weppler, Seigo Nishida, Levi David, Tomoaki Kato, Jose R. Nery, Antonio D. Pinna, Patricia Cantwell, Naveen Mittal, Phillip Ruiz, Camillo Ricordi, Andreas G. Tzakis. Miami, FL; Miami, FL; Miami, FL; Miami, FL.

P97 IN-SITU FULL RIGHT-FULL LEFT SPLITTING: THE ULTIMATE EXPANSION OF THE ADULT DONOR POOL. (Abstract #284)
Dieter C. Broering, Matthias Gundlach, Stefan Topp, Lars Mueller, Xavier Rogiers. Hamburg, Germany.

P98 WARM ISHCEMIA TIME DOES NOT AFFECT DEVELOPMENT OF SEVERE RECURRENT HEPATITIS C FOLLOWING LIVER TRANSPLANTATION. (Abstract #285)
Erik Kerekes, Donald J. Hillebrand, Pedro W. Baron, Ke-Qin Hu, OK Ojogho, Waldo Concepcion. Loma Linda, CA; Loma Linda, CA.

P99 IMPORTANT SURGICAL LESSONS LEARNED FROM ADULT RIGHT LOBE LIVING RELATED LIVER TRANSPLANTS. MINIMIZING SURGICAL COMPLICATIONS. (Abstract #286)
Douglas W. Hanto, James F. Whiting, Scott R. Johnson, Maria H. Alonso, Doan N. Vu, John P. Alspaugh, Joseph F. Buell, Michael J. Hanaway. Cincinnati, OH; Portland, ME; Cincinnati, OH; Cincinnati, OH.

P100 IMMUNOSUPPRESSIVE EFFECT ON RECURRENCE OF PRIMARY BILIARY CIRRHOSIS AFTER LIVER TRANSPLANTATION. (Abstract #287)
Edmund Q. Sanchez, Derek Byers, Shigeru Marubashi, Brian M. Gogel, Marlon F. Levy, Robert M. Goldstein, Ernesto P. Molmenti, Carlos G. Fasola, Thomas A. Gonwa, G. Weldon Tillery, George J. Netto, David L. Watkins, Barbara K. Brooks, Laura L. Christensen, Goran B. Klintmalm. Dallas, TX.

P101 SUSTAINED RESPONSE WITH INTERFERON/ RIBAVIRIN FOR RECURRENT HEPATITIS C AFTER LIVER TRANSPLANTATION. (Abstract #288)
Fredric D. Gordon, Carole Davis, Denise Morin, Urmila Khettry, W. David Lewis, Elizabeth A. Pomfret, James J. Pomposelli, Eric D. Goldberg, Roger L. Jenkins. Burlington, MA; Burlington, MA.

P102 MONOSEGMENT GRAFT IN LIVING DONOR LIVER TRANSPLANTATION. (Abstract #289)
Fumitaka Oike, Seisuke Sakamoto, Mureo Kasahara, Tetsuya Kiuchi, Hiroto Egawa, Shinji Uemoto, Koichi Tanaka. Kyoto, Kyoto, Japan.

P103 RIBAVIRIN MONOTHERAPY IN LIVER TRANSPLANT RECIPIENTS WITH RECURRENT CHRONIC HCV. A PILOT STUDY. (Abstract #290)
Giuseppe Tisone, Elena Torri, Antonino Araco, Alessandro Anselmo, Carlo Camplone, Settimio Zazza, Mario Cepparulo, Giampiero Palmieri, Mario Angelico, Carlo Umberto Casciani. Rome, Italy.

Liver - Preservation, Economics/Public Policy, Donation Allocation, Other I

P104 OUTCOME OF OTHER ORGANS RECOVERED DURING IN SITU SPLIT LIVER PROCUREMENTS. (Abstract #291)
Thiagarajan Ramcharan, Meg Rogers, Raja Kandaswamy, Timothy Sielaff, Rainer W. Gruessner, Jack Lake, William Payne, Abhi Humar. Minneapolis, MN.

P105 RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF SKIN CANCERS AFTER LIVER TRANSPLANTATION. (Abstract #292)
Abigail B. Warshaw, Stacey Supran, Richard B. Freeman.

P106 THE CONSEQUENCES OF PORTAL VEIN THROMBOSIS WITH LENGTHENING WAITING TIMES PRIOR TO ADULT LIVER TRANSPLANTION. (Abstract #293)
Andrew L. Singer, Gideon Zamir, Niraj Desai, James F. Markmann, Abraham Shaked, Kim M. Olthoff. Philadelphia, PA.

P107 NEW COMPUTATIONAL METHODS FOR THE EVALUATION OF LIVING RELATED LIVER DONATION. (Abstract #294)
Bernd B. Frericks, Franco C. Caldarone, Andrea Schenk, Dirk Selle, Bernhard Preim, Heinz O. Peitgen, Michael Galanski, Bjoern Nashan, Juergen Klempnauer. Hannover, Germany; Bremen, Germany; Hannover, Germany.

P108 FIFTY LIVING DONOR LIVER TRANSPLANTS WITH ZERO MORTALITY. (Abstract #295)
Chao-Long Chen, Yaw-Sen Chen, Vanessa H. de Villa, Chih-Chi Wang, Po-Ping Liu, Shih-Hor Wang, Yuan-Cheng Chiang, Yu-Fan Cheng, Tung-Liang Huang, Bruno Jawan, Hak-Kim Cheung, Hock-Liew Eng. Kaohsiung, Taiwan, Province of China.

P109 RECONSIDERING THE IMPACT OF COLD ISCHEMIA TIME ON GRAFT AND PATIENT SURVIVAL AFTER LIVER TRANSPLANTATION. (Abstract #296) ♦
David M. Levi, Seigo Nishida, Tomoaki Kato, Jose Nery, Guy Neff, Les Olson, Andreas G. Tzakis. Miami, FL.

P110 OCTOGENERIAN LIVER DONORS: SHOULD THEY BE EXCLUDED? (Abstract #297)
Bashar A. Aqel, Justin H. Nguyen, James R. Spivey, Jeffery L. Steers, Christopher B. Hughes, Rolland C. Dickson, Denise M. Harnois. Jacksonville, FL; Jacksonville, FL.

P111 ADULT-TO-ADULT LIVING LIVER DONATION HOSPITAL RESOURCE UTILIZATION AND REIMBURSEMENT ECONOMICS: OUR INITIAL EXPERIENCE. (Abstract #298)
Edward Y. Zavala, Kelly Rolfes, Erin Dougherty, Lea Pirro, Jaime Aranda-Michel, Douglas W. Hanto. Cincinnati, OH; Cincinnati, OH; Cincinnati, OH; Cincinnati, OH.

P112 100 DONOR EVALUATIONS AND SURGICAL OUTCOME AFTER LIVE DONOR ADULT LIVER TRANSPLANTATION (LDALT). (Abstract #299) ♦
Elizabeth A. Pomfret, James J. Pomposelli, David L. Burns, Fredric D. Gordon, William D. Lewis, Roger L. Jenkins. Burlingon, MA.

P113 CAN PATIENTS AWAITING LIVER TRANSPLANTATION ELICIT AN IMMUNE RESPONSE TO THE HEPATITIS A VACCINE? (Abstract #300)
Gregory A. Smallwood, Christina T. Coloura, Michael E. de Vera, Enrique Martinez, Andrei C. Stieber, Thomas G. Heffron. Atlanta, GA; Atlanta, GA; Atlanta, GA.

P114 PREGNANCY OUTCOME AFTER LIVER TRANSPLANTATION. (Abstract #301)
Gustavo Braslavsky, Donato Spaccavento, Pedro Trigo, Nora Cejas, Javier Lendoire, Orlando Juarez, Osvaldo Parada, Oscar Imventarza. Buenos Aires, Argentina.

P115 PSYCHOSOCIAL RISK EVALUATION AND OUTCOME AFTER LIVER TRANSPLANTATION. (Abstract #302)
Gustavo Braslavsky, Catherine Rosas, María José Ferrari, Dante Fauceglia, Javier Lendoire, Oscar Imventarza. Buenos Aires, Argentina.

P116 EFFICACY AND SAFETY OF ANTI-HYPERTENSIVE AGENTS IN LIVER TRANSPLANT RECIPIENTS. (Abstract #303)
Iman Bajjoka, Saeed Rasty, Viken Douzdjian, Kimberly Brown, Dilip Moonka, Lawton Shick, Atsushi Yoshida, Marwan Abouljoud. Detroit, MI; Orlando, FL; Detroit, MI; Detroit, MI.

Pancreas and Islets - All Topics I

P117 THE IMPACT OF ELDERLY AND PEDIATRIC DONORS ON OUTCOME AFTER SPK TRANSPLANT. (Abstract #304)
Abhi Humar, Raja Kandaswamy, Thiagarjan Ramcharan, Steven Paraskevas, Roger Denis, Rainer W. Gruessner, Angelika Gruessner, David E.R. Sutherland. Minneapolis, MN.

P118 ANALYSIS OF ACUTE AND CHRONIC REJECTION PATTERNS AFTER PANCREAS TRANSPLANT ALONE(PTA). (Abstract #305)
Abhi Humar, Raja Kandaswamy, Thiagarjan Ramcharan, Steven Paraskevas, Roberto Meirelles, Rainer W. Gruessner, Angelika Gruessner, David E.R. Sutherland. Minneapolis, MN.

P119 PANCREAS TRANSPLANTATION FOR TYPE 2 DIABETES. (Abstract #306)
Amy L. Friedman, Eli A. Friedman. New Haven, CT; Brooklyn, NY.

P120 IMPROVEMENT OF ECHOCARDIOGRAPHIC FUNCTION AND ARTERIAL BLOOD PRESSURE ONE YEAR AFTER SIMULTANEOUS PANCREAS KIDNEY TRANSPLANTATION (SPK). (Abstract #307)
Andreas Kahl, Michael Oppert, Andre Godau, Frank Lorenz, Ralph Bartels, Hans P. Lehmkuhl, Wolfgang Bocksch, Eckard Fleck, Andrea R. Müller, Wolf O. Bechstein, Peter Neuhaus, Ulrich A. Frei. Berlin, Germany; Berlin, Germany; Berlin, Germany.

P121 IMPROVED PANCREAS TRANSPLANTATION OUTCOMES IN AFRICAN AMERICANS (AA) WITH PORTAL VENOUS DRAINAGE. (Abstract #308)
T. Direnzo, Anne M. Wiland, Clarence Foster, Benjamin Philosophe, Gene Schweitzer, Alan Farney, J. Colonna, Jeffrey Fink, Matthew Weir, Stephen T. Bartlett. Baltimore, MD; Baltimore, MD; Baltimore, MD.

P122 USE OF RAPAMYCIN IN PANCREATIC TRANSPLANTATION UNDER TACROLIMUS BASED IMMUNOSUPPRESSION. (Abstract #309)
Ashok B. Jain, Velma Scantlebury, Victor Garrido, Pradip Chakrabarti, Ron Shapiro, Robert Corry. Pittsburgh, PA.

P124 **LONG-TERM PANCREAS ALLOGRAFT SURVIVAL IN MYCOPHENOLATE MOFETIL TREATED PANCREAS RECIPIENTS COMPARED TO AZATHIOPRINE TREATED PANCREAS RECIPIENTS. (Abstract #311)**
David K. Klassen, Jung M. Oh, Anne M. Wiland, Jeffrey C. Fink, Matthew R. Weir, Stephen T. Bartlett. Baltimore, MD; Seoul Knea; Baltimore, MD; Baltimore, MD.

P125 **SAFETY AND EFFICACY OF PERCUTANEOUS PANCREAS ALLOGRAFT BIOPSY: A LARGE SINGLE CENTER SERIES. (Abstract #312)**
David K. Klassen, Matthew R. Weir, Charles B. Cangro, Emilio R. Ramos, Jeffrey C. Fink, Ravinder K. Wali, Rochelle M. Cunningham, Stephen T. Bartlett. Baltimore, MD; Baltimore, MD.

P126 **CONVERSION TO SIROLIMUS-BASED MAINTENANCE IMMUNOSUPPRESSIVE THERAPY IN PANCREAS ALLOGRAFT RECIPIENTS. (Abstract #313)**
David K. Klassen, Anne M. Wiland, Matthew R. Weir, Benjamin Philosophe, Stephen Bartlett. Baltimore, MD; Baltimore, MD; Baltimore, MD.

Heart/Lung - All Topics I

P127 **EARLY ACUTE REJECTION IN PEDIATRIC LUNG TRANSPLANT RECIPIENTS. (Abstract #314)**
Albert Faro, Kelli Harker, Deborah Kahler, Barbara Williams, Edward D. Staples, Gary A. Visner. Gainesville, FL.

P128 **EFFICIENCY OF HYPERIMMUNOGLOBULINE CMV-PROPHYLAXIS IN HEART TRANSPLANT RECIPIENTS. (Abstract #315)**
Alfred A. Kocher, Meinhard Ploner, Barbara Zweytick, Ernst Wolner, Michael Grimm, Marek Ehrlich, Bernhard Schlechta, Georg Wieselthaler, Guenther Laufer.

P129 **FLOW-PRA MONITORING POST HEART TRANSPLANTATION. (Abstract #316)**
Anat R. Tambur, Biljana Surjancev, Susan Shott, Alain Heroux, Walter Kao, Elaine Winkel, Mitchell T. Saltzberg, Maria Rosa Costanzo. Chicago, IL.

P130 **EVEN SLIGHTLY IMPAIRED KIDENY FUNCTION (CREATININE >1.79 mmol/l) ONE YEAR AFTER CARDIAC TRANSPLANTATION HAS A NEGATIVE IMPACT ON LONG TERM SURVIVAL. (Abstract #317)**
Andreas Zuckermann, Meinhard Ploner, Martin Czerny, Keziban Uenal, Ernst Wolner, Guenther Laufer, Micheal Grimm. Vienna, Austria.

P131 **LOW INCIDENCE OF GRAFT ARTERIOSCLEROSIS AFTER CARDIAC TRANSPLANTATION - RISK FACTOR ANALYSIS FOR PATIENTS WITH INDUCTION THERAPY. (Abstract #318)**
Andreas O. Zuckermann, Meinhard Ploner, Martin Czerny, Keziban Uenal, Tudor Birsan, Guenther Laufer, Ernst Wolner, Michael Grimm. Vienna, Austria.

P132 **SENSITIVITY OF THE FLOW CYTOMETRY CROSSMATCH IN HEART TRANSPLANTATION. (Abstract #319) ♦**
Ashraf S. Abdo, Daniel J. Cook, James F. McCarthy, Ehab S. Bishay, Ganesh S. Kumpati, Randall C. Starling, James B. Young, Mohamad H. Yamani, Nicholas G. Smedira, Patrick M. McCarthy. Cleveland, OH.

P133 **RENAL SPARING (CYCLOSPORINE FREE) PROTOCOL FOR CARDIAC TRANSPLANT RECIPIENTS. (Abstract #320)**
Barbara A. Pisani, Zyed Tai, Jose C. Mendez, Robert C. Lichtenberg, Bryan K. Foy, Mark J. Stout, Krystyna Malinowska, G. Martin Mullen, John A. Robinson. Maywood, IL.

P134 **TACROLIMUS/MYCOPHENOLATE MOFETIL VS CYCLOSPORINE/MYCOPHENOLATE MOFETIL AFTER HTX: IMAPACT ON CORONARY VASOMOTOR FUNCTION AND MYCOPHENOLATE ACID TROUGH LEVELS. (Abstract #321) ♦**
Bruno M. Meiser, Jan Groetzner, Johannes Schirmer, Soeren Schenk, Wolfgang von Scheidt, Michael Weis, Volker Klauss, Hans U. Stempfle, Hermann Reichenspurner, Bruno Reichart. Munich, Germany; Munich, Germany; Munich, Germany.

P135 **THE FIRST MULTICENTRE TACROLIMUS HEART PILOT STUDY: THREE YEAR FOLLOW-UP. (Abstract #322)**
Bruno M. Meiser, Bruno Reichart, Mario Viganò, Mauro Rinaldi, Magdi Yacoub, Nicholas R. Banner, Iradj Gandjbakhch, Richard Dorent, Manfred Hummel, Roland Hetzer. Munich, Germany; Pavia, Italy; Harefield, United Kingdom; Paris, France; Berlin, Germany.

P136 **RELATIONSHIP BETWEEN TROPONIN I LEVELS AND STATUS OF VASCULAR ANTITHROMBIN BINDING IN HEART TRANSPLANT PATIENTS. (Abstract #323)**
Carlos A. Labarrere, David Nelson.

P137 **LACK OF CHRONIC CD4 T CELL ACTIVATION IN CALVES TRANSPLANTED WITH THE ABIOCOR TOTAL IMPLANTABLE REPLACEMENT HEART (IRH. (Abstract #324)**
Christina L. Kaufman, L. Madison Ryle, Melo Nicolas, Karla Stevens, Robert D. Dowling. Louisville, KY; Louisville, KY; Louisville, KY.

P138 **BEDSIDE TROPONIN T MEASUREMENT IMPROVES THE SELECTION OF HEART DONORS AND REDUCE THE EARLY GRAFT FAILURE IN RECIPIENTS. (Abstract #325)**
Evgenij V. Potapov, Matthias Loebe, Onnen Grauhan, Ekaterina A. Ivanitskaia, Wolfgang Zuelke, Heidi Kriegler, Ralf Sodian, Miralem Pasic, Yuguo Weng, Roland Hetzer. Berlin, Germany.

P139 **CAN OUTCOME OF LEFT VENTRICULAR ASSIST DEVICE IMPLANTATION AS A BRIDGE TO ORTHOTOPIC HEART TRANSPLANT BE PREDICTED? (Abstract #326)**
Beth L. Tumilty, Krystyna Malinowska, Ramon Durazo-Arviru, Barbara A. Pisani, Jose C. Mendez, Robert C. Lichtenberg, John A. Robinson, Bryan K. Foy, Mamdouh Bakhos, G. Martin Mullen. Maywood, IL.

P140 **WHAT PROGNOSTIC FACTORS IN ADVANCED HEART FAILURE PATIENTS PREDICT ASSIST DEVICE PLACEMENT? (Abstract #327)**
Geetha Bhat, Lori Muncy. Louisville, KY; Louisville, KY.

P141 **HISTOPATHOLOGICAL FINDINGS IN HEART TRANSPLANT PATIENTS UNDER TACROLIMUS-MYCOPHENOLATE MOFETIL VERSUS CYCLOSPORINE-AZATHIOPRINE. (Abstract #328)**
José L. Sgrosso, Jaime Ferrer, Graciela L. Araujo, Ligia Romeo, Coloma Parisi, María C. Vázquez. Santa Fe, Argentina; Buenos Aires, Argentina.

Bone Marrow - All Topics I

P142 **FTY720 INTERACTIONS WITH THE P-GLYCOPROTEIN PUMP IN T CELLS: PRELIMINARY OBSERVATIONS. (Abstract #329)**
Aarati R. Ranade, Vera S. Donnenberg, Gilbert J. Burckart, Heather L. Galant-Haidner, Barry D. Kahan, Albert D. Donnenberg. Pittsburgh, PA; Houston, TX.

P143 **CMV STATUS IN PATIENTS WITH TYPE 1 DIABETES MELLITUS AWAITING ISLET CELL TRANSPLANTATION. (Abstract #330)**
Aileen M. Caulfield, Tatiana Froud, Jacqueline V. Ferreira, Thierry Berney, Andres Boker, Andreas Tzakis, Camillo Ricordi, Rodolfo Alejandro. Miami, FL.

P144 **THE MOTTEP MODEL: INCREASING DONATION RATES AND PREVENTING THE NEED FOR TRANSPLANTATION. (Abstract #331)**
Clive O. Callender, Margruetta B. Hall, Doreen Branch, Patrice V. Miles. Washington, DC.

P145 **IMPROVED ORGAN YIELD USING HIGH DOSE STEROIDS DURING DONOR MANAGEMENT. (Abstract #332)**
Robert C. Kincade, David M. Follette, Richard V. Perez, Wayne D. Babcock. Sacramento, CA; San Francisco, CA.

P146 **PATIENT OUTCOMES REGISTRY FOR TRANSPLANT EFFECTS ON LIFE (PORTEL)- THE FIRST REPORT OF A NATIONWIDE LONGITUDINAL QUALITY OF LIFE STUDY IN TRANSPLANT RECIPIENTS. (Abstract #333)**
Donna Hathaway, Mary M. Prendergast, Lynn Fallon, Amber Ives. Memphis, TN; Deerfield, IL; Cincinnati, OH.

P147 **THE "SPANISH MIRACLE" VS. THE U.S. - A DIFFERENCE IN ORGAN UTILIZATION? (Abstract #334)**
George J. Chang, Harish D. Mahanty, Mounir A. Hajjar, Ryutaro Hirose, Chris E. Freise, Peter G. Stock, Nancy L. Ascher, John P. Roberts. San Francisco, CA.

P148 **LATE POST TRANSPLANT OPERATIONS IN LIVER, KIDNEY AND PANCREAS TRANSPLANTATION PATIENTS. ANALYSIS OF OUTCOME, MORBIDITY AND MORTALITY IN 160 CONSECUTIVE TRANSPLANT PATIENTS. (Abstract #335)**
Hadar J. Merhav, Sigal Eisner, Richard Nakasche, Mordechai Gutman, Amir Szold, Joseph Klauzner. Tel Aviv, Israel.

P149 **LIPOSOMAL AMPHOTERICIN B (AMBISOME®) IS SAFE AND EFFECTIVE IN TREATING INVASIVE FUNGAL INFECTIONS (IFI) IN TRANSPLANT PATIENTS AND IN PROPHYLAXIS AGAINST IFI IN LIVER TRANSPLANT (OLT) PATIENTS. (Abstract #336)**
Hadar J. Merhav, Ruth Wasserlauf, Wassam Houri, Richard Nakasche, Yardena Igra, Patrick Sorkin. Tel Aviv, Israel; Tel Aviv, Israel; Tel Aviv, Israel.

P150 **ORGAN AND TISSUE DONATION ATTITUDE SCALES FOR PHYSICIANS AND NURSES: GETTING THE DOCTOR TO ASK. (Abstract #337)**
Jan Niesing, Jack A. Jenner, Rutger J. Ploeg. Groningen, The Netherlands; Groningen, The Netherlands.

P151 **ORDAS/TIDAS: AN INSTRUMENT TO SUPPORT PHYSICIANS AND NURSES IN ASKING THE DONATION QUESTION FOR POTENTIAL ORGAN AND TISSUE DONORS. (Abstract #338)**
Jan Niesing, Rutger J. Ploeg. Groningen, The Netherlands.

P152 **PERCEPTIONS ABOUT ORGAN DONATION AND TRANSPLANTATION AMONG AFRICAN-AMERICAN HEALTH CARE AND NON-HEALTHCARE PROFESSIONALS: PRELIMINARY FINDINGS FROM A FOCUS GROUP APPROACH. (Abstract #339)**
John D. Thornton, Clarence Spigner, Margaret D. Allen. Seattle, WA; Seattle, WA; Seattle, WA.

P153 **CAN PATIENTS WITH CNS TUMORS SAFELY BE USED IN EXPANDING THE DONOR POOL? (Abstract #340)**
J. F. Buell, J. Trofe, M. J. Hanaway, M. R. First, T. Beebe, V. R. Peddi, H. L. Rilo, E. S. Woodle. Cincinnati, OH.

Immunosuppression, Preclinical Studies I

P154 **EFFECT OF p21 OVER-EXPRESSION ON GRAFT SURVIVAL IN A RAT HEART TRANSPLANT MODEL. (Abstract #341)**
Ashwani K. Khanna. Milwaukee, WI.

P155 **EFFICACY OF DOUBLE FILTRATION PLASMAPHERESIS TO PREVENT XENO-REJECTION IN A DIRECT PERFUSION MODEL OF BIOARTIFICIAL LIVER SUPPORT SYSTEM, BY USING DISCORDANT SPECIES OF ANIMALS. (Abstract #342)**
Carmelo Puliatti, Pierfrancesco Veroux, Walter Morale, Massimiliano Veroux, Maria C. Valvo, Francesco Leone. Catania, Italy.

P156 **SPILT TOLERANCE OBSERVED IN LIMB TISSUE ALLOGRAFTS IN SWINE TREATED WITH A 12-DAY COURSE OF CYCLOSPORINE. (Abstract #343) ♦**
David W. Mathes, Mark A. Randolph, Mario G. Solari, Jamal Nazzal, David H. Sachs, W.P. Andrew Lee. Boston, MA; Boston, MA.

P157 **INHIBITION OF P21 RAS LEADS TO IN VITRO AND IN VIVO IMMUNOSUPPRESSION. (Abstract #344)**
Ping Ji, Yale D. Podnos, Ming-Sing Si, David K. Imagawa. Orange, CA.

P158 **CAMPATH-1H RESISTANT MONOCYTES ARE DEFICIENT IN INDIRECT BUT CAPABLE OF DIRECT ALLOANTIGEN PRESENTATION. (Abstract #345)**
Douglas K. Tadaki, He Xu, Lorna Graham, David M. Harlan, Allan D. Kirk.

P159 **THE NEW ANTINEOPLASTIC DRUG PACLITAXEL HAS IMMUNOSUPPRESSIVE PROPERTIES THAT CAN EFFECTIVELY PROMOTE ALLOGRAFT SURVIVAL IN A RAT HEART TRANSPLANT MODEL. (Abstract #346)**
Edward K. Geissler, Christian Graeb, Marcus N. Scherer, Martin Justl, Erika Frank, Karl-Walter Jauch, Stefan Tange. Regensburg, Germany.

P160 **SIROLIMUS IMPAIRS WOUND HEALING IN AN ANIMAL MODEL. (Abstract #347)**
Fadi Dagher, Devon John, Glyn R. Morgan, Thomas Diflo, Mohamed Wehbe, Kevin Hyman, Neil Theise, Lewis Teperman. New York, NY; New York, NY.

P161 **NOVEL PHARMACODYNAMIC ASSAYS OF LYMPHOCYTE FUNCTION AND PROLIFERATION IN PERIPHERAL BLOOD ARE AFFECTED BY MMF DOSE SCHEDULE AND CORRELATE WITH HISTOLOGICAL SEVERITY OF REJECTION IN RAT HEART TRANSPLANT RECIPIENTS. (Abstract #348)**
Jochen Klupp, Teun van Gelder, Camille Dambrin, Katrin Boeke, Jacob Regieli, Margret Billingham, Randall E. Morris. Stanford, CA; Stanford, CA; Berlin, Germany.

P162 **PHARMACODYNAMIC ASSESSMENT OF IMMUNOSUPPRESSIVE ACTIVITY OF STEROIDS - RESULTS FROM A HUMAN PHASE I PILOT STUDY. (Abstract #349)**
Jochen Klupp, Camille Dambrin, Wolfgang Jacobson, Uwe Christians, Randall E. Morris. Stanford, CA; San Francisco, CA; Berlin, Germany.

P163 **A NOVEL RETINOIC ACID RECEPTOR-ALPHA SELECTIVE AGONIST, ER-38925, SUCCESSFULLY PREVENTED ACUTE AND CHRONIC REJECTION OF MOUSE CARDIAC ALLOGRAFTS. (Abstract #350) ♦**
Ken-ichiro Seino, Katashi Fukao, Hideki Taniguchi, Yasutsugu Takada, Kenji Yuzawa, Masaaki Otsuka, Toshihiko Yamauchi. Tsukuba, Ibaraki; Kawaguchi, Saitama, Japan; Tsukuba, Ibaraki, Japan.

Tolerance I

P164 **IMMUNE STATUS OF CHIMERIC CLASS II$^{-/-}$ BMTX RECIPIENTS TREATED WITH FLT3-L AND COSTIMULATORY BLOCKADE. (Abstract #351)**
Jennifer E. Woodward, Adam T. Schaefer, Mary E. Tarara, Roger D. Sequeria, Jian-Lin Tang, Alison J. Logar, Robert Peach, Abdul S. Rao. Pittsburgh, PA; Princeton, NJ.

P165 **ALLOGRAFT ACCEPTANCE, DONOR-LINKED SUPPRESSION AND MALIGNANCY. (Abstract #352)**
Anne M. VanBuskirk, Jiao-Jing Wang, Charles G. Orosz. Columbus, OH.

P166 **DONOR MHC CLASS II IS REQUIRED FOR TOLERANCE INDUCTION BY CD45RB mAb IN MOUSE RENAL ALLOGRAFTS. (Abstract #353)**
C. A. O'Brien, P. Luke, J. Jian, B. Garcia, R. Zhong, A. M. Jevnikar. London, Canada; London, ON, Canada.

P167 **INDUCTION OF HEMATOPOIETIC CHIMERISM AND MURINE CARDIAC ALLOGRAFT TOLERANCE WITH A NON-MYELOABLATIVE REGIMEN. (Abstract #354)** ♦
Nozomu Shirasugi, Andrew B. Adams, Megan M. Durham, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

P168 **IMMUNOLOGIC TOLERANCE TO VASCULARIZED KIDNEY ALLOGRAFTS ACHIEVED THROUGH STABLE MIXED HEMATOPOIETIC CHIMERISM. (Abstract #355)**
Christian S. Kuhr, Margaret D. Allen, Christian Junghans, Eustacia Zellmer, Christopher L. Marsh, Murad Yunusov, Beverly Torok-Storb, Marie-Terese Little, Rainer Storb. Seattle, WA; Seattle, WA.

P169 **ROLE OF IL-4-MEDIATED SIGNALING IN PROLONGATION OF GRAFT SURVIVAL INDUCED BY PERITRANSPLANT T CELL DEPLETION AND 15-DEOXYSPERGUALIN. (Abstract #356)**
Clement Asiedu, James F. George, Peter D. Ray, Judith M. Thomas. Birmingham, AL.

P170 **THE EFFECT OF IMMUNOREGULATORY CD4$^+$ CELL FROM NEONATAL TOLERANT MICE ON CD8$^+$ ALLOREACTIVITY *IN VITRO*. (Abstract #357)**
Damir Matesic, Todd Rouse, Elizabeth Field. Iowa City, IA; Iowa City, IA.

P171 **ROLE OF STAT4 AND STAT6 SIGNALLING IN ANTI-CD2 MEDIATED IMMUNOSUPPRESSION. (Abstract #358)**
Dass S. Vinay, Brandon J. Wong, Guanyi Lu, Ying Wang, Keith Bishop, Jonathan S. Bromberg, Jeffrey D. Punch. Ann Arbor, MI; New York, NY.

P172 **RADIORESISTANT CD4$^+$ CELLS PRODUCE IL-4 FOLLOWING NONMYELOABLATIVE TOTAL LYMPHOID IRRADIATION TREATMENT OF MICE. (Abstract #359)**
Elizabeth H. Field, Todd Rouse, Tricia Fehr. Iowa City, IA; Iowa City, IA.

P173 **THE ROLE OF IL-4 AND IMMUNOREDIRECTION IN THE DEVELOPMENT OF MIXED-CHIMERISM IN NONMYELOABLATIVE TOTAL LYMPHOID IRRADIATED MICE. (Abstract #360)**
Elizabeth H. Field, Todd Rouse, Tricia Fehr. Iowa Ctiy, IA; Iowa City, IA.

P174 **WHICH DONOR HLA ANTIGENS CAN CAUSE BYSTANDER SUPPRESSION IN LIVER TRANSPLANT RECIPIENTS? (Abstract #361)**
Ewa Jankowska-Gan, Tonja Rhein, Lynn D. DeVito-Haynes, Felix Geissler, Munci Kalayoglu, Hans Sollinger, William J. Burlingham. Madison, WI.

Acute/Chronic Rejection I

P175 **INHIBITION OF *IN VITRO* DONOR-SPECIFIC HUMORAL AND CELLULAR IMMUNE RESPONSES IN AORTIC ALLOGRAFT RECIPIENTS RECEIVING DELAYED COSTIMULATORY BLOCKADE. (Abstract #362)**
Hong Sun, Jennifer E. Woodward, Jian-Lin Tang, Adam T. Schaefer, Mary E. Tarara, Alison J. Logar, Robert Peach, Abdul S. Rao. Pittsburgh, PA; Princeton, NJ.

P176 **ADMINISTRATION OF CTLA4-Ig AND ANTI-CD40L ANTIBODY SUBSEQUENT TO AORTIC ALLOTRANSPLANTATION MITIGATES THE DEVELOPMENT OF CHRONIC REJECTION. (Abstract #363)**
Hong Sun, Jennifer E. Woodward, Jian-Lin Tang, Vladimir Subbotin, Robert Peach, Abdul S. Rao. Pittsburgh, PA; Princeton, NJ.

P177 **THE ROLE OF DIRECT AND INDIRECT ALLORECOGNITION IN THE DEVELOPMENT OF TRANSPLANT VASCULOPATHY. (Abstract #364)**
Akira Yamada, Catharine M. Chase, Mohamed H. Sayegh, Robert B. Colvin, Paul S. Russell, Hugh Auchincloss, Jr.. Boston, MA; Boston, MA; Boston, MA.

P178 **AMD6221, A NOVEL NITRIC OXIDE SCAVENGER, DECREASES HEME PROTEIN NITROSYLATION AND PROLONGS CARDIAC ALLOGRAFT SURVIVAL. (Abstract #365)**
Allan M. Roza, Galen M. Pieper, Gail Hilton, Christopher C. Felix, Simon P. Fricker, Mark B. Adams. Milwaukee, WI; Milwaukee; Langley, BC, Canada.

179 **PASSIVE TRANSFER OF NON-COMPLEMENT-ACTIVATING ALLOANTIBODIES AUGMENTS CARDIAC ALLOGRAFT REJECTION IN IgKO MICE. (Abstract #366)** ♦
Barbara A. Wasowska, Zhiping Qian, Jodi Layton, Fred Sanfilippo, William M. Baldwin, III. Baltimore, MD.

P180 **SIROLIMUS (RAPAMYCIN) MONOTHERAPY PREVENTS GRAFT VASCULAR DISEASE IN NON-HUMAN PRIMATES. (Abstract #367)**
Camille Dambrin, Jochen Klupp, Tudor Bîrsan, Jorge Luna, Takeshi Suzuki, Tuan Lam, Peter Staehr, Bernard Hausen, Laurie Hook, Uwe Christians, Peter Fitzgerald, Gerald Berry, Randall Morris. Stanford, CA; Stanford, CA; San Francisco, CA; Stanford, CA.

P181 **PRAVASTATIN PREVENTS CHRONIC ALLOGRAFT REJECTION IN A RAT MODEL OF KIDNEY TRANSPLANTATION. (Abstract #368)**
Ping Ji, Yale D. Podnos, Ming-Sing Si, Sean Cao, David K. Imagawa. Orange, CA.

P182 **EXPRESSION OF INDUCIBLE AND ENDOTHELIAL NITRIC OXIDE SYNTHASES, FORMATION OF PEROXYNITRITE MODIFIED PROTEINS AND REACTIVE OXYGEN SPECIES IN HUMAN CHRONIC RENAL TRANSPLANT FAILURE. (Abstract #369)**
Ester W.J.A. Albrecht, Coen A. Stegeman, Anton T.M.G. Tiebosch, Adam M. Tegzess, Harry van Goor. Groningen.

P183 **STATINS PREVENT GENERATION OF REACTIVE OXYGEN SPECIES VIA INHIBITION OF PKC AND MAP KINASE. (Abstract #370)**
Faikah Abou-Rebyeh, Carsten Lindschau, Anette Fiebeler, Winfried Gwinner, Anke Schwarz, Hermann Haller.

P184 **THE EFFECT OF ACUTE REJECTION EPISODES ON INTRAGRAFT TGFβ EXPRESSION AND THE SUBSEQUENT DEVEOPMENT OF CHRONIC RENAL ALLOGRAFT INJURY. (Abstract #371)**
Geraint V. Jones, Alenka L. Janezic, Adam Jurewicz, Keshwar Baboolal. Cardiff, Wales, United Kingdom.

Allorecognition, Antigen Presentation, Co-Stimulation and Other I

P185 **COSTIMULATORY BLOCKADE IS INSUFFICIENT FOR THE ESTABLISHMENT OF DONOR CELL CHIMERISM DURING DIRECT ANTIGEN PRESENTATION. (Abstract #372)**
Jennifer E. Woodward, Roger D. Sequeria, Mary E. Tarara, Alison J. Logar, Adam T. Schaefer, Robert Peach, Abdul S. Rao. Pittsburgh, PA; Princeton, NJ.

P186 **ABSENCE OF DONOR-SPECIFIC CYTOTOXICITY IN CHIMERIC CLASS II$^{-/-}$ RECIPIENTS RECEIVING COSTIMULATORY BLOCKADE. (Abstract #373)**
Jennifer E. Woodward, Adam T. Schaefer, Alison J. Logar, Timothy Daskivich, Julie K. Stazer, Robert Peach, Abdul S. Rao. Pittsburgh, PA; Princeton, NJ.

P187 **TRANSCRIPTIONAL PROFILING OF PRIMATE RENAL TRANSPLANTS: MOTIFS DIFFERENTIATING REPERFUSION FROM ALLOSPECIFIC IMMUNITY. (Abstract #374)** ♦
Margot O'Toole, Holly Swiniarski, Sean P. Montgomery, Eric A. Elster, He Xu, Robert L. Kampen, Andrew J. Dorner, Allan D. Kirk. Andover, MA; Bethesda, MD.

P188 **INDIRECT PRESENTATION OF DONOR-DERIVED ANTIGENS BY DENDRITIC CELLS. (Abstract #375)**
Anat R. Tambur, Nancy Delgado, Eytan Mor. Chicago, IL; Petach-Tikva, Israel.

P189 RESISTANCE TO FAS AND TRAIL-MEDIATED APOPTOSIS IN EBV+ B CELL LYMPHOMAS FROM PATIENTS WITH PTLD. (Abstract #376)
Andrew L. Snow, Linda J. Chen, Ronald R. Nepomuceno, Sheri M. Krams, Olivia M. Martinez. Stanford, CA.

P190 EVIDENCE FOR CELLULAR SENESCENCE IN TUBULAR EPITHELIUM OF RODENT AND HUMAN KIDNEYS. (Abstract #377)
Anette Melk, Kieran Halloran, Irwindeep Sandhu, Sita Gourishankar, David Rayner, Lisa Helms, Philip F. Halloran. Edmonton, AB, Canada; Edmonton, AB, Canada.

P191 PRETRANSPLANT DONOR-SPECIFIC HELPER T-CELL REACTIVITY AS A TOOL FOR TAILORING THE INDIVIDUAL NEED FOR IMMUNOSUPPRESSION. (Abstract #378)
Barbara J. van der Mast, Nicole M. van Besouw, Petronella de Kuiper, Lenard M.B. Vaessen, Peter J.H. Smak Gregoor, Jan N.M. IJzermans, Teun van Gelder, Willem Weimar. Rotterdam, The Netherlands; Rotterdam, The Netherlands.

P192 BLOCKADE OF IL-2 RECEPTOR ABROGATES CO-STIMULATION BLOCKADE RESISTANT REJECTION. (Abstract #379)
Jongwon Ha, Thomas R. Jones, Andrew B. Adams, Megan M. Durham, Matthew A. Williams, Phyllis A. Rees, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

P193 TRANSCRIPTIONAL PROFILING OF ALLOGRAFT REJECTION. (Abstract #380)
Scott Damrauer, Thomas Mueller, David Perkins. Boston, MA.

P194 HIERARCHICAL CLUSTER ANALYSIS OF GENE EXPRESSION DURING ALLOGRAFT REJECTION. (Abstract #381)
Thomas Mueller, David Perkins. Boston, MA.

P195 DIRECT ANTIGEN PRESENTATION BY MATURE AND IMMATURE HUMAN ALLOGENEIC DENDRITIC CELLS. (Abstract #382)
Debra A. MacKenzie, Hans W. Sollinger, Debra A. Hullett. Madison, WI.

Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines I

P196 CYTOKINE PRODUCTION AND Th-CELL DRIVING CAPACITY OF MYELOID DENDRITIC CELLS AT DIFFERENT STAGES OF DIFFERENTIATION, AND FOLLOWING LPS STIMULATION OR CD40 LIGATION. (Abstract #383)
Adrian E. Morelli, Alan F. Zahorchak, Adriana T. Larregina, Bridget L. Colvin, Alison J. Logar, Takuya Takayama, Louis D. Falo, Angus W. Thomson. Pittsburgh, PA.

P197 IMMUNE ACTIVATION AND INFILTRATE APOPTOSIS, NOT IMMUNE DEVIATION, IS ASSOCIATED WITH LONG-TERM RAT RENAL ALLOGRAFT TOLERANCE INDUCED BY POST-TRANSPLANT ADMINISTRATION OF DONOR LEUKOCYTES. (Abstract #384)
Yiqun Yan, Suma Shastry, Chuanmin Wang, Geoffrey McCaughan, Craig Richards, Alex Bishop. Sydney, NSW, Australia; Sydney, NSW, Australia.

P198 CYTOKINE GENOTYPING, DONOR-SPECIFIC ANTIBODIES AND ACUTE REJECTION IN RENAL TRANSPLANTATION. (Abstract #385)
Angelica Canossi, Daniela Piancatelli, Anna Aureli, Marilena Di Rocco, Antonina Piazza, Tiziana Del Beato, Gabriella Liberatore, Carlo Umberto Casciani, Domenico Adorno. Sections of L'Aquila and Rome, Italy.

P199 MHC CLASS II DERIVED PEPTIDES PREVENT PRIMING TO THE ALLOIMMUNE RESPONSE IN VIVO. (Abstract #386)
Qinsheng Jiao, Joyce Yu, Charles B. Carpenter, Mohamed H. Sayegh, Barbara Murphy. New York, NY; Boston, MA.

P200 COMPARATIVE ANALYSIS OF MIGRATORY RESPONSES OF LYMPHOID AND MYELOID DENDRITIC CELLS TO CC CHEMOKINES AND IN ALLOGENEIC RECIPIENTS. (Abstract #387) ♦
Bridget L. Colvin, Adrian E. Morelli, Angus W. Thomson. Pittsburgh, PA; Pittsburgh, PA.

P201 GENERATION OF REGULATORY T CELLS BY EX-VIVO ALLOEDUCATION. (Abstract #388)
C. G. Orosz, A. A. Bickerstaff. Columbus, OH.

P202 CHEMOATTRACTION OF T CELLS EXPRESSING CCR5 AND CXCR3 BY PROXIMAL TUBULAR EPITHELIAL CELL CHEMOKINES. (Abstract #389)
Paul Cockwell, Christopher J. Brooks, Caroline O.S. Savage. Birmingham, United Kingdom.

P203 SELECTIVE OVER-EXPRESSION OF INFLAMMATORY MOLECULES IN HEARTS FROM BRAIN-DEAD RATS MAINTAINED WITH ADEQUATE CIRCULATION. (Abstract #390) ♦
Leigh D. Segel, Derek W. von Haag, Jie Zhang, David M. Follette. Davis and Sacramento, CA.

P204 DIFFERENTIAL INFLAMMATORY AND CHEMOKINE RESPONSES BY THE INNATE AND ADAPTIVE IMMUNE RESPONSES AFTER TRANSPLANTATION. (Abstract #391)
Thomas Mueller, Scott Damrauer, Michael Buckley, James Stone, David Perkins. Boston, MA; Boston, MA.

P205 ROBUST CHEMOKINE AND INFLAMMATORY RESPONSES BY THE INNATE IMMUNE RESPONSE AFTER TRANSPLANTATION. (Abstract #392)
Scott Damrauer, Thomas Mueller, Michael Buckley, James Stone, David Perkins. Boston, MA; Boston, MA.

P206 IN-VIVO TRACKING OF ACTIVATED T CELLS IN ADOPTIVELY TRANSFERRED MICE TREATED WITH NON-DEPLETING ANTI-CD4 AND ANTI-CD154 MONOCLONAL ANTIBODIES ALONE OR IN COMBINATION. (Abstract #393)
Martin Wijkstrom, Michelle Lucido, B. J. Hering, Elizabeth Ingulli. Minneapolis, MN; Minneapolis, MN.

P207 EXPRESSION OF T-CELL ATTRACTING CHEMOKINES I-TAC, IP-10 AND MIG IN MURINE CARDIAC ALLOGRAFTS IS NOT INFLUENCED BY CYCLOSPORIN-A TREATMENT. (Abstract #394)
Gerald Brandacher, Robert Öllinger, Wolfgang Steurer, Raimund Margreiter, Ernst R. Werner, Gabriele Werner-Felmayer. Innsbruck, Austria; Innsbruck, Austria.

P208 DOWNREGULATION OF ALLOIMMUNE RESPONSE BY ANTIGEN-SPECIFIC REGULATORY CD4+ T CELL LINE INDUCED BY ALLOMHC CLASS I PEPTIDE. (Abstract #395)
Hector A. DePaz, Olakunle O. Oluwole, Zhuoru Liu, Kris Engelstad, Ming X. Jin, Mark A. Hardy, Soji F. Oluwole. New York, NY.

Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH I

P209 HIGH DOSE THYMOGLOBULIN WITHOUT RADIATION RESULTS IN LONGTERM MIXED CHIMERISM IN MHC-MISMATCHED MONKEYS. (Abstract #396)
A. Bartholomew, C. Sturgeon, M. Siatskas, V. Vidanovic, S. Sosler, K. Ferrer, S. Devine, R. Buffet, J. Tollier, R. Buelow, R. Hoffman. Chicago, IL; Menlo Park, CA.

P210 THE ABSENCE OF GRAFT-VERSUS-HOST DISEASE AFTER DELAYED LEUCOCYTE INJECTION EARLY (3 WEEKS) AND LATE (12 WEEKS) AFTER BONE MARROW TRANSPLANTATION IS BASED ON DIFFERENT IMMUNEREGULATORY MECHANISMS. (Abstract #397)
An D. Billiau, Sabine Fevery, Lutgart Overbergh, Omer Rutgeerts, Jozef Goebels, Chantal Mathieu, Mark Waer. Leuven, Belgium; Leuven, Belgium.

P211 **ADENOVIRAL CO-DELIVERY OF CTLA4-IG AND CD40-IG INTO DONOR LIVER PROMOTES PERMANENT GRAFT ACCEPTANCE L OF ALLOGENIC RECIPIENTS. (Abstract #398)**
Andrea Gambotto, Toshio Miki, Wentao Gao, Yoshihito Takahashi, David A. Geller, Paul D. Robbins, Noriko Murase. Pittsburgh, PA; Pittsburgh, PA.

P212 **ASSOCIATION OF FACTOR 5 LEIDEN MUTATION WITH DELAYED GRAFT FUNCTION, ACUTE REJECTION EPISODES AND LONG-TERM GRAFT DYSFUNCTION IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #399)**
Berthold Hocher, Torsten Slowinski, Ingeborg Hauser, Lutz Fritsche, Klemens Budde, Andreas Kulozik, Hans-H. Neumayer. Berlin, Berlin, Germany; Frankfurt, Hessen, Germany; Berlin, Berlin, Germany.

P213 **MIXED HEMATOPOIETIC CHIMERISM WITHOUT MYELOSUPPRESSION IN A LARGE ANIMAL MODEL: EVIDENCE FOR MULTILINEAGE PROGENITOR CELL ENGRAFTMENT IN LONG-TERM LYMPHOID CHIMERAS. (Abstract #400)**
Brian Lima, Zachary L. Gleit, Sharon Germana, Christian LeGuern, Michael C. Murphy, Qing Chang, Julian Down, David H. Sachs, Christene A. Huang. Boston, MA; Boston, MA.

P214 **IMMUNOSUPPRESSION THROUGH BLOCKADE OF TRANSFERRRIN RECEPTOR (TfR). (Abstract #401)**
Xiaoming Li, Mark A. Braun, Melissa L. Brown, Pilar Mercado, Luca Cicalese, Enrico Benedetti, Cristiana Rastellini. Chicago, IL.

P215 **EVIDENCE IN SUPPORT OF A TROPHIC MECHANISM FOR FC:HSC INTERACTION. (Abstract #402)**
Daniel Cramer, Michael K. Tanner, Carrie L. Schanie, Yimming Haung, Suzanne T. Ildstad. Louisville, KY.

P216 **CONSTRUCTION OF A RECOMBINANT ADENOVIRUS THAT EXPRESSES A SOLUBLE FORM OF CR1 FOR USE IN XENOTRANSPLANTATION. (Abstract #403)**
E. Gamerl, N. R. Thummala, S. W. Lee, M. Krych-Goldberg, R. E. Hauhart, J. Hammel, S. S. Ghosh, J. P. Atkinson, J. Roy Chowdhury, I. J. Fox.

P217 **COMPARATIVE STUDIES OF ISOLATED ISLET TRANSPLANTATION (IIT) IN 2 SEPARATE NON HUMAN PRIMATE (NHP) MODELS. (Abstract #404)**
Francis Thomas, Juan Contreras, Devin Eckhoff, Cheryl Smith, Judith Thomas.

P218 **ALLOANTIBODY AS AN EFFECTOR MECHANISM FOR CD4+ T CELL INITIATED HEPATOCYTE REJECTION. (Abstract #405)**
Philomena Salvemini, Donghong Gao, Charles G. Orosz, Ginny L. Bumgardner. Columbus, OH.

P219 **DENDRITIC CELLS TRANSDUCED WITH ADENOVIRAL IL-10 INHIBIT THE ALLOIMMUNE RESPONSE *IN VITRO* BUT DO NOT PROLONG KIDNEY ALLOGRAFT SURVIVAL IN SHEEP. (Abstract #406)**
Toby Coates, Ravi Krishnan, Mark Siddins, Bu Lang He, Mohan M. Rao, Svjetlana Kireta, Ghee C. Chew, Graeme R. Russ. Woodville, Australia; Woodville, Australia.

Tissue Injury, Preservation I

P220 **INHIBITION OF COLD INDUCED APOPTOSIS IN ENDOTHELIAL CELLS BY BETA 1 AGONISTS: A NEW PARADIGM IN ORGAN PRESERVATION. (Abstract #407)**
Benito A. Yard, Grietje Beck, Yang Xiao, Fokko J. van der Woude.

P221 **LOCALIZATION AND KINETICS OF INTERLEUKIN-6 (IL-6) DURING ISCHEMIA-REPERFUSION INJURY (IRI) OF PORCINE INTESTINE. (Abstract #408)♦**
Felix Braun, Mehdi Hosseini, Sven Laabs, Burckhard Sattler, Eberhard Wieland, Burckhardt Ringe. Göttingen, Germany; Göttingen, Germany; Göttingen, Germany.

P222 **EXTENDED PRESERVATION AND VIABILITY ASSESSMENT OF DONOR LIVERS UTILISING NORMOTHERMIC EXTRACORPOREAL PERFUSION. (Abstract #409)**
Charles J. Imber, Shawn D. St.Peter, Inigo Lopez, David W. Pigott, David A. Hughes, James Mcguire, Peter J. Friend. Oxford, United Kingdom.

P223 **INDUCTION OF HEME OXYGENASE-1 IMPROVES RAT LIVER TRANSPLANTATION SURVIVAL BY INHIBITING APOPTOSIS. (Abstract #410)♦**
Claudio A. Redaelli, Ying-Hua Tian, Martin K. Schilling, Jean-Francois Dufour. Bern, Switzerland; Bern, Switzerland.

P224 **ESTROGENS(E)INHIBITS ISCHEMIA/REPERFUSION INJURY (I/R I) OF THE LIVER. (Abstract #411)**
Juan L. Contreras, Guadalupe Bilbao, Luc Frenette, Devin E. Eckhoff. Birmingham, AL; Birmingham, AL.

P225 **SHORT TERM CERIVASTATIN DRASTICALLY REDUCES ISCHEMIA/REPERFUSION INJURY IN RATS. (Abstract #412)**
Faikah Abou-Rebyeh, Song Rong, Anette Fiebeler, Joon-Keun Park, Dominik Müller, Franziska Hampich, Ralf Dechend, Marlies Elger, Hermann Haller. Hannover, Germany; Berlin, Germany.

P226 **CASPASE 6 INHIBITOR (VEID) PROLONGS SURVIVAL OF RHESUS MACAQUE ISOLATED ISLETS (II). (Abstract #413)**
Jianguo Wu, Peter Ray, Jin He, Cheryl Smith, Judith Thomas, Francis Thomas.

P227 **ISCHEMIC PRECONDITIONING ENHANCES REGENERATIVE CAPACITY OF HEPATOCYTES AFTER PROLONGED ISCHEMIA. (Abstract #414)**
Fumihiko Yamada, Tsuyoshi Abe, Takuro Saito, Takao Tuschiya, Akira Kenjo, Mitsukazu Gotoh. Fukushima, Fukushima, Japan.

P228 **PROTECTIVE EFFECT OF A SELECTIVE PROSTAGLANDIN E1 RECEPTOR AGONIST AGAINST WARM ISCHEMIA AND REPERFUSION INJURY IN RAT KIDNEY. (Abstract #415)**
Fusako Toda, Kazunari Tanabe, Marcia Manu, Tadahiko Tokumoto, Hiroshi Toma.

P229 **BLOCKADE OF THE L-ARGININE-NITRIC OXIDE SYNTHASE PATHWAY WORSENS HEPATIC NECROSIS AND APOPTOSIS IN THE LIVER TRANSPLANT SETTING. (Abstract #416)**
Gautam P. Yagnik, Yoshihito Takahashi, George Tsoulfas, Noriko Murase, David A. Geller. Pittsburgh, PA.

Xenotransplantation I

P230 **SIMPLE BARIUM ALGINATE CAPSULES PROTECT NEONATAL PORCINE ISLET CELLS IN DIABETIC MICE OVER 20 WEEKS. (Abstract #417)**
Abdulkadir Omer, Valerie Duvivier-Kali, Nitin Trivedi, Susan Bonner-Weir, Gordon C. Weir. Boston, MA.

P231 **GENERATION OF TRANSGENIC MICE EXPRESSING AN ANTI-GAL ANTIBODY TRANSGENE. (Abstract #418)**
Ajay Sharma, Jeannine Okabe, Hui Xu, Sharon Wannberg, Hua Wan, Ying Lei, John S. Logan, Guerard W. Byrne.

P232 **ENDOTHELIAL CELL EXPRESSION OF HUMAN FUCOSYLTRANSFERASE IN TRANSGENIC MICE USING THE PUTATIVE HUMAN MCP PROMOTER. (Abstract #419)**
Ajay Sharma, Deling Tao, Hua Wan, John S. Logan, Guerard W. Byrne.

P233 **RESISTANCE OF HEPATOCYTE FUNCTIONS TO PERTURBATION BY COMPLEMENT. (Abstract #420)**
Akiyoshi Kanazawa, Cody A. Koch, Zoie E. Holzknecht, Jeffery L. Platt. Rochester, MN; Rochester, MN; Rochester, MN.

P234 **A GENETICALLY MODIFIED HLA CLASS I MOLECULE ABLE TO INHIBIT NK CELLS WITHOUT STIMULATING ALLOREACTIVE CD8+ T LYMPHOCYTES. (Abstract #421)**
Alexandra Sharland, Amy Patel, Josie Han Lee, Aimee E. Cestra, Gerald L. Waneck. Boston, MA.

P235 **EFFECTIVE REMOVAL OF CIRCULATING ANTI-αGAL ANTIBODIES BY THE INJECTABLE POLYMER GAS914. (Abstract #422)**
Andreas G. Katopodis, Richard Warner, Rudolf O. Duthaler, Markus Streiff, Armin Bruelisauer, Hugh Davies, Emanuele Cozzi, Gilda Chavez, Christian Bruns, Gebhard Thoma, Willy Kinzy, Reinhold Oehrlein. Basel, Switzerland; Cambridge, United Kingdom.

P236 **CYCLOPHOSPHAMIDE INDUCED ANAEMIA AS A RESTRICTIVE FACTOR FOR LONG TERM SURVIVAL IN DISCORDANT KIDNEY XENOTRANSPLANTATION FROM PIGS TO CYNOMOLGUS MONKEYS. (Abstract #423)**
Arman Jalali, Martin Loss, Ralf Lorenz, Jens M. Hecker, Richard Appiah, S. Piepenbrock, Juergen Klempnauer, Michael Winkler. Hannover, Germany; Hannover, Germany.

P237 **ANTI-GAL ANTIBODIES INDUCE EXPRESSION OF A Fc GAMMA RECEPTOR ON PORCINE ENDOTHELIAL CELLS. (Abstract #424)**
Bashoo Naziruddin, Ajay Sharma, Jessie Yin, Hui Xu, Guerard W. Byrne. Princeton, NJ.

P238 **THE NEUTROPHIL: AN UNSEEN THREAT IN XENOTRANSPLANTATION? (Abstract #425)**
D. B. Rouw, M. Midulla, M. F. Morgan, A. N. Warrens. London, United Kingdom.

P239 **ROLE OF CD4+ AND CD8+ T CELLS IN THE REJECTION OF CONCORDANT PANCREAS XENOGRAFTS. (Abstract #426)**
Deng Ping Yin, Howard N. Sankary, LianLi Ma, Jikun Shen, Lei Zhang, James W. Williams, Anita S. Chong. Chicago, IL.

P240 **FcγRs POLYMORPHISMS PREDICT THE ADCC LEVELS AGAINST PIG AORTIC ENDOTHELIAL CELLS (PAEC). (Abstract #427)**
Diana M. Metes, Penelope A. Morel, Joseph Nellis, Abdul S. Rao. Pittsburgh, PA; Pittsburgh, PA.

TRANSPLANT 2001
The Joint American Transplant Meeting
Day-at-a-Glance, Monday, May 14, 2001

6:30 AM - 7:50 AM — **Concurrent Sunrise Symposia**

Page 77 — **Sunrise Symposium I: New Technologies in Transplantation Immunobiology**
Sheraton Chicago Ballroom 4-7, Sheraton

Page 77 — **Sunrise Symposium II: Video Session II: Techniques in Expanding the Liver Donor Pool**
Chicago Ballroom 8-10, Sheraton

Page 77 — **Sunrise Symposium III: Recurrent Disease after Pediatric Transplantation**
Sheraton Ballroom 1-3, Sheraton

8:00 AM - 9:30 AM — **Concurrent Symposia**

Page 77 — **Basic Science Symposium: Innate Immunity**
Sheraton Ballroom 1-3, Sheraton

Page 77 — **Clinical Science Symposium: Clinical Practice Guidelines: The Prevention of Medical Complications after Renal Transplantation**
Sheraton Chicago Ballroom 4-7, Sheraton

10:00 AM - 12:00 PM — **Joint Session**

10:00 AM
Page 77 — **State-of-the-Art Lecture**
Potential Applications of Cloning for Transplantation
Sheraton Chicago Ballroom 4-7, Sheraton

10:30 AM
Page 77 — **Joint Plenary Session**
Sheraton Chicago Ballroom 4-7, Sheraton

11:30 AM
Page 77 — **Award Presentations**
Sheraton Chicago Ballroom 4-7, Sheraton

12:30 PM - 1:30 PM
Page78 — **Parallel Luncheon Workshops**
Sheraton and Intercontinental

12:30 PM - 1:30 PM
Page 78 — **Selected Poster Sessions**
Sheraton

2:00 PM - 3:30 PM — **Concurrent Sessions**

Page 79 — **Concurrent Session 21: Basic Science: Rejection III**
Chicago Ballroom 10, Sheraton

Page 80 — **Concurrent Session 22: Kidney Transplantation: Minimizing Immunosuppression**
Chicago Ballroom 6/7, Sheraton

Page 81 — **Concurrent Session 23: Kidney Transplantation: Live-Donors, Factors and Outcomes**
Chicago Ballroom 8/9, Sheraton

Page 81 — **Concurrent Session 24: Basic Science: Immunosuppression II**
Sheraton Ballroom 1-3, Sheraton

Page 82 — **Concurrent Session 25: Mechanisms of Acute/ Chronic Rejection**
Sheraton Ballroom 4/5, Sheraton

Page 82 — **Concurrent Session 26: T Cells in Ischemia/ Reperfusion Injury**
Empire Room, Intercontinental

Page 83 — **Concurrent Session 27: Cardiac Allograft Rejection: Cell Death and Molecular Markers**
Exchange Room, Intercontinental

Page 83 — **Concurrent Session 28: Living Liver Donors I**
Grand Ballroom, Intercontinental

Page 84 — **Concurrent Session 29: Basic Science: Co-Stimulation**
King Arthur Court Ballroom, Intercontinental

Page 84 — **Concurrent Session 30: Liver Transplantation Outcomes**
Renaissance Ballroom, Intercontinental

4:00 PM - 5:30 PM — **Concurrent Sessions**

Page 85 — **Concurrent Session 31: Regulation of Allreactive T Cell Responses**
Chicago Ballroom 10, Sheraton

Page 85 — **Concurrent Session 32: Viral Infections in Renal Transplantation**
Chicago Ballroom 6/7, Sheraton

Page 86 — **Concurrent Session 33: Kidney Transplantation: Immunotherapy, Economics and Efficacy**
Chicago Ballroom 8/9, Sheraton

Page 86 — **Concurrent Session 34: Basic Science: Tolerance I**
Sheraton Ballroom 1-3, Sheraton

Page 87 — **Concurrent Session 35: Antibodies and Immunomodulation**
Sheraton Ballroom 4/5, Sheraton

Page 87 — **Concurrent Session 36: Bone Marrow Cell Transplantation**
Empire Room, Intercontinental

Page 88 — **Concurrent Session 37: Cardiac Transplantation Complications: Diagnosis & Treatment**
Exchange Room, Intercontinental

Page 88 — **Concurrent Session 38: Liver Transplantation: Allocation**
Grand Ballroom, Intercontinental

Page 89 — **Concurrent Session 39: Pathology, Techniques and Results of Pancreas Transplantation**
King Arthur Court Ballroom, Intercontinental

Page 89 — **Concurrent Session 40: Pediatrics I (Liver)**
Renaissance Ballroom, Intercontinental

8:00 AM - 7:00 PM	**Poster Session II**
5:30 PM -7:00 PM	**Presenters in Attendance** *Beer and Pretzel Reception* *River Exhibition Hall, Sheraton*
Page 90	**Kidney - Acute/Chronic Rejection II**
Page 90	**Kidney - GVH, Complications, Infections II**
Page 91	**Kidney - Immunosuppression B II**
Page 91	**Kidney - Immunosuppression A II**
Page 92	**Kidney - Pediatrics, Recurrent Disease II**
Page 92	**Kidney - Preservation, Donation/Allocation, Economics/Public Policy, Surgical Techniques, and Other II**
Page 93	**Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics**
Page 94	**Liver - Infections, Complications, Recurrent Disease, Surgical Techniques II**
Page 95	**Liver - Preservation, Economics/Public Policy, Donation Allocation, Other II**
Page 95	**Pancreas and Islets - All Topics II**
Page 96	**Heart/Lung - All Topics II**
Page 96	**Bone Marrow - All Topics II**
Page 97	**Immunosuppression, Preclinical Studies II**
Page 97	**Tolerance II**
Page 98	**Acute/Chronic Rejection II**
Page 99	**Allorecognition, Antigen Presentation, Co-stimulation and Other II**
Page 99	**Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines, Adhesion Molcules and Cytokines II**
Page 100	**Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH II**
Page 100	**Tissue Injury, Preservation II**
Page 101	**Xenotransplantation II**
5:45 PM	**ASTS Business Meeting**
Page 89	*Sheraton Ballroom 1-3, Sheraton*

Monday, May 14, 2001

Concurrent Sunrise Symposium
6:30 AM - 7:45 AM

Sunrise Symposium I: New Technologies in Transplantation Immunology

Sheraton Chicago Ballroom 4-7, Sheraton
Chair: Abraham Shaked

6:30 AM **The latest vectors for gene therapy**
Abraham Shaked

6:55 AM **Bioinformatics**
Isaac Kohane

7:20 AM **Analyzing the T cell repertoire by landscape spectratyping**
Jean-Paul Soulillou

Sunrise Symposium II: Video Session II: Techniques in Expanding the Liver Donor Pool

Chicago Ballroom 8-10, Sheraton
Chair: Thomas Heffron

6:30 AM **Current refinements in living donor left lateral segmentectomy**
Thomas Heffron

6:55 AM **Living donor right lobectomy**
Charles Miller

7:20 AM **In-situ cadaver donor split liver procurement**
Hasan Yersiz

Sunrise Symposium III: Recurrent Disease after Pediatric Transplantation

Sheraton Ballroom 1-3, Sheraton
Chair: Steven Webber

6:30 AM **Genetics of FSGS**
Martin Pollak

6:45 AM **Recurrent glomerular disease**
Mark Denton

7:00 AM **Recurrent liver disease in children**
Sue McDiarmid

7:15 AM **Recurrent disease after heart and lung transplantation**
Steven Webber

Concurrent Symposia
8:00 AM - 9:30 AM

Basic Science Symposium: Innate Immunity

Sheraton Ballroom 1-3, Sheraton
Chairs: Angus Thomson and David Perkins

8:00 AM **Innate immunity: From plants to humans**
Albert Bendelac

8:30 AM **CD14-dependent clearance of apoptotic cells - A link between an innate and adaptive immunity**
Christopher Gregory

9:30 AM **Toll-like receptors and regulation of adaptive immune responses**
Rusian Medzhitov

Clinical Science Symposium: Clinical Practice Guidelines: The Prevention of Medical Complications after Renal Transplantation

Chairs: Bertram Kasiske and Gabriel Danovitch

8:00 AM **Post-transplant follow-up: How frequent, how thorough and by whom?**
Gabriel Donovitch

8:15 AM **Evidence that post-transplant cardiovascular disease can be prevented**
Bertram Kasiske

9:00 AM **How should post-transplant bone disease be diagnosed and treated?**
Margaret Bia

9:15 AM **Cancer: Post-transplant screening and prevention**
Connie Davis

9:30 AM **Break**

Joint Plenary Session
10:00 AM - 12:00 PM

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: Mohamed Sayegh and Nancy Ascher

10:00 AM **State-of-the-Art Lecture**
Potential Applications of Cloning for Transplantation
Alan Colman

10:30 AM **SIROLIMUS PREVENTS TUMOR PROGRESSION: mTOR TARGETING FOR THE INHIBITION OF NEOPLASTIC PROGRESSION. (Abstract #428)** *Young Investigators Award*
Fulung Luan, Mary Maluccio, Vijay K. Sharma, Minoru Hojo, Milagros Lagman, Manikkam Suthanthiran. New York, NY; New York, NY.

10:45 AM **FTY720 COMBINED WITH NEORAL® AND CORTICOSTEROIDS IS EFFECTIVE AND SAFE IN PREVENTION OF ACUTE REJECTION IN RENAL ALLOGRAFT RECIPIENTS (INTERIM DATA). (Abstract #429)**
Helio Tedesco, Barry Kahan, Georges Mourad, Yves Vanrenterghem, Josep Grinyo, Willem Weimar, Pascale Pellet, Lawrence Chodoff, Tomasz Sablinski. Sao Paolo, Brazil; Houston; Montpellier, France; Leuven, Belgium; Barcelona, Spain; Rotterdam, The Netherlands; Basel, Switzerland; East Hanover.

11:00 AM **ICOS/B7RP-1 COSTIMULATION IN ACUTE AND CHRONIC ALLOGRAFT REJECTION. (Abstract #430)** *Young Investigators Award*
Engin Ozkaynak, Wei Gao, Nida Shemmeri, Chi Wang, Anthony J. Coyle, Wayne W. Hancock. Cambridge, MA.

11:15 AM **TWO-YEAR INSULIN INDEPENDENCE AND METABOLIC FOLLOW-UP AFTER ISLET-ALONE TRANSPLANTATION IN AUTOIMMUNE DIABETES. (Abstract #431)**
A. M.J. Shapiro, E. A. Ryan, R. V. Rajotte, G. S. Korbutt, T. Kin, K. O'Kelly, G. L. Warnock, D. L. Bigam, N. M. Kneteman, J. R.T. Lakey. Edmonton, AB, Canada.

11:30 AM **Award Presentations**

Parallel Luncheon Workshops

12:30 PM - 1:30 PM

Room locations not available at time of publication. Locations will be printed on the tickets and in the mini-program. Be sure to check hotel location.

24. How to use sirolimus in the clinic
Alan McDonald and Marc Lorber
25. Managing the highly sensitized patient
Manuel Pascual and Robert Bray
26. What you need to know about UNOS
Lawrence Hunsicker and Pat Adams
27. Islet transplantation
Bernhard Hering and Paul Gores
28. Complications of liver
Robert Brown Jr. and Micheal Lucey
29. Pathologic classification of rejection
Kim Solez and Robert Colvin
30. NIH initiatives
Stephen Rose, Larry Agadoa and Judith Massicot-Fisher
31. Expanding the living donor pool - ethical issues
Francis Delmonico and J. Richard Thistlethwaite, Jr.
32. Graft vs. host disease vs. graft vs. leukemia
Megan Sykes and Samuel Strober
33. How to work with the FDA
Amy Rosenberg and Phillip Noguchi
34. Live donor lung
Mark Barr and Marshal Hertz
35. New pathogens in transplant patients
Emilio Ramos and Jay Fishman
36. Protocol biopsies in kidney transplantation
David Rush and Shane Meehan
37. Solid organ transplantation in the HIV positive recipient
Peter Stock and Paul Kuo
38. Alternative costimulatory pathways
Diedier Mandelbrot and Kenneth Newell
39. The endothelium and the immune response to an allograft
David Briscoe and Jordan Pober
40. Gene expression to diagnose AR
Mannikam Suthanthiran and Martha Pavlakis
41. T cell signaling: What's new?
Majed M. Hamawy and David Rothstein
42. How to write a grant?
Hugh Auchincloss and Ali Naji
43. Minimizing immunosuppression
Thomas Gonwa and Gregory Everson
44. Women's career development
Olivia Martinez and Dianne McKay
45. Management of the patient on the kidney waiting list
Gabriel Danovitch and Steven Tomlanovich

Selected Poster Open Sessions

12:30 PM - 1:30 PM

Selected Posters on Heart and Lung

Chicago Ballroom 10, Sheraton
Chairs: Edward Garrity and Jon Kobashigawa

12:30 PM **OUTCOMES OF PREGNANCIES IN FEMALE HEART TRANSPLANT RECIPIENTS. (Abstract #1177)**
Scott W. Cowan, Lisa A. Coscia, Carolyn H. McGrory, Lydia Z. Philips, Michael J. Moritz, Vincent T. Armenti. Philadelphia, PA.

12:35 PM **HLA-CYTOMEGALOVIRUS (CMV) INTERACTION IN RELATION TO REJECTION AND VASCULOPATHY FOLLOWING HUMAN HEART TRANSPLANTATION. (Abstract #1172)**
Mohamad H. Yamani, Ashraf Abdo, James B. Young, Randall C. Starling, Norman B. Ratliff, Robin Avery, Murat Tuzcu, Patrick McCarthy, Daniel Cook. Cleveland, OH.

12:40 PM **EXERCISE TOLERANCE AFTER HEART TRANSPLANTATION: INFLUENCE OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC FUNCTION. (Abstract #1170)**
M. Dandel, R. Ewert, M. Hummel, J. Müller, R. Meyer, R. Hetzer. Berlin, Germany.

12:45 PM **DO BLACK HEART TRANSPLANT RECIPIENTS TREATED WITH TACROLIMUS OR CYCLOSPORINE IMMUNOSUPPRESSION DEMONSTRATE DISPARATE METABOLIC OUTCOMES? (Abstract #1166)**
Patricia A. Uber, Mandeep R. Mehra, N. Vivekananthan, Robert L. Scott, Myung H. Park. New Orleans, LA.

12:50 PM **ADVANCED RECIPIENT AGE AS A RISK FACTOR FOR MORBIDITY FOLLOWING CARDIAC TRANSPLANTATION: DOES SINGLE CENTER EXPERIENCE CORRELATE WITH MULTICENTER REGISTRIES? (Abstract #754)**
Katherine J. Hoercher, Patrick M. McCarthy, Michael K. Banbury, James B. Young, Randall C. Starling.

12:55 PM **A LONGITUDINAL INVESTIGATION OF RETENTION OF PATIENT DIRECTED TRANSPLANT EDUCATION. (Abstract #757)**
Alice J. Bordelon, Patricia A. Uber, Debi Dumas-Hicks, Myung H. Park, Robert L. Scott, Mandeep R. Mehra. New Orleans, LA.

1:00 PM **SENSITIVITY OF THE FLOW CYTOMETRY CROSSMATCH IN HEART TRANSPLANTATION. (Abstract #319)**
Ashraf S. Abdo, Daniel J. Cook, James F. McCarthy, Ehab S. Bishay, Ganesh S. Kumpati, Randall C. Starling, James B. Young, Mohamad H. Yamani, Nicholas G. Smedira, Patrick M. McCarthy. Cleveland, OH.

1:05 PM **TACROLIMUS/MYCOPHENOLATE MOFETIL VS CYCLOSPORINE/MYCOPHENOLATE MOFETIL AFTER HTX: IMAPACT ON CORONARY VASOMOTOR FUNCTION AND MYCOPHENOLATE ACID TROUGH LEVELS. (Abstract #321)**
Bruno M. Meiser, Jan Groetzner, Johannes Schirmer, Soeren Schenk, Wolfgang von Scheidt, Michael Weis, Volker Klauss, Hans U. Stempfle, Hermann Reichenspurner, Bruno Reichart. Munich, Germany; Munich, Germany; Munich, Germany.

Selected Posters on Immunosuppression

Chicago Ballroom 8-9, Sheraton
Chairs: Randall Morris and Flavio Vincenti

12:30 PM **EFFECT OF CONCOMITANT TACROLIMUS AND RAPAMYCIN THERAPY ON IMMUNE FUNCTION IN RENAL TRANSPLANT RECIPIENTS. (Abstract #213)**
Ashwani K. Khanna, Matthew S. Plummer, Cathy M. Bromberek, Sundaram Hariharan. Milwaukee, WI; Milwaukee, WI; Milwaukee, WI; Milwaukee, WI.

12:35 PM **CONVERSION FROM MYCOPHENOLATE TO RAPAMYCIN FOR ACUTE AND CHRONIC REJECTION. (Abstract #1069)**
Radha Vankawala, Ravi K. Kode, Anna M. Damask, Susan Stabler, Mark R. Laftavi, Mysore S. Anil Kumar, Oleh Pankewycz. Philadelphia, PA.

12:40 PM **PRELIMINARY RESULTS OF THE USE OF HUMANIZED ANTI-CD154 IN HUMAN RENAL ALLOTRANSPLANTATION. (Abstract #223)**
Allan D. Kirk, Stuart J. Knechtle, Hans W. Sollinger, Flavio G. Vincenti, Scott Stecher, Kari Nadeau. Bethesda, MD; Madison, WI; San Francisco, CA; Cambridge, MA.

12:45 PM **SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY IS PROMISING IN PATIENTS RECEIVING KIDNEYS FROM OLDER DONORS. (Abstract #652)**
Josep M. Grinyo, Yves Vanrenterghem, Brian Hutchinson, Marco Castagneto, Dominique Durand, Gian B. Sorba, Rainer Oberbauer, Julianna Mannion, the Tri-continental Renal Transplant Study Group. Barcelona, Spain.

12:50 PM **INCIDENCE OF BIOPSY PROVEN CHRONIC ALLOGRAFT NEPHROPATHY AFTER CORTICOSTEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS ON TACROLIMUS-BASED IMMUNSUPPRESSION. (Abstract #1067)**
Martin S. Zand, Urath Suresh, Jonathan Sosnov, Fadi Hijazi, Tibor Nadasdy, Mark Orloff, Richard Demme, Luis Mieles, Amadeo Marcos, Oscar Bronsther. Rochester, NY.

12:55 PM **CYP3A4♦1B AS A PHARMACOGENOMIC PREDICTOR OF LOW TACROLIMUS REQUIREMENTS IN LIVER TRANSPLANT PATIENTS. (Abstract #1115)**
Janet L. Karlix, Heather Myers, Son Nguyen, Clara Johary, Kareem Albekairy, Valerie Greene, Alan Hemming, Willem van der Werf, Alan Reed, Richard Howard. Gainesville, FL; Gaineville, FL.

1:00 PM **QUESTION OF STEROID WITHDRAWAL UNDER TACROLIMUS FOR PRIMARY BILIARY CIRRHOSIS (PBC), PRIMARY SCLEROSING CHOLAGITIS (PSC) AND AUTOIMMUNE HEPATITIS (AIH) AFTER LIVER TRANSPLANTATION AND LONG-TERM SURVIVAL. (Abstract #265)**
Ashok B. Jain, Randeep S. Kashyap, Santosh Potdar, John J. Fung. Pittsburgh, PA.

1:05 PM **SIROLIMUS IMMUNOSUPPRESSION FOR LIVER TRANSPLANTATION IMPROVES RENAL DYSFUNCTION. (Abstract #697)**
George J. Chang, Harish D. Mahanty, David Quan, Chris E. Freise, Peter G. Stock, Nancy L. Ascher, John P. Roberts, Ryutaro Hirose. San Francisco, CA; San Francisco, CA.

Selected Posters on Rejection/Immunosuppressive Therapy

Sheraton 1-3, Sheraton
Chairs: Peter Heeger and Angus Thomson

12:30 PM **PASSIVE TRANSFER OF NON-COMPLEMENT-ACTIVATING ALLOANTIBODIES AUGMENTS CARDIAC ALLOGRAFT REJECTION IN IgKO MICE. (Abstract #366)**
Barbara A. Wasowska, Zhiping Qian, Jodi Layton, Fred Sanfilippo, William M. Baldwin, III. Baltimore, MD.

12:35 PM **OX-40 LIGAND DEFICIENCY RESULTS IN DIMINISHED GRAFT ARTERIAL DISEASE IN CARDIAC ALLOGRAFTS. (Abstract #796)**
Jun-ichi Suzuki, Sarah E. Cole, Andy I. Chen, Arlene H. Sharpe, Peter Libby, Richard N. Mitchell. Boston, MA; Boston, MA.

12:40 PM **IILA-A2 TRANSGENIC C57BL/6 TRACHEA TRANSPLANTATION INTO SYNGENEIC C57BL/6 MICE RESULTS IN OBLITERATIVE AIRWAY DISEASE: ROLE FOR CD4 AND CD8 CELLS. (Abstract #1216)**
Toru Higuchi, Andres Jaramillo, Zahid Kaleem, T. Mohanakumar. St. Louis, MO.

12:45 PM **CD4+ T CELLS PARTICIPATE IN ACUTE RENAL FAILURE INDUCED BY ISCHEMIA/REPERFUSION INJURY. (Abstract #837)**
Helady Sanders, Niels O. Saraiva Camara, Marcelo Franco, Jose O. Medina Pestana, Irene L. Noronha, Alvaro Pacheco-Silva. Sao Paulo, SP, Brazil; Sao Paulo, SP, Brazil; Sao Paulo, SP, Brazil.

12:50 PM **METABOLIC ACTIVITY VERSES DEATH IN THE PATHOGENESIS OF ACUTE VASCULAR REJECTION. (Abstract #1276)**
Zoie E. Holzknecht, Karisha L. Kuypers, Josie M. Williams, Jeffrey L. Platt. Rochester, MN.

12:55 PM **COS BLOCKADE BY ANTI-CD40 AND ANTI-CD86 PREVENTS KIDNEY GRAFT REJECTION IN RHESUS MONKEYS. (Abstract #770)**
Krista G. Haanstra, Els Sick, Seema G. Ramdien-Murli, Jan Ringers, Mark de Boer, Louis Boon, Margreet Jonker. Rijswijk, The Netherlands; Amsterdam, The Netherlands; Leiden, The Netherlands.

1:00 PM **A NOVEL RETINOIC ACID RECEPTOR-ALPHA SELECTIVE AGONIST, ER-38925, SUCCESSFULLY PREVENTED ACUTE AND CHRONIC REJECTION OF MOUSE CARDIAC ALLOGRAFTS. (Abstract #350)**
Ken-ichiro Seino, Katashi Fukao, Hideki Taniguchi, Yasutsugu Takada, Kenji Yuzawa, Masaaki Otsuka, Toshihiko Yamauchi. Tsukuba, Ibaraki; Kawaguchi, Saitama, Japan; Tsukuba, Ibaraki, Japan.

1:05 PM **SUSTAINED REDUCTION OF ANTI-aGAL AND ANTI-PIG HEMOLYTIC ANTIBODIES IN VIVO WITH THE POLYMER GAS914. (Abstract #1268)**
Rafael Manez, Alberto Centeno, Eduardo Lopez-Pelaez, Nieves Domenech, Rudolph Duthaler, Andreas Katopodis. La Coruna, Spain; Basel, Switzerland.

Concurrent Session 21: Basic Science: Rejection III

2:00 PM - 3:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Wayne Hancock and Eugenia Fedoseyeva

2:00 PM **A NOVEL CD154 MONOCLONAL ANTIBODY IN ACUTE AND CHRONIC RAT VASCULARIZED CARDIAC ALLOGRAFT REJECTION. (Abstract #432)**
X. Yuan, V. M. Dong, A. J. Coito, A. M. Waaga, M. Lenhard, A. Chandraker, C. D. Benjamin, M. H. Sayegh. Boston, MA.

2:10 PM **ADENOVIRUS-MEDIATED EXPRESSION OF CD40IG ATTENUATES GRAFT ARTERIOSCLEROSIS. (Abstract #433)**
Patrick Mathieu, Cecile Guillot, Francoise Buzelin, Delphine Bouchet, Maria Castro, Pedro Lowenstein, Jean Paul Soulillou, Ignacio Anegon. Nantes, France; United Kingdom.

2:20 PM **MECHANISMS OF INHIBITION OF CHRONIC REJECTION BY ALLOCHIMERIC THERAPY. (Abstract #434)**
Letitia T. Bridges, Anna Coito, Xiu-da Shen, Feng Gao, Natalya V. Semiletova, Alice Zhao, Jerzy W. Kupiec-Weglinski, Ronald Busuttil, Rafik M. Ghobrial. Los Angeles, CA.

2:30 PM **THE PATHOGENIC ROLE OF NON-MHC ALLOANTIBODIES IN CHRONIC GRAFT REJECTION: I. THE KINETICS OF ANTI-DONOR ANTIBODY PRODUCTION AND ITS' CORRELATION WITH GRAFT REJECTION IN THE LEW-TO-F344 HEART TRANSPLANT MODEL. (Abstract #435)**
Gordon D. Wu, Yang-Sun Jin, Joyce Swensson, Robert Salazar, Vaughn A. Starnes, Donald V. Cramer. Los Angeles, CA.

2:40 PM **SERPIN REGULATION BY A MYXOMA VIRUS ENCODED IMMUNOREGULATORY PROTEIN PREVENTS CHRONIC REJECTION IN RAT RENAL ALLOGRAFTS. (Abstract #436)** *International Young Investigator Award*
Eric Bedard, Jifu Jiang, Haiyan Guan, Liying Li, Peter Kim, Bertha Garcia, Graeme Fraser, Xing Li, Grant McFadden, Alexandra Lucas, Robert Zhong. London, Canada; London, Canada.

2:50 PM **ORAL 1,25-$(OH)_2D_3$ MODULATES THE PROGRESSION OF CHRONIC ALLOGRAFT NEPHROPATHY BY ALTERING TGFβ-1 SIGNALING PROTEINS. (Abstract #437)**
Jacquelyn K. Aschenbrenner, Gretchen J. Malin, Paul F. Laeseke, Hans W. Sollinger, Bryan N. Becker, Debra A. Hullett. Madison, WI; Madison, WI.

3:00 PM **HEPARIN-BINDING EPIDERMAL GROWTH FACTOR-LIKE GROWTH FACTOR (HB-EGF) IS INVOLVED IN THE PATHOGENESIS OF CHRONIC ALLOGRAFT VASCULOPATHY. (Abstract #438)**
T. Koshiba, I. Kim, T. Tsuruyama, S. Higashiyama, K. Tanaka, B. Van Damme, M. Waer, J. Pirenne. Leuven, Belgium; Kyoto; Suita, Japan.

3:10 PM **IMMUNOTOXIN-TREATED RHESUS MONKEYS: A MODEL FOR RENAL ALLOGRAFT CHRONIC REJECTION. (Abstract #439)**
Jose R. Torrealba, Terry Oberly, Jacqueline M. Schultz, Kevin Brunner, David Peters, John H. Fechner, Yinchen Dong, Clifford S. Cho, Luis Fernandez, Stuart J. Knechtle. Madison, WI; Madison, WI.

3:20 PM **APPLICATION OF GENE EXPRESSION PATTERNS USING cDNA MICROARAYS IN A BABOON CHRONIC VASCULOPATHY MODEL. (Abstract #440)**
Einari Aavik, Ajit Mahapatra, Stella Chang, Jennifer Boldrick, Pekka Hayry, Minnie Sarwal. Helsinki, Finland; Stanford, CA; Stanford, CA.

Concurrent Session 22: Kidney Transplantation: Minimizing Immunosuppression

2:00 PM - 3:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: Mark Pescovitz and Lorenzo Gallon

2:00 PM **A PROSPECTIVE, RANDOMISED STUDY OF WITHDRAWAL OF CYCLOSPORINE OR PREDNISONE IN RENAL TRANSPLANT RECIPIENTS TREATED WITH MYCOPHENOLATE MOFETIL, CYCLOSPORINE, AND PREDNISONE: 18 MONTHS FOLLOW-UP DATA. (Abstract #441)** *International Young Investigator Award*
Peter J.H. Smak Gregoor, Ruud G.L. de Sevaux, Gerry Ligtenberg, Andries J. Hoitsma, Ronald J. Hene, Willem Weimar, Luuk B. Hilbrands, Teun van Gelder. Rotterdam, The Netherlands; Nijmegen, The Netherlands; Utrecht, The Netherlands.

2:10 PM **EFFECT OF REDUCING CYCLOSPORINE DOSE BY 50% IN STABLE RENAL TRANSPLANT RECIPIENTS IN THE MYCOPHENOLATE (MMF) ERA : A RANDOMIZED CONTROLLED TRIAL. (Abstract #442)**
John J. Curtis, Manual Pascual, Roberto Kalil, Nina Tokoff-Rubin, Francis Delmonico, Patsy Jones, Mary Lin Farrell, A. B. Cosimi. Birmingham, AL; Boston, MA.

2:20 PM **EFFICACY AND SAFETY OF THREE MONTHS OF TACROLIMUS/STEROIDS/MMF FOLLOWED BY A CONTROLLED WITHDRAWAL OF STEROIDS OR MMF: RESULTS OF A LARGE, PROSPECTIVE, MULTICENTRE TRIAL. (Abstract #443)**
Kaija Salmela, Y. Vanrenterghem, J. van Hooff, J.P. Squifflet, the European Tacrolimus/MMF Renal Transplantation Study Group. Helsinki, Finland.

2:30 PM **MMF MAY BE GIVEN AT LOWER DOSES WHEN USED IN ASSOCIATION WITH SIROLIMUS IN RENAL TRANSPLANT RECIPIENTS. (Abstract #444)**
David W. Holt, Öyvind Östraat, Josep M. Grinyo, Jean M. Cisterne, Walter Land, Jean P. Squifflet, James T. Burke, Rachel Taylor, James Zimmerman, the Sirolimus European Renal Transplant Study Group. London, United Kingdom.

2:40 PM **AN OPEN-LABELED PILOT STUDY OF STEROID WITHDRAWAL FROM KIDNEY TRANSPLANT RECIPIENTS ON SIROLIMUS-CYCLOSPORINE COMBINATION. (Abstract #445)**
Kamran Mahalati, Barry D. Kahan. Houston, TX.

2:50 PM **SIROLIMUS SUBSTITUTION FOR CALCINEURIN-INHIBITORS IN RENAL ALLOGRAFT RECIPIENTS WITH CALCINEURIN-INHIBITOR NEPHROTOXICITY. (Abstract #446)**
Amit Govil, V. Ram Peddi, Sharad Goel, E. Steve Woodle, M. Roy First. Cincinnati, OH.

3:00 PM **MYCOPHENOLATE MOFETIL-TREATED PRIMARY RENAL TRANSPLANT PATIENTS CAN SUCCESSFULLY UNDERGO STEROID WITHDRAWAL UNDER CYCLOSPORINE OR TACROLIMUS REGIMENS. (Abstract #447)**
Mariella O. Goggins, Dilip Samarapungavan, Ravi Parasuraman, Vanji Karthikeyan, Nizar Attallah, K.K. Venkat, Gary Zasuwa.

3:10 PM **COMPARISON OF STANDARD AND LOW DOSE TACROLIMUS IN CADAVERIC KIDNEY TRANSPLANT RECIPIENTS. (Abstract #448)**
Dirk Kuypers, Bart D. Maes, Thierry Messiaen, Pieter Evenepoel, Willy Coosemans, Jacques Pirenne, Yves Vanrenterghem. Leuven, Belgium; Leuven, Belgium.

3:20 PM **STEROID AVOIDANCE (MINIMIZATION) AFTER KIDNEY TRANSPLANTATION. (Abstract #449)**
Arthur J. Matas, Thiagarajan Ramcharan, Steven Paraskevas, Raja Kandaswamy, Abhinav Humar, Kristen Gillingham. Minneapolis, MN.

Concurrent Session 23: Kidney Transplantation: Live-Donors, Factors and Outcomes

2:00 PM - 3:30 PM

Chicago Ballroom 8/9, Sheraton
Chairs: Lloyd Ratner and Carl Haisch

2:00 PM **LIVING KIDNEY DONATION: LONG-TERM (20-37 YRS) CONSEQUENCES. (Abstract #450)** *Young Investigator Award*
Thiagarjan Ramcharan, Lois McHugh, Raja Kandaswamy, Rainer W. Gruessner, William Payne, David E.R. Sutherland, Abhinav Humar, David Dunn, John Najarian, Arthur Matas. Minneapolis, MN.

2:10 PM **THE SUBSTRATES OF ALTRUISM: A PERSONALITY STUDY OF 200 PROSPECTIVE KIDNEY DONORS. (Abstract #451)**
David Edwin, Lloyd Ratner, Edward Kraus, Jay Markowitz, James Burdick. Baltimore, MD; Baltimore, MD; Baltimore, MD.

2:20 PM **NON-DIRECTED DONATION: WHO VOLUNTEERS? WHO IS REJECTED? (Abstract #452)**
Cheryl Jacobs, Catherine Garvey, Deborah Roman, Abhi Humar, Arthur Matas. Minneapolis, MN.

2:30 PM **EXPANDING THE LIVING DONOR POOL: ABO-INCOMPATIBLE LIVING DONOR KIDNEY TRANSPLANTATION USING BOTH A2 AND NON-A2 BLOOD TYPE DONORS. (Abstract #453)**
James M. Gloor, Alvaro A. Pineda, S. Breanndan Moore, Thomas R. Schwab, Matthew D. Griffin, Jorge A. Velosa, Timothy S. Larson, Mikel Prieto, Scott L. Nyberg, Sylvester Sterioff, Joseph P. Grande, Donna J. Lager, Mark D. Stegall. Rochester, MN.

2:40 PM **GENDER IMBALANCE IN LIVING UNRELATED DONOR RENAL TRANSPLANTATION. (Abstract #454)**
Liise K. Kayler, Herwig-Ulf Meier-Kriesche, Jeffrey D. Punch, John C. Magee, Juan D. Arenas, Darrell A. Campbell, Jr., Steven M. Rudich, Alan B. Leichtman, Robert M. Merion. Ann Arbor, MI; Ann Arbor, MI.

2:50 PM **LIVING ANONYMOUS DONATION-THE NEXT STEP IN THE PURSUIT OF LIVING DONORS: IS THE CHASE OBSCURING OUR VISION. (Abstract #455)**
Antonia J.Z. Henderson, Monica A. Landolt, David N. Landsberg.

3:00 PM **THE BENEFIT OF CHILD-TO-PARENT KIDNEY DONATION: AN ANALYSIS OF UNOS DATA. (Abstract #456)**
Eric P. Cohen, John D. Rosendale, Christine J. Haywood-Bong, Sundaram Hariharan. Milwaukee, WI; Richmond, VA.

3:10 PM **LONG-TERM RESULTS IN KIDNEY TRANSPLANTATION FROM HLA-IDENTICAL LIVING DONORS: A SINGLE-CENTER EXPERIENCE. (Abstract #457)**
Hiroaki Shimmura, Kazunari Tanabe, Tadahiko Tokumoto, Nobuo Ishikawa, Shohei Fuchinoue, Hiroshi Toma. Tokyo; Tokyo, Japan.

3:20 PM **DECLINING INFLUENCE OF RACE ON THE OUTCOME OF LIVING DONOR RENAL TRANSPLANTATION. (Abstract #458)**
Stephen R. Smith, David W. Butterly. Durham, NC.

Concurrent Session 24: Basic Science: Immunosuppression II

2:00 PM - 3:30 PM

Sheraton Ballroom 1-3
Chairs: Paul Kuo and Stanislaw Stepkowski

2:00 PM **RAPAMYCIN INHIBITS TUMOR GROWTH AND METASTASIS IN MICE BY ANTIANGIOGENESIS. (Abstract #459)** *International Young Investigator Award*
Philipp v. Breitenbuch, Markus Guba, Edward K. Geissler, Gudrun Koehl, Stefan Farkas, Carl Zuelke, Mathias Anthuber, Karl-Walter Jauch, Markus Steinbauer. Regensburg, Germany.

2:10 PM **EFFECTS OF IMMUNOSUPPRESSIVE DRUGS ON THE GROWTH OF EBV-TRANSFORMED B CELL LYMPHOMAS FROM PATIENTS WITH PTLD. (Abstract #460)**
Ronald R. Nepomuceno, Daniel A. Falco, Sheri M. Krams, Olivia M. Martinez. Stanford, CA.

2:20 PM **CALCINEURIN INHIBITORS AND RAPAMYCIN EXERT OPPOSITE EFFECTS ON EARLY T CELL SIGNALING PROTEIN EXPRESSION. (Abstract #461)**
Clifford S. Cho, Johny Elkahwaji, Eric R. Manthei, Stuart J. Knechtle, Majed M. Hamawy. Madison, WI.

2:30 PM **MATURE DC LOADED WITH APOPTOTIC/NECROTIC LCL PRIME SPECIFIC $CD4^+$ AND $CD8^+$ T CELL RESPONSES IN BOTH EBV-MEMORY AND/OR -NAÏVE INDIVIDUALS. (Abstract #462)**
Diana M. Metes, Walter J. Storkus, Adriana Zeevi, Joseph Nellis, Alison Logar, John J. Fung, Abdul S. Rao. Pittsburgh, PA; Pittsburgh, PA.

2:40 PM **THE IMMUNOSUPPRESSIVE DRUG RAPAMYCIN INDUCES APOPTOSIS IN MONOCYTE- AND CD34-DERIVED DENDRITIC CELLS, BUT NOT IN MONOCYTES AND MACROPHAGES. (Abstract #463)**
Johan W. De Fijter, Andrea M. Woltman, Sylvia W.A. Kamerling, Sandra W. Van der Kooij, Mohamed R. Daha, Leendert C. Paul, Cees Van Kooten. Leiden, The Netherlands.

2:50 PM **SIROLIMUS (RAPAMYCIN), AN IMMUNOSUPPRESSANT THAT INHIBITS LYMPHOCYTE ACTIVATION, PROTECTS AGAINST AORTIC ATHEROSCLEROSIS IN CHOLESTEROL-FED APO E-DEFICIENT MICE. (Abstract #464)**
Steven J. Adelman, Suren N. Sehgal, Pa-Lang Hsu, Merle Elloso, Caroline A. Kopec, Mike D. Basso, Kristen L. Phiel, Neal Azrolan. Radnor, PA.

3:00 PM **COMPARISON OF SIROLIMUS AND MYCOPHENOLATE MOFETIL ON RENAL FUNCTION AND MORPHOLOGY IN POST-ISCHEMIC RAT KIDNEY. (Abstract #465)**
Takeshi F. Andoh, Ihab M. Wahba, Seung-Ok Choi, William M. Bennett. Portland, OR.

3:10 PM **THE EFFECT OF RAPAMYCIN ON TGF-β SIGNAL TRANSDUCTION IN CULTURED MAMMALIAN CELLS. (Abstract #466)**
Andrew R. Hyman, Vijay K. Sharma, Ruchuang Ding, Milagros Lagman, Manikkam Suthanthiran. Toronto, ON, Canada; New York, NY.

3:20 PM **COMBINED RAPAMYCIN/NEORAL THERAPY EFFECTIVELY INHIBITS ALLOGRAFT VASCULOPATHY IN RAT ALLOGRAFTS BUT ATTENUATES CHANGES IN PRO-FIBROTIC GENE EXPRESSION ASSOCIATED WITH RAPAMYCIN ALONE. (Abstract #467)**
Gavin J. Murphy, Gareth R. Bicknell, Richard N. Saunders, Mathew S. Metcalfe, Michael L. Nicholson.

3:20 PM **RAPAMYCIN HAS NO EFFECT ON ESTABLISHED ALLOGRAFT VASCULOPATHY IN A RODENT MODEL OF CHRONIC REJECTION. (Abstract #468)**
Gavin J. Murphy, Gareth R. Bicknell, Richard N. Saunders, Mathew S. Metcalfe, Michael L. Nicholson.

Concurrent Session 25: Mechanisms of Acute/ Chronic Rejection

2:00 PM - 3:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Fred SanFilippo and Jimmy Light

2:00 PM **Abstract #469 WITHDRAWN**

2:10 PM **HISTOLOGIC FINDINGS FROM 3-6 MONTH, 2-YEAR AND 10-YEAR AFTER TRANSPLANTATION SEQUENTIAL PROTOCOL BIOPSIES IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #470)**
Catherine Girardin, Michael J. Mihatsch, Marie-Noelle Peraldi, Emmanuel Morelon, Dominique Droz, Laure-Helene Noel, Nicole Brousse, Henri Kreis. Paris, France; Basel, Switzerland; Paris, France.

2:20 PM **PROSPECTIVE STUDY OF ANTI-CD11A ANTIBODY IN PREVENTION OF DELAYED GRAFT FUNCTION (DGF) IN RECIPIENTS OF HIGH-RISK RENAL CADAVER GRAFTS. (Abstract #471)**
A. Osama Gaber, for the ANTILFA™ Study Group. Memphis, TN.

2:30 PM **CHRONIC GRAFT NEPHROPATHY (CGN) IS A SEQUEL TO ACUTE INFLAMMATION IN RENAL TRANSPLANT RECIPIENTS TREATED WITH CALCINURIN INHIBITORS. (Abstract #472)**
Wieslaw A. Jurewicz, Nagappan Kumar, Geraint Jones, Keshwar Baboolal, Harry Nair, Alenka Janezic. Cardiff, Wales, United Kingdom.

2:40 PM **A NOMOGRAM FOR PREDICTING THE LIKELIHOOD OF DELAYED GRAFT FUNCTION (DGF) IN ADULT CADAVERIC (CAD) RENAL TRANSPLANT RECIPIENTS. (Abstract #473)**
William D. Irish, David A. McCollum, Raymond J. Tesi, Art B. Owen, Mark Schnitzler Fremont, CA; Stanford, CA; St. Louis, MO.

2:50 PM **MEASUREMENT OF FREQUENCY AND PHENOTYPE OF ALLOPEPTIDE REACTIVE MEMORY T CELLS BY ELISPOT ASSAY IN HUMAN RENAL TRANSPLANT RECIPIENTS. (Abstract #474)**
N. Najafian, B. W. Illigens, E. V. Fedoseyeva, G. Benichou, N. Goes, C. B. Carpenter, M. H. Sayegh. Boston; Boston.

3:00 PM **THE IN VITRO PRODUCTION OF IMMUNOGLOBULIN G ANTIBODIES AGAINST HLA CLASS I AND II MOLECULES FROM NORMAL B CELLS. (Abstract #475)**
Carl J. Cardella, Nashrudeen Hack, Sarita Angra, Eva Friedman, Theresa McKnight. Toronto, ON.

3:10 PM **DIFFERENCES IN THE EFFECT OF DELAYED GRAFT FUNCTION ON SUBSEQUENT ACUTE REJECTION IN OLDER COMPARED TO YOUNGER RENAL ALLOGRAFT RECIPEINTS. (Abstract #476)**
Anson J. Joseph, Omar Hamzeh, Abdolreza Hanrian, Lavanya Bellumkonda, Steven A. Blahut, Ravinder Wali, David K. Klassen, Jeffrey C. Fink, Emilio Ramos, Matthew R. Weir. Baltimore, MD.

3:20 PM **RAPAMYCIN INCREASES GLOMERULAR PROFIBROTIC GENE EXPRESSION AFTER CYCLOSPORIN DOSE REDUCTION IN PATIENTS WITH CHRONIC ALLOGRAFT NEPHROPATHY. (Abstract #477)** *International Young Investigators Award*
Richard N. Saunders, Matthew S. Metcalfe, Gavin J. Murphy, Gareth R. Bicknell, Steven A. White, Michael L. Nicholson. Leicester, United Kingdom.

3:20 PM **Abstract # 478 WITHDRAWN**

Concurrent Session 26: T Cells in Ischemia/ Reperfusion Injury

2:00 PM - 3:30 PM

Empire Room, Intercontinental
Chairs: Daniel Shoskes and Hamid Rabb

2:00 PM **FTY720: A NOVEL THERAPEUTIC APPROACH TO THE REDUCTION OF HEPATIC ISCHEMIA REPERFUSION INJURY. (Abstract #479)**
Dean M. Anselmo, Farin F. Amersi, Xiu-Da Shen, Feng Gao, Charles Lassman, Volker Brinkmann, Jerzy W. Kupiec-Weglinski, Ronald W. Busuttil, Douglas G. Farmer. Los Angeles, CA; Los Angeles, CA; Basel, Switzerland.

2:10 PM **FTY720 PREVENTS ISCHEMIC/REPERFUSION INJURY IN MURINE KIDNEY. (Abstract #480)**
Shiro Takahara, Toshiyuki Tanaka, Jing-Ding Wang, Naotsugu Ichimaru, Kiyohide Toki, Koji Yazawa, Sompol Permpongkosol, Haruhito Azuma, Akihiko Okuyama. Suita, Osaka, Japan; Takatsuki, Osaka, Japan.

2:20 PM **ACCELERATED T-CELL HOMING ATTENUATES NEUTROPHIL MEDIATED PRESERVATION-REPERFUSION INJURY DESPITE ENDOTHELIAL ACTIVATION. (Abstract #481)**
Duska Dragun, T Böhler, M. Nieminen-Kelhä, J. Waiser, H.-H. Neumayer, K. Budde. Berlin, Germany.

2:30 PM **THE CD4 T CELL IS AN IMPORTANT MEDIATOR OF RENAL ISCHEMIA REPERFUSION INJURY. (Abstract #482)**
Melissa J. Burne, Frank Daniels, Michael P. O'Donnell, Shamila Mauiyyedi, Robert B. Colvin, Hamid Rabb. Minneapolis, MN; Boston, MA.

2:40 PM **THE ROLE OF T CELLS IN EXPERIMENTAL ISCHEMIA/ REPERFUSION INJURY. (Abstract #483)**
V. M. Dong, J. Ames, M. Gasser, I. Laskowski, A. M. Waaga, H. Yagita, H. Rennke, M. H. Sayegh, N. L. Tilney. Boston, MA; Boston, MA; Tokyo, Japan.

2:50 PM **UPREGULATION OF B7-1 AND B7-2 ALONG THE VASA RECTA AFTER RENAL ISCHEMIA/REPERFUSION INJURY. (Abstract #484)**
Kathleen E. De Greef, Dirk K. Ysebaert, Sven R. Vercauteren, Veerle Persy, Marc E. De Broe. Belgium.

2:50 PM **ANTI-B7-1 AND NOT ANTI-B7-2 PROTECTS THE KIDNEY AFTER ACUTE ISCHEMIA REPERFUSION INJURY. (Abstract #485)**
Kathleen E. De Greef, Dirk K. Ysebaert, Sven R. Vercauteren, Veerle Persy, Katrien Lorré, Marc E. De Broe. Belgium; Ghent, Belgium.

3:00 PM **ATTENUATION OF EARLY GRAFT INJURY BY ANTI-CD28 MONOCLONAL ANTIBODY AND rPSGL-Ig IN RAT RENAL ISOGRAFTS FROM NON HEART-BEATING DONORS (NHBD). (Abstract #486)**
Igor A. Laskowski, Victor M. Dong, Martin Gasser, Grey D. Shaw, Mohamed H. Sayegh, Nicholas L. Tilney. Boston; Boston; Cambridge.

3:10 PM **REDUCTION IN ISCHEMIA AND REPERFUSION INJURY AFTER RAT INTESTINAL TRANSPLANTATION USING SELECTIVE P-SELECTIN (CD62) BLOCKADE WITH P-SELECTIN GLYCOPROTEIN LIGAND-1 Ig (rPSGL-Ig). (Abstract #487)**
Farin Amersi, Xiu-Da Shen, Feng Gao, Jeffrey Ma, Galen Cortina, Dean Anselmo, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski, Gray Shaw, Douglas G. Farmer. Los Angeles, CA; Los Angeles, CA; Cambridge, MA.

3:20 PM **SELECTIN INHIBITOR BIMOSIAMOSE (TBC 1269) IMPROVES FUNCTION AND SURVIVAL OF KIDNEY ALLOGRAFTS. (Abstract #488)**
Robert Langer, Xuimei Qu, Mou-er Wang, Kurt L. Berens, Peter Vanderslice, Richard A. Dixon, Stanislaw M. Stepkowski, Barry D. Kahan. Houston, TX; Houston, TX.

Concurrent Session 27: Cardiac Allograft Rejection: Cell Death and Molecular Markers

2:00 PM - 3:30 PM

Exchange Room, Intercontinental
Chairs: Bruce Rosengard and Sara Shumway

2:00 PM **BLOCKADE OF THE IL-2Rα-CHAIN AFFECTS THE DEATH SIGNALS OF GRAFT INFILTRATING LYMPHOCYTES AFTER CLINICAL HEART TRANSPLANTATION. (Abstract #489)** *International Young Investigator Award*
Carla C. Baan, Aggie H. Balk, Iza C. van Riemsdijk, Teun van Gelder, Pascal J. Vantrimpont, Lex P. Maat, Willem Weimar. Rotterdam, The Netherlands.

2:10 PM **MITOGEN-ACTIVATED PROTEIN KINASES IN HEART FAILURE AND HEART TRANSPLANTATION IN HUMANS. (Abstract #490)**
Biljana Pavlovic-Surjancev, Maria R. Costanzo, Joseph E. Parrillo. Chicago, IL.

2:20 PM **AUGMENTED T CELL APOPTOSIS IN VIVO VIA CD95 AND TNF R1 AND ACTIVATION INDUCED T-CELL DEATH BY ANTI-THYMOCYTE ANTIBODY TREATMENT OF STABLE CARDIAC TRANSPLANT RECIPIENTS. (Abstract #491)** *International Young Investigator Award*
Hendrik Jan Ankersmit, Bernhard Moser, Georg Roth, Ingo Teufel, Roswita Hericis, Michael Grimm, George Boltz-Nitulescu, Ernst Wolner. Vienna, Austria; Vienna, Austria.

2:30 PM **ENHANCED VASCULAR SMOOTH MUSCLE CELL EXPRESSION OF EARLY GROWTH RESPONSE FACTOR-1 AS A RESULT OF ACUTE CELLULAR REJECTION AND GRAFT VASCULOPATHY IN CARDIAC TRANSPLANT RECIPIENTS. (Abstract #492)** *Young Investigator Award*
Michael V. Autieri, Sheri E. Kelemen, Howard J. Eisen.

2:40 PM **NONINVASIVE DETECTION OF APOPTOSIS FOR THE DIAGNOSIS OF CARDIAC ALLOGRAFT REJECTION: CAN WE DO AWAY WITH ENDOMYOCARDIAL BIOPSY? (Abstract #493)**
Jagat Narula, Elmo R. Acio, Navneet Narula, Diana Wood, Jane M. Fitzpatrick, John E. Tomaszewski, Christine Kelly, Francis D. Blakenberg, Louis E. Samuels, William Strauss. Philadelphia, PA; Philadelphia, PA; Stanford, CA.

2:50 PM **EXPRESSION OF CHEMOKINE GENES DURING ACUTE REJECTION OF HUMAN CARDIAC ALLOGRAFTS. (Abstract #494)**
Nader Fahmy, Mohamad H. Yamani, Randall C. Starling, Norman B. Ratliff, James B. Young, Robert L. Fairchild. Cleveland, OH; Cleveland, OH; Cleveland, OH.

3:00 PM **MONITORING OF DONOR SPECIFIC T SUPPRESSOR CELLS IN HEART ALLOGRAFT RECIPIENTS. (Abstract #495)**
Rodica Ciubotariu, Corrado Cancedda, Gaia Cavallaro, Eric A. Rose, Raffaello Cortesini, Nicole Suciu Foca Cortesini. New York, NY; New York, NY; Rome, Italy.

3:10 PM **CD27 INTRAGRAFT GENE EXPRESSION IN REJECTING AND NON REJECTING CARDIAC ALLOGRAFT RECIPIENTS. (Abstract #496)** *International Young Investigator Award*
Natalia Shulzhenko, Andrey Morgun, Angela P. Chinellato, Gisele F. Rampim, Rosiane V.Z. Diniz, Dirceu R. Almeida, Ismael D.C.G. Silva, Maria Gerbase-DeLima. Sao Paulo, SP, Brazil; Sao Paulo, SP, Brazil.

3:20 PM **DIFFERENTIALLY EXPRESSED GENES IN CARDIAC TRANSPLANT BIOPSIES AND IN MIXED LYMPHOCYTE CULTURE. (Abstract #497)**
Andrey Morgun, Natalia Shulzhenko, Ismael D.C.G. Silva, Gisele F. Rampim, Angela P. Chinellato, Ricardo C. Borra, Maria Gerbase-DeLima. Sao Paulo, SP, Brazil.

Concurrent Session 28: Living Liver Donors I

2:00 PM - 3:30 PM

Grand Ballroom, Intercontinental
Chairs: Michael Millis and Charles Miller

2:00 PM **ANALYSIS OF FAILURE IN LIVING DONOR LIVER TRANSPLANTATION (LRT): DIFFERENTIAL OUTCOMES IN CHILDREN AND ADULTS. (Abstract #498)** *Young Investigator Award*
Michael J. Goldstein, Sandip Kapur, Milan Kinkhabwala, Patricia Harren, Diane LaPoint-Rudow, Steven J. Lobritto, Mark Russo, Robert S. Brown, Jr., Alan Weinberg, John F. Renz, Jean C. Emond. New York, NY; San Francisco, CA.

2:10 PM **PEDIATRIC LIVER TRANSPLANTATION: OUTCOME ANALYSIS OF WHOLE, LIVING DONOR, AND *IN-SITU* SEGMENTAL SPLIT LIVER GRAFTS. (Abstract #499)**
Douglas G. Farmer, Farin Amersi, Sue V. McDiarmid, Mina Bak, Michael Weaver, Rafik M. Ghobrial, Adam Schlifke, Hoang Le, Dean Anselmo, A.J. Maxfield, Natalie Amos, Beth Vandenbogaardt, Hasan Yersiz, Ronald W. Busuttil. Los Angeles, CA; Los Angeles, CA.

2:20 PM **SUCCESSFUL RECONSTRUCTION OF COMPLEX VASCULAR AND BILIARY ANOMALIES DURING ADULT AND PEDIATRIC LIVING-DONOR LIVER TRANSPLANTATION (LDLTX). (Abstract #500)**
Jeffrey A. Lowell, Surendra Shenoy, Venkataraman Ramachandran, Todd K. Howard, Connie Ceriotti, Ross Shepherd, Mauricio Lisker-Melman, Jeffrey Crippin. St. Louis, MO.

2:30 PM **LIVING RELATED LIVER TRANSPLANTATION FOR DECOMPENSATED END-STAGE LIVER DISEASE. (Abstract #501)**
Giuliano Testa, Massimo Malago, Silvio Nadalin, Andreja Frilling, Cristoph E. Broelsch. Essen, Germany.

2:40 PM **EMERGENCY LIVING DONOR LIVER TRANSPLANTATION FOR HIGH-URGENCY PATIENTS. (Abstract #502)**
Chung-Mau Lo, Sheung-Tat Fan, Chi-Leung Liu, William I. Wei, Ching-Lung Lai, Boon-Hun Yong, Karl Young, John Wong. Hong Kong, China.

2:50 PM **OUTCOME ANALYSIS IN ADULT-TO-ADULT LIVING DONOR LIVER TRANSPLANTATION USING LEFT LOBE. (Abstract #503)**
Yuji Soejima, Takashi Nishizaki, Shigeyuki Nagata, Satoko Shiotani, Keizo Sugimachi. Fukuoka, Japan.

3:00 PM **RIGHT LOBE LIVING DONOR LIVER GRAFTING - ADDRESSING THE MIDDLE HEPATIC VEIN CONTROVERSY. (Abstract #504)**
Vanessa H. de Villa, Chao-Long Chen, Yaw-Sen Chen, Chih-Chi Wang, Shih-Hor Wang, Po-Ping Liu, Yu-Fan Cheng, Tung-Liang Huang, Bruno Jawan, Hak-Kim Cheung. Kaohsiung, Taiwan.

3:10 PM **RIGHT LOBE DONORS: CORRELATION OF RESIDUAL LIVER VOLUME WITH HEPATIC FUNCTION IN THE IMMEDIATE POSTOPERATIVE PERIOD. (Abstract #505)**
Glyn R. Morgan, Vivian Lee, Glenn Krinsky, Devon John, Thomas Diflo, Hillel Tobias, Lewis Teperman. New York, NY; New York, NY.

3:20 PM **OUTCOME OF SPLIT LIVER TRANSPLANTATION USING EX-SITU OR IN-SITU SPLITS FROM CADAVER DONORS. (Abstract #506)**
Bjoern Nashan, Rainer Lueck, Thomas Becker, Hans-Juergen Schlitt, Gerrit Grannas, Juergen Klempnauer. Hannover, Germany.

Concurrent Session 29: Basic Science: Co-Stimulation

2:00 PM - 3:30 PM

King Arthur Court Ballroom, Intercontinental
Chairs: William Burlingham and Anne VanBuskirk

2:00 PM **PREVENTION OF ARTERIAL ALLOGRAFT VASCULOPATHY WITH CONCURRENT BLOCKADE OF THE CD28 AND CD40 PATHWAY IN NON-HUMAN PRIMATES. (Abstract #507)**
Nozomu Shirasugi, Andrew B. Adams, Megan D. Durham, Paul Tso, Jongwon Ha, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA; Seoul, Korea.

2:10 PM **THE ROLE OF DONOR ANTIGEN AND CD28-B7 T CELL COSTIMULATORY BLOCKADE IN A CLINICALLY RELEVANT LARGE ANIMAL MODEL OF CARDIAC TRANSPLANTATION. (Abstract #508)** *Young Investigator Award*
Richard S. Lee, James R. Rusche, Lynn A. Cheatham, Kazuhiko Yamada, Stuart L. Houser, Michaella E. Maloney, Hannah C. Amoah, Joshua D. Mezrich, Mohamed H. Sayegh, Joren C. Madsen. Boston, MA; Needham, MA; Boston, MA.

2:20 PM **INDEFINITE SURVIVAL OF ALLOGENEIC HEPATOCYTES PROMOTED BY DST AND ANTI-CD40L mAb. (Abstract #509)**
Donghong Gao, Philomena Salvemini, C. G. Orosz, Ginny L. Bumgardner. Columbus, OH.

2:30 PM **ACTIVE REGULATION OF ALLOGRAFT REJECTION THROUGH THE INDIRECT PATHWAY. (Abstract #510)** *Young Investigator Award*
Akira Yamada, Mohamed H. Sayegh, Hugh Auchincloss, Jr.. Boston, MA; Boston, MA.

2:40 PM **ROLE OF NOVEL T CELL COSTIMULATORY PATHWAYS IN CD28 INDEPENDENT ALLOIMMUNE RESPONSES. (Abstract #511)**
Akira Yamada, Victor M. Dong, Masayuki Sho, Koji Kishimoto, Nadar Najafian, Didier A. Mandelbrot, Hideo Yagita, Hugh Auchincloss, Jr., Mohamed H. Sayegh. Boston, MA; Boston, MA; Boston, MA; Tokyo, Japan.

2:50 PM **ARE REGULATOR CELLS INDUCED BY INDUCTIVE ANTI-CD40L BLOCKADE? (Abstract #512)**
Meera J. Nathan, D. Keith Bishop. Ann Arbor, MI; Ann Arbor, MI.

3:00 PM **TOWARDS DONOR-SPECIFIC TOLERANCE IN RENAL ALLOGRAFTS: REGULATORY MECHANISMS IN DONOR-SPECIFIC HYPORESPONSIVENESS. (Abstract #513)**
Richard J. Baker, Maria P. Hernandez-Fuentes, Fai Ng, Afzal N. Chaudhry, Terry Cook, Robert I. Lechler. London, United Kingdom; London, United Kingdom.

3:10 PM **EVIDENCE FOR A HUMAN CD4+ T REGULATORY CELL SPECIFIC FOR AN ALLO-PEPTIDE DERIVED FROM DONOR HLA CLASS I. (Abstract #514)** *Young Investigator Award*
J. Lee, E. Jankowska-Gan, S. Kusaka, A. M. VanBuskirk, W. J. Burlingham. Madison, WI; Columbus, OH.

3:20 PM **THE ROLE OF THE OX40-OX40L COSTIMULATORY PATHWAY IN ALLOGRAFT REJECTION: INTERACTION WITH THE CD28-B7 COSTIMULATORY PATHWAY. (Abstract #515)**
Xueli Yuan, Victor M. Dong, Hideo Yagita, Mohamed H. Sayegh. Boston, MA; Tokyo, Japan.

Concurrent Session 30: Liver Transplantation Outcomes

2:00 PM - 3:30 PM

Renaissance Ballroom, Intercontinental
Chairs. Gregory Gores and Gregory Everson

2:00 PM **COLON CANCER RISK IN LIVER TRANSPLANT RECIPIENTS: THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY (IPITTR) EXPERIENCE. (Abstract #516)**
Michael J. Hanaway, Jennifer Trofe, Joseph F. Buell, Agnes Lo, Rita R. Alloway, Thomas Beebe, E. Steve Woodle. Cincinnati, OH; Cincinnati, OH.

2:10 PM **INFLAMMATORY BOWEL DISEASE AND COLORECTAL CANCER IN PATIENTS WITH PRIMARY SCLEROSING CHOLANGITIS FOLLOWING LIVER TRANSPLANTATION. (Abstract #517)**
Alonso Vera, Bridget K. Gunson, Daniel Candinas, David A. Mayer, John A.C. Buckels, Paul McMaster, James Neuberger, Darius F. Mirza. Birmingham, England, United Kingdom.

2:20 PM **RENAL DYSFUNCTION LATE AFTER LIVER TRANSPLANTATION: A COMPARISON OF TACROLIMUS AND CYCLOSPORINE. (Abstract #518)**
A. J. Cohen, M. D. Stegall, H. E. Bohorquez, N. N. Onaca, C. B. Rosen, R. H. Wiesner, N. N. Zein. Rochester, MN.

2:30 PM **END STAGE RENAL DISEASE (ESRD) FOLLOWING ORTHOTOPIC LIVER TRANSPLANTATION (OLTX) UTILIZING CALCINEURIN INHIBITOR (CNI) BASED IMMUNOTHERAPY: RISK OF DEVELOPMENT AND TREATMENT. (Abstract #519)**
Thomas A. Gonwa, Martin L. Mai, Larry B. Melton, Steven R. Hays, Marlon F. Levy, Robert G. Goldstein, Ernesto P. Molmenti, Carlos G. Fasola, Goran B. Klintmalm. Dallas, TX.

2:40 PM **SUCCESSFUL TREATMENT OF PORTOPULMONARY HYPERTENSION: SHORT TERM RESULTS AND LONG TERM SURVIVAL. (Abstract #520)**
Ernesto P. Molmenti, Michael A. Ramsay, Kirsten J. Ramsay, Kevin Lynch, H.A. Tillmann-Hein, Robert M. Goldstein, Marlon F. Levy, Ken A. Ausloos, Carlos G. Fasola, Shigeru Marubashi, Edmund Q. Sanchez, Brian Gogel, Juan Escobar, Cara East, Goran B. Klintmalm. Dallas, TX; Dallas, TX.

2:50 PM **STEROID-FREE LIVER TRANSPLANTATION THROUGH THYMOGLOBULIN INDUCTION. (Abstract #521)**
James D. Eason, George E. Loss, Jamie Blazek, Satheesh Nair, Andrew L. Mason. New Orleans, LA.

3:00 PM **IMPACT OF CARDIOVASCULAR RISK FACTORS IN LIVER TRANSPLANT RECIPIENTS ON LATE MORTALITY RELATED TO CARDIOVASCULAR DISEASE. (Abstract #522)**
Raquel D. Conceicao, Russell H. Wiesner, Walter K. Kremers, Woong R. Kim, Carlos V. Paya, Charles B. Rosen, Michael R. Charlton. Rochester, MN.

3:10 PM **CYTOKINE GENE POLYMORPHISMS AND ACUTE HUMAN LIVER GRAFT REJECTION. (Abstract #523)**
Michiel C. Warle, Ayar Farhan, Herold J. Metselaar, Wim C.J. Hop, Chris Perrey, Marcel Kap, Sjoerd de Rave, Jaap Kwekkeboom, Pieter E. Zondervan, Jan N.M. IJzermans, Hugo W. Tilanus, Vera Pravica, Ian V. Hutchinson, Gerda J. Bouma. Rotterdam, The Netherlands; Manchester, United Kingdom; Rotterdam; Rotterdam; Rotterdam.

3:20 PM **EXPRESSION OF CD80 ON KUPFFER CELLS IN DONOR-LIVER IS A MAJOR RISK FACTOR FOR ACUTE REJECTION AFTER CLINICAL LIVER TRANSPLANTATION. (Abstract #524)**
Jaap Kwekkeboom, Ben Bruyneel, Hugo W. Tilanus, Jan N. IJzermans, Gerda J. Bouma, Pieter E. Zondervan, Rob de Man, Herold J. Metselaar. Rotterdam, The Netherlands; Rotterdam, The Netherlands; Rotterdam, The Netherlands.

3:30 PM **Break**

Concurrent Session 31: Regulation of Alloreactive T Cell Responses

4:00 PM - 5:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Jeffrey Punch and Anil Chandraker

4:00 PM **EX VIVO ALLOEDUCATION OF MACROPHAGES, BUT NOT DENDRITIC CELLS PROMOTES IN VIVO GENERATION OF REGULATORY T CELLS. (Abstract #525)**
A. A. Bickerstaff, P. A. O'Connell, A. Thomson, C. G. Orosz. Columbus, OH; Pittsburgh, PA.

4:10 PM **LYMPHOCYTE DOWNREGULATION BY ALLOCHIMERIC CLASS I MOLECULES: UPREGULATION OF HEME OXYGENASE-1 AND DEVELOPMENT OF REGULATORY T CELLS. (Abstract #526)**
Natalya V. Semiletova, Xiu-Da Shen, Feng Gao, Alice Zhao, Yuan Zhai, Letitia Bridges, Ronald W. Busuttil, Jerzy W. Kupec-Weglinski, Rafik M. Ghobrial. Los Angeles, CA.

4:20 PM **P-GLYCOPROTEIN BLOCKADE INHIBITS ALLOIMMUNE T CELL RESPONSES VIA BOTH CD4+ T CELL- AND CD14+ APC-DEPENDENT ACTIVATION PATHWAYS. (Abstract #527)**
Markus H. Frank, Mohamed H. Sayegh, David M. Briscoe. Boston, MA; Boston, MA.

4:30 PM **CONTRASTING EFFECTS OF TARGETING CD28 VERSUS B7 IN ALLOGRAFT REJECTION. (Abstract #528)**
V. M. Dong, X. Yuan, A. M. Waaga, M. Lenhard, H. Yagita, M. H. Sayegh, A. Chandraker. Boston, MA; Tokyo, Japan.

4:40 PM **THE LINKER FOR ACTIVTION OF T CELLS (LAT) IS A TARGET FOR MODULATION BY CYCLOSPORIN A AND FK506: LAT IS A POTENTIAL SUBSTRATE FOR CALCINEURIN. (Abstract #529)**
Majed M. Hamawy, Eric R. Manthei, Johny Elkahwaji, Tausif Alam, Stuart J. Knechtle. Madison, WI.

4:50 PM **EFFECTS OF ANTIOXIDANT THERAPY ON ALLOIMMUNE ACTIVATION AND NF-κB-DEPENDENT INFLAMMATORY CYTOKINE GENE EXPRESSION. (Abstract #530)**
Allan M. Roza, Ashwani K. Khanna, Galen M. Pieper, Cara Olds, Rashid Baz, Gail Hilton, Mark B. Adams. Milwaukee, WI; Milwaukee.

5:00 PM **INHIBITION OF IκB KINASE (IKK) IN TRANSPLANTATION. (Abstract #531)** *Young Investigator Award*
W. Gao, K. L. Faia, A. Castro, V. Csizmadia, N. Shemmeri, L. Wang, A. Batzer, V. Palombella, W. W. Hancock. Cambridge, MA; Frankfurt.

5:10 PM **NF-kB ACTIVATION IS NECESSARY FOR CARDIAC ALLOGRAFT REJECTION. (Abstract #532)**
Ping Zhou, Kwang Woo Hwang, Jun Wang, Zhong Guo, Mary Markiewicz, Oliver Kim, Thomas Gajewski, J. Richard Thistlethwaite, Kenneth Newell, Maria-Luisa Alegre. Chicago, IL.

5:20 PM **IDENTIFICATION OF A NOVEL NK ACTIVATION RECEPTOR, RAT NKP30, FROM ALLOGENEIC LIVER TRANSPLANTS. (Abstract #533)**
Christine L. Hsieh, Yasuhiro Ogura, Olivia M. Martinez, Sheri M. Krams. Stanford, CA.

Concurrent Session 32: Viral Infections in Renal Transplantation

4:00 PM - 5:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: Daniel C. Brennen and Robert Rubin

4:00 PM **IMMUNOGENICITY OF INTRADERMAL HEPATITIS B VACCINATION IN RENAL TRANSPLANT RECIPIENTS NOT RESPONDING TO CONVENTIONAL INTRAMUSCULAR VACCINATION. (Abstract #534)**
B. Y. Choy, J. S.M. Peiris, T. M. Chan, S. K.F. Lo, S. L. Lui, K. N. Lai. Hong Kong, China; Hong Kong, China.

4:10 PM **CLINICAL SIGNIFICANCE OF EPSTEIN-BARR VIRUS (EBV) MONITORING USING A QUANTITATIVE POLYMERASE CHAIN REACTION (PCR) IN RENAL TRANSPLANT RECIPIENTS. (Abstract #535)**
Asha Moudgil, Mieko Toyoda, Bradley A. Warady, Mark W. Johnson, Stanley C. Jordan. Washington, DC; Los Angeles, CA; Kansas City, MO; Chapel Hill, NC.

4:20 PM **ASSESSMENT OF THE IMMUNE RESPONSE AGAINST CYTOMEGALOVIRUS USING NEOPTERIN AND A NOVEL WESTERN BLOT. (Abstract #536)**
Thomas F. Mueller, Therese Jungraithmayr, Marco Reschke, Harald Lange, Klaus Radsak. Marburg, Germany; Marburg, Germany; Boston, MA.

4:30 PM **QUANTITATIVE (TAQMAN™) PCR FOR BK VIRUS AND CIDOFOVIR THERAPY: ROLE IN MANAGEMENT OF BKV INDUCED RENAL ALLOGRAFT DYSFUNCTION. (Abstract #537)** *International Young Investigator Award*
Acar Tuzuner, Malika Saxena, Parmjeet Singh Randhawa, Demetrius Ellis, Michael Moritz, Ron Shapiro, Mark L. Jordan, Carlos Vivas, Velma Scantlebury, Ashok Kumar Jain, Jerry McCauley, Micheal D. Green, Sydney Finkelstein, Tom Gonwa, Richard Cohn, Abhay Vats. Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA.

4:40 PM **POLYOMAVIRUS TYPE JC INFECTION IN NATIVE AND ALLOGRAFT KIDNEYS. (Abstract #538)**
Parmjeet S. Randhawa, Fabian Baksh, Naoto Aoki, Patricia A. Swalsky, Velma Scantlebury, Ron Shapiro, Abhay Vats, Carlos Vivas, Mark Jordan, Sydney Finkelstein. Pittsburgh, PA; Tokyo, Japan; Pittsburgh, PA; Pittsburgh, PA.

4:50 PM **HISTOLOGICAL PATTERNS OF POLYOMA VIRUS DISEASE IN 55 RENAL ALLOGRAFTS: CONCURRENT EXAMINATION OF BIOPSIES AND URINES. (Abstract #539)**
Cinthia B. Drachenberg, Rene C. Drachenberg, John C. Papadimitriou, Jeffrey C. Fink, Charles B. Cangro, Ravinder Wali, Rochelle Cunningham, David K. Klassen, Matthew R. Weir, Stephen T. Bartlett, Emilio Ramos. Baltimore.

5:00 PM **RAPID KIDNEY ALLOGRAFT FAILURE IS ASSOCIATED WITH ANTILYMPHOCYTE THERAPY IN PATIENTS WITH POLYOMA VIRUS INTERSTITIAL NEPHRITIS. (Abstract #540)**
Syed Hussain, Barbara Bresnahan, Eric Cohen, Sundaram Hariharan. Milwaukee, WI.

5:10 PM **HEPATITIS C ANTIBODY STATUS AND OUTCOMES IN RENAL TRANSPLANT RECIPIENTS. (Abstract #541)**
Herwig-Ulf Meier-Kriesche, Akinlolu O. Ojo, Diane M. Cibrik, Julie A. Arndorfer, Bruce Kaplan. Ann Arbor, MI.

5:20 PM **FURTHER SUPPRESSION OF IMMUNE RESPONSE IN RENAL ALLOGRAFT RECIPIENTS INFECTED WITH HEPATITIS C. (Abstract #542)**
Ashwani K. Khanna, Matthew S. Plummer, Venkateswar K. Rao. Milwaukee, WI; Milwaukee, WI; Minneapolis, MN.

Concurrent Session 33: Kidney Transplantation: Immunotherapy, Economics and Efficacy

4:00 PM - 5:30 PM

Chicago Ballroom 8/9, Sheraton
Chairs: Jeffrey Lowell and Marquis Hart

4:00 PM **EFFECT OF CANDESARTAN CILEXETIL ON CHRONIC ALLOGRAFT DYSFUNCTION IN RENAL TRANSPLANT RECIPIENTS. (Abstract #543)**
Kazuya Omoto, Kazunari Tanabe, Tadahiko Tokumoto, Hiroaki Shinmura, Hiroshi Toma. Tokyo, Japan.

4:10 PM **COST-ANALYSIS OF THYMOGLOBULIN INDUCTION WITH INTERMITTENT DOSING BASED ON CD3+ LYMPHOCYTE COUNTS IN HIGH-RISK CADAVER RENAL TRANSPLANT RECIPIENTS. (Abstract #544)**
V. Ram Peddi, Margaret Bryant, Prabir Roy-Chaudhury, E. Steve Woodle, M. Roy First. Cincinnati, OH.

4:20 PM **META-ANALYSIS AND ECONOMIC ANALYSIS OF BASILIXIMAB (SIMULECT) IN RENAL TRANSPLANTATION. (Abstract #545)**
Paul A. Keown, Robert Fred Balshaw, Shideh Khorasheh, Alexander Korn, Zoltan Kalo. Vancouver, BC, Canada; Vancouver, BC, Canada; Vancouver, BC, Canada; Basle, Switzerland.

4:30 PM **COST SAVINGS FOR LIFETIME IMMUNOSUPPRESSION FROM MMF IN CADAVERIC RENAL TRANSPLANT. (Abstract #546)**
Mark A. Schnitzler, Karen E. Craig, Robert S. Woodward, Jeffrey A. Lowell, Daniel C. Brennan. St. Louis, MO; Little Rock, AR.

4:40 PM **THE COST OF NEW ONSET DIABETES MELLITUS AMONG RENAL TRANSPLANT RECIPIENTS. (Abstract #547)**
Robert S. Woodward, Mark A. Schnitzler, Jeffrey A. Lowell, Seema Haider, Thasia G. Woodworth, Lissa Lopez-Rocafort, Jack Baty, Daniel C. Brennan. St. Louis, MO; Groton, CT.

4:50 PM **T CELL RECOVERY AND ANTI-DONOR ALLORESPONSES IN PATIENTS WITH RENAL ALLOGRAFT TOLERANCE FOLLOWING COMBINED HLA-IDENTICAL KIDNEY AND BONE MARROW TRANSPLANTATION WITH NON-MYELOBLATIVE CONDITIONING. (Abstract #548)**
J. Shaffer, A. Kraus, A. B. Cosmi, C. Colby, S. Saidman, F. Preffer, D. Dombkowski, F. Delmonico, S. McAfee, N. Tolkoff-Rubin, B. Dey, R. Sackstein, D. H. Sachs, T. R. Spitzer, M. Sykes. Boston, MA.

5:00 PM **IS USING DACLIZUMAB (DAC) COST EFFECTIVE IN KIDNEY TRANSPLANTATION? (Abstract #549)**
Jimmy A. Light, Diana Y. Barhyte, Jacqueline M. Ennis, Jay G. Barbaccia. Washington, DC; Washington, DC; Washington, DC.

5:10 PM **ECONOMIC EVALUATION OF DIRECT PATIENT CARE PHARMACY SERVICES TO RENAL TRANSPLANT CLINIC PATIENTS. (Abstract #550)**
Leslie J. Vollenweider, Joseph T. DiPiro, Marie A. Chisholm. Athens, GA; Augusta, GA; Augusta, GA.

5:20 PM **QUALITY OF LIFE IN ELDERLY RECIPIENTS OF A KIDNEY TRANSPLANT. (Abstract #551)**
Abhi Humar, Roger Denis, Roberto Mierelles, Kristen Gillingham, Arthur Matas. Minneapolis, MN.

Concurrent Session 34: Basic Science: Tolerance I

4:00 PM - 5:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: Ana Maria Waaga and Alan Kirk

4:00 PM **ALLOREACTIVE MEMORY T CELLS ARE RESISTANT TO TOLERANCE INDUCTION VIA COSTIMULATORY BLOCKADE. (Abstract #552)**
Peter S. Heeger, Anna Valujskikh. Cleveland, OH.

4:10 PM **BLOCKING THE COMMON CYTOKINE RECEPTOR γ-CHAIN (γC) SUPPRESSES ALLOSPECIFIC CD8+ T CELL MEMORY AND FACILITATES CARDIAC ALLOGRAFT ACCEPTANCE IN MICE. (Abstract #553)**
Kenneth E. Kokko, Maylene E. Wagener, Zhenhua Dai, Fadi G. Lakkis. Atlanta, GA.

4:20 PM **ANTI-L-SELECTIN PREVENTS TOLERANCE INDUCTION BY INHIBITING T CELL LYMPHOCYTE HOMING TO THE LYMPH NODE. (Abstract #554)**
Yalai Bai, Lihui Qin, Jonathan S. Bromberg. New York, NY.

4:30 PM **INDUCTION OF TOLERANCE BY ALLOCHIMERIC PROTEIN REQUIRES BOTH SECOND AND THIRD SIGNALS. (Abstract #555)**
Stanislaw M. Stepkowski, Barton Trawick, Robert Kirken, Min Wang, Neelam Tejpal, Mou-er Wang, Barry D. Kahan. Housotn, TX; Houston, TX.

4:40 PM **LONG-TERM RESULTS OF CD-154 BLOCKADE FOR INDUCTION OF MIXED CHIMERISM AND KIDNEY ALLOGRAFT TOLERANCE IN NON-HUMAN PRIMATES. (Abstract #556)**
Tatsuo Kawai, Hiroshi Sogawa, Gregory Abrahamian, Svetlan Boskovic, Siew-Lin Wee, Ognjenka Nadazdin, David Andrews, Dicken Ko, Shamila Mauiyyedi, Robert B. Colvin, David H. Sachs, A. Benedict Cosimi. Boston, MA; Boston, MA.

4:50 PM **RAPAMYCIN BLOCKS COSTIMULATION BLOCKADE RESISTANT CD8+ T-CELL MEDIATED ALLOGRAFT REJECTION. (Abstract #557)**
Yongsheng Li, Xin-Xiao Zheng, Yan Tian, Alberto Sanchez, Terry B. Strom. Boston, MA.

5:00 PM **ANTI-CD45RB MEDIATED ENGRAFTMENT REQUIRES INTACT B7-SIGNALING. (Abstract #558)**
Jin Tian, Paolo O. Salvalaggio, Charlotte Ariyan, Scott Fecteau, Liza Halpern, Arlene Sharpe, Didier Mandelbrot, Mohamed H. Sayegh, Giacomo P. Basadonna, David M. Rothstein. New Haven, CT; Worcester, MA; Boston, MA; Boston, MA.

5:10 PM **CHARACTERIZATION OF GENE EXPRESSION PROFILES IN ANTI-CD3 STIMULATED HUMAN T CELLS WITH AND WITHOUT CD28 COSTIMULATION USING OLIGONUCLEOTIDE MICROARRAYS. (Abstract #559)**
Mark D. Denton, Li-Li Hsiao, Mohamed H. Sayegh, Steven R. Gullans.

5:20 PM ***IN VIVO* AND *IN VITRO* CHARACTERISTICS OF RAT EMBRYONIC STEM CELLS USED FOR TOLERANCE INDUCTION. (Abstract #560)**
Fred Faendrich, Xiongbin Lin, Maren Schulze, Michael Bader, Bert Binas. Kiel, SH, Germany; Berlin, B, Germany.

Concurrent Session 35: Antibodies and Immunomodulation

4:00 PM - 5:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Enver Akalin and James Schulak

4:00 PM **TWO DOSES OF DACLIZUMAB ARE SUFFICIENT FOR PROLONGED IL-2Rα-CHAIN BLOCKADE. (Abstract #561)**
Cornelis G. ter Meulen, Iza van Riemsdijk, Ronald J. Hene, Luuk B. Hilbrands, Carla C. Baan, Andries J. Hoitsma. Nijmegen, The Netherlands; Rotterdam, The Netherlands; Utrecht, The Netherlands.

4:10 PM **A PHASE I/II TRIAL OF ANTI-CD11a MONOCLONAL ANTIBODY IN RENAL TRANSPLANTATION. (Abstract #562)**
F. Vincenti, R. Mendez, P. R. Rajagopalan, A. Wilkinson, K. Butt, D. Laskow, D. Slakey, M. Lorber, M. D. Pescovitz, R. Dedrick, P. Walicke, M. R. Garovoy. San Francisco, CA; LA, CA; LA, CA; Berkeley, CA.

4:20 PM **PLASMAPHERESIS (PP) AND INTRAVENOUS IMMUNE GLOBULIN(IG)FOR THE TREATMENT AND PREVENTION OF ANTIBODY-MEDIATED REJECTION IN RENAL TRANSPLANTATION. (Abstract #563)**
Lloyd E. Ratner, Karen King, Henkie P. Tan, Andrea A. Zachary, Milagros D. Samaniego, Edward S. Kraus, Matthew Cooper, Robert A. Montgomery. Baltimore, MD.

4:30 PM **INDUCTION IMMUNOSUPPRESSION FOR CADAVERIC RENAL TRANSPLANTATION UTILIZING SIROLIMUS, BASILIXIMAB AND DELAYED INTRODUCTION OF CYCLOSPORINE. (Abstract #564)**
Richard J. Knight, Charles T. Van Buren, Stephen M. Katz, Barry D. Kahan. Houston, TX.

4:40 PM **BASILIXIMAB (SIMULECT®) CAN BE ADMINISTERED SAFELY AND EFFECTIVELY BY IV BOLUS IN A SINGLE DOSE ON DAY 1 POST RENAL TRANSPLANTION IN PATIENTS RECEIVING TRIPLE THERAPY WITH AZATHIOPRINE. (Abstract #565)**
I. Matl, P. Bachleda, R. Michalsky, P. Navratil, M. Lao, V. Treska, H. Prestele, M. Matthisson, A. Kom. Prague, Czech Republic.

4:50 PM **INFUSION OF KIDNEY RECIPIENTS WITH DONOR BONE MARROW CELLS (DBMC) DIFFERENTIALLY EFFECT THE KINETICS AND MAGNITUDE OF THE IgG ANTI-CMV RESPONSE. (Abstract #566)**
Manuel R. Carreno, Gaetano Ciancio, George W. Burke, James M. Mathew, Carmen Gomez, David Roth, Laphalle Fuller, Andreas G. Tzakis, Camillo Ricordi, Joshua Miller, Violet Esquenazi. Miami, FL.

5:00 PM **PHASE I/II SAFETY-EFFICACY TRIAL OF INTERCELLULAR ADHESION MOLECULE-1 (ICAM-1) ANTISENSE OLIGODEOXYNUCLEOTIDE (ISIS 2302) IN THE PREVENTION OF ACUTE ALLOGRAFT REJECTION. (Abstract #567)**
Murat Kilic, Katz Stephen, Van Buren T. Charles, Welsh Maria, Shanahan R. William, Kahan D. Barry. Houston, TX.

5:10 PM **A DECREASE IN THE SERUM LEVEL OF SOLUBLE-CD25 (S-CD25) PARALLELS THE REAPPEARANCE OF $CD25^{POS}$ T-LYMPHOCYTES IN RENAL TRANSPLANT PATIENTS TREATED WITH DACLIZUMAB. (Abstract #568)**
Cornelis G. ter Meulen, Cor W.M. Jacobs, Ina S. Klasen, Carla T.M.A. Verweij, Luuk B. Hilbrands, Andries J. Hoitsma. Nijmegen, The Netherlands; Nijmegen, The Netherlands.

Concurrent Session 36: Bone Marrow/Cell Transplantation

4:00 PM - 5:30 PM

Empire Room, Intercontinental
Chairs: Xian Chang Li and Noriko Murase

4:00 PM **TIGHTLY REGULATED IMMORTAL HUMAN HEPATOCYTE CELL LINES FOR A BIOARTIFICIAL LIVER. (Abstract #569)**
Naoya Kobayashi, Hirofumi Noguchi, Toshinori Totsugawa, Toshihisa Matsumura, Takamasa Watanabe, Toshiyoshi Fujiwara, Noriaki Tanaka, Masakiyo Sakaguchi, Karen A. Westerman, Philippe Leboulch, Ira J. Fox. Okayama, Japan; Okayama, Japan; Cambridge, MA; Omaha, NE.

4:10 PM **COCULTIVATION OF IMMORTALIZED HUMAN HEPATOCYTES AND LIVER FAT-STORING CELLS INCREASES LIVER-SPECIFIC FUNCTIONS. (Abstract #570)**
Takamasa Watanabe, Naoya Kobayashi, Hirofumi Noguchi, Toshinori Totsugawa, Toshihisa Matsumura, Toshiyoshi Fujiwara, Masakiyo Sakaguchi, Masaki Hikida, Hitoshi Ohmori, Karen A. Westerman, Philippe Leboulch, Noriaki Tanaka. Okayama, Japan; Okayama, Japan; Okayama, Japan; Cambridge, MA.

4:20 PM **MAINTENANCE OF TRNASPLANTED HEPATOCYTES IN VIVO WITH C-MET ACTIVATION. (Abstract #571)**
Kazuo Ohashi, Leonard Meuse, Ralph Schwall, Mark A. Kay. Stanford, CA; South San Francisco, CA.

4:30 PM **REVERSIBLY IMMORTALIZED HEPATOCYTES FUNCTION AS WELL AS PRIMARY HEPATOCYTES TO TREAT HEPATIC ENCEPHALOPATHY. (Abstract #572)**
J. Cai, M. Ito, H. Nagata, C. Gao, D. LaFleur, I. J. Fox. Omaha, NE.

4:40 PM **TURNING BLOOD INTO LIVER: A MODEL TO TEST WHETHER PURIFIED AND EX-VIVO EXPANDED HEMATOPOIETIC STEM CELLS CAN HOME, ENGRAFT, AND DIFFERENTIATE IN VIVO INTO HEPATOCYTES. (Abstract #573)**
Eric Elster, K. Johnson, S. Hoffmann, M. Wells, P. Blair, D. Harlan, A. Kirk, John Chute. Bethesda, MD; Bethesda, MD.

4:50 PM **FUNCTIONAL CHARACTERIZATION OF GRAFT FACILITATING CELLS: FURTHER DELINEATING THE ROLE OF THE CD3ε SURFACE MOLECULE. (Abstract #574)**
Carrie L. Schanie, Daniel E. Cramer, H. Leighton Grimes, Suzanne T. Ildstad. Louisville, KY.

5:00 PM **GENETIC MATCHING BETWEEN FACILITATING CELLS (FC) AND HEMATOPOIETIC STEM CELLS (HSC) AT THE MHC CLASS I K LOCUS IS CRITICAL FOR DURABLE ENGRAFTMENT. (Abstract #575)**
Yiming Huang, Daniel E. Cramer, H. Leighton Grimes, Suzanne T. Ildstad. Louisville, KY.

5:10 PM **RAPAMYCIN IS AS EFFECTIVE AS T-CELL COSTIMULATORY BLOCKADE ON PROMOTING THE INDUCTION OF FULLY MHC-MISMATCHED MIXED CHIMERISM UNDER IRRADIATION-FREE CONDITIONS. (Abstract #576)**
Hakan Sozen, Tao Wu, Ping Lan, Neal Heuss, Hannes Kalscheuer, David E.R. Sutherland, Bernhard J. Hering, Zhiguang Guo. Minneapolis, MN.

5:20 PM **BONE MARROW FROM NONOBESE DIABETIC MICE IS LACKING FUNCTIONAL FACILITATING CELLS. (Abstract #577)**
Paula M. Chilton, Yiming Huang, Hong Xu, Suzanne T. Ildstad. Louisville, KY.

Concurrent Session 37: Cardiac Transplantation Complications: Diagnosis & Treatment

4:00 PM - 5:30 PM

Exchange Room, Intercontinental
Chairs: David Taylor and Si Pham

4:00 PM **SEVERE HYPOGAMMAGLOBULINEMIA FOLLOWING HEART TRANSPLANTATION: IMPACT OF PRE-EMPTIVE USE OF IMMUNOGLOBULIN REPLACEMENT (CYTOGAM) ON INFECTION AND REJECTION OUTCOMES. (Abstract #578)**
Mohamad H. Yamani, Robin Avery, Steven Mawhorter, James B. Young, Ann McNeill, Norman B. Ratliff, Patrick McCarthy, Randall C. Starling. Cleveland, OH.

4:10 PM **LONG TERM RESULTS IN SENSITIZED PATIENTS TREATED WITH PRE CARDIAC TRANSPLANT INTRAVENOUS IMMUNOGLOBULIN G AND PLASMAPHERESIS. (Abstract #579)**
Barbara A. Pisani, Jose C. Mendez, Bryan K. Foy, Mark J. Stout, Robert C. Lichtenberg, Krystyna Malinowska, G. Martin Mullen, John A. Robinson. Maywood, IL.

4:20 PM **LEFT VENTRICULAR DYSFUNCTION DURING ACUTE HEART ALLOGRAFT REJECTION: RELATIONSHIP TO THE ISHLT SEVEN-GRADE SCORING. (Abstract #580)**
M. Dandel, M. Hummel, J. Müller, R. Meyer, R. Ewert, R. Hetzer. Berlin, Germany.

4:30 PM **PLASMAPHERESIS LOWERS THE INCIDENCE OF TRANSPLANT CORONARY ARTERY DISEASE FOLLOWING HEMODYNAMIC COMPROMISING HEART TRANSPLANT REJECTION. (Abstract #581)**
Gregory A. Cogert, Jon A. Kobashigawa, Maria L. Espejo, Jignesh Patel, Jamie Moriguchi, Hillel Laks. Los Angeles, CA; Los Angeles, CA.

4:40 PM **NON-INVASIVE REJECTION MONITORING AFTER HEART TRANSPLANTATION (Tx). A RETROSPECTIVE ANALYSIS OF DATA FROM 734 CARDIAC RECEIPIENTS. (Abstract #582)**
Johannes Müller, Antje Eubel, Michael Dandel, Manfred Hummel, Ralf Ewert, Rudolf Meyer, Roland Hetzer. Berlin, Germany.

4:50 PM **LOW AFFINITY FC RECEPTOR (CD32)MEDIATED PLATELET AGGREGATION/ACTIVATION IN VITRO BY ANTITHYMOCYTEGLOBULIN: A POSSIBLE EXPLANATION FOR INDUCTION THERAPY MEDIATED PLATELET DEPLETION IN VIVO. (Abstract #583)**
Hendrik Jan Ankersmit, Bernhard Moser, Georg Roth, Ivo Volf, George Wieselthaler, Michael Grimm, Ernst Wolner. Vienna, VIE, Austria; Vienna, VIE, Austria.

5:00 PM **TRANSMISSION OF DONOR CANCER INTO CARDIO-THORACIC TRANSPLANT RECIPIENTS. (Abstract #584)**
Joseph F. Buell, Jennifer Trofe, Bruce Rosengard, Michael J. Hanaway, Horacio L. Rilo, Tom Beebe, M. Roy First, E. Steve Woodle. Cincinnati, OH.

5:10 PM **EXPERIENCE WITH 274 CARDIAC TRANSPLANT RECIPIENTS WITH POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDER: A REPORT FROM THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY. (Abstract #585)**
M. J. Aull, J. Trofe, R. R. Alloway, M. Hanaway, M. R. First, L. Wagoner, T. Beebe, E. S. Woodle, J. F. Buell. Cincinnati, OH.

5:20 PM **DIABETES AFFECTS LONG-TERM SURVIVAL AFTER HEART TRANSPLANTATION. (Abstract #586)**
Martin Czerny, Vedat Sahin, Daniel Zimpfer, Juliane Kilo, Harald Baumer, Andreas Zuckermann, Peter Fasching, Ernst Wolner, Michael Grimm.

Concurrent Session 38: Liver Transplantation: Allocation

4:00 PM - 5:30 PM

Grand Ballroom, Intercontinental
Chairs: John Fung and Goran Klintmalm

4:00 PM **SHARING OF LIVERS FOR STATUS 1 RECIPIENTS IN REGION 7 - A GOOD THING. (Abstract #587)**
Abhi Humar, Brenda Durand, Marci Knaack, Lynn Shriver, Anne Kallis, Jack Lake, William Payne. Minneapolis, MN.

4:10 PM **PRELIMINARY RESULTS OF A LIVER ALLOCATION PLAN USING A CONTINUOUS MEDICAL SEVERITY SCORE THAT DE-EMPHASIZES WAITING TIME. (Abstract #588)**
Richard B. Freeman, Richard J. Rohrer, Elizer Katz, W. David Lewis, Roger Jenkins, A. Benedict Cosimi, Francis L. Delmonico, Amy Friedman, Marc I. Lorber, Kevin O' Conner, James Bradley. Boston, MA; Worcester, MA; Burlington, MA; Boston, MA; New Haven, CT; Boston, MA.

4:20 PM **THE MODEL FOR ENDSTAGE LIVER DISEASE (MELD) IN THE PREDICTION OF SURVIVAL IN PATIENTS WITH LIVER CIRRHOSIS. (Abstract #589)**
W. Ray Kim, Patrick S. Kamath, Joanne T. Benson, Walter K. Kremers, E. Rolland Dickson, Russell H. Wiesner. Rochester, MN.

4:30 PM **COMPARISON OF MAYO END-STAGE LIVER DISEASE (MELD) AND CHILD-TURCOTTE-PUGH (CTP) SCORES AS PREDICTORS OF PRE-LIVER TRANSPLANT (LT) DISEASE SEVERITY AND POST-LT OUTCOMES IN UNOS STATUS 2A PATIENTS. (Abstract #590)**
Robert S. Brown, Jr., K. Shiva Kumar, Steven J. Lobritto, Dianne L. Rudow, Patricia Harren, Milan Kinkhabwala, Sandip Kapur, Jean C. Emond. New York, NY.

4:40 PM **PREDICTION OF LIVER TRANSPLANT OUTCOME USING THE MELD SCALE. (Abstract #591)**
W. Ray Kim, Russell H. Wiesner, Patrick S. Kamath, Michael Malinchoc, Walter K. Kremers, Charles B. Rosen, E. Rolland Dickson, Goran B. Klintmalm. Rochester, MN; Dallas, TX.

4:50 PM **NEGATIVE IMPACT OF INCREASED WAITING TIME IN PATIENTS WITH CIRRHOSIS AND HEPATOCELLULAR CARCINOMA UNDERGOING LIVER TRANSPLANTATION. (Abstract #592)**
Omar Galdame, Valeria Descalzi, Alejandra G. Villamil, Juan C. Bandi, Sara Chao, Eduardo Mullen, Guillermo Gallo, Juan Pekolj, Lucas McCormack, Miguel Ciardullo, Eduardo deSantibanes, Luis G. Podesta, Federico Villamil, Adrian C. Gadano. Buenos Aires, Argentina; Buenos Aires, Argentina.

5:00 PM **TRENDS AND GEOGRAPHIC VARIABILITY IN MORTALITY FROM LIVER DISEASE IN NEW YORK STATE: IMPACT OF LIVER TRANSPLANTATION (LT). (Abstract #593)**
Jean C. Emond, Lynn Rogut, Robert S. Brown. New York, NY; New York, NY.

5:10 PM **COMPARISON OF WHOLE CADAVERIC AND *IN-VIVO* SPLIT RIGHT TRISEGMENTAL LIVER GRAFTS. (Abstract #594)**
Rafik M. Ghobrial, Hasan Yersiz, Douglas G. Farmer, Farin Amersi, Marc N. Roseboro, Angeles Baquerizo, Sunil Geevarghese, Susan Lerner, Nicholas Nissen, Ronald W. Busuttil. Los Angeles, CA.

5:20 PM **EX VIVO SPLIT LIVER TRANSPLANTATION - COMPLICATIONS AND OUTCOME. (Abstract #595)**
Huda M. Noujaim, Mike Fink, Brigitte K. Gunson, David A. Mayer, John A.C. Buckels, Daniel Candinas, Paul McMaster, John De Ville DeGoyet, Darius F. Mirza. Birmingham, United Kingdom; Birmingham, United Kingdom.

Concurrent Session 39: Pathology, Techniques and Results of Pancreas Transplantation

4:00 PM - 5:30 PM

King Arthur Court Ballroom, Intercontinental
Chairs: Lillian Gaber and Paul Gorres

4:00 PM **REVISED HISTOLOGIC GRADING SCHEME FOR CHRONIC PANCREAS ALLOGRAFT REJECTION (CR) IN CORE NEEDLE BIOPSIES: PROGNOSTIC SIGNIFICANCE. (Abstract #596)**
John C. Papadimitriou, Cinthia B. Drachenberg, David K. Klassen, Jeffrey C. Fink, Charles B. Cangro, Matthew R. Weir, Emilio Ramos, Ravinder Wali, Rochelle Cunningham, Stephen T. Bartlett. Baltimore, MD.

4:10 PM **SIMULTANEOUS PANCREAS (PA) AND KIDNEY (KD) TRANSPLANTS (TXS) (SPKS) FROM TWO DIFFERENT DONORS (DD) - A REGISTRY REPORT. (Abstract #597)**
Angelika C. Gruessner, David E.R. Sutherland, Barbara J. Bland, Rainer W.G. Gruessner. Minneapolis, MN.

4:20 PM **IMPACT OF ACUTE REJECTION EPISODES ON THE LONG-TERM GRAFT SURVIVAL IN SIMULTANEOUS KIDNEY PANCREAS TRANSPLANT RECIPIENTS. (Abstract #598)**
Sudhakar Reddy, Darcy Davies, Deborah Ormond, Thomas Johnston, Dinesh Ranjan. Lexington, KY; Richmond, VA.

4:30 PM **RISK FACTORS FOR ACUTE REJECTION (AR) IN PANCREAS TRANSPLANT RECIPIENTS: A MULTIVARIATE ANALYSIS. (Abstract #599)**
Abhi Humar, Raja Kandaswamy, Thiagarjan Ramcharan, Steven Paraskevas, Roberto Meirelles, Rainer W. Gruessner, Angelika Gruessner, David E.R. Sutherland. Minneapolis, MN.

4:40 PM **THE IMMUNOLOGIC ADVANTAGE OF PORTAL VENOUS DRAINAGE OF THE PANCREAS ALLOGRAFT IN SIMULTANEOUS PANCREAS-KIDNEY TRANSPLANTATION. (Abstract #600)**
Richard Perez, Christoph Troppmann, John McVicar, J. Michael Cecka. Sacramento, CA; Los Angeles, CA.

4:50 PM **IMMUNOLOGIC PROTECTION OF KIDNEYS BY PORTAL VENOUS DRAINAGE OF THE PANCREAS IN SIMULTANEOUS PANCREAS KIDNEY (SPK) TRANSPLANTATION. (Abstract #601)**
Benjamin Philosophe, Anne M. Wiland, David K. Klassen, Eugene J. Schweitzer, Alan C. Farney, John O. Colonna, Clarence Foster, Adam M. Frank, Bruce E. Jarrell, Stephen T. Bartlett. Baltimore, MD.

5:00 PM **UTILITY OF SURVEILLANCE BIOPSIES FOLLOWING SOLITARY PANCREAS TRANSPLANTATION. (Abstract #602)**
Timothy S. Larson, Dean Y. Kim, Herschel A. Carpenter, Lawrence J. Burgart, Jorge A. Velosa, Mark D. Stegall. Rochester, MN.

5:10 PM **IMPROVED PATIENT SURVIVAL IN KIDNEY-PANCREAS TRANSPLANT RECIPIENTS (KPTx)TREATED WITH HMG CoA REDUCTASE INHIBITORS (HMG). (Abstract #603)**
Todd E. Pesavento, Fernando G. Cosio, Ronald P. Pelletier, Elmahdi A.. Elkhammas, Mitchell L. Henry, Bradley S. Ingram, Ronald M. Ferguson. Columbus, OH; Columbus, OH; Westminster, MD.

5:20 PM **THE DISCOVERY AND MANAGEMENT OF OCCULT REJECTION ON PROTOCOL PANCREAS & KIDNEY BIOPSIES IN SPK TRANSPLANTS EQUALIZED OUTCOMES BETWEEN TAC & CSA TREATED PTS. (Abstract #604)**
C. Marsh, J. Lovato, J. Chawla, C. Davis. Seattle, WA.

Concurrent Session 40: Pediatrics I (Liver)

4:00 PM - 5:30 PM

Renaissance Ballroom, Intercontinental
Chairs: Deidre Kelly and Lynt Johnson

4:00 PM **CIRCULATING EBV-DNA IN THE MONITORING OF EBV INFECTION AND PREVENTION OF PTLD IN PEDIATRIC LIVER TRANSPLANTATION. (Abstract #605)**
Marco Spada, Michela Guizzetti, Wanda Petz, Michele Colledan, Andrea Segalin, Alessandro Lucianetti, Alessandro Bertani, Giuseppe Peloni, Aurelio Sonzogni, Daniele Alberti, Silvia Riva, Maria Luisa Melzi, Bruno Gridelli. Bergamo, Italy; Bergamo, Italy; Bergamo, Italy; Bergamo, Italy.

4:10 PM **LYMPHOCYTE SUBSETS MAY DISCERN TREATMENT EFFECTS IN CHILDREN WITH POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDER (PTLD). (Abstract #606)**
Jessie Ganjoo, Michael Green, Rakesh Sindhi, George Mazariegos, Jorge Reyes. Pittsburgh, PA.

4:20 PM **HEALTH STATUS OF LONG-TERM SURVIVORS OF PEDIATRIC LIVER TRANSPLANTATION. (Abstract #607)**
Jeffrey B. Brown, Margaret E. Knapp, Karan M. Emerick, Timothy A. Sentongo, Joan M. Lokar, Brian M. Stahulak, Peter F. Whitington, Estella M. Alonso. Chicago, IL.

4:30 PM **500 PEDIATRIC LIVER TRANSPLANT RECIPIENTS AT SAINT-LUC UNIVERSITY CLINICS: IMPACT OF ABO, HLA COMPATIBILITY, T-CELL CROSSMATCH AND GENDER ON GRAFT OUTCOME. (Abstract #608)**
Veerle Evrard, Raymond Reding, Dominique Latinne, Fabian Haccourt, Jean-Stephan Rochet, Magda Janssen, Killick Paul, Etienne Sokal, Jean-Bernard Otte.

4:40 PM **PEDIATRIC LIVER TRANSPLANTATION IN 808 CONSECUTIVE CHILDREN: 18 YEARS EXPERIENCE FROM A SINGLE CENTER. (Abstract #609)**
Ashok B. Jain, Randeep S. Kashyap, Santosh Potdar, John J. Fung, Jorge Reyes. Pittsburgh, PA.

4:50 PM **AN ANALYSIS OF PRE AND POSTTRANSPLANTATION RISK FACTORS ASSOCIATED WITH OUTCOME AFTER PEDIATRIC LIVER TRANSPLANTATION. (Abstract #610)**
Marco Spada, Wanda Petz, Michele Colledan, Andrea Segalin, Alessandro Lucianetti, Alessandro Bertani, Michela Guizzetti, Giuseppe Peloni, Bruno Gridelli. Bergamo, Italy.

5:00 PM **PROGRESSIVE PORTAL FIBROSIS IN LONG TERM SURVIVORS OF PEDIATRIC LIVER TRANSPLANT RECIPIENTS. (Abstract #611)**
Annette S.H. Gouw, Marius C. van den Heuvel, Sibrand Poppema, Koert P. de Jong, Maarten J.H Slooff, Paul M.J.G. Peeters. Groningen, The Netherlands; Groningen, The Netherlands.

5:10 PM **MORBIDITY FROM CONGENITAL HEPATIC FIBROSIS AFTER KIDNEY TRANSPLANTATION FOR AUTOSOMAL RECESSIVE POLYCYSTIC DISEASE. (Abstract #612)**
Khalid Khan, Sara Jane Schwarzenberg, Harvey Sharp, Arthur J. Matas, Blanche M. Chavers. Minneapolis, MN; Minneapolis, MN.

5:20 PM **NON-COMPLIANCE IN CHILDREN POST-LIVER TRANSPLANT-WHO ARE THE CULPRITS CHILDREN OR PARENTS? (Abstract #613)**
Kathleen Falkenstein, Louise Flynn, Beverly Kirkpatrick, Adela T. Casas, Stephen P. Dunn. Wilmington, DE.

5:45 PM ASTS Business Meeting

Sheraton Ballroom 1-3, Sheraton

Poster Session II

8:00 AM - 7:00 PM

Presenters in Attendance: 5:30 PM - 7:00 PM
Beer and Pretzel Reception
River Exhibition Hall

♦*Also presented in Selected Poster Session*

Kidney - Acute/Chronic Rejection II

P1 **NEORAL AND TACROLIMUS ARE ASSOCIATED WITH A DECREASE IN CHRONIC ALLOGRAFT FAILURE AS COMPARED WITH SANDIMMUNE. (Abstract #614)**
Herwig-Ulf Meier-Kriesche, Bruce Kaplan. Ann Arbor, MI.

P2 **PRE-TRANSPLANT C-REACTIVE PROTEIN (CRP) AS A PREDICTOR OF CHRONIC ALLOGRAFT NEPHROLOGY(CAN): A CASE-CONTROL STUDY. (Abstract #615)**
Macaulay Onuigbo, Steven A. Blahut, R. Christenson, Matthew R. Weir, Jeffrey C. Fink, M. Gutierrez. Baltimore, MD.

P3 **TGF-BETA1 GENE POLYMORPHISM IN ORGAN DONOR AND KIDNEY GRAFT RECIPIENTS. THE EFFECT ON POSTTRANSPLANT OUTCOME. (Abstract #616)**
Jiri Lacha, Jaroslav A. Hubacek, Petr Potmesil, Ondrej Viklicky, Nina Bendukidze, Uwe Heemann, Stefan Vitko. Prague, Czech Republic; Prague, Czech Republic; Essen, Germany; Prague, Czech Republic.

P4 **TACROLIMUS VERSUS CYCLOSPORINE IN KIDNEY TRANSPLANTATION: FIVE-YEAR GRAFT LOSS IN PATIENTS WITH ACUTE REJECTION AND HYPERCHOLESTEROLEMIA. (Abstract #617)**
John D. Pirsch, Darby Thompson, FK506 Kidney Transplant Study Group. Madison, WI; Potomac, MD.

P5 **RATE OF DECLINE IN GFR IN PATIENTS WITH CHRONIC ALLOGRAFT NEPHROPATHY (CAN): EFFECT OF CALCINEURIN INHIBITORS (CI). (Abstract #618)**
Jorge A. Velosa, Joseph P. Grande, Timothy S. Larson, Matthew D. Griffin, Thomas R. Schwab, James M. Gloor, Erik J. Bergstralh, Carrie L. Loebertmann, Kari M. Thomas, Mark D. Stegall. Rochester, MN.

P6 **DETECTION OF DONOR-SPECIFIC HLA CLASS II ANTIBODIES BY FLOW CYTOMETRY. (Abstract #619)**
Juan C. Scornik, Mai Ta, Agnieszka M. Avizinis. Gainesville, FL; Gainesville, FL.

P7 **ANGIOTENSIN CONVERTING ENZYME (ACE) INHIBITION IN CHRONIC RENAL ALLOGRAFT NEPHROPATHY. (Abstract #620)**
Julie Lin, Anthony M. Valeri, David J. Cohen, Jai Radhakrishnan. New York, NY.

P8 **RENAL ALLOGRAFT OUTCOMES IN AFRICAN-AMERICAN VERSUS CAUCASIAN TRANSPLANT RECIPIENTS IN THE TACROLIMUS ERA. (Abstract #621)** ♦
Karen L. Hardinger, Robert J. Stratta, M. Francesca Egidi, Rita R. Alloway, M. Hosein Shokouh-Amiri, Lillian W. Gaber, Hani P. Grewal, Marsha R. Honaker, Santiago R. Vera, A. Osama Gaber. Memphis, TN.

P9 **QUANTITATION OF CYTOTOXIC LYMPHOCYTE AND CYTOKINE GENE EXPRESSION IN RENAL ALLOGRAFT BIOPSIES BY REAL-TIME TAQMAN PCR. (Abstract #622)**
Katrin Kliem, Athanasios Vergopoulos, Nina Babel, Christian Rosenberger, Michael Oppert, Katrin Vogt, Hans Dieter Volk, Petra Reinke. Berlin, Germany.

P10 **CHRONIC ALLOGRAFT NEPHROPATHY: A SINGLE CENTRE RANDOMISED TRIAL OF CYCLOSPORIN WITHDRAWAL AND MYCOPHENOLATE MOFETIL OR TACROLIMUS SUBSTITUTION IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #623)**
John S. McGrath, Keith P. Graetz, Keith M. Rigg, Magdi Shehata. Nottingham, United Kingdom.

P11 **IS THE IMPROVEMENT IN RENAL GRAFT FUNCTION AFTER MYCOPHENOLATE MOFETIL IN CHILDREN WITH CHRONIC TRANSPLANT NEPHROPATHY LONGSTANDING? (Abstract #624)**
Lilian M.P. Araujo, Francine B.C. Lemos, Daisa S.R. David, Eduardo Mazzucchi, William C. Nahas, Sami Arap, Luiz E. Ianhez, Elias David-Neto. Sao Paulo, Brazil.

Kidney - GVH, Complications, Infections II

P12 **ASSOCIATION OF ANTIBODY INDUCTION AND SHORT AND LONG-TERM CAUSE SPECIFIC MORTALITY AFTER RENAL TRANSPLANTATION. (Abstract #625)** ♦
Herwig-Ulf Meier-Kriesche, Friedrich K. Port, Bruce Kaplan. Ann Arbor, MI.

P13 **CYCLOSPORINE ARTERIOLOPATHY (CAA) IN LONG-TERM CyA TREATED RENAL ALLOGRAFT RECIPIENTS. (Abstract #626)**
Yasuhiro Okabe, Kazunari Tanabe, Tadahiko Tokumoto, Hiroaki Shimmura, Yutaka Yamaguchi, Hiroshi Toma. Tokyo; Kashiwa, Japan.

P14 **NEW SERUM MARKER OF BONE RESORPTION (CrossLaps) AND BONE MASS IN RENAL TRANSPLANT RECIPIENTS (RTR). (Abstract #627)**
Jorge A. Argento, Luis S. Re, Norma Venegas, Alejandra Oviedo, Ana Wittich, Cristina Casco, Maria C. Rial, Domingo H. Casadei. Buenos Aires, Argentina; Buenos Aires, Argentina.

P15 **KIDNEY TRANSPLANTATION IN PATIENTS WHO HAVE LEFT VENTRICULAR FRACTION EJECTION LESS THAN 50 PER CENT: FEATURES AND POST-TRANSPLANT OUTCOME. (Abstract #628)**
Jose L. Melchor, Ramon Espinoza, Carmen Gracida, Araceli Ibarra. Mexico, DF, Mexico.

P16 **LONG-TERM EFFECTS OF RENAL TRANSPLANTATION ON LEFT VENTRICLE AND CAROTID PROPERTIES OF HYPERTENSIVE RENAL FAILURE PATIENTS. (Abstract #629)**
Jose J. DeLima, Marcelo L. Vieira, Eduardo M. Krieger, Silvia G. Lage. São Paulo, SP, Brazil.

P17 **FOLLOW-UP OF HEPATITIS C VIRUS (HCV) VIRAL LOAD AFTER RENAL TRANSPLANTATION. ROLE OF IMMUNOSUPPRESSIVE THERAPY. (Abstract #630)**
Josep M. Campistol, Nuria Esforzado, Josep Costa, Honorati Masanja, Josep M. Barrera, Miquel Bruguera. Barcelona, Spain; Barcelona, Spain; Barcelona, Spain; Barcelona, Spain.

P18 **ASSESSMENT OF TUMOR NECROSIS FACTOR-α IN POST-TRANSPLANT OSTEOPORSIS IN FEMALE RENAL TRANSPLANT RECIPIENTS. (Abstract #631)**
Kathleen M. Tornatore, Robert J. Fountaine, Jennie Hom, Robin DiFrancesco, Rocco C. Venuto. Buffalo, NY.

P19 **D+R- DONOR AND RECIPIENT PAIRING PREDICTS SIGNIFICANT RISK FACTORS FOR ADVERSE OUTCOMES DESPITE PROPHYLAXIS WITH IV GANCICLOVIR FOLLOWED BY ORAL ACYCLOVIR. (Abstract #632)**
Kathleen D. Lake, Daniele K. Gelone, Kinnari S. Shah, Julie A. Arndorfer, Jeffrey D. Punch, John C. Magee, Steven M. Rudich, Alan B. Leichtman. Ann Arbor, MI.

P20 **THE EFFICACY OF ORAL VERSUS INTRAVENOUSLY ADMINISTERED GANCICLOVIR IN THE PRE-EMPTIVE TREATMENT OF CYTOMEGALOVIRUS ANTIGENEMIA IN RENAL TRANSPLANT RECIPIENTS. (Abstract #633)**
Kathryn Tinckam, Ognjenka Djurdjev, Gwen Stephens, Paul Ross, David Landsberg.

P21 **CONVERSION TO SIROLIMUS IN RENAL ALLOGRAFT RECIPIENTS WITH CALCINEURIN-INHIBITOR INDUCED NEPHROTOXICITY. (Abstract #634)**
Klemens Budde, F. Diekmann, J. Waiser, D. Dragun, H. H. Neumayer. Berlin, Germany.

P22 **OXIDATIVE STRESS IN KIDNEY TRANSPLANTED PATIENTS WITH CsA OR FK-506 INDUCED HYPERTENSION. EFFECT OF RAMIPRIL. (Abstract #635)**
Lorenzo Calò, Bruno Giacon, Elisa Pagnin, Walter Huber, Augusto Antonello, Andrea Semplicini. Padova, Italy; Bolzano, Italy; Padova, Italy.

P23 **TREATMENT OF POST TRANSPLANT HYPERTENSION BY LAPARASCOPIC BILATERAL NEPHRECTOMY IMPROVES BLOOD PRESSURE CONTROL AND GRAFT FUNCTION. (Abstract #636)**
Lutz Fricke, Christian Doehn, Paolo Fornara, Wolfram Jabs, Juergen Steinhoff. Luebeck; Luebeck; Halle, Germany.

P24 **CREATININE REDUCTION RATIO AND 24-HOUR URINE CREATININE EXCRETION ON DAY TWO: SIMPLE AND OBJECTIVE PARAMETERS TO DEFINE DELAYED GRAFT FUNCTION. (Abstract #637)**
Mahendra V. Govani, Osun Kwon, Ronald S. Filo. Indianapolis, IN; Indianapolis, IN.

Kidney - Immunosuppression A II

P25 **RISK FACTORS FOR TACROLIMUS-INDUCED DIABETES MELLITUS IN ADULT KIDNEY TRANSPLANT RECIPIENTS. (Abstract #638)**
Franz Winklhofer, Dan Murillo, Dennis Diederich. Kansas City, KS.

P26 **DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL OF STEROID WITHDRAWAL IN KIDNEY TRANSPLANT RECIPIENTS WITH A CYCLOSPORINE/ MYCOPHENOLATE REGIMEN-THREE YEAR FOLLOW UP. (Abstract #639)**
James F. Burke, Barbara B. Francos, George C. Francos. Philadelphia, PA.

P27 **IS IT POSSIBLE TO REDUCE CICLOSPORIN EARLY WITH DACLIZUMAB AND MYCOPHENOLATE MOFETIL IN RENAL TRANSPLANT RECIPIENTS? PRELIMINARY RESULTS IN 169 PATIENTS OF A RANDOMIZED MULTICENTRIC TRIAL. (Abstract #640)**
Henri Kreis, Sophie Puget, Georges Mourad, Dominique Durand, Francois Berthoux, Michel Delahousse, Elisabeth Cassuto, Jean-Marc Chalopin, Denis Glotz, Yvon Lebranchu, Luc Potaux, Jean-Louis Touraine, Paul Vialtel, Philippe Wolf, Bruno Moulin, Rajsingh Purgus. Paris, France.

P28 **SIROLIMUS MONOTHERAPY TO OPTIMIZE ACTIVATION INDUCED CELL DEATH (AICD) IN RENAL TRANSPLANT RECIPIENTS FOLLOWING LYMPHOCYTE DEPLETION WITH RABBIT POLYCLONAL ANTITHYMOCYTE GLOBULIN. (Abstract #641)**
John Swanson, Roslyn B. Mannon, Chris Chamberlain, Douglas K. Tadaki, Barbara S. DiMercurio, Scott Batty, Allan D. Kirk. Washington, DC; Bethesda, MD.

P29 **DIFFERENTIAL INFLUENCE OF NEORAL *VERSUS* SANDIMMUNE ON EVEROLIMUS PHARMACOKINETICS: A CLINICALLY RELEVANT DRUG INTERACTION. (Abstract #642)**
J. Kalbag, J. M. Kovarik, J. Figueiredo, M. Rouilly, D. W. Tudor, L. Frazier, C. Rordorf. East Hanover, NJ; Cincinnati, OH.

P30 **RIFAMPIN (A P450 ENZYME-INDUCER) SIGNIFICANTLY INCREASES THE CLEARANCE OF EVEROLIMUS: IMPLICATIONS FOR COADMINISTRATION. (Abstract #643)**
J. M. Kovarik, S. Hartmann, J. Figueiredo, M. Rouilly, D. W. Tudor, A. Port, C. Rordorf. Basel, Switzerland; Nuess, Germany.

P31 **USE OF THERAPEUTIC DRUG MONITORING TO EVALUATE RAPAMYCIN DOSING REGIMEN IN RENAL TRANSPLANT RECIPIENTS (RTR). (Abstract #644)**
Kathleen M. Tornatore, Khalid Bashir, Jennie Hom, Brian M. Murray, Lisa M. Venuto, Rocco C. Venuto. Buffalo, NY.

P32 **IMPROVEMENT IN THE OUTCOME OF ABO-INCOMPATIBLE RENAL TRANSPLANTATION UNDER TACROLIMUS IMMUNOSUPPRESSION. (Abstract #645)**
Kazunari Tanabe, Tadahiko Tokumoto, Hiroaki Shimmura, Shohei Fuchinoue, Satoshi Teraoka, Hiroshi Toma. Tokyo; Tokyo, Japan.

P33 **PHARMACODYNAMIC MONITORING OF MYCOPHENOLATE MOFETIL (MMF) IN RENAL ALLOGRAFT RECIPIENTS. (Abstract #646)**
Klemens Budde, P. Glander, K. P. Braun, J. Waiser, T. Böhler, U. Hahn, H. Röblitz, I. Mai, H. H. Neumayer. Berlin, Germany.

P34 **HARMFUL INTERACTION OF SAINT JOHN'S WORT WITH THE IMMUNOSUPPRESSANT CYCLOSPORIN - A PROSPECTIVE CLINICAL TRIAL. (Abstract #647)**
Ingrid Mai, Klemens Budde, Hagen Krueger, Lars Rothermund, Hans H. Neumayer, Ivar Roots. Berlin, Germany; Berlin, Germany.

P35 **INDUCTION OF ALLOGRAFT TOLERANCE AND ANTITUMOR EFFECTS THROUGH MIXED LMYPHOHEMATOPOIETIC CHIMERISM IN PATIENTS WITH MULTIPLE MYELOMA AND END-STAGE RENAL DISEASE. (Abstract #648)**
Leo H. Buhler, Thomas R. Spitzer, Megan Sykes, David H. Sachs, Francis L. Delmonico, Nina Tolkoff-Rubin, Susan Saidman, Robert Sackstein, Steven L. McAfee, Bimalangshu Dey, Christine Colby, Dina Weymouth, A. Benedict Cosimi. Boston, MA.

Kidney - Immunosuppression B II

P36 **THE EFFECT OF SIROLIMUS (RAPAMUNE®) ON CYCLOSPORINE TROUGH LEVELS: THE POSSIBLE ROLE OF CHANGES IN SERUM LIPIDS. (Abstract #649)**
James T. Burke, Kyle Matschke, James Zimmerman. Paris, France; Philadelphia.

P37 **RAD (EVEROLIMUS) PHARMACOKINETICS IN DE NOVO RENAL TRANSPLANT PATIENTS: DOSE-PROPORTIONAL, STABLE EXPOSURE OVER 6 MONTHS. (Abstract #650)**
B. Kaplan, J. M. Kovarik, H. Tedesco Silva, B. D. Kahan, R. Mendez, L. McMahon, R. Boger, C. Rordorf, on behalf of the RADB251 Study Group. Livingston, NJ; East Hanover, NJ; Sao Paulo; Houston, TX; Los Angeles, CA.

P38 **RAD (EVEROLIMUS) PHARMACOKINETICS ARE UNALTERED WITH FULL-DOSE *VERSUS* REDUCED-DOSE CYCLOSPORINE. (Abstract #651)**
J. Curtis, B. Nashan, J. M. Kovarik, C. Ponticelli, G. Mourad, J. Figueiredo, M. Attinger, R. Boger, C. Rordort, on behalf of the RADB156 Study Group. Birmingham, AL; Hannover, Germany; East Hanover, NJ; Milan, Italy; Monpellier, France.

P39 **SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY IS PROMISING IN PATIENTS RECEIVING KIDNEYS FROM OLDER DONORS. (Abstract #652) ♦**
Josep M. Grinyo, Yves Vanrenterghem, Brian Hutchinson, Marco Castagneto, Dominique Durand, Gian B. Sorba, Rainer Oberbauer, Julianna Mannion, the Tri-continental Renal Transplant Study Group. Barcelona, Spain.

P40 **LUPUS NEPHRITIS IN AFRICAN AMERICAN KIDNEY TRANSPLANT RECIPIENTS CARRIES A POOR PROGNOSIS. (Abstract #653) ♦**
Lee Erbe, Jeremy Kirtz, Jeffery Rogers, Angello Lin, G. Mark Baillie, P.R. Rajagopalan, Kenneth D. Chavin, Prabhakar K. Baliga. Charleston, SC.

P41 **SAFETY AND PHARMACODYNAMICS OF MULTIPLE DOSES OF FTY720 IN STABLE RENAL TRANSPLANT RECIPIENTS. (Abstract #654)**
Barry Kahan, Lawrence Chodoff, Alan Leichtman, Janet Karlix, Ronald Ferguson, Shamkant Mulgaonkar, Thomas Gonwa, Robert Schmouder, Milbhor D'Silva, Jennifer Dehlinger, Tomasz Sablinski. Houston; East Hanover; Ann Arbor; Gainesville; Columbus; Livingston; Dallas; Horsham, United Kingdom.

P42 **TWO DOSE DACLIZUMAB (ZENAPAX) IS SAFE AND EFFECTIVE INDUCTION THERAPY IN ADULT RENAL TRANSPLANTATION. (Abstract #655)**
Lutf U. Rehman, Ralph C. Atkinson, Peter J. Minich. Nashville, TN.

P43 **RANDOMIZED CONVERSION FROM CYCLOSPORINE TO TACROLIMUS IN RENAL TRANSPLANT PATIENTS: IMPROVEMENT IN RENAL FUNCTION AND CARDIOVASCULAR RISK FACTORS. (Abstract #656)**
Marika A. Artz, Gerry Ligtenberg, Joke I. Roodnat, Maarten H. Christiaans, Luuk B. Hilbrands. Nijmegen, The Netherlands.

P44 **FK506 AND THE RAPAMYCIN ANALOGUE, SDZ RAD DO NOT HAVE ANTAGONISTIC EFFECTS ON LYMPHOCYTES FROM RENAL TRANSPLANT PATIENTS. (Abstract #657)**
Michael P. Delaney, Robert M. Higgins, Alan G. Morris.

P45 **QUANTIFICATION OF INTERLEUKIN-2 mRNA EXPRESSION BY REAL-TIME PCR IN PATIENTS TREATED WITH NEORAL UNDERGOING LIVING-KIDNEY TRANSPLANTATION. (Abstract #658)**
Michael Muller-Steinhardt, Christoph Hartel, Holger Kirchner, Lutz Fricke. Lubeck, Germany; Lubeck, Germany.

P46 **CYTOTOXIC T CELL GENE EXPRESSION PLUS TRAIL IN 1 MONTH PROTOCOL BIOPSIES PREDICTS EFFICACY OF EARLY STEROID WITHDRAWAL. (Abstract #659)**
Kosunarty Fa, Qun Lu, Kode Ravi, Elizabeth Ferry, Billie Fyfe, Mark Laftavi, Mysore S. Anil Kumar, Oleh Pankewycz. Philadelphia, PA.

P47 **IMPACT OF MULTIPLE CLINICAL STUDIES WITH NEW IMMUNOSUPPRESSIVE AGENTS ON THE OUTCOME OF RENAL TRANSPLANTATION AT A SINGLE CENTER. (Abstract #660)**
Paolo Rigotti, Nicola Baldan, Roberto Cadrobbi, Giacomo Sarzo, Lucrezia Furian, Francesco Marchini, Ermanno Ancona. Padova, Italy; Padova, Italy.

Kidney - Pediatrics, Recurrent Disease II

P48 **LAPAROSCOPIC DONOR NEPHRECTOMY FOR ADULTS FOR PEDIATRIC LIVING DONOR KIDNEY TRANSPLANTATION. (Abstract #661)**
Stephen F. Shafizadeh, Elizabeth Ashcraft, G. Mark Baillie, Jeffrey Rogers, Angello Lin, P.R. Rajagopalan, Prabhakar K. Baliga, Valerie Panzarino, John Orak, Louis Kavoussi, Barbara Fivush, Paul Colombani, Lloyd Ratner, Kenneth D. Chavin. Charleston, SC; Charleston, SC; Baltimore, MD.

P49 **LIMITED SURGICAL INTERVENTIONS IN POSTERIOR URETHRAL VALVES CAN LEAD TO GOOD GRAFT OUTCOMES IN PEDIATRIC RENAL TRANSPLANTATION. (Abstract #662)**
Leah Bartsch, Minnie Sarwal, Pamela Orlandi, Peter Yorgin, Kevin Lemley, Amir Belson, Steve Alexander, Oscar Salvatierra. Stanford, CA.

P50 **Abstract #663 WITHDRAWN**

P51 **RENAL TRANSPLANTATION IN SMALL CHILDREN: DONOR SELECTION AND TECHNICAL CONSIDERATION. (Abstract #664)**
Michael Neipp, Gisela Offner, Rainer Lueck, Jochen H. Ehrich, Juergen Strehlau, Hans J. Schlitt, Juergen Klempnauer, Bjoern Nashan. Hannover, Germany.

P52 **UNEXPECTEDLY HIGH INCIDENCE OF POST TRANSPLANT ANEMIA IN PEDIATRIC RENAL TRANSPLANT RECIPIENTS. (Abstract #665)**
Peter D. Yorgin, Amir Belson, John Oehlert, Jaime Sanchez, Amira Y. Al-Uzri, Dan Bloch, Oscar Salvatierra, Steven R. Alexander. Stanford, CA; Charlottesville, VA; Stanford, CA; Stanford, CA.

Kidney - Preservation, Donation/Allocation, Economics/ Public Policy, Surgical Techniques, and Other II

P53 **MINIMALLY INVASIVE OPEN DONOR NEPHRECTOMY (MIODN). (Abstract #666)**
Ignacio T. Castillon-Vela, Robert Naraghi, Paul Asai, Sali Aswad, Umakant Khetan, Alexandre E. Da Silva, Rafael G. Mendez, Roberto Mendez, Hamid Shidban. Los Angeles, CA.

P54 **INCREASED IMMUNOGENICITY AND CAUSE OF GRAFT LOSS OF OLD DONOR KIDNEYS. (Abstract #667)**
Johan W. De Fijter, Marko J.K. Mallat, Ilias I.N. Doxiadis, Jan Ringers, Frits R. Rosendaal, Frans H.J. Claas, Leendert C. Paul. Leiden, The Netherlands; Leiden, The Netherlands; Leiden, The Netherlands; Leiden, The Netherlands.

P55 **TACROLIMUS VERSUS CYCLOSPORINE IN KIDNEY TRANSPLANTATION: FIVE-YEAR GRAFT SURVIVAL AND RENAL FUNCTION. (Abstract #668)**
John D. Pirsch, Darby Thompson, FK506 Kidney Transplant Study Group. Madison, WI; Potomac, MD.

P56 **THE URINE PROTEIN TO CREATININE RATIO AS A PREDICTOR OF 24-HOUR URINE PROTEIN EXCRETION IN RENAL TRANSPLANT PATIENTS. (Abstract #669)**
Shirley Tong, Claudio Rigatto, David N. Rush, Peter Nickerson, John R. Jeffery.

P57 **IN VIVO IL-10 PRODUCTION BY PBMC FROM LONG-TERM KIDNEY TRANSPLANT RECIPIENTS WITH EXCELLENT GRAFT FUNCTION. (Abstract #670) ♦**
Hugo Guzman-Rodriguez, Claudia de Leo, Eduardo Mancilla, Ricardo Correa-Rotter, Alfredo Chew-Wong, Josefina Alberu.

P58 **TGFβ EXPRESSION IN AN EXPERIMENTAL MODEL OF ISCHEMIA-REPERFUSION SYNDROME IN KIDNEY TRANSPLANTATION. (Abstract #671)**
Sergio Lario, Diego F.R. Mendes, Monica Bescos, Pilar Luque, Pablo Inigo, Antonio Alcaraz, Josep M. Campistol. Barcelona, Spain; Barcelona, Spain.

P59 **INFLUENCE OF LEARNING CURVE, SURGEON TYPE, AND TRAINING TYPE ON THE RESULTS OF LAPAROSCOPIC DONOR NEPHRECTOMY AT NINE TRANSPLANT CENTERS. (Abstract #672)**
Joseph F. Buell, Alan Koffron, Michael J. Hanaway, Atsushi Yoshida, David S. Bruce, Paul C. Kuo, Joseph Leventhal, Juan Arenas, Michael Edye, E. Steve Woodle, Stephen T. Bartlett, Eugene Cho, Mark Johnson. Cincinnati, OH.

P60 **1200 LAPAROSCOPIC LIVING DONOR NEPHRECTOMIES: ANALYSIS OF GRAFT LOSS IN THE FIRST YEAR POST TRANSPLANT. (Abstract #673)**
Joseph F. Buell, Mark Johnson, Alan Koffron, Fred Faneli, Stephen Jacobs, David S. Bruce, Atshui Yoshida, Kenneth A. Newell, Juan Arenas, E. Steve Woodle, Joseph Leventhal, Eugene Cho, Michael Edye, Paul C. Kuo. Cincinnati, OH.

P61 **A DONOR SELECTION ALGORITHM FOR NON-HEART-BEATING DONORS ALLOWS HIGH RETRIEVAL RATES AND EXCELLENT GRAFT SURVIVAL. (Abstract #674)**
Joseph F. Buell, Meredith J. Aull, Rino Munda, J. W. Alexander, James Fidler, Horacio Rilo, Douglas Hanto, Agnes Lo, Michael Hanaway, Dave Lewis, M. Roy First, E. Steve Woodle. Cincinnati, OH; Cincinnati, OH.

P62 **QUALITY OF LIFE IN SIROLIMUS-TREATED KIDNEY TRANSPLANT PATIENTS AFTER CYCLOSPORINE ELIMINATION. (Abstract #675)**
Josette Eris, Fang Wang, J. Chen, D. Revicki, the Sirolimus Tri-continental Renal Transplant Study Group. Sydney, Australia; Radnor, PA; Bethesda, MD.

P63 **TGF-BETA 1 GENE POLYMORPHISM MAY BE ASSOCIATED WITH POST-TRANSPLANTATION LYMPHOPROLIFERATIVE DISEASE (PTLD). (Abstract #676)**
Kazuo Mizutani, Brian Hardy, Robert Mendez, James Cicciarelli, Yuichi Iwaki. Los Angeles, CA; Los Angeles, CA.

P64 **LIVE KIDNEY DONATION: FACTORS INFLUENCING INDIVIDUALS' WILLINGNESS TO DONATE. (Abstract #677)**
L. Ebony Boulware, Lloyd E. Ratner, Julie Ann Sosa, Alexander H. Tu, Satish Nagula, Neil R. Powe. Baltimore, MD; Baltimore, MD.

P65 **EFFECTS OF RIBAVIRIN ON CHRONIC HEPATITIS C VIRUS (HCV) INFECTION IN RENAL TRANSPLANT (RT) PATIENTS. (Abstract #678)**
Lionel Rostaing, O. Cointault, D. Ribes, K. Sandres, N. Kamar, D. Durand, J. Izopet.

P66 **PROMOTING EMPLOYMENT FOLLOWING RENAL TRANSPLANTATION. (Abstract #679)**
Lisa R. Raiz, Elizabeth A. Davies, Ronald M. Ferguson. Athens, OH; Columbus, OH.

P67 **LAPAROSCOPIC LIVE DONOR NEPHRECTOMY: GLOBAL STATUS AT THE MILLENIUM. (Abstract #680)**
Lloyd E. Ratner, Vladimir Sinkov, Robert A. Montgomery, Karen Fox-Talbot, Henkie P. Tan, Matthew Cooper, Louis R. Kavoussi. Baltimore, MD.

P68 **IMPACT OF DIRECT PATIENT CARE CLINICAL PHARMACY SERVICES ON RENAL TRANSPLANT PATIENTS COMPLIANCE WITH IMMUNOSUPPRESSIVE MEDICATIONS. (Abstract #681)**
Marie A. Chisholm, Leslie J. Vollenweider, Laura L. Mulloy, Muralidharan Jagadeesan, Joseph T. DiPiro. Athens, GA; Augusta, GA; Augusta, GA.

P69 **TECHNIQUE OF RIGHT LAPAROSCOPIC DONOR NEPHRECTOMY: A SINGLE CENTER EXPERIENCE. (Abstract #682)**
Mark W. Johnson, Kenneth Andreoni, Lynn McCoy, Lisa Scott, Suzanne Thomas, David A. Gerber, Jeffrey H. Fair. Chapel Hill, NC.

P70 **OLD KIDNEY FOR OLD RECIPIENT, IS IT JUSTIFIED? (Abstract #683)**
Reza M. Laftavi, Ravi Kode, Kim D. Phillips, Debbie Sierka, Mysore A. Kumar, Oleh G. Pankewycz. Philadelphia, PA; Philadelphia, PA.

P71 **THE ASSESSMENT OF RENAL ISCHAEMIC INJURY DURING WARM EX-VIVO PERFUSION. (Abstract #684)**
Matthew S. Metcalfe, Wolfgang Rohlke, Michael L. Nicholson. Leicester, United Kingdom; Ulm, Germany.

P72 **COVARIATES INFLUENCING OUTCOMES OF OLD CADAVERIC DONOR KIDNEY TRANSPLANTS. (Abstract #685)**
Elisabetta Bertoni, Alberto Rosati, Lorenzo Di Maria, Maria Zanazzi, Luciano Moscarelli, Maurizio Salvadori. Florence, Italy.

P73 **PULSATILE PRESERVATION IMPROVES TRANSPLANT UTILIZATION OF EXPANDED CRITERIA DONOR KIDNEYS. (Abstract #686)**
Maximilian M. Polyak, Richard D. Hasz, Howard M. Nathan. Philadelphia, PA.

P74 **PERFORMING LAPAROSCOPIC DONOR NEPHRECTOMY IN ALL LIVING DONORS: INITIAL EXPERIENCE INCLUDING OBESE DONORS AND RIGHT-SIDED KIDNEYS. (Abstract #687)**
Mikel Prieto, George K. Chow, James M. Gloor, Humberto E. Bohorquez, Fawad Qureshi, Edwina Baskin-Bey, Sylvester Sterioff, Scott L. Nyberg, Walter K. Kremers, Mark D. Stegall. Rochester, MN.

P75 **PREOPERATIVE EVALUATION OF LIVING DONOR RENAL ANATOMY WITH CT ANGIOGRAPHY/ UROGRAPHY AND 3-D RECONSTRUCTION. (Abstract #688)**
Humberto E. Bohorquez, Mikel Prieto, Terri J. Vrtiska, Andrew LeRoy, Bernard F. King, Patrick G. Dean, Ari J. Cohen, Ainitze Ibarzabal, Mark D. Stegall. Rochester, MN; Rochester, MN.

P76 **HAND ASSISTED TECHNIQUE FACILITATES A TRANSITION FROM OPEN TO FULLY LAPAROSCOPIC DONOR NEPHRECTOMY. (Abstract #689)**
J. DelPizzo, E. Sosa, S. Kapur, W. T. Stubenbord, M. Stifleman, M. Kinkhabwala. New York, NY.

Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics II

P77 **NEURODEVELOPMENTAL STATUS IN VERY YOUNG PEDIATRIC LIVER AND SMALL BOWEL RECIPIENTS. (Abstract #690)**
Deborah M. Thevenin, Christina Lopez-Hernandez, Stephanie E. Eischen, Tomoaki Kato, Andreas G. Tzakis. Miami, FL; Miami, FL.

P78 **EARLY AND LATE GRAFT LOSS AFTER PEDIATRIC LIVER TRANSPLANTATION. (Abstract #691)**
Egbert Sieders, Paul M.J.G. Peeters, Elisabeth M. TenVergert, Koert P. de Jong, Robert J. Porte, Jan H. Zwaveling, Charles M.A. Bijleveld, Annette S.H. Gouw, Maarten J.H. Slooff. Groningen, The Netherlands.

P79 **HISTOPATHOLOGY IN LONG-TERM SURVIVORS OF PEDIATRIC LIVER TRANSPLANTATION. (Abstract #692)**
Jeffrey B. Brown, Hector Melin-Aldana, Julie Chesterton, Peter F. Whitington, Estella M. Alonso. Chicago, IL; Chicago, IL.

P80 **LIMITS OF MMF IN LIVER TRANSPLANTATION - DISCONTINUATION OF A CYA WITHDRAWAL TRIAL. (Abstract #693)**
Gabriela A. Berlakovich, Thomas Windhager, Peter Wamser, Thomas Soliman, Fritz Wrba, Rudolf Steininger, Ferdinand Muehlbacher. Vienna, Austria; Vienna, Austria.

P81 **CONVERSION TO C2 CYCLOSPORINE MONITORING USING NEORAL IMMUNOSUPPRESSION IN MAINTENANCE LIVER TRANSPLANT PATIENT: IMPROVEMENT IN RENAL FUNCTION AND HYPERTENSION. (Abstract #694)**
Gary A. Levy, Catherine O'Grady, Leslie B. Lilly, David Grant, Nigel Girgrah, Paul D. Greig. Toronto, ON, Canada.

P82 **C2 MONITORING IN LIVER TRANSPLANTATION WITH NEORAL IMMUNOSUPPRESSION: EFFECT OF ACHIEVING C2 TARGET EARLY ON EFFICACY AND SAFETY. (Abstract #695)**
Gary A. Levy, Catherine O'Grady, Leslie B. Lilly, David Grant, Nigel Girgrah, Paul D. Greig. Toronto, ON, Canada.

P83 **A ONE-YEAR, MULTICENTER, RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND, PARALLEL GROUP, DOSE-FINDING STUDY TO EVALUATE THE SAFETY, TOLERABILITY AND PHARMACOKINETICS OF RAD IN DE NOVO LIVER TRANSPLANT RECIPIENTS. (Abstract #696)**
RAD International Liver Study Group.

P84 **SIROLIMUS IMMUNOSUPPRESSION FOR LIVER TRANSPLANTATION IMPROVES RENAL DYSFUNCTION. (Abstract #697)♦**
George J. Chang, Harish D. Mahanty, David Quan, Chris E. Freise, Peter G. Stock, Nancy L. Ascher, John P. Roberts, Ryutaro Hirose. San Francisco, CA; San Francisco, CA.

P85 **IMPACT OF DRUG WITHDRAWAL ON GROWTH IN CHILDREN AFTER LIVER TRANSPLANTATION(LTx). (Abstract #698)**
George V. Mazariegos, Alcides A. Salzedas, Amy Smith, Cindy Baird, Kathy Iurlano, Jorge D. Reyes. Pittsburgh, PA.

P86 **THYMOGLOBULIN (T) IN THE MANAGEMENT OF STEROID RESISTANT ACUTE CELLULAR REJECTION (SRACR) IN CHILDREN. (Abstract #699)**
George V. Mazariegos, William McGhee, Rakesh Sindhi, Jorge Reyes.

P87 **GERIATRIC ORTHOTOPIC LIVER TRANSPLANTATION (OLT): A RETROSPECTIVE OUTCOMES REPORT FROM A LARGE SINGLE CENTER REVIEW INVOLVING PATIENTS GREATER THAN 70 YEARS OF AGE. (Abstract #700)♦**
G. Tzimas, G. W. Neff, O. Hung, D. Weppler, D. Levi, S. Nishida, J. Tector, L. Kravetz, J. R. Nery, T. Kato, C. O'Brien, K. R. Reddy, E. R. Schiff, A. G. Tzakis. Miami, FL; Miami, FL.

P88 **PREGNANCY OUTCOMES IN FEMALE LIVER TRANSPLANT RECIPIENTS: TEN YEAR EXPERIENCE FROM THE NATIONAL TRANSPLANTATION PREGNANCY REGISTRY (NTPR). (Abstract #701)**
Vincent T. Armenti, Lisa A. Coscia, Stephen R. Dunn, Lydia Z. Philips, Carolyn H. McGrory, John S. Radomski, Michael J. Moritz. Philadelphia, PA; Philadelphia, PA.

Liver - Infections, Complications, Recurrent Disease, Surgical Techniques II

P89 **TREATMENT OF RECURRENT HEPATITIS C INFECTION WITH RIBAVIRIN MONOTHERAPY AFTER ORTHOTOPIC LIVER TRANSPLANT. (Abstract #702)**
Gordon R. Ingle, Curtis D. Holt, Leonard Goldstein, Gregg Kunder, Ronald W. Busuttil. Los Angeles, CA.

P90 **APPARENT SLOWING OF HEPATITIS C RECURRENCE AND PROGRESSION AFTER LIVER TRANSPLANTATION USING VERY EARLY (14d) STEROID WITHDRAWAL. (Abstract #703)♦**
Nancy Stolpman, Janet Stephens, Thomas Trouillot, James Trotter, Marcelo Kugelmas, Michael Wachs, Thomas Bak, Tracy Steinberg, Igal Kam, Gregory Everson. Denver, CO.

P91 **TREATMENT OF DECOMPENSATED CIRRHOTICS WITH INTERFERON + RIBAVIRIN MAY REDUCE RECURRENCE OF HEPATITIS C AFTER LIVER TRANSPLANTATION. (Abstract #704)**
Gregory T. Everson, Arthur Halprin, Thomas E. Trouillot, James F. Trotter, Marcelo Kugelmas, Justin Skilbred, Christine Tomasi, Carol McKinley, Barb Fey, Cathy Ray, Jim Epp. Denver, CO.

P92 **SIDE TO SIDE CAVO-CAVAL ANASTOMOSIS, END TO SIDE DONOR CELIAC (CA) TO RECIPIENT SPLENIC ARTERY (SA) ANASTOMOSIS, AND DUCT TO DUCT ANASTOMOSIS WITHOUT T-TUBE MAKE LIVER TRANSPLANTATION (OLT) SIMPLER TO PERFORM, REDUCE OPERATING TIME, AND ARE PHYSIOLOGICALLY SOUNDER. (Abstract #705)**
Hadar J. Merhav, Richard Nakasche. Tel Aviv, Israel.

P93 **ANASTOMOSIS OF THE DONOR RIGHT REPLACED HEPATIC ARTERY (RRHA) TO THE DONOR GASTRO DUODENAL ARTERY (GDA) IS SAFE, BETTER SIZE MATCHED AND POSITIONALLY SUPERIOR TO OTHER RRHA RECONSTRUCTION TECHNIQUES. (Abstract #706)**
Hadar J. Merhav, Richard Nakasche. Tel Aviv, Israel.

P94 **PERMISSIVE LIMIT OF GRAFT SIZE FOR SUCCESSFUL LIVING DONOR LIVER TRANSPLANTATION IN ADULTS. (Abstract #707)**
Hiroyuki Furukawa, Tsuyoshi Shimamura, Maeng Bong Jin, Toshiya Kamiyama, Boonchoo Sirichindakul, Michiaki Matsushita, Satoru Todo.

P95 **INTRAOPERATIVE INDOCYANINE GREEN KINETICS PREDICT POSTOPERATIVE GRAFT FUNCTION AFTER ORTHOTOPIC LIVER TRANSPLANTATION FOR CHRONIC HEPATIC FAILURE. (Abstract #708)**
Hubert Hetz, Walter Ploechl, Gabriela A. Berlakovich, Claus G. Krenn, Heinz Steltzer. Vienna, Austria; Vienna, Austria.

P96 **ARTERIAL AND PORTAL VENOUS RECONSTRUCTION AFTER *EX VIVO* SPLIT LIVER TRANSPLANTATION. (Abstract #709)♦**
Huda M. Noujaim, Brigitte B. Gunson, Daniel Candinas, David A. Mayer, John A.C. Buckels, Paul McMaster, John De Ville De Goyet, Darius F. Mirza. Birmingham, United Kingdom; Birmingham, United Kingdom.

P97 **THE ORIGIN OF HEPATITIS C VIRUS REINFECTING LIVER GRAFT: SERUM VERSUS PERIPHERAL BLOOD MONONUCLEAR CELLS-DERIVED VIRUS. (Abstract #710)**
H. Vargas, J. Wilkinson, M. Radkowski, V. Balan, D. D. Douglas, M. E. Harrison, D. C. Mulligan, A. Moss, T. Laskus, J. Rakela. Scottsdale, AZ.

P98 **LONGITUDINAL ANALYSIS OF EPITOPE-ENCODING REGIONS OF HEPATITIS C VIRUS (HCV) FOLLOWING LIVER TRANSPLANTATION. (Abstract #711)**
Hugo R. Rosen, John Rabkin, Sunwen Chou. Portland, OR; Portland, OR.

P99 **INCREASED INCIDENCE OF NEPHROLITHIASIS IN LIVER TRANSPLANT RECIPIENTS WITH CHRONIC HEPATITIS C. (Abstract #712)**
Debbie F. Dick, George E. Loss, Satheesh P. Nair, Andrew L. Mason, James D. Eason. New Orleans, LA.

P100 **IS CHOLEDOCHOCHOLEDOCHOSTOMY WITH T TUBE STILL A SAFE PROCEDURE IN LIVER TRANSPLANTATION? (Abstract #713)**
Javier C. Lendoire, Jose Saul, Gustavo Bianco, Fernando Duek, Gabriel Illanes, Carlos Quarin, Oscar Imventarza. Buenos Aires, Argentina.

P101 **LIVE DONOR ADULT RIGHT LOBE LIVER TRANSPLANTATION-LESSONS LEARNED FROM 49 TRANSPLANTS. (Abstract #714)**
John M. Ham, Robert A. Fisher, Martha Behnke, Adrian H. Cotterell, Sherfield Dawson, III, Amadeo Marcos, Marc P. Posner. Richmond, VA; Rochester, NY.

P102 **CHANGES IN BONE DENSITY IN LIVER TRANSPLANT PATIENTS. (Abstract #715)**
Khondker K. Islam, Steven D. Creech, Rana P. Sokhi, Ravi K. Kondaveeti, James M. Harig, David H. Van Thiel. Maywood, IL; Maywood, IL.

P103 **INCIDENCE AND IMPACT OF HEPATIC ARTERY THROMBOSIS IN LIVER TRANSPLANTATION. (Abstract #716)**
Barbara K. Brooks, Norman G. Diamond, Linda W. Jennings, Edmund Q. Sanchez, Shigeru Marubashi, Brian M. Gogel, Robert M. Goldstein, Ernesto P. Molmenti, Carlos G. Fasola, Thomas A. Gonwa, Goran B. Klintmalm, Marlon F. Levy. Dallas, TX.

P104 **QUANTITATION OF HBV DNA IN LIVER GRAFT FROM HBSAG NEGATIVE, HBCAB POSITIVE DONOR. (Abstract #717)**
Masahiro Shinoda, Go Wakabayashi, Motohide Shimazu, Ken Hoshino, Minoru Tanabe, Yasuhide Morikawa, Masaki Kitajima. Tokyo, Japan.

P105 **DE NOVO LIVER CANCER AFTER LIVER TRANSPLANTATION. (Abstract #718)**
Michael Hanaway, Joseph Buell, Agnes Lo, Jennifer Trofe, Tom Beebe, E. Steve Woodle. Cincinnati, OH.

Liver - Preservation, Economics/Public Policy, Donation Allocation, Other II

P106 **INFLUENCE OF INTRAOPERATIVE STEROIDS ON ISCHEMIA/REPERFUSION INJURY (IRI) AFTER LIVER TRANSPLANTATION (LTX). (Abstract #719)**
J. Pirenne, F. Van Gelder, T. Koshiba, R. Aerts, S. Kimpen, E. Benedetti, M. Schetz, E. Vandermeersch, M. Verhaegen, P. Lauwers, J. Fevery, F. Nevens, T. Roskams. Leuven, Belgium.

P107 **LIVING DONOR LIVER TRANSPLANTATION IN HEPATITIS C PATIENTS: SHORT-TERM RESULTS COMPARED TO CADAVERIC TRANSPLANTATION. (Abstract #720)**
James F. Trotter, Thomas Schiano, Michael Wachs, Leona Kim-Schluger, Thomas Bak, Marcelo Kugelmas, Thomas Trouillot, Gregory T. Everson, Charles Miller, Igal Kam. Denver, CO; New York, NY.

P108 **ADULT-TO-ADULT LIVING DONOR LIVER TRANSPLANTATION USING RIGHT LOBE: SINGLE CENTER EXPERIENCE. (Abstract #721)**
Jeff H. Fair, Mark W. Johnson, David A. Gerber, Kenneth A. Andreoni, Sue Weeks, Michael Fried, Steve Zacks, Amy Strzakla, Roshan Shrestha. Chapel Hill, NC; Chapel Hill, NC; Chapel Hill, NC.

P109 **CLINICAL AND ECONOMIC IMPACT OF LATE STEROID WITHDRAWAL IN A STABLE LIVER TRANSPLANT POPULATION. (Abstract #722)**
Eric W. Mueller, Jill E. Martin, Kelly S. Rolfes, Paula M. Auble, Jaime Aranda-Michel, Fredrick L. Weber, Douglas W. Hanto. Cincinnati, OH.

P110 **INCIDENCE OF BONE COMPLICATIONS AND ASSOCIATED RESOURCE UTILIZATION IN LIVER TRANSPLANT PATIENTS. (Abstract #723)**
Alicia R. Large, Jill E. Martin, Jaime Aranda-Michel, Douglas W. Hanto, James F. Whiting. Cincinnati, OH; Portland, ME.

P111 **PHYSICAL FITNESS AND HEALTH RELATED QUALITY OF LIFE AMONG LIVER TRANSPLANT RECIPIENTS. (Abstract #724)**
Joanne B. Krasnoff, Patricia Painter, Norah A. Terrault, Nancy L. Ascher, John P. Roberts.

P112 **PREDICTING THE PROBABILITY OF PROGRESSION FREE SURVIVAL IN PATIENTS WITH SMALL HEPATOCELLULAR CARCINOMA. (Abstract #725)**
Steve J. Cheng, Richard B. Freeman, Jr., John B. Wong. Boston, MA.

P113 **CHARGE COMPARISONS OF LIVING DONOR RIGHT LOBE LIVER TRANSPLANTATION VERSUS CADAVER DONOR LIVER TRANSPLANTATION. (Abstract #726)**
John M. Ham, Robert A. Fisher, Martha Behnke, Paul Descutner, Adrian H. Cotterell, Sherfield Dawson, III, Marc P. Posner. Richmond, VA.

P114 **FUNCTIONAL STATUS OF DONORS FOLLOWING LIVING DONOR LIVER TRANSPLANTATION. (Abstract #727)**
Kimberly L. Beavers, Jeffrey H. Fair, Roshan Shrestha. Chapel Hill, NC; Chapel Hill, NC.

P115 **EVALUATION OF POTENTIAL LIVING DONORS FOR LIVING DONOR LIVER TRANSPLANTATION. (Abstract #728)**
Kimberly L. Beavers, Michael W. Fried, Steven L. Zacks, Jeffrey H. Fair, Mark W. Johnson, David A. Gerber, Kenneth A. Andreoni, Amy L. Strzalka, Roshan Shrestha. Chapel Hill, NC; Chapel Hill, NC.

P116 **DEVELOPMENT OF EXSANGUINEOUS METABOLIC SUPPORT FOR THE LIVER. (Abstract #729)**
Lauren Brasile, Bart Stubenitsky, Maurits Booster, Gauke Kootstra. Maastricht, The Netherlands; Chicago, IL; Schenectady, NY.

P117 **TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT (TIPS) AND LIVER TRANSPLANTATION. (Abstract #730)**
M. Hosein Shokouh-Amiri, A. Osama Gaber, Santiago R. Vera, Claudio Tombazzi, Nosratollah Nezakatgoo, Hani P. Grewal, Robert J. Stratta, A. Tarik Kızılisik, Bradford Waters, Caroline A. Riely, Pamela Flick, Robert E. Gold, Trine Nymann, Wagdy A. Bagous. Memphis, TN.

P118 **RIGHT LIVING DONOR LIVER TRANSPLANTATION FOR EXTENDED INDICATIONS: A NEW CHANCE FOR NEGLECTED PATIENTS OR A WASTE OF GRAFTS? (Abstract #731)**
Massimo Malago', Giuliano Testa, Hauke Lang, Martin Hertl, Christoph E. Broelsch.

Pancreas and Islets - All Topics II

P119 **THE FATE OF PANCREASES DECLINED AND TRANSPLANTED OUT OF A LOCAL ORGAN PROCUREMENT ORGANIZATION. (Abstract #732)**
David B. Leeser, John Abrams, Richard Hasz, Howard Nathan, Clarence E. Foster. Philadelphia, PA; Philadelphia, PA.

P120 **CAN GRAFT LOSS FROM PANCREAS TRANSPLANT THROMBOSIS BE PREVENTED? THROMBOELASTOGRAM DIRECTED ANTICOAGULATION FOR SIMULTANEOUS PANCREAS/KIDNEY HYPERCOAGULABLE STATE. (Abstract #733)**
George W. Burke, Gaetano Ciancio, Jose Figueiro, Rafael Buigas, David Roth, Warren Kupin, Joshua Miller. Miami, FL.

P121 **PORTAL AND SYSTEMIC INSULIN BEHAVIOUR DURING IV GLUCOSE CHALLENGE FOLLOWING PARATOPIC AND HETEROTOPIC TOTAL PANCREATIC GRAFT IN RATS. (Abstract #734)**
Negib Elian, Franck Zinzindohoue, Catherine Drevillon, Danielle Bailbe, Françoise Carnot, Paul-Henri Cugnenc, Jean-Jacques Altman. Paris, France; Paris, France.

P122 **OUTCOME OF SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION IN AFRICAN AMERICANS WITH TYPE 1 DIABETES. (Abstract #735)**
Jeffrey Rogers, Vicki D. Graham, Kenneth D. Chavin, Prabhakar K. Baliga, Angello Lin, G. Mark Baillie, Elizabeth E. Ashcraft, P.R. Rajagopalan. Charleston, SC; Charleston, SC.

P123 **A COMPARISON OF READMISSION RATES OF SIMULTANEOUS KIDNEY PANCREAS TRANSPLANT RECIPIENTS RECIEVING INDUCTION WITH MUROMONAB-CD3 VERSUS DACLIZUMAB. (Abstract #736)**
Mariano S. Dy-Liacco, Bradley H. Collins, David W. Butterly, Ralph R. Bollinger, Janet E. Tuttle-Newhall. Durham, NC.

P124 **A LOW RATE OF ACUTE REJECTION IN SIMULTANEOUS PANCREAS-KIDNEY TRANSPLANT RECIPIENTS RECEIVING DACLIZUMAB INDUCTION. (Abstract #737)**
Muralikrishna S. Golconda, Stephen C. Rayhill, Lawrence G. Hunsicker. Jacksonville, FL; Iowa City, IA; Iowa City, IA.

P125 **ADDITIONAL SHORT-TERM (5HOURS) PRESERVATION OF PANCREAS BY THE TWO-LAYER (UW SOLUTION/PERFLUOROCARBON+OXYGEN) METHOD PRIOR TO ISLET ISOLATION IMPROVED QUANTITY AND QUALITY OF ISOLATED NON-HUMAN PRIMATE ISLETS. (Abstract #738)**
Shinichi Matsumoto, Ian Sweet, Douglas M. Strong, Yoshikazu Kuroda, R. Brian Stevens. Seattle, WA; Seattle, WA; Seattle, WA; Kobe, Hyogo, Japan.

P126 ISCHEMICALLY DAMAGED HUMAN PANCREASES (Px) CAN BE RESUSCITATED BY THE TWO-LAYER METHOD BEFORE ISLET ISOLATION: IMPLICATIONS FOR CLINICAL ISLET TRANSPLANTATION. (Abstract #739)
R. Brian Stevens, Shinichi Matsumoto, Owen Lawrence, Theodore H. Rigley, Christopher L. Marsh. Seattle, WA; Seattle, WA.

P127 PANCREASE STORAGE WITH THE TWO-LAYER METHOD (TLM) DRAMATICALLY EXTENDS THE ORGAN PRESERVATION PERIOD BEFORE ISOLATION OF HUMAN ISLETS. (Abstract #740)
R. Brian Stevens, Sabrina A. Qualley, Theodore H. Rigley, Christopher L. Marsh, Shinichi Matsumoto.

P128 CYTOKINE GENE POLYMORPHISMS (CGP) IN "PANCREAS TRANSPLANT ALONE" (PTA) RECIPIENTS - CAN THEY PREDICT OUTCOME? (Abstract #741)
Raja Kandaswamy, Abhi Humar, Angelika Gruessner, Rainer W. Gruessner, Bernhard Hering, Arthur J. Matas, David E.R. Sutherland. Minneapolis, MN.

P129 KIDNEY(KD) AND KIDNEY-PANCREAS(KP) TRANSPLANTS IN JEHOVAH'S WITNESSES (JW) - A SINGLE-CENTER EXPERIENCE WITH 50 TRANSPLANTS. (Abstract #742)
Thiagarajan Ramcharan, Arthur Matas, Rainer W. Gruessner, David E.R. Sutherland, Raja Kandaswamy. Minneapolis, MN.

Heart/Lung - All Topics II

P130 MYCOPHENOLIC ACID AND AZATHIOPRINE DO NOT DIFFER IN THEIR INFLUENCE ON THE SURFACE GLYCOSYLATION OF LEUKOCYTES. (Abstract #743)
Guenter Weigel, Andrea Griesmacher, Peter Bertalanffy, Ali Karimi, Michael Grimm, Mathias M. Mueller. Vienna, Austria; Vienna, Austria.

P131 CYTOKINE GENE POLYMORPHISMS ARE NOT ASSOCIATED WITH CLINICAL OUTCOMES AFTER HEART TRANSPLANTATION. (Abstract #744)
Ian S. Gourley, David DeNofrio, William Rand, Shashank Desai, Evan Loh, Malek Kamoun. Philadelphia, PA; Boston, MA; Boston, MA.

P132 CONVERSION FROM CYCLOSPORINE TO TACROLIMUS BENEFITS HEART TRANSPLANT RECIPIENTS AND ALLOWS A SIGNIFICANT DOSE-REDUCTION OF MMF. (Abstract #745)
Iza C. van Riemsdijk, Pascal J. Vantrimpont, Aggie H. Balk, Lex P. Maat, Carla C. Baan, Teun Gelder, Willem Weimar. The Netherlands.

P133 EFFICACY OF SIROLIMUS IN LUNG TRANSPLANT RECIPIENTS WITH ACUTE OR CHRONIC REJECTION. (Abstract #746)
Jason A. Crompton, K. Troy Somerville, Susan Parker, Mary O'Rourke, James Stringham, S.V. Karwande, Barbara Cahill. SLC, UT.

P134 IMPLICATIONS OF LEFT VENTRICULAR DYSFUNCTION EARLY AFTER HEART TRANSPLANTATION. (Abstract #747)
Javier Jimenez, Adriana Rosario, Alejandro Morales, Wakkas Tayara, Navdeep Boparai, Randall Starling, James Young, Patrick McCarthy, Daniel Cook. Cleveland, OH.

P135 OUTCOMES IN HEART TRANSPLANT RECIPIENTS WITH VIRAL GENOME IN THE MYOCARDIUM. (Abstract #748)
Neil Bowles, Branislav Radovancevic, Rajko Radovancevic, Cyndi D. Thomas, Thuy Vu, O. H. Frazier, Jeffrey A. Towbin. Houston, TX; Houston, TX.

P136 RHABDOMYOLYSIS WITH HYDROXY METHYLGLUTARYL COENZYME A (HMG-CoA) REDUCTASE INHIBITORS IN CARDIAC TRANSPLANT PATIENTS. (Abstract #749)
Stuti Sinha, Jill E. Martin, Rosann M. Giesting, Lynne E. Wagoner, Michael B. Bottorff. Cincinnati, OH; Cincinnati, OH.

P137 AN ANALYSIS OF CLINICAL, GENE EXPRESSION, AND PROTEIN DATA FROM PATIENTS WHO WERE WEANED FROM A CARDIAC ASSIST DEVICE. (Abstract #750)
Johannes Müller, Gerd Wallukat, Yu Go Weng, Michael Dandel, Hannelore Morwinski, Peter Ellinghaus, Joachim Huetter, Roland Hetzer. Berlin, Germany; Berlin, Germany; Berlin, Germany; Berlin, Germany; Berlin, Germany; Wuppertal, Germany; Wuppertal, Germany.

P138 THE DONOR CHEST RADIOGRAPH IN LUNG TRANSPLANTATION: PREDICTIVE VALUE AND INTER-OBSERVER VARIABILITY OF DONOR RADIOGRAPHS. (Abstract #751)
James S. Bolton, Marvin C. Borja, Jonathan B. Orens, Patrice A. Becker, Charles M. Wiener, Stephen C. Yang, Richard S. Stuart, John V. Conte.

P139 THE IMPACT OF TRACHEOSTOMY ON CLINICAL OUTCOMES FOLLOWING LUNG TRANSPLANTATION. (Abstract #752)
Siddharth A. Padia, Marvin C. Borja, Rajiv M. Jhaveri, Stephen C. Yang, Jonathan B. Orens, John V. Conte. Baltimore, MD.

P140 HAS TACROLIMUS MADE MULTI-ORGAN CARDIAC TRANSPLANTATION FEASABLE OR DOES THE HEART EXERT A PROTECTIVE EFFECT OVER VISCERAL TRANSPLANTS? (Abstract #753)
Joseph F. Buell, V. Jeevanandrum, David C. Cronin, Atshui Yoshida, Kenneth A. Newell, A. Baker, M. Josephson, E. Steve Woodle, J. R. Thistlethwaite, A. Anderson, J. M. Millis. Cincinnati, OH; Chicago, IL; Chicago, IL.

P141 ADVANCED RECIPIENT AGE AS A RISK FACTOR FOR MORBIDITY FOLLOWING CARDIAC TRANSPLANTATION: DOES SINGLE CENTER EXPERIENCE CORRELATE WITH MULTICENTER REGISTRIES? (Abstract #754) ♦
Katherine J. Hoercher, Patrick M. McCarthy, Michael K. Banbury, James B. Young, Randall C. Starling.

P142 ISCHAEMIC HEART DISEASE AND RENAL DYSFUNCTION IN CARDIAC TRANSPLANT RECIPIENTS. (Abstract #755)
Kottarathil A. Abraham, Catherine McGorrian, Maurice Nelligan, Alfred E. Woods, Peter J. Conlon, John Donohoe. Dublin, Ireland; Dublin, Ireland; Dublin, Ireland.

P143 ACCOMMODATION OF BLOOD GROUP INCOMPATIBLE HEART GRAFTS IN INFANT RECIPIENTS: DONOR A/B ANTIGEN EXPRESSION IN GRAFT BIOPSIES. (Abstract #756)
Lori J. West, Stacey M. Pollock-BarZiv, K. J. Lee, Anne I. Dipchand, John G. Coles, Phillip Ruiz. Toronto, ON, Canada; Miami, FL.

P144 A LONGITUDINAL INVESTIGATION OF RETENTION OF PATIENT DIRECTED TRANSPLANT EDUCATION. (Abstract #757) ♦
Alice J. Bordelon, Patricia A. Uber, Debi Dumas-Hicks, Myung H. Park, Robert L. Scott, Mandeep R. Mehra. New Orleans, LA.

Bone Marrow - All Topics II

P145 PROMISING RESULTS FROM A GLOBAL INITIATIVE TO DECREASE ORGAN SHORTAGE. (Abstract #758)
Leo Roels, Celia Wight, Blanca Miranda, Bernard Cohen. London, ON, Canada; Madrid, Spain; Leiden, The Netherlands.

P146 IMMUNOSUPPRESSION-RELATED COMPLICATIONS POST ISLET CELL TRANSPLANTATION. (Abstract #759)
Lisa C. Rothenberg, Jacqueline V. Ferreira, Aileen M. Caulfield, Tatiana Froud, Robin Harbach, Andres Boker, David A. Baidal, Camillo Ricordi, Rodolfo Alejandro. Miami, FL.

P147 PROPOSED PROTOCOL TO REDUCE INFECTIOUS COMPLICATIONS IN CLINICAL LIVING RELATED SMALL BOWEL TRANSPLANTATION. (Abstract #760)
Luca Cicalese, Pierpaolo Sileri, Noreen Coady, Massimo Asolati, Cristiana Rastellini, Herand Abcarian, Enrico Benedetti. Chicago, IL.

P148 IMPROVEMENT OF ACUTE RENAL DYSFUNCTION AFTER SOLID ORGAN TRANSPLANTATION DURING CALCINEURIN INHIBITOR "HOLIDAY" UNDER ANTI-CD25 MONOCLONAL ANTIBODY COVERAGE. (Abstract #761)
M. Cantarovich, P. Metrakos, J. Barkun, N. Giannetti, R. Cecere, J. Tchervenkov. Montreal, QC, Canada; Montreal, QC, Canada; Montreal, QC, Canada.

P149 KAPOSI'S SARCOMA IN SOLID ORGAN TRANSPLANTATION: ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY EXPERIENCE IN US. (Abstract #762)
M. Hanaway, J. Buell, A. Lo, J. Trofe, T. Beebe, M.R. First, E. S. Woodle. Cincinnati, OH; Cincinnati, OH.

P150 SUPERIOR REJECTION RATES WITH THYMOGLOBULIN INDUCTION IN SIMULTANEOUS PANCREAS KIDNEY TRANSPLANTATION. (Abstract #763)
Mounir A. Hajjar, George J. Chang, Harish D. Mahanty, John P. Roberts, Nancy L. Ascher, Ryutaro Hirose, Peter G. Stock, Chris E. Freise. San Francisco, CA.

P151 POTENTIAL DONOR POOL AND EFFICACY RATES IN SPANISH HOSPITALS. (Abstract #764)
Natividad Cuende, Blanca Miranda, Manuel Alonso, Jose F. Canon, Teresa Naya, Eduardo F. Zincke. Madrid, Spain; Sevilla, Spain.

P152 NON-*ASPERGILLUS* INVASIVE MYCELIA AS EMERGING PATHOGENS IN ORGAN TRANSPLANT RECIPIENTS. (Abstract #765)
S. Husain, R. K. Avery, P. K. Linden, B. Alexander, S. Kusne, E. A. Dominguez, T. Pruett, R. Jacobs, J. Tollemar, D. R. Snydman, M. M. Wagener, N. Singh. Pittsburgh, PA; Cleveland, OH; Durham, NC; Omaha, NE; San Francisco, CA; Huddinge, Sweden; Boston, MA; Charlottesville, VA.

P153 THE EFFECT OF PRIMARY IMMUNOSUPPRESSIVE AGENT ON THE PREDOMINANT CLINICAL MANIFESTATIONS OF *C. NEOFORMANS* INFECTION IN ORGAN TRANSPLANT RECIPIENTS. (Abstract #766)
Shahid Husain, Timothy Gayowski, Marilyn M. Wagener, Nina Singh. Pittsburgh, PA.

P154 HYPOADRENALISM SYNDROME IN ABDOMINAL ORGAN RECIPIENTS AND RESPONSE TO STRESS DOSE CORTICOSTEROIDS. (Abstract #767)
Peter Linden, David J. Kramer, Melissa Saul, Paulo Fontes, Kareem Abu-Elmagd, John Fung. Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA.

Immunosuppression, Preclinical Studies II

P155 THE HIGH MOLAR POTENCY OF TACROLIMUS MAY REFLECT PRE-ASSEMBLY OF THE BINDING PROTEIN WITH CALCINEURIN. (Abstract #768)
Lina Kung, Annette Sedlmeyer, Philip F. Halloran. Edmonton, AB, Canada; Edmonton, AB, Canada; Frankfurt, Germany.

P156 THE EFFECTS OF MYCOPHENOLATE MOFETIL ON CARDIAC ALLOGRAFT SURVIVAL AND CARDIAC ALLOGRAFT VASCULOPATHY IN MINIATURE SWINE. (Abstract #769)
Margaret L. Schwarze, Joshua D. Mezrich, Matthew T. Menard, Stuart L. Houser, Michaella E. Maloney, Edmund P. Pillsbury, David H. Sachs, Joren C. Madsen. Boston, MA; Boston, MA; Boston, MA.

P157 COS BLOCKADE BY ANTI-CD40 AND ANTI-CD86 PREVENTS KIDNEY GRAFT REJECTION IN RHESUS MONKEYS. (Abstract #770) ♦
Krista G. Haanstra, Els Sick, Seema G. Ramdien-Murli, Jan Ringers, Mark de Boer, Louis Boon, Margreet Jonker. Rijswijk, The Netherlands; Amsterdam, The Netherlands; Leiden, The Netherlands.

P158 MMF MONOTHERAPY REDUCES SIGNIFICANTLY THE TRANSENDOTHELIAL MIGRATION OF GRAFT INFILTRATING LEUCOCYTES. A COMPARATIVE STUDY WITH CSA AND FK 506 after EXPERIMENTAL CARDIAC TRANSPLANTATION. (Abstract #771)
Markus H. Richter, Friedrich W. Mohr. Leipzig, Germany.

P159 PHARMACODYNAMIC (PD) MEASUREMENTS OF THE IMMUNOSUPPRESSIVE EFFECTS OF THE COMBINATION OF CYCLOSPORINE (CSA) AND MYCOPHENOLATE MOFETIL (MMF): CORRELATION BETWEEN DRUG DOSE AND LYMPHOCYTE FUNCTION. (Abstract #772)
M. J. Barten, J. F. Gummert, P. Bartsch, M. Boeger, P. Gelhaar, T. Rauch, R. Autschbach, F. W. Mohr. Leipzig, Saxonia, Germany.

P160 SYNERGISTIC EFFECTS OF IVIG AND RAPAMYCIN ON CELL PROLIFERATION AND APOPTOSIS IN THE HUMAN MIXED LYMPHOCYTE REACTION. (Abstract #773)
Mieko Toyoda, Anna Petrosyan, Andy Pao, Stanley C. Jordan. L.A., CA.

P161 MYCOPHENOLIC ACID IMPROVES HOMOCYSTEINE METABOLISM OF HUMAN RENAL PROXIMAL TUBULAR EPITHELIAL CELLS (huRPTEC). (Abstract #774)
Mihaela C. Ignatescu, Josef Kletzmayr, Manuela Foedinger, Walter H. Hoerl, Gere Sunder-Plassmann. Vienna, Austria; Vienna, Austria.

P162 IDENTIFICATION OF ACCEPTABLE HLA ANTIGENS FOR HIGHLY ALLOSENSITIZED PATIENTS BY ELISA-BASED SERUM ANALYSIS AND A MOLECULAR MATCHING ALGORITHM. (Abstract #775)
Mohammed R. Awad, Y. Awadalla, R. J. Duquesnoy. Pittsburgh, PA.

P163 STABLE DOWN-REGULATED DONOR-SPECIFIC T-CELL REACTIVITY IN PATIENTS WITH WELL-FUNCTIONING KIDNEY ALLOGRAFT ALLOWS TAPERING OF IMMUNOSUPPRESSION. (Abstract #776)
Nicole M. van Besouw, Barbara J. van der Mast, Ronella de Kuiper, Peter J.H. Smak Gregoor, Lenard M.B. Vaessen, Jan N.M. IJzermans, Teun van Gelder, Willem Weimar. Rotterdam, The Netherlands; Rotterdam, The Netherlands.

P164 ORAL ADMINISTRATION OF GREEN TEA POLYPHENOLS (GTP) INHIBITS ANTIGEN-SPECIFIC IFN-γ SECRETION AND DELAYS MINOR ANTIGEN DISPARATE SKIN GRAFT REJECTION IN MICE. (Abstract #777)
Jorg Bayer, Tariq Haqqi, Teryn Edwards, Anna Valujskikh, Peter S. Heeger. Cleveland, OH; Cleveland, OH.

P165 PREDICTING THRESHOLDS FOR CYCLOSPORINE+SIROLIMUS IN PIVOTAL TRIALS WITH AN ARRAY OF STIMULATED LYMPHOCYTE/LEUKOCYTE RESPONSES (sLR). (Abstract #778)
Rakesh Sindhi, Jan Allaert. Pittsburgh, PA, United States; San Jose, CA.

Tolerance II

P166 HUMAN LIVER ALLOGRAFT ACCEPTANCE AND THE "TOLERANCE ASSAY": IN VITRO ANTI-DONOR T CELL REACTIVITY SHOWS HYPOREACTIVITY TO DONOR CELLS BUT, UNLIKE DTH, FAILS TO DETECT BYSTANDER SUPPRESSION. (Abstract #779)
Felix E. Geissler, Ewa Jankowska-Gan, Lynn DeVito-Haynes, Tonja Rhein, Munci Kalayoglu, Hans J. Sollinger, William J. Burlingham. Madison, WI; Leipzig, Germany.

P167 **CD25+CD4+ T CELLS ARE INDUCED IN ACQUIRED CENTRAL TOLERANCE. (Abstract #780)**
Jose L. Trani, Joseph W. Markmann, Alexander Schlacterman, Daniel J. Moore, Bryan J. Tran, Ines C. Lin, Andrew J. Caton, James F. Markmann. Philadelphia, PA; Philadelphia, PA.

P168 **A CLINICALLY FEASIBLE PROTOCOL TO INDUCE TOLERANCE TO LIMB ALLOGRAFTS USING MIXED ALLOGENEIC CHIMERISM. (Abstract #781)**
Vijay S. Gorantla, Gustavo Perez-Abadia, Xiaoping Ren, Ramsey Majzoub, Kaustubha A. Prabhune, Cedric G. Francois, Marieke Vossen, Pascal C. Brouha, Claudio Maldonado, Diane J. Pidwell, Warren C. Breidenbach, Darla K. Granger, John H. Barker. Louisville, KY; Louisville, KY; Louisville, KY.

P169 **LYMPH NODE LOCALIZATION IS NECESSARY FOR TOLERANCE INDUCTION IN MICE. (Abstract #782)**
Shaun M. Honig, Yalai Bai, Jonathan S. Bromberg. New York, NY.

P170 **VISUALIZATION AND CHARACTERIZATION OF DONOR MINOR H ANTIGEN-SPECIFIC CD8+ T CELLS IN A LONG-TERM(28yr) ALLOGRAFT ACCEPTOR. (Abstract #783)**
Junchao Cai, Junglim Lee, Ewa Jankowska-Gan, Lynn DeVito-Haynes, Satoshi Kusaka, Jonathan Schneck, Tuna Mutis, Els Goulmy, William J. Burlingham. Madison; Baltimore; Leiden, The Netherlands.

P171 **DONOR MINOR H ANTIGEN-DRIVEN IMMUNE REGULATION IN LONG-TERM (28yr) TOLERANT KIDNEY TRANSPLANT RECIPIENT: ROLE OF MICROCHIMERISM AND CD8+ REGULATORY T CELLS. (Abstract #784)**
Junglim Lee, Ewa Jankowska-Gan, Junchao Cai, William J. Burlingham. Madison, WI.

P172 **INDIRECT ALLOREACTIVITY WITH EPITOPE SPREADING AND THE EFFECTS OF ORAL DONOR MHC CLASS II PEPTIDE FEEDING. (Abstract #785)**
Karl L. Womer, John P. Vella, Mohamed H. Sayegh, Charles B. Carpenter. Boston, MA.

P173 **EN BLOC THYMUS ALLOGRAFTS SUPPORT THYMOPOEISIS IN MINIATURE SWINE. (Abstract #786)**
John C. LaMattina, Naoki Kumagai, Rolf N. Barth, David H. Sachs, Kazuhiko Yamada. Boston, MA.

P174 **DONOR-SPECIFIC REGULATORY MECHANISMS AND LINKED SUPPRESSION IN TACROLIMUS-INDUCED TOLERANCE TO FULLY MHC-MISMATCHED RENAL ALLOGRAFTS IN MINIATURE SWINE. (Abstract #787)**
Naoki Kumagai, Richard S. Lee, Rolf N. Barth, John C. LaMattina, Ryu Utsugi, Hiroshi Kitamura, Shannon G. Moran, David H. Sachs, Kazuhiko Yamada. Boston, MA.

P175 **RENAL ALLOGRAFTS WITH SHORT-COURSE TACROLIMUS THERAPY INDUCE TOLERANCE IN OUTBRED SWINE AND NON-HUMAN PRIMATES. (Abstract #788)**
Rolf N. Barth, John C. LaMattina, Shin Yamamoto, Naoki Kumagai, Katsuhito Teranishi, Hiroshi Kitamura, David H. Sachs, Kazuhiko Yamada. Boston, MA.

P176 **THE ROLE OF MHC AND COSTIMULATORY MOLECULES EXPRESSED ON DONOR CELLS IN MODULATING ALLOIMMUNE RESPONSE IN VIVO. (Abstract #789)**
Koji Kishimoto, Didier A. Mandelbrot, Victor M. Dong, Akira Yamada, Hugh Auchincloss, Jr., Laurence A. Turka, Mohamed H. Sayegh. Boston, MA; Boston, MA; Philadelphia, PA.

P177 **DEFICIENT PRODUCTION OF ANTI-A/B ANTIBODIES AFTER ABO-INCOMPATIBLE INFANT HEART TRANSPLANTATION: CLINICAL NEONATAL B-CELL TOLERANCE? (Abstract #790)♦**
Lori J. West, Stacey M. Pollock-BarZiv, K. J. Lee, Anne I. Dipchand, John G. Coles, Phillip Ruiz. Toronto, ON, Canada; Miami, FL.

Acute/Chronic Rejection II

P178 **EXPRESSION OF THE SIGNAL TRANSDUCTION PROTEIN 14-3-3 GAMMA IN INJURED ARTERIES AND STIMULATED HUMAN VASCULAR SMOOTH MUSCLE CELLS. (Abstract #791)**
Michael V. Autieri, Christopher J. Carbone, Howard J. Eisen.

P179 **FTY720 (FTY) - AN IMMUNOSUPPRESSOR THAT ALTERS LYMPHOCYTE TRAFFICKING - COMPLETELY ABROGATES CHRONIC REJECTION (CR) IN COMBINATION WITH CsA. (Abstract #792)**
T. Koshiba, B. Van Damme, Y. Lu, Y. Yan, O. Rutgeerts, L. Overbergh, C. Mathieu, M. Waer, J. Pirenne. Leuven, Belgium.

P180 **DO LIVING UNRELATED TRANSPLANT (LURT) RECIPIENTS REQUIRE INDUCTION? (Abstract #793)**
Anne M. Wiland, Jeffrey C. Fink, Eugene Schweitzer, Alan Farney, Matthew R. Weir, Benjamin Philosophe, John Colonna, Clarence Foster, Steven Blahut, Stephen T. Bartlett. Baltimore, MD; Baltimore, MD; Baltimore, MD.

P181 **DONOR HYPERTENSION INCREASES IMMUNOGENICITY AND INTENSIFIES CHRONIC CHANGES IN LONG SURVIVING RENAL ALLOGRAFTS. (Abstract #794)**
Johann Pratschke, Grzegorz Kofla, Markus J. Wilhelm, Dustin Paz, Igor Laskowski, Athanasios Vergopoulos, Stefan G. Tullius, Harald J. MacKenzie, Wayne W. Hancock, Peter Neuhaus, Hans-Dieter Volk, Nicholas L. Tilney. Berlin, Germany; Berlin, Germany; Boston, MA; Cambridge, MA.

P182 **CHRONIC REJECTION OF MURINE CARDIAC ALLOGRAFT DISCORDANT AT THE H13 MINOR HISTOCOMPATIBILITY ANTIGEN CORRELATED WITH THE GENERATION OF H13-SPECIFIC CD8+ CTLs. (Abstract #795)**
Junbao Yang, Olack J. Barbara, Zahid Kaleem, Wei Liu, Andres Jaramillo, T. Mohanakumar. St. Louis, MO.

P183 **OX-40 LIGAND DEFICIENCY RESULTS IN DIMINISHED GRAFT ARTERIAL DISEASE IN CARDIAC ALLOGRAFTS. (Abstract #796)♦**
Jun-ichi Suzuki, Sarah E. Cole, Andy I. Chen, Arlene H. Sharpe, Peter Libby, Richard N. Mitchell. Boston, MA; Boston, MA.

P184 **RELEVANCE OF RAT AORTA TRANSPLANTATION AS A MODEL OF ORGAN GRAFT ARTERIOSCLEROSIS. (Abstract #797)**
Emile Andriambeloson, Charles Pally, Madeleine Fringeli, Catherine Cannet, Patrick Gfeller, Hans-Guenter Zerwes, Marc Bigaud.

P185 **COMBINED TREATMENT WITH MYCOPHENOLATE MOFETIL (MMF) AND AN ANGIOTENSIN II (AII) RECEPTOR ANTAGONIST FULLY PROTECTS FROM CHRONIC RENAL ALLOGRAFT REJECTION. (Abstract #798)**
Marina Noris, Marilena Mister, Nadia Azzollini, Norberto Perico, Gianfranco Marchetti, Giuseppe Remuzzi. Bergamo, Italy; Bergamo, Italy.

P186 **DONOR BRAIN DEATH PROMOTES THE DEVELOPMENT OF FIBROSIS IN CHRONIC RAT CARDIAC ALLOGRAFT REJECTION. (Abstract #799)**
Markus J. Wilhelm, Johann Pratschke, Maarten Taal, Francisca Beato, Igor A. Laskowski, Wayne W. Hancock, Nicholas L. Tilney. Boston, MA; Muenster, Germany; Cambridge, MA, Germany.

P187 **ANGIOTENSIN II TYPE I RECEPTOR BLOCKADE REDUCES THE DEVELOPMENT OF CHRONIC REJECTION IN TRANSMYOCARDIAL ARTERIES AFTER CARDIAC TRANSPLANTATION IN A RAT MODEL. (Abstract #800)**
Markus H. Richter, Heike R. Richter, Hans Georg Olbrich, Frankfurt, Germany.

Allorecognition, Antigen Presentation, Co-Stimulation and Other II

P188 **CHIMERIC PATTERNS AFTER ORGAN TRANSPLANTS USING GREEN FLUORESCENT PROTEIN-TRANSGENIC RAT. (Abstract #801)**
Kazunori Tahara, Hiroo Uchida, Yasunaru Sakuma, Yoji Hakamata, Eiji Kobayashi. Kawachi-gun, Tochigi, Japan.

P189 **DEOXYSPERGUALIN (DSG) TREATED DENDRITIC CELLS (DC) UNDERGO TRAIL INDUCED APOPTOSIS. (Abstract #802)**
Jianguo Wu, Steven Sooudi, Jin He, Frank Thomas, Judith Thomas. Birmingham, AL.

P190 **EFFECT OF CO-STIMULATORY PATHWAY BLOCKADE ON RAT ORTHOTOPIC SMALL BOWEL TRANSPLANTATION BY ADENOVIRUS MEDIATED TRANSDUCTION OF CTLA4IG AND CD40IG GENES. (Abstract #803)**
Hayato Echizenya, Kenichiro Yamashita, Megumi Takehara, Katsuhito Konishi, Masaru Nomura, Naoyuki Yanagida, Norihiko Kitagawa, Tokushi Kobayashi, Taku Hashimoto, Nobuyasu Sakihama, Hiroyuki Furukawa, Manabu Inobe, Toshimitsu Uede, Satoru Todo. Sapporo, Hokkaido, Japan; Sapporo, Hokkaido, Japan.

P191 **ANALYSIS OF ALLOIMMUNE T-CELL ACTIVATION IN 4-1BB DEFICIENT MICE. (Abstract #804)**
Seonjoo Park, Hong R. Cho, Duck J. Han, Yong J. Lee, Sung G. Lee, Kyubum Kwack, Byung-Sam Kim, Eun-A Lee, Byoung S. Kwon. Ulsan City, Korea; Seoul, Korea; Ulsan City, Korea.

P192 **IN VITRO GENERATION OF PANCREATIC ENDOCRINE TYPE CELLS FROM MOUSE EMBRYONIC STEM CELLS. (Abstract #805)**
Hsun Ku, Gordon Keller, Jonathan Bromberg.

P193 **CROSS-PRIMING IN CHRONIC REJECTION. (Abstract #806)**
James J. Yun, Michael P. Fischbein, Hillel Laks, Michael C. Fishbein, Charles Wortham, Yoshihito Irie, Abbas Ardehali. Los Angeles, CA; Los Angeles, CA.

P194 **DEVELOPMENT OF CARDIAC ALLOGRAFT VASCULOPATHY IN H-2M DEFICIENT MICE. (Abstract #807)**
James J. Yun, Michael P. Fischbein, Hillel Laks, Michael C. Fishbein, Yoshihito Irie, Abbas Ardehali. Los Angeles, CA; Los Angeles, CA.

P195 **EXPRESSION OF HISTOCOMPATIBILITY ANTIGENS IN MURINE EMBRYONIC STEM (ES) CELL CULTURES. (Abstract #808)**
Inka Held, Brenda W. Kahan, Lynn M. Jacobson, Debra A. Mackenzie, Debra A. Hullett, Jon S. Odorico. Madison, WI.

P196 **THE MIGRATION OF DENDRITIC CELLS IS CLOSELY ASSOCIATED WITH THE EXPRESSION OF THE ACTIN BUNDLING PROTEIN FASCIN. (Abstract #809)** ♦
Kenneth A. West, Patricia Colp, Monther Al-Alwan, Giusepe De Panfilis, Mieke Mommaas, Geoff Rowden. Halifax, Canada; Brescia, Italy; Leiden, The Netherlands.

P197 **PRESENTATION OF DONOR MHC ANTIGENS BY EXOSOMES DERIVED FROM BONE MARROW DENDRITIC CELLS CAN INDUCE ALLOGRAFT TOLERANCE. (Abstract #810)**
Helene Pêche, Sebastian Amigorena, Michele Heslan, Jean-Paul Soulillou, Maria Cristina Cuturi. Nantes, France; Paris, France.

P198 **GLOBAL VISUALISATION OF T CELL MOBILIZATION IN ALLOGRAFTS ASSESSED THROUGH INTEGRATED QUALITATIVE/QUANTITATIVE STUDY OF Vβ TCR TRANSCRIPTS. EARLY ACUTE REJECTION IS MEDIATED BY DIRECT RECOGNITION, WHICH IS DOWN-REGULATED DURING TOLERANCE INDUCTION. (Abstract #811)**
Marina Guillet, Sophie Brouard, Katia Gagne, Fabien Sebille, Maria-Cristina Cuturi, Marc-Andre Delsuc, Jean-Paul Soulillou. Nantes, France; Montpellier, France.

Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines II

P199 **DIFFERENTIAL INTRAGRAFT CYTOKINE mRNA PROFILES DURING REJECTION AND REPAIR OF CLINICAL HEART TRANSPLANTS. (Abstract #812)**
Hester A. de Groot-Kruseman, Carla C. Baan, Wendy M. Mol, Bert G.M. Niesters, Lex P.W.M. Maat, Aggie H.M.M. Balk, Willem Weimar. Rotterdam, The Netherlands.

P200 **IFN-g REGULATES ENDOTHELIAL CELL PRODUCTION OF CHEMOKINES. (Abstract #813)** ♦
Hirohito Kobayashi, Shoji Koga, Hiroshi Toma, Andrew C. Novick, Robert L. Fairchild. Cleveland, OH; Tokyo, Japan.

P201 **RANTES NEUTRALIZATION REDUCES MONONUCLEAR CELL INFILTRATION AND INTIMAL THICKENING IN CARDIAC ALLOGRAFT VASCULOPATHY. (Abstract #814)**
James J. Yun, Michael P. Fischbein, Hillel Laks, Michael C. Fishbein, Yoshihito Irie, Robert M. Strieter, Judith A. Berliner, Abbas Ardehali. Los Angeles, CA; Los Angeles, CA; Los Angeles, CA.

P202 **PRODUCTION OF MIG AND CXCR3 IN CHRONIC REJECTION. (Abstract #815)**
James J. Yun, Michael P. Fischbein, Hillel Laks, Michael C. Fishbein, Yoshihito Irie, Robert L. Strieter, Judith A. Berliner, Abbas Ardehali. Los Angeles, CA; Los Angeles, CA; Los Angeles, CA.

P203 **CYCLOSPORINE A UPREGULATES THE EXPRESSION OF TGF-β1 AND ITS RECEPTORS TYPE I AND TYPE II IN RAT MESANGIAL CELLS. (Abstract #816)**
Johannes Waiser, Kerstin Dell, Torsten Boehler, Jens Gaedeke, Klemens Budde, Hans-Hellmut Neumayer.

P204 **DENDRITIC CELLS DERIVED FROM LIVER B CELL LINEAGE INDUCE T CELL APOPTOSIS THROUGH CASPASE ACTIVATION, BUT NOT VIA FAS OR TNF LIGATION. (Abstract #817)** ♦
Xaioyan Liang, Shiguang Qian, Jinrong Li, C. Andrew Bonham, John J. Fung, Lina Lu. Pittsburgh, PA.

P205 **FRACTALKINE MODULATES T LYMPHOCYTE AND MACROPHAGE FUNCTIONS. (Abstract #818)**
Lisa A. Robinson, Dennis W. Thomas, Chandra Nataraj, Josette M. Tucker, Dhavalkumar D. Patel, Thomas M. Coffman. Durham, NC; Durham, NC.

P206 **ANTI-CD45RB TREATMENT OF NORMAL MICE RESULTS IN REDUCTION OF CD45RB AND L-SELECTIN EXPRESSION ON PERIPHERAL BLOOD T-CELLS. (Abstract #819)**
Lydia Visser, Sibrand Poppema. Groningen, The Netherlands.

P207 **THE EFFECT OF ANTI-CD45 ANTIBODIES IN VITRO. (Abstract #820)**
Lydia Visser, Sibrand Poppema. Groningen, The Netherlands.

P208 **EVIDENCE FOR A ROLE OF FRACTALKINE AND ITS RECEPTOR CX_3CR1 IN ALLOGRAFT TOLERANCE. (Abstract #821)**
Cedric Louvet, Elise Chiffoleau, Jean-Marie Heslan, Maria Cristina Cuturi. Nantes, France.

P209 **DENDRITIC CELLS CONDITIONED BY A VITAMIN D ANALOG ARE RESISTANT TO MATURATIONAL STIMULI EVEN AFTER ANALOG WITHDRAWAL. (Abstract #822)**
Matthew Griffin, Ward Lutz, Vy Phan, Lori Bachman, David McKean, Rajiv Kumar. Rochester, MN; Rochester, MN; Rochester, MN.

P210 **MECHANISM OF BENEFICIAL EFFECT ON T-CELL REGULATORY APOPTOSIS BY MPA. (Abstract #823)**
Michio Nakamura, Warren R. Maley, James F. Burdick. Baltimore, MD.

P211 **PHENOTYPE AND FUNCTION OF DONOR MHC CLASS II+ CELLS INVOLVED IN LIVER ALLOGRAFT ACCEPTANCE UNDER TACROLIMUS IMMUNOSUPPRESSION. (Abstract #824)**
Olga Azhipa, Toyokazu Okuda, Anthony J. Demetris, Thomas E. Starzl, Noriko Murase. Pittsburgh, PA.

P212 **HIGH EXPRESSION OF CHEMOKINES INTERFERON-γ INDUCIBLE PROTEIN OF 10 kDa (IP-10), MONOKINE INDUCED BY INTERFERON-γ(MIG) AND OF THEIR RECEPTOR (CXCR3) IN ACUTE RENAL REJECTION. (Abstract #825)**
Paola Romagnani, Elena Lazzeri, Laura Lasagni, Chiara Beltrame, Michela Francalanci, Lucia Laurenzig, Andrea Buonamano, Mario Rotondi, Mario Serio, Elisabetta Bertoni, Alberto Rosati, Maurizio Salvadori. Florence, Italy; Florence, Italy; Florence, Italy.

Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH II

P213 **DONOR SPECIFIC TRACKING OF ALLOANTIGEN-SPECIFIC, EGFP-ENGINEERED T LYMPHOCYTES INTO RAT KIDNEY TRANSPLANTS. (Abstract #826)**
Grit Schroeder, Kirsten Risch, Markus Hammer, Alexander Fluegel, Josef Brock, Manfred Lehmann, Thomas Ritter, Hans-Dieter Volk. Rostock, Germany; Berlin, Germany; Munich, Germany.

P214 **REVERSIBLY IMMORTALIZED LIVER SINUSOIDAL ENDOTHELIAL SCAVENGER CELLS TO DEVELOP A BIOARTIFICIAL LIVER. (Abstract #827)**
Hirofumi Noguchi, Naoya Kobayashi, Toshinori Totsugawa, Toshihisa Matsumura, Takamasa Watanabe, Toshiyoshi Fujiwara, Masakiyo Sakaguchi, Karen A. Westerman, Phillipe Leboulch, Noriaki Tanaka. Okayama, Japan; Okayama, Japan; Cambridge, MA.

P215 **PROLONGED ISLET ALLOGRAFT SURVIVAL WITHOUT TOXICITY USING ISATX247, A NOVEL CALCINEURIN INHIBITOR. (Abstract #828)**
A.M.J. Shapiro, RJ Rajotte, J.R.T. Lakey, D.L. Bigam, NM Kneteman, RW Yatscoff. Edmonton, AB, Canada; Edmonton, AB, Canada.

P216 **SMALL BOWEL INTRAMURAL SITE FOR ISLET TRANSPLANTATION IN THE PORCINE ALLOGRAFT MODEL. (Abstract #829)**
Jun-ichiro Sageshima, Satoshi Shibata, Nicole Kirchhof, Kunihiko Hiraoka, Hui-Jian Zhang, Thomas Gilmore, Michele Dunning, Jeffry Shearer, Jeffrey Ansite, David E.R. Sutherland, Bernhard J. Hering. Minneapolis, MN.

P217 **VASCULARIZED ISLET-KIDNEY ALLOGRAFTS CURE SURGICALLY-INDUCED DIABETES AND INDUCE TOLERANCE IN MINIATURE SWINE. (Abstract #830) ♦**
Naoki Kumagai, Rolf N. Barth, John J. O'Neil, John C. LaMattina, Ryu Utsugi, Hiroshi Kitamura, Gordon C. Weir, David H. Sachs, Kazuhiko Yamada. Boston, MA; Boston, MA.

P218 **TRANSFECTION AND TRANSGENE EXPRESSION IN A HUMAN KIDNEY DURING EX VIVO PERFUSION. (Abstract #831)**
Lauren Brasile, Bart Stubenitsky, Maurits Booster, Dorian Araneda, Carl Haisch, Gauke Kootstra.

P219 **EFFECTS OF SEQUENTIAL CD154 AND LFA-1 BLOCKADE ON THE SURVIVAL OF ALLOGENEIC ISLET GRAFTS IN NOD MICE. (Abstract #832)**
Thierry Berney, Antonello Pileggi, R.Damaris Molano, Camillo Ricordi, Luca Inverardi. Miami, FL.

P220 **ABSENCE OF INDUCIBLE NITRIC OXIDE SYNTHASE, AND HEME OXYGENASE-1 UPREGULATION RESULT IN IMPROVED ISLET GRAFT FUNCTION. (Abstract #833)**
Antonello Pileggi, R.Damaris Molano, Thierry Berney, Ricardo L. Pastori, Fritz H. Bach, Camillo Ricordi, Luca Inverardi. Miami, FL; Boston, MA.

P221 **PROLONGED SURVIVAL AND ABSENCE OF LYMPHOCYTIC INFILTRATION IN RAT ISLET XENOGRAFTS IN DIABETIC MICE AFTER SHORT COURSE OF CD45RB MONOTHERAPY. (Abstract #834)**
Lydia Visser, Paul De Vos, Sibrand Poppema. Groningen, The Netherlands.

P222 **PIG ISLETS TRANSPLANTED INTO MONKEYS ARE NOT SUBJECT TO ANTIBODY MEDIATED REJECTION AND CAN SURVIVE FOR MORE THAN 50 DAYS. (Abstract #835)**
Margreet Jonker, Josephine K.R.H. Rijkelijkhuizen, Krista G. Haanstra, Jacqueline Wubbe, Anja Roos, Jan Ringers, Eelco Bouwman. Rijswijk, The Netherlands; Leiden, The Netherlands; Leiden, The Netherlands.

Tissue Injury, Preservation II

P223 **CONTINOUS GRAFT MONITORING DURING LIVER TRANSPLANTATION WITH MICRODIALYSIS IN A PIG MODEL. (Abstract #836)**
Grzegorz Nowak, Johan Ungerstedt, Jan Wernerman, Urban Ungerstedt, Bo-Göran Ericzon. Huddinge, Sweden; Stockholm, Sweden; Huddinge, Sweden.

P224 **CD4+ T CELLS PARTICIPATE IN ACUTE RENAL FAILURE INDUCED BY ISCHEMIA/REPERFUSION INJURY. (Abstract #837) ♦**
Helady Sanders, Niels O. Saraiva Camara, Marcelo Franco, Jose O. Medina Pestana, Irene L. Noronha, Alvaro Pacheco-Silva. Sao Paulo, SP, Brazil; Sao Paulo, SP, Brazil; Sao Paulo, SP, Brazil.

P225 **ANTIGEN-INDEPENDENT INJURY TO KIDNEY ALLOGRAFTS FROM NON HEART-BEATING DONORS (NHBD) ACCELERATES CHRONIC HOST ALLODESTRUCTION. (Abstract #838)**
Igor A. Laskowski, Martin Gasser, Johann Pratschke, Wayne W. Hancock, Nicholas L. Tilney. Boston, MA; Cambridge, MA.

P226 **TNF-α AS A CRITICAL COMPONENT OF ENDOTHELIAL ACTIVATION POST REPERFUSION: INHIBITION BY EURO-COLLINS SOLUTION IN A MODEL OF ISCHEMIA-REPERFUSION. (Abstract #839)**
Keith Hunter, Hee Xu, Douglas Tadaki, Uma Basavanna, Robert Kampen, David Harlan, Allan Kirk. Bethesda, MD.

P227 **UNCOUPLING PROTEIN-2 IS UPREGULATED IN STEATOTIC HUMAN LIVERS: IS THIS THE CAUSE OF PRIMARY NONFUNCTION? (Abstract #840)**
Kenneth D. Chavin, Stephen F. Shafizadeh, Kathy Haines, Satish Nadig, Lydia Nichols, Ryan Fiorini, Nicolas P. Mora, Sally Self, Michael Schmidt. Charleston, SC; Charleston, SC; Charleston, SC.

P228 **CORTICOSTEROIDS ENHANCE HEPATIC INJURY FOLLOWING ISCHEMIA/REPERFUSION. (Abstract #841)**
Pierpaolo Sileri, Stefano Schena, Michael Mihalov, Joji Fukada, Cristiana Rastellini, Jaques Pirenne, Enrico Benedetti, Luca Cicalese. Chicago, IL; Chicago, IL; Miami, FL.

P229 **A NEW AND IMPROVED CARDIAC PRESERVATION SOLUTION (PBS11) IN COMPARISON WITH UW, CELSIOR AND ST THOMAS HOSPITAL SOLUTIONS. (Abstract #842)**
Madgy S. Attia, Irfan Ahmed, Claire L. Corps, Mary S. Kamel, Niaz Ahmad, David J. Potts, J.Peter A. Lodge. Leeds, WY, United Kingdom; Leeds, WY, United Kingdom.

P230 **HEME OXYGENASE-1 OVEREXPRESSION EXERTS CYTOPROTECTIVE EFFECTS AGAINST ISCHEMIA/REPERFUSION INJURY VIA ANTI-APOPTOTIC PATHWAY. (Abstract #843)**
Masamichi Katori, Roland Buelow, Bibo Ke, Jefferey Ma, Ana J. Coito, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Fremont, CA.

P231 **DOPAMINE TREATMENT IN BRAIN DEAD DONORS REDUCES MONOCYTE INFILTRATION IN THE KIDNEY. (Abstract #844)**
Meike Schaub, Christian Ploetz, Liu Fang, Benito Yard, Claude Braun, Peter Schnuelle, Fokko J. van der Woude. Mannheim, Germany.

P232 **ROLE OF NITRIC OXIDE AND ENDOTHELIN IN ISCHEMIA/REPERFUSION INJURY IN THE RAT PANCREAS. (Abstract #845)**
Nicholas T. Stowe, Ann V. Robinson, Mike S. Simonson, James A. Schulak. Cleveland, OH; Cleveland, OH; Cleveland, OH.

Xenotransplantation II

P233 **INTRAGRAFT EXPRESSION OF PROTECTION GENES HO-1 AND Bcl-2 IN PRIMARY HEART XENOGRAFTS: A LACK OF PROTECTION EFFECT AGAINST XENOGRAFT HYPERACUTE REJECTION INDUCED BY HYPER-IMMUNE SERA. (Abstract #846)**
Gordon D. Wu, Yang-Sung Jin, Vaughn A. Starnes, Donald V. Cramer. Los Angeles, CA.

P234 **EVALUATION OF DIFFERENT α-GAL GLYCOCONJUGATES FOR USE IN XENOTRANSPLANTATATION. (Abstract #847)**
Alexander Schwarz, Ivona Bakaj, Patrick Birch, Joanna Fesi, Anna Nepomich, Cianna Cooper, Margaret A. Velardo, Aileen Stark, Lisa E. Diamond, John S. Logan, Guerard W. Byrne. Princeton, NJ.

P235 **HUMAN MONOCYTES PLAY AN IMPORTANT ROLE IN INDIRECT ANTIGEN PRESENTATION AND COSTIMULATION TO T CELLS DURING HUMAN ANTI-PORCINE IMMUNE RESPONSES. (Abstract #848)**
He Xu, Douglas K. Tadaki, Patrick J. Blair, Francis Cruzata, David M. Harlan, Allan D. Kirk.

P236 **DEVELOPMENT AND CHARACTERIZATION OF ANTI-GAL B-CELL RECEPTOR TRANSGENIC GAL-/- MICE. (Abstract #849)**
Hui Xu, Ying Lei, Ajay Sharma, Hua Wan, Jeaninne Okabe, John S. Logan, Guerard W. Byrne. Princeton, NJ.

P237 **ANTI-GAL ANTIBODY GENE USAGE IN NAIVE AND POST XENOGRAFT GAL-/- MICE. (Abstract #850)**
Hui Xu, Ajay Sharma, Libing Chen, Caren Harrison, Yuanyuan Wei, Anita S.-F. Chong, John S. Logan, Guerard W. Byrne. Princeton, NJ.

P238 **ANTI-GAL MONOCLONAL ANTIBODIES INDUCE PIG MICROVASCULAR ENDOTHELIAL CELL (PMVEC) ACTIVATION AND APOPTOSIS. (Abstract #851)**
Hui Xu, Bashoo Naziruddin, Yuanyuan Wei, Libing Chen, John S. Logan, Guerard W. Byrne. Princeton, NJ.

P239 **PIG-TO-PRIMATE RENAL XENOTRANSPLANTATION - PREDOMINANT GLOMERULAR INJURY IN LONG TERM FUNCTIONING H-CD55 TRANSGENIC KIDNEYS. (Abstract #852)**
Jan Schmidtko, Martin Loss, Ergin Kilic, Ralf Roland Lorenz, Jens Martin Hecker, Richard Appiah, Robert Kunz, Juergen Klempnauer, Udo Helmchen, Michael Winkler. Hannover, Germany; Hamburg, Germany; Hannover, Germany.

P240 **NATURAL REGULATION OF GALα1-3GAL EXPRESSION. (Abstract #853)**
Joo Ho Tai, Jeffrey L. Platt. Rochester, MN; Rochester, MN; Rochester, MN.

P241 **PERIPHERAL SURVIVAL OF MURINE CD4 T CELLS SELECTED IN PORCINE THYMUS GRAFTS. (Abstract #854)**
Jose-Ignacio Rodriguez-Barbosa, Yong Zhao, Gui-Ling Zhao, Sheng-Ping Wang, David H. Sachs, Megan Sykes. Boston, MA.

P242 **ROLE OF PIG CYTOKINES IN INDUCTION OF XENOGENEIC BONE MARROW CHIMERISM IN A DISCORDANT PIG TO MOUSE MODEL. (Abstract #855)**
Muhammad M. Mohiuddin, Yuru Meng, Verdi J. DiSesa. Chicago, IL.

P243 **ACTIVATION OF HUMAN DENDRITIC CELLS BY PORCINE AORTIC ENDOTHELIAL CELLS: TRANSACTIVATION OF NAIVE T CELLS THROUGH CO-STIMULATION AND CYTOKINE GENERATION. (Abstract #856)**
Partha P. Manna, T. Mohanakumar.

Notes

TRANSPLANT 2001
Joint American Transplant Meeting
Day-at-a-Glance, Tuesday, May 15, 2001

6:30 AM - 7:50 AM — **Concurrent Sunrise Symposia**

Page 105 — **Sunrise Symposium I: Infectious Diseases: Prevention, Diagnosis, and Treatment of Fungal Infections after Transplantation**
Sheraton Ballroom 1-3, Sheraton

Page 105 — **Sunrise Symposium II: Liver Donors: Cadaver Allocation Policy**
Sheraton Chicago Ballroom 4-7, Sheraton

Page 105 — **Sunrise Symposium III: Preventing and Treating Type I Diabetes**
Chicago Ballroom 8-10, Sheraton

8:00 AM - 9:00 AM — **Concurrent Symposia**

Page 105 — **Basic Science Symposium: Immune Regulation**
Sheraton Ballroom 1-3, Sheraton

Page 105 — **Clinical Science Symposium: Infectious Complications of Transplantation: Revisiting the Old and Understanding the New**
Sheraton Chicago Ballroom 4-7, Sheraton

9:30 AM — **Break**

10:00 AM - 12:00 PM — **Joint Session**
Sheraton Chicago Ballroom 4-7, Sheraton

10:00 AM — **ASTS Presidential Address**
Page 105 — *Nancy Ascher*

10:35 AM — **AST Presidential Address**
Page 105 — *Mohamed Sayegh*

11:10 AM — **Awards**

12:30 PM - 1:30 PM — **Parallel Luncheon Worshops**
Page 105 — *Sheraton and Intercontinental*

12:30 PM - 1:30 PM — **Selected Open Poster Sessions**
Page 106 — *Sheraton*

2:00 PM - 3:30 PM — **Concurrent Sessions**

Page 107 — **Concurrent Session 41: Basic Science: Antigen Presentation**
Chicago Ballroom 10, Sheraton

Page 107 — **Concurrent Session 42: Kidney Transplantaion: Clinical Immunosuppression**
Chicago Ballroom 6/7, Sheraton

Page 108 — **Concurrent Session 43: Pediatrics II (Kidney)**
Chicago Ballroom 8/9, Sheraton

Page 108 — **Concurrent Session 44: Kidney Transplantation: Allocation and Public Policy**
Sheraton Ballroom 1-3, Sheraton

Page 108 — **Concurrent Session 45: Diagnosis and Prognosis of Acute/Chronic Rejection**
Sheraton Ballroom 4/5, Sheraton

Page 109 — **Concurrent Session 46: Chemokines and Adhesion Molecules in Graft Infiltration**
Empire Room, Intercontinental

Page 109 — **Concurrent Session 47: Small Bowel Transplantation & Donor Procurement/ Modeling**
Exchange Room, Intercontinental

Page 110 — **Concurrent Session 48: Living Liver Donors II**
Grand Ballroom, Intercontinental

Page 110 — **Concurrent Session 49: Experimental Islet Transplantation**
King Arthur Court Ballroom, Intercontinental

Page 111 — **Concurrent Session 50: Liver Transplantation: Hepatitis B**
Renaissance Ballroom, Intercontinental

Page 111 — **Concurrent Session 51: Basic Science: Allorecognition**
Chicago Ballroom 10, Sheraton

Page 112 — **Concurrent Session 52: Outcome Assessment in Renal Transplantation**
Chicago Ballroom 6/7, Sheraton

Page 112 — **Concurrent Session 53: Cadaver Donation: Factors and Outcomes**
Chicago Ballroom 8/9, Sheraton

Page 113 — **Concurrent Session 54: Basic Science: Tolerance II**
Sheraton Ballroom 1-3, Sheraton

Page 113 — **Concurrent Session 55: Pharmacokinetics of Immunosuppressants**
Sheraton Ballroom 4/5, Sheraton

Page 114 — **Concurrent Session 56: Xenotransplantation: Mechanisms, Infectious Issues**
Empire Room, Intercontinental

Page 114 — **Concurrent Session 57: Infection and Neoplasms after Organ Transplantation**
Exchange Room, Intercontinental

Page 115 — **Concurrent Session 58: Fulimant Hepatic Failure**
Grand Ballroom, Intercontinental

Page 115 — **Concurrent Session 59: Genetic Modulation**
King Arthur Court Ballroom, Intercontinental

Page 116 — **Concurrent Session 60: Liver Transplantation: Infection/Immunology**
Renaissance Ballroom, Intercontinental

8:00 AM - 7:00 PM	**Poster Session III**
5:30 PM - 7:00 PM	**Presenters in Attendance** *Beer and Pretzel Reception* *River Exhibition Hall*
Page 116	**Kidney - Acute/Chronic Rejection III**
Page 117	**Kidney - GVH, Complications, Infections III**
Page 118	**Kidney - Immunosuppression A III**
Page 118	**Kidney - Immunosuppression B III**
Page 119	**Kidney - Pediatrics, Recurrent Disease III**
Page 119	**Kidney - Preservation, Donation/Allocation, Economics/Public Policy, Surgical Techniques, and Other III**
Page 120	**Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics III**
Page 120	**Liver - Infections, Complications, Recurrent Disease, Surgical Techniques III**
Page 121	**Liver - Preservation, Economics/Public Policy, Donation Allocation, Other III**
Page 122	**Pancreas and Islets - All Topics III**
Page 122	**Heart/Lung - All Topics III**
Page 123	**Bone Marrow - All Topics III**
Page 123	**Immunosuppression, Preclinical Studies III**
Page 124	**Tolerance III**
Page 124	**Acute/Chronic Rejection III**
Page 125	**Allorecognition, Antigen Presentation, Co-Stimulation and Other III**
Page 125	**Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines III**
Page 126	**Genetic Modulation, Islet Cell Transplantation and Bone Marrow/GVH III**
Page 127	**Tissue Injury, Preservation III**
Page 127	**Xenotransplantation III**
5:45 PM	**AST Business Meeting**
Page 116	*Sheraton Ballroom 1-3, Sheraton*

Tuesday, May 15, 2001

Concurrent Sunrise Symposia

6:30 AM - 7:50 AM

Sunrise Symposium I: Infectious Diseases: Prevention, Diagnosis, and Treatment of Fungal Infections after Transplantation

Sheraton Chicago Ballroom 1-3, Sheraton
Chairs: Carlos Paya

6:30 AM How the pathogenesis of fungal infection impacts its prevention
Carlos Paya
6:55 AM Old and new tools for diagnosis of fungal infection
Barbara D. Alexander
7:20 AM Treatment and new anti-fungal agents
Jay Fishman

Sunrise Symposium II: Liver Donors: Cadaver Allocation Policy

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: John Roberts

6:30 AM Liver allocation: Why the rules should change
John Roberts
6:50 AM Liver allocation policy at UNOS
Richard Freeman
7:10 AM Continous disease severity scales
Russell Wiesner
7:30 AM Application of MELD model to patients waiting on the UNOS list
Erick B. Edwards

Sunrise Symposium III: Preventing and Treating Type I Diabetes

Supported by an unrestricted educational grant from the Juvenile Diabetes Research Foundation

Chicago Ballroom 8-10, Sheraton
Chairs: James Shapiro

6:30 AM Update on clinical trials of islet transplantation
Bernhard Hering
6:50 AM Treatment trials for new onset diabetes
Kevan Herold
7:10 AM Genetics and gene therapy for type I diabetes
Ron Crystal
7:30 AM Generating a new supply of islets
Alberto Hayek

Concurrent Symposia

8:00 AM - 9:30 AM

Basic Science Symposium: Immune Regulation

Sheraton Ballroom 1-3, Sheraton
Chairs: David Briscoe and Elizabeth Field

8:00 AM Homeostasis of naïve and memory T cell
Charlie Surh
8:30 AM Mechanisms of T cell mediated suppression
Herman Waldmann
9:00 AM Regulatory T cells in transplantation tolerance
Jerzy Kupiec-Weglinski

Clinical Science Symposium: Infectious Complications of Transplantation: Revisiting the Old and Understanding the New

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: Michael Lucey and Geraldine Miller

8:10 AM Management of the hepatic viruses: Should we use the donor and how to treat the virus positive recipient before and after
Hugo Rosen
8:20 AM Biology and management of EBV/PTLD
Jutta Prieksaitis
8:40 AM Polyoma virus and its consequesnces
Parmjeets Randhawa
9:00 AM Pneumocystis prophylaxis
Robin Avery
9:20 AM Immunizations for transplant recipients
Margaret Burroughs
9:30 AM Break

Joint Session

10:00 AM - 12:00 PM

10:00 AM ASTS Presidential Address
Nancy Ascher
10:35 AM AST Presidential Address
Mohamed Sayegh
11:10 AM Awards
12:00 PM Break

Parallel Luncheon Workshops

12:30 PM - 1:30 PM

Room location not available at time of publication. Locations will be printed on the tickets and in the mini-program. Be sure to check hotel location.

46. What can the internet do for you?
Robert Merion and Kim Solez
47. T cell memory
Fadi Lakkis and Andrew Wells
48. Managing the highly sensitized patient
Ronald Kerman and Stanley Jordan
49. Minimizing immunosuppression
Lorenzo Gallon and Donald Hricik
50. Gene array technology to monitor post-transplant events
Evner Akalin and Terry Strom
51. The tolerance assay: Where do we stand
Peter Heeger and Ann VanBuskirk
52. Optimal immunosuppression for children
William Harmon and Robert Ettenger
53. Role of CD8+ cells
Kathryn Wood and Christian Larsen
54. Recent advances in immune profiling
Adriana Zeevi and Nancy Reinsmoen
55. Composite tissue grafts
Scott Gruber and Nadey Hakim
56. The marginal donor
J. Wesley Alexander and Edward Alfey
57. Strategies for non-myeloablative conditioning
Judith Thomas and Megan Sykes
58. Characterization of regulatory T cells
Elizabeth Field and Jeffrey Bluestone
59. Tissue injury as determinant of graft outcome
Philip Halloran and Nicholas Tilney
60. Initial cardiac evaluation of kidney recipients
Connie Manske and David Cohen

61. **Regulation of xenotransplantation**
Hugh Auchincloss and Daniel Salomon
62. **Stem cells - An answer to pancreatic islet shortage**
Susan Bonner-Weir and VJ Ramiya
63. **Mechansims of chronic rejection**
Joren Madsen and Anil Chandraker
64. **The renin angiotensin system: Interface between physiology and immunology**
Thomas Coffman and Harold McKenzie
65. **Complications of renal transplantation: Interactive case presentation**
Roy First and Daniel Brennan
66. **Complications of liver transplantation**
Russell Weisner and John Lake
67. **Complications of pancreas transplantation**
Robert Stratta and Steven Bartlett
68. **Small bowel transplantation**
David Grant and Kareem Abu-Elmadg
69. **The campath antibodies**
Herman Waldman and Peter Friend

Selected Poster Open Sessions

12:30 PM - 1:30 PM

Selected Posters on Liver Transplantation

Sheraton Ballroom 1-3, Sheraton
Chairs: Andreas Tzakis and Byers Shaw

12:30 PM **ARTERIAL AND PORTAL VENOUS RECONSTRUCTION AFTER EX VIVO *SPLIT LIVER TRANSPLANTATION.* (Abstract #709)**
Huda M. Noujaim, Brigitte B. Gunson, Daniel Candinas, David A. Mayer, John A.C. Buckels, Paul McMaster, John De Ville De Goyet, Darius F. Mirza. Birmingham, United Kingdom; Birmingham, United Kingdom.

12:35 PM **MONOSEGMENT GRAFT IN LIVING DONOR LIVER TRANSPLANTATION. (Abstract #289)**
Fumitaka Oike, Seisuke Sakamoto, Mureo Kasahara, Tetsuya Kiuchi, Hiroto Egawa, Shinji Uemoto, Koichi Tanaka. Kyoto, Kyoto, Japan.

12:40 PM **APPARENT SLOWING OF HEPATITIS C RECURRENCE AND PROGRESSION AFTER LIVER TRANSPLANTATION USING VERY EARLY (14d) STEROID WITHDRAWAL. (Abstract #703)**
Nancy Stolpman, Janet Stephens, Thomas Trouillot, James Trotter, Marcelo Kugelmas, Michael Wachs, Thomas Bak, Tracy Steinberg, Igal Kam, Gregory Everson. Denver, CO.

12:45 PM **GERIATRIC ORTHOTOPIC LIVER TRANSPLANTATION (OLT): A RETROSPECTIVE OUTCOMES REPORT FROM A LARGE SINGLE CENTER REVIEW INVOLVING PATIENTS GREATER THAN 70 YEARS OF AGE. (Abstract #700)**
G. Tzimas, G. W. Neff, O. Hung, D. Weppler, D. Levi, S. Nishida, J. Tector, L. Kravetz, J. R. Nery, T. Kato, C. O'Brien, K. R. Reddy, E. R. Schiff, A. G. Tzakis. Miami, Fl; Miami, Fl.

12:50 PM **NITRIC OXIDE AND ISCHAEMIC PRECONDITIONING OF THE LIVER. (Abstract #1148)**
Rahul S. Koti, Wenxuan Yang, Janice Tsui, Alexander M. Seifalian, Brian R. Davidson. London, United Kingdom.

12:55 PM **RECONSIDERING THE IMPACT OF COLD ISCHEMIA TIME ON GRAFT AND PATIENT SURVIVAL AFTER LIVER TRANSPLANTATION. (Abstract #296)**
David M. Levi, Seigo Nishida, Tomoaki Kato, Jose Nery, Guy Neff, Les Olson, Andreas G. Tzakis. Miami, FL.

1:00 PM **SPLIT IN SITU LIVER GRAFTS AND DONORS' AGE. (Abstract #1153)**
Umberto Maggi, Giorgio Rossi, Paolo Reggiani, Lucio Caccamo, Stefano Gatti, Gianni Paone, Ernesto Melada, Maurizio Doglia, Alessia De Simone, Simone Olmetti, Paolo Trezza, Luigi Rainero Fassati. Milano, Italy.

1:05 PM **100 DONOR EVALUATIONS AND SURGICAL OUTCOME AFTER LIVE DONOR ADULT LIVER TRANSPLANTATION (LDALT). (Abstract #299)**
Elizabeth A. Pomfret, James J. Pomposelli, David L. Burns, Fredric D. Gordon, William D. Lewis, Roger L. Jenkins. Burlington, MA.

Selected Posters on Allorecognition/Tissue

Chicago Ballroom 8/9, Sheraton
Chairs: Jonathan Bromberg and Osama Gaber

12:30 PM **INDUCTION OF HEME OXYGENASE-1 IMPROVES RAT LIVER TRANSPLANTATION SURVIVAL BY INHIBITING APOPTOSIS. (Abstract #410)**
Claudio A. Redaelli, Ying-Hua Tian, Martin K. Schilling, Jean-Francois Dufour. Bern, Switzerland; Bern, Switzerland.

12:35 PM **LOCALIZATION AND KINETICS OF INTERLEUKIN-6 (IL-6) DURING ISCHEMIA-REPERFUSION INJURY (IRI) OF PORCINE INTESTINE. (Abstract #408)**
Felix Braun, Mehdi Hosseini, Sven Laabs, Burckhard Sattler, Eberhard Wieland, Burckhardt Ringe. Göttingen, Germany; Göttingen, Germany; Göttingen, Germany.

12:40 PM **SELECTIVE OVER-EXPRESSION OF INFLAMMATORY MOLECULES IN HEARTS FROM BRAIN-DEAD RATS MAINTAINED WITH ADEQUATE CIRCULATION. (Abstract #390)**
Leigh D. Segel, Derek W. von Haag, Jie Zhang, David M. Follette. Davis and Sacramento, CA.

12:45 PM **DENDRITIC CELLS DERIVED FROM LIVER B CELL LINEAGE INDUCE T CELL APOPTOSIS THROUGH CASPASE ACTIVATION, BUT NOT VIA FAS OR TNF LIGATION. (Abstract #817)**
Xaioyan Liang, Shiguang Qian, Jinrong Li, C. Andrew Bonham, John J. Fung, Lina Lu. Pittsburgh, PA.

12:50 PM **COMPARATIVE ANALYSIS OF MIGRATORY RESPONSES OF LYMPHOID AND MYELOID DENDRITIC CELLS TO CC CHEMOKINES AND IN ALLOGENEIC RECIPIENTS. (Abstract #387)**
Bridget L. Colvin, Adrian E. Morelli, Angus W. Thomson. Pittsburgh, PA; Pittsburgh, PA.

12:55 PM **THE MIGRATION OF DENDRITIC CELLS IS CLOSELY ASSOCIATED WITH THE EXPRESSION OF THE ACTIN BUNDLING PROTEIN FASCIN. (Abstract #809)**
Kenneth A. West, Patricia Colp, Monther Al-Alwan, Giusepe De Panfilis, Mieke Mommaas, Geoff Rowden. Halifax, Canada; Brescia, Italy; Leiden, The Netherlands.

1:00 PM **IFN-g REGULATES ENDOTHELIAL CELL PRODUCTION OF CHEMOKINES. (Abstract #813)**
Hirohito Kobayashi, Shoji Koga, Hiroshi Toma, Andrew C. Novick, Robert L. Fairchild. Cleveland, OH; Tokyo, Japan.

1:05 PM **TRANSCRIPTIONAL PROFILING OF PRIMATE RENAL TRANSPLANTS: MOTIFS DIFFERENTIATING REPERFUSION FROM ALLOSPECIFIC IMMUNITY. (Abstract #374)**
Margot O'Toole, Holly Swiniarski, Sean P. Montgomery, Eric A. Elster, He Xu, Robert L. Kampen, Andrew J. Dorner, Allan D. Kirk. Andover, MA; Bethesda, MD.

Concurrent Session 41: Basic Science: Antigen Presentation

2:00 PM - 3:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Charles Orosz and Gilles Benichou

2:00 PM **UNPRIMED CD8+ T CELLS ARE ACTIVATED BY RESTING VASCULAR ENDOTHELIUM TO INDUCE ENDOTHELIAL CELL APOPTOSIS. (Abstract #857)**
Alexander S. Krupnick, Daniel Kreisel, Wilson Y. Szeto, Sicco H. Popma, Bruce R. Rosengard. Philadelphia, PA.

2:10 PM **INTERACTIONS AMONG RECIPIENT MONOCYTES AND DONOR ENDOTHELIUM IN THE MAINTENANCE OF THE INDIRECT PATHWAY OF ALLORECOGNITION. (Abstract #858)** *Young Investigator Award*
Dmitry V. Samsonov, Christopher S. Geehan, Mark D. Denton, Gilles Benichou, Ana Maria Waaga, Mohamed H. Sayegh, David M. Briscoe. Boston, MA; Boston, MA.

2:20 PM **T-CELL AND MONOCYTE INTERDEPENDENCE DURING ALLOIMMUNE RESPONSES. (Abstract #859)**
He Xu, Douglas K. Tadaki, Patrick J. Blair, Francis Cruzata, David M. Harlan, Allan D. Kirk.

2:30 PM **CD8α+ *(LYMPHOID-RELATED)* AND CD8α *(MYELOID)* DENDRITIC CELL SUBSETS DIFFERENTIALLY REGULATE ORGAN ALLOGRAFT SURVIVAL. (Abstract #860)** *Young Investigator Award*
Peta J. O'Connell, Wei Li, Takuya Takayama, Shiguang Qian, Alison J. Logar, Angus W. Thomson. Pittsburgh, PA; Pittsburgh, PA.

2:40 PM **IDENTIFICATION OF THE DENDRITIC CELL SUBSET THAT PRESENTS ANTIGEN TO NAÏVE CD4 T CELLS IN VIVO. (Abstract #861)**
Deborah Ulman, Marc K. Jenkins, Elizabeth Ingulli. Minneapolis, MN; Minneapolis, MN.

2:50 PM **T-LYMPHOCYTES ACTIVATED BY DONOR IMMATURE DENDRITIC CELLS SUPPRESS RECIPIENTS' ANTIGEN-PRESENTING CELLS BY DIRECT CELL-CELL CONTACT. (Abstract #862)**
Wei-Chen Lee, Yang-Jen Chiang, Hui-Chuan Wang, Chen-Rong Lia, Pei-Fang Huang, Long-Bin Jeng, Miin-Fu Chen, Lina Lu, Shiquan Qian. Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Pittsburgh, PA; Pittsburgh, PA.

3:00 PM **ALLOSTIMULATORY CAPACITY OF WILD TYPE, IL-12 DEFICIENT, AND CD40 DEFICIENT DENDRITIC CELLS. (Abstract #863)**
Jeffery E. Mold, D. Keith Bishop. Ann Arbor, MI.

3:10 PM **RETROVIRAL DELIVERY OF TGFβ1 TO ALLOGENEIC MYELOID DENDRITIC CELLS: INHIBITION OF T CELL PRIMING ABILITY AND INFLUENCE ON GRAFT SURVIVAL (Abstract #864)**
Takuya Takayama, Adrian E. Morelli, Wei Li, Shiguang Qian, Hideaki Tahara, Angus W. Thomson. Pittsburgh, PA; Tokyo, Japan.

3:20 PM **A NOVEL CELLULAR TARGET FOR SALICYLATES: ASPIRIN INHIBITS THE MATURATION AND IN VITRO AND IN VIVO FUNCTION OF MURINE MYELOID DENDRITIC CELLS. (Abstract #865)**
Holger Hackstein, Adrian E. Morelli, Adriana T. Larregina, Raymond W. Ganster, Glenn D. Papworth, Alison J. Logar, Simon C. Watkins, Louis D. Falo, Angus W. Thomson. Pittsburgh, PA; Pittsburgh, PA; Pittsburgh, PA; Giessen, Germany.

Concurrent Session 42: Kidney Transplantation: Clinical Immunosuppression

2:00 PM - 3:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: John Pirsch and Elmahdi Elkhammas

2:00 PM **PRAVASTATIN IMPROVES LONG-TERM GRAFT AND PATIENT SURVIVAL AFTER KIDNEY TRANSPLANTATION – AN EXAMINATION OF FIVE-YEAR FOLLOW-UP DATA. (Abstract #866)**
Steven Katznelson, Alan H. Wilkinson, Gabriel M. Danovitch. San Francisco, CA; Los Angeles, CA.

2:10 PM **MMF BASED IMMUNOSUPPRESSION AND CYTOKINE GENOTYPES: EFFECTS ON MONOKINE SECRETION AND ANTIGEN PRESENTATION IN LONG-TERM RENAL TRANSPLANT RECIPIENTS. (Abstract #867)**
Rolf Weimer, Janis Mytilineos, Andreas Feustel, Astrid Preiss, Volker Daniel, Helmut Grimm, Thomas Zimmermann, Manfred Wiesel, Gerd Staehler, Gerhard Opelz. Giessen, Germany; Heidelberg, Germany; Giessen, Germany; Heidelberg, Germany.

2:20 PM **A MULTIVARIATE ANALYSIS OF DISCHARGE IMMUNOSUPPRESSION AND POST-TRANSPLANT MALIGNANCY. (Abstract #868)**
Wida S. Cherikh, H. M. Kauffman, Francis L. Delmonico. Richmond, VA; Boston, MA.

2:30 PM **A LARGE, MULTICENTRE, THREE-ARM STUDY COMPARING IMMEDIATE TACROLIMUS THERAPY, ATG/TACROLIMUS THERAPY, AND ATG/CYCLOSPORIN THERAPY IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #869)**
Michèle Kessler, the Tacrolimus versus microemulsified Cyclosporin Study Group. Vandoeuvre les Nancy, France.

2:40 PM **A CONTEMPORANEOUS COMPARISON OF MMF AND AZA IN THE USRDS DATA BASE. (Abstract #870)**
Mark A. Schnitzler, Karen E. Craig, Robert S. Woodward, Jeffrey A. Lowell, Daniel C. Brennan. St. Louis, MO; Little Rock, AR.

2:50 PM **SUCCESSFUL ABO-INCOMPATIBLE LIVING KIDNEY TRANSPLANTATIONS AFTER SUFFICIENT IMMUNOSUPPRESSION WITH CYCLOPHOSPHAMIDE AND MIZORIBINE. (Abstract #871)**
Kazuharu Uchida, Yoshihiro Tominaga, Toshito Haba, Akio Katayama, Tetsu Sato, Izuru Watanabe, Hiroko Inagaki, Takaaki Kobayashi, Asami Takeda, Kunio Morozumi, Hiroshi Takagi. Nagoya, Japan; Nagoya, Japan; Nagoya, Japan.

3:00 PM **EFFICACY OF EON CYCLOSPORINE CAPSULES COMPARED TO NEORAL CAPSULES IN STABLE RENAL TRANSPLANT RECIPIENTS. (Abstract #872)**
Iman Bajjoka, Kerry Estes, Viken Douzdjian. Detroit, MI; Gainesville, FL; Detroit, MI.

3:10 PM **COMPARISON OF GRAFT OUTCOMES AFTER KIDNEY TRANSPLANT FROM RELATED AND UNRELATED LIVING DONORS UNDER MYCOPHENOLATE MOFETIL PROCOTOLS. (Abstract #873)**
Eytan Mor, Archil Chkhotua, Tirza Klein, Eti Shabtai, Alexander Yussim, Nathan Bar-Nathan, Ezra Shaharabani, Zaki Shapira. Petah-Tiqva, Israel; Tbilisi, Georgia; Petah-Tiqva, Israel.

3:20 PM **KIDNEYS OF NON-HEART BEATING CADAVERS PROVIDED AN EXCELLNT GRAFT SURVIVAL UNDER THE IMMUNOTHERAPY STARTED WITH LOW DOSE CSA. (Abstract #874)**
Yusuke Kubota, Ryoichi Shiroki, Hitomi Sasaki, Toru Ito, Kiyohito Ishikawa, Masanobu Izumitani, Kiyotaka Hoshinaga. Toyoake, Aichi, Japan.

Concurrent Session 43: Pediatrics II (Kidney)

2:00 PM - 3:30 PM

Chicago Ballroom 8/9, Sheraton
Chairs: Mark Benfield and Matthew R. Weir

2:00 PM **FIRST, MULTICENTRE, COMPARATIVE TRIAL OF TACROLIMUS THERAPY VS. MICROEMULSIFIED CYCLOSPORIN THERAPY IN PAEDIATRIC KIDNEY TRANSPLANT RECIPIENTS. (Abstract #875)**
Richard Trompeter, the Tacrolimus Study Group in Paediatric Renal Transplantation. London, United Kingdom.

2:10 PM **OLESTRA DECREASES CYCLOSPORINE ABSORPTION AND TOTAL EXPOSURE THAT IS NOT REFLECTED BY TROUGH CONCENTRATIONS. (Abstract #876)**
K. Troy Somerville, Jenifer Lill, Cynthia J. Terrill, Joseph R. Sherbotie. Salt Lake City, UT; Seattle, WA; Salt Lake City, UT.

2:20 PM **PROMISING EARLY OUTCOMES WITH A NOVEL, COMPLETE STEROID AVOIDANCE IMMUNOSUPPRESSION PROTOCOL IN PEDIATRIC RENAL TRANSPLANTATION. (Abstract #877)**
Minnie Sarwal, Peter Yorgin, Steve Alexander, Maria Millan, Kevin Lemley, Amir Belson, Stella Chang, Pamela Orlandi, Oscar Salvatierra. Stanford, CA.

2:30 PM **CYTOKINE GENE EXPRESSION DURING COMPLETE STEROID AVOIDANCE IN PEDIATRIC RENAL TRANSPLANTATION. (Abstract #878)**
Amir Belson, Stella Chang, Oscar Salvatierra, Minnie Sarwal. Stanford, CA; Stanford, CA.

2:40 PM **DEFINITION OF INTRAGRAFT AND PERIPHERAL BLOOD GENE EXPRESSION (GE) RANGES IN RENAL ALLOGRAFTS BY TAQMAN BASED PCR (T-PCR) QUANTIFICATION. (Abstract #879)**
Juergen Strehlau, Michael Melter, Gisela Offner, Christian v. Schnakenburg, Kay Latta, Bjoern Nashan, Jochen H.H. Ehrich. Hannover, Germany.

2:50 PM **A REJECTION-FREE MILEU PROVIDES ACCELERATED GROWTH, BETTER GRAFT FUNCTION AND SUPERIOR GRAFT SURVIVAL IN CHILDREN. (Abstract #880)**
William E. Harmon, Donald M. Stablein, Amir Tejani. Boston, MA; Potomac, MD; Valhalla, NY.

3:00 PM **THE CONTINUING IMPACT OF ACUTE REJECTION ON CHRONIC REJECTION GRAFT LOSS. (Abstract #881)**
Amir Tejani, Ping-Leung Ho, Lea Emmett, Donald M. Stablein. Valhalla, NY; Potomac, MD.

3:10 PM **rhGH POST-TRANSPLANT: ONE YEAR RANDOMIZED CONTROL STUDY OF NAPRTCS. (Abstract #882)**
Richard N. Fine, Donald N. Stablein, Arthur Cohen, Edward C. Kohaut, Amir Tejani.

3:20 PM **PRE-TRANSPLANT PERITONEAL DIALYSIS (PD) AND GRAFT THROMBOSIS (GT) FOLLOWING PEDIATRIC RENAL TRANSPLANTATION. (Abstract #883)**
William E. Harmon, Ruth A. McDonald, Donald Stablein. Boston, MA; Seattle, WA; Bethesda, MD.

Concurrent Session 44: Kidney Transplantation: Allocation and Public Policy

2:00 PM - 3:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: James Wynn

2:00 PM **RACIAL DIFFERENCES, BY STATE, IN ACCESS TO WAITLISTING AND RENAL TRANSPLANTATION IN THE UNITED STATES. (Abstract #884)**
Valarie B. Ashby, Alan B. Leichtman, Robert A. Wolfe, Akinlolu O. Ojo, Robert M. Merion, Eric W. Young, Philip J. Held, Friedrich K. Port. Ann Arbor, MI; Ann Arbor, MI.

2:10 PM **DOES KIDNEY ALLOCATION BASED ON HLA MATCHING ADVERSELY AFFECT MINORITY ACCES TO TRANSPLANTATION? (Abstract #885)**
Charles T. Van Buren, Teresa Shafer. Houston, TX.

2:20 PM **ANALYSIS OF HLA "BLANKS" IN VARIOUS ETHNIC GROUPS OF PATIENTS ON THE OPTN/UNOS CADAVERIC KIDNEY WAITING LIST. (Abstract #886)**
S. Vaidya, W. S. Cherikh, A. Ting, D. A. Distant. Galveston, TX; Richmond, VA; Brooklyn, NY.

2:30 PM **RACE, NOT HLA MATCHING IS THE MOST IMPORTANT PREDICTOR OF LONG TERM GRAFT SURVIVAL IN US CADAVERIC RENAL TRANSPLANT RECIPIENTS. (Abstract #887)**
Ross B. Isaacs, Steven L. Nock, Clint E. Spencer, Peter I. Lobo. Charlottesville, VA; Charlottesville, VA.

2:40 PM **VARIABILITY IN PHYSICIAN ATTITUDES TOWARDS LISTING FOR RENAL TRANSPLANTATION. (Abstract #888)**
Juan Palma, John P. Vella, Jonathan Himmelfarb, James F. Whiting. Portland, ME; Portland, ME.

2:50 PM **HIGHER RATES OF WAITLISTING DO NOT CORRELATE WITH LOWER RATES OF RENAL TRANSPLANTATION AMONG WAITLISTED PATIENTS. (Abstract #889)**
Alan B. Leichtman, Valarie B. Ashby, Robert A. Wolfe, Akinlolu O. Ojo, Robert M. Merion, Eric W. Young, Friedrich K. Port, Philip J. Held. Ann Arbor, MI; Ann Arbor, MI.

3:00 PM **INSURANCE COVERAGE FOR RENAL TRANSPLANTATION: DOES PAYOR TYPE MAKE A DIFFERENCE? (Abstract #890)**
Lisa R. Raiz, Elizabeth A. Davies, Ronald M. Ferguson. Athens, OH; Columbus, OH.

3:10 PM **COMPARISON OF THE SURVIVAL OF IMPORTED AND LOCALLY SHARED CADAVERIC RENAL ALLOGRAFTS IN THE UNITED STATES (U.S.). (Abstract #891)**
Kevin C. Mange, Jude Maghirang, Wida Cherikh, Roy D. Bloom. Philadelphia, PA; Philadelphia, PA; Richmond, VA.

3:20 PM **FIVE YEAR CADAVER RENAL TRANSPLANT OUTCOME IN THE UK. (Abstract #892)**
R. J. Johnson, J. D. Briggs, M. A. Belger. Bristol, United Kingdom.

Concurrent Session 45: Diagnosis and Prognosis of Acute/Chronic Rejection

2:00 PM - 3:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Douglas Norman and Kim Solez

2:00 PM **mRNA PROFILING OF URINARY CELLS IS PREDICTIVE, DIAGNOSTIC AND PROGNOSTIC OF RENAL ALLOGRAFT STATUS. (Abstract #893)**
Baogui Li, Kouzaburo Yamaji, Darshana Dadhania, Choli Hartono, Jin M. Kong, Ruchuang Ding, Vijay K. Sharma, David Serur, Joseph E. Schwartz, Manikkam Suthanthiran. New York, NY; Stony Brook, NY.

2:10 PM **GRAFT LOSS BY CHRONIC REJECTION CAN BE PREDICTED BY PROXIMAL TUBULAR DYSFUNCTION IN PATIENTS WITH GOOD AND STABLE RENAL FUNCTION 3 YEARS IN ADVANCE. (Abstract #894)**
Alvaro Pacheco-Silva, Niels O. Saraiva Câmara, Marcelo S. Silva, Jose O. Medina Pestana, Sonia Nishida, Aparecido B. Pereira. Sao Paulo, Sao Paulo, Brazil.

2:20 PM **CLINICAL FACTORS ASSOCIATED WITH DONOR-DIRECTED MEMORY T-CELL REACTIVITY MEASURED BY THE ELISPOT ASSAY IN RENAL TRANSPLANT RECIPIENTS. (Abstract #895)**
Donald E. Hricik, Hany H.S. Anton, Atallah Aymen, James A. Schulak, Peter S. Heeger. Cleveland, OH; Cleveland, OH; Cleveland, OH.

2:30 PM **PREVALENCE OF CHRONIC HUMORAL REJECTION IN CHRONIC RENAL ALLOGRAFT DYSFUNCTION. (Abstract #896)**
Tom Theruvath, Susan Saidman, Nina Rubin, Winfred Williams, Shamila Mauiyyedi, Bernard Collins, Robert Colvin, Francis Delmonico, A. Benedict Cosimi, Manuel Pascual.

2:40 PM **NON-INVASIVE EARLY DIAGNOSIS AND SUBSEQUENT MONITORING OF RENAL ALLOGRAFT REJECTION USING URINE NMR SPECTRA: A PRELIMINARY ANALYSIS. (Abstract #897)**
David Rush, Ray Somorjai, Roxanne Deslauriers, Miriam Glogowski, John Jeffery, Peter Nickerson. University of Manitoba, Winnipeg; National Research Council of Canada, Winnipeg.

2:50 PM **SHOULD A TRANSPLANTED LIVING DONOR KIDNEY FUNCTION LIKE IT'S DONOR KIDNEY PAIR? (Abstract #898)**
Jorge A. Velosa, Timothy S. Larson, Thomas R. Schwab, James M. Gloor, Matthew D. Griffin, Sylvester Sterioff, Erik J. Bergstralh, Carrie L. Loebertmann, Mark D. Stegall. Rochester, MN.

3:00 PM **DONOR CYTOKINE GENE POLYMORPHISMS ARE ASSOCIATED WITH INCREASED GRAFT LOSS AND DYSFUNCTION AFTER TRANSPLANT. (Abstract #899)**
David Kahan, Travis Mason, Nipa Gandhi, David Goldman, Ann Marie Melanson, Beth Horth, Michelle Dixon, Stacey Supran, Robert Salomon, Francis L. Delmonico, Kevin O' Connor, Richard J. Rohrer, Richard B. Freeman. Boston, MA; Newton, MA; Boston, MA.

3:10 PM **FLOW CYTOMETRIC PRA DETECTS SENSITIZED PATIENTS UNDETECTED BY AHG-PRA. (Abstract #900)**
M. Karpinski, D. Rush, J. Jeffery, D. Pochinco, S. Dancea, P. Birk, P. Nickerson. Winnipeg, MB, Canada.

3:20 PM **USING CYTODIAGNOSTIC URINALYSIS (CDU) TO MONITOR RENAL ALLOGRAFT STATUS. (Abstract #901)**
Jimmy A. Light, Carole A. Allston. Washington, DC; Washington, DC.

Concurrent Session 46: Chemokines and Adhesion Molecules in Graft Infiltration

2:00 PM - 3:30 PM

Empire Room, Intercontinental
Chairs: Robert Fairchild and Anita Chong

2:00 PM **HOW A GRAFT CONTRIBUTES TO ITS OWN DESTRUCTION. (Abstract #902)**
Wayne W. Hancock, Wei Gao, Vilmos Csizmadia, Kerrie L. Faia, Nida Shemmeri, Andrew D. Luster. Cambridge, MA; Boston, MA.

2:10 PM **IFN-INDUCIBLE CHEMOKINES RELEASED BY INTESTINAL T CELLS ENHANCE INTESTINAL ALLOGRAFT REJECTION. (Abstract #903)**
Jonathan P. Fryer, Zheng J. Zhang, Levent Kaptanoglu, Alnadjim Ziad, David Ivancic, Joe R. Leventhal, Dixon B. Kaufman, Mike A. Abecassis, Frank P. Stuart, Terrance A. Barrett. Chicago, IL.

2:20 PM **A LYTIC IP-10/Fc FUSION PROTEIN INDUCES LONG-TERM SURVIVAL OF ISLET ALLOGRAFTS IN MICE. (Abstract #904)**
Alberto Sanchez-Fueyo, Daya Papalkar, Sylvie Ferrari Lacraz, Yan Tian, Terry B. Strom, Xin Xiao Zheng. Boston, MA.

2:30 PM **NEUTRALIZATION OF MONOKINE INDUCED BY IFN-g, MIG, PREVENTS DEVELOPMENT OF CHRONIC ALLOGRAFT VASCULOPATHY. (Abstract #905)**
Masayoshi Miura, Qiwei Zhang, Robert L. Fairchild. Cleveland, OH.

2:40 PM **ACTIVE CELL MIGRATION AND SELECTIVE CHEMOKINE/RECEPTOR GENE EXPRESSION IN RAT CARDIAC ALLOGRAFTS DEVELOPING TRANSPLANT VASCULOPATHY. (Abstract #906)**
Satoru Kitagawa-Sakakida, Kei Horiguchi, Zhan-Zhuo Li, Masayuki Tori, Hikaru Matsuda, Ryota Shirakura. Osaka, Japan; Osaka, Japan.

2:50 PM **HOMING OF IN VITRO GENERATED DONOR ANTIGEN-REACTIVE CD4 T LYMPHOCYTES TO RENAL ALLOGRAFTS IS VLA-4 BUT NOT LFA-1 DEPENDENT. (Abstract #907)**
Markus H. Hammer, Yuan Zhai, Masamichi Katori, Thomas Ritter, Hans-Dieter Volk, Ronald W. Busuttil, Ana J. Coito, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Berlin, Germany.

3:00 PM **ALLOSPECIFIC CD8+ CTL UPREGULATE CD103 EXPRESSION SUBSEQUENT TO ENTRY INTO RENAL ALLOGRAFTS AND REMAIN SEQUESTERED AT THE GRAFT SITE. (Abstract #908)**
Donghua Wang, Benjamin Philosophe, Stephen T. Bartlett, Gregg A. Hadley. Baltimore, MD.

3:10 PM **CRITICAL ROLE OF CD103 IN PROMOTING ALLOGRAFT DESTRUCTION BY CD8+ T CELLS. (Abstract #909)** *Young Investigator Award*
Ye Feng, Gregg A. Hadley. Baltimore, MD.

3:20 PM **P-SELECTIN GLYCOPROTEIN LIGAND-1 (rPSGL-Ig) MEDIATED BLOCKADE OF ENDOTHELIAL CD62 MOLECULE IN COMBINATION WITH LOW DOSE CYCLOSPORINE PROTECTS LIVER ALLOGRAFTS FROM ACUTE REJECTION. (Abstract #910)**
Farin F. Amersi, Douglas G. Farmer, Xiu-Da Shen, Feng Gao, Judy Melinek, Jeffrey Ma, Jerzy W. Kupiec-Weglinski, Gray P. Shaw, Ronald W. Busuttil. Los Angeles, CA; Los Angeles, CA; Cambridge, MA.

Concurrent Session 47: Small Bowel Transplantation & Donor Procurement/Modeling

2:00 PM - 3:30 PM

Exchange Room, Intercontinental
Chairs: Alan Langnas and Lawrence Hunsicker

2:00 PM **EARLY LIVING RELATED SEGMENTAL BOWEL TRANSPLANTATION AS THERAPY FOR TRAUMA INDUCED IRREVERSIBLE INTESTINAL FAILURE. (Abstract #911)**
Luca Cicalese, Pierpaolo Sileri, Massimo Asolati, Cristiana Rastellini, Marie F. Pasqual, Noreen T. Coady, Herand Abcarian, Enrico Benedetti. Chicago, IL.

2:10 PM **A NOVEL IMMUNOMODULATORY STRATEGY FOR CLINICAL INTESATINAL TRANSPLANTATION: EX-VIVO IRRADIATION WITH ADJUNCT DONOR BONE MARROW INFUSION. (Abstract #912)**
Kareem M. Abu-Elmagd, Jorge D. Reyes, Geoffrey J. Bond, Noriko Murase, Tong Wu, Anthony Demetris, Abdul S. Rao, Melvin Deutch, John J. Fung, Thomas E. Starzl. Pittsburgh, PA; Pittsburgh, PA.

2:20 PM **GRAFT VERSUS HOST DISEASE (GVHD) IN INTESTINAL ALLOGRAFT RECIPIENTS. (Abstract #913)**
George V. Mazariegos, Kareem Abu-Elmagd, Ronald Jaffe, John Peters, Jorge Reyes.

2:30 PM **SIROLIMUS RESCUE THERAPY IN PEDIATRIC LIVER AND INTESTINE TRANSPLANT RECIPIENTS ON TACROLIMUS BASED IMMUNOSUPPRESSION. (Abstract #914)**
Naveen Mittal, Tomoaki Kato, Spiros Delis, Barbara Miller, John Thompson, Andreas Tzakis. Miami, FL; Miami, FL.

2:40 PM **INTESTINAL TRANSPLANTATION BEFORE AND AFTER THE INTRODUCTION OF SIROLIMUS. (Abstract #915)**
Thomas M. Fishbein, Gabriel Gondolesi, Stuart S. Kaufman, Neal S. LeLeiko, Thomas Schiano, Robert Decker, Sukru Emre, Patricia A. Sheiner, Myron E. Schwartz, Charles M. Miller. New York, NY.

2:50 PM **RAPAMYCIN IMMUNOSUPPRESSION IN PEDIATRIC LIVER AND INTESTINAL TRANSPLANT RECIPIENTS - A PRELIMINARY EXPERIENCE. (Abstract #916)**
Clarivet Torres, Simon P. Horslen, Dean Collier, Kishore Iyer, Debra L. Sudan, Alan N. Langnas, Byers W. Shaw, Jr.. Omaha, NE.

3:00 PM **THE USE OF SERUM CITRULLINE AS A MARKER OF ACUTE CELLULAR REJECTION IN ISOLATED SMALL BOWEL TRANSPLANTATION. (Abstract #917)** *Young Investigator Award*
P. A. Pappas, J. M. Saudubray, A. G. Tzakis, D. Rabier, M. R. Carreno, D. Levi, J. R. Nery, T. Kato, N. Mittal, B. Gelman, S. Nishida, J. F. Thompson, P. Ruiz. Miami, FL; Paris, France; Miami, FL; Miami, FL.

3:10 PM **DONORS PER MILLION INACCURATELY DEFINES ACTUAL OPO DONOR POTENTIAL. (Abstract #918)**
Suzanne Conrad, Lori Brigham, Ellen Sheehy, Mark Eakin, Lawrence Hunsicker, Richard Luskin. Falls Church, VA; Iowa City, IA; Falls Church, VA; Arlington, TX; Iowa City, IA; Newton, MA.

3:20 PM **MODELING ORGAN DONOR POTENTIAL FOR ORGAN PROCUREMENT ORGANIZATIONS. (Abstract #919)**
Suzanne Conrad, Lori Brigham, Ellen Sheehy, Mark Eakin, Lawrence Hunsicker. Falls Church, VA; Iowa City, IA; Falls Church, VA; Arlington, TX; Iowa City, IA.

3:20 PM **HOSPITAL DEATH RECORD REVIEW DATA ENABLE OPOs TO TARGET RESOURCES TO INCREASE DONATION. (Abstract #920)**
Suzanne Conrad, Lori Brigham, Mark Eakin, Ellen Sheehy, Lawrence Hunsicker. Falls Church, VA; Iowa City, IA; Falls Church, VA; Arlington, TX; Iowa City, IA.

Concurrent Session 48: Living Liver Donors II

2:00 PM - 3:30 PM

Grand Ballroom, Intercontinental
Chairs: Sandy Feng and Jean Emond

2:00 PM **THE INFLUENCE OF ADULT LIVING DONOR LIVER TRANSPLANTATION ON TRANSPLANT VOLUME IN UNOS REGION 9 (NEW YORK STATE). (Abstract #921)**
Thomas M. Fishbein, Gabriel Gondolesi, Amadeo Marcos, Glyn R. Morgan, Milan Kinkhabwala, Lewis Teperman, Jean Emond, Charles Miller. New York, NY; Rochester, NY; New York, NY; New York, NY.

2:10 PM **A PILOT STUDY OF ADULT LIVING DONOR LIVER TRANSPLANTATION FOR HEPATOCELLULAR CARCINOMA. (Abstract #922)**
Mikiko Ueda, Shinji Uemoto, Tetsuya Kiuchi, Hiroto Egawa, Michihiro Hayashi, Koichi Tanaka. Kyoto, Japan.

2:20 PM **IMPACT OF PORTAL HEMODYNAMICS IN LIVING-DONOR RIGHT-LOBE LIVER TRANSPLANTS IN ADULTS. (Abstract #923)**
Gabriel E. Gondolesi, Robert Shapiro, Pablo Capitanich, Luis Muñoz, Ruoqing Huang, Swan N. Thung, Semo Siljkovic, Thomas M. Fishbein, Patricia A. Sheiner, Myron E. Schwartz, Sukru Emre, Charles M. Miller. New York, NY; New York, NY; New York, NY.

2:30 PM **ASSESSMENT OF THE IMPACT OF LIVING DONOR LIVER TRANSPLANTATION ON QUALITY OF LIFE IN DONORS. (Abstract #924)**
Michael Talamantes, James Trotter, Mary McClure, Michael Wachs, Thomas Bak, Thomas Trouillot, Marcelo Kugelmas, Tracy Steinberg, Gregory Everson, Igal Kam

2:40 PM **DONOR QUALITY OF LIFE AFTER ADULT LIVER TRANSPLANT DONATION. (Abstract #925)**
Leona Kim-Schluger, Sander S. Florman, Thomas Schiano, Patricia A. Sheiner, Sukru Emre, Thomas M. Fishbein, Myron E. Schwartz, Charles M. Miller. New York, NY.

2:50 PM **GRAFT INJURY CORRELATED WITH PORTAL HEMODYNAMICS AND INTRAGRAFT ENDOTHELIN-1 EXPRESSION IN LIVING DONOR LIVER TRANSPLANTATION IN RELATION TO GRAFT SIZE. (Abstract #926)**
Kwan Man, Sheung Tat Fan, Chung Mau Lo, John Wong. Hong Kong.

3:00 PM **A CRITICAL ANALYSIS OF TACROLIMUS DOSING REQUIREMENTS AND CONCENTRATIONS IN LIVING DONOR LIVER TRANSPLANT RECIPIENTS. (Abstract #927)**
David J. Taber, Robert E. Dupuis, Amy L. Fann, Kenneth Andreoni, Roshan Shrestha, David Gerber, Jeffrey Fair, Mark W. Johnson. Chapel Hill, NC; Chapel Hill, NC; Chapel Hill, NC.

3:10 PM **TACROLIMUS DOSAGE ADJUSTMENT IN ADULT RIGHT LOBE LIVER TRANSPLANT RECIPIENTS. (Abstract #928)**
Glyn R. Morgan, Devon John, Thomas Diflo, Lewis Teperman. New York, NY.

3:20 PM **SINGLE DUCT-TO-DUCT ANASTOMOSIS IN ADULT RIGHT LOBE LIVING DONOR LIVER TRANSPLANTATION MINIMIZES BILIARY COMPLICATIONS. (Abstract #929)**
Glyn R. Morgan, Vivian Lee, Glenn Krinsky, Mike Levelle, Thomas Diflo, Devon John, Lewis Teperman. New York, NY; New York, NY.

Concurrent Session 49: Experimental Islet Transplantation

2:00 PM - 3:30 PM

King Arthur Court Ballroom, Intercontinental
Chairs: Philip O'Connell and Anthony M. Jevnikar

2:00 PM **INDUCING TOLERANCE TO MHC-MATCHED ALLOGENEIC ISLET GRAFTS IN DIABETIC NOD MICE BY SIMULTANEOUS ISLET AND BONE MARROW TRANSPLANTATION UNDER NONIRRADIATIVE AND NONMYELOABLATIVE CONDITIONING THERAPY. (Abstract #930)**
Tao Wu, Neal Heuss, Brett Levay-Young, Hakan Sozen, Nicole Kirchhof, David E.R. Sutherland, Bernhard Hering, Zhiguang Guo. Minneapolis, MN.

2:10 PM **LONG-TERM SURVIVAL OF ISLETS AGAINST AUTO- AND ALLO-IMMUNITY IN DIABETIC NOD MICE USING NON-PERMSELECTIVE BARIUM ALGINATE CAPSULES. (Abstract #931)**
Valerie F. Duvivier-Kali, Abdulkadir Omer, Richard J. Parent, John J. O'Neil, Gordon C. Weir. Boston, MA.

2:20 PM **LONG-TERM ISLET GRAFT SURVIVAL IN DIABETIC NOD MICE IS MAINTAINED BY A NOVEL IMMUNOMUDULATOR FTY720. (Abstract #932)**
Philip Lake, Fumin Fu, Jeffrey DeLeo, Christine Hopf, Victor Shi. Summit, NJ.

2:30 PM **A NON-DEPLETING COMBINATION OF ANTI-LFA-1 AND ANTI-CD154 THERAPY IS EFFECTIVE IN HIGH-RESPONDER RECIPIENTS. (Abstract #933)**
Mark R. Nicolls, Marilyne Coulombe, Joshua Beilke, Ronald G. Gill. Denver, CO.

2:50 PM **15-DEOXYSPERGUALIN (DSG) IMPROVES IMMEDIATE FUNCTIONAL ISLET MASS (FIM) AFTER PANCREATIC ISLET TRANSPLANTATION (PIT) IN STREPTOZOTOCIN (STZ)-INDUCED DIABETIC PRIMATES. (Abstract #934)**
Juan L. Contreras, Cheryl Smyth, Devin Eckhoff, Andrew Lobashevsky, Francis Thomas, Judith M. Thomas. Birmingham, AL.

2:50 PM **PROLONGATION OF ALLOGRAFT SURVIVAL IN DIABETIC NOD MICE BY CD154 BLOCKADE AND T CELL SIGNALING MODULATION WITH ANTI-CD45RB ANTIBODY. (Abstract #935)**
R.Damaris Molano, Thierry Berney, Antonello Pileggi, Camillo Ricordi, David M. Rothstein, Giacomo Basadonna, Luca Inverardi. Miami, FL; New Haven, CT.

3:00 PM **ENFORCED c-REL DEFICIENCY PROLONGS ISLET ALLOGRAFT SURVIVAL. (Abstract #936)**
Hua Yang, Dolca Thomas, Daniel Boffa, Baogui Li, Vijay K. Sharma, Milagros Lagman, Hsiou-chi Liou, Manikkam Suthanthiran. New York, NY; New York, NY.

3:10 PM **TRAIL (TUMOR NECROSIS RECEPTOR-APOPTOSIS-INDUCING-LIGAND) INDUCED APOPTOSIS (TIA) IS A MAJOR MECHANISM OF EARLY DONOR ISOLATED ISLET (II) DEATH AND DYSFUNCTION. (Abstract #937)**
Francis T. Thomas, Jin He, Jianguo Wu, Peter Ray, Cheryl Smyth, Judith Thomas.

3:20 PM **THE ROLE OF ISLET SPECIFIC AUTOIMMUNITY IN ISLET ALLOGRAFT DESTRUCTION. (Abstract #938)**
Leila Makhlouf, Koji Kishimoto, Reza Abdi, Neal Rex Smith, Maria Koulmanda, Hugh Auchincloss, Jr, Mohamed H. Sayegh. Boston, MA; Boston, MA; Boston, MA.

Concurrent Session 50: Liver Transplantation: Hepatitis B

2:00 PM - 3:30 PM

Renaissance Ballroom, Intercontinental
Chairs: James Eason and David Grant

2:00 PM **LONG-TERM OUTCOMES IN RECIPIENTS OF HEPATITIS B CORE ANTIBODY POSITIVE LIVER DONORS. (Abstract #939)**
Cosme Manzarbeitia, Vivek Kaul, Thirumalesh Kanchana, Kenneth D. Rothstein, Santiago J. Munoz, Shivakumar Vignesh, Victor Araya, Jorge Ortiz, Rafael Pena, David J. Reich. Philadelphia, PA; Philadelphia, PA.

2:10 PM **OUTCOME OF LIVER TRANSPLANTATION FOR HEPATITIS B VIRUS (HBV) VARIES BY HBV GENOTYPE. (Abstract #940)**
H. Devarbhavi, A. Cohen, M. Charlton, R. Wiesner, R. Dickson, C. Rosen, M. Ishitani. Rochester.

2:20 PM **IS HEPATIC HBVDNA STATUS A PARAMETER TO GUIDE MAINTENANCE/INTERRUPTION OF ANTI-HBV REGIMENS? (Abstract #941)**
Jose Nery, Robert Cirocco, Rajender Reddy, Caio Nery, Deborah Weppler, Phillip Ruiz, Eugene Schiff, Andreas Tzakis. Miami, FL; Miami, FL; Miami, FL.

2:30 PM **ELIMINATION OF DE NOVO HEPATITIS B VIRUS INFECTION IN RECIPIENTS OF HEPATIC ALLOGRAFTS FROM DONORS POSITIVE FOR HEPATITIS B CORE ANTIBODY. (Abstract #942)**
S. Forrest Dodson, Clark Bonham, Thomas Cacciarelli, Dave Geller, Paulo Fontes, John Fung. Pittsburgh, PA.

2:40 PM **FAILURE OF A DOUBLE-REINFORCED COURSE OF HB VACCINATION IN PTS TRANSPLANTED FOR HBV-RELATED CIRRHOSIS. (Abstract #943)**
Giuseppe Tisone, Elena Torri, Antonino Araco, Alessandro Anselmo, Carlo Camplone, Settimio Zazza, Daniele Di Paolo, Mario Angelico, Carlo Umberto Casciani. Rome, Italy.

2:50 PM **SPONTANEOUS DEVELOPMENT OF ANTIBODY TO HEPATITIS B SURFACE ANTIGEN AFTER LIVER TRANSPLANTATION FOR CHRONIC HEPATITIS B USING LAMIVUDINE PROPHYLAXIS. (Abstract #944)**
Chung-Mau Lo, James T.K. Fung, George K.K. Lau, Chi-Leung Liu, Siu-Tim Cheung, Ching-Lung Lai, Sheung-Tat Fan, John Wong. Hong Kong, China.

3:00 PM **PATIENTS TRANSPLANTED FOR CHRONIC HBV USING ANTI-HBcAB POSITIVE DONOR LIVERS WHO ARE TREATED WITH HBIG AND/OR LAMIVUDINE HAVE NO DETECTABLE HBV-DNA IN LIVER BIOPSY TISSUE OR SERA DURING LONG-TERM FOLLOW-UP. (Abstract #945)**
M. Ishitani, H. Devarbhavi, J. Wilkinson, T. Laskus, R. Dickson, J. Rakela. Rochester, MN; Scottsdale, AZ; Jacksonville, FL.

3:10 PM **COMBINATION OF LAMIVUDINE AND HIGH DOSE IV HEPATITIS B IMMUNE GLOBULIN PROVIDES EFFECTIVE PROPHYLAXIS FOR HEPATITIS B PATIENTS UNDERGOING LIVER TRANSPLANTATION. (Abstract #946)**
Victor I. Machicao, Harshad C. Devarbhavi, Consuelo Soldevilla-Pico, Michael B. Ishitani, Rolland C. Dickson. Jacksonville, FL; Rochester, MN; Gainesville, FL.

3:20 PM **EARLY PHARMACOKINETICS OF IV 5% NABI HEPATITIS B IMMUNE GLOBULIN COMBINED WITH LAMIVUDINE IN LIVER TRANSPLANTATION FOR HEPATITIS B CHRONIC LIVER DISEASE. (Abstract #947)**
R. Dickson, R. Reddy, M. Ishtani, N. Terrault, V. Luketic, P. Sheiner, T. Angtuaco, M. Fried, C. Soldevila-Pico, J. Johnson. Jacksonville, FL; Miami, FL; Rochester, MN; San Francisco, CA; Richmond, VA; New York, NY; Chicago, IL; Chapel Hill, NC; Gainesville, FL.

3:30 **Break**

Concurrent Session 51: Basic Science: Allorecognition

4:00 PM - 5:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Elizabeth Field and Sheri Krams

4:00 PM **CROSSREACTIVE TH1 RESPONSES TO DONOR MHC PEPTIDE AND CARDIAC MYOSIN ARE PRESENT DURING CHRONIC REJECTION OF HEART ALLOGRAFTS. (Abstract #948)** *Young Investigator Award*
Gilles Benichou, Koji Kishimoto, Hillary Rolls, Victor Dong, Anna Valujskikh, Peter S. Heeger, Mohamed H. Sayegh, Eugenia V. Fedoseyeva. Boston, MA; Boston, MA; Cleveland, OH.

4:10 PM **THE ROLE OF MHC CLASS II DISPARITIES IN CARDIAC ALLOGRAFT REJECTION: DISSECTING PRIMING VS. EFFECTOR MECHANISMS USING A TCR TRANSGENIC SYSTEM. (Abstract #949)**
Laurence A. Turka, Zihao Wu, Andrew D. Wells, Sigrid Sandner, Peter Langmuir, Mohamed H. Sayegh. Philadelphia, PA; Boston, MA.

4:20 PM **INDIRECT PRESENTATION OF DONOR MHC CLASS II ANTIGEN BY DNA VACCINATION PROLONGS HEART ALLOGRAFT SURVIVAL. (Abstract #950)**
Helene Pêche, Bryce Van Denderen, Jean Christian Roussel, Valérie Guilloteau, Benjamin Trinité, Michèle Heslan, Jean Paul Soulillou, Maria Cristina Cuturi. Nantes, France; Melbourne, Australia.

4:30 PM **PROLONGATION OF GRAFT SURVIVAL BY EXPOSURE TO NON-INHERITED MATERNAL MHC ANTIGENS (NIMA) IN A VASCULARIZED MURINE CARDIAC TRANSPLANTATION MODEL. (Abstract #951)**
Satoshi Kusaka, Ewa Jankowska-Gan, Gilles Benichou, William J. Burlingham. Madison, WI; Boston, MA.

4:40 PM **ARE SECONDARY LYMPHOID ORGANS ESSENTIAL FOR SKIN ALLOGRAFT REJECTION? (Abstract #952)**
Karina Bonin, Claude Daniel. Laval, QC, Canada.

4:50 PM **IN VITRO 'ACCOMMODATION' OF ENDOTHELIAL CELLS BY ALLOSPECIFIC ANTIBODIES IS ASSOCIATED WITH EARLY RELEASE OF LOW CONCENTRATIONS OF ADENOSINE. (Abstract #953)**
Alexandros Delikouras, Lynette D. Fairbanks, Anne Simmonds, Robert I. Lechler, Anthony Dorling. London, United Kingdom; London, United Kingdom.

5:00 PM **THE POLYMORPHISM AT -330 IN THE INTERLEUKIN-2 GENE RESULTS IN A SIGNIFICANT INCREASE IN IL-2 PROTEIN FOLLOWING CO-STIMULATION OF PERIPHERAL BLOOD LYMPHOCYTES WITH ANTI-CD3/CD28. (Abstract #954)**
Steven C. Hoffmann, Eran M. Stanley, E. Darrin Cox, Nancy Craighead, Stephen J. Perfetto, Barbara S. DiMercurio, Dee E. Koziol, David M. Harlan, Allan D. Kirk, Patrick J. Blair. Bethesda, MD; Washington, DC; Bethesda, MD; Bethesda, MD.

5:10 PM **STRAIN DIFFERENCES IN THE ABILITY TO MEDIATE CD28/CD40 INDEPENDENT REJECTION OF SKIN ALLOGRAFTS ARE EXPRESSED IN THE CD8+ T CELL COMPARTMENT. (Abstract #955)**
Matthew A. Williams, Joel Trambley, Andrew B. Adams, Megan M. Durham, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

5:20 PM **LF15-0195 TREATMENT INDUCES ALLOGRAFT TOLERANCE. EVIDENCE FOR REGULATORY CD4+CD25+CD45RC- T CELLS. (Abstract #956)**
Elise Chiffoleau, Patrick Dutartre, Claire Usual, Jean-Paul Soulilllou, Maria-Cristina Cuturi. Nantes, France; Daix, France.

Concurrent Session 52: Outcome Assessment in Renal Transplantation

4:00 PM - 5:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: Philip Halloran and David Rush

4:00 PM **PTLD IN KIDNEY TRANSPLANTATION: A 32 YEAR EXPERIENCE IN 405 PATIENTS FROM THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY. (Abstract #957)** *Young Investigators Award*
Jennifer Trofe, Joseph F. Buell, Michael J. Hanaway, Tom Beebe, M. Roy First, Rita R. Alloway, Agnes Lo, Horacio L. Rilo, E. Steve Woodle. Cincinnati, OH.

4:10 PM **NATIONAL TRANSPLANTATION PREGNANCY REGISTRY: RELATIONSHIP OF TRANSPLANT TO CONCEPTION INTERVAL TO PREGNANCY OUTCOME IN CYCLOSPORINE-TREATED FEMALE KIDNEY RECIPIENTS. (Abstract #958)**
William J. Gaughan, Lisa A. Coscia, Stephen R. Dunn, Carolyn H. McGrory, Michael J. Moritz, Vincent T. Armenti. Philadelphia, PA; Philadelphia, PA.

4:20 PM **DEATH AFTER GRAFT LOSS: A NOVEL ENDPOINT FOR RENAL TRANPLANTATION. (Abstract #959)**
Herwig-Ulf Meier-Kriesche, Bruce Kaplan. Ann Arbor, MI.

4:30 PM **ASSESSING COMPETING RISKS OF FOUR CAUSES OF KIDNEY TRANSPLANT FAILURE. (Abstract #960)**
David W. Gjertson, Dorota M. Dabrowska, Xingping Cui, J. Michael Cecka.

4:40 PM **FAILURE OF HLA MATCHING TO IMPROVE LONG TERM PATIENT SURVIVAL IN US RENAL TRANSPLANT RECIPIENTS. (Abstract #961)**
Ross B. Isaacs, Peter I. Lobo, Clint E. Spencer, Steven L. Nock. Charlottesville, VA; Charlottesville, VA.

4:50 PM **ROUTINE CANCER SCREENING IN THE POST RENAL TRANSPLANT PERIOD. (Abstract #962)**
Bryce Kiberd, Tammy Keough-Ryan, Joseph Lawen. Halifax, NS, Canada; Halifax, NS, Canada.

5:00 PM **GEOGRAPHICAL VARIATION IN SKIN CANCER RISK POST-TRANSPLANTATION: A MATCHED COHORT COMPARISON BETWEEN ENGLAND AND AUSTRALIA. (Abstract #963)**
Helen M. Ramsay, Anthony A. Fryer, Carmel Hawley, Andrew G. Smith, David Nicol, Paul N. Harden. Stoke-on-Trent, United Kingdom; Stoke-on-Trent, United Kingdom; Brisbane, Queensland, Australia.

5:10 PM **LONG TERM PROGNOSTIC SIGNIFICANCE OF CLINICAL RISK STRATIFICATION AND MYOCARDIAL PERFUSION STUDIES (MPS) IN PATIENTS ASSESSED FOR RENAL TRANPLANTATION. (Abstract #964)**
Christian G. Rabbat, Kim Lambert, Eric B. Stanton, David N. Churchill, J. David Russell. Hamilton, ON, Canada.

5:20 PM **DIFFERENCES IN B-LINEAGE POST-TRANSPLANT LYMPHOMAS ACCORDING TO TYPE OF ABDOMINAL ORGAN TRANSPLANT. (Abstract #965)**
Steven Paraskevas, Matthew J. Graczyk, James E. Coad, Rainer W.G. Gruessner. Minneapolis, MN; Minneapolis, MN.

Concurrent Session 53: Cadaver Donation: Factors and Outcomes

4:00 PM - 5:30 PM

Chicago Ballroom 8/9, Sheraton
Chairs: David Hull and Richard Perez

4:00 PM **LONG-TERM GRAFT FUNCTION IN RENAL TRANSPLANTATION USING NON-HEART-BEATING DONORS. (Abstract #966)** *Young Investigator Award*
M. J. Aull, M. R. First, M. Cardi, V. R. Peddi, P. Weiskittel, E. Berilla, L. Meredith, J. Trofe, E. Zavala, R. R. Alloway, E. S. Woodle, J. F. Buell. Cincinnati, OH; Cincinnati, OH.

4:10 PM **LONG-TERM (OVER 15 YEARS) OUTCOME OF NON-HEART-BEATING DONOR KIDNEY TRANSPLANTATION. A SINGLE-CENTER EXPERIENCE. (Abstract #967)**
Fumio Tsukuda, Kazunari Tanabe, Tadahiko Tokumoto, Hiroaki Shimmura, Shohei Fuchinoue, Satoshi Teraoka, Hiroshi Toma. Tokyo; Tokyo, Japan.

4:20 PM **THE EFFECTIVENESS OF IN SITU REGIONAL COOLING IN THE RENAL PROCUREMENTS FROM NON-HEART BEATING DONORS. (Abstract #968)**
Kiyotaka Hoshinaga, Ryoichi Shiroki, Tamio Fujita, Yusuke Kubota, Hitomi Sasaki, Toru Itoh, Kiyohito Ishikawa, Masanobu Izumitani, Tetsuo Kanno. Toyoake, Aichi, Japan; Toyoake, Aichi, Japan; Nagoya, Aichi, Japan.

4:30 PM **DONOR CORONARY ANGIOGRAPHY LEADS TO INCREASED KIDNEY DELAYED GRAFT FUNCTION. (Abstract #969)**
Prabhakar K. Baliga, G. Mark Baillie, Tamala Sill, Jeremy Kirtz, Stephen F. Shafizadeh, Jeffrey Rogers, Angello Lin, P.R. Rajagopalan, Kenneth D. Chavin. Charleston, SC; Charleston, SC.

4:40 PM **DONOR SCORING SYSTEM FOR CADAVERIC RENAL TRANSPLANTION: UNOS DATABASE ANALYSIS. (Abstract #970)**
Scott Nyberg, Walter Kremers, Jeffrey Thostenson, Mikel Prieto, Michael Ishitani, Sylvester Sterioff, Mark Stegall. Rochester, MN.

4:50 PM **INFLUENCE OF DONOR AGE, COLD ISCHEMIA TIME (CIT), AND GENDER ON CADAVERIC KIDNEY GRAFT OUTCOME. (Abstract #971)**
Mark I. Aeder, Charles F. Shield, III, Bradley A. Warady, Daniel Murillo, Nicolas A. Muruve, Paul W. Nelson, Christopher F. Bryan. Westwood, KS.

5:00 PM **USE OF DIABETIC KIDNEYS CAN SAFELY EXPAND THE DONOR POOL. (Abstract #972)**
Yolanda T. Becker, Glen E. Leverson, Anthony M. D'Alessandro, Hans W. Sollinger, Bryan N. Becker. Madison, WI; Madison, WI.

5:10 PM **EXPANDING THE ORGAN DONOR POOL- THE LIMITING FACTOR. (Abstract #973)**
Lauren Brasile, Bart Stubenitsky, Booster Maurits, Dorian Arenada, Carl Haisch, Gauke Kootstra. Maastricht, The Netherlands; Greenville, NC; Schenectady, NY.

5:20 PM **THE IMPACT OF INCREASING DONOR AGE ON LONG-TERM KIDNEY GRAFT SURVIVAL - EACH YEAR COUNTS. (Abstract #974)**
Abhi Humar, William Payne, Thiagarjan Ramcharan, Roger Denis, Kristen Gillingham, Arthur Matas. Minneapolis, MN.

Concurrent Session 54: Basic Science: Tolerance II

4:00 PM - 5:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: Suzanne T. Ildstad and M. Suthanthiram

4:00 PM **NON-MYELOSUPPRESSIVE BONE MARROW TRANSPLANTATION INDUCES TOLERANCE AND CORRECTS HEMOGLOBINOPATHIES. (Abstract #975)**
Andrew B. Adams, Megan M. Durham, Leslie Kean, Jonwong Ha, Matthew A. Williams, Phyllis Rees, Stephen Mittelstaedt, Adam W. Bingaman, David Archer, Thomas C. Pearson, Edmund Waller, Christian P. Larsen. Atlanta, GA; Atlanta, GA; Atlanta, GA.

4:10 PM **IMMUNOLOGIC MECHANISMS IN TOLERANCE INDUCED BY NON-RADIATION BASED IMMUNOSUPPRESSION AND DONOR BONE MARROW. (Abstract #976)**
Douglas A. Hale, Rita Gottschalk, Akihisi Umemura, Takashi Maki, Anthony P. Monaco. Boston, MA.

4:20 PM **LACK OF ALLOGRAFT TOLERANCE DESPITE STABLE CHIMERISM AFTER ALLOGENEIC BONE MARROW TRANSPLANTATION. (Abstract #977)**
Takashi Maki, Akihisa Umemura, Hirofumi Morita, Xian C. Li, Anthony P. Monaco. Boston, MA; Boston, MA.

4:30 PM **Vα14 NKT CELLS ARE ESSENTIAL FOR ACCEPTANCE OF MURINE ISLET ALLOGRAFTS BY DONOR SPECIFIC TRANSFUSION. (Abstract #978)**
Yohichi Yasunami, Yoshiichiroh Nakamura, Megumi Takehara, Toshimitsu Uede, Satoru Todo, Toshinori Nakayama, Masaru Taniguchi, Seiyo Ikeda. Fukuoka, Fukuoka, Japan; Sapporo, Hokkaido, Japan; Sapporo, Hokkaido, Japan; Chiba, Chiba, Japan.

4:40 PM **THYMUS REQUIREMENT FOR TOLERANCE INDUCTION BY ADOPTIVE TRANSFER OF ALLOMHC PEPTIDE-PULSED DENDRITIC CELLS. (Abstract #979)**
Olakunle O. Oluwole, Hector A. DePaz, Ayoola O. Ali, Roshni Gopinathan, Mark A. Hardy, Soji F. Oluwole. New York, NY.

4:50 PM **SYNGENIC BONE MARROW DERIVED IMMATURE DENDRITIC CELLS INDUCED PROLONGATION OF ALLOGRAFT SURVIVAL. (Abstract #980)**
Helene Pêche, Michele Heslan, Claire Usal, Jean-Paul Soulillou, Maria Cristina Cuturi. Nantes, France.

5:00 PM **PEPTIDE LOADED ALLOGENEIC DIVALENT MHC CLASS I MOLECULES VISUALIZE ALLOREACTIVE T-CELLS AND DISCRIMINATING TOLERANT FROM REJECTING GRAFT RECIPIENTS. (Abstract #981)**
Bhavna Sharma, Dirk Behrens, Dieter Kabelitz, Nicholas Zavazava. Kiel, Germany.

5:10 PM **INTRAVASCULAR IN UTERO INJECTION OF ADULT BONE MARROW LEADS TO ACCEPTANCE OF FULL MHC MISMATCHED KIDNEY ALLOGRAFTS. (Abstract #982)**
David W. Mathes, Kazuhiko Yamada, Mark A. Randolph, Mario G. Solari, Anette Wu, David H. Sachs, W.P. Andrew Lee. Boston, MA; Boston, MA.

5:20 PM **INFLUENCE OF FETAL EXPOSURE TO NON-INHERITED MATERNAL MHC ANTIGENS (NIMA) ON ALLORESPONSES IN ADULT OFFSPRINGS. (Abstract #983)**
William Burlingham, Satoshi Kusaka, Ewa Jankowska-Gan, Eugenia Fedoseyeva, Robert Tam, Ben Illigens, Natalie Anosova, Gilles Benichou. Madison, WI; Boston, MA; Costa Mesa, CA.

Concurrent Session 55: Pharmacokinetics of Immunosuppressants

4:00 PM - 5:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Ronald Shapiro and Todd Pesavento

4:00 PM **A COMPARISON OF THE ABILITY OF DIFFERENT MARKERS OF CYCLOSPORINE EXPOSURE TO PREDICT ACUTE REJECTION IN RENAL TRANSPLANT RECIPIENTS. (Abstract #984)**
Catherine M. Clase, Kamran Mahalati, Bryce A. Kiberd, Joseph G. Lawen, Kenneth A. West, Philip Belitsky. Halifax, NS, Canada; Halifax, NS, Canada.

4:10 PM **CYCLOSPORINE IS ASSOCIATED WITH DECREASED ABSOLUTE BIOAVAILABILITY OF MYCOPHENOLIC ACID. (Abstract #985)**
Wei Zhu, Wolfgang Arns, Polly Carpenter, Les Choi, Pratapa Prasad, Somesh Choudhury, Peter Graf, Robert Schmouder. East Hanover, NJ; Koln, Germany; Horsham, United Kingdom; Basel, Switzerland.

4:20 PM **POTENTIAL PHARAMCOKINETIC INTERACTIONS BETWEEN SIROLIMUS AND TACROLIMUS. (Abstract #986)**
James J. Zimmerman, Alain Patat, Marie-Laure Souan, Isabelle Paty, Guy Cadieu. Radnor, PA; Paris, France; Rouffach, France.

4:30 PM **SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY: THERAPEUTIC DRUG MONITORING AND DOSING CONSIDERATIONS. (Abstract #987)**
Josep M. Campistol, Joseph Lawen, Christina Brattström, Paolo Altieri, David Oliveira, Rowan Walker, David Holt, James T. Burke, James Zimmerman, the Sirolimus Tri-continental Renal Transplant Study Group. Barcelona, Spain.

4:40 PM **IMPACT OF ETHNICITY ON THE PHARMACOKINETICS AND EFFICACY OF AN EVEROLIMUS-CYCLOSPORINE-PREDNISONE REGIMEN IN RENAL TRANSPLANTATION. (Abstract #988)**
P. R. Rajagopalan, J. M. Kovarik, B. D. Kahan, D. VanBuren, L. McMahon, R. Boger, C. Rordorf. Charleston, SC; East Hanover, NJ; Houston, TX; Nashville, TN.

4:50 PM **INFLUENCE OF RENAL AND HEPATIC IMPAIRMENT ON EVEROLIMUS PHARMACOKINETICS: ARE DOSE ADJUSTMENTS NECESSARY? (Abstract #989)**
J. M. Kovarik, H. Sabia, M. Rouilly, J. Figueiredo, D. W. Tudor, K. Lasseter, C. Rordorf. Basel, Switzerland; Miami, FL.

5:00 PM **NEORAL®C2 ABSORPTION PROFILING: A SIMPLE, ACCURATE, AND PRECISE PREDICTOR OF REJECTION RISK IN RENAL TRANSPLANTATION. (Abstract #990)**
Paul A. Keown, The International Neoral Renal Transplant Study Group. Vancouver, BC, Canada.

5:10 PM **PHARMACOKINETIC AND PHARMACODYNAMIC STUDIES OF ONE OR TWO DOSES OF DACLIZUMAB IN RENAL TRANSPLANT PATIENTS. (Abstract #991)**
F. Vincenti, M. V. Lantz, J. L. Birnbaum, S. J. Tomlanovich, W. J.C. Amend, D. B. Adey, P. G. Stock, R. Hirose, C. E. Freise, J. P. Roberts, D. Pace. San Francisco, CA; Nutley, NJ.

5:20 PM **CONVERSION OF STABLE RENAL TRANSPLANT RECIPIENTS FROM CYCLOSPORIN MICROEMULSION TROUGH (C-0) TO C-2 MONITORING: IMPLICATIONS FOR EXPOSURE. (Abstract #992)**
Edward Cole, Carl Cardella, Daniel Cattran, Stanley Fenton, Catherine O'Grady, Robert Smith. Toronto, ON, Canada.

Concurrent Session 56: Xenotransplantation: Mechanisms, Infectious Issues

4:00 PM - 5:30 PM

Empire Room, Intercontinental
Chairs: Joseph Leventhal and Jeffrey Platt

4:00 PM **XENOGRAFT ACCOMMODATION: EXPRESSION OF HEME OXYGENASE-1 PROTECTS ENDOTHELIAL CELLS FROM XENOSERUM-MEDIATED APOPTOSIS. (Abstract #993)**
Ning Wang, Jangming Lee, Edda Tobiasch, Simon C. Robson, Fritz H. Bach, Yuan Lin.

4:10 PM **USE OF MOLECULAR MODELING AND SITE-DIRECTED MUTAGENESIS TO ADDRESS THE ROLE OF XENOANTIBODY STRUCTURE IN THE SPECIFICITY OF scFv XENOANTIBODIES FOR THE GAL a1,3GAL CARBOHYDRATE. (Abstract #994)**
Mary K. Keams-Jonker, Michael B. Bolger, Robert Mencel, Namath Hussain, Alice Kearney, Vaughn A. Starnes, Donald V. Cramer. CA; Los Angeles, CA.

4:20 PM **THE IL-2 RECEPTOR ALPHA CHAIN (CD25) PLAYS AN IMPORTANT ROLE IN REGULATING CD40 EXPRESSION DURING HUMAN ANTI-PORCINE CELLULAR RESPONSES. (Abstract #995)**
He Xu, Francis Cruzata, Douglas K. Tadaki, Patrick J. Blair, David M. Harlan, Allan D. Kirk.

4:30 PM **PORCINE BONE MARROW-DERIVED DENDRITIC CELLS STIMULATE XENOGENEIC B CELL PROLIFERATION BY A NON CONTACT-DEPENDENT MECHANISM. (Abstract #996)**
Nada Kanaan, Lori Bachman, David McKean, Christopher McGregor, Matthew Griffin. Rochester, MN; Rochester, MN; Rochester, MN.

4:40 PM **INTER-SPECIFIC TRANSMISSION OF PORCINE ENDOGENOUS RETROVIRUS *IN VIVO*. (Abstract #997)**
Ruhul H. Kuddus, Mary E. Tarara, Abdul S. Rao. Pittsburgh, PA.

4:50 PM **ABSENCE OF ANTI-PERV ANTIBODIES AFTER EXPOSURE TO PIG GRAFTS. (Abstract #998)**
Hui Xu, Ajay Sharma, Yuanyuan Wei, Liping Huang, Jeannine Okabe, Cunqi Cui, Libing Chen, Christopher G.A. McGregor, Marlon Levy, John S. Logan, Guerard W. Byrne. Princeton, NJ; Rochester, MN; Dallas, TX.

5:00 PM **MOLECULAR AND INFECTIVITY ANALYSIS OF PORCINE ENDOGENOUS RETROVIRUS IN A TRANSGENIC PIG LINE. (Abstract #999)**
Cunqi Cui, Jianying Su, Colby Enck, Colleen M. Quinn, John S. Logan, Lisa E. Diamond.

5:10 PM **TOLERIZING ANTI-GAL IgG PRODUCTION WITH TRANSIENT ANTI-CD40L MAB TREATMENT AND INTACT ACTIVE BONE (IAB) TRANSPLANTATION. (Abstract #1000)**
Dengping Yin, Lianli Ma, Lei Zhang, Anita S. Chong. Chicago, IL.

5:20 PM **ASIALO GM1+ CD8+ CELLS MEDIATE CD40/CD28 INDEPENDENT XENOGRAFT REJECTION. (Abstract #1001)**
Joel Trambley, Eric Elwood, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

Concurrent Session 57: Infection and Neoplasms After Organ Transplantation

4:00 PM - 5:30 PM

Exchange Room, Intercontinental
Chairs: Susan Orloff and Donald Dafoe

4:00 PM **A PILOT STUDY USING ALLOGENEIC CYTOTOXIC T CELLS FOR POST-TRANSPLANT LYMPHOPROLIFERATIVE DISEASE. (Abstract #1002)**
Tanzina Haque, Clare Taylor, Gwen M. Wilkie, Angela Iley, Peter L. Amlot, Dorothy H. Crawford. Edinburgh, United Kingdom; London, United Kingdom.

4:10 PM **THE OCCURRENCE OF EPSTEIN-BARR VIRUS NEGATIVE POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDER IN SOLID ORGAN TRANSPLANTATION. (Abstract #1003)**
Thomas V. Cacciarelli, Igor Dvorcik, John J. Fung, Michael A. Nalesnik. Pittsburgh, PA.

4:20 PM **TRANSPLANT TUMOR REGISTRY: DONORS WITH CNS TUMORS. (Abstract #1004)**
H. M. Kauffman, M. A. McBride, W. S. Cherikh, P. C. Spain, F. L. Delmonico. Richmond, VA; Boston, MA.

4:30 PM **MELANOMA IN SOLID ORGAN TRANSPLANTATION: A 25 YEAR EXPERIENCE IN 143 PATIENTS FROM THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY. (Abstract #1005)**
J. Trofe, J. Buell, M. Hanaway, T. Beebe, E. S. Woodle.

4:40 PM **ANALYSIS OF CD8 AND CD4 T-CELL REACTIVITY TO CYTOMEGALOVIRUS - MEASURING REACTIVITY AT THE TOTAL PROTEIN AND EPITOPE LEVEL. (Abstract #1006)** *International Young Investigator Award*
Florian Kern, Nicole Faulhaber, Torsten Bunde, Rudolf Volkmer-Engert, Ines Kretzschmar, Susanna Prösch, Ralph Ewert, Hans-Dieter Volk, Petra Reinke. Berlin, Germany; Berlin, Germany; Berlin, Germany; Berlin, Germany.

4:50 PM **MULTICENTERED CYTOMEGALOVIRUS (CMV) TREATMENT IN TRANSPLANTATION (TX) REGISTRY: FIRST YEAR RESULTS. (Abstract #1007)**
Marian G. Michaels, Christina A. Dingivan, Rachael Henderson, Michael D. Green, CMV Transplant Registry. Pittsburgh, PA; Gaithersburg, MD.

5:00 PM **LINEZOLID IS AN EFFECTIVE TREATMENT FOR VANCOMYCIN-RESISTANT ENTEROCOCCUS *FAECIUM* IN SOLID ORGAN TRANSPLANT RECIPIENTS-REPORT OF A MULTICENTER COMPASSIONATE USE TRIAL. (Abstract #1008)**
Joseph El Khoury, Mary C. Birmingham, Jay A. Fishman. Boston, MA; Buffalo, NY.

5:10 PM *CRYPTOCOCCUS NEOFORMANS* INFECTION IN ORGAN TRANSPLANT RECIPIENTS: IMPACT AND CORRELATES OF OUTCOME. (Abstract #1009)
Shahid Husain, Marilyn M. Wagener, Timothy Gayowski, Nina Singh. Pittsburgh, PA.

5:20 PM EARLY INFECTIOUS COMPLICATIONS IN NON-MYELOABLATIVE ALLOGENEIC BONE MARROW TRANSPLANT RECIPIENTS (NMA-BMT). (Abstract #1010)
Robin K. Avery, David L. Longworth, Sherif B. Mossad, Matt Kalaycio, Brad Pohlman, Ronald Sobecks, Elizabeth Kuczkowski, Laura Bernhard, Jenni Koennecke, Holly Sommer, Melissa McBee, Julie Curtis, Brian Bolwell. Cleveland, OH; Cleveland, OH.

Concurrent Session 58: Fulminant Hepatic Failure

4:00 PM - 5:30 PM

Grand Ballroom, Intercontinental
Chairs: Charles Rosen and Gary Levy

4:00 PM ORTHOTOPIC LIVER TRANSPLANTATION FOR FULMINANT HEPATIC FAILURE. (Abstract #1011)
Shigeru Marubashi, Edmund Q. Sanchez, Brian M. Gogel, Linda W. Jennings, Ernesto P. Molmenti, Carlos G. Fasola, Robert M. Goldstein, Marlon F. Levy, Barbara K. Brooks, Laura L. Christensen, Thomas A. Gonwa, Goran B. Klintmalm. Dallas, TX.

4:10 PM RESULTS OF PHASE I TRIAL OF THE EXTRACORPOREAL LIVER ASSIST DEVICE FOR PATIENTS WITH FULMINANT HEPATIC FAILURE. (Abstract #1012)
J. M. Millis, D. J. Kramer, J. O'Grady, T. G. Heffron, S. Caldwell, M. Hart, P. MaGuire. Chicago, IL; Pittsburgh, PA; London, England, United Kingdom; Atlanta, GA; Charlottesville, VA; San Diego, CA; LaJolla, CA.

4:20 PM A NOVEL STRATEGY TO IMMORTALIZE HUMAN HEPATOCYTES FOR THE DEVELOPMENT OF HEPATOCYTE TRANSPLANTATION. (Abstract #1013)
Toshinori Totsugawa, Naoya Kobayashi, Hirofumi Noguchi, Toshihisa Matsumura, Takamasa Watanabe, Toshiyoshi Fujiwara, Masakiyo Sakaguchi, Masaki Hikida, Hitoshi Ohmori, Karen A. Westerman, Philippe Leboulch, Noriaki Tanaka. Okayama, Japan; Okayama, Japan; Okayama, Japan; Cambridge, MA.

4:30 PM URGENT LIVING DONOR LIVER TRANSPLANTATION FOR THE TREATMENT OF FULMINANT HEPATIC FAILURE. (Abstract #1014)
Tsuyoshi Shimamura, Maeng B. Jin, Toshiya Kamiyama, Michiaki Matsushita, Hiroyuki Furukawa, Satoru Todo. Sapporo, Hokkaido, Japan.

4:40 PM LIVERS FROM MORBIDLY OBESE DONORS; RESULTS OF 97 ATTEMPTED LIVER PROCUREMENTS - HALF PROVIDED TRANSPLANTS! (Abstract #1015)
David J. Reich, Vivek Kaul, Leonard Braitman, Howard M. Nathan, Richard D. Hasz, John D. Abrams, Jorge A. Ortiz, Victor R. Araya, Kenneth D. Rothstein, Santiago J. Munoz, Cosme Manzarbeitia. Philadelphia; Philadelphia.

4:50 PM MARGINAL FATTY LIVERS MAY BE AN UNTAPPED RESOURCE TO EXPAND THE DONOR POOL. (Abstract #1016)
Kenneth D. Chavin, Stephen F. Shafizadeh, Ryan Fiorini, Jeremy Kirtz, Satish Nadig, Julia K. Haines, Lydia Nichols, Nicolas P. Mora, G. Mark Baillie, Jeffrey Rogers, Angello Lin, Prabhakar K. Baliga. Charleston, SC; Charleston, SC.

5:00 PM OLDER RECIPIENTS OF ORGANS FROM OLDER DONORS HAVE LESS REJECTION AFTER LIVER TRANSPLANTATION. (Abstract #1017)
Nicholas Onaca, Ari J. Cohen, Humberto E. Bohorquez, Scott L. Nyberg, Michael B. Ishitani, J. E. Hay, David J. Brandhagen, Michael R. Charlton, Russell H. Wiesner, Charles B. Rosen. Rochester, MN.

5:10 PM RECONSIDERING THE IMPACT OF DONOR AGE ON GRAFT AND PATIENT SURVIVAL AFTER LIVER TRANSPLANTATION. (Abstract #1018)
David M. Levi, Seigo Nishida, Tomoaki Kato, Jose Nery, Guy Neff, Les Olson, Andreas G. Tzakis. Miami, FL.

5:20 PM UNCONTROLLED NON-HEART BEATING DONOR LIVER TRANSPLANTATION. (Abstract #1019)
Alejandra Otero, Francisco Suarez, Manuel Gomez-Gutierrez, Anton Fernandez-Garcia, Francisco Arnal, Carlos Fernandez-Selles, Jose Garcia-Buitron, Joaquin Alvarez, Rafael Manez. La Coruna, Spain; Madrid, Spain.

Concurrent Session 59: Genetic Modulation

4:00 PM - 5:30 PM

King Arthur Court Ballroom, Intercontinental
Chairs: David Geller and Xin Xiao Zheng

4:00 PM TREATMENT WITH ANTISENSE OLIGODEOXYRIBONUCLEOTIDES TARGETING FAS mRNA ATTENUATES RENAL ISCHEMIA-REPERFUSION INJURY. (Abstract #1020) *Young Investigator Award*
Linlin Ma, Hong Zhang, Lianfu Wang, Nicholas M. Dean, John J. Fung, Shiguang Qian. Pittsburgh, PA; San Digo, CA.

4:10 PM GENE TRANSFER OF THE ANTI-APOPTOTIC BCL-2 GENE CONFERS CYTOPROTECTION TO ISOLATED PORCINE PANCREATIC ISLETS (PPI) EXPOSED TO XENOREACTIVE ANTIBODIES (XA) AND COMPLEMENT. (Abstract #1021)
Juan L. Contreras, Guadalupe Bilbao, Cheryl Smyth, Devin E. Eckhoff, Xiaoling Jiang, Francis T. Thomas, David Curiel, Judith M. Thomas. Birmingham, AL.

4:20 PM ADENOVIRUS-MEDIATED GENE TRANSFER OF IMMUNOREGULATORY MOLECULES IN A RAT MODEL OF CHRONIC ALLOGRAFT REJECTION IMPROVES LONG-TERM RENAL ALLOGRAFT OUTCOME. (Abstract #1022)
Jun Yang, Anja Reutzel-Selke, Christoph Steier, Anke Jurisch, Stefan G. Tullius, Hans-Dieter Volk, Thomas Ritter. Berlin, Germany.

4:30 PM LONG-TERM ACCEPTANCE OF RAT CARDIAC ALLOGRAFTS BY COMBINED ADENOVIRUS MEDIATED CTLA4IG AND CD40IG GENE THERAPY. (Abstract #1023)
Kenichiro Yamashita, Naoyuki Yanagida, Hayato Echizenya, Megumi Takehara, Tokushi Kobayashi, Taro Masunaga, Taku Hashimoto, Hideyasu Sakihama, Nun Hua, Hiroyuki Furukawa, Toshimitsu Uede, Satoru Todo. Sapporo, Hokkaido, Japan; Sapporo, Hokkaido, Japan.

4:40 PM SOLUBLE MHC CLASS I GENE TRANSFER EFFECTIVELY INHIBITS MULTIPLE ASPECTS OF THE ANTIDONOR IMMUNE RESPONSE IN ACTIVELY SENSITIZED RAT TRANSPLANT RECIPIENTS. (Abstract #1024)
Marcus N. Scherer, Christian Graeb, Stefan Tange, Karl-Walter Jauch, Edward K. Geissler. Regensburg, Germany.

4:50 PM INHIBITION OF APOPTOSIS IN PANCREATIC BETA CELLS AND ISLETS BY DIRECT TRANSFER OF HEME OXYGENASE-1 PROTEIN FUSED TO A PROTEIN TRANSDUCTION DOMAIN (PTD). (Abstract #1025)
Melina Ribeiro, Dagmar Klein, Sundararajan Jayaraman, Jennifer Embury, Antonello Pileggi, R.Damaris Molano, Luca Inverardi, Camillo Ricordi, Ricardo L. Pastori. Miami, FL.

5:00 PM ADENOVIRAL INDOLEAMINE 2,3-DIOXYGENASE TRANSFER PROLONGS SURVIVAL OF MOUSE SKIN ALLOGRAFTS. (Abstract #1026)
Toshio Miki, Andrea Gambotto, Hong Sun, Alessia Tandin, Annastasia M. Kovscek, John J. Fung, Luis A. Valdivia. Pittsburgh, PA.

5:10 PM CARBON MONOXIDE GENERATED BY HEME OXYGENASE-1 SUPPRESSES ENDOTHELIAL CELL APOPTOSIS THROUGH A P38 MAPK DEPENDENT MECHANISM. (Abstract #1027)
Sophie Brouard, Leo E. Otterbein, Josef Anrather, Edda Tobiasch, Fritz H. Bach, Augustine M.K. Choi, Miguel P. Soares. Boston, MA; Yale, CT; New York, NY.

5:20 PM IMMUNOMODULATION BY DENDRITIC CELLS TRANSFECTED WITH CTLA4-IG IS ENHANCED BY NF-κB DECOY OLIGODEOXYRIBONUCLEOTIDES. (Abstract #1028)
C. Andrew Bonham, Lansha Peng, Zongyou Chen, Lina Du, Paul D. Robbins, Shiguang Qian, Lina Lu. Pittsburgh, PA; Pittsburgh, PA.

Concurrent Session 60: Liver Transplantation: Infection / Immunology

4:00 PM - 5:30 PM

Renaissance Ballroom, Intercontinental
Chairs: Emily Blumberg and Douglas Hanto

4:00 PM LIVER TRANSPLANTATION OF HIV POSITIVE PATIENTS IN THE ERA OF HIGHLY ACTIVE ANTIRETROVIRAL THERAPY (HAART). (Abstract #1029) *Young Investigator Award*
C. Andrew Bonham, S. Forrest Dodson, Margaret V. Ragni, Guilherme Costa, John J. Fung. Pittsburgh, PA; Pittsburgh, PA.

4:10 PM SOLID ORGAN TRANSPLANTATION IN PATIENTS WITH HUMAN IMMUNODEFICIENCY SYNDROME (HIV). A SUCCESSFUL CASE SERIES PRESENTATION. (Abstract #1030)
Nicholas Hadges, Guy W. Neff, Sundararajan Jayarama, David Levi, Seigo Nishida, Joe Tector, Lesli Kravetz, Olivia Hung, Debbie Weppler, Jose R. Nery, Tomoaki Kato, Lorraine Dowdy, Scott Koonce, Chris O'Brien, K. R. Reddy, Eugene R. Schiff, Andreas G. Tzakis. Miami, FL; Miami, FL; Miami, FL; Miami, FL.

4:20 PM THE CLINICAL IMPACT OF HUMAN HERPESVIRUS-6 INFECTION FOLLOWING LIVER TRANSPLANTATION. (Abstract #1031)
A. Humar, D. Kumar, A. Caliendo, G. Levy, G. Moussa, T. Mazzulli. Toronto, ON, Canada; Toronto, ON, Canada; Atlanta, GA.

4:30 PM LONG-TERM ORAL GANCICLOVIR DOES NOT PREVENT PRIMARY CYTOMEGALOVIRUS INFECTION BUT STRONGLY SUPPRESSES VIRAL REPLICATION AND DISEASE. (Abstract #1032)
Arie P. van den Berg, Elisabeth B. Haagsma, Ids J. Klompmaker, Robert J. de Knegt, Hauw The, Maarten J.H. Slooff. Groningen, The Netherlands.

4:40 PM SIGNIFICANCE OF SERIAL QUANTITATIVE MONITORING OF EBV GENOME LOAD BY REAL-TIME PCR IN SHORT TERM AND LONG TERM FOLLOW-UP OF EBV RELATED DISORDER IN RECIPIENTS OF LIVING DONOR LIVER TRANSPLANTATION. (Abstract #1033)
Hiroto Egawa, Tadashi Matsukura, Akiko Yokoi, Toyoichiro Kudo, Koichi Tanaka. Kyoto, Japan; Kyoto, Japan.

4:50 PM A PROSPECTIVE, RANDOMIZED, DOUBLE-BLINDED EVALUATION OF SELECTIVE BOWEL DECONTAMINATION IN LIVER TRANSPLANTATION. (Abstract #1034)
Walter C. Hellinger, Salvador Alvarez, Joseph D. Yao, Peter C. O'Brien, James R. Spivey, Jeffery L. Steers. Jacksonville, FL; Rochester, MN.

5:00 PM ALLOSPECIFIC T SUPPRESSOR CELLS IN LIVER TRANSPLANT RECIPIENTS. (Abstract #1035)
Paola Cinti, Elvira Renna Molajoni, Pasquale Berloco, Massimo Rossi, Raffaello Cortesini. Rome, Italy.

5:10 PM LOSS OF PUTATIVE HEPATIC STEM CELLS IS A HALLMARK OF END-STAGE CHRONIC LIVER ALLOGRAFT REJECTION. (Abstract #1036)
Marius C. van den Heuvel, Koert P. de Jong, Marian L.C. van der Horst, Sibrand Poppema, Maarten J.H. Slooff, Annette S.H. Gouw. Groningen, The Netherlands.

5:10 PM INADEQUATE REACTION OF PUTATIVE HEPATIC STEM CELLS CONTRIBUTES TO DUCTOPENIA IN CHRONIC LIVER ALLOGRAFT REJECTION. (Abstract #1037)
Marius C. van den Heuvel, Koert P. de Jong, Marian L.C. van der Horst, Sibrand Poppema, Maarten J.H. Slooff, Annette S.H. Gouw. Groningen, The Netherlands.

5:20 PM PEDIATRIC AND ADULT EVALUATION OF DACLIZUMAB AS INDUCTION THERAPY FOLLOWING LIVER TRANSPLANTATION WHILE WITHOLDING CALCINERUIN INHIBITION. (Abstract #1038)
Thomas G. Heffron, Gregory A. Smallwood, Todd Pillen, Michael E. de Vera, Laurel Davis, Enrique Martinez, Renee Romero, Andrei C. Stieber. Atlanta, GA.

5:45 PM AST Business Meeting

Sheraton Ballroom 1-3, Sheraton

Poster Session III

8:00 AM - 7:00 PM

Presenters in Attendance: 5:30 PM - 7:00 PM
Beer and Pretzel Reception
River Exhibition Hall

♦*Also presented in Selected Poster Session*

Kidney - Acute/Chronic Rejection III

P1 FACTORS ASSOCIATED WITH HISTOLOGICAL RESOLUTION OF ACUTE REJECTION IN KIDNEY TRANSPLANT RECIPIENTS UNDERGOING PLANNED PAIRED PROTOCOL BIOPSIES. (Abstract #1039)
Lillian W. Gaber, Patricia A. Cowan, M. Francesca Egidi, Robert J. Stratta, M. H. Shokouh-Amiri, Santiago H. Vera, Hani P. Grewal, A. Osama Gaber. Memphis, TN; Memphis, TN.

P2 URINARY TGFβ LEVELS PREDICT URINARY PROTEIN/CREATININE RATIO IN TACROLIMUS AND CYCLOSPORINE-TREATED RENAL TRANSPLANT RECIPIENTS (RTR'S) WITH ENHANCED PLATELET SECRETION AND ELEVATED CIRCULATING TGFβ LEVELS. (Abstract #1040)
Mariana S. Markell, Moro O. Salifu, Frieda Wolf, Anna Babinska, Nabil Sumrani, Joon H. Hong, Dale A. Distant, Bruce G. Sommer, Elizabeth Kornecki, Eli A. Friedman. Brooklyn, NY.

P3 SUBCLINICAL REJECTION AFTER KIDNEY TRANSPLANTATION: A COMPARISON OF TACROLIMUS AND CYCLOSPORINE. (Abstract #1041)
James M. Gloor, Ari J. Cohen, Donna J. Lager, Matthew D. Griffin, Timothy S. Larson, Thomas R. Schwab, Jorge A. Velosa, Mikel Prieto, Scott L. Nyberg, Sylvester Sterioff, Joseph P. Grande, William J. Lund, Mark D. Stegall. Rochester, MN.

P4 INTRAGRAFT GENE EXPRESSION IN CHRONIC KIDNEY ALLOGRAFT NEPHROPATHY. (Abstract #1042)
Mark D. Stegall, Patrick G. Dean, David I. Schwartz, Anis Khair, Timothy S. Larson, Jorge A. Velosa. Rochester, MN.

P5 **THYMOGLOBULIN IS AS EFFECTIVE AS OK-T3 MONOCLONAL ANTIBODY IN THE TREATMENT OF HISTOLOGICALLY SEVERE ACUTE REJECTION IN KIDNEY ALLOGRAFT RECIPIENTS. (Abstract #1043)**
Jonathan Rudick, Joseph Eustace, Jay S. Markowitz, Henkie Tan, Laura Lees, Robert Montgomery, Lloyd Ratner, Milagros Samaniego. Baltimore, MD.

P6 **OBESITY DOES NOT PORTEND A BAD OUTCOME FOR KIDNEY TRANSPLANT RECIPIENTS. (Abstract #1044)**
Richard J. Howard, Alan W. Hemming, Willem J. Van der Werf, Shiro Fujita, Pamela R. Patton, Juan C. Scornik. Gainesville, FL; Gainesville, FL.

P7 **HIGH PRODUCING TUMOR NECROSIS ALPHA (TNF-A) GENE POLYMORPHISM IS ASSOCIATED WITH CHRONIC RENAL ALLOGRAFT DYSFUNCTION (CRAD). (Abstract #1045)**
David Kahan, Nipa Gandhi, David Goldman, Robert Salomon, Ann Marie Melanson, Beth Horth, Michelle Dixon, Debbie Chabot, Ronald Perrone, Kim Salm, Richard B. Freeman. Boston, MA; Deerfield, IL.

P8 **COMPREHENSIVE LIPID ANALYSIS: A NOVEL MEANS OF PRETRANSPLANT RISK STRATIFICATION IN RENAL TRANSPLANTATION. (Abstract #1046)**
Richard Perez, Steve Watkins, Christoph Troppmann, Brian Gallay, J Bruce German. Sacramento, CA; West Sacramento, CA; Sacramento, CA; Davis, CA.

P9 **PRETRANSPLANT HLA ANTIBODIES DETECTED BY FLOW PRA IN RAPAMYCIN TREATED RENAL ALLOGRAFT RECIPIENTS ARE A SIGNIFICANT RISK FACTOR FOR REJECTION. (Abstract #1047)**
Ronald Kerman, Howard Gebel, Chris Garcia, Stephanie Rasmussen, Shauna Garner, Charles Van Buren, Stephen Katz, Richard Knight, Barry Kahan, Robert Bray. Houston, TX; Shreveport, LA; Atlanta, GA.

P10 **NO PATHOLOGIC DIAGNOSIS AFTER KIDNEY BIOPSY: WHAT NEXT? (Abstract #1048)**
Steven Paraskevas, Roger Denny, Arthur J. Matas. Minneapolis, MN.

P11 **GENETIC ADAPTIVE NEURAL NETWORK MODEL FOR PREDICTING GRAFT FAILURE FOLLOWING KIDNEY TRANSPLANTATION. (Abstract #1049)**
Viken Douzdjian, Ashutosh Tewari, Marwan Abouljoud. Detroit, MI; Detroit, MI.

P12 **A SURROGATE MARKER FOR LONG-TERM RENAL ALLOGRAFT SURVIVAL: 6 MONTH SERUM CREATININE (sCr). (Abstract #1050)**
William D. Irish, Raymond J. Tesi, Mark A. Schnitzler. Fremont, CA; St. Louis, MO.

Kidney - GVH, Complications, Infections III

P13 **SERUM ALBUMIN AT 6 WEEKS POST-TRANSPLANT PREDICTS RENAL ALLOGRAFT SURVIVAL INDEPENDENT OF CREATININE VALUE. (Abstract #1051)** ♦
Mariana S. Markell, Moro O. Salifu, Manish Jotwani, Halim Ghali, Nabil Sumrani, Joon H. Hong, Dale A. Distant, Bruce G. Sommer, Eli A. Friedman. Brooklyn, NY.

P14 **GRAFT LOSS AND RECURRENT REJECTIONS IN FLOW CYTOMETRIC CROSS-MATCH POSITIVE PRIMARY RENAL TRANSPLANTS. (Abstract #1052)**
M. Karpinski, D. Rush, J. Jeffery, D. Pochinco, S. Dancea, P. Birk, P. Nickerson. Winnipeg, MB, Canada.

P15 **DONOR TRANSMITTED INFECTION (DTI) AFTER CADAVER RENAL TRANSPLANTATION. (Abstract #1053)**
Paul E. Morrissey, Angelito Yango, Reginald Y. Gohh, Anthony P. Monaco. Providence, RI.

P16 **HOSPITAL RESOURCE UTILIZATION ASSOCIATED WITH TREATMENT OF CYTOMEGALOVIRUS (CMV)-RELATED READMISSION POST RENAL TRANSPLANT. (Abstract #1054)**
Rachael Henderson, David Carlin, Shelah Leader.

P17 **POST-TRANSPLANT LYMPHOMA AFTER ABDOMINAL ORGAN TRANSPLANTION: AN ASSOCIATION WITH CYTOMEGALOVIRUS. (Abstract #1055)**
Matthew J. Graczyk, Steven Paraskevas, Rainer W.G. Gruessner, James E. Coad. Minneapolis, MN; Minneapolis, MN.

P18 **PRETRANSPLANT INFLAMMATION AND RENAL ALLOGRAFT SURVIVAL: THE ROLE OF RECIPIENT CYTOMEGALOVIRUS (CMV) SEROSTATUS AND DIALYSIS MODALITY. (Abstract #1056)**
Richard Perez, Sarah Taranto, Christoph Troppmann, Brian Gallay, John McVicar, Matthew McIntosh. Sacramento, CA; Richmond, VA; Sacramento, CA; Potomoc, MD.

P19 **INFECTION-RELATED COMPLICATIONS AFTER KIDNEY TRANSPLANTATION ARE NOT REDUCED BY LIVE DONATION. (Abstract #1057)**
Robert M. Grossberg, Debbie Cohen, Alyson Rossino, Roy D. Bloom, Emily A. Blumberg.

P20 **THE EFFECT OF MODERN IMMUNOSUPPRESSION ON THE DEVELOPMENT AND BEHAVIOUR OF POSTTRANSPLANT CANCERS IN KIDNEY TRANSPLANT RECIPIENTS. (Abstract #1058)**
Rolf Loertscher, Francine Tremblay, Jean Tchervenkov, Peter Metrakos, Sarkis Meterissian. Montreal, QC, Canada; Montreal, QC, Canada.

P21 **GANCICLOVIR PROPHYLAXIS DELAYS THOUGH DOES NOT PREVENT CYTOMEGALOVIRUS (CMV) INFECTION IN HIGH-RISK RENAL TRANSPLANT RECIPIENTS. (Abstract #1059)**
Roy Bloom, Karen Krok, Debbie Cohen, Emily Blumberg. Phila, PA.

P22 **TACROLIMUS-ASSOCIATED POSTTRANSPLANT DIABETES IN RENAL TRANSPLANT RECIPIENTS: A POSSIBLE ROLE OF HEPATITIS C INFECTION. (Abstract #1060)**
S. Baid, F. L. Delmonico, M. L. Farrell, N. Tolkoff-Rubin, W. Williams, D. Ko, A. B. Cosimi, M. Pascual. Boston, MA.

P23 **HERPES ZOSTER IN RENAL TRANSPLANTATION: A SINGLE CENTER RETROSPECTIVE CASE REVIEW. (Abstract #1061)**
Sita Gourishankar, Jill McDermid, Patricia Hibberd, Jutta Preiksaitis. Edmonton, AB, Canada; Boston, MA.

P24 **TREATMENT OF ACTIVE CMV INFECTION WITH ORAL GANCICLOVIR IN RENAL ALLOGRAFT RECIPIENTS: MONITORING EFFICACY WITH QUANTITATIVE CMV-PCR. (Abstract #1062)**
Stanley C. Jordan, Ashley A. Vo, Suphamai Bunnapradist, Elaine Kamil, Mieko Toyoda. L.A., CA.

P25 **THE FATE OF PATIENTS WHO LOST THEIR KIDNEY TRANSPLANT IN THE 1990'S: THE EFFECT OF GRAFT LOSS ON SUBSEQUENT SURVIVAL. (Abstract #1063)**
Stuart M. Flechner, Barbara Mastroianni, Kathy Savas, Navdeep Boparai, David A. Goldfarb, Charles Modlin, Andrew C. Novick. Cleveland, OH.

P26 **GRAFT FUNCTION AFTER DIAGNOSIS OF BK VIRUS IN ADULT KIDNEY TRANSPLANT RECIPIENTS UNDER TACROLIMUS-BASED IMMUNOSUPPRESSION. (Abstract #1064)**
Velma P. Scantlebury, Ron Shapiro, Gusphyl Justin, Luis S. Re. Pittsburgh, PA; Buenos Aires, Argentina.

P27 **THROMBOTIC MICROANGIOPATHY (TMA) IN RENAL ALLOGRAFT BIOPSIES. (Abstract #1065)**
Sadia Saboor, Muhammad Qadir, Praveen Chander, Rafik A. El Sabrout, Khalid M.H. Butt, Veronica Delaney.

Kidney - Immunosuppression A III

P28 LONG-TERM RESULTS OF LIVING-RELATED HLA-IDENTICAL RENAL TRANSPLANTATIONS WITHOUT STEROIDS OR CALCINEURIN-DEPENDANT DRUGS. (Abstract #1066)
Marie N. Peraldi, Sylvie LeCoz Regnier, Emmanuel Morelon, Henri Kreis. Paris, France.

P29 INCIDENCE OF BIOPSY PROVEN CHRONIC ALLOGRAFT NEPHROPATHY AFTER CORTICOSTEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS ON TACROLIMUS-BASED IMMUNSUPPRESSION. (Abstract #1067) ♦
Martin S. Zand, Urath Suresh, Jonathan Sosnov, Fadi Hijazi, Tibor Nadasdy, Mark Orloff, Richard Demme, Luis Mieles, Amadeo Marcos, Oscar Bronsther. Rochester, NY.

P30 MMF-TACROLIMUS COMBINATION IN KIDNEY TRANSPLANTATION: CRITERIA FOR MPA TOXICITY DISCRIMINATION. (Abstract #1068)
Michel Mourad, Jacques Malaise, Djamila Chaib-Eddour, Josiane König, Raf Scheppers, Pierre Wallemacq, Jean-Paul Squifflet. Brussels, Belgium; Brussels, Belgium.

P31 CONVERSION FROM MYCOPHENOLATE TO RAPAMYCIN FOR ACUTE AND CHRONIC REJECTION. (Abstract #1069) ♦
Radha Vankawala, Ravi K. Kode, Anna M. Damask, Susan Stabler, Mark R. Laftavi, Mysore S. Anil Kumar, Oleh Pankewycz. Philadelphia, PA.

P32 3 YEAR FOLLOWUP OF RANDOMIZED MULTICENTER KIDNEY TRANSPLANT STUDY COMPARING TACROLIMUS(TAC)+ AZATHIOPRINE(AZA) VS CYCLOSPORINE MODIFIED(CSA)+ MYCOPHENOLATE MOFETIL(MMF) VS TAC+MMF. (Abstract #1070)
P. Halloran, N. Ahsan, C. Johnson, T. Gonwa, M. Stegall, M. Hardy, R. Metzger, C. Shield, L. Rocher, J. Scandling, J. Sorenson, L. Mulloy, J. Light, C. Corwin, G. Danovitch, M. Wachs, P. VanVeldhuisen, K. Owen, K. Salm, D. Tolzman, S. King, W. Fitzsimmons. Edmonton, AB, Canada.

P33 IMPROVED RECOVERY FROM DGF IN NEORAL TREATED RENAL TRANSPLANT RECIPIENTS MONITORED BY ABSORPTION PROFILING VERSUS TROUGH LEVELS. (Abstract #1071)
Kamran Mahalati, Joe Lawen, Bryce Kiberd, Philip Belitsky. Halifax, NS, Canada.

P34 RAPAMYCIN AND REDUCED DOSE CYCLOSPORIN PRODUCE EFFECTIVE IMMUNOSUPPRESSION FOR DENOVO RENAL TRANSPLANT RECIPIENTS. (Abstract #1072)
Richard S. Saunders, Gavin J. Murphy, Matthew S. Metcalfe, Rachel Kimber, Michael L. Nicholson. Leicester, United Kingdom.

P35 PREDNISONE METABOLISM IS SLOWER IN SOLID ORGAN TRANSPLANT RECIPIENTS THAN IN NORMALS AND IS PROPORTIONAL TO CYCLOSPORINE METABOLISM. (Abstract #1073)
S. Jeng, T. Pham, S. Tabibzadeh, T. Chanchairujira, W. Jusko, R. Steiner. San Diego, CA; Buffalo, NY; San Diego, CA.

P36 PHARMACOKINETICS (PK) AND TOLERABILITY OF TACROLIMUS AND SIROLIMUS COMBINATION THERAPY IN STABLE RENAL TRANSPLANT RECIPIENTS. (Abstract #1074)
Sundaram Hariharan, Stephen J. Tomlanovich, Ronald S. Filo, Mary Dessimoz, Wayne Wisemandle, Robert W. Townsend. Milwaukee, WI; San Francisco, CA; Indianapolis, IN; Deerfield, IL.

P37 SAFETY AND EFFICACY OF THYMOGLOBULIN INDUCTION WITH INTERMITTENT DOSING BASED ON CD3+ LYMPHOCYTE COUNTS IN HIGH-RISK CADAVER RENAL TRANSPLANT RECIPIENTS. (Abstract #1075)
V. Ram Peddi, Margaret Bryant, Prabir Roy-Chaudhury, E. Steve Woodle, M. Roy First. Cincinnati, OH.

Kidney - Immunosuppression B III

P38 BLOOD TACROLIMUS LEVELS AND CALCINEURIN PHOSPHATASE ACTIVITY EARLY AFTER RENAL TRANSPLANTATION. (Abstract #1076)
Pernille B. Koefoed-Nielsen, Maria B. Gesualdo, Johan V. Povlsen, Else Marie Heinsvig, Jørgen H. Poulsen, Kaj A. Jørgensen. Aarhus N, Denmark; Aarhus C, Denmark; Hershey, PA.

P39 STANDARDIZED IMMUNE MONITORING IN THE EARLY PHASE AFTER KIDNEY TRANSPLANTATION. (Abstract #1077)
Gantuja Bold, Wolf Dietrich Doecke, Gottfried May, Klaus Peter Platz, Didier Paulin Bitti, Hans Dieter Volk, Petra Reinke. Berlin, Germany.

P40 ALLOSPECIFIC HUMAN T SUPPRESSOR CELLS IN RECIPIENTS OF KIDNEYS FROM LIVE DONORS. (Abstract #1078)
Luca Poli, Renzo Pretagostini, Paola Cinti, Elvira Renna Molajoni, Raffaello Cortesini. Rome, Italy

P41 INTRAOPERATIVE ATGAM ♦ (PHARMACIA) - A SAFE AND EFFECTIVE APPROACH FOR INDUCTION THERAPY IN RENAL TRANSPLANT RECIPIENTS. (Abstract #1079)
Khalid Bashir, Brian M. Murray, Joseph P. Gerbasi, Rocco C. Venuto, Daniel Leary. Buffalo, NY.

P42 ABBREVIATED PHARMACOKINETICS OF MYCOPHENOLATE MOFETIL IN RENAL TRANSPLANT RECIPIENTS. (Abstract #1080)
John Johnson, Paula Lavery, Rolf Loertscher. Montreal, QC, Canada.

P43 PEAK WHOLE BLOOD TACROLIMUS CONCENTRATIONS CORRELATE BEST WITH THE AREA-UNDER-THE-CONCENTRATION-TIME CURVE. (Abstract #1081)
John Johnson, Paula Lavery, Rolf Loertscher. Montreal, QC, Canada.

P44 SURROGATE ESTIMATES OF CYCLOSPORINE (CsA) AREA UNDER THE CURVE (AUC) IN RENAL TRANSPLANT RECIPIENTS. (Abstract #1082)
Rumeyza Kazancioglu, Shannon Shockley, Simin Goral, Irene Feurer, J. Harold Helderman, David VanBuren. Nashville, TN.

P45 LOW DOSE TACROLIMUS and SIROLIMUS vs ABSORPTION PROFILE MONITORED CYCLOSPORINE THERAPY IN RENAL TRANSPLANTATION. (Abstract #1083)
Tammy M. Keough-Ryan, Bryce A. Kiberd, Catherine M. Clase, Joseph Lawen, Alan S. MacDonald. Halifax, NS, Canada.

P46 HIGH MYCOPHENOLIC ACID (MPA) EXPOSURE IN KIDNEY RECIPIENTS ON LONG TERM MYCOPHENOLATE MOFETIL (MMF) TREATMENT. (Abstract #1084)
Rob S. Engelbertink, Peter J.H. SmakGregoor, Jan N.M. IJzermans, Willem Weimar, Teun van Gelder. Rotterdam, The Netherlands.

P47 CHRONIC RENAL GRAFT FAILURE: BETTER GRAFT FUNCTION AFTER WITHDRAWAL OF CALCINEURIN INHIBITORS AND SWITCH TO MYCOPHENOLATE MOFETIL - 6 MONTHS FOLLOW UP IN 22 RANDOMIZED RENAL TRANSPLANT RECIPIENTS. (Abstract #1085)
Barbara M. Suwelack, Ulf M.W. Gerhardt, Uta Hillebrand, Martin Hausberg, Helge Hohage. Münster, Germany.

P48 THYMOGLOBULIN VERSUS OKT3 INDUCTION IN HIGH RISK KIDNEY TRANSPLANT RECIPIENTS. (Abstract #1086)
Viken Douzdjian, Iman Bajjoka, Ravi Parasuraman, Marwan Abouljoud. Detroit, MI; Detroit, MI.

Kidney - Pediatrics, Recurrent Disease III

P49 **OUTCOME OF RENAL TRANSPLANTATION (TX) IN PEDIATRIC PATIENTS (PTS) TREATED WITH MYCOPHENOLATE MOFETIL (MMF). (Abstract #1087)**
Salam I. Gharaybeh, Samhar I. Al-Akash, Eleonora M. Lima, Robert E. Ettenger.

P50 **CAUSES OF ANEMIA IN PEDIATRIC (PED) RENAL TRANSPLANT (TX) PATIENTS. (Abstract #1088)**
Eleonora M. Lima, Samhar I. Al-Akash, Salam I. Gharaybeh, Robert B. Ettenger. Los Angeles, CA.

P51 **LONG TERM OUTCOME OF PEDIATRIC RENAL TRANSPLANT RECIPIENTS WHO SURVIVE INTO ADULTHOOD. (Abstract #1089)**
Sharon M. Bartosh, Hans W. Sollinger. Madison, WI; Madison, WI.

P52 **INITIAL IMMUNOSUPPRESSION WITH MYCOPHENOLATE (MMF) OR TACROLIMUS (FK506) IS NOT ASSOCIATED WITH INCREASED RISK FOR POST TRANSPLANT LYMPHOPROLIFERATIVE DISORDER (PTLD) IN PEDIATRIC KIDNEY TRANSPLANTATION. (Abstract #1090)**
Vikas R. Dharnidharka, Ping-Leung Ho, Donald M. Stablein, William E. Harmon, Amir H. Tejani. Gainesville, FL; Potomac, MD; Boston, MA; Valhalla, NY.

P53 **STEROID WITHDRAWAL USING TACROLIMUS AND MMF IN PEDIATRIC RENAL TRANSPLANTATION. (Abstract #1091)**
Yoshihiko Watarai, Ichiro Takeuchi, Junri Shindo, Satoshi Sasaki, Tetsuya Tabata, Tomoaki Usuki, Katsuya Nonomura, Tomohiko Koyanagi. Sapporo, Japan; Sapporo, Japan.

Kidney - Preservation, Donation/Allocation, Economics/ Public Policy, Surgical Techniques, and Other III

P54 **BASILIXIMAB INDUCTION (Bmab) IS MORE EFFECTIVE THAN OKT3 IN RECIPIENTS OF KIDNEYS FROM DONORS OLDER THAN 55 YEARS IN REDUCING ACUTE REJECTION (AR) AND IMPROVING GRAFT SURVVAL (GS). (Abstract #1092)**
Radha Vankawala, Ravi K. Kode, Oleh G. Pankewycz, Aryavarta M.S. Kumar, Mark R. Laftavi, Harry Lam, Anna M. Damask, Billie Fyfe, Mary Beth Tomeny, Debbie Seirka, Kim Phillips, Elizabeth Ferry, Mysore S. Anil Kumar.

P55 **GRAFT DYSFUNCTION IN RECIPIENTS WITH KIDNEY OF HYPERTENSIVE CADAVERIC DONORS. THE ROLE OF ZERO HOUR RENAL BIOPSY. (Abstract #1093)**
Paolo F. Schena, Giovanni Stallone, Loreto Gesualdo, Salvatore Di Paolo, Antonio Schena, Francesco P. Selvaggi, Battaglia Michele, Di Tonno Pasquale, De Ceglie Giovanni, Pace G.. Bari, Italy; Bari, Italy.

P56 **A RANDOMIZED TRIAL OF EXERCISE TRAINING FOLLOWING RENAL TRANSPLANTATION. (Abstract #1094)**
Patricia L. Painter, Steven L. Tomlanovich, Lisa A. Hector, Karen A. Ray, Nancy L. Ascher.

P57 **HAND-ASSISTED LAPAROSCOPIC DONOR NEPHRECTOMY: A SINGLE CENTER EXPERIENCE. (Abstract #1095)**
Paul L. Tso, C. Daniel Smith, Gene D. Branum, Thomas C. Pearson, Christian P. Larsen. Atlanta, GA.

P58 **HLA MATCHING FOR KIDNEY TRANSPLANTATION SIGNIFICANTLY ENHANCES SURVIVAL, REDUCES ACUTE REJECTION AND MINIMISES SENSITISATION. (Abstract #1096)**
Philip A. Dyer, Susan Martin, Judith E. Worthington, Salim Al-Maket, Afshin Tavakoli, Hany N. Riad.

P59 **NON-COMPLIANCE IN RENAL TRANSPLANT RECIPIENTS: A MULTIVARIATE ANALYSIS OF RISK FACTORS AND IMPACT IN RENAL FUNCTION AND ACUTE REJECTION. (Abstract #1097)**
Rafael Reyes-Acevedo, Oscar Ron-Torres, Irene Lopez, Luis Romo-Franco, Imelda Davila, Alfredo Chew-Wong. Aguascalientes, Ags., Mexico; Aguascalientes, Ags., Mexico.

P60 **THE PATHOGENESIS OF POST-TRANSPLANT (TX) OSTEODYSTROPHY AS DETECTED BY EARLY ALTERATIONS IN BONE REMODELING. (Abstract #1098)**
Eudocia Rojas, Raul G. Carlini, Paul H. Clesca, Anabella Arminio, Jose Weisinger, Keith A. Hruska, Bellorin Ezequiel. Caracas, DF, Venezuela; St Louis, MO; Caracas, DF, Venezuela.

P61 **OUTCOME OF CONGESTIVE HEART FAILURE DUE TO LEFT VENTRICULAR SYSTOLIC DYSFUNCTION (DECREASED EJECTION FRACTION) IN PATIENTS WITH FUNCTIONING RENAL TRANSPLANT. (Abstract #1099)**
Gregory S. Wang, Lavanya Bellumkonda, Riple Hansalia, Emilio Ramos, David Klassen, Jeffrey C. Fink, Steven Blahut, Michael Fisher, Matthew Weir, Ravinder R. Wali. Baltimore, MD.

P62 **PREDOMINANCE OF FEMORAL NECK BONE LOSS BY DEXA SCAN IN PATIENTS AFTER KIDNEY TRANSPLANT. (Abstract #1100)**
Lalit M. Verma, Flores Raymond, Fink Jeffrey, Weir R. Matthew, Ravinder K. Wali. Baltimore, MD.

P63 **MORBIDITY IN RECIPIENTS WITH RENAL ALLOGRAFTS FUNCTIONING FOR OVER TWENTY YEARS. (Abstract #1101)**
Richard P. Baker, Afshin Tavakoli, Hany N. Riad, Philip A. Dyer, Nethar P. Mallick, Robert W.G. Johnson. Manchester, United Kingdom.

P64 **MICROPARTICLE FLOW CYTOMETRIC PRA ANALYSIS: WHEN NEGATIVE COULD MEAN POSITIVE AND POSTIVE COULD MEAN NEGATIVE. (Abstract #1102)**
Robert A. Bray, Howard M. Gebel. Atlanta, GA; Shreveport, LA.

P65 **INCREASE IN BLOOD LIPIDS IN DE NOVO RENAL TRANSPLANT PATIENTS FOLLOWING CERTICAN™ (RAD) ADMINISTRATION IS MANAGEABLE AND NOT ASSOCIATED WITH INCREASED CARDIOVASCULAR RISK. (Abstract #1103)**
H. Tedesco-Silva, B. Kaplan, R. Mendez, B. Kahan, D. Van Buren, R. Boger, the RAD 251 Study Group. Sao Paulo, Brazil.

P66 **SIGNIFICANT PROTEINURIA OCCURS TERMINALLY IN "NORMAL" CADAVER KIDNEY DONORS WITHOUT APPARENT RENAL DISEASE. (Abstract #1104)**
Robert W. Steiner, Sandra F. Leyden. San Diego, CA.

P67 **THE NEW RELIABLE METHOD FOR PREDICTING IMMEDIATE GRAFT FUNCTION AFTER KIDNEY TRANSPLANTATION. (Abstract #1105)**
Roman Danielewicz, Maciej Kosieradzki, Artur Kwiatkowski, Wojciech P. Polak, Michal Wszola, Slawomir Fesolowicz, Irena Wegrowicz-Rebandel, Grzegorz Michalak, Janusz E. Walaszewski, Wojciech A. Rowinski.

P68 **ISCHEMIA INJURY AND RENAL VIABILITY ASSESSMENT USING GLUTATHIONE S-TRANSFERASE DURING MACHINE PRESERVATION. (Abstract #1106)**
Wojciech P. Polak, Artur Kwiatkowski, Roman Danielewicz, Maciej Kosieradzki, Michal Wszola, Wojciech Lisik, Grzegorz Michalak, Slawomir Fesolowicz, Leszek Paczek, Janusz E. Walaszewski, Wojciech A. Rowinski. Warsaw, Poland.

P69 **EXPERIENCE WITH LAPAROSCOPIC DONOR NEPHRECTOMY [LDN] VS OPEN NEPHRECTOMY [ON]. (Abstract #1107)**
Shamkant Mulgaonkar, Moushumi Vaidya, Otto Leiti, Debra Morgan, Eleanor Simchera, Penny Defranco, Luigi Bonomini, Gary Friedman, Stuart Geffner. Livingston, NJ.

P70 OLDER RECIPIENTS DEMONSTRATE A STRONGER ALLORESPONSE TO BOTH MARGINAL AND 'NORMAL' DONOR ORGANS COMPARED TO YOUNGER RECIPIENTS IN AN EXPERIMENTAL MODEL. (Abstract #1108)
Anja Reutzel-Selke, Ulrike Bachmann, Anke Jurisch, Kirstin Atrott, Johann Pratschke, Peter Neuhaus, Hans-Dieter Volk, Stefan G. Tullius. Berlin, Germany.

P71 BLOOD PRESSURE CHARACTERISTICS OF AN EXPANDING AND AGING RENAL TRANSPLANT DONOR POOL. (Abstract #1109)
Stephen Textor, Velosa Jorge, Larson Tim, Stegall Mark, Prieto Mikel, Taler Sandra, Schwartz Lora. Rochester, MN.

P72 EXPRESSION OF STRESS-INDUCIBLE PROTEINS AFTER HUMAN KIDNEY TRANSPLANTATION: POSSIBLE CORRELATION WITH DONOR CRITERIA AND COLD ISCHEMIA TIME. (Abstract #1110)
Thorsten Vowinkel, Christian August, Heiner Wolters, Marc Schult, Jens Brockmann, Stefan Heidenreich, Hideo A. Baba, Norbert Senninger, Karl-Heinz Dietl. Münster, Germany; Münster, Germany; Münster, Germany.

P73 CYSTATIN C MORE ACCURATELY REFLECTS GLOMERULAR FILTRATION RATE THAN DOES SERUM CREATININE FOLLOWING RENAL AND NON-RENAL WHOLE ORGAN TRANSPLANTATION. (Abstract #1111)
Timothy S. Larson, Christopher K. Buehrig, Jan H. Bergert, Gregory R. Pond, Erik J. Bergstrahl, Jorge A. Velosa, Mark D. Stegall. Rochester, MN.

P74 GENDER DIFFERENCES IN THE SIGNIFICANCE OF A POSITIVE B-CELL CROSSMATCH FOR RENAL ALLOGRAFT SURVIVAL. (Abstract #1112)
Vahakn B. Shahinian, Andrew A. House, Stephen H. Leckie, William T. Howson, Norman Muirhead, Patrick P. Luke, David J. Hollomby, Michael Bloch, Anthony M. Jevnikar. London, Canada.

P75 COMPARISON OF TRENDS IN LIVING-DONOR RENAL TRANSPLANTS BETWEEN CAUCASIANS AND AFRICAN AMERICANS. (Abstract #1113)
Viken Douzdjian, Aaron Rabinovitch, Ravi Parasuraman, Atsushi Yoshida, Marwan Abouljoud. Detroit, MI; Detroit, MI.

P76 FACTORS AFFECTING THE FIRST 3-YEAR RENAL ALLOGRAFT FUNCTION AFTER LIVE DONOR RENAL TRANSPLANTATION. (Abstract #1114)
Yu Seun Kim, Myoung Soo Kim, Dong Kee Kim, Sung Min Myoung, Soon Il Kim, Kiil Park. Seoul, Korea; Wonju, Korea; Seoul, Korea.

P239 COMPARISON OF OPEN, LAPAROSCOPIC AND HAND-ASSIST LIVE DONOR NEPHRECTOMY. (Abstract #1114.5) ♦
Ergun Velidedeoglu, Ali Naji, Kenneth L. Brayman, Noel N. Williams, Niraj M. Desai, Luis Campos, Maral Palanjian, Martin Wocjik, Roy D. Bloom, Robert Grossman, Kevin C. Mange, Jo Buyske, Clyde F. Barker, James F. Markman. Philadelphia, PA; Philadelphia, PA

Liver - Immunosuppression, Acute/Chronic Rejection, GVH, Pediatrics III

P77 CYP3A4♦1B AS A PHARMACOGENOMIC PREDICTOR OF LOW TACROLIMUS REQUIREMENTS IN LIVER TRANSPLANT PATIENTS. (Abstract #1115) ♦
Janet L. Karlix, Heather Myers, Son Nguyen, Clara Johary, Kareem Albekairy, Valerie Greene, Alan Hemming, Willem van der Werf, Alan Reed, Richard Howard. Gainesville, FL; Gaineville, FL.

P78 EARLY REJECTION IN FULMINANT HEPATIC FAILURE. (Abstract #1116)
Pedro Trigo, Javier C. Lendoire, Gustavo Braslavsky, Marcelo Amante, Nora Cejas, Oscar C. Imventarza. Buenos Aires, Argentina.

P79 TACROLIMUS IS EFFECTIVE IN BOTH DUAL AND TRIPLE REGIMENS AFTER LIVER TRANSPLANTATION. (Abstract #1117)
Miguel Garcia-Gonzalez, A. Bernardos, M. Gomez, J. Ortiz de Urbina, P. Lopez Cillero, F. San Juan, P. Parrilla, J. I. Herrero. Madrid, Spain.

P80 PRE-AND POSTTRANSPLANT PREDICTORS OF RENAL DYSFUNCTION FOLLOWING LIVER TRANSPLANTATION. (Abstract #1118)
A. J. Cohen, M. D. Stegall, W. K. Kremers, C. O. Zein, H. Bohorquez, N. N. Onaca, C. R. Rosen, R. H. Wiesner, N. N. Zein. Rochester, MN.

P81 SIROLIMUS-BASED STEROID-FREE IMMUNOSUPPRESSION FOR LIVER TRANSPLANTATION. (Abstract #1119)
Norman Kneteman, Vincent Bain, David Bigam, Glenda Meeberg, Daniel Kosoy, AM James Shapiro, Mang Ma, Winnie Wong, Klaus Gutfreund. Edmonton, AB, Canada; Edmonton, AB, Canada; Edmonton, AB, Canada.

P82 LONG-TERM RESULTS OF EARLY STEROID WITHDRAWAL FOLLOWING LIVER TRANSPLANTATION. (Abstract #1120)
Paul D. Greig, David R. Grant, Norman R. Kneteman, Vivian C. McAlister, Andre F. Roy, Charles H. Scudamore, Jean I. Tchervenkov, William J. Wall, Eric M. Yoshida. Toronto, ON; Edmonton, AB.; Halifax, NS; Montreal, QC; Vancouver, BC; Montreal, QC; London, ON, Canada.

P83 TRANSIENT VASCULAR PATHOLOGICAL ALTERATIONS IN THE EARLY POSTTRANSPLANT PERIOD OF ISOLATED SMALL BOWEL ALLOGRAFT RECIPIENTS: RELATIONSHIP TO HUMORAL SENSITIZATION AND OTHER CLINICAL FEATURES. (Abstract #1121)
P. Ruiz, M. Garcia, P. Pappas, V. Esquenazi, T. Kato, N. Mittal, D. Weppler, D. Levi, S. Nishida, J. Nery, J. Miller, A. Tzakis. Miami, FL; Miami, FL.

P84 REDUCED GRAFT LOSS IN PEDIATRIC LIVING-RELATED LIVER TRANSPLANTATION. (Abstract #1122)
Puneet Gupta, David Cronin, J. Michael Millis, Lynda Brady. Chicago, IL; Chicago, IL.

P85 BENEFITS OF CYCLOSPORINE MICROEMULSION (NEORAL®) C-2 MONITORING ARE SUSTAINED AT ONE YEAR IN DE NOVO LIVER TRANSPLANT RECIPIENTS. (Abstract #1123)
Stephen G. Pollard, the NEO-INT06 International Study Group.

P86 RELATIONSHIP BETWEEN CYCLOSPORINE MICROEMULSION (NEORAL) C-2 LEVELS AND EXPOSURE IN DE NOVO AND MAINTENANCE PEDIATRIC LIVER TRANSPLANT RECIPIENTS. (Abstract #1124)
Stephen P. Dunn, Kathleen Falkenstein, Oliver David, Gerard Cooney. Wilmington, DE; London.

P87 PREDICTORS OF SURVIVAL AFTER HEPATIC RETRANSPLANTATION IN CHILDREN. (Abstract #1125)
Sukru Emre, Sasan Roayaie, Ravinder Anand, The SPLIT Research Group. New York, NY; Potomac, MD.

Liver - Infections, Complications, Recurrent Disease, Surgical Techniques III

P88 PILOT STUDY UTILIZING AMANTADINE, INTERFERON-α, AND RIBAVIRIN IN RECURRENT HEPATITIS C FOLLOWING LIVER TRANSPLANTATION. (Abstract #1126)
Michael E. de Vera, Gregory A. Smallwood, Laurel Davis, Andrei C. Stieber, Thomas G. Heffron. Atlanta, GA.

P89 ADULT LIVING DONOR LIVER TRANSPLANT: THE UNIVERSITY OF COLORADO EXPERIENCE. (Abstract #1127)
Michael E. Wachs, Thomas E. Bak, Gregory T. Everson, James Trotter, Thomas Trouillot, Marcelo Kugelmas, Tracy Steinberg, Mary McClure, Tim Brackett, Igal Kam. Denver, CO; Denver, CO.

P90 **DOES HEPATITIS C INFECTION PROMOTE IMMUNOSUPPRESSION? (Abstract #1128)**
Pam M. Kimball, Mitch L. Shiffman. Richmond, VA.

P91 **SCLEROMYXEDEMA AND MYONECROSIS FOLLOWING LIVER TRANSPLANTATION. (Abstract #1129)**
Pedro W. Baron, Donald Hillebrand, John Rambharose, Ke-Qin Hu, Okechukwu Ojogho, Ken Berger, James Pappas, Sandra Nehlsen-Cannarella, Waldo Concepcion. Loma Linda, CA; Loma Linda, CA; Loma Linda, CA; Loma Linda, CA.

P92 **THE RACE OF THE RECIPIENTS DOES NOT AFFECT THE OUTCOME OF LIVER TRANSPLANTATION. (Abstract #1130)**
Rita Lepe, Laura Kulik, Pierpaolo Sileri, Thelma E. Wiley, Luca Cicalese, Patricia Gaddis, Thomas J. Layden, Enrico Benedetti. Chicago, IL; Chicago, IL.

P93 **EVALUATION OF CALCIUM AND VITAMIN D SUPPLEMENTATION IN LIVER TRANSPLANT RECIPIENTS. (Abstract #1131)**
Cheryl Reid, Robert E. Dupuis, Amy L. Fann, David J. Taber, Amy Stzralka, Patricia Odell, Roshan Strestha, Steven Zacks, Mark W. Johnson, Jeffrey Fair. Chapel Hill, NC; Chapel Hill, NC; Chapel Hill, NC.

P94 **COMPARISON OF TRANSVERSE HEPATECTOMY AND LIVER TRANSPLANTATION FOR POLYCYSTIC LIVER DISEASE. (Abstract #1132)**
Sherfield Dawson, III, Robert Fisher. Richmond, VA.

P95 **EVALUATION OF THE INTRA-OPERATIVE FLOWMETRY AND THE ROLE OF THE LEGATURE OF THE SPLENIC ARTERY IN ADULT LIVING DONOR LIVER TRANSPLANTATION (ALDLTx): A WAY TO REDUCE SINUSOIDAL HYPERTENSION? (Abstract #1133)**
Roberto Troisi, Guy Cammu, Oreste Cuomo, Bernard De Hemptinne. Ghent, Belgium.

P96 **COMPARISON OF EMOTIONAL AND COGNITIVE FUNCTIONING IN PATIENTS PRE- AND POST-LIVER TRANSPLANT. (Abstract #1134)**
Robin C. Hilsabeck, Meghan D. Carlson, William Perry, Tarek I. Hassanein.

P97 **THE EFFECT OF DONOR AGE ON OUTCOME AFTER LIVER TRANSPLANT. (Abstract #1135)**
Sasan Roayaie, Thomas M. Fishbein, Myron E. Schwartz, Patricia A. Sheiner, Charles M. Miller, Sukru Emre. New York, NY.

P98 **AN ANALYSIS OF AUTOPSIES OF PATIENTS AWAITING LIVER TRANSPLANTATION: WHAT HAVE WE LEARNED? (Abstract #1136)**
Susan M. Lerner, Nicholas N. Nissen, Denise Arthur, Charles Lassman, Galen Cortina, Michael Fishbein, Angeles Baquerizo, Sunil Geevarghese, Pauline Chen, Douglas G. Farmer, Rafik M. Ghobrial, Hasan Yersiz, Ronald W. Busuttil. Los Angeles, CA.

P99 **ADULT LIVER TRANSPLANTATION: AN ANALYSIS OF THE CAUSES OF DEATH IN AUTOPSIED PATIENTS. (Abstract #1137)**
Susan M. Lerner, Nicholas N. Nissen, Denise Arthur, Charles Lassman, Galen Cortina, Michael Fishbein, Angeles Baquerizo, Sunil Geevarghese, Pauline W. Chen, Douglas G. Farmer, Rafik M. Ghobrial, Hasan Yersiz, Ronald W. Busuttil. Los Angeles, CA.

P100 **ANALYSIS OF 50 CONSECUTIVE RIGHT LOBES LIVER TRANSPLANTS FROM LIVING DONORS. (Abstract #1138)**
Gabriel Gondolesi, Thomas M. Fishbein, Cal Matsumoto, Sukru Emre, Patricia A. Sheiner, Myron E. Schwartz, Leona Kim-Schluger, Charles M. Miller. New York, NY.

P101 **USE OF RITUXIMAB(MONOCLONAL ANTI CD20 ANTIBODY)FOR THE TREATMENT OF PTLD. (Abstract #1139)**
Spiros Delis, Shogo Kobayashi, Tomoaki Kato, Guy Neff, Jose R. Nery, Joseph Tecter, Lisa Babinsky, David M. Levi, Seigo Nishida, Philip Ruiz, Andreas G. Tzakis. Miami, FL; Miami, FL; Miami, FL.

P102 **OUTCOME OF SPLIT IN SITU GRAFTS IN ADULT AND PEDIATRIC PATIENTS AFTER LIVER TRANSPLANTATION. (Abstract #1140)**
Umberto Maggi, Giorgio Rossi, Paolo Reggiani, Lucio Caccamo, Stefano Gatti, Gianni Paone, Ernesto Melada, Maurizio Doglia, Luigi Rainero Fassati. Milano, Italy.

P103 **LONG-TERM SURVIVAL OF EARLY STAGE CHOLANGIOCARCINOMA IN PATIENTS WITH PSC USING COMBINED LIVER TRANSPLANTATION, PANCREATICODUODENECTOMY AND BRACHYTHERAPY. (Abstract #1141)**
Youmin Wu, Frederick C. Johlin, Jr., Stephen C. Rayhill, Michael Voigt, Warren Schmidt, RouYee Chenhsu, Douglas LaBrecque. Iowa City, IA.

Liver - Preservation, Economics/Public Policy, Donation Allocation, Other III

P104 **LIVING DONOR LIVER TRANSPLANTATION AT THE UNIVERSITY OF ESSEN. (Abstract #1142)**
Massimo Malagó, Giuliano Testa, Camino Valentin-gamazo, Hauke Lang, Christoph Broelsch. Essen, Germany.

P105 **VOLUMETRIC EVALUATION OF RIGHT LOBE LIVING LIVER DONORS: A NOVEL SURGICAL APPROACH FOR THE RADIOLOGIST. (Abstract #1143)**
Alan Koffron, Mario Ferrario, Albert Nemcek, James Carr, Andres Blei, Ikuo Hirano, Jonathan Fryer, Ricardo Superina, Dixon Kaufman, Joseph Leventhal, Frank Stuart, Michael Abecassis. Chicago, IL.

P106 **NEUROPSYCHIATRIC FUNCTIONING FOLLOWING LIVER TRANSPLANTATION FOR ACUTE LIVER FAILURE. (Abstract #1144)**
Elizabeth W. Jackson, John Ryan, Mark W. Johnson, David Gerber, Kenneth Andreoni, Steven Zacks, Jeffrey Fair, Roshan Shrestha, Michael W. Fried. Chapel Hill, NC; Chapel Hill, NC.

P107 **HEPATITIS A: A MAIN CAUSE OF LIVER TRANSPLANTATION IN FULMINANT HEPATIC FAILURE. (Abstract #1145)**
Oscar C. Imventarza, Javier C. Lendoire, Marcelo Dip, Pedro Trigo, Gustavo Bianco, Guillermo Cervio, Jose Saul, Mirta Ciocca, Gustavo Braslavsky, Luis Rojas. Buenos Aires, Argentina.

P108 **COMPARISON BETWEEN PORTAL PRESSURE AND WEDGED HEPATIC VENOUS PRESSURE IN THE TRANSPLANTED LIVER. (Abstract #1146)**
Paulo C.B. Massarollo, Marilia D.G. Brescia, Andre Beer, Jr., Oswaldo I. Pereira, Sergio Mies. Sao Paulo, SP, Brazil.

P109 **PROSPECTIVE STUDY OF THE EVOLUTION OF ANTI-HBs+ RECIPIENTS OF ANTI-HBc+ HEPATIC GRAFTS. (Abstract #1147)**
David del Pozo, Rafael Barcena Marugan, Santos del Campo Terron, Javier Moreno Garcia, Marisa Mateos L., Rosa Nash, Emilio de Vicente, Fernando Garcia-Hoz. Madrid, Spain; Madrid, Spain; Madrid, Spain.

P110 **NITRIC OXIDE AND ISCHAEMIC PRECONDITIONING OF THE LIVER. (Abstract #1148)** ♦
Rahul S. Koti, Wenxuan Yang, Janice Tsui, Alexander M. Seifalian, Brian R. Davidson. London, United Kingdom.

P111 **HEPATOCELLULAR CARCINOMA ABLATIVE REGIMEN FOR OPTIMIZING SURGICAL RESECTION, TRANSPLANT, AND PALLIATION. (Abstract #1149)**
Robert A. Fisher, Melodie L. Anderson, John M. Ham, Luke G. Wolfe, Mitchell L. Shiffman, Richard K. Sterling, A. Scott Mills, Marc P. Posner.

P112 THE PSYCHOLOGICAL TOLL OF SELECTION AND WAITING FOR LIVER TRANSPLANTATION. (Abstract #1150)
Robert M. Weinrieb, Deborah H.A. Van Horn, Michael R. Lucey. Philadelphia, PA; Philadelphia, PA; Philadelphia, PA.

P113 CAUSES OF LATE HEPATIC ALLOGRAFT LOSS AND DEATH FOLLOWING LIVER TRANSPLANTATION. (Abstract #1151)
Raquel D. Conceicao, Russell H. Wiesner, Charles B. Rosen, Woong R. Kim, Jane M. Anderson, Michael B. Ishitani, Michael R. Charlton. Rochester, MN.

P114 1 AND 4 YEAR PATIENT SURVIVAL AFTER ADULT FIRST LIVER TRANSPLANTATION IN THE UK, 1994 TO 2000. (Abstract #1152)
Sanjaya P. Wijeyekoon, Lynn P. Copley, Jan van der Meulen, Giles J. Toogood.

P115 SPLIT IN SITU LIVER GRAFTS AND DONORS' AGE. (Abstract #1153) ♦
Umberto Maggi, Giorgio Rossi, Paolo Reggiani, Lucio Caccamo, Stefano Gatti, Gianni Paone, Ernesto Melada, Maurizio Doglia, Alessia De Simone, Simone Olmetti, Paolo Trezza, Luigi Rainero Fassati. Milano, Italy.

P116 OUTCOME OF LIVER TRANSPLANTATION FOR HEPATIC FAILURE AFTER HEPATIC RESECTION. (Abstract #1154)
Yuichiro Otsuka, Pauline W. Chen, Douglas G. Farmer, Mitsugi Shimoda, Susan Lerner, Nicholas Nissen, Farin Amersi, Rafik M. Ghobrial, Roland W. Busuttil. Los Angeles, CA.

Pancreas and Islets - All Topics III

P117 THE IMPACT OF PANCREAS TRANSPLANTATION ON PATIENT EMPLOYMENT OPPORTUNITIES. (Abstract #1155)
Richard J. Knight. Houston, TX.

P118 EVALUATION OF THE CONTRAST ENHANCED MAGNETIC RESONANCE ANGIOGRAPHIC (CE-MRA) APPEARANCE OF THE DYSFUNCTIONING AND NORMAL FUNCTIONING PANCREATIC ALLOGRAFT. (Abstract #1156)
R. Wolf, J. J. Homan van der Heide, Th. Kok, W. J. Sluiter, R. L. Kamman, R. J. Ploeg, W. J. Boeve.

P119 SIGNIFICANCE OF ELEVATED SERUM LIPASE AS AN INDICATOR OF REJECTION IN PANCREAS TRANSPLANTATION. (Abstract #1157)
Pradip K. Chakrabarti, Amitabh Gautam, Ron Shapiro, Shigetaka Inoue, Robert J. Corry. Pittsburgh, PA.

P120 IMPACT OF COLD ISCHEMIA TIME (CIT) ON ALLOGRAFT SURVIVAL, FUNCTION AND SURGICAL COMPLICATIONS FOLLOWING PANCREAS TRANSPLANTATION. (Abstract #1158)
Pradip K. Chakrabarti, Amitabh Gautam, Ron Shapiro, Robert J. Corry. Pittsburgh, PA.

P121 SIMULTANEOUS KIDNEY-PANCREAS TRANSPLANTATION WITH PORTAL-ENTERIC DRAINAGE AND TACROLIMUS/MYCOPHENOLATE MOFETIL-BASED IMMUNOSUPPRESSION. (Abstract #1159)
Robert J. Stratta, M. Hosein Shokouh-Amiri, M. Francesca Egidi, Hani P. Grewal, A. Tarik Kizilisik, N. Nezakatgoo, Lillian W. Gaber, A. Osama Gaber. Memphis, TN.

P122 THYMOGLOBULIN INDUCTION OR REJECTION THERAPY IN PANCREAS TRANSPLANTATION: A SINGLE CENTER EXPERIENCE. (Abstract #1160)
Jennifer Trofe, Robert J. Stratta, Agnes Lo, Lillian W. Gaber, M. Hosein Shokouh-Amiri, Hani P. Grewal, M. Francesca Egidi, Rita R. Alloway, A. Osama Gaber. Memphis, TN.

P123 NON-IMMUNOLOGIC ALLOGRAFT PANCREATITIS—PROGNOSTIC SIGNIFICANCE? (Abstract #1161)
Rochelle M. Cunningham, David K. Klassen, Charles B. Cangro, Matthew R. Weir, Stephen T. Bartlett. Baltimore, MD.

P124 FATE OF THE PANCREAS AFTER ASYNCHRONOUS KIDNEY LOSS IN PATIENTS UNDERGOING SIMULTANEOUS KIDNEY/PANCREAS TRANSPLANTATION (SPK). (Abstract #1162)
Srinath Chinnakotla, Rodney J. Taylor, Arnaud DeRoover, Robert J. Stratta, John P. Leone.

P125 MULTIVISCERAL TRANSPLANTATION WITHOUT THE LIVER. (Abstract #1163)
Tomoaki Kato, Andreas G. Tzakis, Shogo Kobayashi, Lisa Babinsky, Seigo Nishida, David M. Levi, Jose R. Nery. Miami, FL.

P126 THE NATIONAL TRANSPLANTATION PREGNANCY REGISTRY: PREGNANCY OUTCOMES IN FEMALE PANCREAS-KIDNEY RECIPIENTS. (Abstract #1164)
Lisa A. Coscia, Carolyn H. McGrory, Lydia Z. Philips, John S. Radomski, Michael J. Moritz, Donald C. Dafoe. Philadelphia, PA.

P127 TWO DOSES OF DACLIZUMAB PROVIDES EFFECTIVE INDUCTION FOR SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION. (Abstract #1165)
Willem J. Van der Werf, Alan I. Reed, Alan W. Hemming, Shiro Fujita, Pamela R. Patton, Janet Crabtree, Titte Srinivas, Richard J. Howard. Gainesville, FL.

Heart/Lung - All Topics III

P128 DO BLACK HEART TRANSPLANT RECIPIENTS TREATED WITH TACROLIMUS OR CYCLOSPORINE IMMUNOSUPPRESSION DEMONSTRATE DISPARATE METABOLIC OUTCOMES? (Abstract #1166) ♦
Patricia A. Uber, Mandeep R. Mehra, N. Vivekananthan, Robert L. Scott, Myung H. Park. New Orleans , LA.

P129 AN INVESTIGATION OF THE PREVALENCE AND CLINICAL CORRELATES OF VENTRICULAR ASYNCHRONY AFTER HEART TRANSPLANTATION. (Abstract #1167)
Srinivas Potluri, Ananth Prasad, Patricia A. Uber, Robert L. Scott, Myung H. Park, Mandeep R. Mehra. New Orleans, LA.

P130 CORRELATION BETWEEN THE GLOMERULAR FILTRATION RATE, SERUM CREATININE, CREATININE CLEARANCE AND THE CALCULATED CREATININE CLEARANCE IN HEART TRANSPLANT PATIENTS. (Abstract #1168)
M. Cantarovich, E. Cyr, R. Chartier, N. Giannetti, R. Cecere. Montreal, QC, Canada; Montreal, QC, Canada; Montreal, QC, Canada.

P131 MONITORING OF ANTI-HLA CLASS I AND II ANTIBODIES BY FLOW CYTOMETRY AT THE TIME OF POST TRANSPLANT ENDOMYOCARDIAL BIOPSY. (Abstract #1169)
Joel Fernandez, Mark Weston, William LeFor, Mayra Lopez-Cepero. Tampa, FL; Tampa, FL.

P132 EXERCISE TOLERANCE AFTER HEART TRANSPLANTATION: INFLUENCE OF LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC FUNCTION. (Abstract #1170) ♦
M. Dandel, R. Ewert, M. Hummel, J. Müller, R. Meyer, R. Hetzer. Berlin, Germany.

P133 TRANSPULMONARY PRESSURE GRADIENT IS A SIGNIFICANT PREDICTOR FOR MORTALITY AFTER ORTHOTOPIC CARDIAC TRANSPLANTATION. (Abstract #1171)
Michael S. Gorlitzer, Nico Fiegl, Michaela Lanzenberger, Jan Ankersmit, Meinhart Ploner, Andreas Zuckermann, Sharokh Taghavi, Georg Wieselthaler, Juliane Kilo, Ernst Wolner, Michael Grimm. Vienna, Vienna, Austria.

P134 HLA-CYTOMEGALOVIRUS (CMV) INTERACTION IN RELATION TO REJECTION AND VASCULOPATHY FOLLOWING HUMAN HEART TRANSPLANTATION. (Abstract #1172) ♦
Mohamad H. Yamani, Ashraf Abdo, James B. Young, Randall C. Starling, Norman B. Ratliff, Robin Avery, Murat Tuzcu, Patrick McCarthy, Daniel Cook. Cleveland, OH.

P135 **INDUCTION WITH BASILIXIMAB PRESERVES RENAL FUNCTION FOLLOWING CARDIAC TRANSPLANTATION. (Abstract #1173)**
Ank E. Vriesendorp, Paul B. Rosenberg, Mark H. Drazner, Daniel Dries, Pat A. Kaiser, J. Michael DeMaio, Daniel L. Meyer, W. Steves Ring, Clyde W. Yancy. Dallas, TX; Dallas, TX; Dallas, TX.

P136 **INFLUENCE OF AGE ON LUNG TRANSPLANT RECIPIENTS. (Abstract #1174)**
Salem M. Al-faifi, Carlos A. Gutierrez, Cecilia B. Chaparro, Charlie K. Chan, Shaf Keshavjee, Tom K. Waddell, Michael A. Hutcheon. Toronto, ON, Canada; Toronto, ON, Canada.

P137 **SIROLIMUS AND TACROLIMUS IN CLINICAL CARDIAC TRANSPLANTATION: A PILOT STUDY. (Abstract #1175)**
Xiao-shi Qi, Pi-Chong Lee, Richard Kaplon, Alice F. Loo, Steve Mallon, Kushagra Katariya, Eugene J. Bauerlein, Si M. Pham. Miami, FL; Miami, FL; Miami, FL.

P138 **CARDIAC GRAFT GROWTH AFTER HEART TRANSPLANTATION IN CHILDREN. (Abstract #1176)**
Sylvie Di Filippo, François Sassolas, Jean Ninet, Gérard Champsaur, André Bozio. Lyon, France.

P139 **OUTCOMES OF PREGNANCIES IN FEMALE HEART TRANSPLANT RECIPIENTS. (Abstract #1177)** ♦
Scott W. Cowan, Lisa A. Coscia, Carolyn H. McGrory, Lydia Z. Philips, Michael J. Moritz, Vincent T. Armenti. Philadelphia, PA.

P140 **PROPHYLACTIC ANTI-THYMOCYTE GLOBULIN TREATMENT IS ASSOCIATED WITH COMPLEMENT DEPOSITION IN EARLY CARDIAC TRANSPLANT BIOPSIES. (Abstract #1178)**
William M. Baldwin, III, Lauren P. Armstrong, Milagros D. Samaniego, Salma Rahimi, Kasper K. Edward, Conte V. John, E. Rene Rodriguez, Hruban H. Ralph. Baltimore, MD; Baltimore, MD; Baltimore, MD.

P141 **HEMODYNAMIC EVALUATION OF WORKING HEART TRANSPLANTS IN A CERVICAL HEART-LUNG TRANSPLANTATION MODEL IN RATS. (Abstract #1179)**
Ning Wang, Jangming Lee, Yuan Lin.

Bone Marrow - All Topics III

P142 **RAPID INFECTIOUS DISEASE SCREENING OF CADAVER DONORS FOR HIV, HBV AND HCV USING THE LIGHTCYCLER PCR TECHNOLOGY. (Abstract #1180)**
Robert E. Cirocco, Riena Rodriguez, Les Olsen, George W. Burke, Violet Esquenazi, Andreas Tzakis, Joshua Miller. Miami, FL.

P143 **RISK FACTORS FOR GANCICLOVIR (GCV) RESISTANT CMV IN CMV D+/R- SOLID ORGAN TRANSPLANT RECIPIENTS. (Abstract #1181)**
Robin K. Avery, Belinda Yen-Lieberman, Carlos M. Isada, Robert Schilz, Nell Lurain, Debra Kohn, Janet Maurer, Sherif B. Mossad, Alan J. Taege, Steven D. Mawhorter, Steven M. Gordon, Roy Chemaly, Jennifer Long, David L. Longworth. Cleveland, OH; Cleveland, OH; Cleveland, OH; Chicago, IL; Cleveland, OH.

P144 **POST-TRANSPLANT ALLOSENSITIZATION PATTERNS IN TRANSPLANT. (Abstract #1182)**
R. P. Pelletier, R. M. Ferguson, A. M. VanBuskirk, D. Xia, P. K. Hennessy, P. W. Adams, C. G. Orosz. Columbus, OH.

P145 **CRYPTOCOCCOSIS IN SOLID ORGAN TRANSPLANT RECIPIENTS. (Abstract #1183)**
Regis Vilchez, Ron Shapiro, Kenneth McCurry, Robert Kormos, John Fung, Shimon Kusne.

P146 **INTRALUMINAL DEPOSITION OF ADENOSINE CRYSTALS IN ABDOMINAL ORGANS FOLLOWING PERFUSION WITH UNIVERSITY OF WISCONSIN (UW) SOLUTION. (Abstract #1184)**
Stefan G. Tullius, Alexander Filatencov, Johann Pratschke, Sven Jonas, Hussein Al-Abadi, Thomas Steinmueller, Peter Neuhaus. Berlin, Germany.

P147 **IN VITRO ASSESSMENT OF CALCINEURIN ACTIVITY AS A THERAPEUTIC INDEX OF IMMUNOSUPPRESSION INDUCED BY CICLOSPORINE OR TACROLIMUS IN CHRONIC GVH DISEASE. (Abstract #1185)**
Sylvia Sanquer, Homa Rafi, Philippe Beaune, Mathieu Kuentz, Catherine Cordonnier. Paris, France; Paris, France; Créteil, France.

P148 **NOVEL LYMPHOCYTE HOMEOSTASIS IN PEDIATRIC AND ADULT TRANSPLANT PATIENTS AFTER T CELL DEPLETION. (Abstract #1186)**
Thomas F. Mueller, Guenter Klaus, Scott O. Grebe, Anette Borutta, Katharina M. Mostert, Harald Lange, Barbara Reckzeh. Marburg, Germany; Marburg, Germany; Marburg, Germany; Boston, MA.

P149 **EVALUATION OF TWO-LAYER(UNIVERSITY OF WISCONSIN SOLUTION/ PERFLUOROCHEMICAL)COLD STORAGE METHOD FOR PRESERVATION OF THE CANINE SMALL BOWEL. (Abstract #1187)**
Toshiaki Tsujimura, Yasuyuki Suzuki, Tsuyoshi Takahashi, Isao Yoshida, Yasuki Tanioka, Yasuhiro Fujino, Yonson Ku, Yoshikazu Kuroda. Kobe, Japan.

P150 **EFFECT OF HYPERPARATHYREOIDISM ON CAROTID INTIMA-MEDIA THICKNESS AFTER RENAL TRANSPLANTATION. (Abstract #1188)**
Uta Hillebrand, Ulf M.W. Gerhardt, Karl H. Rahn, Helge Hohage, Barbara M. Suwelack.

P151 **EFFICACY OF FENOLDOPAM MESYLATE IN THE PREVENTION OF RADIOCONTRAST NEPHROPATHY IN DIABETICS WITH CHRONIC RENAL INSUFFICIENCY: A MULTICENTER, RANDOMIZED, PLACEBO-CONTROLLED TRIAL. (Abstract #1189)**
Vandana S. Mathur, James A. Tumlin, Andrew Wang, Patrick T. Murray. San Francisco, CA; Atlanta, GA; Durham, NC; Chicago, IL.

P152 **CAUCASIAN RACE AND PEDIATRIC AGE ARE RISK FACTORS FOR POST TRANSPLANT LYMPHOPROLIFERATIVE DISORDER (PTLD) IN ALL ORGAN SYSTEMS. (Abstract #1190)**
Vikas R. Dharnidharka, William E. Harmon, Brenda Hutson, Richard J. Howard. Gainesville, FL; Boston, MA; Gainesville, FL; Gainesville, FL.

Immunosuppression, Preclinical Studies III

P153 **PRECLINICAL EFFICACY OF A NOVEL CALCINEURIN INHIBITOR: ISA^{TM} 247. (Abstract #1191)**
Mark D. Abel, Launa J. Aspeslet, Derrick G. Freitag, Philip F. Halloran, Norman M. Kneteman, Selvaraj S. Naicker, Daniel J. Trepanier, Robert T. Foster, Randall W. Yatscoff. Edmonton, AB, Canada; Edmonton, AB, Canada.

P154 **ISA^{TM} 247: A NOVEL CALCINEURIN INHIBITOR WITH MINIMAL RENAL TOXICITY. (Abstract #1192)**
Mark D. Abel, Launa J. Aspeslet, Derrick G. Freitag, Philip F. Halloran, Norman T. Kneteman, Selvaraj S. Naicker, Daniel J. Trepanier, Robert T. Foster, Randall W. Yatscoff. Edmonton, AB, Canada; Edmonton, AB, Canada.

P155 **LF 15-0195, A NOVEL IMMUNOSUPPRESSIVE AGENT PREVENTS REJECTION AND INDUCES TOLERANCE IN A MOUSE CARDIAC ALLOGRAFT MODEL. (Abstract #1193)**
Dejun Zhou, Catherine O'Brien, Bertha Garcia, Anthony Jevnikar, Patrick Dutartre, Calvin Stiller, Robert Zhong. London, ON, Canada; London, ON, Canada; London, ON, Canada; London, Canada; Daix, France.

P156 **COMBINED FTY720/CYCLOSPORINE A TREATMENT ENHANCES GRAFT SURVIVAL AND LOWERS THE PERIPHERAL LYMPHOCYTE COUNT: A COMPARISON OF HEART AND SKIN TRANSPLANTATION MODELS IN DA TO LEWIS RATS. (Abstract #1194)**
Zariana Nikolova, Akiko Hof, Yves Baumlin, Robert P. Hof. Basel, Switzerland.

P157 **TOXICITY OF RAPAMYCIN, TACROLIMUS, AND DACLIXUMAB IN THE NON-HUMAN PRIMATE. (Abstract #1195)**
Sean P. Montgomery, Doug Hale, He Xu, Doug K. Tadaki, Justin P. Berning, John Leconte, Allan Kirk. Bethesda, MD.

P158 **MEMBRANE TRANSPORT OF MYCOPHENOLATE MOFETIL AND ITS ACTIVE METABOLITE, MYCOPHENOLIC ACID IN MDCK AND MDR1-MDCK CELL MONOLAYERS. (Abstract #1196)**
Taiji Sawamoto, Teun van Gelder, N. Okamura, Wolfgang Jacobsen, Uwe Christians, Leslie Z. Benet. San Francisco; Palo Alto.

P159 ***IN VIVO* EVALUATION OF THE NOVEL IMMUNOSUPPRESSANT A-285222 IN NON-HUMAN PRIMATES. (Abstract #1197)**
Tudor Bîrsan, Camille Dambrin, Laurie Hook, Janeth C. Villanueva, Kennan C. Marsh, Stevan W. Djuric, Karl W. Mollison, Randall E. Morris. Stanford, CA; Abbott Park, IL.

P160 ***EX VIVO* EVALUATION OF THE IMMUNOSUPPRESSIVE EFFECT OF THE LEFLUNOMIDE DERIVATIVE FK 778 ON WHOLE BLOOD LYMPHOCYTES OF NON-HUMAN PRIMATES. (Abstract #1198)**
Tudor Bîrsan, Camille Dambrin, Jochen Klupp, John D. Patz, Randi Shorthouse, Randall E. Morris. Stanford, CA; Davis, CA.

P161 ***EX VIVO* EVALUATION OF THE IMMUNOSUPPRESSIVE EFFECT OF THE NOVEL CALCINEURIN INHIBITOR ISATX 247 ON WHOLE BLOOD LYMPHOCYTES OF NON-HUMAN PRIMATES. (Abstract #1199)**
Tudor Bîrsan, Camille Dambrin, Janeth C. Villanueva, Randall W. Yatscoff, Randall E. Morris. Stanford, CA; Edmonton, AB, Canada.

P162 **IMMUNOTHERAPY WITH ANTI-CD3 MAB ALONE OR IN COMBINATION WITH ANTI-CD154 MAB AND/OR RAPAMYCIN DOES NOT INDUCE LONG-TERM ISLET ALLOGRAFT SURVIVAL IN DIABETIC NOD MICE. (Abstract #1200)**
Tao Wu, David E.R. Sutherland, Jeffrey A. Bluestone, Bernhard J. Hering, Zhiguang Guo. Minneapolis, MN; San Francisco, CA.

Tolerance III

P163 **NON-SPECIFIC CARDIAC ALLOGRAFT ACCEPTANCE IS INDUCED BY NEONATAL INFUSION OF ALLOGENEIC FETAL LIVER CELLS WITHOUT ALTERING ALLOREACTIVITY TO SKIN GRAFTS AND *IN VITRO* ASSAYS. (Abstract #1201)**
Lori J. West, Kesheng Tao, Lydia Mai. Toronto, ON, Canada.

P164 **ALLOANTIGEN REQUIREMENTS FOR TOLERANCE INDUCTION VIA NOTCH SIGNALLING. (Abstract #1202)**
Ken K. Wong, Gerard F. Hoyne, Jonathan R. Lamb, Margaret J. Dallman. London, United Kingdom; Edinburgh, United Kingdom.

P165 **ANTI-LFA-1 INDUCED ISLET ALLOGRAFT SURVIVAL DOES NOT REQUIRE STAT6. (Abstract #1203)** ♦
Marilyne Coulombe, Mark R. Nicolls, Ronald G. Gill. Denver, CO.

P166 **COSTIMULATORY MOLECULE-EXPRESSING DENDRITIC CELLS POLARIZED BY PROSTAGLANDIN E PROLONG CARDIAC ALLOGRAFT SURVIVAL IN MICE. (Abstract #1204)** ♦
Mark L. Jordan, Susan M. Specht, Patrick P. Luke, Adrian Morelli, Zhiliang Wang, Angus W. Thomson. Pittsburgh, PA; London, ON, Canada; Pittsburgh, PA.

P167 **MHC CLASS II COMPATIBILITY BETWEEN BONE MARROW AND SECONDARY HEART GRAFT DONORS IS ESSENTIAL FOR TOLERANCE BASED ON MIXED CHIMERISM. (Abstract #1205)**
Mark D. Jager, Kai Timrott, Tung-Yu Tsui, Heiko Aselmann, Andrea Deiwick, Michael Neipp, Juergen Klempnauer, Kurt Wonigeit, Hans J. Schlitt. Hannover, Germany; Hong Kong, Hong Kong.

P168 **FTY720 AND CYCLOPHOSPHAMIDE GIVEN PRE-TRANSPLANT WITH DONOR PERIPHERAL BLOOD STEM CELLS INDUCES TOLERANCE TO KIDNEY ALLOGRAFT IN RHESUS MONKEYS. (Abstract #1206)**
Masaaki Kimikawa, Yuichi Sato, Yasuo Ishii, Yojiro Kato, Kei Eguchi, Keiji Terao, Seiichi Suzuki, Satoshi Teraoka. Shinjuku-ku, Tokyo, Japan; Tsukuba, Ibaragi, Japan; Setagaya-ku, Tokyo, Japan.

P169 **THE ROLE OF DONOR ANTIGEN IN MODULATING ALLOIMMUNE RESPONSES IN THE STRINGENT MOUSE SKIN TRANSPLANT MODEL. (Abstract #1207)**
Masayuki Sho, Koji Kishimoto, Nader Najafian, Akira Yamada, Sigrid E. Sandner, Giacomo P. Basadonna, Hugh Auchincloss, David M. Rothstein, Mohamed H. Sayegh. Boston, MA; Boston, MA; New Haven, CT.

P170 **INDUCTION OF T HELPER CELL ANERGY BY ALLOSPECIFIC HUMAN T SUPPRESSOR CELLS. (Abstract #1208)**
Joel LeMaoult, Chih-Chao Chang, Jianda Yuan, Rodica Ciubotariu, Cortesini Raffaello, Nicole Suciu Foca Cortesini. New York, NY; Rome, Italy.

P171 **SPLENOCYTES FROM WILD-TYPE BUT NOT IL-4 KO TOTAL LYMPHOID IRRADIATED MICE INDUCE IMMUNOREDIRECTION OF TCR-TRANSGENIC CD4 CELLS. (Abstract #1209)**
Shawn M. Rigby, Todd Rouse, Tricia Fehr, Elizabeth H. Field. Iowa City, IA; Iowa City, IA.

P172 **SUCCESSFUL TOLERANCE INDUCTION UNDER CD40 LIGATION IN A RODENT SMALL BOWEL TRANSPLANT MODEL: FIRST REPORT OF A STUDY WITH THE NOVEL ANTIBODY AIL F5. (Abstract #1210)**
Thomas M. Fishbein, Christopher Benjamin, Liqing Wang, Adel Tarcsalfalva, Charles M. Miller, Peter Boros. New York, NY; Cambridge, MA.

P173 **LONG-TERM LIMB ALLOGRAFT SURVIVAL USING ANTI-CD40 LIGAND ANTIBODY IN A MURINE LIMB TRANSPLANTATION MODEL. (Abstract #1211)**
Thomas H. Tung, Susan E. Mackinnon, Thalachallour Mohanakumar. St. Louis, MO.

P174 **EVIDENCE FOR TOLERANCE *IN VITRO* AND *IN VIVO* IN SWINE RECEIVING STEM-CELL TRANSPLANTS WITHOUT MYELOSUPPRESSIVE CONDITIONING. (Abstract #1212)**
Zachary L. Gleit, Yasushi Fuchimoto, Christene Huang, Elizabeth Melendy, David H. Sachs. Boston, MA.

Acute/Chronic Rejection III

P175 **T-CELL DEPENDENT AND T-CELL INDEPENDENT IMMUNE MECHANISMS OF CHRONIC RENAL ALLOGRAFT REJECTION IN A NUDE RAT MODEL. (Abstract #1213)**
Martina Koch, Corinna Doege, Annice Heratizadeh, Peter Sotony, Michael Mengel, Juergen Strehlau, Bjoern Nashan. Hannover, Germany; Budapest, Hungary; Hannover, Germany; Hannover, Germany.

P176 **ADENOVIRUS MEDIATED BCL-2 TRANSFECTION INHIBITS APOPTOSIS IN A RAT HETEROTOPIC HEART TRANSPLANT MODEL. (Abstract #1214)**
Murray H. Kown, Christina L. Jahncke, Douglas N. Miniati, Murata Seiichiro, Francis G. Blankenberg, H. W. Strauss, Grant Hoyt, Robert C. Robbins. Stanford, CA; Stanford, CA; Stanford, CA.

P177 **COLD ISCHEMIA AUGMENTS ALLOGENEIC MEDIATED INJURY IN RAT KIDNEY TRANSPLANTS. (Abstract #1215)**
Ewout A. Kouwenhoven, Ron W.F. de Bruin, Ingeborg M. Bajema, Richard L. Marquet, Jan N.M. IJzermans. Rotterdam, The Netherlands.

P178 **HLA-A2 TRANSGENIC C57BL/6 TRACHEA TRANSPLANTATION INTO SYNGENEIC C57BL/6 MICE RESULTS IN OBLITERATIVE AIRWAY DISEASE: ROLE FOR CD4 AND CD8 CELLS. (Abstract #1216)** ♦
Toru Higuchi, Andres Jaramillo, Zahid Kaleem, T. Mohanakumar. St. Louis, MO.

P179 **EARLY APPLICATION OF MATRIX METALLOPROTEINASE INHIBITOR AMELIORATES CHRONIC ALLOGRAFT NEPHROPATHY IN RATS. (Abstract #1217)**
Erwei Song, Jens Lutz, Yousheng Yao, Balazs Antus, Shanying Liu, Peter Hamar, Uwe Heemann. Essen, Germany.

P180 **SEQUENTIAL ACTIVATION PATTERNS OF MACROPHAGES IN CHRONIC ALLOGRAFT NEPHROPATHY. (Abstract #1218)**
Erwei Song, Balazs Antus, Yousheng Yao, Jens Lutz, Uwe Heemann. Essen, Germany.

P181 **APOPTOSIS IN ACUTE RENAL ALLOGRAFT REJECTION IS MEDIATED BY THE AT2-RECEPTOR (Abstract #1219)**
Jens Lutz, Kirsten Risch, Balazs Antus, Manfred Lehmann, Uwe Heemann. Essen, Germany; Rostock, Germany.

P182 **INHIBITION OF ANGIOGENESIS-RELATED ENDOTHELIAL ACTIVITY BY LEFLUNOMIDE. (Abstract #1220)**
W. James Waldman, Alice Bickerstaff, Gayle Gordillo, Kathleen Orosz, Deborah A. Knight, Charles G. Orosz. Columbus, OH; Columbus, OH.

P183 **PERMANENT RECIPIENT CD8+ T CELLS DEPLETION DOES NOT ABROGATE THE DEVELOPMENT OF CARDIAC ALLOGRAFT VASCULOPATHY. (Abstract #1221)**
Wilson Y. Szeto, Alyssa M. Krasinskas, Daniel Kreisel, Alexander S. Krupnick, Sicco H. Popma, Bruce R. Rosengard. Philadelphia, PA; Philadelphia, PA.

P184 **CASPASE ACTIVATION AND APOPTOSIS OCCURS IN THE ABSENCE OR PRESENCE OF $CD8^+$ CTL. (Abstract #1222)**
Yasuhiro Ogura, Olivia M. Martinez, Carlos O. Esquivel, Sheri M. Krams. Stanford, CA.

Allorecognition, Antigen Presentation, Co-Stimulation and Other III

P185 **ALLOGENEIC CHIMERISM IN NONOBESE DIABETIC MICE REVERSES THE ABNORMAL DEVELOPMENTAL PHENOTYPE OF MYELOID LINEAGE BONE MARROW CELLS. (Abstract #1223)**
Paula M. Chilton, Hong Xu, Yiming Huang, Haval Shirwan, Suzanne T. Ildstad. Louisville, KY.

P186 **IN VIVO DETECTION OF ACUTE REJECTION IN RAT RENAL ALLOGRAFT BY MR IMAGING WITH ULTRASMALL SUPERPARAMAGNETIC IRON OXIDE. (Abstract #1224)**
Qing Ye, Dewen Yang, Mangay Williams, Donald S. Williams, Charchai Pluempitiwiriyawe, Jose' M.F. Moura, Chien Ho.

P187 **OSTEOPROTEGERIN INHIBITS CD3/CD28 ACTIVATED T CELLS BUT NOT DENDRITIC CELL-MEDIATED COSTIMULATION. (Abstract #1225)**
Ravi Krishnan, Svjetlana Kireta, Julie Johnston, Graeme Russ.

P188 **DESTRUCTIVE INFILTRATION OF HUMAN ISLET ALLOGRAFTS IN HU-PBL-NOD-*SCID* MICE. (Abstract #1226)**
Scott J. Banuelos, Leonard D. Shultz, Robert C. Harland, Bonnie Lyons, Aldo A. Rossini, Dale L. Greiner, Michael C. Appel. Worcester, MA; Bar Harbor, ME; Worcester, MA.

P189 **NEOINTESTINE ENGINEERING BY TRANSPLANTATION OF ENTEROCYTES ON A BIODEGRADABLE POLYMER. (Abstract #1227)**
W. De Faria, S. Salgar, P. Ruiz, R. Vianna, N. Wasserberg, C. Gandia, S. Santiago, C. Ricordi, J. Miller, A. Tzakis. FL.

P190 **HUMAN T CELLS EXPRESS CD86 AND ACQUIRE APC FUNCTION AFTER STIMULATION WITH HUMAN OR PORCINE APC. (Abstract #1228)**
Sicco H. Popma, Wilson Y. Szeto, Daniel Kreisel, Alexander S. Krupnick, Jonni S. Moore, Bruce R. Rosengard. Philadelphia, PA.

P191 **DIVERSITY OF SELF VS. NON-SELF HLA REPERTOIRE INFLUENCES GRAFT ACCEPTABILITY. (Abstract #1229)**
Steven K. Takemoto, Rene Duquesnoy, Elaine F. Reed, J. Michael Cecka.

P192 **PREVENTION OF ACUTE RAT LUNG ALLOGRAFT REJECTION AND TOLERANCE INDUCTION BY CTLA4IG. (Abstract #1230)**
Takeshi Shiraishi, Yohichi Yasunami, Megumi Takehara, Toshimitsu Uede, Katsunobu Kawahara, Takayuki Shirakusa. Fukuoka City, Fukuoka, Japan; Fukuoka City, Fukuoka, Japan; Sapporo, Hokkaido, Japan.

P193 **NON-PARENCHYMAL CELLS IN LIVER SUPPRESS THE FUNCTION OF DENDRITIC CELLS. (Abstract #1231)**
Wei-Chen Lee, Yang-Jen Chiang, Chen-Rong Lia, Pei-Fang Huang, Hui-Chuan Wang, Long-Bin Jeng, Miin-Fu Chen, Shiquan Qian, Lina Lu. Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Taipei, Taiwan; Pittsburgh, PA, United States; Pittsburgh, PA.

P194 **HIERARCHY OF CD2-CD48 AND 2B4-CD48 RECEPTOR-LIGAND INTERACTION IN PROLONGATION OF ALLOGRAFT SURVIVAL. (Abstract #1232)**
Yalai Bai, Yinong Wang, Lihui Qin, Jonathan S. Bromberg. New York, NY.

P195 **MECHANISM OF ALLOGRAFT ACCEPTANCE IN SENSITIZED CD40L-DEFICIENT RECIPIENTS. (Abstract #1233)**
Yuan Zhai, Bibo Ke, Xiu-Da Shen, Feng Gao, Mohamed H. Sayegh, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Boston, MA.

Lymphocyte Activation, Lymphocyte-Down-Regulation, Chemokines/Adhesion Molecules and Cytokines III

P196 **CORRELATION OF CYTOKINE GENE POLYMORPHIC INHERITANCE AND IN VITRO CYTOKINE PRODUCTION IN ANTI-CD3/CD28 STIMULATED PERIPHERAL BLOOD LYMPHOCYTES. (Abstract #1234)**
Steven C. Hoffmann, Eran M. Stanley, E. Darrin Cox, Barbara S. DiMercurio, Dee E. Koziol, David M. Harlan, Allan D. Kirk, Patrick J. Blair. Bethesda, MD; Washington, DC.

P197 **ETHNICITY GREATLY INFLUENCES THE DISTRIBUTION OF CYTOKINE GENE POLYMORPHISMS. (Abstract #1235)**
Eran M. Stanley, Steven C. Hoffmann, E. Darrin Cox, Barbara S. DiMercurio, David M. Harlan, Allan D. Kirk, Patrick J. Blair.

P198 **DIFFERENTIAL REGULATION OF ALLOGENEIC T CELL PROLIFERATION AND APOPTOSIS BY IMMATURE HEPATIC DENDRITIC CELL SUBSETS. (Abstract #1236)**
Peta J. O'Connell, Alison J. Logar, Alem Truneh, Angus W. Thomson. Pittsburgh, PA; King of Prussia, PA; Pittsburgh, PA.

P199 **BLOCKADE OF RHO-KINASE INHIBITS T CELLS ACTIVATION FOLLOWING TCR-CD3 LIGATION AND ALLO-IMMUNE STIMULATION. (Abstract #1237)**
Pierre-Louis F. Tharaux, Thomas M. Coffman. Durham, NC.

P200 **DELAYED CELLULAR INFILTRATION AND ACUTE REJECTION OF CARDIAC ALLOGRAFTS FROM ICAM-1-/- DONORS. (Abstract #1238)**
Qiwei Zhang, Masayoshi Miura, Robert L. Fairchild. Cleveland, OH; Cleveland, OH.

P201 **ANTIBODY AGAINST β7 INTEGRINS, BUT NOT β7-DEFICIENCY ATTENUATES INTESTINAL ALLOGRAFT REJECTION IN MICE. (Abstract #1239)**
Richard Kellersmann, Robert Zhong, Bertha Garcia, David Grant, Werner Mueller, Norbert Wagner, Anne Kellersmann, Karin Ulrichs, Arnulf Thiede, Andrew Lazarovits. Wuerzburg, Germany; London, Canada; Toronto, Canada; Cologne, Germany.

P202 **CYTOKINE TRANSCRIPTS UPREGULATED IMMEADIATELY FOLLOWING HUMAN RENAL ALLOGRAFT REPERFUSION. (Abstract #1240)**
Robert L. Kampen, Steven C. Hoffmann, Shashi Amur, Patrick J. Blair, Barbara S. DiMercurio, Douglas K. Tadaki, David M. Harlan, Allan D. Kirk. Bethesda, MD; Foster City, CA.

P203 **NOTCH PROVIDES A SURVIVAL SIGNAL IN ACTIVATED T CELLS. (Abstract #1241)**
Scott H. Adler, Warren S. Pear, Laurence A. Turka. Philadelphia, PA; Philadelphia, PA.

P204 **ALLO-ANTIGENS STIMULATION IN THE PRESENCE OF IL-10 PROMOTES REGULATORY CELL DEVELOPMENT AND PROLONG ALLOGRAFT SURVIVAL. (Abstract #1242)**
Wei Li, Lina Lu, Satwant K. Narula, John J. Fung, Shiguang Qian. Pittsburgh, PA; Kenilworth, NJ.

P205 **SELECTIN INHIBITOR BIMOSIAMOSE (TBC 1269) ALONE OR IN COMBINATION WITH OTHER AGENTS PROLONGS KIDNEY ALLOGRAFT SURVIVAL. (Abstract #1243)**
Mou-er Wang, Stanislaw M. Stepkowski, Kurt L. Berens, Peter Vanderslice, Richard A. Dixon, Barry D. Kahan. Houston, TX; Houston, TX.

P206 **A NOVEL TUMOR NECROSIS FACTOR-a SUPPRESSANT, ONO-SM362, PREVENTS LIVER FAILURE AND PROMOTES LIVER REGENERATION AFTER MASSIVE HEPATECTOMY. (Abstract #1244)**
Toshiro Ogata, Maeng Bong Jin, Masao Sunahara, Naoyuki Takada, Tsuyoshi Shimamura, Hiroyuki Furukawa, Michiaki Matsushita, Satoshi Watanabe, Satoru Todo. Sapporo, Hokkaido, Japan; Sapporo, Hokkaido, Japan.

P207 **AN INHIBITOR OF GLYCOSAMINOGLYCAN-CYTOKINE INTERACTION DELAYS REJECTION OF VASCULARIZED SKIN ALLOGRAFTS. (Abstract #1245)**
Vijay S. Gorantla, Gustavo Perez-Abadia, Xiaoping Ren, Haldun I. Orhun, Edwin E. Quan, Claudio Maldonado, Mukunda Ray, John H. Barker, Rafael Fernandez-Botran. Louisville, KY; Louisville, KY.

P208 **THE ROLE OF NATURALLY OCCURRING HUMAN $CD4^+CD25^+$ SUPPRESSOR CELLS IN THE REGULATION OF ALLORESPONSES IN MAN. (Abstract #1246)**
Wan-Fai Ng, Phillip Duggan, Niels Olsen-Saraiva-Camara, Guseppe Matarese, Giovanna Lambardi, A. David Edwards, Robert I. Lechler. London, United Kingdom; London, United Kingdom.

Genetic Modulation, Islet/Cell Transplantation and Bone Marrow/GVH III

P209 **PREVENTION OF AUTOIMMUNE DESTRUCTION OF ISLET TRANSPLANTS IN NONOBESE DIABETIC (NOD) MICE. (Abstract #1247) ♦**
Maria Koulmanda, Andi S. Qipo, Neal R. Smith, Hugh Auchincloss, Jr.

P210 **IL-1R KNOCK-OUT ISLETS RESIST CYTOKINE-INDUCED APOPTOSIS AND DEMONSTRATE ACCELERATED POST-TRANSPLANT GRAFT FUNCTION. (Abstract #1248)**
Marshall Baker, Jan Chen, Tracy Carr, Dixon B. Kaufman. Chicago, IL.

P211 **CASPASE INHIBITION IN MURINE ISLETS BLOCKS NITRIC OXIDE DEPENDENT CYTOKINE-INDUCED APOPTOSIS WITHOUT PRESERVING ISLET FUNCTION. (Abstract #1249)**
Marshall S. Baker, Xiaojuan Chen, Xiao-Chun Cao, Dixon B. Kaufman. Chicago, IL.

P212 **THE COMBINED USE OF TRANSGENIC (MIEP-*lacZ*) AND KNOCK-OUT (TNFRt1-/-) MICE PROVIDES A NOVEL STRATEGY FOR THE MECHANISTIC STUDY OF CMV IMMEDIATE EARLY (IE) GENE REGULATION. (Abstract #1250)**
Piyush Golia, Zhang Zheng, Soo Jung Kim, Isabelle DePlaen, Gail Thomas, Mary Hummel, Dixon B. Kaufman, Jonathan P. Fryer, Joseph R. Leventhal, Frank P. Stuart, Michael M. Abecassis. Chicago, IL.

P213 **EVALUATION OF HUMAN PANCREAS PRESERVED BY THE TWO-LAYER (UW SOLUTION/ PERFLUOROCARBON) METHOD PRIOR TO ISLET ISOLATION. (Abstract #1251)**
Shinichi Matsumoto, Jessica Chawla, JoAnna Reems, Yoshikazu Kuroda, R. Brian Stevens. Seattle, WA; Kobe, Hyogo, Japan; Seattle, WA.

P214 **A REAPPRAISAL OF THE PASSENGER LEUKOCYTE MODEL OF TISSUE IMMUNOGENICITY. (Abstract #1252)**
Joshua Beilke, Marilyne Coulombe, Amy Bolwerk, Ronald G. Gill. Denver, CO.

P215 **IMMUNOMODULATION BY AAV-CTLA4Ig GENE THERAPY: GENE EXPRESSION IS CONTROLLED BY THE TARGET ORGAN. (Abstract #1253)**
Wei Li, Jiuang Li, Xiao Xiao, Zongyou Chen, Lina Lu, John J. Fung, Shiguang Qian. Pittsburgh, PA.

P216 **ALLOCHIMERIC PROTEIN-INDUCED TOLERANCE IS MEDIATED BY POTENT REGULATORY T HELPER 2 CELLS. (Abstract #1254) ♦**
Barton Trawick, Robert Kirken, Min Wang, Neelam Tejpal, Mou-Er Wang, Barry D. Kahan, Stanislaw M. Stepkowski. Houston, TX; Houston, TX.

P217 **LOW-DOSE FK506 CAN ENHANCE THE EFFECTS OF RECOMBINANT ADENO-ASSOCIATED VIRUS MEDIATED hCTLA4Ig GENE TRANSFER. (Abstract #1255)**
Zhenfan Yang, Xiaobing Wu, Tung-Yu Tsui, Yunde Hou, John. M. Luk, Sheung-Tat Fan. Hong Kong; Beijing, China.

P218 **IMPROVEMENT IN VASCULAR ENDOTHELIAL CELL-TARGETED GENE TRANSFER BY USING AN ACTIN BASED HYBRID PROMOTER FOR TRANSPLANT VASCULAR APPLICATIONS. (Abstract #1256)**
Yoshio Nitta, Christine Halbert, A. Dusty Miller, Akiko Iwata, Jun-Ichi Miyazaki, Lakshmi K. Gaur, Margaret D. Allen. Seattle, WA; Osaka, Japan.

P219 **DONOR CELL PRETREATMENT ENHANCES THE INDUCTION OF ALLOGENEIC MIXED HEMATOPOIETIC CHIMERISM IN C57BL/6 AND NOD MICE BY USING NONIRRADIATIVE AND NONMYELOABLATIVE APPROACHES. (Abstract #1257)**
Tao Wu, Hakan Sozen, Ping Lan, Neal Heuss, Hannes Kalscheuer, David E.R. Sutherland, Bruce R. Blazar, Bernhard J. Hering, Zhiguang Guo. Minneapolis, MN.

Tissue Injury, Preservation III

P220 **MATRIX METALLOPROTEINASES INHIBITION DECREASES LIVER ISCHEMIA/REPERFUSION INJURY IN RATS. (Abstract #1258)**
Raffaele Cursio, Bernard Mari, Marie Christine Saint-Paul, Krystel Louis, Vincent Dive, Patrick Auberger, Jean Gugenheim. Nice, France; Nice, France; Nice, France; Saclay, France.

P221 **DEFINING THE ROLE OF A TAILORED LUMINAL SOLUTION FOR EXTENDED SMALL BOWEL PRESERVATION. (Abstract #1259)**
Yasuhiro Fujimoto, David W. Olson, David L. Bigam, Karen L. Madsen, Janice Zeng, Laurence D. Jewell, Norman M. Kneteman, Thomas A. Churchill. Edmonton, AB, Canada; Edmonton, AB, Canada; Edmonton, AB, Canada; Edmonton, AB, Canada.

P222 **THE IMPORTANCE OF OSMOTIC AND ONCOTIC SUPPORT IN SMALL BOWEL PRESERVATION: A MORPHOLOGIC, METABOLIC, AND FUNCTIONAL STUDY. (Abstract #1260)**
David W. Olson, Brian Stewart, Michelle Carle, Karen Madsen, Jay Zhu, David Bigam, Norman Kneteman, Thomas A. Churchill. Edmonton, AB, Canada; Edmonton, AB, Canada; Edmonton, AB, Canada.

P223 **COMPARING THE EFFICACY OF UNIVERSITY OF WISCONSIN (UW) AND CELSIOR (Cls) SOLUTIONS FOR HEART PRESERVATION WITH AND WITHOUT CYCLOSPORINE A (CsA). (Abstract #1261)**
Thomas N. Masters, Alexander A. Fokin, Lieven Pool, Jutta Schaper, Francis Robicsek.

P224 **PROTECTIVE EFFECT OF LOW DOSE CYCLOSPORINE-A ON ISCHEMIC-REPERFUSED KIDNEY. (Abstract #1262)**
Tong-yu Zhu, Kathy K.W. Au-Yeung, Yaw L. Siow, Karmin O. Shanghai, China; Hong Kong, SAR, China.

P225 **HEPATOCYTE GROWTH FACTOR (HGF) GENE THERAPY OF KIDNEY FIBROSIS USING ELECTROPORATION INTO SKELETAL MUSCLE IN VIVO. (Abstract #1263)**
Toshiyuki Tanaka, Shiro Takahara, Naotsugu Ichimaru, Yoshitaka Isaka, Toshiki Moriyama, Haruhito Azuma, Kiyohide Toki, Koji Yazawa, Jing-Ding Wang, Sompol Permpongkosol, Enyu Imai, Akihiko Okuyama. Suita, Osaka, Japan; Suita, Osaka, Japan; Takatsuki, Osaka, Japan.

P226 **ADRENOMEDULLIN, A NOVEL VASODILATIVE PEPTIDE, ATTENUATES ISCHEMIA REPERFUSION INJURY OF THE LIVER. (Abstract #1264)**
Tsunenori Sakurai, Maeng Bong Jin, Keisa Takeda, Tsuyoshi Shimamura, Hiroyuki Furukawa, Miri Fujita, Satoru Todo. Sapporo, Hokkaido, Japan; Sapporo, Hokkaido, Japan.

P227 **HYPOXIA-INDUCIBLE FACTOR-1 (HIF-1) IS UP-REGULATED BY A COMBINATION OF COLD AND WARM ISCHEMIA IN RAT CARDIAC ISOGRAFTS. (Abstract #1265)**
Walter Mark, Deborah S. Stroka, Desley Neil, Stefan Schneeberger, Ruediger Seiler, Tobias Burkhardt, Raimund Margreiter, Daniel Candinas. Birmingham, United Kingdom; Innsbruck, Austria.

Xenotransplantation III

P228 **INDUCTION OF HEME OXYGENASE-1 WITH COBALT PROTOPORPHYRIN FAILS TO PROLONG HAMSTER-TO-RAT KIDNEY XENOGRAFT SURVIVAL. (Abstract #1266)**
Patrick G. Dean, Dean Y. Kim, Dora I. Ninova, Karl A. Nath, Mark D. Stegall. Rochester, MN; Rochester, MN.

P229 **IN VIVO CELLULAR AND MOLECULAR REQUIREMENTS FOR CELL-MEDIATED CARDIAC XENOGRAFT REJECTION. (Abstract #1267)**
Masayuki Obatake, Michelle Kushida, Jennifer Zhang, Peter C.W. Kim. Toronto, ON, Canada.

P230 **SUSTAINED REDUCTION OF ANTI-αGAL AND ANTI-PIG HEMOLYTIC ANTIBODIES IN VIVO WITH THE POLYMER GAS914. (Abstract #1268) ♦**
Rafael Manez, Alberto Centeno, Eduardo Lopez-Pelaez, Nieves Domenech, Rudolph Duthaler, Andreas Katopodis. La Coruna, Spain; Basel, Switzerland.

P231 **THE IDEAL HUMAN ABO BLOOD GROUPS FOR PORCINE-HUMAN XENOTRANSPLANTATION. (Abstract #1269)**
Rizwan A. Manji, Jacqueline S. Manji, Arvind Koshal, Greg S. Korbutt, Raymond V. Rajotte. Edmonton, AB, Canada.

P232 **CD4+ T CELLS ARE SUFFICIENT FOR REJECTION OF NEONATAL PORCINE ISLET XENOGRAFTS AND ARE DEPENDENT ON HOST CLASS II MHC MOLECULES. (Abstract #1270)**
Gina R. Rayat, Zachary Johnson, Gregory S. Korbutt, Ray V. Rajotte, Ronald G. Gill. Denver, CO; Edmonton, AB, Canada.

P233 **THE TCR REPERTOIRE OF PROLIFERATING T CELLS IS RESTRICTED BY IMMUNODOMINANT ANTIGENS IN THE DIRECT HUMAN ANTI-PIG PATHWAY. (Abstract #1271)**
Sicco H. Popma, Wilson Y. Szeto, Daniel Kreisel, Alexander S. Krupnick, Alyssa M. Krasinskas, Jonni S. Moore, Bruce R. Rosengard. Philadelphia, PA; Philadelphia, PA.

P234 **HIGH LEVELS OF NATURAL ANTI-PIG HEMOLYTIC ANTIBODIES ARE ASSOCIATED WITH HYPERACUTE REJECTION OF hDAF TRANSGENIC PIG TO CYNOMOLGUS MONKEY CARDIAC AND RENAL XENOGRAFTS. (Abstract #1272)**
Tuan Lam, Bernard Hausen, Laurie Hook, Katrin Boeke, Camille Dambrin, John Higgins, Gerry Berry, Hugh Davies, Emanuele Cozzi, Randall Morris. Stanford, CA; Stanford, CA; Cambridge, United Kingdom.

P235 **THE ROLE OF DN REGULATORY T CELLS IN LONG-TERM CARDIAC XENOGEAFT SURVIVAL INDUCED BY PRETRANSPLANT DONOR SPECIFIC TRANSFUSION AND A SHORT COURSE OF ANTI-CD4 MONOCLONAL ANTIBODY. (Abstract #1273)**
Wenhao Chen, Megan S. Ford, Li Zhang. Toronto, ON, Canada.

P236 **BASILIXIMAB AND RITUXIMAB INCREASE SURVIVAL IN PIG-TO-BABOON KIDNEY XENOTRANSPLANTATION. (Abstract #1274)**
Y. T. Becker, D. Hullett, A. Friedl, S. O'Herrin, A. L. Usborne, H. W. Sollinger. Madison, WI; Madison, WI.

P237 **DURATION OF CARDIAC XENOGRAFT SURVIVAL IN BONE MARROW CHIMERAS DEPENDS UPON THE TIMING OF TRANSPLANTATION. (Abstract #1275)**
Yuru Meng, Muhammad M. Mohiuddin, Yong Qin, Verdi J. DiSesa.

P238 **METABOLIC ACTIVITY VERSES DEATH IN THE PATHOGENESIS OF ACUTE VASCULAR REJECTION. (Abstract #1276) ♦**
Zoie E. Holzknecht, Karisha L. Kuypers, Josie M. Williams, Jeffrey L. Platt. Rochester, MN.

Notes

TRANSPLANT 2001
Joint American Transplant Meeting
Day-at-a-Glance, Wednesday, May 16, 2001

6:30AM - 7:50 AM	Concurrent Sunrise Symposia
Page 130	**Sunrise Symposium I: Xenotransplantation: Into the Clinic** *Sheraton Chicago Ballroom 4-7, Sheraton*
Page 130	**Sunrise Symposium II: Special Issues for Female Transplant Pratients** *Sheraton Ballroom 1-3, Sheraton*
Page 130	**Sunrise Symposium III: Cardiovascular Disease after Transplantation** *Chicago Ballroom 8-10, Sheraton*

8:00 AM - 9:00 AM	Plenary Sessions
Page 130	**Basic Science** *Sheraton Ballroom 1-3, Sheraton*
Page 130	**Clinical Science** *Sheraton Chicago Ballroom 4-7, Sheraton*

9:00 AM	Break

9:30 AM - 10:30 AM	Joint Session
Page 131	**What's Hot, What's New: Clinical** **What's Hot, What's New: Basic** *Sheraton Chicago Ballroom 4-7, Sheraton*

11:00 AM - 12:30 AM	Concurrent Sessions
Page 131	**Concurrent Session 61: Mechanisms of Ischemia/Reperfusion Injury II** *Chicago Ballroom 10, Sheraton*
Page 131	**Concurrent Session 62: Liver Transplantation: General** *Chicago Ballroom 6/7, Sheraton*
Page 132	**Concurrent Session 63: Basic Science: Tolerance III** *Chicago Ballroom 8, Sheraton*
Page 132	**Concurrent Session 64: Immunosuppression and Immune Responses** *Chicago Ballroom 9, Sheraton*
Page 133	**Concurrent Session 65: Donor Management and Outcomes** *Sheraton Ballroom 1-3, Sheraton*
Page 133	**Concurrent Session 66: New Maintenance Immunosuppressive Agents** *Sheraton Ballroom 4/5, Sheraton*

Adjorn

Wednesday, May 16, 2001

Concurrent Sunrise Symposia
6:30 AM - 7:50 AM

Sunrise Symposium I: Xenotransplantation: Into the Clinic

Sheraton Chicago Ballroom 4-7, Sheraton
Chair: Ira Fox

6:30 AM Extracorporeal perfusion for hepatic failure - Whole liver vs. transgenic perfusion
Marlon Levy

6:50 AM Xenogeneic hepatocyte transplantation
Ira Fox

7:10 AM Clinical trials of xenogeneic neural cell transplants
Albert Edge

7:30 AM New discoveries about PERV and PERV transmission
Clive Patience

Sunrise Symposium II: Special Issues for Female Tranplant Patients

Sheraton Ballroom 1-3, Sheraton
Chairs: Jill Lindberg and Dianne McKay

6:30 AM Pregnancy, fertility, and contraception
Thomas Easterling

6:50 AM Hormone replacement and bone disease
Jill Lindberg

7:10 AM Hormone replacement and heart disease
Nanette Wenger

7:30 AM Malignancies in women after transplantation
Joseph F. Buell

Sunrise Symposium III: Cardiovascular Disease after Transplantation

Chicago Ballroom 8-10, Sheraton
Chair: John Curtis

6:30 AM Diabetes as a risk factor
Robert Rosenson

6:55 AM Management of post-transplant hypertension
John Curtis

7:20 AM Management of post-transplant hyperlipidemia
Steven Katznelson

Plenary Session: Basic
8:00 AM - 9:30 AM

Sheraton Ballroom 1-3, Sheraton
Chair: Anthony Jevnikar and Peter Stock

8:00 AM **ABROGATION OF ACUTE REJECTION IN THE ABSENCE OF LOCALLY SYNTHESISED COMPLEMENT C3. (Abstract #1277)**
Julian R. Pratt, Shamim A. Basheer, Steven H. Sacks. London, United Kingdom.

8:15 AM **THE IDENTIFICATION OF A G-PROTEIN-COUPLED RECEPTOR EDG-6 AS A TARGET OF FTY720, A NOVEL TRANSPLANTATION DRUG. (Abstract #1278)** *Young Investigator Award*
Shizhong Chen, Gabriela E. Garcia, Rong Liao, Volker Brinkmann, Lili Feng. La Jolla, CA; Houston, TX; Basel, Switzerland.

8:30 AM **ADENOVIRALLY OVER-EXPRESSED REDOX FACTOR-1(REF-1) PROTECTS AGAINST POST-ISCHEMIC LIVER INJURY BY REDUCING OXIDATIVE STRESS AND NF-kB DNA BINDING ACTIVITY. (Abstract #1279)**
International Young Investigator Award
Michitaka Ozaki, Kaikobad Irani, Seiichi Suzuki. Setagaya, Tokyo, Japan; Baltimore, MD.

8:45 AM **CD40 AND ANGIOGENESIS: A LINK BETWEEN ALLOIMMUNE RESPONSES AND NON-IMMUNE MECHANISMS OF ALLOGRAFT REJECTION? (Abstract #1280)** *Young Investigator Award*
Marlies E.J. Reinders, Masayuki Sho, Michael Melter, Christopher Geehan, Cees van Kooten, David M. Briscoe. Boston, MA; Boston, MA; Leiden, The Netherlands.

9:00 AM **BLOCKADE OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) INHIBITS THE RECRUITMENT OF INFLAMMATORY CELLS AND THE REJECTION OF HUMAN SKIN ALLOGRAFTS. (Abstract #1281)**
Marlies E.J. Reinders, Masayuki Sho, Michael Melter, Christopher Geehan, David M. Briscoe. Boston, MA.

9:15 AM **THE CD40 LIGAND AND CD28 T CELL COSTIMULATION PATHWAYS ARE REQUIRED FOR NON-ANTIGENIC WARM ISCHEMIA/REPERFUSION INJURY IN MOUSE LIVER MODEL. (Abstract #1282)**
Young Investigator Award
Xiu-Da Shen, Bibo Ke, Feng Gao, Judy Melinek, Farin Amersi, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA; Los Angeles, CA.

Plenary Session: Clinical
8:00 AM - 9:30 AM

Sheraton Chicago Ballroom 4-7, Sheraton
Chairs: John Roberts and Sue McDiarmid

8:00 AM **EVIDENCE FOR GENETIC SUSCEPTIBILITY TOWARDS DEVELOPMENT OF POST TRANSPLANT LYMPHOPROLIFERATIVE DISORDER (PTLD) IN SOLID ORGAN TRANSPLANT PATIENTS. (Abstract #1283)** *International Young Investigator Award*
Nina Babel, Athanasios Vergopoulos, Ian Hutchinson, Conny Hoeflich, Stephen Oertel, Hanno Riess, Hans Dieter Volk, Petra Reinke. Berlin, Germany; United Kingdom.

8:15 AM **DEVELOPMENT OF A PEDIATRIC END STAGE LIVER DISEASE (PELD) SCORE. (Abstract #1284)**
Sue V. McDiarmid, Ravinder Anand, SPLIT Research Group. Los Angeles, CA; Potomac, MA.

8:30 AM **MAYO END STAGE LIVER DISEASE MODEL (MELD) SCORE PREDICTS LIVER TRANSPLANT WAITING LIST MORTALITY: IMPLICATIONS FOR LIVER ALLOCATION POLICY. (Abstract #1285)**
Russell H. Wiesner, Eric B. Edwards, Patrick S. Kamath, Woong R. Kim, Walter K. Kremers, Richard B. Freeman, Michael Malinchoc, Todd K. Howard, John R. Lake. Rochester, MN; Richmond, VA; Boston, MA; St. Louis, MO; Minneapolis, MN.

8:45 AM **INCREASED B7 COSTIMULATORY MOLECULE EXPRESSION IN AFRICAN-AMERICAN KIDNEY ALLOGRAFT RECIPIENTS: ASSOCIATION WITH CYTOKINE POLYMORPHISMS. (Abstract #1286)**
Anne Hutchings, Lisa Guay-Woodford, Judith M. Thomas, Wendy M. Purcell, Ian V. Hutchinson, Mark R. Benfield. Birmingham, AL; Birmingham, AL; Birmingham, AL; Bristol, United Kingdom; Manchester, United Kingdom.

9:00 AM **SPLIT-LIVER TRANSPLANTATION IN THE UNITED STATES: CREATION OF A NATIONAL REGISTRY AND PRELIMINARY OUTCOMES. (Abstract #1287)**
Jean C. Emond, Nancy L. Ascher, Ronald W. Busuttil, American Society of Transplant Surgeons.

9:15 AM **Break**

Joint Session

9:30 AM - 10:30 AM

Chairs: Marc Lorber and Laurence Turka

What's Hot, What's New: Clinical
Donald Hricik
What's Hot, What's New: Basic
Jonathon Bromberg

10:30 **Break**

Concurrent Session 61: Mechanisms of Ischemia/ Reperfusion Injury II

11:00 AM - 12:30 PM

Chicago Ballroom 10, Sheraton
Chairs: Anita Coito and Douglas Farmer

11:00 AM **A NON-TRANSGENIC APPROACH TO TISSUE ENGINEERING FOR THE MODULATION OF ISCHAEMIA /REPERFUSION INJURY IN KIDNEY TRANSPLANTATION. (Abstract #1288)**
Julian R. Pratt, Jun Dong, Miriam E. Jones, Richard A.G. Smith, Steven H. Sacks. London, United Kingdom; Royston, United Kingdom.

11:10 AM **ENDOTHELIN RECEPTOR BLOCKADE MITIGATES THE ADVERSE EFFECT OF PRE-RETRIEVAL WARM ISCHEMIA ON GRAFT FUNCTION TWO MONTHS FOLLOWING RENAL TRANSPLANTATION IN RATS. (Abstract #1289)**
Sharon R. Inman, Wanda K. Plott, Ray A. Pomilee, Jodi A. Antonelli, Richard M. Lewis. Austin, TX; Maywood, IL.

11:20 AM **BRAIN DEATH AS A RISK FACTOR IN TRANSPLANTATION-INFLUENCE OF DONOR PRETREATMENT ON ORGAN FUNCTION AFTER EXPERIMENTAL KIDNEY TRANSPLANTATION. (Abstract #1290)**
Johann Pratschke, Grzegorz Kofla, Markus J. Wilhelm, Athanasios Vergopoulos, Igor Laskowski, Gray Shaw, Stefan G. Tullius, Hans-Dieter Volk, Peter Neuhaus, Nicholas L. Tilney. Berlin, Germany; Berlin, Germany; Boston, MA; Cambridge, MA.

11:30 AM **BRAIN DEATH DOES NOT AFFECT HEPATIC ALLOGRAFT FUNCTION AND SURVIVAL AFTER ORTHOTOPIC TRANSPLANTATION IN A CANINE MODEL. (Abstract #1291)**
Philippe L. Compagnon, Hongbing Wang, Susanne L. Lindell, Mary S. Ametani, James H. Southard, Anthony M. D'Alessandro.

11:40 AM **NEUTRALIZATION OF KC AND MACROPHAGE INFLAMMATORY PROTEIN-2 ATTENUATES RENAL ISCHEMIA/REPERFUSION INJURY. (Abstract #1292)**
Masayoshi Miura, Xi Fu, Qiwei Zhang, Daniel G. Remick, Robert L. Fairchild. Cleveland, OH; Ann Arbor, MI.

11:50 AM **SUCCESSFUL SIX DAY KIDNEY PRESERVATION BY TROPHIC FACTOR SUPPLEMENTED SIMPLE COLD STORAGE. (Abstract #1293)**
Jonathan F. McAnulty, Ted W. Reid, Ken R. Waller, Christopher J. Murphy. Madison, WI; Lubbock, TX.

12:00 PM **A HYPOXIA RELATED GENE DETECTED BY mRNA DIFFERENTIAL DISPLAY AFTER ISOLATED INTESTINAL PERFUSION AND INTESTINAL TRANSPLANTATION IN PIGS. (Abstract #1294)**
Mehdi Hosseini, Felix Braun, Sven Laabs, Eberhard Wieland, Burckhardt Ringe. Göttingen, Germany; Göttingen, Germany.

12:10 PM **HYPOXIA MEDIATES THE INDUCTION OF TGF-β1 VIA HYPOXIA INDUCIBLE FACTOR-1α AFTER HEART TRANSPLANTATION. (Abstract #1295)**
Carla C. Baan, Aggie H. Balk, Wendy M. Mol, Jasper Snaathorst, Annemiek M. Peeters, Lex P. Maat, Bert G. Niesters, Willem Weimar. Rotterdam, The Netherlands.

12:20 PM **HGF PROTECTS HEPATOCYTES AGAINST HYPOXIA/ REOXYGENATION-INDUCED CELL DEATH BY REDUCING MITOCHONDRIAL GENERATION OF REACTIVE OXYGEN SPECIES THROUGH ACTIVATION OF PI3K-AKT PATHWAY. (Abstract #1296)**
Michitaka Ozaki, Yuko Nagata, Seiichi Suzuki. Setagaya, Tokyo, Japan.

Concurrent Session 62: Liver Transplantation: General

11:00 AM - 12:30 PM

Chicago Ballroom 6/7, Sheraton
Chairs: Maureen Martin and Ruud Krom

11:00 AM **MAINTAINING THE CELIAC TRUNK WITH THE LEFT GRAFT FOR IN-SITU SPLIT LIVER TRANSPLANTATION (SLT). (Abstract #1297)**
Murat Kilic, Philip Seu, John A. Goss. Houston, TX.

11:10 AM **LESSONS LEARNED IN BILE DUCT RECONSTRUCTION: A COMPARATIVE ANALYSIS OF ROUX-EN-Y HEPATICOJEJUNOSTOMY VERSUS CHOLEDOCHOCHOLEDOCHOSTOMY IN PATIENTS WITH PRIMARY SCLEROSING CHOLANGITIS. (Abstract #1298)**
Susan M. Lerner, Nicholas N. Nissen, Chung-Bao Hsieh, Cheng-Yuan Hsia, Gregg Kunder, Angeles Baquerizo, Sunil Geevargese, Pauline W. Chen, Douglas G. Farmer, Rafik M. Ghobrial, Hasan Yersiz, Ronald W. Busuttil. Los Angeles, CA.

11:20 AM **A COST-EFFECTIVENESS ANALYSIS OF BILIARY ANASTOMOSIS WITH OR WITHOUT T-TUBE AFTER ORTHOTOPIC LIVER TRANSPLANTATION. (Abstract #1299)**
Mitsugi Shimoda, Sammy Saab, Marcia Morrisey, Mark R. Ghobrial, Douglas G. Farmer, Steven-Huy B. Han, Rudolph A. Bedford, Leonard I. Goldstein, Paul Martin, Ronald W. Busuttil. Los Angeles, CA; Los Angeles, CA.

11:30 AM **ADDITION OF EPOPROSTENOL (EPO) INTO THE PRESSURIZED UW REDUCES THE INCIDENCE OF BILIARY STRICTURE (BS) AFTER LIVER TRANSPLANTATION (LTX). (Abstract #1300)**
J. Pirenne, F. Van Gelder, R. Aerts, W. Coosemans, S. Kimpen, D. Van Hees, T. Koshiba, T. Roskams, W. Van Steenbergen, P. Yap, J. Fevery, F. Nevens. Leuven, Belgium.

11:40 AM **MODIFIED PIGGYBACK TECHNIQUE IMPROVES GRAFT AND PATIENT SURVIVAL AFTER LIVER TRANSPLANTATION. (Abstract #1301)**
Jeroen de Jonge, Geert Kazemier, Sjoerd de Rave, Allard J. Rinksma, Herold J. Metselaar, Jan N.M. IJzermans, Hugo W. Tilanus. Rotterdam, The Netherlands.

11:50 AM **MUTATIONAL PROFILING OF HEPATOCELLULAR CARCINOMA PREDICTS TUMOR BEHAVIOR FOLLOWING ORTHOTOPIC LIVER TRANSPLANTATION. (Abstract #1302)**
C. Andrew Bonham, Sydney D. Finkelstein, Igor Dvorchik, J. Wallis Marsh. Pittsburgh, PA; Pittsburgh, PA.

12:00 PM **EVIDENCE FOR DIFFERENT MECHANISMS FOR TUMOUR GROWTH FOLLOWING LIVER RESECTION AND TRANSPLANTATION. (Abstract #1303)**
Claire L. Corps, Janusz Strzelczyk, Shaw S. Somers, J. Peter A. Lodge. Leeds, United Kingdom.

12:10 PM **ABO INCOMPATIBLE LIVER TRANSPLANTATION WITH NO IMMUNOLOGICAL GRAFT LOSSES UTILIZING PLASMAPHERESIS, SPLENECTOMY, AND QUADRUPLE IMMUNOSUPPRESSION. (Abstract #1304)**
Douglas W. Hanto, Annie H. Fecteau, Maria H. Alonso, John F. Valente, James F. Whiting. Cincinnati, OH.

12:20 PM **SIROLIMUS IS NOT ASSOCIATED WITH HYPERCHOLESTEROLEMIA IN THE ABSENCE OF PREDNISONE IN LIVER TRANSPLANT RECIPIENTS. (Abstract #1305)**
James F. Trotter, Michael Wachs, Thomas Trouillot, Thomas Bak, Marcelo Kugelmas, Gregory T. Everson, Igal Kam.

Concurrent Session 63: Basic Science: Tolerance III

11:00 AM - 12:30 PM

Chicago Ballroom 8, Sheraton
Chairs: Soji Oluwole and Takashi Maki

11:00 AM **FAILURE TO INDUCE ALLOGRAFT ACCEPTANCE IN PERFORIN-DEFICIENT MICE. (Abstract #1306)** *Young Investigator Award*
Anirban Bose, Yoshihiko Inoue, Fadi G. Lakkis. Atlanta, GA.

11:10 AM **ACTIVE IMMUNE REGULATION IS AN IMPORTANT MECHANISM OF ALLOGRAFT TOLERANCE. (Abstract #1307)**
Alberto Sanchez-Fueyo, Martina Weber, Evia Csizmadia, Sylvie Ferrari Lacraz, Yongsheng Li, Terry B. Strom, Xin Xiao Zheng. Boston, MA.

11:20 AM **IL13 GENE TRANSFER POTENTIATES THE EFFICACY OF REGULATORY T CELLS BY UPREGULATING THE EXPRESSION OF PROTECTIVE MOLECULES IN THE INFECTIOUS TOLERANCE PATHWAY. (Abstract #1308)**
Bibo Ke, Xiu-Da Shen, Feng Gao, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA.

11:30 AM **IN VITRO CHARACTERIZATION OF T REGULATORY (Tr) CELLS IN THE INFECTIOUS TOLERANCE PATHWAY IN TRANSPLANT RECIPIENTS. (Abstract #1309)**
Yuan Zhai, Xiu-Da Shen, Feng Gao, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. Los Angeles, CA.

11:40 AM **DIFFERENT MECHANISMS FOR THE RAPID INDUCTION OF $CD4^+$ AND $CD8^+$ DONOR-SPECIFIC THYMOCYTE TOLERANCE ACROSS A FULL MHC BARRIER THROUGH BMT AND COSTIMULATORY BLOCKADE. (Abstract #1310)**
J. Kurtz, H. Ito, J. Shaffer, M. Sykes. Boston, MA.

11:50 AM **ANTIGEN SPECIFIC B CELLS ARE REQUIRED TO INDUCE SKIN GRAFT TOLERANCE FOLLOWING INTRAVENOUS INJECTION OF SOLUBLE ANTIGEN. (Abstract #1311)**
Anna Valujskikh, Peter S. Heeger. Cleveland, OH.

12:00 PM **RENAL ALLOGRAFT ACCEPTANCE IN RHESUS MONKEY IS MEDIATED BY ACTIVE IMMUNE REGULATION MECHANISM. (Abstract #1312)**
Masaaki Katayama, Ewa Jankowska-Gan, Satoshi Kusaka, John H. Fechner, William J. Burlingham, Stuart J. Knechtle. Madison, WI.

12:10 PM **B7/CTLA4 PATHWAY IS ESSENTIAL TO GENERATE REGULATORY CELLS AFTER INTRATRACHEAL DELIVERY OF ALLOANTIGEN. (Abstract #1313)**
Yoshinobu Akiyama, Nozomu Shirasugi, Kennji Matsumoto, Masaki Kitajima, Masanori Niimi. Tokyo, Japan; Tokyo, Japan; Tokyo, Japan.

12:20 PM **THE MECHANISM OF ANTI-GAL-PRODUCING B CELL TOLERANCE INDUCED BY MIXED CHIMERISM. (Abstract #1314)**
Toshiyasu Kawahara, Hideki Ohdan, Guiling Zhao, Megan Sykes. Boston, MA; Hiroshima, Japan.

Concurrent Session 64: Immunosuppression and Immune Responses

11:00 AM - 12:30 PM

Chicago Ballroom 9, Sheraton
Chairs. Ginny L. Bumgardner and Adriana Zeevi

11:00 AM **ANTI-CD20 MONOCLONAL ANTIBODY (RITUXIMAB) FOR PEDIATRIC POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDERS: A PRELIMINARY MULTICENTER EXPERIENCE. (Abstract #1315)**
Steven A. Webber, Richard N. Fine, William McGhee, William Harmon, Minnie Sarwal, Albert Faro, Frederick J. Fricker, James Y. Coe, Oscar Salvatierra, Jr., Brigitta Mueller, George M. Mazariegos, Jorge Reyes. Pittsburgh, PA.

11:10 AM **THE USE OF RITUXAN (ANTI-CD20 MOAB) IN TRANSPLANTATION. (Abstract #1316)**
Hans W. Sollinger, Yolanda Tai Becker, John D. Pirsch.

11:20 AM **SIROLIMUS FOR PROPHYLAXIS OF REJECTION AND RESCUE IN TRANSPLANTED CHILDREN RECEIVING TACROLIMUS. (Abstract #1317)**
Rakesh Sindhi, Jessie Ganjoo, Steven Webber, Tracy Daugherty, William McGhee, Brenda Cooperstone, David Holt, George Mazariegos, John Fung, Jorge Reyes. Pittsburgh, PA; St. Davids, PA; London, United Kingdom.

11:30 AM **SUPPRESSION OF CD154-EXPRESSION BY CALCINEURIN INHIBITORS AFTER ORGAN TRANSPLANTATION IS INCOMPLETE. (Abstract #1318)**
Mark L. Kuijf, Herold J. Metselaar, Hugo W. Tilanus, Gerda J. Bouma, Roel de Weger, Margreet Jonker, Jaap Kwekkeboom. Rotterdam, The Netherlands; Rijswijk, The Netherlands; Utrecht, The Netherlands.

11:40 AM **PHASE 1 EVALUATION OF A NOVEL CALCINEURIN INHIBITOR ISA_TX 247. (Abstract #1319)**
Mark D. Abel, Lauma J. Aspeslet, Derrick G. Freitag, Philip F. Halloran, Norman M. Kneteman, Selvaraj Naicker, Daniel J. Trepanier, Robert T. Foster, Randall W. Yatscoff. Edmonton, AB, Canada; Edmonton, AB, Canada.

11:50 AM **IDENTIFICATION OF EBV-SPECIFIC CD8+ T CELLS USING MHC/PEPTIDE TETRAMERS IN PEDIATRIC TRANSPLANT RECIPIENTS. (Abstract #1320)** *Young Investigator Award*
Daniel A. Falco, Ronald R. Nepomuceno, Penelope A. Robbins, Sheri M. Krams, Peter Lee, Mark Davis, Steve Alexander, Kenneth Cox, Oscar Salvatierra, Carlos O. Esquivel, Olivia M. Martinez. Stanford, CA.

12:00 PM **DONOR REACTIVE DTH RESPONSES IN TRANSPLANT PATIENTS. (Abstract #1321)**
R. P. Pelletier, R. M. Ferguson, A. M. VanBuskirk, D. Xia, P. K. Hennessy, C. G. Orosz. Columbus, OH.

12:10 PM **DONOR DOWN-REGULATED T CELL RESPONSES IN TRANSPLANT PATIENTS. (Abstract #1322)** *Young Investigator Award*
R. P. Pelletier, R. M. Ferguson, A. M. VanBuskirk, D. Xia, P. K. Hennessy, C. G. Orosz. Columbus, OH.

12:20 PM **SUCCESSFUL COMPOSITE TISSUE TRANSPLANTATION—NERVE AND FUNCTIONAL RECOVERY AFTER TWO YEARS. (Abstract #1323)**
Linda C. Cendales, Warren C. Breidenbach, Darla K. Granger. Louisville, KY; Louisville, KY.

Concurrent Session 65: Donor Management and Outcomes

11:00 AM - 12:30 PM

Sheraton Ballroom 1-3, Sheraton
Chairs: Steve Bartlett and Nicholas Feduska

11:00 AM **THE HAND-ASSISTED MODIFICATION OF THE TRADITIONAL LAPAROSCOPIC DONOR NEPHRECTOMY RETAINS ITS ADVANTAGES OVER OPEN DONOR NEPHRECTOMY WHILE ADDRESSING SHORTCOMINGS. (Abstract #1324)**
Joseph F. Buell, David S. Bruce, Michael J. Hanaway, Atshui C. Yoshida, David C. Cronin, Rino Munda, Kenneth A. Newell, E. Steve Woodle. Cincinnati, OH; Chicago, IL.

11:00 AM **106 CASES OF RIGHT LAPAROSCOPIC DONOR NEPHRECTOMY: A MULTICENTER EXPERIENCE. (Abstract #1325)**
Joseph F. Buell, Alan Koffron, Michael Edye, Mark Johnson, Juan Arenas, Paul Kuo, Lynt Johnson, David S. Bruce, Joseph Leventhal, Eugene Cho, Stephen T. Bartlett, Michael J. Hanaway, E. Steve Woodle.

11:10 AM **LAPAROSCOPIC DONOR NEPHRECTOMY FOR RIGHT KIDNEYS, WHY NOT? (Abstract #1326)**
Mikel Prieto, George K. Chow, Humberto E. Bohorquez, Mark D. Stegall. Rochester, MN.

11:20 AM **SAFETY OF HEPATITIS B CORE ANTIBODY POSITIVE CADAVERIC DONORS IN RENAL TRANSPLANTATION: MULTIVARIATE UNOS ANALYSIS. (Abstract #1327)**
Suphamai Bunnapradist, Tse-Ling Fong, Stanley C. Jordan, Yong W. Cho. Los Angeles, CA; Los Angeles, CA.

11:30 AM **TRANSMISSION OF DONOR MALIGNANCY DURING RENAL TRANSPLANTATION: A REPORT FROM THE ISRAEL PENN INTERNATIONAL TRANSPLANT TUMOR REGISTRY. (Abstract #1328)**
M. J. Aull, R. R. Alloway, J. Trofe, M. R. First, V. R. Peddi, E. S. Woodle, J. F. Buell. Cincinnati, OH.

11:40 AM **COMPARISON OF OUTCOMES OF LIVER-KIDNEY TRANSPLANT VERSUS THE CONTRALATERAL KIDNEY USED FOR KIDNEY TRANSPLANT OR KIDNEY-PANCREAS TRANSPLANT. (Abstract #1329)**
Tse-Ling Fong, Stanley C. Jordan, Suphamai Bunapradist, Yong W. Cho. Los Angeles, CA; Los Angeles, CA.

11:50 AM **NOVEL PRESERVATION SOLUTION IMPROVES EARLY FUNCTION IN THE COLD STORED AND MACHINE PRESERVED KIDNEY. (Abstract #1330)**
Maximilian M. Polyak, Ben O. Arrington, William T. Stubenbord, Sandi Kapur, Milan Kinkhabwala. New York, NY.

12:00 PM **THE PAIRING EFFECT DEMONSTRATES DONOR TISSUE QUALITY AS THE KEY DETERMINANT OF EARLY AND LATE RENAL ALLOGRAFT FUNCTION. (Abstract #1331)**
Sita Gourishankar, Gian S. Jhangri, Robert B. Huizinga, Sandra M. Cockfield, Philip F. Halloran. Edmonton, AB, Canada.

12:10 PM **A MULTIVARIATE ANALYSIS OF RISK FACTORS FOR SLOW GRAFT FUNCTION (SGF) AFTER KIDNEY TRANSPLANT. (Abstract #1332)**
Abhi Humar, Thiagarjan Ramcharan, Steven Paraskevas, Roger Denis, Roberto Mierelles, Kristen Gillingham, Arthur Matas. Minneapolis, MN.

12:20 PM **SUCCESSFUL LONG TERM OUTCOMES IN ADULT RECIPIENTS OF PEDIATRIC EN BLOC KIDNEYS. (Abstract #1333)**
Jade S. Hiramoto, Henry R. Randall, Chris E. Freise, Peter N. Bretan, Stephen Tomlanovich, Peter G. Stock, Ryutaro Hirose. San Francisco, CA; Louisville, KY.

Concurrent Session 66: New Maintenance Immunosuppressive Agents

11:00 AM - 12:30 PM

Sheraton Ballroom 4/5, Sheraton
Chairs: Gabriel Danovitch and William Marks

11:00 AM **FTY720 MEDIATES REVERSIBLE REDUCTION OF LYMPHOCYTE COUNTS IN HUMAN RENAL ALLOGRAFT RECIPIENTS–EVIDENCE FOR ALTERED LYMPHOCYTE TRAFFICKING FOR THE MECHANISM OF ACTION OF FTY720. (Abstract #1334)** *Young Investigator Award*
Torsten Boehler, Johannes Waiser, Manuela Schuetz, Duska Dragun, Klemens Budde, Hans- H. Neumayer. Berlin, Berlin, Germany.

11:10 AM **ONE YEAR RESULTS OF A MULTICENTER, OPEN-LABEL TRIAL ON SAFETY AND EFFICACY OF CERTICAN™ (RAD) USED IN COMBINATION WITH SIMULECT®, CORTICOSTEROIDS, AND FULL OR REDUCED DOSE NEORAL® IN RENAL TRANSPLANTATION. (Abstract #1335)**
J. Curtis, B. Nashan, C. Ponticelli, G. Mourad, R. Boger, the RAD 156 Study Group. Birmingham, AL.

11:20 AM **EXPOSURE-RESPONSE RELATIONSHIPS FOR EVEROLIMUS IN DE NOVO RENAL TRANSPLANTATION: TOWARD DEFINING A THERAPEUTIC RANGE. (Abstract #1336)**
J. M. Kovarik, C. Rordorf, L. McMahon, S. Berthier, R. Boger. East Hanover, NJ.

11:30 AM **INTERNATIONAL, DOUBLE-BLIND, PARALLEL GROUP STUDY OF THE SAFETY AND EFFICACY OF CERTICAN™ (RAD) VERSUS MYCOPHENOLATE MOFETIL (MMF) IN COMBINATION WITH NEORAL® AND STEROIDS. (Abstract #1337)**
S. Vitko, R. Margreiter, W. Weimar, J. Dantal, H. Viljoen, N. Cambon, R. Boger, the RAD 201 Study Group. Videnska, Czech Republic.

11:40 AM **FTY720 METABOLISM IN HUMANS. (Abstract #1338)**
Robert Schmouder, Robert Dannecker, Somesh Choudhury, Denise Barilla, Tomasz Sablinski. East Hanover, NJ; Basel, Switzerland; East Hanover, NJ.

11:50 AM **NORTH/SOUTH AMERICAN, DOUBLE-BLIND, PARALLEL GROUP STUDY OF THE SAFETY AND EFFICACY OF CERTICAN™ (RAD) VERSUS MYCOPHENOLATE MOFETIL (MMF) IN COMBINATION WITH NEORAL® AND CORTICOSTEROIDS. (Abstract #1339)**
B. Kaplan, H. Tedesco-Silva, R. Mendez, B. Kahan, D. Van Buren, R. Boger, the RAD 251 Study Group. Ann Arbor, MI.

12:00 PM **PROLONGED, CONSISTENT ORAL ABSORPTION OF FTY720. (Abstract #1340)**
Robert Schmouder, Somesh Choudhury, Denise Barilla, Patricia Ledford, Guy Taccard. East Hanover, NJ; Basel, Switzerland.

12:10 PM **LONGITUDINAL INFLUENCE OF EVEROLIMUS ON CYCLOSPORINE ASSESSED OVER 6 MONTHS IN TWO BLINDED *DE NOVO* KIDNEY TRANSPLANT TRIALS. (Abstract #1341)**
J. M. Kovarik, B. Kaplan, S. Vitko, L. McMahon, M. Attinger, R. Boger, C. Rordorf. East Hanover, NJ; Livingston, NJ; Praha, Czech Republic.

12:20 PM **EFFECT OF IMPAIRED HEPATIC FUNCTION ON THE SYSTEMIC EXPOSURE OF FTY720. (Abstract #1342)**
Denise Barilla, Somesh Choudhury, Patricia Ledford, Joaquim Figueiredo, Robert Schmouder. East Hanover, NJ; Basel, Switzerland.

Adjorn

A LETTER OF THANKS TO THE TRANSPLANT COMMUNITY

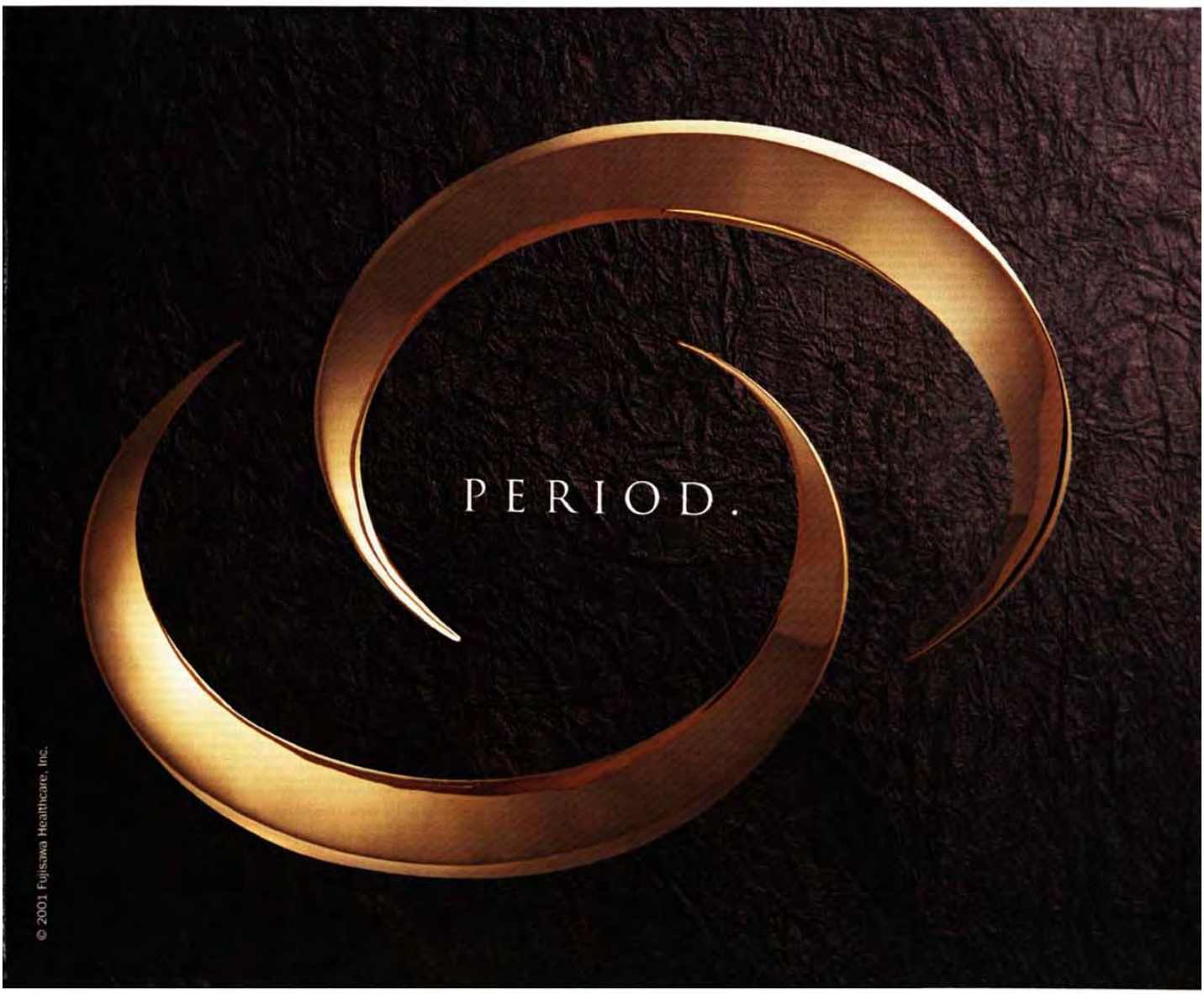
We at Wyeth Laboratories would like to take this opportunity to express our thanks to you for your dedication to excellence in the field of transplant medicine. It is this dedication that has produced the extraordinary success rates in short-term survival of both graft and patient in solid organ transplant. We feel fortunate for the opportunity to join with you in your continued pursuit of excellence in transplant.

Perhaps the most important challenge in transplant today is in bringing the long-term success rate up to a level commensurate with that seen over the shorter term. As we are all aware, the lack of available organs for transplant places a tremendous burden on our community to improve the life of the graft and the quality of life of the patient. Wyeth Laboratories is committed to working with you to develop regimens which will help ensure that every organ—and every patient—will have the best opportunity for long-term survival.

Yours truly,
Wyeth Transplantation

80513-00 May 2001

THERE'S NO SUBSTITUTE.



PROGRAF®
tacrolimus capsules and injection

Fujisawa Healthcare

Please see brief summary of prescribing information on adjacent page.

PROGRAF®

tacrolimus capsules
tacrolimus injection (for intravenous infusion only)

Revised October, 1998

The following is a brief summary; see current package insert for complete information.

WARNING

Increased susceptibility to infection and the possible development of lymphoma may result from immunosuppression. Only physicians experienced in immunosuppressive therapy and management of organ transplant patients should prescribe Prograf. Patients receiving the drug should be managed in facilities equipped and staffed with adequate laboratory and supportive medical resources. The physician responsible for maintenance therapy should have complete information requisite for the follow-up of the patient.

INDICATIONS AND USAGE:

Prograf is indicated for the prophylaxis of organ rejection in patients receiving allogeneic liver or kidney transplants. It is recommended that Prograf be used concomitantly with adrenal corticosteroids. Because of the risk of anaphylaxis, Prograf injection should be reserved for patients unable to take Prograf capsules orally.

CONTRAINDICATIONS:

Prograf is contraindicated in patients with a hypersensitivity to tacrolimus. Prograf injection is contraindicated in patients with a hypersensitivity to HCO-60 (polyoxyl 60 hydrogenated castor oil).

WARNINGS:

(See boxed WARNING.)

Insulin-dependent post-transplant diabetes mellitus (PTDM) was reported in 20% of Prograf-treated kidney transplant patients. The median time to onset of PTDM was 68 days. Insulin dependence was reversible in 15% of these patients at one year and in 50% at two years post transplant. Black and Hispanic kidney transplant patients were at an increased risk of development of PTDM.

Insulin-dependent post-transplant diabetes mellitus was reported in 18% and 11% of Prograf-treated liver transplant patients and was reversible in 45% and 31% of these patients at one year post transplant, in the U.S. and European randomized studies, respectively. Hyperglycemia was associated with the use of Prograf in 47% and 33% of liver transplant recipients in the U.S. and European randomized studies, respectively, and may require treatment (see **ADVERSE REACTIONS**).

Prograf can cause neurotoxicity and nephrotoxicity, particularly when used in high doses. Nephrotoxicity was reported in approximately 52% of kidney transplantation patients and in 40% and 36% of liver transplantation patients receiving Prograf in the U.S. and European randomized trials, respectively. More overt nephrotoxicity is seen early after transplantation, characterized by increasing serum creatinine and a decrease in urine output. Patients with impaired renal function should be monitored closely as the dosage of Prograf may need to be reduced. In patients with persistent elevations of serum creatinine who are unresponsive to dosage adjustments, consideration should be given to changing to another immunosuppressive therapy. Care should be taken in using tacrolimus with other nephrotoxic drugs. In particular, to avoid excess nephrotoxicity, Prograf should not be used simultaneously with cyclosporine. Prograf or cyclosporine should be discontinued at least 24 hours prior to initiating the other. In the presence of elevated Prograf or cyclosporine concentrations, dosing with the other drug usually should be further delayed.

Mild to severe hyperkalemia was reported in 31% of kidney transplant recipients and in 45% and 13% of liver transplant recipients treated with Prograf in the U.S. and European randomized trials, respectively, and may require treatment. Serum potassium levels should be monitored and potassium-sparing diuretics should not be used during Prograf therapy.

Neurotoxicity, including tremor, headache, and other changes in motor function, mental status, and sensory function were reported in approximately 55% of liver transplant recipients in the two randomized studies. Tremor occurred more often in Prograf-treated kidney transplant patients (54%) compared to cyclosporine-treated patients. Tremor and headache have been associated with high whole-blood concentrations of tacrolimus and may respond to dosage adjustment. Seizures have occurred in adult and pediatric patients receiving Prograf (see **ADVERSE REACTIONS**). Coma and delirium also have been associated with high plasma concentrations of tacrolimus.

As in patients receiving other immunosuppressants, patients receiving Prograf are at increased risk of developing lymphomas and other malignancies, particularly of the skin. The risk appears to be related to the intensity and duration of immunosuppression rather than to the use of any specific agent. A lymphoproliferative disorder (LPD) related to Epstein-Barr Virus (EBV) infection has been reported in immunosuppressed organ transplant recipients. The risk of LPD appears greatest in young children who are at risk for primary EBV infection while immunosuppressed or who are switched to Prograf following long-term immunosuppression therapy. Because of the danger of oversuppression of the immune system which can increase susceptibility to infection, combination immunosuppressant therapy should be used with caution.

A few patients receiving Prograf injection have experienced anaphylactic reactions. Although the exact cause of these reactions is not known, other drugs with castor oil derivatives in the formulation have been associated with anaphylaxis in a small percentage of patients. Because of this potential risk of anaphylaxis, Prograf injection should be reserved for patients who are unable to take Prograf capsules.

Patients receiving Prograf injection should be under continuous observation for at least the first 30 minutes following the start of the infusion and at frequent intervals thereafter. If signs or symptoms of anaphylaxis occur, the infusion should be stopped. An aqueous solution of epinephrine should be available at the bedside as well as a source of oxygen.

PRECAUTIONS:

General

Hypertension is a common adverse effect of Prograf therapy (see **ADVERSE REACTIONS**). Mild or moderate hypertension is more frequently reported than severe hypertension. Antihypertensive therapy may be required; the control of blood pressure can be accomplished with any of the common antihypertensive agents. Since tacrolimus may cause hyperkalemia, potassium-sparing diuretics should be avoided. While calcium-channel blocking agents can be effective in treating Prograf-associated hypertension, care should be taken since interference with tacrolimus metabolism may require a dosage reduction (see **Drug Interactions**).

Renally and Hepatically Impaired Patients

For patients with renal insufficiency some evidence suggests that lower doses should be used.

The use of Prograf in liver transplant recipients experiencing post-transplant hepatic impairment may be associated with increased risk of developing renal insufficiency related to high whole-blood levels of tacrolimus. These patients should be monitored closely and dosage adjustments should be considered. Some evidence suggests that lower doses should be used in these patients.

Myocardial Hypertrophy

Myocardial hypertrophy has been reported in association with the administration of Prograf, and is generally manifested by echocardiographically demonstrated concentric increases in left ventricular posterior wall and interventricular septum thickness. Hypertrophy has been observed in infants, children and adults. This condition appears reversible in most cases following dose reduction or discontinuance of therapy. In a group of 20 patients with pre- and post-treatment echocardiograms who showed evidence of myocardial hypertrophy, mean tacrolimus whole blood concentrations during the period prior to diagnosis of myocardial hypertrophy ranged from 11 to 53 ng/mL in infants (N=10, age 0.4 to 2 years), 4 to 46 ng/mL in children (N=7, age 2 to 15 years) and 11 to 24 ng/mL in adults (N=3, age 37 to 53 years)

In patients who develop renal failure or clinical manifestations of ventricular dysfunction while receiving Prograf therapy, echocardiographic evaluation should be considered. If myocardial hypertrophy is diagnosed, dosage reduction or discontinuation of Prograf should be considered.

Information for Patients

Patients should be informed of the need for repeated appropriate laboratory tests while they are receiving Prograf. They should be given complete dosage instructions, advised of the potential risks during pregnancy, and informed of the increased risk of neoplasia. Patients should be informed that changes in dosage should not be undertaken without first consulting their physician.

Patients should be informed that Prograf can cause diabetes mellitus and should be advised of the need to see their physician if they develop frequent urination, increased thirst or hunger.

Laboratory Tests

Serum creatinine, potassium, and fasting glucose should be assessed regularly. Routine monitoring of metabolic and hematologic systems should be performed as clinically warranted.

Drug Interactions

Drug interaction studies with tacrolimus have not been conducted. Due to the potential for additive or synergistic impairment of renal function, care should be taken when administering Prograf with drugs that may be associated with renal dysfunction. These include, but are not limited to, aminoglycosides, amphotericin B, and cisplatin. Initial clinical experience with the co-administration of Prograf and cyclosporine resulted in additive/synergistic nephrotoxicity. Patients switched from cyclosporine to Prograf should receive the first Prograf dose no sooner than 24 hours after the last cyclosporine dose. Dosing may be further delayed in the presence of elevated cyclosporine levels.

Drugs That May Alter Tacrolimus Concentrations

Since tacrolimus is metabolized mainly by the CYP3A enzyme systems, substances known to inhibit these enzymes may decrease the metabolism of tacrolimus with resultant increases in whole blood or plasma concentrations. Drugs known to induce these enzyme systems may result in an increased metabolism of tacrolimus and decreased whole blood or plasma concentrations. Monitoring of blood concentrations and appropriate dosage adjustments are essential when such drugs are used concomitantly.

Drugs that may increase tacrolimus concentrations: **Calcium Channel Blockers**: diltiazem, nicardipine, nifedipine, verapamil, **Antifungal Agents**: clotrimazole, fluconazole, itraconazole, ketoconazole, **Macrolide Antibiotics**: clarithromycin, erythromycin, troleandomycin, **Gastrointestinal Prokinetic Agents**: cisapride, metoclopramide, **Other Drugs**: bromocriptine, cimetidine, cyclosporine, danazol, methylprednisolone, protease inhibitors.

Drugs that may decrease tacrolimus blood concentrations: **Anticonvulsants**: carbamazepine, phenobarbital, phenytoin, **Antibiotics**: rifabutin, rifampin. This list of drugs is not all-inclusive.

Interaction studies with drugs used in HIV therapy have not been conducted. However, care should be exercised when drugs that are nephrotoxic (e.g., ganciclovir) or that are metabolized by CYP3A (e.g., ritonavir) are administered concomitantly with tacrolimus. Grapefruit juice affects CYP3A-mediated metabolism and should be avoided.

Other Drug Interactions

Immunosuppressants may affect vaccination. Therefore, during treatment with Prograf, vaccination may be less effective. The use of live vaccines should be avoided; live vaccines may include, but are not limited to measles, mumps, rubella, oral polio, BCG, yellow fever, and TY 21a typhoid.[1]

Carcinogenesis, Mutagenesis and Impairment of Fertility

An increased incidence of malignancy is a recognized complication of immunosuppression in recipients of organ transplants. The most common forms of neoplasms are non-Hodgkin's lymphomas and carcinomas of the skin. As with other immunosuppressive therapies, the risk of malignancies in Prograf recipients may be higher than in the normal, healthy population. Lymphoproliferative disorders associated with Epstein-Barr Virus infection have been seen. It has been reported that reduction or discontinuation of immunosuppression may cause the lesions to regress.

No evidence of genotoxicity was seen in bacterial (Salmonella and E. coli) or mammalian (Chinese hamster lung-derived cells) *in vitro* assays of mutagenicity, the *in vitro* CHO/HGPRT assay of mutagenicity, or *in vivo* clastogenicity assays performed in mice; tacrolimus did not cause unscheduled DNA synthesis in rodent hepatocytes.

Carcinogenicity studies were carried out in male and female rats and mice. In the 80-week mouse study and in the 104-week rat study no relationship of tumor incidence to tacrolimus dosage was found. The highest doses used in the mouse and rat studies were 0.8 - 2.5 times (mice) and 3.5 - 7.1 times (rats) the recommended clinical dose range of 0.1 - 0.2 mg/kg/day when corrected for body surface area.

No impairment of fertility was demonstrated in studies of male and female rats. Tacrolimus, given orally at 1.0 mg/kg (0.7 - 1.4X the recommended clinical dose range of 0.1 - 0.2 mg/kg/day based on body surface area corrections) to male and female rats, prior to and during mating, as well as to dams during gestation and lactation, was associated with embryolethality and with adverse effects on female reproduction. Effects on female reproductive function (parturition) and embryolethal effects were indicated by a higher rate of pre-implantation loss and increased numbers of undelivered and nonviable pups. When given at 3.2 mg/kg (2.3 - 4.6X the recommended clinical dose range based on body surface area correction), tacrolimus was associated with maternal and paternal toxicity as well as reproductive toxicity including marked adverse effects on estrus cycles, parturition, pup viability, and pup malformations.

Pregnancy: Category C

In reproduction studies in rats and rabbits, adverse effects on the fetus were observed mainly at dose levels that were toxic to dams. Tacrolimus at oral doses of 0.32 and 1.0 mg/kg during organogenesis in rabbits was associated with maternal toxicity as well as an increase in incidence of abortions; these doses are equivalent to 0.5 - 1X and 1.6 - 3.3X the recommended clinical dose range (0.1 - 0.2 mg/kg) based on body surface area corrections. At the higher dose only, an increased incidence of malformations and developmental variations was also seen. Tacrolimus, at oral doses of 3.2 mg/kg during organogenesis in rats, was associated with maternal toxicity and caused an increase in late resorptions, decreased numbers of live births, and decreased pup weight and viability. Tacrolimus, given orally at 1.0 and 3.2 mg/kg (equivalent to 0.7 - 1.4X and 2.3 - 4.6X the recommended clinical dose range based on body surface area corrections) to pregnant rats after organogenesis and during lactation, was associated with reduced pup weights.

No reduction in male or female fertility was evident.

There are no adequate and well-controlled studies in pregnant women. Tacrolimus is transferred across the placenta. The use of tacrolimus during pregnancy has been associated with neonatal hyperkalemia and renal dysfunction. Prograf should be used during pregnancy only if the potential benefit to the mother justifies potential risk to the fetus.

Nursing Mothers

Since tacrolimus is excreted in human milk, nursing should be avoided.

Pediatric Patients

Experience with Prograf in pediatric kidney transplant patients is limited. Successful liver transplants have been performed in pediatric patients (ages up to 16 years) using Prograf. The two randomized active-controlled trials of Prograf in primary liver transplantation included 56 pediatric patients. Thirty-one patients were randomized to Prograf-based and 25 to cyclosporine-based therapies. Additionally, a maximum of 122 pediatric patients were studied in an uncontrolled trial of tacrolimus in living related donor liver transplantation. Pediatric patients generally required higher doses of Prograf to maintain blood trough concentrations of tacrolimus similar to adult patients.

ADVERSE REACTIONS:

Liver Transplantation

The principal adverse reactions of Prograf are tremor, headache, diarrhea, hypertension, nausea and renal dysfunction. These occur with oral and IV administration of Prograf and may respond to a reduction in dosing. Diarrhea was sometimes associated with other gastrointestinal complaints such as nausea and vomiting.

Hyperkalemia and hypomagnesemia have occurred in patients receiving Prograf therapy. Hyperglycemia has been noted in many patients, some may require insulin therapy.

Kidney Transplantation

The most common adverse reactions reported were infection, tremor, hypertension, decreased renal function, constipation, diarrhea, headache, abdominal pain and insomnia.

The following adverse events were reported in >15% incidence among liver and kidney transplantation patients: NERVOUS SYSTEM: headache, tremor, insomnia, paresthesia; GASTROINTESTINAL: diarrhea, nausea, constipation, vomiting; CARDIOVASCULAR: hypertension; UROGENITAL: creatinine increased, urinary tract infection; METABOLIC AND NUTRITIONAL: hyperglycemia, hypomagnesemia; HEMIC AND LYMPHATIC: anemia; MISCELLANEOUS: pain, fever, back pain, abdominal pain, peripheral edema, asthenia; RESPIRATORY SYSTEM: dyspnea; SKIN: pruritus, rash.

In addition among liver transplantation patients: GASTROINTESTINAL: LFT abnormal, anorexia; UROGENITAL: kidney function abnormal, BUN increased, oliguria; HEMIC AND LYMPHATIC: leukocytosis, thrombocytopenia; MISCELLANEOUS: ascites; RESPIRATORY SYSTEM: atelectasis, pleural effusion.

In addition among kidney transplantation: NERVOUS SYSTEM: dizziness; GASTROINTESTINAL: dyspepsia; CARDIOVASCULAR: chest pain; METABOLIC AND NUTRITIONAL: hypophosphatemia, diabetes mellitus, hyperlipemia; HEMIC AND LYMPHATIC: leukopenia; MISCELLANEOUS: infection; RESPIRATORY SYSTEM: cough increased; MUSCULOSKELETAL: athralagia.

Less Frequently Reported Adverse Reactions

The following adverse events were reported in the range of 3% to less than 15% incidence in either liver or kidney transplant recipients who were treated with tacrolimus in the Phase 3 comparative trials.

NERVOUS SYSTEM (see **WARNINGS**) abnormal dreams, agitation, amnesia, anxiety, confusion, convulsion, depression, dizziness, emotional lability, encephalopathy, hallucinations, hypertonia, incoordination, myoclonus, nervousness, neuropathy, psychosis, somnolence, thinking abnormal; SPECIAL SENSES: abnormal vision, amblyopia, ear pain, otitis media, tinnitus; GASTROINTESTINAL: anorexia, cholangitis, cholestatic jaundice, dyspepsia, dysphagia, esophagitis, flatulence, gastritis, gastrointestinal hemorrhage, GGT increase, GI perforation, hepatitis, ileus, increased appetite, jaundice, liver damage, liver function test abnormal, oral moniliasis, rectal disorder, stomatitis; CARDIOVASCULAR: angina pectoris, chest pain, deep thrombophlebitis, abnormal ECG, hemorrhage, hypotension, postural hypotension, peripheral vascular disorder, phlebitis, tachycardia, thrombosis, vasodilatation; UROGENITAL (see **WARNINGS**): albuminuria, cystitis, dysuria, hematuria, hydronephrosis, kidney failure, kidney tubular necrosis, nocturia, pyuria, toxic nephropathy, oliguria, urinary frequency, urinary incontinence, vaginitis; METABOLIC/NUTRITIONAL: acidosis, alkaline phosphatase increased, alkalosis, ALT (SGPT) increased, AST (SGOT) increased, bicarbonate decreased, bilirubinemia, BUN increased, dehydration, GGT increased, healing abnormal, hypercalcemia, hypercholesterolemia, hyperlipemia, hyperphosphatemia, hyperuricemia, hypervolemia, hypocalcemia, hypoglycemia, hyponatremia, hypophosphatemia, hypoproteinemia, lactic dehydrogenase increase, weight gain; ENDOCRINE (see **PRECAUTIONS**): Cushing's syndrome, diabetes mellitus; HEMIC/LYMPHATIC: coagulation disorder, ecchymosis, hypochromic anemia, leukocytosis, leukopenia, polycythemia, prothrombin decreased, serum iron decreased, thrombocytopenia; MISCELLANEOUS: abdomen enlarged, abscess, accidental injury, allergic reaction, cellulitis, chills, flu syndrome, generalized edema, hernia, peritonitis, photosensitivity reaction, sepsis; MUSCULOSKELETAL: arthralgia, cramps, generalized spasm, joint disorder, leg cramps, myalgia, myasthenia, osteoporosis; RESPIRATORY: asthma, bronchitis, cough increased, lung disorder, pneumothorax, pulmonary edema, pharyngitis, pneumonia, respiratory disorder, rhinitis, sinusitis, voice alteration; SKIN: acne, alopecia, exfoliative dermatitis, fungal dermatitis, herpes simplex, hirsutism, skin discoloration, skin disorder, skin ulcer, sweating.

There have been rare spontaneous reports of myocardial hypertrophy associated with clinically manifested ventricular dysfunction in patients receiving Prograf therapy.

Please see the current package insert for complete information regarding adverse effects.

Prograf capsules (tacrolimus capsules) 0.5 mg

Oblong, light yellow, branded with red "0.5 mg" on the capsule cap and "617" on the capsule body, supplied in 60-count bottles (NDC 0469-0607-67) and 10 blister cards of 10 capsules (NDC 0469-0607-10), containing the equivalent of 0.5 mg anhydrous tacrolimus.

Prograf capsules (tacrolimus capsules) 1 mg

Oblong, white, branded with red "1 mg" on the capsule cap and "617" on the capsule body, supplied in 100-count bottles (NDC 0469-0617-71) and 10 blister cards of 10 capsules (NDC 0469-0617-10), containing the equivalent of 1 mg anhydrous tacrolimus.

Prograf capsules (tacrolimus capsules) 5 mg

Oblong, grayish/red, branded with white "5 mg" on the capsule cap and "657" on the capsule body, supplied in 100-count bottles (NDC 0469-0657-71) and 10 blister cards of 10 capsules (NDC 0469-0657-10), containing the equivalent of 5 mg anhydrous tacrolimus.

Store and Dispense

Store at 25°C (77°F), excursion permitted to 15°C-30°C (59°F-86°F)

Prograf Injection (tacrolimus injection) 5mg (for IV infusion only)

Supplied as a sterile solution in 1 mL ampules containing the equivalent of 5 mg of anhydrous tacrolimus per mL, in boxes of 10 ampules (NDC 0469-3016-01)

Store and Dispense

Store between 5°C and 25°C (41°F and 77°F)

Rx only

Made in Ireland for Fujisawa Healthcare, Inc., Deerfield, IL 60015-2548
by Fujisawa Ireland, Ltd., Killorglin, Co. Kerry, Ireland

REFERENCE
1. CDC Recommendations of the Advisory Committee on Immunization Practices. Use of vaccines and immune globulins in persons with altered immunocompetence. MMWR 1993;42(RR-4):1-18

Fujisawa Healthcare

Z140304

TRANSPLANT
2001
ABSTRACTS

PLENARY SESSION I: BASIC

Abstract# 1

MEMORY BUT NOT NAIVE T CELLS REJECT VASCULARIZED CARDIAC ALLOGRAFTS IN THE ABSENCE OF SECONDARY LYMPHOID ORGANS. Geetha Chalasani,[1] Bogumila T. Konieczny,[1] Zhenhua Dai,[1] Fadi G. Lakkis.[1] [1]*Renal Division, Emory University & VAMC, Atlanta, GA.*

Allospecific memory T cells are an important impediment to tolerance induction because of their unique ability to respond to donor antigens under much less stringent conditions than naive T cells. Here, we tested the hypothesis that memory but not naive T cells readily mount an effective immune response in the absence of secondary lymphoid tissue. To do so, we transplanted fully vascularized, fully allogeneic BALB/c hearts into B6 alymphoplastic mice (aly/aly) that had undergone splenectomy. These mice lack lymph nodes, mucosal lymphoid tissue, and the spleen and are incapable of rejecting skin or cardiac allografts. Two days after transplantation, 20 million T lymphocytes were adoptively transferred from mice that harbor either naive or memory CD8+ T cells that are allospecific (2C, TCR-transgenic). The memory phenotype was verified by flow analysis (CD44 high, CD62L low, CD25 low, and 1B11 low) and by the ability of the lymphocytes to mount a rapid CTL recall response. Allograft function was monitored daily.

Results: Allograft survival was as follows:

No Adoptive Transfer: > 120 days (n = 6)

Adoptive Transfer of Naive T Cells: > 120 days (n = 6)

Adoptive Transfer of Memory T Cells: 9, 40, 43, & 43 days

Histopathologic analysis revealed no evidence of rejection in the first two groups while grade 3B to 4 rejection was observed in each mouse that received memory T cells. Immunohistochemistry confirmed that the majority of cells infiltrating the rejected grafts were CD3+. Rejection was not observed in splenectomized aly/aly mice even when excess naive T cells were transferred, indicating that memory T cells are qualitatively different from naive T cells.

Conclusion: This is the first experimental proof that memory T cell recall is independent of secondary lymphoid organs. These findings highlight an important difference between memory and naive T cells and underscore the need to develop novel tolerance-induction strategies that inhibit both naive and memory T cell activation.

Abstract# 2

HEME OXYGENASE-1 GENE TRANSFER PREVENTS FAS/FAS LIGAND-INDUCED APOPTOSIS IN VITRO AND IMPROVES ALLOGRAFT FUNCTION IN VIVO. Bibo Ke,[1] Xiu-Da Shen,[1] Judy Melinek,[1] Feng Gao,[1] Thomas Ritter,[2] Hans-Dieter Volk,[2] Roland Buelow,[3] Ronald W. Busuttil,[1] Jerzy W. Kupiec-Weglinski.[1] [1]*Surgery, Dumont-UCLA Transplant Ctr., Los Angeles, CA;* [2]*Humboldt Univ., Berlin, Germany;* [3]*Sangstat, Fremont, CA.*

Background: Apoptosis via Fas/FasL pathway plays an important role in acute allograft rejection. The expression of heme oxygenase (HO-1), an anti-oxidant molecule, may prevent apoptosis and ameliorates the development of chronic rejection. In this study, we analyzed putative HO-1-based cytoprotective mechanisms against apoptosis *in vitro* and following Ad-HO-1 gene transfer in a rat orthotopic liver transplant (OLT) model.

Methods: In *in vitro* cytotoxicity assay, Hela cells were transfected with Ad-FasL + Ad-HO-1/Ad-βGal at multiplicity of infection (MOI) 5, 10, and 20; cytotoxicity was measured by ELISA reader. In OLT model, DA donor livers were perfused *ex-vivo* with $5x10^{10}$ pfu of Ad-HO-1 or Ad-βGal, and transplanted into LEW rats. Animals were sacrificed at 3, 7 and 10 days. OLTs were harvested for histology, and sGOT levels were measured. Intragraft expression of HO-1 and anti-apoptotic (Bcl-xl/Bag-1) genes was assessed by Western blots; cytokine gene expression was analyzed by competitive-template RT-PCR.

Results: Cytotoxicity to Fas-bearing YAC-1 target cells at MOI of 5, 10 and 20, was 39%, 48% and 76%, respectively in Ad-FasL + Ad-βGal group, as compared with 4%, 6% and 14%, respectively in Ad-FasL + Ad-HO-1 group ($p<0.001$). OLTs in Ad-βGal group showed progressive signs of severe acute rejection, with necrosis/hemorrhage, and <25% of hepatic parenchyma viable by day 10. In contrast, OLTs in Ad-HO-1 group exhibited mild to moderate rejection, with >90% of parenchyma preserved. These correlated with decreased sGOT levels in Ad-HO-1 group. The expression of HO-1, Bcl-xl and Bag-1 was enhanced throughout in Ad-HO-1 transduced OLTs, as compared with Ad-βGal controls. Intragraft expression of mRNA coding for IL-2/IFN-γ remained depressed, whereas that of IL-4/IL-10 reciprocally increased in Ad-HO-1 group.

Conclusion: Ad-HO-1 gene transfer prevents Fas/FasL-induced apoptosis *in vitro*, and ameliorates histological signs of acute rejection/improves hepatic function in rat OLT recipients. Local induction of anti-oxidant HO-1 promotes intragraft expression of Th2-dependent protective molecules with anti-apoptotic (Bcl-xl/Bag-1) function. These results provide the rationale for refined novel therapeutic approaches based on new concepts of immunosuppression.

Abstract# 3

REGULATORY ROLE OF CTLA4 AND CYTOKINES IN PHYSIOLOGIC TERMINATION OF ALLOIMMUNE RESPONSES IN VIVO. nader najafian,[1] Masayuki Sho,[1] Akira Yamada,[1] Koji Kishimoto,[1] Victor M. Dong,[1] Sigrid E. Sandner,[1] Mohamed H. Sayegh.[1] [1]*Renal Division, Brigham and Women's Hospital, Boston, MA.*

There are three principal control mechanisms for the physiologic termination of T cell responses after activation by antigens: anergy, cytokine-mediated regulation and apoptosis. While the importance of CTLA4 mediated negative signal in terminating autoimmune responses is well established, its role in terminating physiologic alloimmune responses is unknown. We investigated the role of CTLA4 and cytokines in physiologic rejection processes in vivo. To this end, we used two different heart transplant models: B10-D2 hearts into BALB/C recipients (minor mismatched combination) and C57BL/6 hearts into BALB/C recipients (major mismatched combination). The recipients were either wild type (WT), STAT4 KO (impaired Th1 response) or STAT6 KO (impaired Th2 response) BALB/C mice. Recipients received a blocking anti-CTLA4 mAb to inhibit CTLA4 mediated negative signaling in vivo. Heart allograft survival is summarized as follows (n=4-8/group):

Recipient	MST±SEM	Recipient+anti-CTLA4	MST±SEM	P value
1 WT(minor)	96±13	1a.WT (minor)	10 6±0 4	p<0 0001 vs 1
2.STAT4 KO (minor)	88±21	2a.STAT4KO (minor)	9.8±0.2	p=0 0035 vs 2
3 STAT6 KO (minor)	49±22	3a.STAT6KO (minor)	9.6±0.4	p=0 0078 vs 3
4 WT (major)	7 6±0 4	4a.WT (major)	5 7±0 2	p=0 0001 vs 4
5.STAT4 KO (major)	8 5±0.8	5a.STAT4KO (major)	5 75±0 25	p=0 01 vs 5
6 STAT6 KO (major)	7 2±0 4	6a.STAT6KO (major)	6±0	p=0.023 vs 6

In the major mismatched groups, there was abrupt rejection of allografts regardless of the recipient phenotype.In the minor mismatched groups there was significant prolongation of allograft survival in all recipients. However, graft survival was significantly shorter in STAT6 KO (p=0.03) but not STAT4 KO as compared to WT recipients. Regardless of the cytokine milieu, blocking CTLA4 accelerates the rejection process significantly in all recipients, particularly in the minor mismatched combination where the alloreactive T cell clone size is smaller. This is the first report establishing the negative regulatory functions of CTLA4 signaling pathway in physiologic termination of Th1 and Th2 alloimmune responses in vivo.

Abstract# 4

TYPE III IMMUNE IMMUNE REACTIONS IN XENOTRANSPLANTATION. Yoshihiro Miyata,[1] Christine L. Lau,[2] R. Duane Davis,[2] Jeffrey L. Platt,[1] Zoie E. Holzknecht.[1] [1]*Surgery, Mayo Clinic, Rochester, MN;* [2]*Surgery, Duke University, Rochester, MN.*

Cardiac and renal xenograft rejection occurs when natural antibodies of the recipient bind to and activate complement on the surface of donor endothelium. This sequence of events is typical of a Type II immune reaction and results in cellular damage through the activation of complement. The rejection of pulmonary xenografts, however, occurs in the absence of substantial antibody deposits and unlike cardiac and renal xenografts, is not prevented when complement is depleted from the recipient. In light of these differences, we asked whether a different mechanism of immune reaction might occur in xenotransplanted lungs.

Results: Blood taken from baboons after xenotransplantation of porcine lungs contained immune complexes consisting of baboon IgM and porcine von Willebrand factor. *Immunocomplexed baboon IgM was specific for Galα1-3Gal. Immune* complexes were also seen in baboon blood, albeit to a lesser extent, after xenotransplantation of porcine hearts and kidneys. Immunopathology of pulmonary xenografts revealed a significant decrease of von Willebrand factor in the microvasculature as early as 15 minutes after transplantation and an overall decrease in Galα1-3Gal antigen in the lung. Transplanted hearts and kidneys had a slight decrease in microvascular von Willebrand factor and very little, if any, decrease in Galα1-3Gal antigen. Deposition of porcine von Willebrand factor and baboon C3 was detected in the spleens and livers of pulmonary xenotransplant recipients less than 24 hours after transplantation.

Conclusions: Although Type II immune reactions occur in xenotransplants, these results show, for the first time, that Type III immune reactions, defined as those which result in formation of soluble, circulating immune complexes, occur as well. Moreover, immune complex formation can lead to the deposition of complexes in distant organs. These findings may explain the fate of xenoreactive antibody following pulmonary xenotransplantation and may also constitute a novel complication of xenotransplantation.

PLENARY SESSION II: CLINICAL

Abstract# 5

PREMILINARY RESULTS FROM A HUMAN TOLERANCE TRIAL USING CAMPATH-1H. Allan D. Kirk,[1,2] S. John Swanson,[1,2] Roslyn B. Mannon,[1] D. Scott Batty,[1,2] Wendy Bernstein,[2] Lee Brettman,[3] Chris Chamberlain,[1] Barbara S. DiMercurio,[1] Keith Hunter,[1] Robert Kampen,[1] David Kleiner,[1] Douglas K. Tadaki,[1] David M. Harlan.[1] [1]*NIH/NMRC, Bethesda, MD;* [2]*WRAMC, Washington, DC;* [3]*Millennium, Cambridge, MA.*

Campath-1H is a depleting, humanized Mab specific for CD52. In a recent human renal transplant trial Campath-1H allowed for greatly reduced maintenance immunosuppression. As preoperative T cell depletion has been shown to induce tolerance in primates, we initiated a study to evaluate the toleragenic potential of Campath-1H. Six patients have undergone pretransplant depletion with Campath-1H

followed by living donor renal allografts. Patients have been monitored physiologically, by flow cytometry, and by protocol biopsy including RNA analysis to determine the onset of immune engagement and identify an appropriate time to begin maintenance therapy cognizant of the therapy's potential affect on AICD. All patients received 3 doses of 0.3 mg/kg beginning 3-5 days prior to transplant. Campath-1H was well tolerated by all recipients. Following the first dose of 0.3mg/kg, no peripheral T or B cells were present, and peripheral monocytes were decreased by >95%. Neutrophil or platelet depletion did not occur. Inguinal lymph nodes at surgery showed T cell depletion. Allograft function was immediate and excellent. Protocol biopsies at 1 week revealed no evidence of rejection. Most patients experienced a rise in monocytes (not lymphocytes) during the 3rd week accompanied by a moderate, predominantly monocytic, interstitial infiltrate, elevated intragraft TNF-α, and renal dysfunction. This was initially treated in 2 patients with a steroid taper and conversion to prednisone (5-7.5 mg/day) and sirolimus (trough levels 6-10ng/ml). One patient received a 3-day course of steroids followed by monotherapy sirolimus. Two patients were treated preemptively with monotherapy sirolimus beginning on days 7 and 17. The remaining patient experienced an early recurrence of FSGS requiring steroids and plasmapheresis. All patients have resumed or maintained normal renal function (mean creat 1.35 mg/dl). Six-month biopsies show no evidence of rejection. Lymphocyte repopulation has occurred over 8 months with all TCR V-beta families represented. These data indicate that Campath-1H is a potent, well tolerated depleting agent that allows for at least a 2 week rejection free window post transplant in the absence of any maintenance immunosuppression. While complete tolerance does not occur, immunosuppressive requirements thereafter are minimal.

Abstract# 6

RANDOMIZED CONTROLLED TRIAL OF HAND-ASSISTED LAPAROSCOPIC VERSUS OPEN SURGICAL LIVE DONOR NEPHRECTOMY. J. Stuart Wolf, Jr.,[1] Robert M. Merion,[1] Alan B. Leichtman,[1] Darrell A. Campbell, Jr.,[1] John C. Magee,[1] Jeffery D. Punch,[1] Jeremiah G. Turcotte,[1] John W. Konnak.[1] *[1]University of Michigan, Ann Arbor, MI.*

Introduction and Objectives: Laparoscopic live donor nephrectomy for renal transplantation is being performed in increasing numbers with the goals of broadening organ supply while minimizing pain and duration of convalescence for donors. Relative advantages in terms of recovery provided by laparoscopy over standard open surgery have not been rigorously assessed in a randomized fashion. We hypothesized that laparoscopic as compared to open surgical live donor nephrectomy provides briefer, less intense, and more complete convalescence.

Methods: Of 105 volunteer, adult, potential living-renal donors interested in the laparoscopic approach, 70 were randomized between hand-assisted laparoscopic and open surgical live donor nephrectomy at a single referral center. Objective data, and subjective recovery information obtained with telephone interviews and validated questionnaires administered 2 weeks, 6 weeks, and 6 to 12 months post-operatively, were compared between the 23 laparoscopic and 27 open surgical patients. Primary endpoints were measures of the duration and intensity of donor convalescence.

Results: There was 47% less analgesic use ($p = 0.004$), 35% shorter hospital stay ($p = 0.0001$), 33% more rapid return to non-strenuous activity ($p = 0.006$), 23% sooner return to work ($p = 0.037$), and 73% less pain six weeks post-operatively ($p = 0.004$) in the laparoscopy group. Laparoscopic patients experienced complete recovery sooner ($p = 0.032$) and had fewer long-term residual effects ($p = 0.0015$). Operative time was 65% longer in the laparoscopy group ($p < 0.0001$). Mean hospital cost was 24% greater ($p = 0.0005$) in the laparoscopy group, but global cost, which included estimated loss of occupational income during the recovery period, was only 2% greater ($p = 0.55$).

Conclusions: Laparoscopic donor nephrectomy is associated with a briefer, less intense, and more complete convalescence compared to the open surgical approach. Although operative time and hospital costs are increased, global costs of the procedures are equivalent.

Abstract# 7

INHIBITION OF CROSSMATCH (CMX) POSITIVITY BETWEEN DONOR-RECIPIENT PAIRS USING INTRAVENOUS GAMMAGLOBULIN (IVIG) WITH SUBSEQUENT TRANSPLANTATION. Stanley C. Jordan,[1] Ashley A. Vo,[1] Suphamai Bunnapradist,[1] Dolly Tyan.[1] *[1]Kidney Transplant & Transplant Immunology, Cedars-Sinai Medical Center, L.A., CA.*

Introduction: Sensitization to HLA class I antigens with CMX positivity is a significant immunologic barriers to successful transplantation. Here, we explored the ability of IVIG to eradicate positive CMXs between donor-recipient pairs. Patients & Methods: 15 highly-sensitized patients (PRA >40%) presented for cadaver (CAD)(#5) or living-donor(LD) (#10) transplantation. All were kidney allograft recipients except for 1 who received a combined heart-liver transplant. All had positive CDC and FACS CMXs with their potential donors. For potential LD recipients,in vitro IVIG CMXs were done to determine if IVIG inhibited the CMXs. If so, the potential recipients were given IVIG 2gm/kg and CMXs repeated within 24 hrs. If (-), transplants were performed in 24-48 hrs. A second dose was given in most patients at 1 month post transplant. For CAD transplants, patients were offered organs with whom they had positive CMXs. IVIG 2gm/kg was given 4-5 hrs. prior to transplantation and repeated at 1 month. Mean sCr, AR episodes, graft and patient survival were determined. Results: All LD CMXs were abrogated by IVIG therapy with CMXs post-IVIG becoming negative. These patients were transplanted within 24-48 hrs. Repeat CMXs on CAD recipients after IVIG showed abrogation in most cases. Two patients required dialysis post-transplant. 4/15 patients developed AR episodes at (7, 7, 10 & 49 days) post transplant. Two responded to pulse steroids only and 2 required OKT3 . In addition to IVIG induction, 4 received Zenapax and 1 Thymoglobulin. Mean sCrs were as follows: 1M: 1.36 mg/dl + 0.3; 3M: 1.29 mg/dl + 0.44; 6M: 1.41 mg/dl + 0.60; 12M (#7): 1.67 mg/dl + 0.76 (P=NS). No allografts were lost, 1 patient died of pulmonary edema 1 year post transplant with a functioning graft. Conclusions: 1). IVIG treatment of in vitro CMXs can predict in vivo responses to IVIG. 2). IVIG treatment of highly-HLA sensitized patients who exhibit + CMXs with prospective donors abrogrates these responses and allows for successful transplantation. 3). AR episodes occurred in 26% of IVIG treated patients and responded to standard therapy. Excellent allograft function was noted in all but one patient. 4). Although the exact mechanism(s) responsible for the observed beneficial effects are not know, they most likely relate to IVIG's inhibition of anti-HLA antibodies and subsequent deletion of alloreactive B-cells.

Abstract# 8

TEN YEAR EXPERIENCE WITH INTESTINAL TRANSPLANTATION. A. Langnas,[1] S. Chinnakotla,[1] D. Sudan,[1] S. Horslen,[1] B. Shaw,[1] K. Iyer,[1] I. Fox.[1] *[1]Department of Surgery, University of Nebraska, Omaha, NE.*

In 1990 we initiated an Intestinal Transplant Program at our institution. 220 patients have been listed for intestinal transplant with 76 deaths on the waiting list. 103 intestinal transplants (38 isolated small bowel (SB) and 65 liver/small bowel (L/SB))were performed in 11 adults and 92 children. The mean age of the adult and pediatric recipients was 35 and 3.7 years. The indication for transplantation for L/SB recipients was decompensated liver disease due to TPN in all but 1 patient(biliary atresia). For recipients of isolated SB transplants the indications were potentially reversible TPN liver disease, loss of vascular access and sepsis. The median age of the donors was 0.3 years for recipients of L/SB transplants and 2.1 years for recipients of isolated SB transplants. The mean donor recipient weight ratio was 0.9 (range 0.45 - 2.1) for recipients of L/SB transplant and 0.6 (range 0.43 - 0.92) for recipients of isolated SB transplants. The mean cold ischemia time was 9.8 hours (range 7 - 16 hours). The transplant procedures were modified over the 10 years. The L/SB procedure is now an en-bloc technique incorporating the liver, duodenum, head of pancreas and SB. No colon or stomach has been transplanted. 1 reduced size L/SB procedure was performed. Immune suppression consisted of Cyclosporine in the first 5 transplants and Tacrolimus in the remaining. Simulect was used routinely in the last 14 consecutive intestinal transplants. Sirolimus was used in 11pts. Results:The first 5 recipients initially received Cyclosporine, one of these patients is still alive (9 years). In the 87 patients who received primary transplantation under Tacrolimus therapy, the 2-year actuarial survival was 63%. The 2-year survival for recipients for SB transplants was 79% and for L/SB recipients 52%. The 2-year graft survival for recipients of isolated SB transplants was 68%. 5 patients who had undergone prior transplantation then underwent L/SB transplant of whom only 1 is alive (5 years). Rejection episodes occurred in 63% of L/SB recipients and 74% of isolated SB recipients. Chronic rejection was diagnosed in 3 pts who underwent isolated SB transplantation. PTLD occurred in 7 L/SB and 3 isolated SB transplant recipients. GVHD was diagnosed in 2 of the L/SB recipient patients. Summary:Intestinal transplantation remains a difficult clinical endeavor; however the results of this experience continue to improve as we gain a better understanding of the surgical, immune suppression and post-operative management.

CONCURRENT SESSION 1:
CYTOKINE REGULATION OF ALLOIMMUNE RESPONSES

Abstract# 9

ABSENCE OF BOTH TUMOR NECROSIS FACTOR RECEPTORS P55 AND P75 ON DONOR HEARTS DIMINISHES GRAFT ARTERIAL DISEASE ALTHOUGH P55 OR P75 SINGLE DEFICIENCY DOES NOT. Jun-ichi Suzuki, Sarah E. Cole, Peter Libby, Richard N. Mitchell. [1]*Vascular Medicine and Atherosclerosis Unit, Brigham and Women's Hospital, Harvard Medical School, Boston, MA.*

Background. Graft arterial disease (GAD) remains the leading cause of allograft failure in organ transplantation. Tumor necrosis factor (TNF) promotes organ rejection; the activities of TNF are mediated by two functionally distinct receptors, p55 and p75. Major biological responses are mediated by p55; p75 has been proposed to both mediate and antagonize the inflammatory effects. Methods. To test hypotheses that TNF receptor deficiency can attenuate GAD, heterotopic cardiac transplantation was performed using TNF receptor p55 and p75 double deficient (TNFRDKO) mice and p55 or p75 single deficient mice. C57BL/6 (B/6) WT or KO mice and B6.C-H2bm12/KhEg (Bm12) WT mice (class II mismatch) were used to assess the effects of TNF receptor deficiency on GAD (score: 0:none to 4:most severe) 8 weeks after transplant. Cytokine expression in the allografts was analyzed using RNase protection assay. Results. Severe GAD occurred in WT combinations (B/6 WT to Bm12 WT mice, n= 9; GAD= 2.3±1.6; Bm12 WT to B/6 WT mice, n=8; GAD= 2.4±1.4) or when p55 or p75 single deficient hearts were used as donors. However, B/6 TNFRDKO hearts in Bm12 WT hosts exhibited significantly diminished GAD (n=8; GAD= 1.4±1.1, P<0.05), while Bm12 WT hearts into B/6 TNFRDKO recipients did not (n=8; GAD= 2.6±1.0). Interferon-gamma expression was markedly reduced when TNFDKO hearts were used as donors. Conclusion. The absence of both TNF receptors in donor hearts attenuates GAD and is associated with diminished interferon-gamma expression. Single receptor deficiency did not diminish GAD or modulate interferon-gamma expression. Thus, both p55 and p75 signals on donor organs are involved in the development of the allograft arteriopathy.

Abstract# 10

FAILURE TO INDUCE NEONATAL TOLERANCE IN MICE THAT LACK BOTH IL-4 AND IL-13 BUT NOT IN THOSE THAT LACK IL-4 ALONE. Yoshihiko Inoue, Maylene E. Wagener, Bogumila T. Konieczny, Fadi G. Lakkis. [1]*Renal Division, Emory University & VAMC, Atlanta, GA.*

Although it is generally accepted that neonatal tolerance to a foreign antigen is the consequence of T helper type 2 (Th2) immunity, it is uncertain whether IL-4 plays a dominant role in the tolerance process because another cytokine, IL-13, also contributes to the development of a Th2 response. Here, we addressed this uncertainty by testing whether tolerance to the male H-Y antigen can be induced in newborn mice that lack IL-4 (IL-4-/-) or lack both IL-4 and IL-13 (IL-4/IL-13-/-).

Methods: We injected newborn female wild-type (wt), IL-4-/-, and IL-4/IL-13-/- mice i.p. with 50 million syngeneic male spleen cells. Control mice did not receive any i.p. injections. Four weeks later, full-thickness syngeneic male skin grafts were transplanted to all mice. Tolerance was deemed to be present if female mice accepted the first skin graft (> 80 days), failed to reject a second syngeneic male graft but rejected third party skin, and failed to mount a CTL response upon rechallenge with syngeneic male splenocytes. Mouse strains were as follows: wt (B6 or BALB/c), IL-4-/- (B6), and IL-4/IL-13-/- (BALB/c).

Results: Control female wt, IL-4-/-, and IL-4/IL-13-/- mice that did not receive neonatal splenocyte injection rejected their skin grafts promptly (median graft survival = 24, 20, and 16 days, respectively; n = 5/group). Neonatal splenocyte injection lead to immunologic tolerance as defined above in female B6 wt and IL-4-/- mice (n = 10/group), indicating that IL-4 is not essential for tolerance induction. In contrast, neonatal tolerance could not be achieved in any of the female BALB/c IL-4/IL-13-/- mice studied (median graft survival = 24 days; n = 5) although it was uniformly induced in female BALB/c wt mice (n = 5).

Conclusion: IL-4 alone does not play a dominant role in the induction of neonatal tolerance to a minor histocompatibility antigen. Instead, the findings indicate that cooperation between IL-4 and IL-13 is responsible for this form of immunologic tolerance.

Abstract# 11

THE CONTRIBUTION OF IFN-γ TO ISLET ALLOGRAFT SURVIVAL FOLLOWING ANTI-CD4 THERAPY. Alexander C. Wiseman, Mark R. Nicolls, Andrew S. Diamond, Josh Beilke, Ron G. Gill. [1]*Divisions of Nephrology, Pulmonary, and Immunology, University of Colorado Health Sciences Center, Denver, CO.*

We have previously demonstrated that acute islet allograft rejection is a CD4-mediated process that can be abrogated with anti-CD4 therapy. While traditionally regarded as a pro-inflammatory cytokine, IFN-γ has been shown to act in a regulatory fashion in some models of allograft tolerance. To determine if the efficacy of anti-CD4 therapy in islet transplantation is dependent upon IFN-γ, we transplanted islets into allogeneic streptozotocin-induced diabetic recipients deficient in either IFN-γ (BALB/c IFN-γ$^{-/-}$ C57Bl/6 IFN-γ$^{-/-}$"GKO") or the IFN-γ receptor (129 IFN-γR$^{-/-}$"GRKO") and compared allograft survival with BALB/c, C57Bl/6, and 129 wild-type (WT) controls. Recipients were given anti-CD4 mAb (GK1.5) on days -1, 0, 1, and 2 relative to transplantation.

Recipient	Survival (days)	Mean +/- S.E.M.	p value
C57Bl/6 WT	28, 33, 33, 40, 41, 43, 49, 55	40 3 +/- 3.2	
C57Bl/6 GKO	13 x 2, 14, 15, 17 x 3, 20, 21 x 2	16 8 +/- 1 0	<.0001
BALB/c WT	26, 42, 42, 43, 46, >100 x 5	69 9 +/- 10 2	
BALB/c GKO	8, 12, 13, 14 x 3, 27, 30, 36, 43, 46 x 2	25 3 +/- 4 2	003
129 WT	41, 47 x 2, 48, 49, 58, 79, >100	58 6 +/- 7 2	
129 GRKO	26, 28, 31, 32, 41, 42	33 3 +/- 2 7	001

Absence of host IFN-g or IFN-g receptors significantly diminished the efficacy of anti-CD4 therapy in prolonging islet allograft survival. Importantly, IFN-g production by 129 GRKO mice was similar to wild-type controls. This suggests that, since graft survival in the GRKO recipient is diminished despite intact IFN-g production and islet allograft responsiveness, the protective effect of IFN-g is independent of its effects upon the allograft itself. The novel finding that either IFN-g deficiency or IFN-g receptor deficiency in the recipient leads to resistance to anti-CD4 therapy suggests that IFN-g is a critical regulatory cytokine that facilitates anti-CD4-mediated islet allograft prolongation

Abstract# 12

NEUTROPHILS ACCELERATE THE REJECTION OF FULLY MHC-DISPARATE CARDIAC ALLOGRAFTS IN THE ABSENCE OF INTERFERON-γ. Masayoshi Miura,[1] Qiwei Zhang,[1] Robert L. Fairchild.[1] [1]*Urological Institute, Cleveland Clinic Foundation, Cleveland, OH.*

OBJECTIVES: We have previously shown that interferon-g (IFN-g) is critical for the optimal recruitment of T cells into class II disparate skin grafts. In contrast, the absence of IFN-g is known to result in more rapid rejection of MHC-mismatched heart grafts. The goal of this study was to examine potential mechanisms mediating the rapid rejection of cardiac allografts in the absence of interferon-g.

METHODS: A/J (H-2a) heart grafts were heterotopically transplanted into wild-type C57BL/6 (H-2b) or C57BL/6.IFN-g-/- (GKO) recipients. A group of recipients received 100 ug aliquots of anti-Ly6G mAb (RB6-8C5) at days -2, -1, and +2. Control recipients received rat IgG. Allografts were harvested at 6h, days 1, 2, 3, 4, 5, 6, 7 and 8 post-transplant for RNA and histological analyses. Frozen sections of allografts were stained with anti-Ly6G, anti-CD4 (GK1.5) or anti-CD8 (53-6.7) mAbs for immunohistochemical analyses. Temporal expression of chemokine genes in allografts was tested by RNase protection assay.

RESULTS: WT C57BL/6 recipients rejected A/J cardiac allografts at day 8-9 post-transplant. In contrast, GKO recipients rejected the allografts at day 6 post-transplant. This rapid rejection was associated with intense thrombosis and hemorrhagic infarction. Allografts from GKO recipients also had a 600% increase in the number of infiltrating neutrophils when compared to WT at the time of allograft rejection for each recipient. In contrast, infiltration with CD8+ T cells in allografts from GKO recipients was significantly decreased to 20% that observed in allografts from the WT. Numbers of infiltrating CD4+ T cells was low but equivalent in allografts from both recipient groups. In GKO recipients, intra-allograft expression of Mig was undetectable, expression of IP-10 and RANTES was very low and expression of MIP-1a, MIP-1b, MIP-2 and MCP-1 was also decreased compared to WT. Lymphotactin, KC and GCP-2 were at equivalent levels. Treatment with anti-Ly6G mAb restored allograft survival in GKO recipients to day 8-9 post-transplant.

CONCLUSIONS: IFN-g is necessary for optimal recruitment of T cells into fully MHC-disparate cardiac allografts. During the acute rejection process, IFN-g attenuates neutrophil infiltration into cardiac allografts and neutrophil mediated tissue damage.

Abstract# 13

THE ROLE OF TH1 CYTOKINES IN TOLERANCE REVISITED: EFFECT OF T CELL CLONE SIZE. Koji Kishimoto,[1] Victor M. Dong,[1] Nader Najafian,[1] Terry B. Strom,[2] Laurence A. Turka,[3] Mohamed H. Sayegh.[1] [1]*Medicine, Brigham and Women's Hospital, Boston, MA;* [2]*Medicine, Beth Israel Deaconess Medical Center, Boston, MA;* [3]*Medicine, University of Pennsylvania, Philadelphia, PA.*

Recent studies demonstrated that the Th1 cytokine IFN-γ is necessary for tolerance induction by CD28-B7 T cell costimulatory blockade in MHC-mismatched donor/recipient combinations. However, it is not known whether this is true for tolerance across minor histocompatibility barriers. We hypothesized that while the anti-proliferative action of IFN-γ may be necessary for induction of tolerance in conditions of high alloreactive T cell clone size (MHC mismatched combination), IFN-γ is not necessary for tolerance when the alloreactive T cell clone size is relatively small (minor mismatched combination). Indeed, flow cytometry studies of CFSE labeled lymphocytes responding to donor antigen in vivo show that the frequency of alloreactive T cells is 22% versus 3.5% against major and minor mismatch stimulators, respectively. In order to test our hypothesis IFN-γ KO or wild type (WT) C57BL/6 mice were used as recipients of BALB/c (MHC mismatch) or 129 (minor mismatch) heart grafts. Recipients were treated with either CTLA4Ig (to block CD28-B7) or MR1 (anti-CD154 mAb) on day 0, 2, 4, and 6 posttransplant. Graft survival was as follows.

Group	Donor	Recipient	Treatment	Graft survival (days)	Group	Donor	Recipient	Treatment	Graft survival (days)
1	BALB/c	WT	None	6 00± 32	7	129	WT	None	9 33± 99
2	BALB/c	WT	CTLA4Ig	>99 00±1 00	8	129	WT	CTLA4Ig	>100
3	BALB/c	WT	MR1	>100	9	129	WT	MR1	>100 (NS vs group 8)
4	BALB/c	IFN-γ KO	None	6 50± 29	10	129	IFN-γ KO	None	>24.43±12 64 (p= 0367 vs group 7)
5	BALB/c	IFN-γ KO	CTLA4Ig	12.50±.50	11	129	IFN-γ KO	CTLA4Ig	>100 (NS vs group 8)
6	BALB/c	IFN-γ KO	MR1	10 25±1 49	12	129	IFN-γ KO	MR1	>66 29±10.21 NS vs group 10, (p= 0211 vs group 11)

Our data show that in IFN-γ KO recipients, while both CTLA4Ig and MR1 could not prevent acute rejection in MHC mismatched combination (groups 4, 5, 6), both costimulatory blockade strategies prevented acute rejection in minor mismatched combination (groups 10, 11, 12). Moreover, in minor mismatched combination CD154 blockade was not as effective as CTLA4Ig in IFN-γ KO (groups 11 and 12). These studies indicate that the requirement of IFN-γ for tolerance induction is not universal; both the alloreactive T cell clone size as well as the tolerance strategy itself are important determinants of this requirement.

Abstract# 14

CYTOKINE EXPRESSION BY DENDRITIC CELLS DRIVES DIFFERENTIATAION OF VARIOUS T CELL SUBSETS.

Lianfu Wang, Xiaoyan Liang, Shiguang Qian, C. Andrew Bonham, John J. Fung, Lina Lu. [1]*Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, Pittsburgh, PA.*

T cell differentiation is critical for immune responses. It is postulated that cytokines secreted by adjacent cells drive T cells toward a particular differentiation pathway. Reports by our laboratory and others have demonstrated that DC derived from different cell lineages and under the influence of local cytokine microenvironment differentially direct polarization of T cells. We have described three subsets of mouse DC that are propagated from: 1) bone marrow (BM) in response to GM-CSF+IL-4 (mature myeloid DC), 2) liver nonparenchymal cells (NPC) in GM-CSF with immature phenotype (immature myeloid DC), and 3) liver NPC in IL-3 and CD40L that are derived from the B cell lineage ($B220^+$ lymphoid DC). The aim of this study was to examine the roles of cytokines produced by these DC in determining T cell differentiation. The production and mRNA expression of cytokines by B10 ($H2^b$) DC with or without stimulation by LPS, or by C3H ($H2^k$) spleen T cells stimulated by irradiated DC at S/R ratio of 1/10 for three days, were determined by ELISA and RNase protection assays, respectively. DC not stimulated by LPS produced low levels of cytokines. After LPS stimulation, mature BM-derived myeloid DC secreted large amounts of IL-12 (p70), TNF-α, IL-10 and nitric oxide (NO), and moderate amounts of IFN-γ, which were largely correlated with mRNA expression, thus both IL-12 p35 and p40 could readily be detected. Mature myeloid DC promote Th1 differentiation characterized by secretion of IL-2 and IFN-γ, but no IL-4. Immature liver-derived myeloid DC expressed mRNA for IL-1α, TGF-β and IL-6, moderately for IL-5, IL-9, IL-13, with little message fe for IL-12 (both p35 and p40). They stimulated T cells that mainly released TGF-β, suggesting Th3 polarization. Interestingly, liver $B220^+$ lymphoid DC produced large amounts of IFN-γ and IL-10, and minimal for TNF-α, IL-12 and NO. They expressed mRNA for IFN-γ, IL-10 and IL-12 p35, but not for IL-12 p40. T cells cultured with these cells synthesized IL-10, IFN-γ and TGF-β, but no IL-2 and IL-4, a cytokine profile consistent with T regulatory 1 (Tr1) cells. These data suggested that cytokins produced by different DC subsets upon activation are important in regulating T cell differentiation. IL-12 produced by mature myeloid DC appears to be important in determining Th1 polarization. Production of IL-10 instead of IL-12 in DC may favor generation of Tr1 cells.

Abstract# 15

IL-10 ENHANCES CCR5 BUT DOWN-REGULATES CCR7 EXPRESSION BY MYELOID DENDRITIC CELLS: IMPACT ON CHEMOTACTIC AND IN VIVO HOMING RESPONSES.

Takuya Takayama,[1] Nobuyuki Onai,[2] Motohiro Hirano,[2] Kouji Matsushima,[2] Adrian E. Morelli,[1] Hideaki Tahara,[2] Angus W. Thomson.[1] [1]*Department of Surgery, Transplant Institute, University of Pittsburgh, Pittsburgh, PA;* [2]*Department of Surgery and Bioengineering, University of Tokyo, Tokyo, Japan.*

IL-10 inhibits the phenotypic and functional maturation of dendritic cells (DC) and has been reported to confer tolerogenic properties on these important professional antigen-presenting cells (APC). Purpose: To ascertain the influence of mammalian (m) and viral (v) IL-10 on CC chemokine receptor (CCR) expression by myeloid DC and its impact on their migratory properties, both in vitro and in vivo. Methods: Murine bone marrow-derived myeloid DC were exposed to either mouse (m) or viral (v) IL-10 during their in vitro generation in response to GM-CSF+IL-4. CCR mRNA expression was determined by reverse transcriptase polymerase chain reaction, while chemotactic responses to recombinant mouse CC chemokines were determined in transwell plates. In vivo migration of fluorochrome (PKH-26)-labeled DC was assessed after their s.c. injection and detection in draining lymphoid tissue cell suspensions. Retroviral transduction of DC to overexpress CCR7 was by the centrifugal enhancement method. Results: Both m and vIL-10 downregulated the expression of CCR7 mRNA, while mIL-10 upregulated CCR5 transcripts. These changes in CCR7 and CCR5 expression were associated with inhibition and augmentation respectively, of DC chemotaxis toward their respective agonists, macrophage inflammatory protein (MIP)-3β and MIP-1α. At the same time, the in vivo migration of IL-10 treated DC from peripheral sites to draining lymph nodes of allogeneic recipients was significantly impaired. Anti-mIL-10R mAb reversed the effects of mIL-10 on CCR expression, and restored DC homing ability. Retroviral transduction of m and vIL-10-treated DC to overexpress transgenic CCR7 partially restored the cells' lymphoid tissue homing ability in allogeneic recipients. CCR7 gene transfer did not, however, reinstate the capacity of IL-10-treated DC to prime host naive T cells for ex vivo proliferative responses or T helper (Th) 1 cytokine (IFNγ) production in response to rechallenge with (donor) alloAg. Conclusions: These findings suggest that, in addition to their capacity to subvert DC maturation/function and confer tolerogenic potential on these important APC, mIL-10 and vIL-10 reversibly regulate DC migratory responses via modulation of CCR expression.

Abstract# 16

SPONTANEOUS DEVELOPMENT OF TGFβ-REGULATED ALLOIMMUNITY BY MURINE RENAL ALLOGRAFT RECIPIENTS.

J. J. Wang,[1] M. E. Wakely,[1] A. A. Bickerstaff,[1] C. G. Orosz.[1] [1]*Surgery, The Ohio State University, Columbus, OH.*

DBA/2 ($H\text{-}2^d$) kidneys transplanted into nephrectomized C57BL/6 ($H\text{-}2^b$) mice are spontaneously accepted for more than 60 days without immunosuppression. In contrast, non-immunosuppressed cardiac allografts in the same strain combination are spontaneously rejected within 7-10 days. Transient treatment of cardiac allograft recipients with anti-CD4 mAb, or gallium nitrate permits prolonged cardiac allograft acceptance that is associated with the production of DBA/2-reactive alloantibodies and the expression of DBA/2-reactive DTH responses that are counter-regulated by both TGFβ and IL10 (DTH responses restored by co-localized TGFβ- or IL10-reactive antibodies). At 60 days post-transplant, the recipients of accepted renal allografts similarly display DBA/2-reactive alloantibodies and DBA/2-reactive DTH responses that are counter-regulated by TGFβ, but not by IL10. Furthermore, at 60 days post-transplant, the accepted cardiac allografts display histologically-detectable TGFβ and IL10 throughout the graft interstitium. Naive, non-transplanted hearts do not display these bioactive mediators. In contrast, naive, non-transplanted kidneys constitutively display not only histologically detectable TGFβ, but also high levels of the TGFβ activators urokinase plasminogen activator (uPA) and thrombospondin. The accepted renal allografts continue to display these agents. These data demonstrate that 1) both renal and cardiac allograft acceptance is associated with the development of TGFβ-mediated immune regulation of donor-reactive cell-mediated immunity, 2) unlike hearts, kidneys appear to be predisposed to TGFβ-mediated immune regulation, which may allow their acceptance by allogeneic recipients in the absence of immunosuppression.

Abstract# 17

EXPRESSION OF IL-10 IN THE LIVER ALLOGRAFTS IN A COMPOSITE AUXILIARY PARTIAL LIVER/SMALL BOWEL TRANSPLANTATION MODEL.

Saiho Ko,[1] Yoshiyuki Nakajima,[1] Hiromichi Kanehiro,[1] Hideki Kanokogi,[1] Tetsuhiro Kanamura,[1] Michiyoshi Hisanaga,[1] Yukio Aomatsu,[1] Mitsuo Nagao,[1] Naoya Ikeda,[1] Yasuyuki Urizono,[1] Tsunehiro Kobayashi,[1] Takamune Shibaji,[1] Sanehito Ogawa,[1] Hiroshige Nakano.[1] [1]*First Department of Surgery, Nara Medical University, Kashihara, Nara, Japan.*

Liver allografts are uniquely capable of inducing tolerance, while small bowel allografts are strongly immunogeneic. We have established a model of composite auxiliary partial liver/small bowel transplantation, and evaluated the tolerogeneic effect of the liver allografts on rejection of composite small bowel allografts. **Methods.** Allogeneic transplantation from BN ($RT1^n$) donors to LEW ($RT1^l$) recipients was performed. It has been known that the isolated small bowel allograft induces severe acute rejection, while the liver allograft induces spontaneous long-term acceptance in this strain combination. En-bloc auxiliary composite partial (30 %) liver/small bowel allografts were harvested from the donors and transplanted to the abdominal aorta and IVC of the recipients heterotopically using microsurgical techniques (group A, n=6). As controls, isolated small bowel transplantation was performed (group B, n=5). Histologic scores of small bowel allograft rejection on day 7 were compared between the groups. Expressions of IFN-gamma and IL-10 mRNA in the liver /small bowel allografts and spleen were evaluated by RT-PCR. **Results.** While severe histologic signs of rejection were observed on day 7 in the group B, histology of the small bowel and partial liver allografts in group A showed no or minimal signs of rejection at the same time. Two recipients in the group A were observed for long-term, and their allografts survived for more than 100 days. RT-PCR study showed the strong expressions of both IFN-gamma and IL-10 in the small bowel allografts in the group B. Composite partial liver allografts in group A showed strong expression of IL-10 without showing IFN-gamma expression. The spleen showed positive signals of IFN-gamma and IL-10 expression in both groups. **Conclusion.** Heterotopic auxiliary partial liver allografts prevented the acute rejection of composite small bowel allografts. Selective expression of IL-10 in the liver allografts may play a crucial role in the tolerogeneic effect of the liver in this transplantation model.

CONCURRENT SESSION 2:
COMPLICATIONS OF RENAL TRANSPLANTATION

Abstract# 18

PHARMACOKINETIC INTERACTION OF CHLORAMPHENICOL WITH CALCINEURIN INHIBITORS. A. Scott Mathis,[2] Nita Shah,[1] Gary S. Friedman,[1] Jenivieve Villadolid,[2] Kathryn Kalafatas.[2] *[1]Transplant Department, Saint Barnabas Medical Center, Livingston, NJ; [2]College of Pharmacy, Rutgers, The State University of New Jersey, Piscataway, NJ.*

Introduction: With the emergence of multi-drug resistant organisms, chloramphenicol has reemerged into clinical practice. Infections in transplant recipients may be difficult to manage due to drug interactions and additive toxicity of immunosuppressive agents and anti-infective agents. Several case reports have described a pharmacokinetic interaction between chloramphenicol and calcineurin (CNI) inhibitors. We undertook this evaluation to characterize and compare the effects of chloramphenicol on cyclosporine and tacrolimus trough concentrations (TC).

Methods&Results: 6 adult renal or pancreas/kidney transplant recipients received 11 courses of chloramphenicol for the management of E. faecium (n=5) or Staph Epi (n=1) infections [peritoneal cavity (n=3), wound (n=2), and blood (n=1)]. We retrospectively evaluated daily CNI TC and compared them with baseline CNI TC prior to initiating chloramphenicol. Data reported are mean ± standard deviation (SD) or percent change. Of the 6 patients receiving chloramphenicol, 3 received cyclosporine (6 episodes) and 3 received tacrolimus (5 episodes). In both groups, mean duration of chloramphenicol treatment was 12 days, and patient age, weight, height and sex were similar. Chloramphenicol daily dose ranged from 2333 ± 516 to 2667 ± 577 (mean 2475 ± 504) mg/day in the cyclosporine group, and 2167 ± 764 to 2750 ± 500 (mean 2585 ± 601) mg/day in the tacrolimus group (P=NS). Mean cyclosporine TC were increased maximally to 41.3% above baseline on day 4 (P=NS). On day 10, mean cyclosporine TC were 30.9% lower than baseline (P=0.044), reaching maximal decreases of 47.4% below baseline values on day 12 (P=0.025) despite no significant change in mean daily cyclosporine dose from baseline. In contrast, tacrolimus TC increased to 99% above baseline on day 2 (P=0.023), 151% on day 3 (P=0.006), 161% on day 4 (P=0.028), 191% on day 5 (P=0.103), and to 207% on day 6 (P=0.034). No significant change was observed in mean daily tacrolimus dose from baseline.

Conclusions: This is the largest systematic evaluation of an interaction of chloramphenicol with the CNIs. Although TC fluctuations with both CNIs were observed, tacrolimus TC appears to be more significantly increased. Careful TC monitoring should be instituted if chloramphenicol is to be used with the CNIs, as it appears that the patient may be at risk for fluctuations in CNI levels.

Abstract# 19

LOSARTAN DRAMATICALLY REDUCES MASSIVE PROTEINURIA AFTER RENAL TRANSPLANTATION: A MULTICENTRIC AND PROSPECTIVE STUDY. Beatriz Dominguez-Gil,[1] Pablo Iñigo,[2] Milagros Ortiz,[1] Maria P. Sierra,[1] Fernando Anaya,[3] D. del Castillo,[4] Amado Andres,[1] Jose M. Campistol,[2] Jose M. Morales.[1] *[1]Nephrology, 12 de Octubre Hospital, Madrid, Madrid, Spain; [2]Renal Transplant Unit, Clinic Hospital, Barcelona, Barcelona, Spain; [3]Nephrology, Gregorio Marañón Hospital, Madrid, Madrid, Spain; [4]Nephrology Service, Reina Sofia Hospital, Cordoba, Spain.*

It has been recently published that Losartan reduces proteinuria in native and transplanted kidneys. However, there is no information about its efficacy in patients with massive proteiuria. We present an open, multicentric and prospective study evaluating the efficacy of Losartan in the treatment of nephrotic range proteinuria in kidney transplant patients. We included 34 patients (Age 43.48 ± 14.1; 67.6% males, 29.4% females) who had developed a nephrotic range proteinuria (>=3.5 g/day) from different etiologies (including chronic allograft nephropathy, de novo or recurrent glomerulonephritis, transplant glomerulopathy). Treatment with Losartan was started after renal artery stenosis of the graft was excluded by duplex-ultrasonography.

	Initial	Final	p
Proteinuria(g/day)	6 33 ± 3 89	2 44 ± 2 2	<0 0005
Serum Creatinine (mg/dl)	1 82 ± 0 85	1 92 ± 1	0 07
MAP (mmHg)	106 9 ± 11 05	102 4 ± 9 06	0 1

MAP: Mean arterial pressure

After a mean follow-up period of 7.97 ± 5.49 months, proteinuria decreased in 31 (91%) patients, being this reduction significative (>=50%) in 25 of them. At the end of the study, medium dose of Losartan was 60 mg. None of the patients developed hyperkalemia. In conclusion, Losartan significantly reduces nephrotic range proteinuria in kidney transplant patients with an adequate tolerance in terms of maintenance of a stable renal function and without a significative change in blood pressure levels, a factor that could otherwise explain the reduction in proteinuria.

Abstract# 20

HETEROGENEITY OF BONE HISTOLOGY IN HYPERCALCEMIC KIDNEY TRANSPLANT RECIPIENTS: DIAGNOSTIC VALUES OF BIOCHEMICAL MARKERS OF BONE REMODELING. Marie Lafage-Proust,[2] Lionel Rostaing,[1] Jean Cisterne,[1] Pierre Bories,[1] Anne Caillot-Augusseau,[2] Dominique Durand.[1] *[1]Nephrology, CHU, Toulouse, France; [2]Bone Biology, INSERM 9901, Saint-Etienne, France.*

Hypercalcemia (HCa) occurs in about 10% of renal transplanted patients. Bone lesions associated with HCa remain unknown. The aim of our study was to determine the etiology of sustained HCa in patients after renal transplantation and evaluate the diagnostic value of biochemical markers of bone remodeling. We analyzed bone iliac crest biopsies (BB) from 18 patients (pts)(mean age: 44±11) who had undergone renal transplantation (RT) and remained hypercalcemic (serum calcium at the time of BB: 2.79±0.21mmol/l) at least 3 months after RT (mean duration between TR and BB: 28±-30 months) Mean creatinine clearance was 82±22 ml/min. Beside intact PTH, the following bone biochemical markers were measured: Bone alkaline phosphatase (BAP, µg/l)), N-terminal fragment of type I Procollagen (P1NP, µg/l)), intact osteocalcin (Oc, ng/ml) for evaluating bone formation and urinary deoxypyridinoline (D-pyr, mMol /mMol creat), serum(s,10^3 x pmol/l) and urinary (u, µg /mMol creat) C-terminal fragment of type I collagen (CTX) for evaluating bone resorption.

Bone histomorphometry found 9 pts with pure secondary hyperparathyroidism (HPT), 4 with adynamic bone disease (ABD) and 5 with a mineralization impairment (MI: Osteoid Volume >5%, osteoid thickness >12µm and mineralization lag time >50days) including 4 mixed osteopathy and one osteomalacia. Results are expressed as mean ±SD, Kruskall-Wallis statistical analysis.* vs HPT, °vs MI. PTH was 121±31 pg/ml in HPT, 132±56 in MI and 50±39 *, ° in ABD. Biochemical markers of bone remodeling: cf table. Serum CTX and BAP correlated best with Bone Formation Rate BFR/BS (r= 0.78, p<0.0002 and r=0.66, p<0.01, respectively) whereas PTH did not.

In conclusion, hypercalcemic renal transplant recipients exhibited a wide range of bone histologic lesions that were not predicited by 1-84 intact PTH levels. Serum CTX and BAP best correlated with bone turn-over, however accurate prediction of mineralization impairment could not be obtained with biochemical markers.

	Oc	P1NP	BAP	D-Pyr	uCTX	sCTX
HPT	36 5±22 1	208±128	38±14	111 3±5 9	641±531	14 9±3 5
ABD	11 2±4 3°°	47±42°°	17±6°°	4 5±0 8°°	186±34	3 8±.4°°
MI	36 8±16 8	216±179	42±39	10 9±5 7	450±592	13 5±7 3

Abstract# 21

LYMPHOCELES AFTER KIDNEY TRANSPLANTATION - WHAT'S THE BEST APPROACH FOR TREATMENT? Brian Grubbs,[1] Dan Zapzalka,[1] David Hunter,[1] Arthur Matas,[1] Abhi Humar.[1] *[1]Surgery, University of Minnesota, Minneapolis, MN.*

Background: Lymphoceles occurring post kidney transplant may account for significant morbidity. Lymphoceles are usually diagnosed during the work up for posttransplant renal dysfunction. Several treatment options are available in the management of these and we sought to find the most efficacious.

Results: Between January 1984 through January 1997, 122 lymphoceles were diagnosed in 2,601 kidney transplant recipients (incidence = 4.7%). Mean time to diagnosis was 78 ± 94 days following transplantation. The most frequent clinical manifestation was a rising creatinine (52%). Other presenting symptoms were ipsilateral leg swelling (26%), abdominal swelling (15%), wound drainage (10%), and local pain (7%). The first treatment intervention was as follows: simple aspiration (19%), drain placement with or without sclerosis (45%), surgical drainage (open or laparoscopic) with peritoneal window (27%), and other (9%). Success rates with the initial procedure were as follows: simple aspiration (30%), drain placement (38%), surgical drainage (55%). Of the 55 lymphoceles treated with drain placement, 34 also had injection of a sclerosing agent. If sclerosis was done, success rates for drain placement increased to 47%. When sclerosis was attempted, there was an average of 3.5 sclerosis performed on each patient. Drains were left in lymphocele cavities an average of 21 ±20 days. 73 lymphoceles recurred after the initial treatment, necessitating a second procedure. Surgery was the most common second procedure and was successful in 74% of cases. 31 patients required a third intervention, and 7 required 4 or more interventions to eventually resolve the lymphocele.

Conclusion: Lymphoceles are diagnosed in about 5% of patients following kidney transplantation. They may require more than 1 intervention to resolve. While surgical drainage had the highest success rate, drain placement followed by sclerosis was effective in almost 50% of cases. This should likely be the first intervention for most patients; a surgical drainage procedure should be performed if the lymphocele has not resolved by 3 attempts at sclerosis.

Abstract# 22

CARDIOVASCULAR (CV) RISK PROFILE IN RENAL ALLOGRAFT RECIPIENTS WITH *DE NOVO* POST-TRANSPLANT DIABETES (PTDM). Fernando G. Cosio,[1] Todd E. Pesavento,[1] Kwame Osei,[3] Mitchell L. Henry,[2] Ronald M. Ferguson.[2] *[1]Medicine, The Ohio State University, Columbus, OH; [2]Surgery, The Ohio State University, Columbus, OH; [3]Endocrinology, The Ohio State University, Columbus, OH.*

PTDM, defined as the need for any hypoglycemic treatment post-transplant (Tx), is due in part to the diabetogenic effects of immunosuppressants. We showed recently that 6% of CsA treated Tx patients develop PTDM during the first year post-Tx. Beyond that time the incidence of PTDM raises continuously. Thus, 16% of 1874 Tx recipients followed for 5.7±4 years developed PTDM (ASN2000, In Press). The incidence of PTDM correlates significantly with, increasing age, increasing weight, and African American race. Furthermore, since 1995 the incidence of PTDM has increased significantly. In this study we compared the CV risk profile of 3 groups of adult Tx recipients: 1) Patients with PTDM (N=201); 2) patients with pre-Tx DM (mainly AODM, N=398); and 3) patients without DM (NoDM, N=1269). PTDM and DM patients were significantly older than NoDM (47+12, 47+12 and 40+13 years respectively, ANOVA <.0001). The % of African Americans was higher in PTDM (24%) and DM (23%) than in NoDM (16%) (Chi sq, p=.001). PTDM and DM were also significantly heavier than NoDM (81+20, 78+19 and 73+19 Kg, ANOVA<.0001). The average cholesterol levels post-Tx were not significantly different among groups. However, TG levels were significantly higher in PTDM and in DM than in NoDM (307+169, 290+237, 276+169 mg/dl, Kruskal-Wallis <.0001). Of interest, high TG level preceded the diagnosis of PTDM, and 19% of patients with TG levels >250 mg/dl subsequently developed PTDM compared to 11% of patients with lower TG levels (chi sq, p<.0001). Pulse pressure (PP), a strong correlate of CV morbidity, was significantly higher in PTDM and in DM than in NoDM (59+13, 63+14, 53+11 mmHg, ANOVA <.0001). PP declined progressively post-Tx in NoDM. In contrast, in PTDM PP rose progressively reaching the higher levels of DM patients within 3 years post-Tx. Patient survival was significantly worse in DM (23% dead) than in PTDM (17%) and in NoDM (15%) (Log Rank =.0001). Conclusion, PTDM is associated with an unfavorable CV risk profile, similar to that of DM and including: older age, dyslipidemia, obesity, and high PP. Elevated TG levels and rising PP precede frank hyperglycemia suggesting that these parameters may be early indicators of insulin resistance. PTDM is not associated with significantly reduced survival likely due to the short follow up time.

Abstract# 23

PERIOPERATIVE MI IN RENAL TRANSPLANT RECIPIENTS.

Connie L. Manske, Than Oo, Arthur J. Matas.

The University of Minnesota employs an aggressive pretransplant cardiac screening program, including coronary angiography for diabetic transplant candidates and patients over 50 with symptoms or positive stress tests. In spite of pretransplant cardiac screening of high risk renal transplant recipients, the incidence of peritranspiant myocardial infarction (MI) remains significant. Therefore, we studied all renal allograft recipients transplanted at the University of Minnesota from May 99 to Apr 00 to determine the rate of postoperative MI and the characteristics of patients at risk. The transplant database was used to identify patients who sustained an MI within 6 months of receiving a renal transplant, and patient charts were reviewed to obtain demographic and historical data.

Of 229 patients receiving a renal transplant during the one year period, 17 (7%) had an MI within 6 months of transplantation. Of these, 12 were within 3 days, and 14 within one month, of surgery. Two MIs were immediately fatal. Four additional MIs were associated with patient death but occurred in the setting of postoperative complications including sepsis and MOSF (1), mesenteric thrombosis (1) PE (1), and traumatic injury (1). One additional patient sustained an uncomplicated perioperative MI and died two months later from surgical complications of a small bowel obstruction. Patient demographics were: 10(59%) male, 10(59%) diabetes, 14(83%) over age 50. Ten (59%) had a cadaveric donor (two received a simultaneous pancreas transplant). Twelve patients (71%) had a history of previous CABG (7), angioplasty (2) or MI (4). Only 3 patients had not undergone coronary angiography prior to transplantation. Two of these patients were nondiabetic women aged 50 and 56 who had normal stress tests and one was a 28 year old male with no risk factors.

Conclusion: 5% of 229 renal transplant recipients sustained a perioperative MI. The majority were uncomplicated, but two were fatal. The majority of MIs occurred in patients with revascularized ischemic heart disease, which suggests that even revascularization cannot reduce the MI risk to zero in some patients with diffuse coronary artery disease. Further study is needed to determine whether high risk patients might benefit from perioperative anticoagulation, beta blockade or other treatment strategies.

Abstract# 24

SIROLIMUS-BASED IMMUNOSUPPRESSION FOR IMMUNOPROPHYLAXIS OF ACUTE ALLOGRAFT REJECTION IN PATIENTS WITH CALCINEURIN-INHIBITOR INDUCED HEMOLYTIC UREMIC SYNDROME.

Alan Leichtman, the Sirolimus HUS Compassionate Use Study Investigators. [1]*Department of Nephrology, University of Michigan Medical Center, Ann Arbor, MI.*

Hemolytic uremic syndrome (HUS) may develop in 5-10% of renal allograft recipients treated with calcineurin inhibitors (CI). We report the outcome of 15 renal transplant recipients with a history of HUS who received sirolimus through a compassionate use program that provided access for immunoprophylaxis of acute allograft rejection. Conversion to sirolimus (mean maintenance dose 6.77 mg/d; range 1.0-20.0 mg/d) occurred at varying times post-transplantation, with cyclosporine or tacrolimus having been completely eliminated in 10 subjects. Cyclosporine was minimized but continued in 1 additional recipient, while the remaining 4 did not receive CI immediately prior to starting sirolimus. HUS was CI-associated in 13/14 patients (92.9%) for whom this information was available. There were no reported recurrences of HUS. Ten of 15 patients (66.7%) had greater than 6 months follow-up. After initiation of sirolimus, one- and three-month mean (±sd) Nankivell GFR improved from 47.1±19.0 (n=10) to 57.9±21.2 (n=9) mL/min, respectively, and remained stable at 67.9±34.5 mL/min (n=8) through the time of last recorded follow-up (345±274 days). During that time, none of the patients experienced acute rejection. One functioning allograft was lost due to death from a myocardial infarction on day 15 of sirolimus therapy. Overall patient and graft survivals were both 93.3% (n=15). In 3 cases, sirolimus was discontinued at treatment days 15, 27, 117, due to death, an adverse event possibly related to sirolimus, and a patient request unrelated to sirolimus, respectively. All but one of the 10 patients who continued to receive sirolimus for >6 months had been discontinued from CI therapy; none required re-initiation. HUS is historically associated with poor renal allograft survival. The above data suggest that sirolimus-based immunosuppression is a promising alternative for immunoprophylaxis of acute allograft rejection in patients with CI-induced HUS.

Abstract# 25

THE TREATMENT OF HYPERLIPIDEMIA IN RENAL TRANSPLANT RECIPIENTS: DO WE PRACTICE WHAT WE PREACH?

Sandra M. Cockfield, Loreen Wales, Denise Lam. [1]*Nephrology and Immunology, University of Alberta, Edmonton, AB, Canada.*

With increasing renal allograft survival, greater emphasis is being placed on the prevention and management of complications such as premature cardiovascular disease (CVD). Recently published clinical practice guidelines (J Am Soc Nephrol, 2000) concluded that renal transplant recipients (RTR) are at high risk for CVD and that interventions could target several potentially modifiable risk factors. To determine whether our current management of post-transplant dyslipidemias meet these guidelines, we reviewed the charts of 530 adult RTR, transplanted between January 1981 and June 1999, to obtain cross-sectional data on CVD risks including age, gender, blood pressure, diabetes, smoking, obesity, immunosuppressive medications, and lipid profiles drawn in 1998-1999. The population was 63.8% male with a mean age of 47.7±13.2 yrs. Diabetes was present in 24.2%, hypertension in 90.5%, and known CVD in 18.5%. RTRs were classified for risk for CVD: 32.1% of RTRs were considered very high risk (VHR) on the basis of known CVD or diabetes; the remaining 67.9% were considered high risk (HR). Of those considered HR, 95% had at least 1 CVD risk factor (male≥45years, female≥55years, hypertension, smoking, LDL>3.4 mM or HDL<0.9 mM). Although 90.5% of RTRs had a total cholesterol performed in 1999-9 (92.4% of VHR, 89.7% of HR), full lipid panels were performed in only 63.4% (69.4% VHR, 60.6% HR). Dyslipidemias were common; mean cholesterol was 5.3±1.1 mM, LDL-C was 3.0±0.9 mM, TG 2.1±1.2 mM, and TC:HDL 4.1±1.4. A significant proportion of patients in both groups failed to meet recommended lipid targets and yet were untreated.

Risk group	Lipid parameter	% RTR	% untreated
VHR	LDL >2.5 mM	56.5%	57.8%
	TC HDL >4	48.3%	51.8%
	TG >2.0 mM	36.4%	42.9%
HR	LDL >3.0 mM	44.4%	75.6%
	TC HDL >5	25.3%	81.8%
	TG >2.0 mM	43.4%	74.0%

Current screening for and management of dyslipidemias in RTRs does not meet published recommendations. RTRs may benefit from the development of specific algorithms to address this modifiable risk factor on a consistent basis, given their increased risk for premature CVD.

Abstract# 26

THE USE OF MONO/POLICLONAL ANTIBODIES DOES NOT NEGATIVELY INFLUENCE THE OUTCOME OF LIVER DISEASE AT FIVE YEARS AFTER RENAL TRANSPLANTION IN HEPATITIS C VIRUS POSITIVE PATIENTS. Beatriz Dominguez-Gil,[1] Nuria Esforzado,[2] Miguel A. Muñoz,[1] Amado Andres,[1] Federico Oppenheimer,[2] Jose L. Rodicio,[1] Jose M. Campistol,[2] Jose M. Morales.[1] *[1]Nephrology, 12 de Octubre Hospital, Madrid, Madrid, Spain; [2]Renal Transplant Unit, Clinic Hospital, Barcelona, Barcelona, Spain.*

The impact of immunosuppresion on liver disease due to Hepatitis C virus (HCV) infection is not well established. It has been suggested that mono/policlonal antibodies could favour chronic liver disease in these patients. Our current immunosupressive protocol limits the use of ATG/OKT3 for induction in high immunological risk patients (hyperimmunized patients, second transplants) or for the treatment of corticoresistant acute rejection. In the present study, we compare the evolution of HCV + kidney transplant patients who received ATG/OKT3, with that of HCV + kidney transplant patients who did not, both groups of patients being treated with a cyclosporine based protocol. We include 263 HCV + patients who were transplanted in our units during March 1990-December 1998. Ninety one patients were treated with antilymphocyte preparations as the protocol described above (Group I). The rest of the patients did not receive such medication (Group II).

	GROUP I (N=91)	GROUP II (N=172)	p
Follow-up (months)	65 1 ± 29 9	65.4 ± 32.7	NS
HCV RNA +	60/66 (91%)	119/130 (91 5%)	NS
Chronic Liver Disease *	5 (5 8%)	22 (13%)	NS
Hepatonecrosis	16 (18%)	33 (20%)	NS
Severe Liver Disease **	5 (5 7%)	6 (6.3%)	NS
Death due to Liver Disease	2	1	NS
Patient Survival ***	84%	92%	

* ALT levels greater than 2.5 times the upper normal limit. **Hepatocelular insufficiency and/or portal hypertension. ***Five Years Life Table analysis. NS:Non significative

In summary, our results suggest that therapy with ATG/OKT3 in selected high risk patients did not adverselly afect the evolution of liver disease due to HCV infection after kidney transplantation at five years. However, the possible deleterious effect of this approach on liver function could be observed later on.

CONCURRENT SESSION 3:
IMMUNOSUPPRESSION/TOLERANCE

Abstract# 27

STEALTH, THE 3RD MODEL OF PRIMATE TOLERANCE: EVIDENCE FOR SUSTAINED TH2 CYTOKINE PRODUCTION IN THE LONG TERM (LT) SURVIVORS. Judith M. Thomas, Andrew Lobashevsky, Cheryl A. Smyth, William Hubbard, Devin Eckhoff, Juan L. Contreras, David Neville, Francis T. Thomas. *[1]University of Alabama at Birmingham, Birmingham, AL.*

Introduction: The two major approaches to primate tolerance are costimulation blockade and mixed chimerism. Recent studies proposed a 3rd model of primate donor-specific tolerance, termed STEALTH. The acronym signifies Specific Tolerance by early Evasion of APC-Lymphocyte interactions and TH2 deviation. The model utilizes neither chimerism nor costimulatory blockade to achieve stable tolerance without chronic immunosuppression (CI), but capitalizes upon a unique synergy between anti-CD3 immunotoxin (IT) and Deoxyspergualin (DSG). This peritransplant treatment protocol depletes tissue and circulating T cells while inhubiting NF-κB dependent maturation of dendritic cells and proinflammatory cytokines responses. Here, we update the LT survival of MHC-mismatched kidney allografts (KTx) and report complex dynamics of T cell repopulation and an enduring TH2 cytokine deviation in LT survivors.

Methods: LT KTx rhesus macaque recipients in this study (n=14) were transplanted 1.4-4.5 years ago. All were given an allograft mismatched for MHC class I & II alleles. All exhibited normal LT KTx function without intrinsic kidneys. All received IT (as either IgG or F(Ab)), beginning the day of KTx. No CI was used. 13/14 received DSG during the 1st 2 weeks post-KTx; 1*/13 received only F(Ab) IT. Systemic plasma cytokine levels of IL10, IL-4, and IFNγ were tested during 1- 4.5 yr. Follow up, and dynamics of T cell repopulation examined in T^{memory}(CD45RO+), T^{naive} (CD45RA+), recent thymic emigrants (RTE) (CD103+), and $TH2^{memory}$ (CRTH2+ populations.

Results: Among the LT KTx, 12/14 are alive and with normal KTx function at: >1673, >1452, >1541, >1141, >1107, >744, >745, >>716, 690*,>638,>603, and >533 days. Two LT KTx have been lost; one to urinary stones at d.1470 and one at d.230 to obstructive ureteral fibrosis at the site of a retained stent. T cell repopulation occurred in 3 tandem waves of: 1) early peripheral expansion of T^{memory} and T^{naive} cells and compression of the former, 2) expansion of RTE and 3) appearance of $TH2^{memory}$. Plasma levels of IL4 and IL10 were significantly elevated over normal (10-20 fold) throughout follow up. In contrast, IFNg was 6-fold decreased in the first year and 2- fold increased during 1-4.5 years post-KTx.

Conclusion: The results confirm stable LT function of MHC-mismatched KTx after tolerance induction by STEALTH, a model that appears to merge paradigms of Danger and Cytokine Deviation. T cells recover in a tri-phasic pattern that includes peripheral expansion, RTE, and generation of TH2 T^{memory} cells in concert with continuous TH2 cytokine deviation showing high IL10 and IL4 production for years.

Abstract# 28

CAN IMMUNOLOGIC UNRESPONSIVENESS IN RHESUS MONKEYS PREDICT A SUCCESSFUL TOLERANCE STRATEGY IN HUMANS? Stuart J. Knechtle, John H. Fechner, Clifford S. Cho, Kevin G. Brunner, Yinchen Dong, Majed M. Hamawy. *[1]Department of Surgery, University of Wisconsin, Madison, WI.*

The nonhuman primate renal allotransplant model is considered the closest animal model to human renal transplantation and well suited for preclinical development of immunosuppressive and tolerance strategies. Based on results in nonhuman primates, clinical trials have been designed and performed. We have had experience with several such strategies in both nonhuman primates and humans. Donor-recipient combinations of monkeys, ages 1-3 years, have been chosen based on DNA typing of class II alleles, MLC, and 1-DIEF of MHC class I. The table summarizes four induction strategies using no long-term immunosuppression, and results are shown only for monkeys with >1-year graft survival. All of these donor-recipient pairs had defined MHC disparity and untreated controls reject in 5-9 days. Although 12 of these 17 rhesus monkey renal allograft recipients eventually succumbed to chronic rejection after more than a year, all remained free of acute rejection. The monkey treated with only cyclosporine was 1 of 3 such monkeys with the others succumbing to acute rejection prior to 200 days. One-third of monkeys treated with immunotoxin (IT) survived >1 year. One of 8 monkeys treated with anti-CD154 and 1 of 3 monkeys treated with ATG induction survived >1 year.

Baseline induction	Graft survival time (days)
Cyclosporine for 60 days (levels 150-300 mg/ml)	582, >588
Anti-CD3 immunotoxin (last dose on day 2)	728, 846, 1489 889, 1038, >1364 712, 601, 413, 435, 630, >742, >1028
Anti-CD154 (last dose on day 28)	>1359
Rabbit ATG	1227

These results imply that compared to human renal transplantation where discontinuation of all immunosuppression results in acute rejection in >90% of cases, it is relatively easier to prevent acute rejection in the rhesus monkey renal allograft model. These results have implications for preclinical modeling of human immunosuppressive and tolerance strategies. Although the monkey model most closely represents the human situation, the age of monkeys may be important and it should be anticipated that tolerance in humans will be more difficult to achieve.

Abstract# 29

NEW INSIGHTS INTO THE INTERACTION BETWEEN T CELL COSTIMULATION BLOCKADE AND CONVENTIONAL IMMUNOSUPPRESSION IN VIVO. Masayuki Sho,[1] Nader Najafian,[1] Victor M. Dong,[1] Koji Kishimoto,[1] Akira Yamada,[1] Sigrid E. Sandner,[1] Mohamed H. Sayegh.[1] *[1]Laboratory of Immunogenetics and Transplantation, Brigham and Women's Hospital, Boston, MA.*

In order to develop T cell costimulation blockade strategies clinically we investigated systematically the interactions between CD40-CD154 or B7-CD28 blockade with conventional immunosuppressive agents in vivo. We used a vascularized fully MHC-mismatched cardiac allograft model. BALB/c recipients of C57BL/6 hearts were treated with anti-CD154 mAb (MR1) or CTLA4Ig. Additional groups were treated with cyclosporine (CsA or CsA30; 20mg/kg, day 0-3 or 30-33), FK506 (FK or FK30; 1.5mg/kg, day 0-3 or 30-33), methylprednisolone (MP; 20mg/kg, day 0-7), or rapamycin (RPM; 0.3mg/kg, day 0-3). Graft survival was compared and long-term graft morphology was evaluated to assess chronic rejection.

Group	Treatment	MST±SE	Group	Treatment	MST±SE	Group	Treatment	MST±SE
I	None	7 0±0.6	VII	sMR1+CsA	22.0±3.7	XIII	mMR1	>100
II	sMR1	62.0±40 6	VIII	sMR1+FK	17.5±4.3	XIV	mMR1+CsA	>100
III	CsA	13 0±7 3	IX	sMR1+RPM	>100	XV	mMR1+CsA30	>100
IV	FK	11 0±0.6	X	sMR1+MP	37.5±16.5	XVI	mMR1+FK	>90
V	MP	14.5±6 2	XI	sMR1+CsA+RPM	>100	XVII	mMR1+FK30	>90
VI	RPM	22 0±6.2	XII	sMR1+CsA+MP	17 0±3.8			

The effect of single dose MR1 (sMR1:0.25mg, day 0) was abrogated by CsA, FK, or CsA+MP (p=0.017, p=0.003, p=0.003 respectively vs. group II). Interestingly, multiple doses of MR1 (mMR1; 0.5mg on day 0, 0.25mg on day 2,4,6) induced long-term survival and overcame the opposite effects of calcineurin inhibitors in both induction and maintenance phases (group XIII-XVII). RPM acted synergistically with sMR1 (p=0.037 vs. group II, p=0.0005 vs. group VI) and significantly less fibrosis and interstitial inflammation were seen in heart grafts suggesting that sMR1+RPM prevented chronic rejection. Furthermore, the additional CsA to sMR1+RPM did not affect long-term survival (Group XI). CTLA4Ig (0.25mg on day 2) induced long-term survival (MST>100 days) and was not affected by any immunosuppressive drugs. Our data provide new insights into important interactions between conventional immunosuppressive drugs and T cell costimulatory blockade. We conclude that the wide spread view that calcineurin inhibitors abrogate the effects of T cell costimulatory blockade should be revisited. These results should have relevant clinical implications in organ transplantation.

Abstract# 30

DENDRITIC CELLS TREATED WITH ANTISENSE OLIGODEOXYRIBONUCLEOTIDES TARGETING CD80 OR CD86 mRNA PROLONG CARDIAC ALLOGRAFT SURVIVAL.

Xiaoyan Liang,[1] Lina Lu,[1] Zongyou Chen,[1] Hong Zhang,[2] Nicholas M. Dean,[2] John J. Fung,[1] Shiguang Qian.[1] *[1]Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, Pittsburgh, PA; [2]ISIS Pharmaceutical, San Diego, CA.*

Immature DC expressing low costimulatory molecules (CM) induce donor-specific hyporesponsiveness. However, their immunosuppressive effect is limited *in vivo* due to rapid upregulation of CM upon interaction with host cells. Treatment of DC with antisense oligodeoxyribonucleotides (ODN) targeting CM mRNA may enhance tolerogenicity. DC propagated from B10 ($H2^b$) bone marrow in GM-CSF and IL-4 were administered anti-CD80 or -CD86 ODN. DC phenotype and allostimulatory function were determined by flow and MLR. Cytokines were measured by ELISA. The tolerogenicity of DC was evaluated in a B10→C3H ($H2^k$) vascularized cardiac transplant model. After exposure to FITC-ODN (140 μM), 80% of DC exhibited fluorescence. ODN concentration could be reduced to 0.2 μM with the addition of lipofectamine (10 μl/ml). The fluorescence was sustained as long as being viable in culture (d 14). Compared to scrambled ODN, DC exposed to anti-CD80 or -CD86 demonstrated a decreased incidence of CD80 or CD86 expression (from 70.6% to 48.2% and 37.5% to 16.9%, respectively), and stimulated lower T cell proliferative responses, with correspondent downregulation of IL-2 and IFN-γ release, but upregulation of IL-4 production. Addition of antisense ODN at the initiation of DC propagation produced optimal suppressive effect, suggesting that ODN inhibited rather than reversed DC maturation. Survival of B10 heart allografts in C3H recipients was significantly prolonged in groups injected (d -7, i.v., 2 x 10^6) with DC treated with anti-CD80 or CD86, compared with scrambled ODN ($p<0.05$), or no ODN treatment groups ($p<0.05$) (Table). In conclusion, treatment of DC with antisense ODN targeting expression of CM is an effective approach to arresting DC maturation and enhancing their tolerogenicity.

Treatment	Survival (MST) (day)
None	9, 10, 11 x 2, 12, 14 (11.2 ± 0.7
DC	9 x 2, 6 x 4, 7 x 2, 8 (6.2 ± 1)
DC-anti-CD80	16, 18 x 4, 19, 20 (18.2 ± 1.2)
DC-scrambled anti-CD80	7 x 2, 9 x 2, 10 (8.4 ± 1.1)
DC-anti-CD86	16, 17 x 3, 21 x 2, 22, 45 (22 ± 9.6)
DC-scrambled anti-CD86	8 x 3 (8 ± 0)

Abstract# 31

EFFECT OF INCREASING ANTI-CD40L (IDEC 131) ANTIBODY DOSE AND CONCOMITANT T-CELL DEPLETION ON PRIMATE ALLOGRAFT SURVIVAL AND HISTOLOGY.

Steffen Pfeiffer,[1] George L. Zorn, III,[1] Agnes M. Azimzadeh,[1] James Atkinson,[2] Robert Newman,[3] Richard N. Pierson, III.[1] *[1]Cardiac and Thoracic Surgery, Vanderbilt University Medical Center, Nashville, TN; [2]Pathology, Vanderbilt University Medical Center, Nashville, TN; [3]IDEC Pharmaceuticals Inc., San Diego, CA.*

Objective: To evaluate α CD40L dosing and additional T-cell depletion on primate cardiac allograft survival and histology. **Methods:** MHC-class II mismatched cynomolgus recipients (SI>5) of heterotopic hearts received humanized α CD40L-Ab: 40, 60, or 120mg/kg in the first two weeks, then 20mg/kg monthly. 6 animals also received rabbit anti-human ATG (days 0,3,7), profoundly depleting peripheral T-cells. Rejection episodes (infiltrates, graft dysfunction) were not treated. **Results:** No α CD40L-related complications were seen. Low dose regimen prevented early (≤15 days) graft loss, with trough Ab levels of >100 mg/ml on day 5 & 14. Giving the second Ab-dose on day 7 was associated with 66% early graft loss (D7 trough Ab: 280+/-170 mg/ml). An additional dose on day 3 reduced early graft loss (D7 trough Ab: 420+/-40 mg/ml). Increasing intensity of αCD40L reduced but did not prevent early appearance of intense lymphocytic infiltrates in most grafts, but delayed or prevented graft dysfunction. Grafts failed late with cardiac allograft vasculopathy (CAV). Additional periop ATG prevented early graft loss and early lymphocytic infiltrates; prolonged (CD40L-associated) graft protection did not prevent subsequent loss due to CAV. **Conclusions:** 1) αCD40L Ab monotherapy attenuates graft injury despite lymphocytic infiltrates. 2) Frequent early dosing rather than high trough levels appear critical. 3) A short course of peripheral T-cell depletion and ongoing monthly αCD40L therapy is not sufficient to induce tolerance or prevent allograft vasculopathy in this model 4) As used, periop ATG does not abrogate αCD40L effect, or induce tolerance.

Group (n)	Days of αCD40L	Graft survival, days (median)	Bx Grade ≥3 (n)
Historical controls (4)		5,5,5,6 (5)	100% (4/4)
Low dose (3)	0,5,14,28,bi-weekly	56,106,245 (106)	100% (6/6)
Moderate dose (9)	0,7,14,28,monthly	6,8,8,10,13,15,38,95,269* (13)	70% (7/10)
High frequent dose (5)	0,3,7,14,28,monthly	12,41,43,58,112 (43)	44% (4/9)
M/HF+ATG (6)	0,3,7,14,28,monthly	25*,41,41*,74,93,142· (74)	14% (1/7)

* died with beating graft, · alive, *Rx d/c POD 180

Abstract# 32

LONG-TERM METABOLIC STUDIES IN STREPTOZOTIN (STZ) INDUCED PRIMATE RECIPIENTS AFTER TOLERANCE INDUCTION TO ALLOGENIC PANCREATIC ISLET TRANSPLANTS (PIT).

Judith M. Thomas, Juan Conteras, Cheryl Smyth, Devin Eckhoff, Andrew Lobashevsky, Francis T. Thomas. *[1]University of Alabama at Birmingham, Birmingham, AL.*

Introduction: Isolated islet transplantation is associated with loss of functional islet mass (FIM or Kg) and Insulin Secretory Reserve (AIR or Acute Insulin Response to glucose), both early and late post-transplant. These changes decrease short and long-term success and compromise a limited supply of donor islets. Rejections, recurrence of autoimmunity and toxicity by immunosuppressive drugs have all been postulated to play a role in graft failure. A promising therapy for IDDM is the induction of immune tolerance to islet grafts, potentially providing stable, long-term function without chronic immunosuppression (CI). Recently, we demonstrated long-term PIT tolerance without CI in STZ-induced diabetic primates treated with F(Ab) IT plus DSG. In the present study, the durability of FIM and AIR was assessed over a 1-1.5 year period. **Methods:** IDDM was induced in 3-4 kg rhesus monkeys with STZ (140 mg/kg/iv). All STZ-treated animals developed fasting glucose levels >250mg/dl, undetectable stimulated C-peptide levels (<0.5 ng/dl), abnormal intravenous glucose tolerance test (IV-GTT) and required at least 2-5 units of human insulin/kg/day. Mean pre-Tx Kg was 0.28+/- 0.09 and mean AIR was 2.4 +/- 2.3. Islets were isolated by the Ricordi method with minor modifications. Overnight cultured, MHC-mismatched allogeneic islets were infused intraportally at a mean 25,000 IEQ/kg in STZ diabetic recipients. Donors were selected to have 2-3 mismatches at Mamu-A and DRB genes by PCR-SSP typing. 7 animals received F(Ab) IT plus DSG (2.5 mg.kg/iv for 15 days). 2 received F(Ab) IT alone; 2 received DSG alone; and 2 received F(Ab) IT plus DSG without PIT. No CI was given. **Results:** All PIT recipients became normoglycemic without exogenous insulin after the transplant. 1 of 7 recipients in the F(Ab) IT plus DSG group developed acute rejection on day 70. One presented with hyperglycemia on day 335 and has since received exogenous insulin. One animal died with normal islet function following an invasive multi-catheter procedure that demonstrated a 50% insulin secretion step-up across the liver but not the intrinsic pancreas on day 187. 4 animals are normoglycemic at days 566, 532, 475 and 386 post-transplant. None given DSG alone or F(Ab) IT alone survived longer that 70 days. PIT under F(Ab) IT plus DSG tolerance induction resulted in stable restoration of normal metabolic parameters and durable FIM & AIR (mean one yr Kg - 1.78 and AIR- 37.6 ($p>0.10$ vs. 15, 30, 90, 120 and 240 day values). **Conclusions:** Stable FIM and AIR occurred in 4 of 5 long-term PIT survivors for >1 year. These results in a preclinical primate tolerance model suggest that excellent and durable FIM and AIR is achievable without CI and with single donor PIT.

Abstract# 33

SURVIVAL OF PANCREATIC ISLET ALLOGRAFTS IN DIABETIC CYNOMOLOGYS MONKEYS.

Maria Koulmanda,[1] Andi S. Qipo,[1] Neal R. Smith,[2] Tatsuo Kawai,[1] Jack Oneil,[3] Jim Rusche,[4] Dicken Ko,[1] Gordon Weir,[3] Terry Strom,[5] Hugh Auchincloss, Jr..[1] *[1]Surgery, MGH/HMS; [2]Pathology, MGH/HMS; [3]JDC/HMS; [4]Repligen; [5]BIDMC/HMS, Boston, MA.*

To study islet transplantation in non human primates we performed, 2 autografts, 1 allograft without immunosuppression and 7 allografts with different immunosuppressive protocols. Diabetes was induced by either 80% pancreatectomy followed by Streptozotocin (STZ) or by 55mg/kg STZ alone. The animals were maintained on daily insulin until they were transplanted. The majority of the islets were infused into the portal vein and the rest under the left kidney capsule.

Blood glucose and C-peptide levels were monitored along with a number of other biochemical and hematological tests. The islets under the capsule were biopsied 2-3 times during each experiment, and again at the time of sacrifice at which time both the kidney and the liver were examined.

Using C-peptide levels and blood glucose monitoring, we found that either pancreatectomy plus STZ or STZ alone was sufficient to induce diabetes with no evidence of regeneration of host pancreatic islets. Immunosuppression was give to groups 3 to 6: gr 3 received ATG (-2, -1 and 0) CsA (day 0-28) and MMF (day 0-14); gr 4 received CTLA4Ig alone (day 0-15); grs 5 and 6 received RPM (-2 to 28) and anti-CD40L twice a week for 4 weeks, gr 6 also received anti-CD8 MAb on days 0, 3 and 7 post-transplantation.

GROUPS	TREATMENT	GRAFT SURVIVAL (Days)	ISLETS (IE/kg)
1-Autografts	None	>120, >100	7,500 , 3,000
2-Allografts	None	4	12,000
3-Allografts	ATG/CsA/MMF	16, 36	5,600, 5,500
4-Allografts	CTLA4Ig	14, >15	14,500, 12,500
5-Allografts	RPM, anti-CD40L	42, >50	11,500, 12,500
6-Allografts	RPM, anti-CD40L, anti-CD8	99	10,200

These results demonstrate that: (1) islet autografts can be transplanted successfully and can function for prolonged periods in monkeys; (2) islet allografts function briefly but are rejected in less than a week in the absence of immunosuppression; and (3) islet allograft survival can be prolonged for up to 99 days with brief post-transplant immunosuppression. One of the autografts with 3,000 islets/kg showed evidence of survival and function, but with imperfect blood glucose control. This suggests that more than about 5,000 islets/kg are needed to achieve insulin independence in monkeys.

Abstract# 34

PIRFENIDONE DECREASES TGF-β1 EXPRESSION AND AMELIORATES FIBROSIS IN CHRONIC CYCLOSPORINE NEPHROTOXICITY. Fuad S. Shihab,[1] William M. Bennett,[2] Hong Yi,[1] Takeshi F. Andoh.[2] *[1]Division of Nephrology, University of Utah, Salt Lake City, UT; [2]Solid Organ and Cellular Transplantation Services, Legacy Hospital, Portland, OR.*

Pirfenidone (PFD) is a newly developed compound that can prevent fibrosis in the kidney, lung and peritoneum. We examined the effect of PFD on chronic cyclosporine (CsA) nephrotoxicity which is characterized by progressive tubulointerstitial fibrosis. Salt-depleted pair-fed rats were treated with olive oil (VH), VH+PFD (250 mg/kg/day), CsA (7.5 mg/kg/day) or CsA+PFD and were sacrificed at 28 days.

Concommitant therapy with PFD in CsA-treated rats improved CsA-induced decrease in GFR although it did not reach statistical significance. Also, PFD ameliorated significantly (by 50%) CsA-induced interstitial fibrosis (P<0.05 vs. CsA group). We have previously shown that apoptosis plays a role in this model. The number of apoptosis (+) cells by TUNEL, which was increased in the CsA group (P<0.05 vs VH group), was significantly decreased (by 75%) in the CsA+PFD group (P<0.05 vs CsA group). In this experiment, there was a significant correlation between interstitial fibrosis and apoptosis in all groups (r=0.85, P<0.01).

We also looked at the mRNA expression of TGF-β1, plasminogen activator inhibitor (PAI)-1 and the proteoglycan biglycan by Northern blot analysis. Values are mean±SD; N=4-5/group; *P<0.05 vs VH group; #P<0.05 vs CsA group; mRNA values were GAPDH corrected.

	VH	VH+PFD	CsA	CsA+PFD
TGF-β1	0 85±0 10	0 78±0 01	2 12±0 47*	0 82±0 10#
PAI-1	0 11±0 06	0.10±0 02	0 59±0 20*	0.11±0 02#
Biglycan	0 17±0 04	0 16±0 05	0 49±0 24*	0 17±0 06#

These results demonstrate that PFD treatment significantly protects animals from CsA-induced fibrosis and apoptosis. PFD also ameliorated CsA-induced decrease in GFR. These effects were specific to CsA since PFD treatment in VH rats had no significant effect. We have previously shown that TGF-b1 is involved in the fibrosis of chronic CsA nephrotoxicity by increasing matrix production and by decreasing matrix degradation by means of upregulating PAI-1, an inhibitor of the plasmin protease system. PFD decreased the expressions of both TGF-b1 and PAI-1 in CsA-treated rats to the levels observed in VH-treated rats. This was associated with amelioration of CsA-induced fibrosis. Thus, PFD seems to be protective in chronic CsA nephrotoxicity and may have a beneficial effect in other models of fibrosis.

Abstract# 35

ATORVASTATIN PREVENTS CHRONIC REJECTION IN RAT CARDIAC ALLOGRAFTS. Ping Ji,[1] Yale D. Podnos,[1] Ming-Sing Si,[1] Earl Steward,[1] David K. Imagawa.[1] *[1]Div. of Transplantation, University of California, Irvine, Orange, CA.*

Background: Atorvastatin, a new HMG-CoA reductase inhibitor, has been shown to ameliorate coronary transplant vasculopathy following orthotopic heart transplant. In a rat cardiac allograft model, heterotopic abdominal cardiac transplants performed across minor histocompatibility barriers reproducibly develop transplant vasculopathy. We have previously demonstrated the efficacy of pravastatin in ameliorating transplant vasculopathy in this model. This study sought to determine if atorvastatin affects the development of transplant vasculopathy and, if so, to further investigate the mechanism of action.

Methods: F344 rats served as recipients of Lewis allografts. Group 1 consisted of syngeneic cardiac transplant rats (n=6) receiving no immunosuppression. Group 2 (n=6) received only cyclosporine (CsA) (1 5 mg/kg/day for 10 days) to prevent acute rejection treatment. Group 3 consisted of experimental rats (n=8) receiving adjunctive atorvastatin (20 mg/kg/day for 4 months) in addition to cyclosporine (CsA) (1.5 mg/kg/day for 10 days). Transplanted grafts were harvested at 120 days. Cardiac grafts were evaluated by H&E, Verhoeff's Van Elastic and Masson's Trichrome stains. Host anti-donor IgG and IgM alloantibodies were monitored by flow cytometry. Results are expressed as the relative mean channel fluorescence, calculated as the fluorescence obtained with experimental serum sample divided by the mean fluorescence of cells incubated with control serum. Intragraft expression of anti-oxidant hemoxygenase-1 (HO-1) gene was analyzed by Western blot.

Results:

	Histology score	IgG	IgM	HO-1
Syngeneic	0 5±0 3	1 00±0 5	1 00±0 5	-
CsA	2 8±0 7	4 33±1 2	1 83±0 9	-
CsA+Atorvastatin	1 0±0 6	1 58±0 7	1 49±0 7	+
Group 3 vs 2	p<0 05	p<0 01	p=ns	

Conclusion: Atorvastatin prevented transplant vasculopathy in a rat model of chronic rejection. This efficacy is associated with attenuation of host anti-donor IgG alloantibodies. Grafts treated with atorvastatin showed upregulation of the protective HO-1 gene, possibly playing an important role in preventing arteriosclerosis and chronic rejection of heart allografts.

CONCURRENT SESSION 4:
REJECTION I

Abstract# 36

HEME OXYGENASE 1 (H0-1) GENE TRANSFER DELAYED ALLOGRAFT ACUTE REJECTION IN A RAT MODEL. Christine Chauveau,[1] Cecile Braudeau,[1] Cecile Guillot,[1] Claire Usal,[1] Suhasini Iyer,[2] Jean Paul Soulillou,[1] Marie Cristina Cuturi,[1] Roland Buelow,[2] Ignacio Anegon.[1] *[1]INSERM U437, Nantes, France; [2]SangStat Medical Corporation, Fremont, CA.*

HO-1 is the rate limiting enzyme in heme degradation to carbon monoxide (CO), iron and bilirubin. It has recently been shown to be involved, through its catalytic by-products, in the inhibition of apoptosis, immune responses and inflammation.

To evaluate the effect of HO-1 overexpression in allogeneic immune responses, we generated a recombinant adenovirus coding for HO-1 (AdHO-1) and we transduced (1010 pfu) rat hearts at transplantation in an allogeneic combination (LEW.1W [RT1u]→[LEW.1A [RT1a]). Hearts nontransduced or transduced with a non-coding virus (Addl324) were rejected in 11+/-0.6 (n=11) and 7.5+/-0.7 (n=8) days, respectively, whereas hearts transduced with AdHO-1 survived 27 +/- 3.7 days (n=11). Immunohistological analysis showed that grafts injected with AdHO-1 or Addl324 displayed comparable infiltration by total leukocytes, macrophages, MHC class II+,αβTCR+, CD4+ and CD8+ cells. In contrast, an increase in CD25+ cells was detected in AdHO-1-injected hearts. While MLR responses of total splenocytes from rats transplanted with AdHO-1-treated hearts against LEW.1A or third-party (BN) dendritic cells were inhibited as compared to those from Addl324-treated controls, T cell responses were comparable. Systemic immune responses against cognate antigens were not inhibited in recipients of AdHO-1 treated grafts, as evaluated at day 12 by proliferation of lymph node cells against KLH previously injected in the footpad at day 0. Exposure of recipients to CO (500 ppm for 8 hrs/day) resulted in graft survival prolongation equivalent to that observed for AdHO-1-treated grafts. This suggests that CO released by HO-1 activity may mediate graft protection. Inhibition of graft rejection after HO-1 overexpression was not dependent on NOS production since administration of the NOS inhibitor L-NMMA did not reduce graft survival of AdHO-1-treated grafts (22+/- 4 days n=4).

HO-1 expression has been previously associated with inhibition of allogeneic chronic rejection and induction of accommodation of xenogeneic hearts. Our results show that HO-1 overexpression resulted in inhibition of acute allograft rejection and was associated with inhibition of MLR responses and accumulation of CD25+ cells in the grafts. This protective effect was possibly mediated through release of CO.

Abstract# 37

SELECTIVE BLOCKADE OF CD28-B7 INTERACTION, BUT NOT OF CTLA4-B7, WITH A SCFV-ALPHA 1 ANTITRYPSIN FUSION PROTEIN. Bernard Vanhove,[1] Geneviève Laflamme,[2] Flora Coulon,[1] Daniel Olive,[1] Roland Buelow,[2] Jérôme Tiollier,[2] Jean-Paul Soulillou.[1] *[1]U437, INSERM, Nantes, France; [2]Sangstat Europe, Lyon, France; [1]U119, INSERM, Marseille, France.*

Among several other co-stimulatory molecules, CD28 and CTLA-4 participate in the regulation of T cell activation. CD28 ligation by CD80 and CD86 (B7 molecules) stabilizes interaction with APC and directly signals T cells for activation, whereas engagement of CD152 (CTLA-4) by B7 inhibits expression of activation antigens, cytokine production, and proliferation, and is required for unresponsiveness to be induced. Therefore, having a molecule selectively blocking CD28 without interfering with the CTLA-4/B7 pathway might result in a reagent useful for inducing tolerance in vivo. In order to evaluate this concept, we collected a series of anti-human CD28 antibodies and compared the activity of their Fab fragments. One of them inhibited the CD28-dependant adhesion of T cells to B7-transfected mouse fibroblasts by 90% at 3 µg/ml. We produced in E. Coli and CHO cells a monovalent single chain Fv recombinant form of this antibody that we fused with the human alpha-1-antitrypsin, in order to prolong its half-live in vivo. In allogenic mixed lymphocyte reactions (MLR), monovalent anti-CD28 Fab fragments or recombinant protein, but not parental divalent antibody or Fab2 fragments, inhibited proliferation by 50 to 95%, in a dose-response relationship. Messenger RNA for INFγ and IL-2 were reduced in these MLR whereas IL10 was increased, as compared with control reactions. Interestingly, monovalent anti-CD28 did inhibit superantigen-induced proliferation of T cells measured after 24h by up to 100%, whereas anti-B7 antibodies or CTLA4Ig started to reduce proliferation only 3 days after stimulation. Secondary T cell proliferative responses to specific alloantigen were also deeply inhibited by addition to the primary culture of monovalent anti-CD28, but not of anti-B7 antibodies. This effect could be reversed by addition of IL-2. Our data suggest that blocking CD28, while leaving CTLA4/B7 interaction undisturbed, reduces T cell proliferation and induces an alloantigen-specific T cell hyporesponsiveness.

Abstract# 38

BLOCKADE OF THE MEMBRANE LYMPHOTOXIN PATHWAY INHIBITS CD8⁺ T CELL-MEDIATED REJECTION. Jun Wang, Zhong Guo, Lingzhong Meng, Qiang Wu, Oliver Kim, John Hart, Gang He, J. Richard Thistlethwaite, Maria-Luisa Alegre, Yang-Xin Fu, Kenneth A. Newell. [1]*The University of Chicago, Chicago, IL.*

Rejection mediated by CD8⁺ T cells is relatively resistant to blockade of the CD28 and CD40 pathways. Herein the effect on CD8⁺ T cell-mediated intestinal allograft rejection of agents that block the LIGHT costimulatory and/or the membrane lymphotoxin (mLT) pathways was examined. **Methods.** Allografts from B6C3F1 donors were transplanted into C57BL/6 wild-type, CD4$^{-/-}$CD8$^{-/-}$ or mLT deficient (LTα$^{-/-}$) recipients. Recipients were treated with LTβRIg (binds LIGHT and mLT), an anti-LTβ mAb (binds mLT only), or CTLA4Ig. Rejection was scored from 0 (normal) to 3 (severe rejection) based on histology at day 14. **Results.** All syngeneic grafts displayed normal histology (rejection grade (RG) 0). LTβRIg inhibited rejection by both CD4$^{-/-}$ and CD8$^{-/-}$ recipients (RG 1.0±0.9 vs 2.6±0.8 control, $p < 0.01$ and 1.0±0 vs 3.0±0 control, $p < 0.001$ respectively). Although neither LTβRIg nor CTLA4Ig alone inhibited rejection in wild-type mice (RG 2.4±0.5 and 2.6±0.7 respectively vs 2.4±1.0 control), the combination did (RG 1.1±1.1, $p = 0.02$). Treatment of mice with an anti-LTβ mAb inhibited rejection in CD4$^{-/-}$ but not CD8$^{-/-}$ mice (RG 0.3±0.5, $p < 0.01$ and 2.0±0, p = ns). There was also a trend toward decreased rejection in LTα$^{-/-}$ mice (RG 1.5±0.5, $p = 0.07$). Disruption of these pathways did not result in an intrinsic T cell defect in that RAG$^{-/-}$ recipients reconstituted with T cells from naive LTα$^{-/-}$ mice rejected allografts promptly and the proliferation of CD8⁺ or CD4⁺ T cells from transplanted LTβRIg-treated mice was not impaired in an allogeneic MLR. Further supporting an effect on non-T cells, intragraft Mig and IP-10 expression were dramatically reduced in LTβRIg-treated CD4$^{-/-}$ and wild-type recipients as determined by real-time RT-PCR. Little Mig or IP-10 mRNA was detected in treated or untreated CD8$^{-/-}$ recipients. **Conclusions.** 1. These data provide the first evidence that mLT regulates CD8⁺ T cell-mediated rejection and suggest that targeting this pathway may be of therapeutic value in transplantation. 2. Results obtained with an anti-LTβ mAb suggest that mLT is more important for CD8⁺ than for CD4⁺ T cell-mediated rejection. 3. Combining agents that inhibit CD4⁺ T cells (i.e., CTLA4Ig) with agents that block mLT may be beneficial therapeutically. 4. Disruption of the mLT pathway affects T cells indirectly. 5. Mig and IP-10 may play a greater role in rejection mediated by CD8⁺ T cells than that mediated by CD4⁺ T cells.

Abstract# 39

INDUCTION OF TH2 RESPONSE TO CARDIAC MYOSIN PREVENTS ACUTE REJECTION OF ALLOGENEIC HEART GRAFTS IN MICE. Eugenia V. Fedoseyeva,[1] Koji Kishimoto,[2] Hillary Rolls,[1] Victor Dong,[2] Anna Valujskikh,[3] Peter S. Heeger,[3] Mohamed H. Sayegh,[2] Gilles Benichou.[1] [1]*Schepens Eye Research Inst, Harvard Medical School, Boston, MA;* [2]*Brigham and Women's Hospital, Harvard Medical School, Boston, MA;* [3]*Case Western Reserve University School of Medicine, Cleveland, OH.*

We have previously demonstrated that heart allotransplantation leads to the induction of Th1 type autoimmunity to cardiac myosin (CM), a heart-specific antigen known to be the target autoantigen in autoimmune myocarditis. Here, we show that in single MHC class I-mismatched combination A/J (Kk AkEk) - A.TL (Ks AkEk), induction of Th2 type CM-specific response either via pre-transplant immunization of adult recipient mice with CM emulsified in IFA or via CM/IFA immunization of neonates results in blockade of acute rejection and significant prolongation of heart allograft survival (100 ± 25 days and 71 ± 19 days, respectively). Histological examination of surviving donor hearts from adult CM/IFA-treated mice displayed well-preserved epicardium and moderate cell infiltration confined to endocardium. T cell responses induced in mice with prolonged heart survival were analyzed using ELISPOT. The frequencies of both IFNg (Th1 cytokine)- and IL-5 (Th2 cytokine)-producing T cells specific to donor MHC antigens and to CM were evaluated. Spleen cells from untreated recipients that acutely rejected the allografts exhibited vigorous expansion of IFNg-producing alloreactive and CM-specific T cells (Th1 response) but no significant IL-5-production (Th2 response). In contrast, in non-rejecting mice, the frequency of alloreactive Th1 cells was reduced to levels observed in naive mice (100 IFNg spots per million of T cells) while high numbers of IL-5-producing CM- and allo-specific T cells were recorded. Our data show that single injection of CM in IFA resulted in induction of Th2 type CM- and allo-specific responses while Th1 alloimmunity was downregulated. No expansion of alloreactive and CM-specific Th2 cells was detected in control mice injected with IFA alone that acutely rejected the grafts (11 days). Our study provides the first demonstration that modulation of organ-specific autoimmunity to CM in the absence of any other immunosuppression leads to abrogation of acute rejection, a finding which may provide the basis for the design of novel selective therapy in transplantation.

Abstract# 40

STRATEGY FOR PREVENTION OF VASCULAR REJECTION: INHIBITION OF ECTODOMAIN SHEDDING OF HEPARIN-BINDING EPIDERMAL GROWTH FACTOR-LIKE GROWTH FACTOR BLOCKS MIGRATION AND PROLIFERATION OF VASCULAR SMOOTH MUSCLE CELLS IN RAT AORTIC ALLOGRAFTS. Ildeok Kim,[1] Shigeki Higashiyama,[2] Masanori Nakamura,[3] Hiroto Egawa,[1] Koichi Tanaka.[1] [1]*Transplantation and Immunology, Kyoto University, Kyoto, Japan;* [2]*Biochemistry, Osaka Univ. Facul. Med., Suita, Japan;* [3]*Nippon Organon K K., Osaka, Japan.*

Introduction: Heparin-binding epidermal growth factor-like growth factor (HB-EGF) has been recognized to play a significant role in development of atherosclerosis as a potent mitogen and a chemotactic factor for vascular smooth muscle cells (SMCs). We previously demonstrated that HB-EGF might be also involved in vascular rejection in clinical liver transplantation. We recently showed that ectodomain shedding of HB-EGF, which induced mitogenic and chemotactic effect through transactivation of EGF receptor, was blocked by a kind of matrix metalloproteinase (MMP) inhibitor . The purpose of this study is to analyze the localization of HB-EGF positive cells and to investigate the effect of a MMP inhibitor in rat vascular rejection model. Methods: F344 (RT1^{lvl}) and LEW (RT1^{l}) rats were used as donors and recipients, respectively. Orthotopic abdominal aortic transplantation (AoTx) was performed, and recipients were divided into three groups as follows. Group I (n=6); Syngeneic AoTx (F344 to F344), Group II (n=9); Allogeneic AoTx (F344 to LEW), Group III (n=6); Allogeneic AoTx (F344 to LEW) treated with a MMP inhibitor KB-R7785 (100 mg/kg/day, i.p., from a day before transplant to a day before harvest). Aortic grafts were harvested 2 months after transplant, stained by H-E, EVG and immunostained for α-actin in SMCs, monocytes/macrophages, and HB-EGF. Results: No vasculopathy was observed in Group I. In Group II, intimal thickening and medial narrowing with migration and proliferation of vascular SMCs were observed. In Group III, neither migration nor proliferation of vascular SMCs were observed although ED-1-positive foamy cells were detected in subendothelial space. HB-EGF was significantly stained in neointima and residual vascular SMCs of media in Group II, and also stained in vascular SMCs of media in Group III, whereas minimal HB-EGF staining was detected in vascular SMCs of media in Group I. Conclusions: HB-EGF was significantly localized in vasculopathy of rat aortic allografts and inhibition of ectodomain shedding of HB-EGF completely blocked migration and proliferation of vascular SMCs. Ectodomain shedding of HB-EGF may be suggested to play a crucial role in pathogenesis of vascular rejection.

Abstract# 41

INHIBITION OF DONOR BRAIN DEATH-RELATED INFLAMMATION OF TANSPLANTED KIDNEYS BY RECOMBINANT SOLUBLE P-SELECTIN GLYCOPROTEIN LIGAND (sPSGL). Martin Gasser,[1] Ana Maria Waaga,[2] Igor Laskowski,[1] Miriam S. Lenhard,[2] Gray D. Shaw,[3] Wayne W. Hancock,[4] Nicholas L. Tilney.[1] [1]*Dept. of Surgery, Surgical Research Laboratory, Brigham and Women's Hospital, Harvard Med School, Boston, MA;* [2]*Dept. of Nephrology, Laboratory of Immunogenetics and Transplantation, Brigham and Women's Hospital;* [3]*Genetics Institute, Cambridge, MA;* [4]*Millenium Inc., Cambridge, MA.*

Because grafts of potentially diminished quality are increasingly accepted to reduce the severe shortage of donor organs, it has become apparent that several donor-associated risk factors may influence adversely short and long-term outcome after transplantation (Tx). We have previously shown that donor brain death (BD) leads to accelerated acute kidney allograft rejection. sPSGL-Ig inhibits selectin activity and subsequent inflammatory events in organs damaged by I/R injury. To define its effects on late graft changes, we compared kidney allografts from BD- and living donors in a chronic rejection (CR) model. After induction of gradual onset BD F344 donor rats were monitored over 6 hrs, the kidneys were then grafted into LEW recipients. Kidneys from living donors (LD, group I) and normotensive BD-donors (MAP>80 mmHg, group II) served as controls. To determine the effect of sPSGL-Ig, donors were treated i.v. (50μg) 3 hrs after BD. A second dose was given to the recipient after Tx (n=8, group III). All recipients received low dose cyclosporin A (1.5 mg/kg/10d) as a standard treatment protocol in this CR model. Grafts were analyzed 6 hrs after BD and before Tx by semiquantiative RT-PCR. Protein levels (24 hr urine) were measured serially. After 200 d post Tx the kidneys were examined histologically and in the RNAse Protection assay for cytokines. RT-PCR analysis showed higher transcription of ICAM-1, IL-1β, MCP-1, TNF-α, β, IFN-γ, IL-2, 3, 4, 5, 6 and TGF-β in untreated BD controls before Tx whereas those treated with sPSGL-Ig were virtually normal. Protein levels from animals in group I and II, but not in group III, increased progressively from 12 weeks after Tx (107.8 and 201 mg vs 21.5mg). Histologically, grafts from sPSGL-Ig treated animals showed only minor changes 200 d after Tx whereas those from untreated controls revealed moderate (group I) to severe (group II) signs of CR with upregulated cytokines and chemokines. The results suggest that treatment with sPSGL-Ig inhibits donor-BD associated early and late events evolving in renal allografts. This may be therefore of particular interest for use in grafts of potentially diminished quality.

Abstract# 42

ANTI-HLA ANTIBODY-MEDIATED ACTIVATION OF AIRWAY EPITHELIAL CELLS INDUCES THE PRODUCTION OF SEVERAL FIBROGENIC GROWTH FACTORS FOLLOWED BY APOPTOTIC CELL DEATH. Andres Jaramillo, Leiying Zhang, Elbert P. Trulock, G. Alexander Patterson, T. Mohanakumar. [1]*Surgery;* [2]*Medicine;* [3]*Pathology & Immunology, Washington University School of Medicine, St. Louis, MO.*

Bronchiolitis Obliterans Syndrome (BOS) is the most common cause of long-term morbidity and mortality after lung transplantation (LT). BOS is identified histologically by fibrosis of the lamina propria and lumen. Our studies indicate that development of anti-HLA antibodies (Ab) after LT plays an important role in the pathogenesis of BOS. Airway epithelial cells (AEC) have been shown to be immunological targets during lung allograft rejection. In addition, AECs have the ability to produce several fibrogenic growth factors. The objectives of this study were: 1) to determine the ability of anti-HLA class I Abs to activate signal transduction and proliferation of AECs, 2) to determine the ability of anti-HLA-activated AECs to induced the up-regulation of proliferation of lung fibroblasts, and 3) to identify the fibrogenic growth factors produced by AEC upon anti-HLA Ab binding.

Binding of the W6/32 anti-HLA mAb stimulated DNA synthesis in AECs after 24 hours. This was induced through the activation of intracellular calcium influx and tyrosine phosphorylation. The activation of AECs also resulted in growth factor production at 24 h post-activation measured by means of the up-regulation of proliferation of lung fibroblasts in trans-well experiments. Several anti-growth factor blocking Abs showed significant inhibition of lung fibroblast proliferation i.e. anti-PDGF: 63% inhibition, anti-HB-EGF: 59% inhibition, anti-FGF: 59% inhibition, and anti-IGF-1: 48% inhibition. Interestingly, anti-HLA mAb binding also resulted in a significant up-regulation of Annexin-5 expression (apoptosis) by the AECs after 48 hours.

Our findings indicate that activation of AECs by anti-HLA Abs induces the production of a complex profile of growth factors that result in increased fibroblast proliferation. Further, activated AECs undergo apoptotic cell death. Activation of AECs by anti-HLA Abs produced after LT in vivo may be a contributing factor in the immunopathogenesis of BOS following LT.

Abstract# 43

ACTIVATION OF T CELLS IN AN INTESTINAL ALLOGRAFT TRIGGERS REJECTION BY INFLUENCING HOST T CELL ACTIVATION AND MIGRATION. Zheng J. Zhang,[1] Terrance A. Barrett,[1] Levent Kaptanoglu,[1] David Ivancic,[1] Frank P. Stuart,[1] Jonathan P. Fryer.[1] [1]*Surgery and Medicine, Northwestern University, Chicago, IL.*

Small bowel (SB) allograft rejection is more aggressive than rejection in other organ allografts. The large lymphoid cell population in SB grafts may play an important role. To study how donor T cells may influence host alloresponses, we utilized a single class I MHC antigen (L^d) disparate model, in which SB grafts from DO11.10 OVA specific T cell receptor (TCR) transgenic mice(BALB/c background, L^{d+}) were transplanted into BALB/c (L^{d+}), isografts) or BALB/cH-2dm2 (dm2) mutant mice (L^d allografts). Severe rejection developed by POD20 in 80% of DO11.10 to dm2 allografts if transgenic T cells in the grafts were activated by OVA peptide (200ug, ip on POD 5,6, and 7), while only 20% of allografts that were not given OVA developed rejection by POD30. No isografts rejected by POD30 regardless of OVA administration. This result indicates that donor T cell activation following SB transplant accelerates SB allograft rejection. In follow-up studies to evaluate the effect of graft T-cell activation on host-derived effector T cells in spleen and draining lymph nodes (LN), the DO11.10 to dm2 SB allografts were sacrificed on POD 5 before OVA injection or POD 8 with/without OVA injection (POD 5, 6, and 7). Lymphocytes from host spleen and mesentery LN (MLN), donor MLN or Peyer's patches (PP) were harvested and stained with mAbs to mouse L^dThy1, and CD45/CD69/CD25. Tri-color flow cytometric analysis were performed to distinguish the relative populations of donor (L^{d+}) and host T cells (L^d), and to determine the phenotypes of host T cells. We found that donor T cells migrated to the host spleen by POD5. At POD8, the proportion of donor T cells increased dramatically after OVA injection from 4% to 42%, but decreased (10% to 3%) without OVA. The proportion of $CD69^+$ and $CD25^+$ host T cells increased remarkably after activation of donor T cells. Furthermore, the percentage of activated host T cells, and B cells in the graft mucosa was also increased. These results indicate that activation of T cells residing in an intestinal allograft can cause accumulation of donor T cells in host lymphoid tissues, activate host T cells in these sites, alter recipient T cell trafficking, and trigger allograft rejection.

Abstract# 44

OVER EXPRESSION OF SMAD2 AND CO-LOCALIZATION WITH TGF-B1 IN CHRONIC REJECTION (CR). Xiao L. Jiang, Shenglin Ma, Clement Asiedu, Juan L. Contreras, Devin Eckhoff, Francis T. Thomas, Judith M. Thomas. [1]*University of Alabama at Birmingham, Birmingham, AL.*

Introduction: CR is the leading cause of long-term (LT) kidney transplant (KTx) failure with contemporary immunosuppression. Molecular mechanisms regulating CR are uncertain. A hallmark of CR is intragraft expression of TGF-b1. This cytokine regulates a diverse array of biological processes including proliferation, extracellular matrix production, fibrosis, and apoptosis. TGF-b1 initiates a signaling cascade by binding the type-II TGF-b receptor (TbRII), an interaction that induces TbRII to heterodimerize and activate the TGF-b receptor type 1 (TbRI). Activated TbRI phosphorylates transcription factor Smad2 to facilitate heterodimer formation with Smad4, nuclear translocation, and multigenic transcription. Previously, we showed active TGF-b1 expression was increased in biopsies of LT KTx from rhesus monkeys with CR. In contrast, TGF-b1 was not detected in LT KTx exhibiting robust tolerance and freedom from CR. Here we examined the hypothesis that Smad2 expression might play a role in CR. **Methods:** Western blot analysis and immunohistochemistry were used to investigate essential components of the TGF-b1 signaling pathway, including TbRII, Smad2, and collagen III in monkey kidney cells. We studied biopsies from 5 LT KTx survivors bearing MHC class I and class II mismatched KTx with or without CR. Two LT recipients, 95C163 and 93B628, were induced with only anti-CD3 immunotoxin (IT as either IgG or F(Ab) conjugate) on day 0 (95C163) or pre-Tx day -7 (93B628). They exhibited clinical and histological CR and succumbed at 721 and 1398 days post-KTx, respectively. In contrast, 3 LT recipients, AXE, TIJ and T4P, were induced with IT plus 2-weeks of DSG and exhibited no evidence of CR at 1543, 1511, and 1470 days post-KTx, respectively. We also used the COS 7 monkey kidney cell line as a controlled *in vitro* model to examine Smad2 expression with/without gene transfer of active TGF-b1 vector at MOI of 50-500. **Results:** The KTx biopsies of LT CR^+ KTx exhibited a 10-fold increase in Smad2 protein expression compared to those without Cr. Smad2 was detected in ~ 15% of the interstitial cells in the CR^+ KTx biopsies but was absent from CR KTx biopsies. Serial sections revealed cellular co-expression of active TGF-b1, and TbRII, and collagen III. In COS 7 cells, over-expression of active TGF-b1 by gene transfer upregulated steady-state expression of Smad2, TGF-b1, TbRII, and collagen III. **Conclusion:** The results show TGF-β1 upregulates Smad2 expression in primate kidney cells and that Smad2 is upregulated in CR in LT primate KTx. They suggest a regulatory role for Smad2 in excessive activation of the TGF-b1 signaling pathway in the pathology of CR.

Concurrent Session 5:
Kidney Transplantation: Recipient Factors and Outcomes

Abstract# 45

RENAL FUNCTION IN THE FIRST YEAR AFTER TRANPLANTATION PREDICTS LONGTERM SURVIVAL. Christopher P. Johnson,[1] Maureen McBride,[2] Wida S. Cherikh,[2] Christine B. Tolleris,[2] Barbara A. Bresnahan,[3] Sundaram Hariharan.[3] [1]*Transplant Surgery, Medical College of Wisconsin, Milwaukee, WI;* [2]*United Network for Organ Sharing, Richmond, VA;* [3]*Division of Nephrology, Medical College of Wisconsin, Milwaukee, WI.*

Background: In a previous analysis of the UNOS/OPTN database for renal transplantation, we reported that between 1988 and 1996, the half-life (t½) for cadaveric donor (CD) transplants increased from 7.9 to 13.8 yrs. In the current study we examined the relationship between renal function in the first year, (expressed as Cr_{6mo}, Cr_{1yr} and ΔCr (the change in Cr between 6mo and 1yr) and long term outcome. Our aim was to define the relationship such that t½ can be predicted within the first year after transplantation especially for CD transplants. **Methods and Results:** Between 1988 and 1998 105,743 adult renal transplants (73.4 % CD, 26.6 % LD), were reported to the OPTN registry. Demographic and immunological characteristics for CD transplants have not changed substantially over the past 10 years with the exception of donor age. Over this 10yr period, Cr_{1yr} has steadily improved (1.81 to 1.6) as has Cr_{6mo} (1.82 to 1.67). In 1988, 41% of CD transplants had $Cr_{1yr} \leq 1.5$, compared to 51% of transplants in 1998. The differences are even greater when adjusted for donor age. Interestingly, there has not been a significant change in Δ Cr over the past 10 yrs (mean Δ 0.17 in 1988 and 1998). The t1/2 for CD transplants according to Cr_{1yr} and delta Cr. is shown.

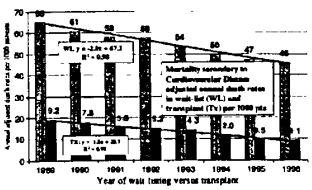
Serum Creatinine (mg/dl) at 1 yr	1/2 life (Yrs)	Δ Creatinine (mg/dl) 1yr-6mo	1/2 life (Yrs)	1 Yr Serum Creatinine and Δ creatinine.	1/2 life (Yrs)
<1.0	11.2	no change	10	≤1.5 and <0.3	11.6
1.0-1.5	10.9	0.1-0.2	9.6	≤1.5 and ≥0.3	9.9
1.6-2.0	9.2	0.3-0.4	8.0	>1.5 and <0.3	8.9
2.1-2.5	7.2	0.5-0.9	6.3	>1.5 and ≥0.3	6.0
2.6-3.0	5.3	1.0-1.9	3.2		
>3.0	3.2	1.9-2.9	1.9		
		>3.0	1.6		

In a Cox Regression analysis for long-term graft survival, both serum Cr at 1 yr and Δ Cr were important predictors of t½. **Conclusions:** The improvements in t½ seen over the past 10 yrs may be due to achieving better renal function within the first 6 months. Even though the Δ Cr has not changed over 10 yrs., it remains a strong predictor of t½. By using a combination of $Cr_{1yr} \geq 1.6$ and $\Delta Cr \geq 0.3$ it is possible to identify a subset of transplant recipients early on, with a predictably shortened t½, which in turn may be eligible for secondary intervention trials.

Abstract# 46

HAVE THE IMPROVEMENTS IN SURVIVAL IN RENAL TRANSPLANTATION EXCEEDED THE IMPROVEMENTS IN SURVIVAL ON DIALYSIS? Herwig-Ulf Meier-Kriesche,[1] Friedrich K. Port,[1] Akinlolu O. Ojo,[1] Julie A. Arndorfer,[1] Diane M. Cibrik,[1] Bruce Kaplan.[1] *[1]Department of Internal Medicine, University of Michigan, Ann Arbor, MI.*

Background: Both transplant and dialysis outcomes have improved over recent years. In addition recent studies have clearly documented a survival benefit of transplantation versus maintenance hemodialysis. One question that has not been addressed is whether the survival improvements seen in transplantation and the survival improvements seen in dialysis have been of similar magnitude. The reception among many nephrologists has been that survival improvements in transplantation have exceeded those of dialysis. This study specifically addressed this question. Methods: Data was collected from the U.S. Renal Transplant Registry and the U.S. Renal Data System (USRDS). 96,850 patients placed on the waiting list from 1988-1996 were analyzed Annual adjusted mortality rates were calculated by calendar year of placement on the renal transplant waiting list and year of transplant for renal transplant recipients. Data were plotted and curve fitting was used to estimate the slope of the change of the adjusted mortality rates over the period studied. Results: Annual adjusted death rates for both wait-listed patients and transplant recipients decreased for both groups throughout the study period. Slope analysis of the improvement for both groups reveals a similar improvement in mortality. The relative risk for patient death improved by 30% for transplant recipients, while it improved by 23% for wait-listed patients. Cause-specific mortality slope analysis for cardiovascular and infectious deaths showed similar progressive and equal improvements. Conclusion: Mortality rates have improved in a similar fashion for both transplant recipients and wait-listed patients on dialysis. This applies both for overall mortality and for major cause-specific mortality. These favorable trends most likely represent equal advances in transplantation, dialysis and general medical care.



Abstract# 47

THE IMPACT OF RENAL GRAFT DYSFUNCTION ON GRAFT SURVIVAL IS VARIABLE AND DEPENDENT ON THE TYPE OF INJURY RESPONSIBLE FOR THE DYSFUNCTION. Viken Douzdjian,[1] Ravi Parasuraman,[2] Atsushi Yoshida,[1] Marwan Abouljoud.[1] *[1]Surgery, Henry Ford Hospital, Detroit, MI; [2]Nephrology, Henry Ford Hospital, Detroit, MI.*

The literature on the impact of delayed graft function (DGF) on acute rejection (AR) or renal graft survival is inconsistent. DGF alone, AR alone or DGF+AR may represent different manifestations of various types and degrees of injury to the graft. The impact of these manifestations on graft survival may not be an all-or-none phenomenon and may depend on the nature of the injury. The purpose of this study is to identify donor and recipient factors which predict DGF alone, AR alone and DGF+AR and to evaluate whether the impact of these conditions on graft failure is influenced by these factors. Between 1/1990 and 08/2000 674 kidney transplant were performed at our center. Of these, 357(53%) had no DGF/no AR, 108(16%) had DGF alone, 135(20%) had AR alone and 74(11%) had DGF+AR. A logistic regression analysis model with 17 donor and recipient covariates was developed for each of the following tested outcomes: DGF alone, AR alone, DGF+AR and death-censored graft failure. The following covariates were found to be independent predictors (P<0.05 shown; rr=relative risk, <0.95 or >1.05 shown). For DGF alone: older donor (P=0.02) and longer cold ischemic time (P=0.02). For AR alone: older recipient (P=0.006) and diabetes (P=0.02, rr=2.99). For DGF+AR: longer time on dialysis prior to transplant (P=0.009), Black recipient (P=0.01, rr=2.43) and older donor (P=0.02). For graft failure: Black recipient (P=0.004, rr=2.43), diabetes (P=0.02, rr=2.42), older donor (P=0.03) and longer time on dialysis prior to transplant (P=0.03). All of these factors remained predictors when antibody induction (used vs not used), type of calcineurin inhibitor (tacrolimus vs cyclosporine) and CellCept(used vs not used) were incorporated in the analyses. The 5 year actuarial death-censored graft survival was 81% for DGF alone, 69% for AR alone and 45% for DGF+AR. In conclusion, the risk factors associated with DGF alone, AR alone or DGF+AR were distinctly different. The impact of DGF alone, AR alone or DGF+AR on graft failure varied depending on the risk factors associated with each of these states and not necessarily the presence of that state per se.

Abstract# 48

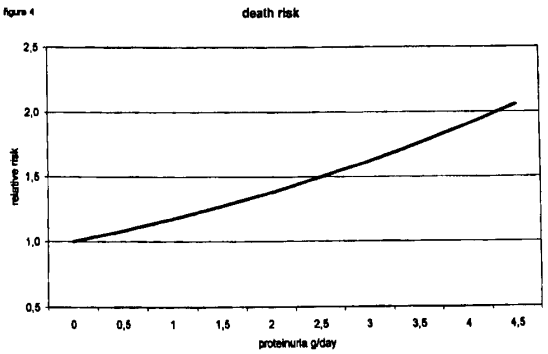
PROTEINURIA AFTER RENAL TRANSPLANTATION INCREASES NOT ONLY THE RISK FOR GRAFT FAILURE BUT ALSO THE DEATH RISK. Joke I. Roodnat,[1] Paul G.H. Mulder,[2] Teun Gelder van,[1] Jacqueline Rischen-Vos,[1] Iza C. van Riemsdijk,[1] Jan N.M. IJzermans,[3] Willem Weimar.[1] *[1]Internal Medicine I, University Hospital Rotterdam-Dijkzigt, Rotterdam; [2]Epidemiology and Biostatistics, Erasmus University Rotterdam, Rotterdam; [3]General Surgery, University Hospital Rotterdam-Dijkzigt, Rotterdam, The Netherlands.*

Background: Proteinuria has been shown to be associated with an increased (cardiovascular) death risk.

Methods: In the population transplanted in Rotterdam before 1995 we studied the influence of proteinuria, at one year after transplantation, on the failure risk in all 722 recipients of a kidney graft still surviving with a functioning graft. Proteinuria was analyzed both as a categorical and as a continuous variable.The other one year variables were: serum cholesterol, serum creatinine and blood pressure. Both immunological and non-immunological general variables were included. A multivariate Cox proportional hazards analysis was performed. Results: The influence of proteinuria as a categorical variable on graft failure rate censored for death was significant and showed no interaction with any of the other variables. Proteinuria had an adverse effect on the graft failure rate (RR=2.03). Proteinuria as a continuous variable also significantly influenced the risk and there was interaction with original disease (glomerulonephritis, hypertension and systemic diseases). The risk increased with an increasing amount of proteinuria. The influence of proteinuria as a categorical variable on the rate ratio (RR) for patient failure was significant without interaction. Proteinuria almost doubled the death risk. The influence of proteinuria as a continuous variable was significant too without interaction. Proteinuria increased both the cardiovascular and non-cardiovascular death risks.

Conclusion: Proteinuria after renal transplantation is an important independent variable increasing the risk of both graft failure and death.

figure 4



Abstract# 49

FUNCTIONAL RESERVE AND HYPERFILTRATION AFTER CADAVERIC RENAL TRANSPLANTATION. Elisabetta Bertoni,[1] Alberto Rosati,[1] Maria Zanazzi,[1] Lorenzo Di Maria,[1] Luciano Moscarelli,[1] Marco Gallo,[1] Maurizio Salvadori.[1] *[1]Renal Transplantation, Renal Unit Careggi University Hospital, Florence, Italy.*

Hyperfiltration (Hy)and kidney functional reserve is well known in the case of living renal transplantation both for the donor (residual kidney)and the recipient(transplanted kidney).Aim of this study was to evaluate the degree of Hy after cadaveric renal transplantation and which factors had the main influence on Hy.

80 cadaveric renal transplants with an actual follow-up of at least 1 year were studied(40 patients received a graft from a donor >55 years,40 from a donor <55 years).Creatinine clearance evaluated according Gault-Cockcroft(CrClGC)strictly correlated with creatinine clearance measured (p<0.0001)both at 6 months and at 1 year(53.3 ±14.29ml/min vs 52.15±11.29 ml/min at 6 months).Then CrClGC was a good estimate of glomerular filtration rate(GFR).

1/2 CrClGC (one kidney)of younger donors was 44.16±13.8 ml/min with respect to 37.21±8.6 ml/min of older donors(p=0.008).The overall increase of CrClGC after 6 months was 56.83% and remained stable at 1 year(58.9%).In the case of older donors the increase was 40.5% with respect to an increase of 69.02% in the case of younger donors(p<0.01).The GFR increase both at 6 months and at 1 year either in recipients from young and old donors was strictly and inversely related to the donor calculated GFR (r= -0.719;p< 0.0001).Delayed graft function (DGF)and/or rejections were the two main causes of a smaller increase in GFR:32.25% for patients experiencing DGF vs 75% for patients not experiencing DGF and/or rejections (p=0.0029).

Cadaveric renal transplants do have an hyperfiltration due to a functional reserve.The hyperfiltration develops early post-transplantation, then stabilizes.Old kidneys,DGF and rejections are the main causes of a reduced hyperfiltration,that anyway is strictly and inversely related to the donor GFR.It is well documented that the hyperfiltration degree is well related to the stimulus coming by the recipient weight.Our data demonstrate that the hyperfiltration stimulus could exert a more powerful effect on kidneys with lower GFR. The hyperfiltration state is stable for at least one year.Our prospective study has 1 year observational period.Whether the hyperfiltration could induce a chronic allograft failure or not remains to be stated.

Abstract# 50

EFFECT OF CALCIUM AND CHOLECALCIFEROL TREATMENT ON BONE MINERAL DENSITY IN RENAL TRANSPLANT RECIPIENTS. Martin Wissing,[1] Nilufer Broeders,[1] André Schoutens,[2] Bernard Stallenberg,[3] Brigitte Borré,[1] Daniel Abramowicz.[1] *[1]Nephrology, Hopital ULB-Erasme, Brussels, Belgium; [2]Nuclear Medicine, Hopital ULB-Erasme, Brussels, Belgium; [3]Radiology, Hopital ULB-Erasme, Brussels, Belgium.*

Background: Recipients of renal transplants have been reported to loose 5 to 10% of their bone mass during the first year after transplantation. Post-transplant osteoporosis results in an increased incidence of bone fractures. We conducted a prospective randomised trial to compare the effect of calcium supplementation with or without cholecalciferol (vitamin D3) on bone mineral density (BMD) in renal transplant patients treated with a low-dose glucocorticoid immunosuppressive regimen.

Methods: From January 1999 to August 2000 we randomised 87 renal transplant recipients (47 men and 40 women) to receive either 400 mg daily oral calcium (group Ca, N=44) or the same dose of calcium in association with a monthly dose of 25000 IU of vitamin D3 (group Ca-VitD, N=43). Patients received tacrolimus (N=74) or cyclosporine microemulsion (N=13) based immunosuppression. Prednisolone was tapered to 5 mg by 6 months and stopped in low-risk patients. BMD was measured by dual energy absorptiometry at baseline and at 1 year. Patients were also monitored for serum levels of calcium, phosphorus, parathormone and 25-OH vitamin D.

Results: Calcium and vitamin D3 supplementation was well supported and none of the patients had to interrupt the treatment because of clinical intolerance. Calcium supplementation had to be permanently discontinued because of the development of hypercalcemia in 5 patients of the Ca group and 4 patients of the Ca-VitD group (P=NS). At submission of the abstract 23 patients have completed BMD measurements at 1 year of follow-up. In the Ca group (N=12) lumbar spine BMD was 1.03±0.12 and 0.99±0.15 gr/cm^2 at baseline and 1-year respectively (P=NS). In the Ca-VitD group lumbar spine BMD was 1.03±0.12 and 1.03±0.16 gr/cm^2 at baseline and 1-year respectively (P=NS). Similarly, femoral neck BMD remained stable in both groups. Vitamin D3 supplementation significantly increased 25-OH vitamin D serum levels (14.3±8.3 ng/ml in Ca patients vs. 27.5±12.2 ng/ml in Ca-VitD patients at 6 months; P<0.0001).

Conclusions: Our preliminary data suggest that kidney transplant recipients supplemented with calcium with or without vitamin D3 do not loose bone mass during the first post-transplant year when treated with low-dose prednisolone. Vitamin D3 therapy corrects 25-OH vitamin D levels without increasing the incidence of hypercalcemia.

Abstract# 51

PRETRANSPLANT DIALYSIS MODALITY IS ASSOCIATED WITH LONG TERM RENAL ALLOGRAFT SURVIVAL AFTER CADAVERIC BUT NOT LIVING DONOR TRANSPLANTION. Brian J. Gallay,[1] J. Michael Cecka,[2] Richard V. Perez.[3] *[1]Division of Transplant Medicine, UC Davis Medical Center, Sacramento, CA; [2]Immunogenetics Center, UCLA Medical Center, Los Angeles, CA; [3]Division of Transplant Surgery, UC Davis Medical Center, Sacramento, CA.*

The effect of pretransplant dialysis modality on early cadaveric renal transplant function has been studied, but little data are available for the effect of dialysis modality on long term graft survival after either cadaveric or living donor transplantation. We addressed this question by studying the UNOS database registry for renal transplants from 1994 to 1999. Delayed graft function (DGF) and early acute rejection (AR) rates as well as projected graft survival (including death with functioning graft) were compared for 10,400 patients on peritoneal dialysis (PD) and 29,826 patients on hemodialysis (HD) before cadaveric transplant. Projected graft survival was also studied for living donor transplants in 3937 PD and 10,253 HD patients. Pretransplant dialysis modality did not affect living donor transplant survival, but cadaveric graft survival at one and three years post-transplant as well as projected half-life were significantly better for PD compared to HD recipients (see table).

Dialysis type	DGF (%)	AR at discharge (%)	1 yr survival (%)	3 yr survival (%)	half-life (yrs)
PD	18 8	10 4	88	79	12 9
HD	27 5	10 7	86	76	9 5
p value (log-rank test)	<0 01	0 361	<0 001	<0 01	<0 01

Although early rates of acute rejection were similar, the PD group experienced less DGF. Confirming previously reported data, we found that the HD group included more African-American patients than the PD group (29% vs 18%) and that HD patients were on average four years older than the PD patients. Although these factors may contribute to the different graft survival rates after cadaveric transplantation, they do not appear to affect graft survival after living donor transplantation.

Conclusion: This analysis suggests an effect on long term cadaveric renal allograft survival associated with pretransplant dialysis modality. This effect is not observed for living donor transplant survival. We suggest that factors associated with pretransplant HD may increase the risk of DGF and in turn affect long term cadaveric transplant survival, but they do not affect long term survival of immediately functioning grafts. An immediate pretransplant event such as HD performed before transplant surgery may be one such factor.

Abstract# 52

ELEVATED BODY MASS INDEX (BMI) DOES NOT ADVERSELY EFFECT THE OUTCOME IN RENAL TRANSPLANT PATIENTS. Stuart Greenstein, Wanda Chin, Richard Schechner, Vivian Tellis.

Obesity has been defined by the World Health Organization as a BMI (kg/m^2) greater than 25. It has been used to deny or delay renal transplantation because of reported increased surgical complications and poorer outcomes. However, studies supporting this practice have not been based on consecutive patients nor consistent immunosuppressive therapy.

METHODS: We reviewed the records of 656 patients (age≥18) transplanted at our center from 1/1/90 to 8/31/00. Immunosuppression, by standard protocol, included prednisone (low dose from day 1) and cyclosporine or tacrolimus. Periodic adjustments were made to reflect prevailing practices. Patients were divided into three groups: (1) non-obese, BMI < 25 (mean= 21.7); (2) mildly obese, BMI≥25 (mean=27.5); and (3) obese, BMI ≥30 (mean=35.0). Records were evaluated for race, gender, HLA mismatching (Mm), anastamotic time (AT) and donor source. We analyzed the immediate outcomes of length of hospital stay (LOS) and wound infections. Actuarial patient and graft survival were calculated by using the cumulative log rank test. Significance was determined using Student's test.

RESULTS: Groups were not significantly different for race, gender, and HLA mismatching. The non-obese group was found to be significantly younger and to have a greater percentage of living donors (p<0.001). The disparity in donor source reflected the surgeons' assessment that the use of shorter live-donor blood vessels in obese patients posed an added threat to the kidney. The obese group was found to have a significantly longer mean anastomotic time when compared to the non-obese group (p<0.001). There was no significant correlation between obesity and mortality, graft survival rates or length of hospital stay. Patient survival rates were excellent for all groups at 86% or greater at ten years. The main causes of death were myocardial infarction (n=19) and infection (n=17). Two diabetic non-obese patients developed fascial necrosis. There were no other wound infections in any group.

BMI (Avg)	N	%Pt Survival 1 yr	5 yr	10 yr	%Gft Survival 1 yr	5 yr	10 yr	Mean Age	Sex (F M)	% Black	% Cad	Mean HLA Mm	Mean AT (min)	Mean LOS (days)
<25 (21 7)	330	98 10	95 10	91 90	86 80	78 90	70 40	40 4	40 60	33 3	70	3 2	46 5	12 1
25-30 (27 5)	213	96 20	93 00	88 30	86 70	78 00	67 80	45 7	43 57	36 2	100	3 1	47 7	11 8
>30 (35 0)	113	95 60	91 90	86 90	82 30	73 20	60 60	45 2	40 60	34 5	100	3 0	51 4	12 8

CONCLUSION: Obesity has no significant adverse impact on immediate renal transplant outcomes or long-term patient or graft survival. We conclude that it should not be the sole criteria in denying these patients access to transplantation.

Abstract# 53

OVERWEIGHT IN RENAL TRANSPLANT RECIPIENTS IS ASSOCIATED WITH AN INCREASED RISK OF PROTEINURIA AND HYPERTENSION. Chew-Wong Alfredo, Ron Oscar, Ricalde Guadalupe, Romo Luis, Parra Ana Laura, Reyes-Acevedo Rafael. *[1]Nephrology and Transplants, Hospital de Especialidades Miguel Hidalgo, Aguascalientes, Mexico.*

Overweight is a recognized risk factor for hypertension and proteinuria in general population.

AIM: To evaluate overweight effect in renal transplant recipients (RTR).

PATIENTS AND METHODS: 86 from 200 consecutive patients who received a renal transplant in our center from January 1995 to August 2000 were randomly selected for this retrospective analysis. Overweight was defined as Body Mass Index (BMI)≥27 measured in the last visit (LV).

RESULTS:All RTR received CsA, AZA and PDN, living related donor 95%.HLA matching was similar in boths groups.

	BMI < 27(n=52)	BMI ≥27 (n=34)	p
Age (years)	29 ± 11	35 ± 13	0.03
Male (%)	56	67	0.27
Acute Rejection (%)	21	26	0.56
Hypertension (%)	59	85	0.01
Proteinuria (%)	3	20	0.03
Δ BMI at month and LV	2.3 ± 2.6	4 ± 2.8	0.01
Δ SCr at month and LV (mg/dl)	-0.4 ± 0.6	-0.8 ± 0.5	0.01
SCr at month (mg/dl)	1.4 ± 0.4	1.8 ± 0.5	0.00
SCr in LV (mg/dl)	1.5 ± 0.8	1.4 ± 0.6	0.44
Chronic Rejection (%)	8	6	0.74
Follow-up (months)	22 ± 16	22 ± 18	0.99

Cox analysis identifies overweight as an associated risk factor for development of proteinuria ($p<0.05$, relative risk 4.7).

CONCLUSIONS: Overweight is clearly associated with higher frequency of hypertension and proteinuria in RTR, suggesting such patients develop renal graft hyperfiltration. This findings suggest that overweight may be deleterious for the long-term graft outcome.

Concurrent Session 6: Kidney Transplantation: Factors Affecting Clinical Outcomes

Abstract# 54

IMPROVED RENAL FUNCTION WITH CYCLOSPORINE ELIMINATION IN SIROLIMUS-TREATED RENAL TRANSPLANT RECIPIENTS: ONE-YEAR RESULTS FROM A PHASE II TRIAL. Donald E. Hricik, for the Rapamune Renal Function Study Group. *[1]Division of Nephrology, University Hospitals of Cleveland, Cleveland, OH.*

Background: Sirolimus (Rapamune®), in combination with cyclosporine (CsA), has been shown to reduce the incidence of acute rejection episodes in recipients of renal allografts. When administered as primary therapy in combination with azathioprine or mycophenolate mofetil, sirolimus has a favorable safety profile compared with CsA. We evaluated the effect of CsA elimination on renal function, acute rejection, and safety in sirolimus-treated renal transplant recipients.

Methods: This Phase II, open-label, randomized study was conducted in 17 centers in the United States and Europe. 246 first cadaveric renal allograft recipients were enrolled; 97 patients were randomized to full-dose CsA (microemulsion) plus fixed-dose sirolimus (2 mg/day) [Group A], and 100 patients were randomized to reduced-dose CsA plus concentration-controlled sirolimus (10-20 ng/mL, IMX or LC-MS/MS assays) [Group B]. Patients with delayed graft function which resolved later than day 7 post-transplant were assigned to a rescue group [Group C, n=49]. All patients received standard corticosteroids At the end of month 2 after transplantation, patients in Group B who did not have an acute rejection episode had CsA tapered and eliminated over a 1-month period.

Results: At 12 months after transplantation, renal function was significantly better in sirolimus-treated patients who underwent CsA elimination (Group B) compared with patients who continued to receive full-dose CsA (Group A), as evidenced by improved serum creatinine (1.53 vs 1.92 mg/dL, respectively, *p=0.002*) and Nankivell GFR (68.3 vs 55.6 mL/min, respectively, *p<0.001*). Intent-to-treat analysis at 2 months after transplantation showed a similar rate of biopsy-confirmed acute rejection between Groups A and B (13.4% vs 10.0%, respectively; *p=NS*). At 12 months, graft survival (93% vs 94%), patient survival (97% vs 95%), and the incidence of biopsy-confirmed acute rejection episodes (22% vs 21%) were not significantly different between Groups A and B. Hypertension, edema, hypomagnesemia, and dyspnea were reported significantly less frequently in patients randomized to undergo CsA elimination compared with patients in Group A (*p<0.05*).

Conclusion: Concentration-controlled sirolimus, with early elimination of CsA, is safe and results in improved renal function as well as fewer adverse events. Reduced exposure to CsA does not result in an increased incidence of acute rejection episodes.

Abstract# 55

CHRONIC ALLOGRAFT NEPHROPATHY UNIFORMLY AFFECTS HLA NON-IDENTICAL LIVING-RELATED, LIVING-UNRELATED AND CADAVERIC RECIPIENTS. Nancy R. Krieger,[1] Dennis Heisey,[1] Barbara J. Voss,[1] Brian N. Becker,[1] Yolanda T. Becker,[1] Jon S. Odorico,[1] Anthony M. D'Alessandro,[1] Munci Kalayoglu,[1] John D. Pirsch,[1] Hans W. Sollinger,[1] Stuart J. Knechtle.[1] *[1]Transplantation, University of Wisconsin, Madison, WI.*

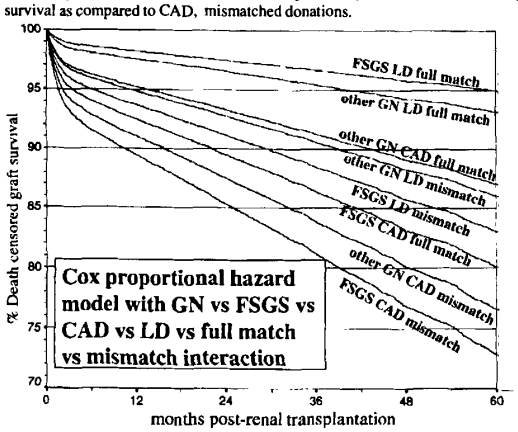
Chronic allograft nephropathy (CAN) remains a major barrier to long-term allograft survival. We retrospectively analyzed risk factors in the development of CAN in cadaveric (CAD), living-related (LRD), and living-unrelated (LUD) recipients at our center. Between January 1, 1990 and May 31, 2000, 2140 kidney alone transplants were performed, 61% CAD, 31% LRD, and 8% LUD. Overall 5-year patient and graft survival was 87% and 74%. Of these 2140 recipients, 306 developed CAN. The overall 5-year incidence of CAN was 18% (19% CAD, 17% LUD, 16% LRD, and 2% HLA-identical). Follow-up ranged from 6 mos to 10 years (mean, 4 years). Using multivariate proportional hazards analysis, we examined the impact of transplant number, recipient and donor age, HLA matching, type of donor (LRD, LUD or CAD), discharge creatinine, one-year creatinine, delayed graft function (DGF), pretransplant dialysis, and bacterial and CMV infections on the incidence of CAN. In this analysis, risk factors for CAN (in LRD, LUD or CAD) included the number of transplants (p=.0001), discharge creatinine (p=.0001), DGF (p=.003), B and DR mismatches (p=.0001), recipient age (p=.0074), donor age (p=.0025), bacterial (p=.001) and CMV infections (p=.002). When stratifying for these risk factors, although the LRD HLA-identical recipients had a significantly lower incidence of CAN (p=.0006), the incidence of CAN in the CAD, LUD and HLA non-identical LRD recipients, including one-HLA haplotypes, did not differ (p=NS). In addition, the diagnosis of CAN resulted in a 12-fold increase in the risk of graft loss (p<.001). However, overall graft survival was significantly less for CAD recipients than all other groups (p=.001). In conclusion, these results demonstrate the importance of both immunologic and nonimmunologic factors on the development of CAN, with HLA B and DR matching, the number of transplants, and discharge creatinine the most significant factors. Suprisingly, HLA non-identical LRD recipients have a similar 5-year incidence of CAN as compared with LUD and even CAD recipients. Despite this similar incidence of CAN, overall graft survival is significantly less in CAD recipients, suggesting factors in addition to CAN influence the increase in graft loss in CAD transplant recipients.

Abstract# 56

AN HLA-IDENTICAL LRD KIDNEY TRANSPLANT IS NOT A RISK FACTOR FOR RENAL ALLOGRAFT LOSS IN PATIENTS WITH FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS).

Diane M. Cibrik,[1] Bruce Kaplan,[1] Alan B. Leichtman,[1] Darrell A. Campbell,[2] Herwig-Ulf Meier-Kriesche.[1] *[1]Department of Internal Medicine, University of Michigan, Ann Arbor, MI; [2]Department of Surgery, University of Michigan, Ann Arbor, MI.*

Previous literature suggests that the recurrence of FSGS after renal transplantation is more common in recipients who have received an HLA-identical LRD transplant. To address the question if FSGS patients can safely receive a 6-antigen match LRD kidney transplant, we analyzed 19259 adult primary renal transplant recipients registered in the USRDS database between 1988 and 1997. Of these patients, 2414 patients had FSGS as their primary diagnosis as compared to16845 patients who had glomerulonephritis (GN). Methods: A Cox proportional hazard interaction model was used to estimate death censored graft survival among FSGS patients with an HLA-identical LRD kidney transplant. The model included a triple interaction term comparing FSGS vs GN vs living donation (LD) vs cadaveric donation (CAD) vs full match vs mismatch. Annually adjusted death censored graft loss rates per 1000 patients (ADGL) were calculated. Results: FSGS patients receiving an HLA-identical LRD kidney transplant had the lowest ADGL rate, losing 10.5 grafts per 1000 patients per year. Not significantly different but higher (14.3) was the ADGL rate for LD, full match GN recipients. The ADGL rate was significantly higher in FSGS recipients who received a LD, mismatched transplant (36.5). FSGS patients who received a CAD, full match graft (44.1) or CAD, mismatched graft (63.2) had significantly higher ADGL rates. Conclusion: HLA-identical LRD kidney transplants are not a risk factor for graft loss in FSGS patients but are associated with significantly better death censored graft survival as compared to CAD, mismatched donations.



Abstract# 57

IMPACT OF MYCOPHENOLATE MOFETILE (MMF) ON CHRONIC ALLOGRAFT NEPHROPATHY (CAN) IN CHILDREN.

Thomas Henne, Gisela Offner, Kai Latta, Barbara Enke, Christian V. Schnakenburg, Jochen H.H. Ehrich, Juergen Strehlau. *[1]Dept. Pediatric Nephrology, Hannover Medical School, Hannover, Germany.*

Background: CAN remains the most common cause of late allograft loss. Previous acute rejection (AR) episodes and insufficient immunosuppression (IS) have been incriminated as causative factors and up to date no drug has proven to halt its progression. The purpose of this study was to evaluate the impact of MMF on biopsy proven CAN and to correlate its efficacy to histology and intragraft and peripheral blood gene expression (GE).

Methods: Thirty-six children (mean 13.1+ 3.6 y) with a progressive decline in glomerular filtration rate (GFR) of 16.9+12.6 ml/min/1.73m2/year and biopsy confirmed CAN 4,3+/-2.9 years after transplantation were included in this study. MMF was added to conventional IS consisting of ciclosporine (CsA,n=26) or tacrolimus (n=1) and prednisone or replaced azathioprine in triple IS (n=9). Initial dosage of CsA and tacrolimus was unaltered and later reduced by 10-20% in 13 patients. Alterations of GFR were correlated to histology and intragraft and blood GE measured by Taqman PCR based quantification of Fas-ligand (FasL), NF-kB p65, IL-10, TGF-b1 and GAPDH.

Results: One year after conversion 22 children (61%) showed a rise in GFR (mean 7.5+5.6 ml/min/1.73m2), 8 (22%) remained stable and 6 (17%)showed a further decline of GFR (7.4+7 2 ml/min/1.73 m2) The response rate to MMF was identical in patients with unaltered and reduced CsA. Mean CsA trough levels prior and 1 year post conversion were 113 vs. 98 ng/ml. MMF side effects required dose reduction in 14 children, discontinuation was unnecessary. Patients responding to MMF with increasing GFR showed augmented intrarenal GE of NF-kB (median 1,52+/-1,25% of GAPDHx100) as compared to children with stable or declining GFR (0,80+/-0,45%, p<0,05). A similar trend was observed in FasL GE (median 0,29+/-0,14% vs. 0,03+/-0,03% of GAPDHx1000, p<0,08). GE in patients with increasing GFR reached a similar range as observed in AR (median NF-kB 1.96+/-1,56%; FasL 0,31+/-0,23%) despite the fact, that biopsies did not confirm AR. Intragraft GE was not reflected by peripheral blood GE.

Conclusions: Co-treatment with MMF reversed the progressive loss of GFR in a large proportion of children with late CAN for at least one year. Intrarenal signs of cellular activation by enhanced NF-kB and FasL transcription rates seem to indicate patients responding more favorable to treatment. Peripheral blood GE failed to identify or monitor patients response to MMF.

Abstract# 58

ANTI-CD20 MONOCLONAL ANTIBODY RESCUE THERAPY FOR REFRACTORY ANTIBODY-MEDIATED REJECTION.

Milagros Samaniego,[1,2] Henkie Tan,[1] Karen King,[1] Robert Montgomery,[3] Mark Haas,[1] Lloyd Ratner,[3] Andrea Zachary.[2] *[1]Dept. of Pathology, Medicine and Surgery, Johns Hopkins Sch of Med., Baltimore, MD.*

Anti-CD20 monoclonal antibody (Rituxan) has been used successfully in 4 patients (Pts) with steroid resistant acute rejection (Rej). We report 2 Pts with biopsy-proven antibody-mediated Rej (Ab-Rej) that failed treatment (Rx) with plasmapheresis (PP) and intravenous immunoglobulin (IVIg), yet responded to a regimen of PP and Rituxan. Both Pts are female cadaveric transplant recipients and had an initial episode of Ab-Rej treated with PP and IVIg 2 months (mos) prior to relapse and subsequent Rx with Rituxan. As per our protocol, Pts received PP 3-times/week until the titer of HLA-Ab dropped by at least-two-fold dilution, followed by IV Rituxan (375 mg/m^2/week) until allograft function improved (Pt 1=2 doses; Pt 2=1 dose). HLA-Ab titers (as per AHG) decreased from 1:16 to 1:2 in Pt 1 following Rituxan Rx. Pre-Rituxan, Pt 2 had a flow cytometry MFC shift of 800.

At 6-mos of follow-up, both Pts remain Ab-Rej free, their CD20 counts remain undetectable and their serum creatinine (Scr) is unchanged from levels at discharge. HLA-Ab titers have remained stable in Pt 1. Follow-up biopsies demonstrate histological improvement in both Pts.

In our limited experience, a regimen of PP and Rituxan has been a safe and clinically effective rescue Rx for Ab-Rej. Although CD20 is not normally expressed in resting plasma cells (PC), its expression is rapidly upregulated in PC exposed to IFN-γ and TNF-α. Because Ab activates macrophages and cellular Rej usually accompanies Ab-Rej, the resulting cytokine milieu can enhance the response to Rituxan by the upregulation of its target on activated PC.

In our Pts, Rx with Rituxan reduced the number of PP sessions required for a therapeutic response. Whether PP is necessary to ensure the success of Rituxan is uncertain. We believe that the removal of long-lived, high affinity Ab is pivotal to abort the injury cascade that follows Ab-deposition in the allograft and have included PP as part of our protocol.

Ab-Rej is recognized as a recalcitrant process that usually results in graft loss in the absence of an effective Rx. The design of randomized trials aimed to test the safety and effectiveness of different Rx protocols is warranted.

Patient #	Time post-Tx at Rej	PRA/# mismatches	CIT	Anti-HLA Ab specificities	# of PP sessions pre-/post-Rituxan	CD 20 counts pre/post-Rituxan	Scr pre/post-Rituxan
1	29 mos	0% / 6	19 hrs	DRw52	30 / 4	4% / 0%	3.0 / 2.0
2	3 mos	73% / 0	20 hrs	DQ7	24 / 4	36% / 0%	3.0 / 1.4

Abstract# 59

SYNERGISTIC HLA-DR MATCHING EFFECT AMONG SENSITIZED RECIPIENTS WHO RECEIVED ELDERLY DONOR KIDNEYS.

Yong W. Cho,[1] J. Michael Cecka.[1] *[1]UCLA Immunogenetics Center, UCLA School of Medicine, Los Angeles, CA.*

Over the past 12 years, the utilization of elderly donor kidneys has tripled in the US. It has been reported that HLA matching does not improve the survival of older donor kidneys. We evaluated the HLA-DR matching effect on the outcome of elderly cadaveric donor kidney allografts. Materials and Methods. From 1994 to 1997, 2,471 kidney allografts from cadaveric donors aged over 60 were reported to UNOS. In order to evaluate early and late risk factors, two-stage logistic regression analyses were applied: the first stage covered graft survival during the first year and the second the period 1-3 years after transplant. Patient death was regarded as graft loss.

Results. During the first year, HLA-DR mismatching had a deleterious effect among sensitized recipients (odds ratio (OR) = 2.05) followed by regraft (OR=1.71), older recipient (OR=1.47), and Black (OR=1.28). From 1-3 years, only the significant risk factors were Black recipient (OR=1.93) or young recipient (OR=1.89). During the first year, 501 grafts failed and the leading causes of failure were death with functioning graft (26%), acute rejection (21%), and primary failure (14%). Between 1-3 years, 165 grafts failed due to chronic rejection (39%), death with functioning graft (28%), and unknown causes (15%).

Conclusions. HLA-DR matching for sensitized patients had a dramatic beneficial effect on graft survival during the first posttransplant year. Thus, early graft losses can be significantly reduced by HLA-DR matching for sensitized patients willing to receive elderly donor kidneys.

Factors	First year (n=1,992) OR (95%CI)	P value	During 1-3 yrs (n=1,475) OR (95%CI)	P value
R age < 30 vs 30-59	1 32 (0 83-2 09)	0 25	1 89 (1 02-3 48)	0 04
R age ≥ 60 vs 30-59	1 47 (1 18-1 84)	0 001	1 20 (0 83-1 74)	0 33
Regraft vs primary	1 71 (1 23-2 38)	0 002	1 28 (0 73-2 24)	0 38
Black recip vs others	1 28 (1 02-1 61)	0 04	1 93 (1 37-2 73)	<0 001
CIT > 30 vs ≤ 30 hrs	1 15 (0 93-1 43)	0 21	1 24 (0 88-1 74)	0 22
DR matching & pPRA				
match & ≤ 20%	1 0		1 0	
match & > 20%	0 84 (0 53-1 33)	0 45	0 92 (0 46-1 83)	0 80
mismatch & ≤ 20%	1 15 (0 90-1 47)	0 25	0 81 (0 57-1 17)	0 26
mismatch & > 20%	2 05 (1 45-2 89)	<0 001	1 18 (0 67-2 08)	0.56

Abstract# 60

IMPROVED RESULTS IN HIGH IMMUNOLOGIC RISK RENAL ALLOGRAFT RECIPIENTS USING DACLIZUMAB (ZENAPAX ®) VERSUS TRADITIONAL ANTI-LYMPHOCYTE THERAPY IN MEMBER CENTERS OF THE SOUTH-EASTERN ORGAN PROCUREMENT FOUNDATION. Francis H. Wright,[1] Leroy R. Thacker, II,[2] Thomas G. Peters.[3] *[1]Methodist Specialty & Transplant Hospital, San Antonio, TX; [2]South-Eastern Organ Procurement Foundation, Richmond, VA; [3]Jacksonville Transplant Center at Shands Jacksonville, Jacksonville, FL.*

Aim: This study compares the incidence of treatment of acute rejection episodes in high immunologic risk renal allograft recipients receiving induction therapy with daclizumab (Zenapax®) or traditional anti-lymphocyte products

Methods: Transplant center members of the South-Eastern Organ Procurement Foundation (SEOPF) were queried and fourteen provided patient information for the 1998 calendar year. Patients were defined as high immunologic risk recipients if they were non-Caucasian, received a repeat transplant, had delayed graft function requiring dialysis in the first week post-transplant or had a panel reactive antibody level > 0. A total of 502 high immunologic risk recipients were identified: 222 (44.2%) received daclizumab induction and 280 (55.8%) received other induction. The incidence of acute rejection treatment in the first month and first year post-transplant were determined. Differences were analyzed with a univariate Fisher's Exact Test and utilizing a multivariate logistic regression model.

Results: In the first post transplant follow-up period, 18 of 222 (8.1%) of the daclizumab patients received treatment for rejection compared to 47 of 280 (16.8%) of other induction therapy patients (Odds Ratio=0.4374, p=.0047). In the first year post transplant, 30 of 222 (13.5%) of the daclizumab patients received rejection treatment compared to 62 of 280 (22.1%) of the other patients (Odds Ratio=0.5494, p=.0146). When adjusted for covariates, there was a statistically significant lower incidence treatment for acute rejection during the first post operative period for daclizumab patients (Odds Ratio=0.5150, p=.0495). One year follow up approached but did not achieve statistical significance (Odds Ratio=0.6415, p=.1050).

Conclusion: Daclizumab induction in high immunologic risk renal transplant recipients is effective in reducing the incidence of treatment for early acute rejection episodes.

Abstract# 61

LONG-TERM OUTCOME AFTER TREATMENT OF EARLY REFRACTORY ACUTE HUMORAL REJECTION IN KIDNEY TRANSPLANTATION. Tom Theruvath,[1] Francis Delmonico,[1] Susan Saidman,[2] Nina Tolkoff-Rubin,[1] Winfred Williams,[1] Shamila Mauiyyedi,[2] Marylin Farrell,[1] Bernard Collins,[2] Robert Colvin,[2] A. Benedict Cosimi,[1] Manuel Pascual.[1] *[1]Transplantation Unit, Massachusetts General Hospital, Boston, MA; [2]Dpt of Pathology, Massachusetts General Hospital, Boston, MA.*

Background. Acute humoral rejection (AHR) is associated with de novo production of donor specific antibodies (DSA) and C4d deposits in peritubular capillaries (PTC). Its occurrence carries a 50-80% short-term risk of graft loss. Recently, plasmapheresis (PPh) and tacrolimus-mycophenolate (TAC-MMF) rescue has been reported to effectively reverse refractory AHR. However, the long-term consequences of this therapy remain to be determined.

Methods. Between 7/95 and 7/99, 232 renal transplants (Tx) were performed under cyclosporine-based immunosuppression. Ten consecutive patients (4.3% of the overall population) who developed refractory AHR in the first month post-Tx (acute rejection resistant to antilymphocyte therapy, de novo serum anti-HLA DSA and C4d deposits in PTC) were treated with PPh and TAC-MMF rescue. All patients were sequentially monitored with assays for DSA including T and B cell cytotoxic crossmatches and cytotoxic antibody screens using recipient sera.

Results. With the institution of PPh, tacrolimus (levels: 12-15 ng/ml) and MMF (2 g/day) DSA titers decreased resulting in initial reversal of refractory AHR in 9/10 patients. With a mean follow-up of 38 +/- 18 months (16-60 months) patient and graft survival are 100% and 80% (creatinine: 1.7 +/- 0.5 mg/dl). One graft loss was due to refractory AHR at day 10, the other to CMV-associated glomerulopathy at day 290. Of the eight patients with functioning grafts, only one (12.5%) has developed biopsy-proven chronic rejection (with no C4d deposits in PTC). Long-term monitoring of DSA titers revealed persistently undetectable levels of DSA in all eight patients, i.e. no evidence of *accommodation* was found. In contrast, DSA was demonstrated in both patients with failed allografts. Lung cryptococcoma infection occurred in one patient 3 years post-transplant. Long-term levels of TAC and dose of MMF are maintained at 7.8 +/- 1.4 ng/ml and 1.6 +/- 0.4 g/day, respectively.

Conclusion. After treatment of early refractory AHR, the combination of TAC-MMF suppressed long-term DSA production and resulted in excellent allograft function in most patients. Careful monitoring for late infectious complications and adjustment of TAC and MMF dosages are important aspects of the long-term management of these patients.

Abstract# 62

SERUM CREATININE (CR) AT ONE AND SIX MONTHS PREDICTS LONG-TERM ALLOGRAFT SURVIVAL: A SINGLE CENTER ANALYSIS. Sundaram Hariharan,[1] Yong-Ran Zhu,[2] Allan M. Roza,[2] Mark B. Adams,[2] Christopher P. Johnson.[2] *[1]Division of Nephrology, Medical College of Wisconsin, Milwaukee, WI; [2]Transplant Surgery, Medical College of Wisconsin, Milwaukee, WI.*

Background: Although long-term renal allograft survival has gradually improved over the past 10 years, chronic allograft nephropathy remains a serious problem. There is need for criteria which can identify subsets of individuals within the first year after transplantation with predictably shortened half-life (t1/2), such that secondary intervention trials can be designed more effectively. Our hypothesis is that serum Cr at 6 months (6 mo Cr), together with ΔCr (1-6 mo) (the change in Cr between 1 and 6 months) can be used to predict t1/2.

Methods and Results: Between 1989 and 1997, 806 patients received cadaver donor transplants at our institution. Donor and recipient databases were used to identify demographic, immunological and post-transplant outcome variables. 750 recipients had functioning grafts after 1yr. The mean serum Cr for these recipients at 1mo and 6mo was 1.88 and 1.97. Recipients were stratified into 4 categories according to serum Cr 6 mo, and Δ Cr (1-6 mo). Five-year graft survival and t1/2 (conditioned on 1yr survival) are given below:

Combination of 6mo Cr and ΔCr (1-6mo)	% of total	5 year Graft Survival	Projected Half-life (Yrs)
6mo Cr< 1 6 and Δ Cr<0 3	35%	86 7%	17 3
6mo Cr<1 6 and Δ Cr≥0 3	19%	77 1%	13 2
6mo Cr≥1 6 and Δ Cr<0 3	35%	68 2%	8 2
6mo Cr≥1 6 and Δ Cr≥0 3	11%	52 8%*	5 5*

*p<.001 vs. first group

The differences in t1/2 are even greater when censoring for death with a functioning graft. Within high-risk groups such as African American recipients or donor age ³50, 6moCr and D Cr correlate with t1/2.

Conclusion: The combination of 6 month Cr ³1.6 and D Cr (1-6 mo) identifies a subset of renal allograft recipients with a markedly shortened t1/2. These patients could potentially be enrolled in secondary intervention trials as early as 6 monthS after transplantation.

CONCURRENT SESSION 7:
XENOTRANSPLANTATION: PRECLINICAL NON-HUMAN PRIMATE

Abstract# 63

DIFFERENT IGG SUBCLASSES OF ANTI-GAL MABS DIFFERENTIALLY INDUCE XENOGRAFT REJECTION. Dengping Yin,[1] LianLi Ma,[1] Jikun Shen,[1] Hui Xu Xu,[2] John Logan,[2] Guerard Byrne,[2] Anita S. Chong.[1] *[1]Department of General Surgery, Rush Presbyterian St. Luke's Med Ctr, Chicago, IL; [2]Nextran, Princeton, NJ.*

There is increasing evidence that elicited xenoreactive IgG, play a major role in acute xenograft rejection. We have produced a series of anti-Gal hybridomas from α1,3galactosyltransferase-knockout (GT-Ko) mice after rat to mouse cardiac transplantation. In this study, we have characterized the ability of two anti-Gal IgG mAbs to elicit xenograft rejection.

We first developed a xenotransplantation model involving Lewis rat hearts into double GT and Rag knockout mice. Xenografts survived for >30 days suggesting that NK cells and macrophages cannot directly elicit acute xenograft rejection (AXR) in the absence of T and/or B cells.

TREATMENT	DOSE (mg/mouse)	FREQUENCY	SURVIVAL
NONE	0		>30 days (x4)
IgG1	0 5	1x, d0	>30 days (x4)
	0 5	daily, d0-rej	3 days (x4)
IgG3	0 5	1x, d0	7, 7, 60, 60 mins
	0 125	1x, d0	3 5, 3 5, 4 hours
	0 05	daily, d0-rej	1, 1, 2 2, 2 days
	0 02	daily, d0-rej	>4 days (x4)

Anti-Gal IgG3, but not IgG1, induced complement dependent lysis *in vitro*. *In vivo* studies revealed that high doses of IgG3 anti-Gal mAb elicited hyperacute rejection, while low doses elicited AXR. IgG1 anti-Gal mAb was unable to elicit hyperacute rejection but was able to elicit AXR. Histological examination suggested that IgG3-mediated rejection resembled complement-mediated vascular rejection while IgG1-mediated rejection was associated with leukocyte infiltration. We are currently conducting further investigations into the mechanisms by which anti-Gal Abs of different IgG subclasses elicit AXR.

Abstract# 64

THE LEVEL OF ANTI-GAL IgM BEFORE TRANSPLANTATION CORRELATES WITH XENOGRAFT SURVIVAL IN hDAF PIG-TO-BABOON HETEROTOPIC HEART TRANSPLANTATION.

Rafael Manez,[1] Fabian Crespo,[1] Alberto Juffe,[1] Alberto Centeno,[1] Eduardo Lopez-Pelaez,[1] Emanuele Cozzi,[2] David J. White.[2] *[1]Transplantation, Juan Canalejo Medical Center-University of La Coruna Health Institute, La Coruna, Spain; [2]Imutran (a Novartis Pharma AG Company), Cambridge, United Kingdom.*

Purpose: hDAF pig organs transplanted into non-human primates do not undego hyperacute rejection in most of the cases. However, these xenografts are rejected by an acute humoral xenograft rejection (AHXR). In this study we investigate the role of anti-Gal IgM and IgG and anti-pig hemolytic antibodies (APA) in the AHXR of hDAF pig hearts transplanted into baboons.

Material and Methods: Thirteen baboons underwent heteropic heart xenotransplantation with hDAF pig organs. Four xenografts had a hyperacute rejection and other two baboons died on days 5 and 6 after transplantation because of renal failure with a beating xenograft. The remaining 7 xenografts failed because AHXR and constitute the study group. All recipients received an immunosuppression protocol that included a four-dose course with cyclophosphamide at induction only, Neoral, ERL,and tapering steroids. Serum anti-Gal IgM, IgG and anti-pig hemolytic antibodies were measured daily.

Results: AHXR occurred at a median of 16 days (range 10 - 29) after transplantation. A correlation was found between the level of anti-Gal IgM before transplantation and xenograft survival (r: -0.76, $p < 0.05$). Three baboons had a prolonged survival (median 24 days; range 19 - 29), compared with the other four (median survival 11.5 days; range 10 - 16) ($p < 0.05$). The level of anti-Gal IgM before transplantation in baboons with extended survival was significantly lower (median 560; range 360 - 585) than in those with shorter survival (median 925; range 794 - 928) ($p < 0.05$). At the time of AHXR, higher levels of anti-Gal IgM were found in recipients with reduced survival (median 696; range 361 - 823) compared to those with prolonged survival (median 350; range 211 - 507) ($p = 0.07$). Similarly, APA titers at the time of AHXR were higher in earlier AHXR (median 722; range 330 - 1095) than in later (median 159, range 12 - 335) ($p=0.07$).

Conclusion: The level of anti-Gal IgM antibodies before transplantation correlates with the survival of heterotopic hDAF pig hearts transplanted into baboons, with lower levels associated with longer survivals. The differences in relative values of anti-Gal and APA at the time of rejection suggest a greater contribution of anti-Gal antibodies to earlier AHXR and non-Gal antibodies to later AHXR.

Abstract# 65

INFUSION OF GAL TYPE 6 OLIGOSACCHARIDES IN BABOONS RESULTS IN TOTAL DEPLETION OF ANTI-PIG ANTIBODIES.

Katsuhito Teranishi,[1] Bernd Gollackner,[1] Leo Buhler,[1] Cristoph Knosalla,[1] David H. Sacks,[1] Michel Awwad,[2] David K.C. Cooper.[1] *[1]Transplantation Biology Research Center, Massachusetts General Hospital, Boston, MA; [2]Biotransplant Inc., Charlestown, MA.*

[PURPOSE] Anti-Gal antibodies (Ab) play a key role in the rejection of pig cells or organs transplanted into primates. Extracorporeal immunoadsorption (EIA) of anti-Gal Ab through a Gal type 6 oligosaccharide column depletes Ab, but Ab returns within the next few days through continuing production by B/plasma cells. We have investigated the effect on anti-Gal Ab return after EIA of (i) costimulatory blockade and (ii) the continuous intravenous (i.v.) infusion of bovine serum albumin conjugated to Gal type 6 oligosaccharides (BSA-Gal).

[METHODS] Porcine peripheral blood mobilized progenitor cells (PBPC) obtained by leukapheresis were infused i.v. into baboons. Group1 (n=3) received whole body and thymic irradiation, splenectomy, anti-thymocyte globulin, cobra venom factor, cyclosporine, mycophenolate mofetil, porcine hematopoietic growth factors, and EIA before transplantation of high doses (2-4x10^10 cells/kg) of PBPC; Group2 (n=4) received the Group1 regimen plus anti-CD154 mAb therapy, and Group3 (n=3) received the Group2 regimen plus a continuous i.v. infusion of BSA-Gal for up to 30 days.

[RESULTS] Group1: sensitization to both Gal and new non-Gal porcine antigens occurred within 20 days; Group2: Gal-reactive Ab returned to its pre-PBPC level within 30 days, but no induced Ab to Gal or non-Gal determinants developed; Group3: anti-Gal Ab was unmeasurable or at very low level during BSA-Gal therapy. After discontinuation of BSA-Gal, Ab did not return to pre-PBPC level for more than 40 days, and no sensitization developed. In one baboon, however, Ab to Gal type 2, but not type 6, returned during BSA-Gal therapy.

[CONCLUSIONS] (i) The induced Ab response to Gal and non-Gal epitopes was prevented by anti-CD154 mAb therapy. (ii) BSA-Gal therapy maintained depletion of anti-Gal Ab. (iii) Some polymorphism in specificity of anti-Gal Ab was identified, indicating that the infusion of a combination of type 6 and type 2 BSA-Gal may be required. This therapy is the first that results in a prolonged absence of both natural and induced anti-pig Ab, and may have potential in prolonging pig-to-primate xenograft survival.

Abstract# 66

GAS 914, A POLYLYSINE CONTAINING αGAL, REDUCES THE SEVERITY OF ACUTE HUMORAL REJECTION IN hDAF PIG TO PRIMATE HETEROTOPIC HEART XENOTRANSPLANTATION.

Rafael Manez,[1] Alberto Centeno,[1] Eduardo Lopez-Pelaez,[1] Carmen Ruiz de Valbuena,[1] Alberto Juffe,[1] Beverly Holmes,[2] Rudolph Duthaler,[3] Andreas Katopodis.[3] *[1]Transplantation, Juan Canalejo Medical Center-University of La Coruna Health Institute, La Coruna, Spain; [2]Imutran (a Novartis Pharma AG Company), Cambridge, United Kingdom; [3]Transplantation Research, Novartis Pharma AG, Basel, Switzerland.*

Introduction: Acute humoral xenograft rejection (AHXR) is the most important cause of xenograft failure of hDAF pig organs transplanted into non-human primates. In the present study we investigate the impact of GAS 914, a polylysine molecule containing aGal structures that neutralizes anti-αGal antibodies, in the AHXR of hDAF pig organs transplanted into baboons.

Materials and Methods: Eighteen baboons underwent heterotopic heart xenotransplantation into the abdomen with hDAF transgenic pig organs. Eleven baboons (Group A) received an immunosuppression protocol that included a four-dose course with cyclophosphamide for induction only, Neoral, ERL sodium mycophenolate, and tapering steroids. The other five baboons (Group B) received the same immunosuppression and GAS 914 i.v. or s.c at 5mg/kg/day on days -17, -14 and -11, 1 mg/kg/day on days -8, and from day -5 to 0, and 1 mg/kg/12 hours from day 1 to xenograft failure.

Results: Four xenografts from Group A underwent hyperacute rejection, whilst this type of rejection was not observed in Group B. After excluding hyperacute rejections Group A median survival was 13 days (range 5-29) and in Group B 19 days (range 6-45). All Group A xenografts failed due to AHXR with the exception of two animals that died on days 5 and 6 from renal failure. In Group B no xenograft failed because of AHXR. Four animals died from various causes (two from bleeding possibly related to cyclophosphamide toxicity on days 6 and 8, one from xenograft rupture after left atrium thrombosis on day 19 and one from fungal sepsis on day 45). One xenograft is still functioning on day 20. Although some deposits of IgM, C3, C4 and C5b-9 were found in all Group B xenografts postmortem, these deposits and the tissue damage were significantly less than in those Group A xenografts that failed because of AHXR.

Conclusion: Treatment with GAS 914 for 17 days before and daily after transplantation prevents hyperacute rejection and reduces the severity of AHXR of hDAF pig xenografts in baboon recipients.

Abstract# 67

THE COMBINED EFFECTS OF PHARMACOLOGIC NEUTRALIZATION OF ANTI-αGAL ANTIBODIES AND COMPLEMENT INHIBITION ON SURVIVAL OF hDAF TRANSGENIC PIG RENAL GRAFTS IN CYNOMOLGUS MONKEYS.

Bernard Hausen,[1] Tuan Lam,[1] Laurie Hook,[1] Katrin Boeke,[1] Uwe Christians,[2] Wolfgang Jacobsen,[2] John Higgins,[1] Emanuele Cozzi,[3] Hugh Davies,[1] Rudolf Duthaler,[4] Richard Harrison,[1] Andreas Katopodis,[4] Randall Morris.[1] *[1]Cardiothoracic Surgery and Pathology, Stanford University, Stanford, CA; [2]Biopharmaceutical Sciences, University of California, San Francisco, CA; [3]Imutran Ltd (A Novartis Pharma AG Company), Cambridge, United Kingdom; [4]Novartis Pharma AG, Basel, Switzerland.*

INTRODUCTION: We have shown that in cynomolgus monkeys with very high levels of anti-pig hemolytic natural antibodies (NAb), 7/10 human Decay Accelerating Factor (hDAF) pig organs undergo immediate graft dysfunction with pathology compatible with hyperacute rejection (HAR). Now we evaluate GAS914 (a polymeric form of the αGal trisaccharide) for the neutralization of anti-pig NAb and prevention of HAR. We also study the combined effects of GAS914 and fluid phase complement inhibition using TP10 (soluble complement receptor type 1) on humoral xenograft rejection.

METHODS: Ten monkey recipients of life-supporting hDAF pig kidney xenografts were treated with 2 doses of CyP induction and continuous baseline immunosuppression: CsA (target troughs 300-600 ng/ml), steroids, and sodium mycophenolate (troughs 3-6 ug/ml). GAS914 was used at induction only (days -5, -3 at 5 mg/kg; -2, -1 at 1 mg/kg) or with daily treatment postop (1 mg/kg). Anti-pig Ab were measured by an anti-αGal Ab ELISA and an anti-pig erythrocyte hemolytic Ab assay (APAA). TP10 was administered on day -1 and then daily.

RESULTS: GAS914 induction completely elimininated NAb in the blood by day 0. TP10 completely inhibited complement activation (complete absence of SC5b-9 levels). The predominant histopathologic features in the renal xenografts at necropsy (see Table) were thrombotic microangiopathy, a paucity of cellular rejection, and lack of HAR.

CONCLUSIONS: These are the first data to show that GAS914 prevents HAR of hDAF pig renal grafts in monkeys with high NAb levels. Although cellular rejection was very well controlled, graft loss due to humoral rejection in the absence of anti-αGal Ab and complement suggests better control of anti-graft Ab will improve graft survival.

Group	Treatment	Survival in days (Histopathology)
1 (n=4)	GAS914 at induction and daily treatment + baseline immunosuppression	6 (humoral), 12 (ATN), 31 (humoral), 37 (ATN)
2 (n=2)	GAS914 at induction and daily treatment + TP10 + baseline immunosuppression	3 (humoral), 15 (humoral)
3 (n=4)	GAS914 at induction + TP10 + baseline immunosuppression	10 (cellular, humoral), 20 (humoral), 32 (humoral), 37 (humoral)

Abstract# 68

C1-INHIBITOR (C1-INH) FOR TREATMENT OF ACUTE VASCULAR XENOGRAFT REJECTION (AVR) IN CYNOMOLGUS RECIPIENTS OF PORCINE KIDNEYS. Jens M. Hecker,[1] Ralf Lorenz,[1] Richard Appiah,[2] Martin Loss,[1] Michael Przemeck,[2] Jan Schmidtko,[1] Arman Jalali,[1] Burkhard Vangerow,[2] Horst Rueckoldt,[2] Juergen Klempnauer,[1] Michael Winkler.[1] *[1]Klinik fuer Viszeral- und Transplantationschirurgie, Medizinische Hochschule Hannover, Hannover, Germany, [2]Zentrum Anaesthesiologie, Medizinische Hochschule Hannover, Hannover, Germany.*

At present, the major barrier to successful discordant xenotransplantation of unmodified or complement regulator transgenic porcine xenografts is acute vascular xenograft rejection (AVR). AVR has been shown to be associated with the intragraft deposition of induced recipient xenoreactive antibodies and complement activation. In a life supporting pig to primate kidney xenotransplantation (X-KTX) setting using either unmodified or h-CD55 transgenic donor organs and cynomolgus monkeys as recipients the efficacy of supplemental CI-INH administration for treatment and prophylaxis of AVR was investigated. Methods: Study A: X-KTX were performed using h-CD55-transgenic pigs as organ donors. Postoperative maintenance immunosuppression comprised of CyP, CyA and low dose steroids according to previously established protocols. Episodes of AVR were either treated with boli of CyP and steroids (n=4 animals; group I) or with the same regimen supplemented by a three days course of CI-INH (n=4 animals; group II). Study B: 3 X-KTX were performed using unmodified (n=3) pigs as donors. Postoperative maintenance immunosuppression comprised of CyA, MMF and low dose steroids supplemented by a 14 days course of intravenous CI-INH. Results: Study A: No HAR was observed. In 8 out of 12 animals stable initial graft function at postoperative day 4 was achieved. In all 8 animals one or more episodes of AVR were diagnosed. In group I 0 out of 4 episodes of AVR responded to treatment. In group II AVR was successfully reversed in 6 out of 7 episodes. Recipient survival in group I was 9, 11, 11, 15 days; in group II 18, 21, 28, 68 days. Study B: In the three animals receiving non-transgenic organs and continuous CI-INH treatment no HAR was observed with initial graft function in all 3 experiments. Recipient survival in group B was 5, 13 and 15 days. All animals died with a functioning graft (latest creatinine 96, 112 and 96 umol/L) due to fulminant septicaemia. Conclusion: We conclude that, in our model, CI-INH therapy can be helpful in the treatment of AVR following discordant porcine kidney xenotransplantation. The optimal dose and duration of CI-INH treatment for prophylaxis of AVR has yet to be determined.

Abstract# 69

EVALUATION OF THE EXTRACORPOREAL LIVER PERFUSION SYSTEM USING HUMAN DECAY ACCELERATING FACTOR TRANSGENIC PIG LIVER IN DIRECT CROSS-CIRCULATION WITH BABOON. Takakazu Matsushita,[1] Iwao Ikai,[1] Ryuta Nishitai,[1] Nagato Katsura,[1] Hiroshi Okabe,[1] Satoshi Yamanokuchi,[1] Koichi Matsuo,[1] Tomohiro Shiotani,[1] Hiroaki Terajima,[1] Yoshio Yamaoka.[1] *[1]Department of Gastroenterological Surgery, Kyoto University Graduate School of Medicine, Kyoto, Japan.*

PURPOSE. We have developed from porcine liver an extracorporeal liver perfusion system (ECLP) as a liver assist device for treatment of patients with acute liver failure. The aim of this study is the evaluation of the safety of direct cross-circulation between baboons and ECLP using human decay accelerating factor (hDAF) transgenic pig livers. *MATERIALS AND METHODS:* Livers were isolated from 5 hDAF pigs (Imutran) and 6 normal pigs as controls, and were perfused with fresh baboon blood in ECLP. Eleven healthy baboons were directly connected with the ECLP. We discontinued cross-circulation when liver perfusion pressure was elevated (hepatic artery pressure > 200 mmHg, or portal vein pressure > 60 mmHg), a large amount of exudate was observed on the liver surface, or thrombocytopenia occurred. We planned to maintain the cross-circulation for 24 hours. *RESULTS.* In the first case using a normal pig, we performed cross-circulation for 10 hours, and then hemolysis and macroscopic hematuria occurred. Two days later, the baboon died of acute renal failure. Based on this result, it was decided that hemolysis would also be considered as a criteria for discontinuation of the cross-circulation. In the last 5 cases using normal pig livers, the cross-circulation was discontinued after 4.4 +/- 1.2 hours due to high perfusion pressure (n=2) and hemolysis (n=3). When hDAF livers were used, little hemolysis occurred and the perfusion pressure did not increase, allowing a cross-circulation for at least 24 hours in three cases. In the other 2 cases the cross-circulation was discontinued because of thrombocytopenia and massive exudate. The duration of the cross-circulation was 21 +/- 5.8 hours At the end of these experiments, all baboons except the first case were alive and did not show any serious complications. Through the perfusion period, bile production of the pig livers was preserved in all cases. Total bile output in hDAF cases was greater than in control cases. *CONCLUSIONS:* hDAF expression lead to stable perfusion pressure and little hemolysis of porcine liver perfused with baboon blood. Direct cross-circulation between non-human primate and hDAF pig liver could be performed for more than 24 hours. These data indicate that an ECLP using an hDAF transgenic pig liver gives superior performance over that demonstrated with a normal pig liver, and shows promise for use in a liver assist device.

Abstract# 70

XENOGENEIC THYMOKIDNEY TRANSPLANTATION IN A PIG-TO-BABOON MODEL: EVIDENCE OF SPECIFIC T CELL UNRESPONSIVENESS. Rolf N. Barth,[1] John C. LaMattina,[1] Shin Yamamoto,[1] Naoki Kumagai,[1] Leo Buhler,[1] Hiroshi Kitamura,[1] Michel Awwad,[2] David K.C. Cooper,[1] Megan Sykes,[1] David H. Sachs,[1] Kazuhiko Yamada.[1] *[1]Transplantation Biology Research Center, Massachusetts General Hospital, Boston, MA; [2]BioTransplant,Inc., Boston, MA.*

Xenotransplantation is impeded from clinical application by barriers of humoral and cellular rejection. The induction of specific tolerance in the xenogeneic model of pig-to-rodent thymic transplantation and in the allogeneic model of fully-mismatched miniature swine composite thymokidney transplantation have previously been reported from this laboratory. Here we test this approach in the clinically relevant pig-to-primate model. **METHODS.** Composite thymokidney grafts were created 40-80 days prior to transplant by the autologous implantation of thymic tissue under the renal capsule of transgenic hDAF (human decay accelerating factor) swine. We performed six xenotransplants of hDAF swine composite thymokidneys to baboons. Baboons were treated with regimens including thymectomy or thymic irradiation, splenectomy, extracorporal immunoadsorption, mycophenolate mofetil, T cell depletion (cyclophosphamide, ATG, FN18-CRM9, LoCD2b), cobra venom factor, and anti-CD40L. Some animals received additional thymic tissue into the omentum. They were followed for indicators of xenograft rejection, T cell depletion and reconstitution, anti-Gal antibody levels, and MLR responses. **RESULTS:** Thymokidney xenografts survived up to 27 days with evidence of viable thymic epithelium under the renal capsule and in the omental implants, and with evidence of Hassal's corpuscles but few host lymphocytes. Two animals demonstrated cellular donor unresponsiveness and normal alloresponses in MLR assays after immunosuppression had been stopped. Three animals expired from early complications. Rejected grafts demonstrated humoral damage without evidence of cellular infiltrate. After graftectomy, one animal maintained donor-specific cellular unresponsiveness for 2 months and stable anti-Gal antibody levels, suggesting that this animal was not sensitized to xenoantigen. **CONCLUSION:** Composite thymokidney xenotransplantation from hDAF swine-to-baboon can induce donor-specific T cell unresponsiveness and stable anti-Gal antibody levels indicating non-sensitization after xenotransplantation. The presence of viable donor swine thymic epithelium could play a role in donor-specific cellular tolerance. Further strategies to address humoral rejection could prolong graft survival and result in long-term tolerance to xenografts.

Abstract# 71

XENOGENEIC PIG ISLETS ESCAPE REJECTION IN IMMUNOSUPPRESSED PRIMATES BUT FAIL TO SUSTAIN NORMOGLYCEMIA. Martin Wijkstrom,[1] Nicole Kirchhof,[1] Kristin J. Pilon,[1] Raja Kandaswamy,[1] Sue Clemmings,[1] Alison Trexler,[1] Tom Gilmore,[1] David E.R. Sutherland,[1] Bernhard J. Hering.[1] *[1]Department of Surgery, University of Minnesota, Minneapolis, MN.*

We have previously shown immediate function prior to cellular rejection of adult porcine islets transplanted to diabetic, non-immunosuppressed Rhesus monkeys (RM). This follow-up study evaluates the efficacy of clinically applicable immunosuppression in this preclinical model.

Methods: Recipients were streptozotocin-diabetic RM (n=3; 1.6, 2.4 and 2.2 U insulin/kg/d respectively pre-transplant) and adult pigs served as islet donors. After culture for 2 days, 19-27 K islet equivalents (IE)/kg BW were transplanted intraportally. Immunosuppression: Antithymocyte globulin (Thymoglobulin® 1.5 mg/kg IV, days-2 through 2), daclizumab (1.0 mg/kg IV, days 0 and 14), tacrolimus (target trough 5-8 ng/mL, day -2 onwards), and rapamycin (target trough 12-15 ng/mL, day -2 onwards.) The anti-inflammatory agent sTNFR:Fc was given at 0.5 mg/kg SC on days 0,3, 7 and 10. Insulin was administered through day 14. **Results:** Body weight loss during the study peroid was 8 ± 5 % without other signs of general toxicity. RM #1 showed normoglycemia on markedly reduced insulin requirements (0.21 U/kg/d) until xenoislet dysfunction was noted with blood glucose (BG) >200 mg/dL on day 22. RM #2 showed partial graft function with a mean BG of 194 mg/dL on 0.95 U/kg/d. A second transplant (10 K IE/kg) performed on day 15 did not improve glycemic control until sacrifice on day 30. In RM #3 daily insulin requirements were 1.1 U/kg/d, but steadily increasing until sacrifice on day 8. Porcine C-peptide, negative pre-transplant, was present in all animals (0.68 ± 0.45 ng/mL) until sacrifice. T cells were depleted with absolute lymphocyte counts of 200, 242 and 322 per mm^3 by day 4. Histology showed an abundance of insulin-positive islets in each recipient. Associated peri-islet infiltrates were composed of T cells and macrophages. An infiltrate was absent or minor in 61 % of islets in RM #1, 16 % in RM #2, and 89 % in RM #3. Complement and antibody deposits were rarely seen in RM #3, but not in RM #1 and 2. Pre- and post-transplant serum levels of IgG and IgM XNAbs (determined by flow cytometry with porcine PBMCs as targets) were unchanged and similar in all recipients. **Conclusions:** T cell directed immunosuppression prevents rejection of porcine islets for up to three weeks in primates. Glycemic control and histological analysis of the graft revealed conflicting results. The immunobiology of xenoislet dysfunction is currently being addressed with molecular methods.

CONCURRENT SESSION 8:
LUNG TRANSPLANTATION: BENCH-TO-BEDSIDE

Abstract# 72

QUANTITATIVE MEASUREMENT OF TNF-α IN BRONCHOALVEOLAR LAVAGE SAMPLES: A POTENTIAL MARKER OF LUNG TRANSPLANT REJECTION. Tehmina Z. Ali,[1] Sean M. Studer,[2] Jodi L. Layton,[1] Jonathan B. Orens,[2] William M. Baldwin,[1] Barbara A. Wasowska.[1] *[1]Pathology, The Johns Hopkins School of Medicine, Baltimore, MD; [2]Medicine, The Johns Hopkins School of Medicine, Baltimore, MD.*

Background: Lung graft rejection is usually evaluated by pulmonary function tests, transbronchial biopsy and bronchoalveolar lavage (BAL). To improve the diagnostic sensitivity of BAL, others have assessed biomarkers of inflammmation with varied results. This may be due to the use of techniques that stimulate alveolar macrophage production of these markers. In order to avoid manipulation of macrophages, the predominant cell constituent of BAL, we measured the expression of TNF-α mRNA in fresh BAL samples, as a potential marker for rejection.

Methods: TNF-α mRNA was quantitated by Real-Time PCR in 14 BAL samples from 12 double lung transplant recipients (single lung transplants were excluded because of the possible confounding effects of disease in the native lung). TNF-α was quantitated using the standard curve method. TNF-α mRNA amount was divided by the amount of GAPDH mRNA of the same sample to normalize the TNF-α value. The TNF-α expression levels were correlated with lung function at the time of bronchoscopy and classified as one of the following: no rejection (stable), acute rejection (AR) or bronchiolitis obliterans syndrome (BOS).

Results: Of the 14 samples, 9 were from patients in stable condition, 1 from an AR and CMV pneumonitis patient, 1 from a patient with pneumonia and 3 from patients having BOS. The mean TNF-α level for the stable patients was 29.2(+/-13.49) units with no levels greater than 50 units. One stable patient with two sequential BALs in a 3 month interval had low levels of 19.61 and 36.83 units. The patient with AR+CMV infection had level of 717.4 units; after treatment with Ganciclovir and Cytogam for CMV and a burst of immunosuppressive agents the level fell to 22.72. The levels of TNF-α in the three patients with BOS were 99.15, 116.21 and 237.45 units. The patient with pneumonia had a TNF-α level of 75.5 units.

Conclusion: Quantitaive Real-Time PCR measurements of TNF-α mRNA in fresh BAL is a potential marker for AR, infection and BOS in double lung transplant recipients. Additional studies are underway to confirm these findings.

Abstract# 73

INDIRECT RECOGNITION OF MISMATCHED DONOR HLA CLASS II PEPTIDES IN PEDIATRIC LUNG TRANSPLANT RECIPIENTS WITH BRONCHIOLITIS OBLITERANS SYNDROME. Kim C. Lu,[1] Eric Mendeloff,[1] Charles B. Huddleston,[1] T. Mohanakumar.[1,2] *[1]Department of Surgery, Washington University School of Medicine, St. Louis, MO; [2]Department of Pathology and Immunology, Washington University School of Medicine, St. Louis, MO.*

Purpose: Indirect recognition of mismatched donor MHC class II peptides has been documented in multiple animal and adult human models of solid organ rejection. Therefore, we determined whether indirect recognition of mismatched donor HLA class II peptides correlates with bronchiolitis obliterans syndrome (BOS) in pediatric lung transplant recipients.

Methods. 17 pediatric patients were studied. 4 normals, 4 lung recipients without BOS, and 9 lung recipients with BOS gave peripheral blood. Lymphocytes were obtained by density gradient centrifugation and were then incubated with interleukin 2 (50 units/ml) for seven days. Limiting dilution analysis was performed using synthetic peptides with sequences derived from the hypervariable regions of donor HLA DR molecules. Tritiated thymidine uptake was measured after 48 hours of culture with peptide.

Results. In normals, the average T cell precursor frequency against HLA class II peptides was $3.19 \times 10^{-5} \pm 2.15 \times 10^{-5}$. ($p<0.05$) In pediatric lung recipients without BOS, the average T cell precursor frequency against mismatched donor HLA class II peptides was $2.21 \times 10^{-5} \pm 2.65 \times 10^{-5}$. ($p<0.05$) In recipients with BOS, the frequency was $8.95 \times 10^{-5} \pm 7.31 \times 10^{-5}$. Thus, the average T cell precursor frequency in pediatric BOS patients was significantly higher than the corresponding frequency in either normal patients or lung recipients without BOS.

Conclusions. There are significantly more T cells sensitive to mismatched donor HLA class II peptides in pediatric BOS patients than in normals or in those without BOS. Indirect recognition of mismatched donor HLA class II peptides may play an important role in the development of bronchiolitis obliterans syndrome in pediatric lung tranplant patients.

Abstract# 74

INTERLEUKIN 6 AND TUMOR NECROSIS FACTOR-α POLYMORPHISMS CORRELATE WITH BRONCIIIOLITIS OBLITERANS SYNDROME IN LUNG TRANSPLANT RECIPIENTS. Kim C. Lu,[1] Rachel L. Lecha,[1] Aviva Aloush,[1] Eric Mendeloff,[1] Elbert P. Trulock,[2] G. A. Patterson,[1] T. Mohanakumar.[1,3] *[1]Department of Surgery, Washington University School of Medicine, St. Louis, MO; [2]Department of Pulmonary and Critical Care Medicine, Washington University School of Medicine, St. Louis, MO; [3]Department of Pathology and Immunology, Washington University School of Medicine, St. Louis, MO.*

Purpose. Polymorphisms in the promotor regions of cytokines have been shown to correlate with levels of *in vitro* production of those cytokines. In addition, the "high" expression polymorphism of TGF-β has been associated with lung allograft fibrosis. In this study, we determined whether cytokine polymorphisms correlate with bronchiolitis obliterans syndrome (BOS) in lung transplant recipients.

Methods: 120 patients were studied. 29 normals, 54 lung recipients without BOS, and 37 lung recipients with BOS gave peripheral blood. Lymphocytes were obtained by density gradient centrifugation. DNA was purified with the QIAamp® DNA Blood Mini Kit (QIAGEN Inc, Valencia, CA). To identify cytokine polymorphisms for TNF-α, TGF-β, IL-6, IL-10, and IFN-γ, polymerase chain reactions were performed using the Cytokine Genotyping Tray (One Lambda, Inc., Canoga Park, CA). Statistics were done with Chi-squared analysis.

Results: The distribution of TNF-α and IL-6 polymorphisms correlated strongly with BOS status; p values were 0.042 and 0.005, respectively. Further subgroup analysis of IL-6 revealed that 62% of lung recipients without BOS had the "high" expression IL-6 polymorphisms, while 83% of those with BOS had those same polymorphisms. p value=0.001. Subgroup analysis for TNF-α was not significant. Surprisingly, IL-10 and IFN-γ polymorphisms did not correlate with BOS status in our patient population. In addition, the distribution of TGF-β polymorphisms only showed a trend. (p=0.086)

Conclusion: The distributions of polymorphisms of TNF-α and IL-6 correlate with BOS in lung transplant recipients. In particular, the high expression polymorphisms of IL-6 correlate very strongly with BOS status. This may have implications in tailoring immunosuppression following lung transplantation.

Abstract# 75

CELL-MEDIATED IMMUNITY TO COLLAGEN V IN LUNG TRANSPLANT RECIPIENTS: CORRELATION WITH COLLAGEN V RELEASE INTO BAL FLUID. David S. Wilkes,[1] Kathleen M. Heidler,[1] Kazu Yasufuku,[1] Lynn DeVito-Haynes,[2] Ewa Jankowska-Gan,[2] Keith Meyers,[2] Robert Love,[2] William J. Burlingham.[2] *[1]Department of Medicine, Indiana University School of Medicine, Indianapolis, IN; [2]Department of Surgery and Medicine, University of Wisconsin School of Medicine, Madison, WI.*

Background: Type V collagen [col(V)] is an important constituent of the lung extracellular matrix (ECM) and is located in the perivascular and peribronchiolar tissues which are targets of rejection activity. Analysis of IgG2 present in the BAL fluid led to the discovery that col(V) stimulates a specific local antibody response in lung transplant recipients. Recently, oral administration of col(V) was found to mitigate pathology and prolong survival of MHC-mismatched allografts in rodent lung transplant models.

Hypothesis: Col(V) is a critical "self" antigen involved in the rejection of human lung allografts.

Experimental Design: We postulated that a systemic cell-mediated immune response specific for col(V) would coincide with the release of col(V) fragments into the BAL fluid, and would parallel the development of anti-col(V) antibodies. BAL samples from unilateral and bilateral lung transplant recipients (n=12) were tested for the presence of col(V) fragments using a sensitive ELISA test. PBMC samples were tested for anti-col(V) reactivity in the "trans-vivo" human-to-mouse DTH assay. This quantitative test detects a swelling response in the footpad of SCID mice which is mediated by human antigen-specific T memory cells, and which requires autologous human monocytes.

Results: Patients (n=3) tested <1 yr or (n=5) those tested >3 yr post-transplant had no col(V)-specific DTH response. Reactivity was also low/absent to collagen II [col(II)], a collagen found mainly in cartilage and not in the lung. However, patients (n=4) tested 1.5-2.0 yrs post-transplant showed a strong DTH reactivity to col(V); in 3/4 this response was entirely specific for col(V) vs. col(II). The col(V)-specific DTH response was correlated with the levels of col(V) fragments in the BAL fluid as detected by Western blot.

Conclusion: Our data support the hypothesis that col(V) is antigenic for cell-mediated immunity in the lung transplant patient. Strategies targeting this response may be useful in prevention of chronic rejection of the lung.

Abstract# 76

UPREGULATION OF P-GP PROTECTS GRAFT INFILTRATING T-CELLS FROM APOPTOSIS: A MECHANISM FOR IMMUNOSUPPRESSIVE DRUG RESISTANCE. Vera S. Donnenberg,[1] Gilbert J. Burckart,[1] John W. Wilson,[1] Adriana Zeevi,[1] Bartley P. Griffith,[1] Aldo Iacono,[1] Albert D. Donnenberg.[1] *[1]University of Pittsburgh, Pittsburgh, PA.*

Introduction. Immunosuppressive agents target alloreactive T cells through inhibition of cytokine-mediated activation, ultimately resulting in apoptosis of alloreactive cells. We have previously shown that allograft infiltrating T cell are resistant to apoptosis (AST2000). Johnstone et al. have demonstrated that upregulation of P-glycoprotein (P-gp) in cell lines results in protection from apoptosis (Blood 93:1075, 1999). In the present study we investigated changes in in vivo apoptosis of graft infiltrating T cells from lung transplant patients as a function of their P-gp expression.

Methods. We assayed broncho-alveolar lavage (BAL) and peripheral blood mononuclear cells (PBMC) obtained from 27 patients studied on 94 occasions. PBMC were obtained from 18 healthy subjects and peripheral blood progenitor cells (PBPC) from 4 GCSF mobilized autografts were used as a positive control of physiological P-gp function. 4-color flow cytometry was used to measure surface expression of CD3, CD4, CD8, and CD14 together with P-gp function (decrease in R123 fluorescence), apoptosis (Annexin-V binding), expression of pro-apoptotic FAS and anti-apoptotic bcl2, naive/memory markers (CD45RO, CD62L), and activation markers (CD25, CD122, CD71, HLADR, CD43).

Results. Graft infiltrating BAL T cells had highly upregulated constitutive P-gp activity compared to PBMC (CD4 25±4% P-gp active, CD8 25±3% P-gp active, $p<0.003$ and $p<0.00005$ ANOVA, compared to patient PBL (CD4 9±3%, CD8 8±2%). We found that apoptosis was significantly higher in T cells (CD4 and CD8) with low or no constitutive P-gp activity (slope= -0.25 $p=0.008$ and slope= -0.23 $p=0.00005$, respectively). BAL T cells expressed an array of markers that are usually associated with increased susceptibility to apoptosis (CD45RO+/CD62L-/fasbright/bcl2dim).

Discussion and Conclusion. Most immunosuppressive agents are P-gp substrates. We have shown that: 1) BAL T cells upregulate P-gp to levels comparable to those observed in hematopoietic progenitor cells, which confers resistance to immunosuppressive drugs; and 2) Apoptosis is significantly inhibited in the same cells which have upregulated their P-gp despite the clear demonstration of pro-apoptotic and activation markers. Upregulation of P-gp and the subsequent protection from apoptosis suggests a potential mechanism for preferential survival of graft-infiltrating T cells.

Abstract# 77

LONG TERM RENAL FUNCTION IN PEDIATRIC LUNG TRANSPLANT RECIPIENTS. S. P. Hmiel ,[1] Stuart C. Sweet,[1] Anne M. Beck.[1] *[1]Pediatrics, Washington University at St. Louis Children's Hospital, St. Louis, MO.*

Introduction: Nephrotoxicity is a significant complication of long-term calcineurin inhibitor use. Renal function was assessed in pediatric lung transplant recipients with at least one-year survival.

Methods: Retrospective chart review of 127 patients who received first lung transplants between 1/1/90 and 6/1/99, and survived one year. Glomerular filtration rate (GFR) was estimated at baseline, 6, 12, and 18 months, and annually thereafter by Schwartz formula (based on height and plasma creatinine), without correction for nutritional status/body habitus. GFR was estimated in 127 patients, with ages between 0.16 and 23 years At the time of data extraction, 63 recipients (49.6%) were living. Ten recipients received two organs (8 heart-lung, 2 liver-lung), with 59 transplants due to cystic fibrosis (CF). Living donor lobar transplants accounted for 13 (10%) of transplants.

Results: Mean serum creatinine increased after transplantation, from 0.48 ± 0.23 to 0.87 ± 0.36 at 12 mos. and reaching 1.39 ± 0 9 at seven years after transplant. Mean GFR fell from 166±5.7 ml/min/1.73m^2 (mean±S.E.M) at transplant, to 89±2.7 at 12 mos, 85±3.1 at 18 mos, and 79.7± 3.1 at 24 mos, with a slow decline thereafter to 69±4.9 at 60 months post transplant. By 36 mos., 16% of recipients could be classified with chronic renal insufficiency, with GFR<50 ml/min/1.73m^2. Recipients with CF had lower mean GFR at all times post transplant, despite higher pre-transplant GFR. Patients older than 12 years at transplant had lower GFR post-transplant; but disproportionately had CF as their underlying diagnosis. No difference was observed between living and deceased recipients.

Conclusion: Pediatric lung transplant recipients demonstrate loss of renal function after transplant similar to that observed in other solid organ transplant recipients receiving calcineurin inhibitors, which is most dramatic in older patients with CF.

Abstract# 78

COMMUNITY ACQUIRED RESPIRATORY VIRUSES IN LUNG TRANSPLANT PATIENTS: INCIDENCE, DIAGNOSTIC METHODS AND OUTCOMES. Tony N. Hodges,[1] Fernando Torres,[1] Adriana Weinberg,[2] Shaobing Li,[2] Martin R. Zamora.[1] *[1]Pulmonary Sciences/Lung Transplantation Program, University of Colorado Health Sciences Center, Denver, CO; [2]Clinical Virology Laboratory, University of Colorado Health Sciences Center, Denver, CO.*

Purpose: To prospectively define the epidemiology, performance characteristics of diagnostic tests, clinical manifestations and acute sequelae of community acquired respiratory viruses (CRV) in a lung transplant (LTx) cohort.

Methods: From November 1999 to May 2000 we performed prospective viral surveillance on LTx patients (n=93) at our institution. Weekly telephone symptom surveys were performed by two nurses for upper (URI) or lower (LRI) respiratory symptoms. Patients who developed URI signs / symptoms (s/s) (rhinorrhea, sore throat or cough) underwent nasal washes (NW); those with s/s of LRI (wheezing, dyspnea or hypoxia) underwent bronchoalveolar lavage (BAL). Infection was documented by standard methodology; positive rapid antigen (EIA), direct fluorescent antibody or rapid shell vial culture (RSVC). Samples were then analyzed by polymerase chain reaction (PCR) (Hexaplex, Prodese). All pts received the influenza vaccine prior to November. A total of 96 NW and 48 BAL were performed in 72 symptomatic pts. All documented infections were LRI, except 1 RSV and 2 influenza A which were URIs.

Summary:

Isolates by standard methods	#pts	#episodes	incidence
RSV	11	13	11 8%
Parainfluenza	7	9	7 5%
Influenza A	10	11	10 9%
Any virus	28		30 1%

PCR detected 7 additional cases. The sensitivity of PCR was 89.3%, the specificity was 100%. Twenty-five episodes of biopsy-proven acute rejection developed within 90d. Fourteen episodes of superinfection occurred within 13d in 9 pts. Pneumococcus, Pseudomonas and Aspergillus were the isolated pathogens.

Conclusions: There was a significant incidence of CRV in a symptomatic LTx cohort CRV LRI is commonly associated with AR in < 90d and bacterial and fungal superinfection. PCR is more sensitive and has a higher degree of specificity than standard methodology in detecting CRV infection in symptomatic LTX patients. Prospective screening and early treatment may impact the acute and chronic sequelae of CRV in lung transplant recipients.

Abstract# 79

EMERGENCE OF GANCICLOVIR RESISTANT CYTOMEGALOVIRUS IN LUNG TRANSPLANT RECIPIENTS. Ashby Jordan,[1] Sangeeta M. Bhorade,[1] Nell S. Lurain,[2] Julie Leischner,[1] Jaime Villanueva,[1] Wickii T. Vigneswaran,[3] Edward R. Garrity.[1] *[1]Medicine, Loyola University Medical Center, Maywood, IL; [2]Immunology/ Microbiology, Rush University Medical Center, Chicago, IL; [3]Surgery, Loyola University Medical Center, Maywood, IL.*

The emergence of ganciclovir (GCV) resistant cytomegalovirus (CMV) in lung transplantation may lead to increased morbidity and mortality in this transplant population. We compared lung transplant recipients with GCV resistant CMV disease to recipients with GCV sensitive CMV disease at Loyola University Medical Center from 12/96 to 6/00. All 91 lung transplant recipients had routine CMV cultures and antiviral susceptibility testing performed monthly for the first six months after transplantation and every three months thereafter. Resistance was determined both phenotypically by plaque reduction assay and genotypically by direct sequencing of PCR products amplified from the UL97 and DNA polymerase genes. CMV disease/ syndrome was defined as clinical disease with either a positive blood culture or biopsy in the presence of clinical symptoms. All patients received at least 90 days of ganciclovir prophylaxis. Immunosuppressive therapy included tacrolimus (TAC), azathioprine (AZA) or mycophenolate mofetil (MMF) and prednisone (PRED) +/- daclizumab (DAC). 29/91 (32%) patients developed CMV disease/syndrome during this period. 5/91 (6%) patients developed GCV resistant CMV. Patients with GCV resistant CMV had earlier onset of CMV disease, had a trend towards longer courses of IV GCV and had more episodes of CMV disease than patients without resistant CMV disease. There was a trend to increased and early mortality in the resistant group compared to the non-resistant group (3/5 (60%) vs. 8/24 (33%)). The emergence of ganciclovir resistant CMV disease in the lung transplant population may increase morbidity after lung transplantation. Appropriate strategies for the treatment and prevention of resistant CMV are needed.

	D+/R- CMV serostatus	Time to CMV disease (days)	Cumulative IV ganciclovir (days)	Prior CMV episodes
GCV resistant CMV recipients(5)	4/5 (80%)	181 +/- 69	22 +/- 11	2 4 +/- 0 6
GCV sensitive CMV recipients (24)	10/24 (42%)	257 +/- 152	16 +/- 8	1 6 +/- 0 8

Abstract# 80

***NOCARDIA* INFECTION IN LUNG TRANSPLANT RECIPIENTS.**

Shahid Husain,[1] Kenneth R. McCurry,[2] Nina Singh,[3] James H. Dauber,[4] Shimon Kusne.[1] *[1]Infectious Disease, University of Pittsburgh Medical Center, Pittsburgh, PA; [2]Cardiothoracic Surgery, Univerisity of Pittsburgh Medical Center, Pittsburgh, PA; [3]Infectious Disease, VA Medical Center, Pittsburgh, PA; [4]Pulmonary and Critical Care, UPMC, Pittsburgh, PA.*

Background: Unique clinical characteristics, risk factors, and outcome of lung transplant recipients with *Nocardia* infection have not been well defined.

Methods: Cases of *Nocardia* infections in 473 consecutive lung transplant recipients from January 1991-November 2000 at our institution were reviewed.

Results: Nocardiosis developed in 9(1.9%) of 473 lung transplant recipients. Median time to onset was 34 months (range 5.6-102.8 months). Biopsy proven rejection episodes preceded *Nocardia* infection in 56%(5/9) of the patients. *Nocardia* species included, *N.farcinica* in 33%(3/9), *N.nova* in 33%(3/9),*N. asteroides* complex in 22%(2/9), and *N. brasiliensis* in 11%(1/9). All patients had pulmonary involvement; disseminated infection involving central nervous system and bone was noted only in one patient with *N farcinica*. 56%(5/9) of the patients were receiving bactrim prophylaxis and 11%(1/9) were on dapsone. All available isolates of patients who had breakthrough infection on sulfonamide prophylaxis remained susceptible to sulfonamide drugs. Native lung was involved in 75%(3/4) of patients who underwent single lung transplantation. All isolate (6/6) were susceptible to amikacin, imipenem and sulfamethoxazole. Median duration of intravenous therapy was 60 days, and that of maintenance therapy, 180 days. Of patients who survived the acute episode, none (0/6) experienced a recurrence at a median follow up of 19.9 ±8.4 months. Overall mortality was 33%(3/9), all (3/3) of the patients with *N farcinica* but, none(0/6) of those with other *Nocardia* species died.

Conclusion: *Nocardia* infections have unique epidemiologic and clinical characteristics in lung transplant recipients that include, predominance of non-*asteroides* species as pathogens and a predilection to involve the native lung. Over one half of the cases represented breakthrough infection with bactrim susceptible isolates. Deaths occurred exclusively in patients with *N.farcinica*, a species known to be more virulent.

CONCURRENT SESSION 9:
LIVER TRANSPLATATION: HEPATITIS C CLINICAL OUTCOMES

Abstract# 81

THE OUTCOME OF LIVERS FROM HCV+ DONORS.

Ergun Velidedeoglu,[1] Niraj M. Desai,[1] Luis Campos,[1] Kim M. Olthoff,[1] Avi Shaked,[1] Lisa M. Forman,[2] Frederick A. Nunes,[2] Gilian A. Zeldin,[2] Charmaine A. Stewart,[2] Emily Blumberg,[2] John Abrams,[3] Michael R. Lucey,[2] James F. Markmann.[1] *[1]Surgery, University of Pennsylvania Health System, Philadelphia, PA; [2]Hepatology, University of Pennsylvania Health System, Philadelphia, PA; [3]Gift of Life Donor Program, Philadelphia, PA.*

Introduction: An epidemic of hepatitis C virus infection (HCV) has made end-stage liver disease due to HCV infection the most common indication for liver transplantation. The growing prevalence of HCV infection in the general population has also resulted in an increased frequency of potential organ donors that carry (or have been exposed to) the virus. The survival of grafts from HCV+ donors has not been studied in detail. **Methods:** Two study populations were examined retrospectively to determine the outcome of liver grafts procured from HCV+ donors. First, we evaluated the Kaplan-Meier survival of all 13 HCV+ and 103 HCV- grafts that were transplanted at our institution to HCV+ recipients during the 5-year period from 1/1/95 to 12/31/99. In parallel, we analyzed a subset of the UNOS liver transplant database from the same time period which was comprised of adult patients for whom donor and recipient HCV serologies were known (n=13,782). We thus determined the fate of HCV+ and HCV- grafts in HCV+ and HCV- recipients. A Cox proportional hazards analysis was performed to identify variables independently predictive of graft survival. **Results:** For transplants performed at our institution, we found no statistically significant difference in the Kaplan-Meier graft survival of HCV- and HCV+ grafts transplanted to HCV+ recipients (3 year 71.6% vs 69.2%, p = 0.68). The incidence of biopsy proven recurrent HCV post transplant was similar in recipients receiving either HCV+ or HCV- grafts (4/13 vs 18/103, X^2 p = .211). Analysis of UNOS data revealed that the survival of HCV+ grafts (n=194) was equivalent to the survival of HCV- (n=5,384) grafts in HCV+ recipients (3 year 70% vs 67%, p = 0.91). In contrast, survival of HCV- livers in HCV+ recipients was significantly poorer than control patients (n=8,171,3 year 74.2%) in which both donor and recipient were HCV negative (p <0.0001). Multivariate analysis of all patients found recipient but not donor HCV status to be independently predictive of graft survival. **Conclusions:** Analysis of data from a single center and the nationwide UNOS database suggests that transplantation of liver allografts from HCV+ donors to HCV+ recipients results in graft survival comparable to HCV- grafts transplanted to HCV+ recipients. In contrast, recipient HCV positivity is an independent predictor of graft failure compared with patients transplanted for other causes of liver disease. More liberal use of HCV+ donors may assist in expanding donor pool.

Abstract# 82

ORTHOTOPIC LIVER TRANSPLANTATION FOR HEPATITIS C: ANALYSIS OF ALLOGRAFT SURVIVAL USING THE UNOS DATABASE.

Lisa M. Forman,[1,2] Michael R. Lucey.[1] *[1]Gastroenterology, University of Pennsylvania, Philadelphia, PA; [2]Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania, Philadelphia, PA.*

BACKGROUND: Recurrence of HCV infection after liver transplantation is universal and leads to cirrhosis in 20% of recipients by 5 years. Prior studies, while limited by small sample sizes, have not shown a negative impact of HCV on graft survival in the first 5 years. The primary aim of this study was to characterize the influence of HCV on allograft survival after primary liver transplantation. METHODS: A review of all primary adult transplants in the UNOS database from 1992 to 1998, with follow-up through 1999, was performed. Patients with missing HCV serology were excluded as were patients with a diagnosis of HCV but negative serology. Allograft survival was defined as time from transplantation until death or retransplantation. RESULTS: Mean follow-up time was 889 days. HCV positive and negative recipients had similar clinical and biochemical characteristics. The 5-year actuarial allograft survival of HCV positive and HCV negative recipients was 56.7% and 65.6%, respectively(p<0.001). HCV infection was associated with an increased rate of allograft failure (hazards ratio 1.15, 95%CI 1.08-1.22, p<0.001). This effect persisted after adjustment for clinical and biochemical parameters such as ICU status, age, sex, race, bilirubin, and creatinine (Cox proportional hazard methods). Allograft survival at 5 years was significantly less in HCV-infected patients compared to all other diagnostic subgroups except malignancy.

	1-Year Actuarial Survival	5-Year Actuarial Survival
HCV+	78.0%	56.7%
HCV-	78.5%	65.6%
HBV+	78.6%	66.3%
Cryptogenic	71.2%	64.8%
Autoimmune	77.8%	67.6%
ETOH	78.5%	63.2%
Cholestatic	81.3%	70.3%
Metabolic	75.9%	66.2%
Malignancy	72.2%	42.3%

CONCLUSIONS: By five years, HCV infection significantly reduces allograft survival.

Abstract# 83

IMPACT OF HEPATITIS C ON LIVER TRANSPLANT SURVIVAL OVER TEN YEARS- A REPORT OF THE SEOPF LIVER COMMITTEE.

Jon W. Jones,[1] Members of the SEOPF Liver Committee.[2] *[1]Transplant Center, Carolinas Medical Center, Charlotte, NC.*

Hepatitis C is the most common cause for liver transplantation today. The infection is almost universally recurrent in the allograft. Liver failure due to recurrent aggressive Hepatitis C is well recognized, but the long-term impact of Hepatitis C on allograft survival is unknown. We report patient and graft survival of Hepatitis C (+) recipients of liver allografts over 10 years compared to epatitis C (-) recipients.

The SEOPF Registry was used to examine data on liver transplants performed between Oct. 1, 1987 and Dec. 31, 1998. A total of 7,203 liver transplants were performed at member centers. Primary diagnosis and survival data availability reduced the number for analysis to 5,695. Of these 1,714 (30%) were hepatitis C (+) and 3,981 (70%) were hepatitis C (-). Donor and recipient characteristics were compared by chi-square or ANOVA, while graft and patient survival were compared using Kaplan-Meier estimates. Comparison of survival curves were performed with both log-rank and Wilcoxon tests. A multivariate Cox proportional hazards model was used to assess the impact of Hepatitis C on survival using known variables.

Hepatitis C (+) recipients differed from Hepatitis C (-) recipients significantly (p=0.0001) for the following characteristics: Age 48 yrs vs 42 yrs, Weight 83 kg vs 71 kg, Race 88% vs 83% caucasian, Gender 67% vs 56% male. The donors for each group differed by: Age 33 vs 29 yrs, Vasopressor use in 55% vs 48%. Patient survival was significantly worse in the Hepatitis C (+) recipients [Log-rank (p=0.0001) Wilcoxon (p=0.0003)] over the 10 yr study period. Overall survival for Hepatitis C (+) vs Hepatitis C (-) recipients at 1yr: 86% vs 89%, 5 yrs: 70% vs 76%, 10 yrs: 54% vs 56% . Using the Cox regression model and adjusting for demographic variables and immunosuppression used: Hepatitis C(+) recipients had a significantly higher risk for graft failure(RR 1.25, p=0.0006); other variables of significance were repeat transplant RR 2.671, Recipient caucasian race RR 0.778, and Donor caucasian race RR 0.763. Previous studies have shown equivalent survival for Hepatitis C (+) vs (-) recipients at 1 and 5 yrs. This study demonstrates a significant negative impact of Hepatitis C in liver transplant patient survival over 10 years. While statistically significant however, this difference is not clinically important and is encouraging for Hepatitis C (+) liver transplant recipients.

Abstract# 84

ADVERSE EFFECTS OF HCV RECURRENCE IN OVER FIVE HUNDRED LIVER TRANSPLANTATIONS. Rafik M. Ghobrial,[1] Douglas G. Farmer,[1] Randy Stedman,[2] Hasan Yersiz,[1] Charles Lassman,[3] Natale Danino,[1] Eric Collisson,[1] Steve Han,[4] Sammy Saab,[4] Ronald W. Busuttil.[1] *[1]Dumont-UCLA Liver Transplant Center, UCLA School of Medicine, Los Angeles, CA; [2]Anesthesiology, UCLA School of Medicine, Los Angeles, CA, [3]Pathology, UCLA School of Medicine, Los Angeles, CA; [4]Hepatology, UCLA School of Medicine, Los Angeles, CA.*

Background: Hepatitis C virus (HCV) is the most common indication for OLT. The full effect of HCV recurrence on OLT is yet to be determined. This study, evaluated recurrence rates, effects on pt and graft survival and outcome of HCV (+) donors.

Methods: Retrospective review of 511 pts who underwent OLT for HCV. Recurrence was estimated by biopsies. Univariate and Kaplan Meier methods were utilized for analyses. Median follow-up was 30.2 months.

Results: Of 511 pts, 431 (84.3%) received one transplant and 80 (15.7%) required retransplantation. Fifty-nine (11.5%) pts received livers from HCV (+) donors. Overall 1, 3 and 5 yr pt survivals were 84.8%, 76.4% and 68.4%, respectively. For graft survival analysis, graft loss was defined as graft failure leading to pt death or a need for retransplantation. Graft survival rates were 75%, 65.5% and 57.5% at 1, 3 and 5 years, respectively. Excluding follow-up time, univariate comparison identified HCV recurrence as a negative predictor for pt (Risk ratio, RR 1.54, P 0.178) and graft survival (RR 1.84, P 0.007). Time factored analysis demonstrated that recurrence significantly decreased graft (88%, 85% and 81% without recurrence vs 85%, 78% and 67% with recurrence, P 0.49), but not pt, survival at 1, 3 and 5 years, respectively. However, pts who recurred within 12 months post OLT exhibited a significantly elevated mortality rate (death rate 7.5, RR 3.3) when compared to those who recurred at 24 (DR 4, RR 1.7) or 36 (DR 1, RR 0.4) months, respectively. Similarly, graft failure rates (GFR) were significantly increased when recurrence occurred within 12 months (GFR 12, RR 2.9) when compared to recurrence at 24 (GFR 5.9, RR 1.4) or 36 (3.6, RR 0.9) months posttransplantation. Additionally, HCV (+) donors significantly decreased median time to recurrence (22.8 vs 37.7 months, P 0.05) and increased recurrence rates (36%, 55% and 71% vs 26%, 41% and 53%, P 0.05) at 1, 2 and 4 years, respectively when compared to HCV (-) donors.

Conclusions: HCV recurrence significantly reduces graft survival and may impact long-term pt survival. Early HCV recurrence adversely affects pt and graft survival. HCV (+) donors are accompanied with early recurrence and should be utilized with caution.

Abstract# 85

HCV VIRAL LOAD AT ONE MONTH AFTER LIVER TRANSPLANTATION IS A PROGNOSTIC FACTOR FOR THE DEVELOPMENT OF RECURRENT HEPATITIS C AND LIVER FAILURE AFTER TRANSPLANTATION. Francisco Suarez,[1] Alejandra Otero,[1] Manuel Gomez-Gutierrez,[1] Francisco Arnal,[1] Carlos Fernandez-Selles,[1] Jose Luis Vazquez-Iglesias,[1] Rafael Manez.[1] *[1]Gastroenterology and Transplantation, Juan Canalejo Medical Center-University of La Coruna Health Institute, La Coruna, Spain.*

Introduction: Liver transplantation (LTx) for hepatitis C virus (HCV) infection is associated with a virtually universal recurrence of infection and disease after transplantation. Although it has been shown that HCV viral load in blood increases after transplantation compared with levels previous to the transplant surgery, the consequences of these changes in the recurrence of liver disease are unclear. In this study we investigate the impact of HCV viremia in the development of liver disease after transplantation.

Material and Methods: Between 1995 and 1998 a total of 134 liver transplants were performed at our institution. Of these, 42 (31%) were for HCV-related cirrhosis. In these patients, HCV viral load was studied before and at 1, 3, 6, 12, 24 and 36 months after LTx using the Amplicor HCV test. Biopsy samples were obtained at 3 months, one year and annually thereafter, besides when it was clinically indicated. A multivariate stepwise Cox's regression was performed to identify factors independently associated with hepatitis C recurrence-related mortality.

Results: With a follow-up of at least two years, recurrent hepatitis C in the grafted liver was observed in 35 (83%) of the patients. Patients with recurrent hepatitis had higher HCV viral load at one month after transplantation (median 4.4 x 10^5 range 684 to 59.9 x 10^6) compared with non-recurrent patients (median 2.8 x 10^5 range 121 to 8.3 x 10^5; $p < 0.05$). Nine patients (21%) died. In 6 patients (14%) the cause of death was a liver failure secondary to HCV recurrence, in two a tumor recurrence, and in one bacterial sepsis. HCV viral load at one month (RR: 1.01; 95% CI: 1-1.02) and fibrosis in the liver biopsy at one year (RR: 2.7; 95% CI: 1.3-5.7) were the factors associated with hepatitis C recurrence related death.

Conclusion: HCV viral load at one month after transplantation is a prognostic factor for the development of both, recurrent hepatitis C and liver failure after transplantation. Therapies addressed to reduce this viral load may have an impact in the recurrence of the disease and its related mortality.

Abstract# 86

INCREASED MORTALITY RISK IN HEPATITIS C-POSITIVE LIVER TRANSPLANT RECIPIENTS WHO DEVELOP POSTTRANSPLANT DIABETES MELLITUS. S. Baid, A. B. Cosimi, M. L. Farrell, D. A. Schoenfeld, S. Feng, D. Ko, R. T. Chung, N. Tolkoff-Rubin, M. Pascual. *[1]Renal and Transplantation Units, Massachusetts General Hospital, Boston, MA.*

Background: Recent studies have linked hepatitis C virus (HCV) infection to post-transplant diabetes mellitus (PTDM) after liver transplantation (OLT). Both HCV infection and PTDM have been associated with an increased susceptibility to infections and may be risk factors for enhanced patient (pt) mortality after OLT. The aim of the current study was to analyze risk factors of PTDM and pt mortality after OLT.

Methods: Between 1/91 to 12/98, 185 OLT were performed in 176 adult pts at our institution. Eighteen pts who died within one month after OLT or who were lost to follow-up were excluded, leaving 47 HCV (+) and 111 HCV (-) pts for analysis. We analyzed demographics, etiology of liver failure, pretransplant alcohol abuse, prevalence of diabetes mellitus, clinical course, and immunosuppressive and anti-rejection treatments in the two groups. Cox proportional-hazards model was used to identify determinants of PTDM and mortality. Pt survival was analyzed using Kaplan-Meier actuarial survival curves.

Results: The prevalence of PTDM was significantly higher in HCV (+) than in HCV (-) pts (64% vs. 28%, p=0.0001). HCV infection (HR 2.5, p=0.001) and each methylprednisolone bolus (HR 1.09, p=0.02) were found to be independent risk factors for the development of PTDM. The cumulative mortality in HCV (+) vs HCV (-) patients was 43% vs 27% (p=0.06), and the Kaplan-Meier survival curves revealed significantly worse long-term survival for HCV (+) pts (p=0.02). Death in 75% (12/16) of HCV (+) pts was related to infectious complications, as opposed to only 23% (6/26) in HCV (-) pts (p=0.001). The cumulative mortality in HCV (+) PTDM (+) vs HCV (+) PTDM (-) pts was 56% vs 14% (p=0.01). By multivariate analysis, PTDM was found to be an independent risk factor for overall mortality and infection-related mortality (respective HR: 3.67, p<0.0001 and 7.18, p=0.002).

Conclusions: HCV infection and methylprednisolone boluses were independent risk factors for developing PTDM after liver transplantation. A higher cumulative mortality was found in HCV (+) recipients who developed PTDM. PTDM emerged as an independent risk factor for overall mortality and for infection-related mortality. Limitation of steroid bolus therapy to only biopsy-proven rejection is mandatory after liver transplantation, particularly in HCV-infected recipients.

Abstract# 87

CRYOGLOBULINEMIA PREDICTS POOR OUTCOME FOLLOWING LIVER TRANSPLANTATION IN PATIENTS WITH HEPATITIS C. Stephen C. Rayhill,[1] Warren N. Schmidt,[2] Adel Bozorgzedeh,[1] Daniel Katz,[1] Michael D. Voight,[2] Douglas R. LaBrecque,[2] Rachel Miller,[2] Mohammed Ibrahim,[1] Patricia A. Kirby,[3] Frank A. Mitros,[3] Youmin Wu.[1] *[1]Surgery, University of Iowa, Iowa City, IA; [2]Medicine, University of Iowa, Iowa City, IA; [3]Pathology, University of Iowa, Iowa City, IA.*

Patients with hepatitis C (hep C) who have measurable cryoglobulinemia (cryo) suffer more rapidly progressive fibrosis than those lacking cryo (no cryo). It is unknown whether the presence of cryo in transplant patients similarly predicts poor outcome. Therefore, we evaluated the effect of the presence of cryo in our transplant recipients.

Methods: Using our longitudinal database, survival for all recipients of liver transplants for cirrhosis due to Hep C was analyzed (1993 to the present) based on the presence or absence of cryo. The population was also analyzed after excluding patients who lost their grafts for reasons, such as PNF, other than recurrent Hep C (non-HCV losses excluded). Overall patient and graft survival, and survival without biopsy proven hep C recurrence (without HCV recurrence), were computed using Kaplan-Meier estimates and compared using the log rank test. Cox multivariate analysis was used to determine relative risks. **Results:**

	All Patients			non-HCV Losses		
1 Year Survival	**No Cryo**	**Cryo**	**p**	**No Cryo**	**Cryo**	**p**
n	34	26		29	23	
Patient	90%	75%	0.1	100%	82%	0.02
Graft	84%	68%	0.1	100%	77%	0.007
Patient without HCV recurrence	54%	30%	0.02	60%	32%	0.02
Graft without HCV recurrence	51%	28%	0.03	60%	31%	0.01

In patients with cryo, the relative risk of death, graft loss, and biopsy proven recurrence was at least 4, 3, and 4. Correction for recipient and donor age in the multivariate model strengthened the findings.**Conclusions:** Patients with cryoglobulinemia are at greater risk for early hepatitis C recurrence and consequently, early patient and graft loss.

Abstract# 88

EARLY RECURRENCE OF HEPATITIS C AFTER LIVER TRANSPLANTATION WITH DACLIZUMAB INDUCTION. Gustavo Marino,[1] Vinod K. Rustgi,[1] Carlos E. Marroquin,[2] Jeffrey S. Plotkin,[3] Paul C. Kuo,[2] Amy Lu,[2] Scott Batty,[4] Lynt B. Johnson.[2] *[1]Hepatology, Georgetown University, Washington, DC; [2]Surgery, Georgetown Univerisity, Washington, DC; [3]Anesthesiology, Georgetown University, Washington, DC; [4]Surgery, Walter-Reed Hospital, Washington, DC.*

Daclizumab (D) has reduced the frequency of rejection in kidney transplant recipients; however its use in liver transplant recipients has not been clearly defined.

Aim: To evaluate the outcomes of liver transplantation (OLT) in patients (pts) with hepatitis C (Hep C) receiving daclizumab induction therapy.

Materials and methods: 26 consecutive pts with Hep C undergoing OLT were analyzed. 13 pts received D(Zenapax) 1mg/kg IV, immediately before and two weeks post-transplant. Additional immunosuppression was based on a standard protocol of steroids with calcineurin inhibitors. Mycophenolate mofetil was used selectively. Pts were followed an average of 14 months (6 - 25). The diagnosis of recurrence of Hep C was confirmed with liver biopsy. Multivariate analysis and T-test were performed where appropriate

Results: Demographic data, incidence of recurrent Hep C, rejection and laboratory values at 6 months after OLT are in Table 1. Graft survival at 6 months was 92% (12/13) in the D group vs. 100% (13/13) in the non-D group. Hep C recurrence was significantly higher in the D group (53.8% vs 15.4%; p= 0.048); the mean interval from OLT until Hep C recurrence was 143 days; all cases of recurrence in this group developed a severe cholestatic pattern. One patient developed graft failure and underwent retransplantation 15 months after the first procedure, and died 10 months after re-OLT. A second patient developed graft failure and died.

Induction therapy	Zenapax (D)	Non-Zenapax (Non-D)
N	13	13
Age (mean)	53	47
Sex (f/m)	3/10	5/8
Recurrent Hep C	7 (53.8%) *	2 (15.4%)
Rejection	4 (30.8%)	6 (46.1%)
T Bil@6 months	4.93 *	0.78
Alk phos@6 months	200.4 *	91

*p<0.05

Conclusions: A higher incidence of early hepatitis C recurrence was found on patients receiving daclizumab during induction therapy. IL-2 inhibitors should be used cautiously in this setting.

Abstract# 89

PEGYLATED (40 kDa) INTERFERON ALFA-2A (PEGASYS®) IN POST-LIVER TRANSPLANT RECIPIENTS WITH ESTABLISHED RECURRENT HEPATITIS C: A PRELIMINARY REPORT. Caroline Riely,[1] Peter Ferenci,[2] Markus Peck-Radosavljevic ,[2] Wolfgang Vogel,[3] Michael Voigt,[4] Ian M. Marks,[5] Stephen C. Pappas.[5] *[1]Univ of Tennessee, Memphis, TN; [2]Univ of Vienna, Vienna, Austria; [3]Med. Univ Innsbruck, Innsbruck, Austria; [4]Univ of Iowa, Iowa City, IA; [5]Roche Lab, Inc, Nutley, NJ.*

Background: Recurrent infection with hepatitis C virus (HCV) is a significant cause of allograft dysfunction and progression to end-stage liver disease and allograft failure after orthotopic liver transplantation (OLT). The effectiveness of standard IFN in patients with established recurrent HCV has not been satisfactory, leading to low sustained viral response (SVR) rates. **Objective:** To investigate the safety and efficacy of PEGASYS® in patients with established recurrent hepatitis C post-OLT. **Methods:** Previously untreated HCV-infected post-OLT recipients who were transplanted between 6 and 60 months prior to study initiation were randomized to one of two groups in a 1:1 ratio: Group A: PEGASYS® 180 µg qw for 48 weeks (n=28); Group B: untreated (n=28). Patients were stratified according to viral load (HCV RNA ≤ 1 x 10^6 IU/ml [low] vs. > 1 x 10^6 IU/ml [high]). A positive response to therapy is defined as HCV RNA below the level of detection (< 50 IU/ml as assessed by AMPLICOR MONITOR v2.0) or a 2 $\log_{10}$ drop compared to baseline; SVR is defined as HCV RNA < 50 IU/ml 24 weeks following the end of therapy. **Results:** The overall baseline characteristics of the patients were as follows: mean age 52.9 ± 8.7 years, 79% male, 89% high viral load, 79% HCV genotype-1. Twenty-eight patients have completed at least 4 weeks of the study. The response to therapy is summarized in the table. PEGASYS® treatment has been well tolerated with no unexpected adverse events (AEs); no rejection episodes have been observed in either study group. **Conclusion:** These preliminary findings reveal a trend for early (week 4) antiviral effects of PEGASYS® in the post-OLT population. These results demonstrate a similar response and AE profile to those observed in non-liver transplant patients and support continued studies investigating the use of PEGASYS® in post-OLT patients with recurrent HCV.

Group	HCV RNA Neg. (Week 4)	HCV RNA 2-log drop (Week 4)	HCV RNA Neg. (Week 12)	HCV RNA 2-log drop (Week 12)	Rejection episodes (Week 12)
PEGASYS®	2/14 (14%)	5/14 (36%)	2/4 (50%)	3/4 (75%)	0
Untreated	0/11 (0%)	0/11 (0%)	0/5 (0%)	0/5 (0%)	0

Abstract# 90

IMPROVEMENTS FROM STEROID WITHDRAWAL AFTER PANCREAS TRANSPLANTATION: 1-YEAR RESULTS OF A PROSPECTIVE, RANDOMIZED OPEN-LABEL STUDY. Rainer W.G. Gruessner,[1] David E.R. Sutherland,[1] Elizebeth Parr,[1] Abhinav Humar,[1] Raja Kandaswamy,[1] Angelika C. Gruessner.[1] *[1]Surgery, University of Minnesota, Minneapolis, MN.*

Purpose: In a preliminary analysis, we had recently reported favorable results for steroid withdrawal after pancreas (Pa) transplantation (Tx). We now present the 1-year results of a prospective, randomized open-label study regarding graft and patient (pt) survival, incidence of rejection (rej), lipid metabolism, and quality of life. Methods and Patients: Only pts with functioning Pa grafts between 6 and 36 months post Tx were enrolled. All pts had no rej episodes (RE) 6 months prior to enrollment. A total of 50 pts [25 Pa after Kidney (Kd) Txs (PAK), 25 simultaneous Pa/Kd Txs (SPK)] were randomized to standard immunosuppressive therapy (S) or steroid withdrawal (W). Post Tx-preenrollment immunosuppression consisted of tacrolimus (8 to 10 ng/ml), MMF (1.5 g/day), and steroids (5 mg). In the PAK group (n=25), 12 pts were randomized to S, 13 to W; in the SPK group (n=25), 11 to S and 14 to W. There were no differences in demographics between the S and W groups for both PAK and SPK. Results: Pt and graft survival at 1 year was 100% in the S and W groups for both PAK and SPK. There were two RE. 1 Pa rej (PAK_S), 1 Kd rej (PAK_W); the differences in RE between groups were not significant. PAK group: Compared to preenrollment, serum creatinine (CR), glucose (Gluc), hemoglobin (Hgb) A1C, cholesterol, triglyceride, and urinary amylase (UA) levels were not significantly different in the W and S groups; but cholesterol and triglyceride levels were significantly lower in the W vs S group. SPK group: Compared to preenrollment, CR, Gluc, Hgb A1C, and UA levels were not significantly different in the W and S groups; but cholesterol and triglyceride levels were significantly lower in the W vs S group and significantly lower in the W (but not S) group vs preenrollment. Steroid related side-effects and quality of life improved in both W (but not S) groups. Conclusion: 1) 1-year of steroid withdrawal did not result in a higher incidence of rej. There were no significant differences in pt and graft survival, graft loss from rej, and incidence of RE between S and W groups. 2) There was a significant improvement in cholesterol and triglyceride levels in the W groups (both PAK and SPK) vs S groups and for SPK_W (but not PAK_W) vs preenrollment. 3) A lower incidence of steroid-associated side effects resulted in an improvement in quality of life for the W (but not S) groups. 4) Steroid withdrawal after Pa Tx is safe and effective.

Abstract# 91

RAPID CORTICOSTEROID WITHDRAWAL IN SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. Dixon B. Kaufman,[1] Joseph R. Leventhal,[1] Lorenzo G. Gallon,[2] Michele A. Parker,[2] Frank P. Stuart.[1] *[1]Surgery, Northwerstern University, Chicago, IL, [2]Medicine, Northwestern University, Chicago, IL.*

This is the first reported US experience of rapid corticosteroid withdrawal in SPK tx. A cohort of 32 consecutive SPK tx recipients were studied in which only 6 days of corticosteroids were given. All pts received induction therapy with rabbit ATG (15 mg/kg x 8 doses over 14 d); maintenance therapy was with either tacrolimus (target trough: 9-11 ng/ml) / MMF (3 gm/d, n=15) or tacrolimus / sirolimus (4 mg/d, no levels, n=17). Tx was performed 2/00 - 11/00 (mean f/u: 5.0 mo). Once withdrawn off steroids, none of the pts had to be placed back on prednisone. The outcomes were compared to a control group of 87 SPK tx (performed 7/95 - 12/99, mean f/u. 32.0 mo). The control pts all received a standard steroid taper with MMF/tacrolimus(same levels and doses as study group), and induction with equine ATG (n=50) or an IL-2R antagonist (n=37). Demographics were the same in both groups. Graft loss was defined as death or return to dialysis (kidney) or need for insulin (pancreas). Kaplan-Meier analysis was used.

Group (N)	Patient	Kidney	Pancreas	Rejection	CrCl (3 mo)	WBC (base)	WBC (3 mo)
Steroid-free (32)	100%	100%	100%	3.3%	77±25	8.6±2.2	3.8±2.2
Controls (87)	98.8%	96.5%	93.1%	12.7%	73±17	8.5±2.5	8.5±2.5

The actuarial six month patient, kidney and pancreas survivals, and rejection rates were better in the steroid withdrawal group, but not statistically significantly different. Renal allograft function (3 mo post-tx), as defined by creatinine clearance (CrCl), was similar. Mean WBC (3 mo post-tx) was significantly lower in the steroid-withdrawal group. The tolerability of MMF versus sirolimus in the steroid withdrawal group was also analyzed. Data on cholesterol and triglyceride levels 3 mo post tx showed no differences in the MMF and sirolimus groups. Platelet counts were reduced in the sirolimus group only (baseline 307K vs 270K 3 mo post-tx). Interestingly, there was no difference in the WBC (3 mo post-tx) in the MMF and sirolimus groups (4.2K versus 3.5K, respectively), however, 2 MMF pts were converted to sirolimus for leukopenia (n=2). Two additional MMF pts were converted to sirolimus for lower GI symptoms (n=2). One pt was converted from sirolimus to MMF for GI symptoms. This study showed excellent early results of rapid corticosteroid withdrawal in SPK tx using induction therapy and either MMF/tacrolimus or sirolimus/tacrolimus maintenance immunosuppression.

Abstract# 92

A MULTICENTER, OPEN-LABEL, COMPARATIVE TRIAL OF 2 DACLIZUMAB DOSING STRATEGIES VS. NO ANTIBODY INDUCTION IN COMBINATION WITH TACROLIMUS, MYCOPHENOLATE MOFETIL, AND STEROIDS IN SIMULTANEOUS KIDNEY-PANCREAS TRANSPLANTATION: 6 MONTH ANALYSIS. R. J. Stratta,[1] R. R. Alloway,[2] A. Lo,[2] E. Hodge,[3] The PIVOT Investigators. [1]*Surgery-Transplant, University of Tennessee-Memphis, Memphis, TN,* [2]*Medicine, University of Cincinnati, Cincinnati, OH;* [3]*Roche, Laboratories.*

This study is designed to determine the safety and efficacy of 2 dosing regimens of daclizumab as an adjunctive immunosuppressive agent compared to no antibody induction in simultaneous kidney-pancreas transplant (SKPT) recipients receiving tacrolimus, mycophenolate mofetil, and steroids as primary immunosuppression.

Methods: This multicenter, prospective, open-label, randomized study currently has 263 patients enrolled. Eligible SKPT patients were randomized into 3 groups: daclizumab 1mg/kg/dose every 14 days for 5 doses (Group I), daclizumab 2mg/kg/dose every 14 days for 2 doses (Group II), and no antibody induction (Group III). The primary endpoint is a composite of the incidence of presumed or biopsy-proven acute kidney or pancreas allograft rejection, graft loss, or death within the first 6 months post-transplant.

Results: This is an interim analysis of the first 182 SKPT patients enrolled in the study with a minimum follow-up of 6 months [Group I (n=76), Group II (n=81), and Group III (n=25)]. Demographic and transplant characteristics were similar among the 3 groups At 6 months, patient, kidney, and pancreas graft survival rates were similar among the 3 groups. The probability of either kidney or pancreas allograft rejection at 6 months was 25%, 13%, and 41% in Groups I, II, and III, respectively. The median time to first acute rejection of either the kidney or pancreas was 23, 58, and 25 days in Groups I, II, and III, respectively. At 6 months, the actuarial event-free survival (no acute rejection, allograft loss, or death) was 62%, 79%, and 48% in Groups I, II, and III, respectively. There were no differences in the incidence of infectious complications among the groups and no serious adverse events associated with daclizumab were observed. All 3 groups have excellent renal and pancreas function at 6 months.

Conclusions: The 2-dose regimen appears to be as effective as the 5-dose regimen in preventing acute rejection and is associated with the lowest acute rejection rates and the highest rate of event-free survival. The onset of acute rejection appears to be delayed in patients receiving the 2-dose regimen. Definitive conclusions on the benefits of daclizumab compared to no antibody induction await completion of patient enrollment and follow-up.

Abstract# 93

TACROLIMUS (TAC)AND MYCOPHENOLATE MOFETIL (MMF) +/- ANTIBODY INDUCTION IN SIMULTANEOUS PANCREAS KIDNEY (SPK) TRANSPLANTATION: ONE YEAR RESULTS. G. W. Burke,[1] D. B. Kaufman, D. S. Bruce, D. Sutherland, C. P. Johnson, A. O. Gaber, R. M. Merion, E. Schweitzer, C. L. Marsh, S. A. Gruber, E. Alfrey, J. P. Leone, W. Conception, M. D. Stegall, P. S. Gores, G. Danovitch, P. J. Nunnally, A. K. Henning, W. E. Fitzsimmons. [1]*University of Miami, Miami, FL.*

A prospective, randomized, multicenter study was conducted to assess the effect of antibody induction in SPK transplantation in patients receiving a maintenance immunosuppressive regimen of TAC (target troughs 10-15 ng/mL beyond month 3), MMF (2 gms/d) and a steroid taper. Of 174 patients transplanted, 87 received induction therapy and 87 received no induction therapy. The induction agent used was based on standard practice at each investigational site. Results at 1 year of follow-up are reported below (note that death with a functioning graft counted as graft loss).

Parameter	Induction (n=87)	Non-induction (n=87)
One Year Patient Survival	96.6%	96.6%
One Year Kidney/Pancreas Survival	96.6%/83.9%	92.0%/83.9%
Any Treated Rejection	24.1%	29.9%
Biopsy-Confirmed and Treated Rejection	19.5%	25.2%
Biopsy-Confirmed and Treated Acute Kidney Rejection	12.6%	21.8%
Antilymphocyte Therapy for Rejection	12.6%	19.5%

The odds of a biopsy-confirmed and treated acute kidney rejection were 5.3 times greater in black patients than in non-black patients, despite higher doses and comparable median trough concentrations of TAC. This effect was independent of treatment received. At month 6, there were no differences in the incidence of serious infections, including tissue invasive CMV disease, but CMV viremia/syndrome was higher in the induction group (13.8% vs 5.8%). The median serum creatinine in both treatment groups was 1.3 mg/dL at 1 year. Pancreatic function, assessed by serum amylase, hemoglobin A_1C and glucose was similar between treatment groups. Less than 10% of patients discontinued TAC and approximately 25% of patients discontinued MMF. In conclusion, TAC and MMF with or without antibody induction provides excellent safety, tolerability and efficacy in SPK transplant patients. Refinement of the immunosuppression regimen is merited in black patients due to a higher likelihood of rejection. Induction therapy resulted in an increased incidence of CMV viremia/syndrome (p=0.07) at 6 months, but fewer biopsy-confirmed and treated acute kidney rejection episodes (p=0.08)in the first year.

Abstract# 94

MANAGEMENT OF TACROLIMUS-INDUCED HYPERGLYCEMIA FOLLOWING PANCREAS TRANSPLANTATION. Benjamin Philosophe,[1] Anne M. Wiland,[1] David K. Klassen,[1] Eugene J. Schweitzer,[1] Alan C. Farney,[1] John O. Colonna,[1] Clarence Foster,[1] Adam M. Frank,[1] Bruce E. Jarrell,[1] Stephen T. Bartlett.[1] [1]*Surgery, University of Maryland Medical Center, Baltimore, MD.*

The use of tacrolimus with steroids has markedly improved outcome following pancreas transplantation. In some patients however, the benefits have been hampered by tacrolimus-induced islet toxicity resulting in hyperglycemia. We have identified twenty-five patients (8 SPK, 7 PTA, 5 PAK, 5 SPLK) with hyperglycemia and functioning pancreas grafts based on elevated C-peptide levels. Islet cell toxicity was confirmed histologically in 50% of these patients. Four therapeutic approaches were undertaken to correct the hyperglycemia. Group 1- adding a hypoglycemic agent with no changes in immunosuppression. Group 2- lowering tacrolimus dose only. Group 3- changing from tacrolimus to cyclosporine based immunotherapy. Group 4- changing to rapamycin-based immunotherapy (either lowering or eliminating tacrolimus). Results: For group 1, 2/3 patients responded. For group 2, 1/5 patients responded and 1 had a rejection as a result of the change. In group 3, 6/8 patients responded, but 4/8 had rejection following the immunosuppression changes. In contrast, all patients treated with rapamycin replacement (group 4) responded to the changes; 1/8 had >50% reduction in insulin requirement and 7 of 8 patients were rendered euglycemic without the need for exogenous insulin. Futhermore, 0/8 rejected following the changes in immunosuppression (p=0.07 in comparison to group 3). Conclusion: Tacrolimus-induced hyperglycemia can be successfully managed by changing to rapamycin-based immunosuppresion without signficant risk of ensuing rejection.

Abstract# 95

TACROLIMUS VERSUS CYCLOSPORINE IN PRIMARY SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. PRELIMINARY RESULTS OF A MULTICENTRE TRIAL. F. Saudek,[1] J. Malaise,[1] R. Margreiter,[1] EUROSPK Study Group.[1] [1]*EURO SPK Central Office, Brussels, Belgium.*

We present the 6 months interim analysis of an open, prospective, randomised, parallel-group study,that has been designed to include 200 SPK transplant recipients from 10 centres in Europe and 1 in Israel.Following induction with antithymocyte globulin, patients were either given tacrolimus(Tacro)or ciclosporine-microemulsion (Ciclo)concomitant with mycophenolate mofetil and steroids.In total,174 patients with at least 6 months follow-up form the basis of this intent-to-treat analysis,87 patients were in the Tacro group and 87 patients in the Ciclo group.Despite randomisation, some baseline characteristics differed significantly between the groups:on average,patients in the Tacro group were older,less likely to receive dialysis before transplantation and more likely to have a portal venous drainage of their pancreas graft.The rate of biopsy-proven rejection was 29.0% in the Tacro group compared with 41.9% in the Ciclo group.At Month 6, patient survival was 98.9%(Tacro) and 100.0%(Ciclo).Graft survival at 6 months for the kidney was 97.4% in the Tacro group and 93.7% in the Ciclo group, for the pancreas it was 96.5% in the Tacro group and 83.4% in the Ciclo group (p=0.0027).The most frequently reported adverse events were urinary tract infection (38.5%),CMV-infection (31.6%), abdominal infection (12.0%)and abdominal drain contamination(21.0%) with no significant difference between the two groups. Irrespective of the cornerstone immunosuppressant used, the incidence of urinary tract infection was greater in patients with bladder drainage of the pancreatic graft than with the enteric drainage(70% vs 32%, p=0.0001).The incidence of abdominal infection was increased in patients previously treated with peritoneal dialysis vs. hemodialysis or no dialysis (24% vs. 9%, p= 0.017).At 6 months, serum creatinine levels were 1.35 mg/dL (Tacro) and 1.47 mg/dL (Ciclo).Fasting glucose,C-peptide and HbA1C were 96 mg/dl,3.4 ng/mL and 5.5% in the Tacro group compared with 91 mg/dl, 5.3 ng/ml and 5.5% in the Ciclo group.The mean length of initial hospital stay was 32 days in the Tacro group and 41 days in the Ciclo group (p=0.0076).Mean daily MMF dose was 1454 mg in the Tacro group and 1857 mg in the Ciclo group.CONCLUSION: Pancreas graft survival at 6 months following SPK is better and hospitalisation is shorter on Tacrolimus-based therapy than on Ciclosporin-based therapy. Peritoneal dialysis and bladder drainage increase the risk of infection in primary SPK.

Abstract# 96

EXPERIENCE WITH RAPAMYCIN IN PANCREAS TRANSPLANTATION. Jon S. Odorico, John D. Pirsch, Yolanda T. Becker, Bryan N. Becker, Hans W. Sollinger. *[1]Department of Surgery, University of Wisconsin-Madison, Madison, WI.*

Objective: To present a single center experience of rapamycin immunotherapy in pancreas transplantation where rapamycin was utilized as either induction, rejection rescue, or conversion therapy.

Study Population: Thirty-five patients who underwent pancreas transplantation (25 simultaneous pancreas-kidney, SPK; 10 solitary pancreas, SP) were treated for a mean of 5.0 months with rapamycin (mean dose 3.1 mg daily, range 2-8 mg) Rapamycin was used as part of an induction and maintenance immunosuppressive protocol in 18 patients: in 11 SPK transplants, rapamycin was used in a three drug protocol with Neoral/Prednisone. In 3 SPK transplants who experienced post-operative delayed renal graft function and in 4 SP transplants who demonstrated pretransplant renal insufficiency or mycophenolate mofetil (MMF) intolerance, it was used as part of a four drug immunosuppressive protocol in combination with low dose tacrolimus/MMF/ Prednisone. All patients received anti-T cell antibody induction therapy with an anti-IL-2 receptor monoclonal antibody (16) or Thymoglobulin (2). Of the remaining 17 patients, 5 were treated with rapamycin as rescue therapy for acute rejection and 12 were converted from their primary maintenance therapy for either renal dysfunction/ calcineurin nephrotoxicity (9), calcineurin neurotoxicity (1), glucose intolerance (3), and MMF-induced GI toxicity (2).

Results: In follow-up, there were 2 deaths, not directly attributable to rapamycin therapy. Of the 18 patients who received rapamycin as primary therapy, 5 suffered acute rejection episodes (4 cellular, 1 humoral), and one patient lost both grafts. Of the 5 patients who were converted for acute renal allograft rejection rescue, 4 remain free of recurrent rejection in follow-up, and one recurrent rejection was successfully treated. While one patient eventually lost a kidney graft and was retransplanted, the remaining 4 kidneys and 5 pancreases continue to function. In 5 of 35 patients, rapamycin was discontinued for leukopenia (1), poor wound healing (1), stable renal function (1), acute rejection (1), and unknown (1).

Conclusion: Early experience with rapamycin demonstrates successful induction and maintenance immunotherapy in pancreas transplant patients who are unable to receive full dose calcineurin or antimetabolite therapy. Furthermore, this study suggests rapamycin-based immunosuppression is effective as conversion therapy in a small series of difficult patients after pancreas transplantation.

Abstract# 97

>10 YEAR FOLLOW-UP AFTER PANCREAS TRANSPLANTATION. Rainer W.G. Gruessner,[1] David E.R. Sutherland,[1] David L. Dunn,[1] John S. Najarian,[1] Angelika C. Gruessner.[1] *[1]Surgery, University of Minnesota, Minneapolis, MN.*

Purpose: Long-term outcome after Pa Tx is not well established. We retrospectively analyzed all Txs with >10 yrs PA graft function to identify common denominators. Methods/Patients: Between 7/78 and 10/90, we performed 379 Pa Txs Of those, 81 (21.4%) had Pa graft function for >10 years: 39 (48%) were simultaneous Pa/kidney (Kd) Txs (SPK), 23 (28%) Pa Txs alone (PTA) and 19 (24%) Pa after Kd Txs (PAK) According to the recipient category, Pa graft function >10 yrs was highest for SPK, followed by PAK and PTA. Donor source was cadaveric (CAD) in 63 (78%) and living related (LR) in 18 (12%) Txs. There were 71 (88%) primary and 10 (12%) reTxs. Bladder drainage (BD) was more common (62;77%) than enteric drainage (ED) (16; 20%). BD was primarily used in SPK and PAK, ED in PTA. At 5- and 10-yrs post Tx, these laboratory parameters were analyzed: serum glucose, hemoglobin (Hgb) A1C, creatinine, cholesterol, triglycerides, and urinary amylase (UA). Results: *Patient Survival*: There were 7 deaths (SPK 1, PAK 3, PTA 3), all with functioning grafts. Causes included malignancy (2), trauma (1), suicide (1), and others (3). *Graft Survival* For patients (pts) with a functioning graft at 10 yrs, 15-yr graft survival was 81% for SPK, 74% for PTA, and 70% for PAK. Most Pa graft losses were due to chronic rejection (6/7): 2 pts were successfully retransplanted. For SPK pts, 9 Kd graft losses occurred (8/9 to chronic rejection): 7 were Kd reTx'ed. No significant differences were noted between 5 and 10 yrs for serum glucose, Hgb A1C, creatinine, cholesterol, and triglycerides; only UA levels significantly decreased over time. Of note, 30% of pts developed squamous/basal cell skin cancers >10 yrs, 10% developed other malignancies including astrocytoma, breast Ca, parotis Ca, and seminoma. Conclusions: 1)>10 yr Pa graft function was more common for SPK vs PAK and PTA, primary Txs vs reTxs, LR vs CAD Txs, and initial UA >3000 vs ≤3000 U/hr. 2) Most Pa graft failures >10 yrs were due to death with functioning graft or chronic rejection. 3) 15-yr graft survival rates ranged between 70% (PAK) and 81% (SPK). 4) Serum glucose, Hgb A1C, creatinine, cholesterol and triglyceride levels were not different at 5 vs 10 yrs and most pts have remained on cyclosporine/imuran maintenace therapy. 5) In pts with >10-yr Pa graft function, excellent graft and patient survival rates as well as excellent metabolic long-term function can be obtained.

Abstract# 98

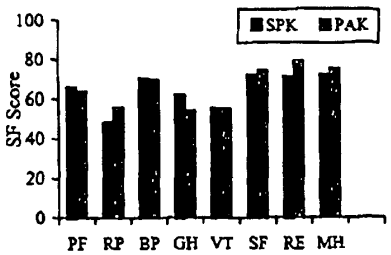
PANCREAS AFTER KIDNEY (PAK) VS. SIMULTANEOUS PANCREAS KIDNEY (SPK) TRANSPLANTS: A COMPARISON OF WAITING TIMES, COST, AND QUALITY OF LIFE. Abhi Humar,[1] Raja Kandaswamy,[1] Thiagarjan Ramcharan,[1] Steven Paraskevas,[1] Rainer W. Gruessner,[1] Angelika Gruessner,[1] David E.R. Sutherland.[1] *[1]Surgery, University of Minnesota, Minneapolis, MN.*

Background: Previously we have shown that PAK tx had fewer surgical complications vs. SPK tx This analysis was performed to determine differences in waiting times, costs, and quality of life.

Results: Between 1994-2000, 205 cadaver PAKs and 193 cadaver SPKs were performed. Demographics are shown. The mean waiting time for a PAK tx (time from being listed for a pancreas to the actual tx) was 167 days vs. 244 days for SPK tx (p=0.01). The waiting time for the kidney tx for PAK recipients was difficult to estimate, since most had received a living donor (LD) kidney. The mean hospital stay was 10.7 days for PAK vs. 17.5 days for SPK recipients (p=0 0001). The mean length of stay after the LD kidney tx was 6.2 days Readmissions to hospital during the first 3 months posttx were 0.9 readmissions for SPK vs. 1.2 for PAK recipients (this included the 3 months after the kidney tx). The average cost of the PAK tx (including the LD kidney) was $63,542; for SPK, $54,160. Using the SF-36 questionnaire, no significant differences in quality of life were noted between the groups.

Conclusion: A PAK transplant is a viable option for uremic diabetics. Decreased waiting time is a significant advantage. Performing a sequential vs. simultaneous procedure does not significantly increase total length of hospitalization or costs. Quality of life is similar.

	SPK	PAK	p
Total #	n=193	n=205	—
Donor age (kidney) (yrs)	32.8	40.9	0.01
%Cadaver(kidney)	100%	28%	0.01
Donor age (pancreas)	25.6	25.8	ns
%Retransplants (pancreas)	7.3%	17.3%	0.04



CONCURRENT SESSION 11:
CONTROL OF ALLOREACTIVE T CELLS

Abstract# 99

CORRELATION BETWEEN TH1 AUTOIMMUNE RESPONSES TO CARDIAC MYOSIN AND CHRONIC REJECTION OF HEART ALLOGRAFTS. Victor Dong,[2] Koji Kishimoto,[2] Hillary Rolls,[1] Masayuki Sho,[2] Mohamed H. Sayegh,[2] Gilles Benichou,[1] Eugenia V. Fedoseyeva.[1] *[1]Shepens Eye Research Inst, Harvard Medical School, Boston, MA; [2]Brigham and Women's Hospital, Harvard Medical School, Boston, MA.*

We have previously demonstrated that in fully mismatched C57BL/6 (H-2b) - BALB/ c (H-2d) donor-recipient combination, potent autoimmunity to cardiac myosin (CM) is induced following heart transplantation. Here, we investigated whether transplantation-induced autoimmune response may contribute to the development of chronic rejection in mice. To address this question, recipient BALB/c mice were treated with MR1 (anti-CD40L) mAb with one of the following regimens: 1) single i.p injection of 0.25 mg/mouse at day 0, 2) multiple i.p. injections of 0.25mg/mouse at day 0, 2, 4 and 6 post-transplant, and 3) co-administration of a single dose of MR1 mAb with donor-specific splenocytes, 106 cells/mouse (MR1/DST). The frequencies of IFNg (Th1 cytokine)-producing T cells specific to donor MHC antigens and to CM autoantigen were evaluated by ELISPOT. In all groups, mice that developed chronic rejection (100% disease, average vessel score 3.3 ± 0.6) exhibited consistent Th1 type immune responses to CM in the absence of Th1 direct alloreactivity

Additionally, STAT4KO recipients deficient in Th1 immunity, unlike their wild type counterparts, BALB/c, did not develop post-transplant immune response to CM These data were consistent with our previous observations showing that CM autoimmunity is mediated by IFNg-producing Th1 type T cells Costimulatory blockade (CTLA4-Ig treatment) resulted in significant prolongation of cardiac allograft survival in BALB/c mice, STAT4KO (Th1 cytokine deficiency) and STAT6 (Th2 cytokine deficiency). However, while CTLA4-Ig-tolerized wild type BALB/c and STAT6 mice developed chronic rejection at late periods after heart allotransplantation, no or minimal chronic rejection was observed in STAT4KO-CTLA4-Ig-tolerized mice. This suggests that the initial lack of tissue-specific autoimmunity in STAT4KO mice may have contributed to the prevention of chronic rejection in these mice. Taken together, our data show a strong correlation between post-transplant autoimmune response to CM and chronic cardiac allograft rejection.

Abstract# 100

MOUSE CD4+ AND CD8+ T CELLS DISPLAY DIFFERENCES IN ACTIVATION AFTER DIRECT STIMULATION BY ALLOGENEIC VASCULAR ENDOTHELIUM. Daniel Kreisel,[1] Alexander S. Krupnick,[1] Wilson Y. Szeto,[1] Sicco H. Popma,[1] Alyssa M. Krasinskas,[2] Bruce R. Rosengard.[1] [1]*Surgery, University of Pennsylvania, Philadelphia, PA;* [2]*Pathology and Laboratory Medicine, University of Pennsylvania, Philadelphia, PA.*

Purpose. Despite apparent advantages of transgenic technology and the availability of murine in vivo models of allograft rejection only few studies have examined whether mouse vascular endothelium can activate alloreactive T cells. This has been partially due to difficulties in growing mouse vascular endothelium. We have recently described a method to culture pure mouse vascular endothelium from thoracic aorta. The purpose of this study was to examine the capacity of vascular endothelium to trigger CD4+ and CD8+ direct allorecognition. Methods: Purified CFSE-labeled CD4+ or CD8+ T cells of CBA/J (H-2Kk) origin were co-cultured with fully allogeneic C57BL/6 (H-2Kb) vascular endothelium free of professional APCs for various periods of time. Results: Mouse vascular endothelium expresses low levels of MHC class I and moderate levels of CD80 at a resting state. Activation with IFN-gamma leads to upregulation of MHC class I, induction of MHC class II and no changes in CD80 expression. Neither resting nor activated endothelium expresses CD40 or CD86. CD8+ T cells proliferate vigorously after 5 and 7 days of co-culture with resting (33% at 5 days, 51% at 7 days) or activated endothelium (55%, 56%), upregulate CD25 and CD69 and differentiate to a Tc1 phenotype as evidenced by production of IFN-gamma and IL-2 and lack of IL-4 production. In addition, proliferating CD8+ T cells acquire FasL expression as well as intracellular perforin stores. Alternatively, CD4+ T cells show only minimal proliferation after 5 and 7 days of co-culture with activated endothelium (7%, 10%). This hyporesponsiveness can be overcome when professional antigen presenting cells are added to the cocultures on day 0. Conclusion: Mouse vascular endothelial cells appear to be potent at triggering CD8+ direct allorecognition, while having a far diminished capacity in activating alloreactive CD4+ T cells directly. Studies examining the underlying mechanisms for the hyporesponsiveness of alloreactive CD4+ T cells to activated vascular endothelium despite the presence of signal 1 (MHC class II) and signal 2 (CD80) are ongoing. In addition, the capacity of mouse vascular endothelium to trigger allograft rejection in vivo is being studied.

Abstract# 101

DIFFERENTIAL LOCALIZATION OF INTERLEUKIN-2- AND -15 RECEPTOR CHAINS IN MEMBRANE RAFTS OF HUMAN T CELLS. Jens Goebel,[1] Kathy Forrest,[1] Lorri Morford,[2] Thomas L. Roszman.[2] [1]*Pediatrics, University of Kentucky, Lexington, KY;* [2]*Immunology and Microbiology, University of Kentucky, Lexington, KY.*

Lipid microdomains ("rafts") are increasingly recognized as critical sites for the initiation of signal transduction into T lymphocytes and other cells. However, current knowledge about the role of rafts in T cells is largely limited to antigen receptor (TCR) and costimulatory signaling. We therefore examined whether rafts might also be involved in signal transduction through cytokine receptors (Rs). Human T lymphocytes were isolated and treated with low-dose phytohemagglutinin to induce the expression of the high-affinity interleukin-2 (IL-2) R. After their separation into cytoplasmic, membrane and raft fractions by mechanical disruption and buoyant density centrifugation, Western blots of equal amounts of protein from each fraction demonstrated at best scarce amounts of IL-2R α- or -γ-chains in rafts. In contrast, the IL-2R β-chain was found to be enriched in these domains. The α-chain of the IL-15R, which shares its β- and γ-chains with the IL-2R, was also absent from rafts. These findings were reproduced by confocal microscopy, as the examination of human T cells with cholera toxin B (CTB)-labeled rafts revealed consistent co-localization of CTB with the IL-2/15R β-chain, but not with the α- or γ-chains. Lastly, anti-phosphotyrosine blots demonstrated that in human T cells, raft disruption by pretreatment with methyl-β-cyclodextrin attenuated the IL-2-induced tyrosine phophorylation of several proteins including STAT5, suggesting functional relevance of the demonstrated localization of the IL-2/15R β-chain within rafts. These studies provide novel information about the relationship between cytokine Rs and rafts in human T cells and demonstrate for the first time that individual chains of such Rs can differ regarding their selective localization in these membrane domains. This information also extends our understanding of cytokine R signaling as a key mechanism in alloimmunity and identifies rafts, or selected cytokine R chains enriched in them, as potential targets of novel immunosuppressive strategies.

Abstract# 102

ISLET GRAFT SURVIVAL IS INCREASED IN MICE TREATED WITH AN IL-15 MUTANT/Fc PROTEIN THROUGH THE CONTROL OF CD8⁺ T-CELL PROLIFERATION. Sylvie Ferrari Lacraz,[1] Xin Xiao Zheng,[1] Alberto Sanchez-Fueyo,[1] Wlodzimierz Maslinski,[1] Terry B. Strom.[1] [1]*Division of Immunology, Beth Israel Deaconess Medical Center, Boston, MA.*

Introduction: As costimulation blockade does not always induce permanent engraftment or even tolerance, we and others have suggested that "escape" from costimulation blockade might represent a CD8⁺ T-cell dependent process. Insofar, as recent data have indicated an important role for IL-15/IL-15R in the activation and survival of CD8⁺ T-cells, we hypothesize that CD4⁺-independent CD8⁺ T-cell mediated rejection is an IL-15/IL-15R dependent process. To study this hypothesis, we have utilized a treatment with an IL-15 mutant/Fcγ2a protein, which acts in part as a long lived, high affinity IL-15 receptor site specific antagonist and has the potential for destroying IL-15R⁺ leukocytes. Data: In a murine islet allograft model with recipients rendered diabetic by injection of streptozotocin, we have observed that the IL-15 mutant/Fcγ2a protein protected allografts from predominantly CD8⁺ T-cell infiltration and decreased CTL-related gene expression. We were thus interested to study the effect of this IL-15 mutant/Fcγ2a protein on CD4⁺-independent alloreactive CD8⁺ T-cells in a murine islet allograft model in CD4KO recipients. All allograft from Balb/c ($H2^d$) donors to C57Bl/6 ($H2^b$) CD4KO recipients (n=6) were accepted permanently when mice were treated with IL-15 mutant/Fcγ2a, whereas 50% of untreated recipients (n=10) rejected their graft with a mean survival time of 35 days. Analysis revealed that IL-15 mutant/Fcγ2a protein blocked a CD8⁺ T-cell rejection process by blunting the proliferation of alloreactive CD8⁺ T-cells *in vivo*. Interpretation: These findings suggest that treatment with IL-15 mutant/Fcγ2a protein may be an additional strategy to prevent resistant CD8⁺ T-cell driven rejection.

Abstract# 103

T CELL HOMEOSTATIC PROLIFERATION - FUNCTIONAL GAIN AND ROLE OF COSTIMULATION. Hrefna Gudmundsdottir, Laurence A. Turka.

Previous reports (Ernst, 1999) have shown that T cells proliferate when transferred into lymphopenic hosts (homeostatic proliferation) and that this requires contact with MHC-self peptide. We wanted to determine if; 1) signaling through B7-CD28 was important in the homeostatic proliferation of naive antigen (Ag) specific CD4 T cells, and 2) if T cells acquire function comparable to memory cells as a result of homeostatic proliferation.

In order to avoid the expression of endogenous TCR and generation of memory T cells to unknown peptides, we used T cells from recombination deficient DO11.10 mice (DO11/RD), that express a TCR transgene which recognizes OVA peptide. In order to determine the role of B7/CD28 signaling on proliferation the cells were labeled with CFSE, transferred to congeneic RAG k.o. mice, and treated with CTLA4Ig or control Ig. For functional studies T cells were harvested 4 weeks after adoptive transfer and function compared to freshly isolated naive or memory DO11.10/RD cells, generated by immunizing the mice with OVA peptide 4 weeks earlier.

Three to five weeks after adoptive transfer we identified two populations of T cells based on CFSE brightness. One that had divided 1-3 times and another one that had divided multiple (>8) times. Blockade of B7/CD28 interactions strongly suppressed the accumulation of multiply divided cells (74.5 ± 11.0% to 9.7 ± 4.3% ($p<0.0005$), but had no effect on the minimally divided population. Thus B7 signals are required for the expansion of multiply dividing cells during homeostatic proliferation.

By 4-6 weeks, homeostatic proliferation was accompanied by the development of a memory surface phenotype (CD45RBlo, CD44hi and CD62lo). Most importantly, homeostatic proliferation was associated with the development of functional responses characteristic of memory cells, including proliferation to low doses of peptide and B7-independence of this response. Finally, the capacity of these cells to make IL-2 and IFNγ was greater than that of naive cells ($p<0.02$)(table, n=6-10) and approached that of true memory cells.

In conclusion our data suggests that 1) signals generated by B7 are important for maximal proliferation during lymphopenia and 2) the cells acquire phenotypic and functional characteristics comparable to that of memory cells despite the absence of Ag specific stimulation.

(%)	Memory	Homeostatic Proliferation	Fresh Naive
IL-2	33 ± 6	16 ±14	0.3 ± 0.3
IFNγ	15 ± 11	7 ± 5	1 ± 1

Abstract# 104

CD40: TURNING OFF "OFF SIGNALS" TO REGULATE GENE TRANSCRIPTION. Michael Melter,[1,3] Peter H. Lapchak,[1,3] Soumitro Pal,[2,3] Christopher Geehan,[1,3] Debabrata Mukhopadhyay,[2,3] David M. Briscoe.[1,3] *[1]Division of Nephrology, Children's Hospital, Boston, MA; [2]Department of Pathology, Beth Israel Deaconess Medical Center, Boston, MA; [3]Harvard Medical School, Boston, MA.*

CD40 is known to regulate many genes via the activation of NF-kB and/or AP-1 transcription factors. We recently demonstrated that CD40 ligation results in induced expression of vascular endothelial growth factor (VEGF) predominantly via transcriptional mechanisms. We have now identified that this activation is restricted to a 68bp spanning sequence of the VEGF promoter conferring inducibility by CD40. A computerized search of genomic DNA libraries and MatInspector revealed that the identified sequence does not contain any putative binding sites recognized by known CD40 regulatory proteins. However, we defined a 43bp sequence within this region that has a 97% homology to a particular cytosine-guanine rich region (CpG island) that is known to bind the abundant chromosomal protein MeCP2. MeCP2 is a potent transcriptional repressor with high specific affinity for methylated CpG. We next addressed whether this CpG binding site was functional for CD40-dependent transcription. By electrophoretic mobility shift assays, we found that the unmethylated 43 bp DNA sequence failed to form a complex with recombinant human MeCP2 protein or nuclear extracts from endothelial cells (EC). In contrast, both MeCP2 protein as well as nuclear extracts from EC formed a complex with methylated 43bp DNA. Thus, this site does function as an "off signal" for gene transcription. Next, we compared the ability of nuclear extracts from untreated or sCD40L-treated (30 mins to 2 hrs) EC to interact with this region. Interestingly, treatment of endothelial cells with sCD40L resulted in a significant reduction of the formed complex (up to 70%), suggesting that CD40 signals decrease the binding of MeCP2 to this region of the VEGF promoter. We are currently assessing whether this process involves NF- kB, AP-1 or a novel transactivating mechanism(s). Taken together, these findings suggest that in the quiescent state, binding of MeCP2 to a CpG island maintains an "off signal" for VEGF gene transcription. Activation of CD40 decreases binding of MeCP2 to the CpG island, thus removing this repression of gene transcription. This is the first demonstration that CD40 signals regulate genes by altering repressors of activation. We believe that this novel mechanism may be involved in CD40-dependent activation of multiple genes involved in the alloimmune inflammatory reaction.

Abstract# 105

COSTIMULATION PATHWAYS IN THE DIFFERENTIATION OF ALLOREACTIVE CYTOTOXIC T CELLS. Yuan Zhai, Feng Gao, Ronald W. Busuttil, Jerzy W. Kupiec-Weglinski. *[1]Surgery, The Dumont-UCLA Transplant Ctr., Los Angeles, CA.*

Background: The role of costimulation signals in CD4 T cell activation/differentiation has been extensively studied. Relatively little is known on the role of costimulation pathways in the generation of alloreactive CD8 T cells. In this study, we took advantage of mAb-based blockade of CD28 vs. CD40L in wild-type (WT) graft recipients, as well as CD28 and CD40L knock-out (KO) hosts, to examine putative roles of these two T cell costimulation molecules in the differentiation of primary alloreactive CTLs in vivo.

Methods: Balb/c skin grafts were placed onto WT or CD40L KO (B6/129)/CD28 KO (C57BL6) mice. Spleens were harvested at day 10, and FACS-based analysis was performed to examine CTL differentiation (CD8+CD44+CD62L-) in vivo.

Results: The induction of primary CTLs by allogeneic skin grafts triggered the generation of CD44+CD62L- cells in CD8+ spleen, but not lymph node, cell population of the recipient mice (<5% in naive vs. 40% in engrafted hosts). In vivo detection of CTLs was correlated with in vitro CTL recall assay (cytolysis of Balb/c ConA blasts after 6-day stimulation) in which only splenocytes from skin graft recipients were able to efficiently lyse donor target cells. The induction of allogeneic CTLs was independent of CD4 T cell help, as mAb-induced specific CD4 T cell depletion failed to affect the generation of CD8+CD44+CD62L- effector population in skin-grafted mice. The role of costimulation pathways in the induction of allogeneic CTLs was then addressed by analyzing the effects of CTLA4-Ig vs. CD40L mAb (MR1) treatment during the 10-day induction phase in WT mice. Blocking any of the two molecules inhibited the generation of the CTL effectors. Indeed, no allogeneic CTLs were detectable in either CD28 KO or CD40L KO skin grafted mice. The effects of costimulation blockade were also examined during the induction of CTLs from primed CD8 T cells in vitro. In contrast to primary CTLs, CTLA4-Ig completely blocked the generation of CTLs from allo-primed T cells; CD40L-targeted therapy reduced CTL generation by ca. 40%.

Conclusion: Allogeneic APCs may directly stimulate CD8+ T cells without CD4+ T cell help. Both CD28 and CD40L signaling pathways are required for primary allogeneic CD8+ CTL differentiation. However, primed T cells depend on CD28 rather than CD40L to differentiate into mature CTL effector cells. Experiments determining relationship between the two costimulation pathways in CD8+ T cell activation, parallel or sequencial, are currently underway.

Abstract# 106

FASL IS REQUIRED FOR COSTIMULATION INDEPENDENT IFN-γSECRETION AND CYTOTOXICITY. Joel Trambley,[1] Matthew A. Williams,[1] Angello Lin,[1] Thomas C. Pearson,[1] Christian P. Larsen.[1] *[1]Surgery, Emory University School of Medicine, Atlanta, GA.*

We have shown that combined CD40/CD28 blockade induces longer allograft survival in fasL deficient C57BL/6.gld (B6.gld) recipients than in C57BL/6(B6) mice. As fasL has roles in apoptosis, cytotoxicity, and costimulation, we sought to define the function of fasL in costimulation blockade resistant alloimmunity.

To determine if fasL was an effector molecule in graft rejection, Balb/c skin grafts were harvested from costimulation blockade(500µg each anti-CD40L (MR1) and CTLA4-Ig days 0,2,4,6) treated B6 and B6.gld recipients 19 days post transplant. Immunohistochemical staining of grafts from either B6 or B6.gld mice showed few CD4+ cells. In contrast, grafts from B6 mice showed more CD8+ cells than those taken from B6.gld mice, suggesting that fasL is important prior to effector function.

To assess the role of fasL in early T cell activation, B6 and B6.gld T cells were stained with CFSE and adoptively transferred into untreated or costimulation blockade treated lethally irradiated Balb/c recipients. While costimulation blockade decreased the number of divisions achieved by both B6 and B6.gld T cells, no significant differences were seen within the treatment groups, suggesting that early T cell activation is comparable in B6 and B6.gld mice.

We next used the ELISpot assay to determine the number of donor specific IFN-γ secreting T cells in B6 and B6 gld mice during costimulation blockade resistant rejection. T cells from the spleens of untreated B6 and B6.gld recipients of Balb/c skin grafts showed similar numbers of donor specific IFN-γ producing cells on day 26 post transplant. While treated B6 mice also demonstrated an easily detectable, though decreased (~40% of untreated) response, almost no IFN-γ producing T cells were found in treated B6.gld mice (<5% or untreated). B6.gld mice treated with costimulation blockade also showed significantly lower ex vivo cytolytic activity day 26 (<5% specific lysis, 100:1 E:T) than treated B6 mice (15%). On day 19 post transplant, however, neither B6 nor B6.gld recipients showed detectable levels of ex vivo lytic activity when treated with costimulation blockade.

Overall, these data support a costimulatory role for fasL in the alloresponse of mice treated with combined CD28/CD40 blockade. Further, they suggest that fasL is not important in early activation events, but plays a role in the persistance of T cell actiation or the reactivation of T cells at the time of rejection in costimulation blockade treated allograft recipients.

Abstract# 107

RECOMBINANT DIMERIC MHC CLASS I MOLECULES STAIN IN VITRO GENERATED ALLOREACTIVE T-CELLS AND SHOW A CRITICAL ROLE FOR CD8. Kyoung-Ae Yoo-Ott, Birgit Fricke, Judith Steude, Nicholas Zavazava. *[1]Immunology, University of Kiel, Kiel, Germany.*

The aim of this study was examine whether MHC class I molecules can be utilized to visualize alloreactive T cells. The extracellular domains of a rat MHC class I molecule (RT1.Al) were amplified by polymerase chain reaction and cloned to the Fc-region of an IgG2c molecule. The construct was ligated into an expression vector for expression in mammalian cells. A rat lymphoblastoid B-cell line was transfected by electroporation. A divalent rat MHC class I fusion molecule (RT1.Al-Fc) comprising the extracellular domains of the rat RT1.Al and the rat IgG2c Fc-region was secreted at at >15 µg/ml into the supernatant. The secreted class I dimeric molecules were peptide loaded and had the expected molecular mass of 160 kDa. The single chain had a molecular mass of about 67 KDa. The dimer was biotynilated and used to stain alloreactive T-cells generated in vitro. Anti-RT1.Aa CTL and anti-RT1.Al CTL were generated in vitro. These cells were >99 % CD8 and cytotoxic towards PHA-stimulated blast-cells. >85 % of the anti RT1.Al CTL stained to the dimer but not the anti-RT1.Aa CTL. Binding of the dimer to the CTL was augmented by an anti-CD8a antibody, but abrogated by an anti-CD8b antibody. Conversely, staining of the CD8 molecule on the surface of these cells was strongly abrogated by pre-incubating the cells with the dimer, clearly implicating CD8 in the binding and recognition of MHC dimers and differential roles for the CD8a and CD8b chains. Regulation of dimer binding to T cells was temperature dependent.Confocal microscopy of the CTL indicated co-capping of both CD8 and the dimer on the cell surface of the CTL. Staining of these cells with the dimer blocked cytolysis of target cells by the CTL and induced apoptosis by Fas/FasL interaction. Thus, these data indicate an important role for MHC dimers in studying alloreactive CTL and also imply a critical role for the heterodimeric CD8 chains.

CONCURRENT SESSION 12:
RISK ANALYSIS IN RENAL TRANSPLANTATION

Abstract# 108

A STUDY OF CYTOKINE SECRETION: EFFECT OF GENOTYPIC VARIATIONS IN THE TNF-A, IL-10 AND IFN-G GENES ON THE IN-VITRO CYTOKINE PRODUCTION. RELEVANCE FOR LATE-ONSET PTLD. Athanasios Vergopoulos,[1] Nina Babel,[2] Ian Hutchinson,[3] Conny Hoeflich,[1] Hans Dieter Volk,[1] Petra Reinke.[2] [1]*Department of Medical Immunology, Charite-Campus Mitte;* [2]*Department of Nephrology, Charite-Campus Virchow, Berlin, Germany;* [3]*School of biological sciences, University of Manchester, United Kingdom.*

PTLD is a devastating complication of solid organ transplantation. Following transplantation, the host:EBV equilibrium is shifted in favour of the virus. Differential inhibition by the immunosuppressive drugs may be one factor, a cytokine environment favouring B cell growth may be another. Disturbances in immunoregulatory balance, be they genetically determined, acquired, and/or iatrogenic, may serve as important contributing factors in the development of PTLD. With the goal of identifying such factors, we compared patterns of LPS/ConA-induced cytokine production in whole blood of transplant patients (Tx-pts) with late-onset PTLD, Tx-pts without any complications, MGUS pts, and healthy individuals. All pts were under comparable immunosuppression. In order to address whether genetic polymorphisms might influence the level of expression we also genotyped our subjects for polymorphisms in cytokine genes making use of RFLP and ARMS methodologies. Healthy controls had higher levels of LPS-induced TNF-a and ConA-induced IL-10 and IFN-g than Tx-pts. PTLD pts exhibited higher LPS/ConA-inducible TNF-a and IL-10 as well as lower ConA-inducible IFN-g producing capacity in comparison with Tx-pts without complications (n.s.). We observed that the G allele for the -308 TNF-a polymorphism, the A allele for the -1082 IL-10 polymorphism, and the T allele for the +874 IFN-g polymorphism were more frequent in the PTLD group compared to the other Tx groups. We demonstrated, that in healthy individuals the GG (-308) TNF-a, the AA (-1082) IL-10 and the AA (+874) IFN-g genotypes are associated with significantly higher in-vitro TNF-a, IL-10 and IFN-g production, respectively, compared to other genotypes (p less than 0,05). In summary, we provide evidence contradicting the hitherto described association between cytokine gene polymorphisms and the level of cytokine production in vitro The discrepancy with previous findings may reflect differences in the nature of the stimulated cell populations. We conclude that cytokine gene polymorphisms may be responsible for the particular cytokine phenotype in our PTLD pts. Further studies will be required to establish the effect of gene polymorphisms on cytokine gene regulation.

Abstract# 109

IMPACT OF DE NOVO PRODUCED ALLOREACTIVE CYTOTOXIC ANTIBODIES AFTER KIDNEY TRANSPLANTATION. Marco Miozzari,[1] Samuel Henz,[1] Margrith Disler,[2] Daniela Garzoni,[1] Daniel Hertner,[1] Markus Fopp,[2] Rudolf P. Wuthrich.[1] [1]*Division of Nephrology, Kantonsspital;* [2]*Regional Blood Bank Center, St. Gallen, Switzerland.*

The de novo occurrence of alloantibodies (panel reactive antibodies, PRA) in the early posttransplant period could trigger acute and chronic rejection of kidney allografts and compromise graft survival. To assess the impact of alloantibody production on graft survival we analyzed 118 sequential kidney transplant recipients with no history of previous PRA-positivity for the emergence of PRA within the first two month after transplantation. The transplantations were performed between 1990 and 2000, with ABO-compatibility and a negative pretransplant crossmatch (B- and T-lymphocytes). The impact of de novo appearing PRA-positivity on the rejection rate, on the course of the serum creatinine, and on graft survival was assessed by multivariate Cox regression, by methods for repeated data analysis, and by Kaplan-Meier survival analysis In 13 (11%) of the 118 analyzed cases a PRA-response could be detected. The overall 5-year graft-survival for all 118 patients was 83%, but it was only 68% in PRA-positive versus 86% in the PRA-negative patients (p=0.001). After multivariate adjustment the probability of acute rejection (within one year posttransplant) was found to be significantly higher in the PRA-positive recipients (Hazard Ratio HR = 4.4, 95% CI 1.8-11.0, p=0.001). The risk of graft failure was also significantly higher in PRA-positive patients even after adjustment for acute rejections (HR = 4.7, 95 % CI 1.6-14.4, p=0.007). De novo formation of PRA and the presence of rejections in the first year after transplantation were both identified as independent predictors of the combined outcome graft failure or death (HR = 2.7, 95% CI 1.1-6.8, p=0.03). PRA-positive recipients also had higher serum creatinine levels than PRA-negative recipients, with an average difference of 124 µmol/l (95% CI 35-216 µmol/l, p=0.006). We conclude that there is a strong association between the de novo formation of PRA in the early posttransplant period and accelerated graft failure. The beneficial effect of a prompt identification of PRA-positive patients for the purpose of a more intense immunosuppressive regimen must be analyzed prospectively in a larger number of patients.

Abstract# 110

TNFβ GENE POLYMORPHISMS PREDICT URINARY TRACT INFECTIONS FOLLOWING RENAL TRANSPLANTATION. Pam M. Kimball,[1] Cecil Rhodes.[1] [1]*Surgery, MCV, Richmond, VA.*

NcoI digestion of the TNFb gene reveals 2 polymorphisms, termed high and low, that regulate cytokine synthesis and secretion. It has been speculated that the TNFb low gene may predispose individuals to infection. If true, this may be useful to predict infection susceptibility prior to renal transplantation (RTX). We determined the frequency of the TNFb low gene in 112 ESRD patients awaiting transplantation and in 25 consecutive RTX recipients. In addition, we correlated the presence of the TNFb low gene with the incidence of urinary tract infections (UTI) and rejection within 60 days after RTX. RTX immunosuppression consisted of MMF and prednisone with concommittant CsA (n=12) or Tacrolimus (n=13). UTIs and rejection after RTX were diagnosed microbiologically and histologically, respectively. Genomic DNA was extracted by chloroform/ethanol from peripheral leukocytes and amplified using TNFb specific primers. Amplified product was digested with NcoI enzyme. Digest products were separated by agarose gel electrophoresis and visualized with ethidium bromide. The entire population of patients was comprised of 63% African Americans (AA) and 37% Caucasians (Cau). TNFb low gene was present in 77.7% of the total population. However, TNFb low gene distribution was racially disparate and found among 83% AA versus 63% Cau (p=0.02). Following RTX, UTI's were not demonstrated among any AA or Cau patient with TNFb high gene. In contrast, the incidence of UTIs was 50% among patients with TNFb low gene and, thus, is greater than among patients with TNFb high gene (50% vs 0%, p<0.01). More African Americans than Caucasians experienced UTIs (p< 0.05). The frequency of TNFb low and UTI were equivalent in CsA versus Tacrolimus therapy (p=ns). Infections consisted of E. coli, enterococcus, lactobacillus and yeast. All infections were resolved medically. The presence of TNFb low gene was not associated with the incidence or severity of rejection. The incidence of rejection was 21% for TNFb low and 18% for TNFb high (p=ns). All rejections were mild and reversed with steroids. Actuarial 1 yr graft survival was statistically equivalent among TNFb low versus TNFb high groups (100% vs 90%, p=ns). In summary, the TNFb low gene was strongly associated with UTI following RTX. In contrast, the TNFb high gene was strongly predictive of freedom from UTI in the early postoperative period. Neither TNFb low or high genes correlated with rejection or 1 yr graft survival.

Abstract# 111

ROLE OF ANTI-β2 GLYCOPROTEIN 1 ANTIBODIES IN ESRD PATIENTS WITH ANTIPHOSPHOLIPID ANTIBODY SYNDROME. Smita Vaidya,[1] Todd Y. Cooper,[1] John A. Daller,[2] Kristene K. Gugliuzza.[2] [1]*Pathology, University of Texas Medical Branch, Galveston, TX;* [2]*Surgery, University of Texas Medical Branch, Galveston, TX.*

Introduction: ESRD patients with antiphospholipid antibody syndrome (APAS) are at high risk for the development of post-transplant renal thrombosis. Positive titer of anticardiolipin antibodies (ACA) is considered a major characteristics of APAS. However, several studies have suggested that antibodies to β2 glycoprotein (β-GP1) may have stronger association with APAS than ACA. In this study we have determined association of antibody to β2 GP1 in ESRD patients with APAS.

Method. One hundred and seventy eight patients currently listed at our center for cadaver kidney transplants, were tested for ACA and anti-β2 GP1 antibodies by ELISA. Twenty-five of these patients had clotting disorders as determined by frequent A-V shunt thrombosis (N=15), microrenal angiopathy (N=4), frequent abortion and lupus (N=3), thrombocytopenia (N=3).

Results: Eleven of 178 (6%) had positive titer of β2 GP1 antibody and 10 of these 11 (91%) had APAS. In contrast, 40 out of 178 (23%) had positive titers of ACA and 21 (53%) had APAS. Of these 21 APAS patients with ACA titers, 6 patients were also positive for anti-β2 GP1. Four patients with various clotting disorders but no evidence of ACA in their serum, had extremely high titers of anti-β2 GP1 antibodies. Of the 25 patients with APAS, 15 (60%) patients had only ACA, 4 (16%) had only β2 GP1 antibodies and 6 (24%) had both ACA and β2 GP1 antibodies. The sensitivity and specificity of ACA to predict APAS are 84% and 89% respectively. In contrast, the sensitivity and specificity of β2 GP1 to predict APAS are 40% and 99%.

Conclusion: APAS in the ESRD patients should be characterized by not only the presence of ACA but also the presence of β2 GP1 antibodies in the association with history of clotting disorder.

Abstract# 112

FASTING AND POST-METHIONINE LOAD HOMOCYSTEINE ARE ASSOCIATED WITH PROGRESSION OF CAROTID ATHEROSCLEROSIS IN RENAL TRANSPLANT RECIPIENTS.

Andrew A. House,[1] David Freeman,[1,2] Anthony M. Jevnikar,[1,2] Norman Muirhead,[1] David J. Hollomby,[1] Kelly B. Zarnke,[1] J. David Spence.[1,2]
[1]*Medicine, University of Western Ontario & LHSC, London, ON, Canada;* [2]*Robarts' Research Institute, University of Western Ontario & LHSC, London, ON, Canada.*

Homocysteine (Hcy) is an intermediary amino acid which has a strong association with cardiovascular morbidity and mortality. Recent evidence supports such an association in renal transplant recipients (RTR), leading some to conclude that Hcy is prothrombotic and/or atherogenic. To support the latter, we measured Framingham and other traditional risk factors, fasting (fHcy) and post-methionine load (PML) Hcy in 24 non-diabetic RTR with stable renal function, and measured the progression of carotid atherosclerosis over one year using Doppler ultrasound. Spearman's rho correlation coefficients were generated for all risk factors and rate of carotid plaque progression, baseline fHcy and PML Hcy were compared with 1 year values using paired t-tests.

Results: 23 RTR (12F/11M) completed the study, with baseline mean (SD) age 60.6 (9.5) yrs, CrCl 70.9 (23.2) mL/min, total plaque area 0.77 (0.66) cm^2. Baseline and 1-yr fHcy were 11.6 (4.1) and 11.7 (4.0) µmol/L (p=0.75), while PML Hcy were 25.0 (7.4) and 28.5 (6.9) µmol/L (p=0.001). As expected, Hcy was highly correlated with both Cr and folate status. The rate of progression was 0.10 (0.37) cm^2/yr. The strongest correlates with plaque progression were average fHcy (R=0.44, p=0.04) and PML Hcy (R=0.43, p=0.04). None of the traditional risk factors had a significant positive correlation with plaque progression.

Conclusions: Both fHcy and PML Hcy are strongly associated with the progression of carotid atherosclerotic plaque in stable, non-diabetic renal transplant recipients, suggesting that Hcy is indeed atherogenic. Further support for a causal relationship will be sought in a randomized trial of folate-based vitamin therapy and this experimental model.

Abstract# 113

CLINICAL CHARACTERISTICS OF SIROLIMUS ASSOCIATED PNEUMONITIS IN RENAL TRANSPLANT PATIENTS.

Emmanuel Morelon,[1] Marc Stern,[2] Marie-france Mamzer-Bruneel,[1] Marie-Noelle Péraldi,[1] Henri Kreis.[1] [1]*Transplantation department, Necker hospital, Paris, France;* [2]*Pneumology, Foch hospital, Suresnes, France.*

Side effects of sirolimus, a new immunosuppressive drug, are mainly thrombocytopenia and hyperlipidemia. We report eight renal transplant patients who developed interstitial pneumonitis during sirolimus therapy.

Seven out of eight patients experienced clinical symptoms including dry cough, exertional dyspnoea, fatigue and weakness whereas only four patients had fever. One patient presented hemoptysis with respiratory distress. Crepitations were found in 5 patients while clinical examination was normal in the three others. Arterial blood gases were altered in 6 patients. Chest X-rays and CT scans disclosed in all patients bilateral asymetric infiltrates predominantly in the lower lobes with a parenchymal condensation in two cases. At least one broncho-alveolar lavage was performed for each patient. 7 of 8 patients presented a moderate to marked alveolar lymphocytosis, associated with intrapulmonary hemorrhage in one case. Alveolar lymphocyte sub-populations, analysed by flow cytometry in three patients, disclosed a majority of CD4 positive T cells. One patient presented only a pure alveolar hemorrhage. Specific stainings and cultures of lavage fluids samples remained negative for bacteria, mycobacteria, fungi, parasites and viruses in all patients. Transbronchial lung biopsies, performed in two patients, displayed an aspect of bronchiolitis obliterans with organizing pneumonia (BOOP) in one patient, and alveolar lymphocytic infiltration in the other. Despite repeated investigations no other etiology of interstitial pneumonitis was found. As drug-induced pneumonitis was suspected, sirolimus was discontinued in seven patients and only decreased in one patient. Clinical and radiological status dramatically improved within a few weeks, and interstitial pneumonitis completely resolved within three months. Therefore, in these eight cases we assumed that the diffuse interstitial pneumonia was related with sirolimus treatment on the following grounds: 1 - Occurence of interstitial pneumonitis during sirolimus therapy. 2 - Absence of any other causes 3 - Resolution of the pneumonia within three months of the discontinuation of sirolimus.

In conclusion, sirolimus associated pneumonitis is an alternative to the diagnosis of opportunistic infection in renal transplant patients taking the drug. Discontinuation or dosage reduction of sirolimus lead to a complete resolution of the symptoms without any persisting lesions.

Abstract# 114

RENAL TRANSPLANTATION IN HIV+ PATIENTS.

Peter G. Stock,[1] Michelle Roland,[2] Laurie Carlson,[1] Stephen Tomlanovich,[2] Willliam Amend,[2] Tom Coates,[2] Chris Freise,[1] John P. Roberts,[1] Nancy L. Ascher.[1]
[1]*Surgery, UCSF, San Francisco, CA;* [2]*Medicine, UCSF, San Francisco, CA.*

Background: People infected with HIV have been denied access to transplantation due to concerns that immunosuppression will accelerate HIV disease and allocate a scarce resource on recipients with an unknown prognosis. Dramatic improvements in HIV related morbidity and mortality secondary to HAART therapy has prompted a re-evaluation of transplantation (tx) as a treatment option for HIV infected people with end-stage renal disease.

Methods: Eligible patients were HIV+, met standard transplant criteria, had undetectable plasma HIV-1 RNA levels for 3 months, a CD4+ T-cell count greater than 200 cells/ml for 6 months, and no history of opportunistic infection/neoplasm (OIs). Outcome measures include: graft function, incidence of OIs, CD4 counts, HIV viral load, genital HPV disease, HHV8 infection, and pharmacokinetic (PK) analysis of protease inhibitors (PI), non-nucleoside reverse transciptase inhibitors (NNRTI) and immunosuppressive agents.

Results: Five patients have received 5 kidney tx [hypertension/HIV nephropathy (3), diabetes mellitus (1), membranoproliferative GN (1)]. All are alive and well (92-242 days) with functioning grafts (creatinine 0.6-1.9). Complications include rejection (1), delayed graft function (1), development of post-tx diabetes (1), and a wound infection (1). HIV viral loads have remained undetectable. CD4 counts have remained stable. There have been no OIs. Mild anal atypia to HPV infection developed in one patient (wk 4) with a normal baseline. High CsA levels requiring decreased dosages occured in patients on PIs, but not in patients on NNRTIs. PI/NNRTI levels have been affected but remain within adequate ranges.

Conclusions: There has not been any evidence of clinical, virologic or immunologic HIV progression. All patients have excellent allograft function. Short-term data suggest that renal tx and immunosuppression is safe in select patients with stable HIV disease.

Abstract# 115

RISK FACTORS FOR PNEUMOCYSTIS CARINII PNEUMONIA IN KIDNEY TRANSPLANT RECIPIENTS. A CASE CONTROL STUDY.

Marcelo V. Radisic, Roberta Lattes, Jennifer Fiore Chapman, María del Carmen Rial, Olga E. Guardia, Fabiana Seu, Domingo H. Casadei.

Objective: To analyze risk factors for Pneumocystis carinii pneumonia (PCP) in kidney transplant recipients (KTx)

Methods: PCP cases diagnosed between July 1994 and July 2000 were matched with two controls (previous and subsequent KTx who did not develop PCP during same follow-up period). PCP diagnosis was established by monoclonal antibodies or silver staining in broncho-alveolar lavage samples. Demographics, organ origin, HLA mismatches,use of poli or monoclonal anti CD3 antibodies (Po/MoAb)for induction or rejection treatment,rejection episodes,cumulative steroid dose for rejection treatment,immunosuppressive regimens and other infections were analyzed.All patients were on trimethoprim-sulfamethoxazole 960 mg qd. prophylactic regimen during the first year after transplantation. Statistical analysis was performed by chi square, t-test,Fischer's exact test and Rank sum test.

Results: Fifty-one patient records were analyzed. PCP:17 Controls:34. Follow-up 16.9±25 mo. Median time to PCP:5.4 months. No significant differences were seen in gender (male 10 vs 15),mean age (39 7 vs 35.4),organ origin (CD 13 vs. 19), HLA mismatches or Po/MoAb use in induction treatment.Significant differences were observed in rejection history:17 vs.26 (p=0.02), steroid resistant rejection 9 vs.7 (Odds ratio [OR] 4.33; 95% Confidence Interval [95CI]= 1.25-15.01; p=0.019),Po/MoAb for rejection treatment 7 vs.3 (OR 7.23; 95CI= 1.67-30.79;p=0.006)

Previous or concomitant CMV 9 vs. 8 (OR 3.65; 95CI =1.08-12-35 p=0.03),tuberculosis 6 vs 1 (OR 18; 95CI= 2.45- 122 p=0.0016), bacterial infections requiring admission 12 vs.10 (OR 5.76; 95CI= 1.64-20.00; p=0.005) or HCV 7 vs. 4 (p=0.03). Immunosuppressive regimen with FK-mycophenolate mophetil (MMF)-Steroids 5 vs. 1 (OR 13.3; Std Err 15.9 p=0.06)and MMF as single variable 8 vs 7(OR 3.4; 95CI 0.99-11.84 p=0.05) were more frequently used in cases.

Conclusions: In spite of TMS-SMX prophylaxis overimmunosuppression is a strong risk factor for PCP. The association with other immunomodulating infections seems to enhance this risk.

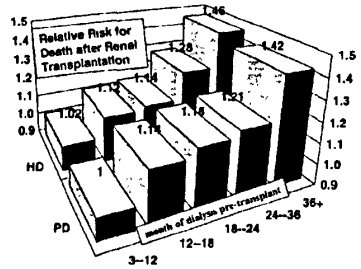
Abstract# 116

THE ASSOCIATION OF WAITING TIME WITH POOR RENAL TRANSPLANT OUTCOME IS UNRELATED TO DIALYSIS MODALITY.

Herwig-Ulf Meier-Kriesche,[1] Friedrich K. Port,[1] Julie A. Arndorfer,[1] Alan B. Leichtman,[1] Diane M. Cibrik,[1] Bruce Kaplan.[1]
[1]*Department of Internal Medicine, University of Michigan, Ann Arbor, MI.*

Waiting time on dialysis prior to renal transplantation (tx) has been shown to be a risk factor for graft and patient survival. We hypothesized that hemodialysis (HD) has deleterious effects on kidney grafts perhaps mediated by pro-inflammatory activities of HD and that these are less with peritoneal dialysis (PD). Based on USRDS data

from 1988 to 1997 we analyzed 43,164 primary adult renal transplants of whom 32,043 were on HD and 11,121 on PD prior to tx. ***Methods:*** Cox proportional hazard models were used to investigate the risk for graft loss and patient death. All analyses were corrected for sex, race, HLA mismatch, panel reactive antibody, delayed graft function, cold ischemia time, induction therapy, dialysis time, etiology of end stage renal disease, immunosuppressive regimen, CMV risk group, donor type and era effect. Patients were assigned to HD vs PD group in intention to treat fashion according to treatment modality at 3 months after dialysis start. Waiting time and HD vs PD were treated as categorical variables and used as interaction terms in the analysis. ***Results:*** Longer waiting time on dialysis was a significant risk factor for mortality ($p<0.001$) regardless of dialysis modality. The relative risk for death after renal tx increased significantly with increasing duration of pre tx dialysis time in both patients on HD and patients on PD. Longer waiting time on dialysis was also a significant risk factor for death censored graft survival ($p<0.001$) regardless of dialysis modality. ***Conclusions:*** Longer waiting times on dialysis negatively impact post tx graft and patient survival regardless of pre-tx dialysis modality. Pro-inflammatory states caused by HD are unlikely to explain the dose dependent negative effect of pre-tx dialysis, as PD confers a virtually identical risk pattern. We speculate that the duration of uremia might cumulatively mediate the worse outcomes with longer waiting times.



CONCURRENT SESSION 13:
IMMUNOSUPPRESSION I

Abstract# 117

EFFECTS OF THE NOVEL IMMUNOMODULATOR FTY720 ON CIRCULATING B CELLS, NK CELLS, AND T CELLS EXPRESSING THE CHEMOKINE RECEPTORS CCR2, CCR5, CXCR4 AND CXCR3 IN KIDNEY TRANSPLANT PATIENTS.

Leonard M.B. Vaessen,[1] Wendy M. Mol,[1] Jan N.M. IJzermans,[2] Teun van Gelder,[1] Willem Weimar.[1] *[1]Internal Medicine; [2]Surgery, University Hospital Rotterdam-Dijkzigt, Rotterdam, The Netherlands.*

FTY720 (FTY), induces a decrease in peripheral blood lymphocyte numbers. It has been suggested that FTY alters lymphocyte recirculation and homing by modulating chemokine receptors. We investigated the effect of FTY on peripheral blood mononuclear cell counts and on the expression of CCR2,CCR5, CXCR4 and CXCR3 on T cells. The patients participated in a multicenter, dose finding study of FTY versus Mycophenolate Mofetil (MMF) in combination with Neoral and corticosteroids in renal transplant patients. Seven patients were enrolled, divided over 3 groups. Group 1: 2 patients, FTY maintenance dose 0.25 mg/day, Group 2: 3 patients, maintenance dose 0.5 mg/day; Group 3: 2 patients, 2 gm/day MMF. FTY was discontinued after 12 weeks. Total numbers of T cells, T subsets CD4 and CD8, NK cells, B cells, monocytes and chemokine receptor positive T cells were determined with flow cytometry in fresh whole blood samples. In the patients treated with 0.5mg FTY/day (Group 2) we observed a strong decrease in the number of circulating T cells (to 23% of baseline) and B cells (to 30% of baseline). This effect was not observed in Groups 1 and 3. CD4 T cells were more affected than CD8 T cells, resulting in CD4/CD8 ratios of 0.22 in Group 2. Numbers of NK cells and monocytes were not affected by FTY treatment (84% and 131% of baseline respectively During FTY treatment only 33% of the T cells expressed CXCR4. Compared to the values at baseline (82%), after discontinuation of FTY (87%) and in Group 3 (69-92%) this was significantly ($p= 0.03$) lower. This decrease in the proportion of CXCR4 positive T cells (34%) was also found in Group 1, however in this group the total number of T cells and B cells was not reduced by FTY. For the receptors CCR2 and CXCR3 no difference was observed during FTY treatment in the Groups 1 and 2, compared to baseline or compared to Group 3. The proportion of CCR5 positive T cells tended to increase during FTY treatment from 13% at baseline, 37% during FTY and 45 % after FTY. In Group 3, 16-26 % of the T cells expressed CCR5.

We conclude that FTY720 remodels the T cell compartment in the peripheral blood. Under FTY CXCR4 negative, CD8 positive T cells stay in the circulation. At a dose of 0.5 mg/day FTY reduces the number of circulating T and B cells to 20-30% of baseline counts, whereas NK cells and monocytes are not affected.

Abstract# 118

PROTEASOME TARGETING IN TRANSPLANTATION.

Kerrie L. Faia,[1] Wei Gao,[1] Vilmos Csizmadia,[1] Nida Shemmeri,[1] Christine S. Pien,[2] Julian Adams,[2] Peter Elliott,[2] Wayne W. Hancock.[1] *[1]Transplantation, Millennium Pharmaceuticals, Inc., Cambridge, MA; [2]Late Stage Dis and Non Clin, Millennium Pharmaceuticals, Inc., Cambridge, MA.*

The proteasome is a large, multi-component protease central to progression of the cell cycle, activation of gene transcription, antigen processing and other crucial cellular processes. Regulating levels of cellular proteins by modulation of proteasome function is a fundamentally new approach to control of alloresponses, though agents such as CsA and rapamycin may have indirect effects on proteasome function. We tested the actions of a novel, highly specific proteasome inhibitor, PS-519 (1 mg/kg/d), alone or in conjunction with a subtherapeutic dose of CsA, in a murine heterotopic vascularized cardiac allograft model (BALB/c to B6); mean survival times were #1, control grafts, 7 d; #2, low CsA, 10 d; #3, PS-519, 14 d ($p<0.01$ vs. #1 or #2), and #4, PS-519/low CsA, 50 d ($p<0.001$ vs. all groups). Comparisons at day 7 showed that PS-519 markedly decreased leukocyte infiltration, myocyte injury and intragraft expression of IFN-γ, IP-10 and CXCR3 mRNA. Inhibition of proteolytic activities of the 26S proteasome was shown by Western blot studies in which the stability of IkBα, phosphorylated IkBα and IkBβ proteins was demonstrated in PS-519-treated recipients, preventing NF-κB activation. Compared to normal hearts, allografts in control mice or those receiving CsA or PS-519 showed similar increases in graft levels of the 20S core protein, indicating PS-519 does not prevent proteasome synthesis. Rather, inhibition of proteasome activation was shown by the ability of PS-519 to prevent intragraft expression of the IFN-γ-inducible β2i and β5i subunits, responsible for the trypsin- and chymotrypsin-like activities of the 26S proteasome, respectively. Proteasome inhibition was confirmed by assay of proteasomal chymotryptic activity in recipient blood, native and transplanted heart samples. Compared to levels in untreated allograft recipients, CsA had no effect on blood and led to only a 23% decrease in cardiac proteasome activity, whereas PS-519 completely inhibited all detectable blood activity and lowered cardiac activity by 87% ($p<0.005$). Our data demonstrate that (i) proteasome activation occurs post-transplant, leading to activation of NF-κB and transcription of multiple pro-inflammatory cytokines and chemokines; and (ii) blockade of the active site of the proteasome can prolong allograft survival, alone, or in synergy with CsA.

Abstract# 119

ANGIOTENSIN II (AII) REGULATION OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) AND ITS RECEPTORS FLT-1 AND FLK-1 IN CHRONIC CYCLOSPORINE NEPHROTOXICITY.

Fuad S. Shihab,[1] William M. Bennett,[2] Hong Yi,[1] Takeshi F. Andoh.[2] *[1]Division of Nephrology, University of Utah, Salt Lake City, UT; [2]Solid Organ and Cellular Transplantation Services, Legacy Hospital, Portland, OR.*

VEGF is a multifunctional cytokine involved in angiogenesis, wound healing and inflammation. We recently showed that VEGF is upregulated in chronic CsA nephrotoxicity (CCN) (*Transplantation* in press). In addition, we previously showed that AII blockade ameliorates CCN. In this experiment, we studied the role of AII blockade on the expression of VEGF and its tyrosine kinase receptors Flt-1 and Flk-1 in CCN.

Salt-depleted pair-fed rats were treated with vehicle (VH), CsA 15 mg/kg/d, CsA +N (nilvadipine), +HH (hydralazine+HCTZ), +L (losartan), or +E (enalapril) for 7 or 28 days. BP, GFR and CsA blood levels were similar in the treated groups. Tubulointerstitial fibrosis and afferent arteriolopathy, the hallmarks of CCN, were improved in the +L and +E groups ($P<0.05$), a phenomenon not observed in the +N and +HH groups.

We studied the mRNA expressions of VEGF, Flt-1 and Flk-1 by Northern blot and the protein expression of VEGF by Western blot. Values are mean±SEM; N=5-6/group; *P<0.05 vs VH; #P<0.05 vs CsA; mRNA values were GAPDH corrected; protein values were normalized

	VH	CsA	CsA+N	CsA+HH	CsA+E	CsA+L
VEGFmRNA-7d	0.16±0.03	0.29±0.05*	0.25±0.02*	0.28±0.05*	0.17±0.02#	0.15±0.09#
VEGFprotein-7d	7.3±1.3	10.9±1.5*	10.4±4.1	11.9±0.8*	6.6±4.3	7.2±2.6#
Flt-1 mRNA-7d	0.13±0.07	0.53±0.15*	0.32±0.14*#	0.27±0.04*#	0.30±0.16*#	0.25±0.18*#
Flk-1 mRNA-7d	0.10±0.04	0.67±0.29*	0.44±0.27*	0.35±0.07*	0.28±0.23#	0.21±0.16#
VEGFmRNA-28d	0.33±0.05	0.50±0.01*	0.43±0.03*	0.50±0.09*	0.39±0.01*#	0.38±0.03#
VEGFprotein-28d	7.8±1.5	11.6±1.7*	9.0±1.3	9.9±0.7*	8.3±1.3#	8.5±0.4#
Flt-1 mRNA-28d	0.15±0.12	0.49±0.14*	0.17±0.03#	0.19±0.08#	0.15±0.02#	0.17±0.06#
Flk-1 mRNA-28d	0.25±0.10	0.42±0.07*	0.31±0.12	0.49±0.15*	0.20±0.11#	0.28±0.04#

While lowering BP decreased VEGF, specific AII blockade with AII receptor antagonist (L) or ACE inhibitor (E) significantly decreased VEGF mRNA and protein to VH level. The expression of Flk-1 followed that of VEGF. On the other hand, Flt-1 mRNA was equally downregulated in all treated groups. The effects of L and E were similar and confirm prior data that show that AII induces VEGF expression through activation of the AT1 receptor. Since AII is known to upregulate TGF-β expression, the interactions between AII, TGF-β and VEGF will need to be further studied in this model.

Abstract# 120

PICEATANNOL, A SELECTIVE SYK/ZAP BLOCKER, IN COMBINATION WITH SUBTHERAPEUTIC DOSES OF CYCLOSPORIN A PROLONGS ALLOGRAFT SURVIVAL IN RATS.

Gokhan Yagci,[1] Luis Fernandez,[1] Nobuhiro Ishido,[1] Stuart J. Knechtle,[1] Majed M. Hamawy.[1] *[1]Surgery, University of Wisconsin, Madison, WI.*

Current immunosuppressive agents have significantly improved graft survival. However, these agents lack specificity and are associated with toxicity. Thus, there is a need for new specific immunosuppressive agents. T cells are central for the immune response to allografts. The activation of T cells by alloantigens stimulates the critical kinases Syk and Zap, which are found predominantly in T and B cells but not in cells of nonhematopoietic origin. We examined whether piceatannol (Pic), a selective Syk/Zap blocker, alone or in combination with subtherapeutic doses of CsA, would prevent graft rejection in the ACI>>LEW kidney transplant model. METHODS: Group 1 (n=6) received Pic 30 mg/Kg/day IV and CsA 2 mg/kg/day IM from day -3 to + 7 post transplant. Pic dose was then reduced to 10 mg/Kg/day and the combined treatment was resumed until day +60. Group 2 (n=7) received 2 mg/Kg/day CsA alone from day -3 to +60. Group 3 (n=4) received Pic alone using the same protocol as for group 1. Group 4 (n=2) received only the vehicle DMSO. Graft rejection was defined as either serum creatinine level over 2 mg/dl or animal death, whichever happened earlier. RESULTS: Pic alone was not effective in prolonging allograft survival. However, Pic in combination with a subtherapeutic dose of CsA significantly prolonged graft survival ($p<0.05$). Strikingly, excellent graft function is still present (> 100 days) in animals in Group 1 despite the withdrawal of the immunosuppressive regimen at day 60. In contrast, immunosuppression withdrawal led to an immediate deterioration in the function of the graft in the one rat that survived on the low dose of CsA (Group 2). No long term weight loss or increase in serum creatinine and liver enzymes was evident in Group 1. CONCLUSIONS: Pic, when combined with subtherapeutic doses of CsA, successfully prolonged kidney allograft survival; thus, therapeutic strategies that target Syk/Zap signaling pathways could potentially be used to ameliorate the undesirable side effects of calcineurin inhibitors.

Group #	Treatment	Graft Survival (days)
1 (n=6)	2 mg/Kg/day CsA (d -3 to +60) + 30 mg/Kg/day Pic (d -3 to +7), 10 mg/Kg/day Pic (d +8 to +60)	>18, >18, >26, >27, >100, >100 ($p<0.05$)
2 (n=7)	2 mg/Kg/day CsA (d -3 to +60)	8, 9, 9,9,9,10, 65
3 (n=4)	30 mg/Kg/day Pic (d -3 to +7), 10 mg/Kg/day Pic (d +8 to +60)	7,8,8,9
4 (n=2)	DMSO	7,7

Abstract# 121

INHIBITION OF JAK3 ALONE BLOCKS ALLOGRAFT REJECTION AND IS SYNERGISTIC IN COMBINATION WITH CYCLOSPORINE BUT NOT RAPAMYCIN.

Stanislaw M. Stepkowski,[1] Erwin-Cohen Rebecca,[2] Fariba Behbod,[2] Mou-Er Wang,[1] Barry D. Kahan,[1] Robert A. Kirken.[1] *[1]Division of Organ Transplant, University of Texas-Houston, Houston, TX; [2]Department of Integrative Biology, University of Texas-Houston, Houston, TX.*

Purpose: The serine-threonine phosphatase calcineurin (CaN) is inhibited by cyclosporine A (CsA) and the interleukin-2 (IL-2) responsive serine-threonine kinase, mammalian target of rapamycin (mTOR), is blocked by rapamycin (RAPA). Because of ubiquitous expression of these enzymes, their inhibition produces adverse effects. We examine the hypothesis that selective inhibition of Janus tyrosine kinase 3 (Jak3), which is activated by T cell growth factors (TCGFs) including IL-2, IL-4, IL-7, IL-9, and IL-15, may provide a unique target for immunosuppression **Results:** Human or rat T-cells were stimulated for 72 hrs with PHA, rested for 16 hrs in the presence of Jak3 inhibitor AG490 or PNU804, and stimulated with 100 nM IL-2. Each of the Jak3 inhibitors (~20 μM) blocked IL-2- and other TCGFs-mediated cell growth as measured by phosphotyrosine Western blot. Both agents also individually inhibited activation of Jak3 substrates, namely, signal transducers and activators of transcription (Stat)5a/b, adapter protein SHC, and Erk1/2, as measured by phospho-Western blots. In contrast, neither agent could affect the T cell receptor (TCR)-induced activation of Lck or Zap70. Moreover, although Stat5a/b DNA binding to an oligonucleotide probe was greatly impaired by AG490 or PNU804, neither drug blocked the non-Jak3 mediated TNF-α-induced NFκB DNA binding. Untreated ACI ($RT1^a$) recipients rejected Lewis (LEW; $RT1^l$) heart allografts with a mean survival time (MST) of 8.8 ± 0.8 days. A course of 7 days of daily injections of 10 or 20 mg/kg AG490 prolonged survivals to 14.6 ± 1.5 and 18.0 days, respectively. Although CsA (10 mg/kg) alone (p.o. days 0-2) extended survivals to 12.2 ± 0.8 days, the addition of 20 mg/kg AG490 produced a MST of 57.6 ± 39.0 days. The combination index (CI) value of 0.01 indicated a synergistic interaction (CI<1 is synergistic and CI>1 is antagonistic). RAPA in combination with AG490 produced only an additive effect (CI=1.0-1.4). Similar results were obtained by PNU804 in combination with CsA or RAPA. **Conclusion:** Inhibition of Jak3 alone prolongs allograft survival and the effect is potentiated by inhibition of CaN but not mTOR.

Abstract# 122

SYNERGISTIC EFFECT OF TACROLIMUS WITH FK778 OR FK779 IN PREVENTION OF ACUTE HEART ALLOGRAFT REJECTION AND IN REVERSAL OF ONGOING ACUTE HEART ALLOGRAFT REJECTION IN THE RAT.

Kupa K. Bilolo,[1] Shijie Qi,[1] Jun Ouyang,[1] Xiang Wang,[1] Dasheng Xu,[1] Pierre Daloze,[1] Ihor Bekersky,[2] William E. Fitzsimmons,[2] Huifang Chen.[1] *[1]Laboratory of Experimental Surgery, Research Center of CHUM, Notre-Dame Hospital, University of Montreal, Montreal, QC, Canada; [2]Fujisawa Healthcare Inc., Deerfield, IL.*

FK778 (MNA715) and FK779 (MNA279) are new derivatives of LEF's active metabolite A771726. Both FK778 and FK779 have potent immunosuppressive effect to prevent allograft and xenograft rejection in animal models. In this study, the mono- and combination therapy of tacrolimus with FK778 or FK779 was examined in prevention of acute heart allograft rejection and in reversal of ongoing heart allograft rejection in the rat. All drugs were administered orally for 14 days after surgery. Eighteen groups (n=6) were involved in the first part of heart allografting model and 10 groups were involved in the second part of study. Lewis (LEW, RT1) recipients received Brown Norway rat hearts in whole study. The naïve control group showed a mean survival time (MST) of 6.5±0.6 days. There were dose-responses to monotherapy of tacrolimus 0.5, 1.0, 1.5 mg/kg/day (7.8±0.8, p=0.013; 16.4±6.3, p=0.004; 21.0±8.8 days, p=0.002); FK778 10, 20, 30 mg/kg/day (9.6±0.9, p=0.0001; 14.2±2.6, p=0.0001; 16.0±0.7 days, p=0.0001); and FK779 10, 20, 30 mg/kg/day (12.8±0.8, p=0.0001; 29.6±17.4, p=0.009; 25.4±2.4 days, p=0.0001) respectively. Results with the combination therapy indicate that a additive to synergistic interaction was produced when compared with monotherapy of tacrolimus, FK778 or FK779: tacrolimus 0.5 mg/kg/day + FK778 10 mg/kg/day (15.2±1.7 days, combination index (CI) = 1.022), tacrolimus 0.5 mg/kg/day + FK778 20 mg/kg/day (19.5±4.4 days, CI = 0.874), tacrolimus 1.0 mg/kg/day + FK778 10 mg/kg/day (25.0±5.4 days, CI = 0.680), tacrolimus 1.0 mg/kg/day + FK778 20 mg/kg/day (28.7±7.0 days, CI = 0.631). These results were repeatable in tracrolimus combined with FK779 treatment. In the second part of the study of reversal of ongoing acute heart allograft rejection model, the combined therapy of tacrolimus 0.5 mg/kg/day + FK778 20 mg/kg/day (48.0±24 days, CI=0.85) and tacrolimus 1.0 mg/kg/day + FK778 20 mg/kg/day (49.8±20.5 days, CI=0.874) represented synergistic interaction compared with monotherapy of tacrolimus or FK778. Combination therapy of tacrolimus and MNAs produces a synergistic effect in prevention of heart allograft rejection and in reversal of ongoing heart allograft rejection in the rat

Abstract# 123

TREATMENT WITH A SHORT COURSE OF LF15-0195 AND CONTINOUS CYCLOSPORINE A INHIBITS ACUTE XENOGRAFT REJECTION IN A RAT-TO-MOUSE CARDIAC TRANSPLANTATION MODEL.

Hao Wang,[1,3] Karoline A. Hosiawa,[2,4] Bertha Garcia,[3] Patrick Dutartre,[6] Calvin Stiller,[5] David J. Kelvin,[2,4] Robert Zhong.[1,2,3,4] *[1]The Multi-Organ Transplant Program, London Health Sciences Centre, London, ON, Canada; [2]Department of Microbiology and Immunology, The University of Western Ontario, London, ON, Canada; [3]Department of Pathology, The University of Western Ontario, London, ON, Canada; [4]Immunology and Transplantation Group, John P. Robarts Research Institute, London, ON, Canada; [5]Department of Medicine, The University of Western Ontario, London, ON, Canada; [6]Immunology Research, Fournier Laboratories, Daix, France.*

Searching for a novel immunosuppressive agent to effectively prevent acute vascular rejection (AVR) is essential for success in clinical xenotransplantation. The present study was undertaken to study the efficacy of LF15-0195 (LF), a new immunosuppressive analogue of 15-deoxyspergualin (DSG) in prevention of AVR in a rat-to-mouse cardiac xenograft model. We previously reported that Lewis rat hearts transplanted into BALB/c mice developed typical AVR in 6 days (*Nature Medicine* 2000; 6: 549). In this study, we found that high dose of CsA (15mg/kg, daily, S.C.) was not able to inhibit AVR and the graft was rejected in 11.3±1.9 days. Graft histology and immunohistology showed typical AVR, characterized by hemorrhage, fibrin deposition, thrombosis and massive deposition of IgG and IgM. Serum xenoreactive antibodies (xAbs) were markedly elevated in these animals. In contrast, treatment with LF alone (2mg/kg/day, day -1 to 14, S.C.) significantly prolonged graft survival to 19.3±0.7 days. Notably, xAbs was significantly inhibited and the rejection pattern of these grafts was switched from AVR to cell-mediated rejection (CMR). When CsA was added to LF, the graft survival was further prolonged to 58.5±17.3 days. The pathology showed striking attenuation of both AVR and CMR. Sequential studies on days 6 and 14 showed that the combination of LF and CsA completely inhibited antibody production and T cell infiltration. However, infiltration by NK cells and macrophages was remarkably enhanced when LF treatment was discontinued on day 14. We conclude that 1) LF can effectively prevent AVR by markedly inhibiting xAb production; 2) treatment with short course of LF and continuous CsA inhibits antibody production and T cell activation and 3) this therapy is less effective to inhibit innate immunity. These data suggest that this novel agent may be of value to prevent AVR in a pig-to-primate xenotransplantation model.

Abstract# 124

NEW MECHANISMS OF IMMUNOSUPPRESSION OF SIROLIMUS *IN VIVO*: DIFFERENTIAL SUPPRESSION OF MULTIPLE IMMUNE FUNCTIONS OF T AND B CELLS AND MONOCYTES IN NON-HUMAN PRIMATES. Camille Dambrin,[1] Tudor Bîrsan,[1] Jochen Klupp,[1] Laurie Hook,[1] Tuan Lam,[1] Uwe Christians,[2] Randall Morris.[1] *[1]Cardiothoracic Surgery, Stanford University, Stanford, CA; [2]Biopharmaceutical Sciences, University of California, San Francisco, CA.*

Background: Although the mechanisms of the immunosuppressive action of sirolimus (SRL) have been studied *in vitro*, SRL's effects after administration *in vivo* have not been described. We use novel whole blood flow cytometric (FCM) assays to quantitate immune functions and to describe new mechanisms of action of SRL.

Methods: Cynomolgus monkeys (NHP, n=5) were treated PO once daily with 1-1.5 mg/kg of SRL over several weeks. At steady state, SRL-treated NHP were sedated with ketamine and heparinized blood was collected before and at 2, 10, 24 hr after treatment. SRL plasma levels were measured by LC/MS. Blood was collected from 2 untreated NHP to control for effects of ketamine on immune functions. Blood samples collected from all NHP were stimulated with different mitogens in culture. Lymphocyte proliferation was quantitated by FCM (expression of proliferating cell nuclear antigen [PCNA] in SG M phase cells). FCM quantitated T cell functions: expression of CD25 (IL-2R), CD71 (transferrin receptor), adhesion/costimulatory molecules (CD11a, CD154), FAS (CD95) and expression of intracellular cytokines (IL-2 and IFNγ). Intracellular cytokine production in monocytes (TNFα) and B cell lymphocyte activation (CD69) were also measured.

Results: SRL not only inhibited lymphocyte proliferation, but also inhibited the expression of T cell surface molecules. At 2 hr after dosing, inhibition of expression of CD25, CD71, CD95, and CD154 was maximal (81%, 86%, 76% and 82%, respectively). At trough SRL levels, lymphocyte proliferation and T cell surface molecule expression were inhibited by 70 - 80% compared to controls. In contrast, SRL did not suppress T cell or monocyte intracellular cytokine production or B cell CD69 expression.

Conclusion: Using novel whole blood FCM assays of T, B and monocyte functions, we measured the effects of SRL administered *in vivo*. We describe the first results showing that SRL inhibits some T cell functions (expression of multiple cell surface molecules) but not others (intracellular cytokine expression) and does not alter monocyte function or suppress B cell CD69 expression. This more complete profile of action of SRL can be used to rationally combine SRL with other immunosuppressants to create more effective and safer means to control rejection.

Abstract# 125

MECHANISMS OF INDUCTION OF ALLOGRAFT TOLERANCE IN RAT BY SHORT-TERM TREATMENT WITH LF15-0195. Elise Chiffoleau,[1] Patrick Dutartre,[2] Claire Usual,[1] Jean-Paul Soulillou ,[1] Maria-Cristina Cuturi.[1] *[1]U437, INSERM, Nantes, France; [2]FOURNIER laboratories, Daix, France.*

Since the discovery of 15-deoxyspergualin (15-DSG) in the early 80's, only a limited number of new analogues have been described in the literature. LF08-0299 was first reported as having long term stability in water solutions and able to induce long term immune tolerance in a rat heart allograft model. Structural modifications led to LF15-0195 which demonstrates a more potent immunosuppressive activity than 15-DSG and LF08-0299. In this report, we tested the effects of this new compound in a model of rat heart allograft and analyzed the anti-donor response during the first week after transplantation.

LEW.1A rat received a fully MHC mismatched LEW.1W heart allograft. LF15-0195 was administered daily for 20 days from the day of grafting at 3mg/kg i.p. Graft infiltrating cells were studied at day 5 after transplantation by immunohistology and the cytokines production by quantitative RT/PCR (Taq Man). Anti-donor antibodies in sera from untreated or LF treated recipients, were assessed against donor splenocytes. A 20 day treatment with LF15-0195 induces long-term tolerance in 95% of recipients. This tolerance is donor-specific since donor skin grafts were accepted, whereas third party skin grafts were rejected. Graft leukocyte infiltrate on day 5 after transplantation, was significantly reduced (50% decrease) in allografts from LF15-0195 treated recipients as compared to allografts from untreated recipients. Intragraft cytokine expression by quantitative RT/PCR, revealed a reduced expression of macrophage-related cytokines (IL10 and TNFα) as well as IFNγ in allografts from LF15-0195 treated recipients. TGFβ and IL13 mRNA expressions were unmodified while a strong inhibition in iNOS protein expression was also noted in LF15-0195 treated recipients. Moreover, we show that in contrast to untreated recipients that had developed strong anti-donor alloantibody response at day 7 after transplantation, LF treated recipients did not develop any anti-donor alloantibodies.

These results suggest that LF15-0195 is a powerful modulator of allograft rejection and is able to induce donor-specific allograft tolerance. During the first week after transplantation, it inhibit recruitment as well as activation of macrophages in the graft. Production of IFNγ and anti-donor antibodies were also dramatically decreased. Further experiments will help to explain the precise links between these properties and the final induction of long term immune tolerance

CONCURRENT SESSION 14:
REJECTION II

Abstract# 126

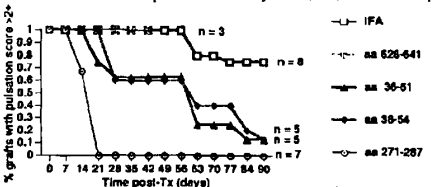
ACUTE REJECTION OF A SKIN GRAFT EXPRESSING A DEFINED MINOR ANTIGEN. Benjamin D. Ehst,[1] Marc K. Jenkins.[1] *[1]Department of Microbiology, University of Minnesota, Minneapolis, MN.*

The underlying cellular interactions causing rejection of minor histocompatibility antigen (mHAg)-mismatched allografts are not well understood. To address this issue we have developed a model of graft rejection across a single mHAg barrier where we can directly visualize the interactions between APC and T cells specific for peptides derived from the minor antigen *in vivo*. Transgenic B6 mice were generated expressing a membrane-bound form of chicken ovalbumin (OVA). Tail skin grafting from one of these transgenic lines, mOVA007, onto normal B6 recipients creates a single minor antigen rejection model. Splenocytes from the transgenic line failed to produce a primary MLR when cultured with B6 APC *in vitro*, consistent with the expected response against a mHAg. B6 recipients completely reject mOVA007 skin by three weeks post-transplant (mean survival time[MST]=15.5 days) while syngeneic B6 control skin is accepted indefinitely. mOVA007 skin graft recipients also mount a humoral response characterized by the production of anti-OVA IgG1 antibodies without any detectable IgG2a (indicative of a host environment containing limited IL-12), although the rejection process is not dependent on antibody production since B cell deficient recipients reject mOVA007 grafts without delay (MST=13.5 days). In addition, TCRα-/- recipients fail to reject the transgenic skin and MHC Class II deficient recipients show significantly prolonged graft survival (MST=38 days). Adoptive transfer of OVA-specific CD8+ and CD4+ TCR transgenic T cells into recipient mice one day prior to grafting results in a slight acceleration of mOVA007 skin rejection. Tracking of these transferred cells via a congenic Thy1 marker reveals activation and expansion almost exclusively in the draining nodes of mOVA007 graft recipients starting around day 4 after grafting, peaking near day 8 and falling dramatically by the time the rejection is complete. Immunohistochemical analysis of the grafted skin shows accumulation of the transferred cells in mOVA007 grafts without appearance in control B6 grafts. These results demonstrate that rejection of a single mHAg-expressing skin graft can occur in a rapid, acute fashion dependent on TCRab+ T cells. It also suggests that activation is confined to the lymph nodes draining the graft bed, reaches a peak about a full week before the graft is completely lost and can occur under conditions where IL-12-dependent CTL activity may be limited.

Abstract# 127

IMMUNE RESPONSES TO HEAT SHOCK PROTEINS (HSP) WORSEN ALLOGRAFT SURVIVAL AND ENHANCE IFN-γ AND ALLOANTIBODY PRODUCTION. Milagros Samaniego,[1] Salma Rahimi,[1] Suyi Cao,[1] Lauren Armstrong,[1] Fred Sanfilippo,[1] William Baldwin.[1] *[1]Dept. of Pathology, Johns Hopkins Sch. of Med., Baltimore, MD.*

Heat shock protein 70 (HSP70) is a cytosolic chaperone that is upregulated in acute rejection and cellular injury, expresses numerous T- and B cell epitopes and is known to elicit specific immune responses. To investigate if immune responses against HSP70 contribute to allograft rejection, BN rats were immunized with HSP70 peptides (HSP-Pep), transplanted on day 21 with an LxBN (F1) cardiac graft (Tx) and treated with CsA. The HSP-Pep chosen for immunization contain a known binding motif for rat Class II MHC (aa 36-51, 38-54, 271-287) and elicit a strong MHC class II-restricted IgG response. HSP-Pep aa 628-641 is not immunogenic and serves as a control Pep. HSP-sensitized rats had a shorter graft survival due to severe rejection than rats immunized with Incomplete Freund's adjuvant (IFA) or control Pep (see figure).



HSP-sensitized rats produced high titers of allogeneic Class I and Class II-IgG by day 7 post-Tx, whereas no production of allo-IgG was detected in control rats by day 42 post-Tx. Furthermore, HSP-Pep immunization resulted in high serum levels of IFN-γ detectable by day 7 post-immunization. After transplant, HSP-sensitized rats had an early and sustained elevation of serum IFN-γ that persisted until rejection. In control rats, IFN-γ was only detectable by the time of rejection.

We conclude that HSP-Pep are important immunogens in transplantation. HSP-Pep may contribute to the pathogenesis of acute rejection by acting as natural adjuvants that elicit the production of IFN-γ, prime the immune system and enhance the adaptive response to allogeneic MHC molecules. This effect of HSP-Pep is not suppressed by CsA. Because HSP70 is upregulated and processed during ischemia and viral infections, the generation of HSP-Pep immune responses may be responsible, in part, for the increased immunogenicity of allografts exposed to such conditions.

Abstract# 128

CD4 FAS-FAS-L CYTOTOXICITY REPRESENTS ONE OF THE EFFECTOR PATHWAYS RESPONSIBLE FOR THE ACUTE REJECTION OF MINOR TRANSPLANTATION ANTIGENS.

Murielle Surquin,[1] Alain Le Moine,[1] Veronique Flamand,[2] Michel Goldman,[2] Daniel Abramowicz.[1] *[1]Nephrology, Erasme Hospital, Brussels, Belgium; [2]Immunology, Erasme Hospital, Brussels, Belgium.*

Cytotoxicity mediated by CD8 cells and the production of Th1 and Th2 cytokines are known to contribute to the rejection of minor transplantation antigens Whether CD4 Fas-FasL mediated cytotoxicity also plays a role as an effector mechanism in this setting has not been studied to date. We investigated this issue during the rejection of β2 microglobulin (β2m)-derived minor transplantation antigens in mice. β2m-derived peptides are constitutively presented by both MHC class I and class II molecules. These antigenic complexes represent minor transplantation antigens as indicated by the rejection of β2m-positive grafts ($\beta2m^{+/+}$) by genetically β2m-deficient recipient mice ($\beta2m^{-/-}$).

We first studied if β2m peptides-MHC class II complexes alone are sufficient to trigger rejection. $\beta2m^{-/-}$ recipient mice were grafted with $\beta2m^{+/+}$ skins harvested from donor mice genetically devoid of the Transporter-associated with Antigen Processing I molecule ($Tap1^{-/-}$). These mice are able to present β2m peptides in association with MHC class II, but not MHC class I molecules. $\beta2m^{-/-}$ mice previously primed towards β2m rejected $Tap1^{-/-}\beta2m^{+/+}$ and wild-type $Tap1^{+/+}\beta2m^{+/+}$ skins equally rapidly (median survival time: 13±6 versus 15±5 days; P=NS). This indicates that effector pathways directed solely at β2m peptides-MHC class II molecules can lead to rapid rejection. Next, we showed that CD4 cells play a major role in this setting, as double knock-out $\beta2m^{-/-}/CD8^{-/-}$ mice primed to β2m antigens also rapidly reject a $Tap1^{-/-}\beta2m^{+/+}$ skin. In order to evaluate the role of Fas/FasL mediated CD4 cytotoxicity during this rejection process, we transplanted $\beta2m^{+/+}$ skins lacking a functional Fas molecule (B6 lpr donor strain). Fas-deficient $\beta2m^{+/+}$ skins transplanted on $\beta2m^{-/-}$ mice showed a significant prolongation of survival time when compared to wild type $\beta2m^{+/+}$ skin grafts (median survival time: 13±6 versus 30±9 days, respectively; P<0.01).

We conclude that CD4 Fas-FasL cytotoxicity represents one of the effector mechanisms responsible for the acute rejection of β2m-associated minor transplantation antigens.

Abstract# 129

INFILTRATION OF ALLOGRAFTS BY NATURAL KILLER CELLS PRECEDES T CELLS: CROSS TALK BETWEEN THE INNATE AND ADAPTIVE IMMUNE SYSTEM? Joyce Popoola,[1] Kathryn J. Wood,[2] Steven H. Sacks,[1] Wilson Wong.[1] *[1]Nephrology and Transplantation, Guy's Campus, King's College, London, United Kingdom; [2]Nuffield Department of Surgery, University of Oxford, Oxford, United Kingdom.*

Much is known about the role of the adaptive immune response in the rejection process. In contrast the innate response, e.g. Natural Killer (NK) cells remain a mystery in solid organ transplants. We therefore set out to determine the kinetics of graft infiltrating NK cells and to assess whether they are functional in the early stages post transplantation,in terms of cytokine production.

Fully allogeneic donor BALB/c ($H2^d$) or CBA ($H2^k$) mouse hearts were transplanted into C57BL/6 ($H2^b$) recipients. Cardiac allografts were harvested from recipient animals at different time points post transplant Graft infiltrating cells were isolated using collagenase digestion followed by centrifugation through a ficoll gradient for analysis using 2 colour flow cytometry. Cells were stained for DX5 (pan NK cell marker), CD4 and CD8 expression. NK cells were also permeabilised using saponin and stained for intracellular IL2 and IFNγexpression.

Rejection of allografts takes place between the 9th and 11th day post transplant. In the first 5 days, the majority of the graft infiltrating cells consist of NK cells

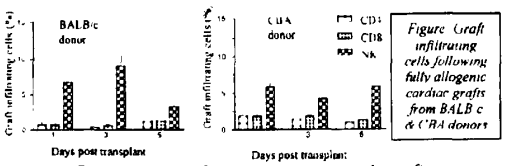


Figure Graft infiltrating cells following fully allogenic cardiac grafts from BALB c & CBA donors

The NK to CD4/CD8 cell ratio falls with time suggesting that graft rejection may have been initiated by NK cells in the first few days, after which T cells take over. Intracellular cytokine analysis on freshly isolated graft infiltrating NK cells (unstimulated ex-vivo) showed production of IFNγ but not IL2 within 1 day of transplantation. Since NK cell infiltration precedes that of T cells, they may play a crucial role in the rejection process, possibly through IFNγ production in the early post-transplant period during which T cell priming takes place. Therefore, the innate (NK cell) response may play a crucial role in the initiation of the adaptive (T cell) response in the rejection process

Abstract# 130

MECHANISMS OF NITRIC OXIDE MEDIATED CYTOTOXICITY IN CARDIAC ALLOGRAFT REJECTION: PRESERVATION OF ACONITASE, A CRITICAL MITOCHONDRIAL ENZYME, WITH CSA. Allan M. Roza,[1] Galen M. Pieper,[1] Gail Hilton,[1] Cara L. Olds,[1] Christopher C. Felix,[2] Eugene A. Konorev,[2] Mark B. Adams.[1] *[1]Division of Transplant Surgery, Department of Surgery, Medical College of Wisconsin, Milwaukee, WI; [2]Biophysics Research Institute, Medical College of Wisconsin, Milwaukee, WI.*

We have shown that oxidative and nitrosative (nitric oxide; NO) stresses contribute to rat cardiac allograft destruction.The mechanisms of NO mediated cytotoxicity are unknown. We hypothesized that NO derived from iNOS and its derivative, peroxynitrite, inhibit mitochondrial respiration by inactivating the critical electron transport chain enzyme, aconitase METHODS:WF rats receiving a Lew strain heterotopic cardiac allograft were either untreated or received low dose CsA (2.5mg/kg im daily) until rejection Induction of myocardial iNOS protein was determined by Western blotting. NO production was verified by measurement of plasma nitrate+nitrite (end products of NO) and directly within the myocardium by electron paramagnetic resonance (EPR) spectroscopy. Myocardial immunoreactive nitrotyrosine was analysed as a marker of peroxynitrite production. Myocardial aconitase enzyme activity was directly measured. Controls were isografts (Lew-Lew) or native hearts of isograft and allograft recipients. RESULTS: Untreated allografts rejected at 6.6±0.2 days (n=6); with CsA allografts rejected at 12 6±0.5 days(n=14;p<0 01). In untreated allografts vs. controls we noted: (1) A time dependent increase in myocardial iNOS protein beginning at day 4 post-transplant. (2) Localization of iNOS to the myocardium including the cardiomyocyte by immunoreactive staining. (3) A 5x increase in plasma nitrate+nitrite by day 5. (4) Myocardial NO production (EPR nitrosylheme protein signal)on day 4 and peaking by day 6. (5) Confirmation of peroxynitrite production on day 6. (6) A time dependent decrease in myocardial aconitase enzyme activity as early as day 4 (15%) and maximal by day 6 (43%). Treatment with CsA (1) prevented induction of iNOS (2) eliminated nitrate+nitrite and EPR signals for nitrosylated protein and (3) preserved aconitase enzyme activity. CONCLUSIONS. These data confirm production of iNOS derived NO and its toxic derivative, peroxynitrite, and inhibition of aconitase within the rejecting myocardium In vitro, purified aconitase is inactivated by peroxynitrite. EPR studies to confirm nitrosylation of aconitase are underway. This is the first report of inactivation during rejection of a mitochondrial protein important for respiration.We conclude that both heme and non-heme proteins are molecular targets for NO cytotoxicity in early rejection.These targets are preserved with CsA treatment

Abstract# 131

DIVERGING SIGNALING PATHWAYS FOR REGENERATIVE AND PROFIBROTIC EFFECTS OF THROMBIN IN PROXIMAL TUBULAR CELLS (PTEC). Giuseppe Grandaliano, Paola Pontrelli, Michele Ursi, Loreto Gesualdo, Paolo F. Schena. *[1]Div. of Nephrology, DETO, University of Bari, Bari, Italy.*

Interstitial fibrin deposition is a common histopathological finding in acute and chronic graft rejection. We have previously demonstrated that thrombin, released upon coagulation cascade priming, may induce DNA synthesis in cultured PTEC and regulate the PTEC fibrinolytic and profibrotic activity, through the expression of PAI-1. The intracellular mechanisms leading to these cellular effects of thrombin in PTEC are still largely unclear. We have previously demonstrated that thrombin activates the cytoplasmic tyrosine kinase c-src. Thus, in the present study, we evaluated the effect of a specific src inhibitor, PP1, and its inactive analogue, PP3, on thrombin-induced DNA synthesis and PAI-mRNA abundance. Interestingly, PP1, but not PP3, inhibited both thrombin-elicited DNA synthesis and PAI-1 gene expression. Since src may activate Protein kinase C (PKC) in several cell types, we investigated the effect of PKC activation on the two target effects. Stimulation of PKC by PMA induced a striking induction of both DNA synthesis and PAI-1 mRNA levels. Downregulation of this signaling enzyme, by prolonged PMA incubation, completely inhibited thrombin-induced proliferation and PAI-1 expression. To further investigate the signaling pathways linking thrombin-induced PKC activation to the observed nuclear events, we evaluated the effect of PMA and thrombin on jun-N-terminal kinase (JNK), a stress kinase involved in several cell responses. Both agonists induced JNK phosphorylation in a time-dependent manner. Moreover, thrombin-elicited JNK activation was abolished by prolonged PMA pre-incubation. Interestingly, pretreatment with curcumin, a specific JNK inhibitor, caused a marked inhibition of thrombin-induced PAI-1 gene expression, but did not influence the increased DNA synthesis in response to the coagulation factor.

In conclusion, our data would suggest that both thrombin-induced proliferation and PAI-1 gene expression depend upon src and subsequent PKC activation, but the signaling pathways for these cellular responses diverge downstream. Indeed, the profibrotic, but not the regenerative effect, depends upon JNK activation. These findings suggest a potential therapeutic approach to reduce the fibrogenic effect of thrombin and potentiate its regenerative action in acute and chronic graft rejection.

Abstract# 132

LIGATION OF HLA CLASS I INDUCES TYROSINE PHOSPHORYLATION OF $P60^{SRC}$, $P125^{FAK}$ AND PAXILLIN IN HUMAN ENDOTHELIAL CELLS. Yiping Jin, Elaine F. Reed. [1]*Pathology, UCLA, Los Angeles, CA.*

The development of anti-HLA antibodies (ab) following transplantation is positively associated with chronic rejection and transplant atherosclerosis.In previous studies we have shown that ligation of HLA class I molecules with anti-HLA antibodies stimulates fibroblast growth factor receptor expression (FGFR) and proliferation of endothelial cells (EC) and smooth muscle cells (SMC) suggesting a possible mechanism for the development of transplant atherosclerosis. To further explore the contribution of anti-HLA ab to the development of chronic rejection we have examined the ability of ab to class I antigens to induce tyrosine phosphorylation of intracellular proteins in EC. For this, EC were treated with for various periods of time with the anti-class I mAb W6/32 and cell lysates were electrophoresed and immunoblotted with the anti-phosphotyrosine antibody 4G10. Treatment of EC with mAb W6/32 resulted in time dependent increase in tyrosine phosphorylation of proteins at approximate molecular masses of 42, 60, 80,120 kDa. To investigate the nature of the phosphorylated proteins we assessed the role of the Src family of protein kinases in the class I signaling pathway. Increased tyrosine phosphorylation of Src (p60) was detected at 1 minute following treatment with mAb W6/32, reaching a maximum level after 15 minutes.Similarly, Fyn (p59) a member of the Src family of protein kinases was rapidly phosphorylated upon class I ligation.Treatment with anti-class I antibodies also stimulated tyrosine phosphorylation of paxillin (p68) and Focal Adhesion Kinase (FAK)(p125). Addition of cytochalasin D an actin depolymerizing agent remarkably decreased tyrosine phosphorylation of FAK and its association with paxillin. Taken together, our findings suggest that anti-HLA class I antibodies may stimulate FGFR expression through tyrosine phosphorylation of Src and Focal Adhesion proteins. This signaling pathway involving actin cytoskeletal reorganization may be required for class I mediated induction of FGFR expression and subsequent EC and SMC proliferation.

Abstract# 133

INTRAGRAFT GENE EXPRESSION IN ACUTE REJECTION OF KIDNEY ALLOGRAFTS. Patrick G. Dean,[1] Mark D. Stegall,[1] David I. Schwartz,[1] Anis Khair,[1] Timothy S. Larson,[1] Jorge A. Velosa.[1] [1]*The Transplant Center, Mayo Clinic and Foundation, Rochester, MN.*

Acute cellular rejection (AR) is a complex process involving both infiltrating host immune cells and the response to this inflammation by the graft. While certain aspects of allograft rejection have been described in detail, the recent development of large gene chip arrays allows for a broader analysis of intragraft gene expression. The aim of the current study was to study intragraft gene expression during AR with special emphasis on gene expression "profiles" and genes not commonly associated with rejection.

Methods. Core kidney biopsy tissue was obtained from 10 patients 7-10 days after kidney transplantation for elevations in serum creatinine. Baseline immunosuppression for all patients consisted of tacrolimus, mycophenolate mofetil, and prednisone. No patient received induction antibody therapy. High-density gene expression arrays (HuGeneFL GeneChip; Affymetrix, Santa Clara, CA) were utilized to determine relative intragraft gene expression profiles in these biopsy samples. Total RNA was extracted from snap frozen biopsy samples, reverse transcribed, labeled with biotin and fragmented according to established protocol. Following hybridization onto the HuGeneFL GeneChip, samples were stained, washed, and scanned. Statistical analyses were performed using the log-average ratio and Wilcoxon rank sum methods. Those values with a p value of less than 0.05 were considered significantly changed.

Results. Five patients were found to have AR and 5 had no evidence of rejection. Of the 5,600 unique genes studied, 252 (4.5%) were upregulated, 198 (3.5%) were downregulated, and 5,150 (92.0%) were not significantly changed in patients with AR compared to those without AR. Significant elevations in RNA levels for IL-4, IL-8, $\alpha 4$ integrin and Fas ligand were seen in patients with AR. Interestingly, significant alterations in gene expression were seen for 113 genes that are seemingly unrelated to rejection [signal transduction (n=34), cellular metabolism (n=33), oncogenes (n=46)].

Conclusions. A "profile" of inflammatory gene expression was demonstrated in the patients with AR. However, the results using the GeneChip differed slightly from those previously described in AR utilizing quantitative PCR (no increase in granzyme B or perforin). The GeneChip microarray provides a powerful tool for the analysis of acute rejection. Further investigation into the seemingly unrelated genes may provide new insights into the mechanisms of acute rejection.

Abstract# 134

MICROARRAY EXPRESSION ANALYSIS OF ACUTE REJECTION IN HUMAN PANCREAS ALLOGRAFTS. Lynn M. Jacobson, Roger W. Sands, Jon S. Odorico. [1]*Department of Surgery, Division of Multiorgan Transplantation, University of Wisconsin-Madison, Madison, WI.*

A better understanding of pancreas allograft rejection would advance our ability to diagnose and control the rejection process. To examine the molecular events associated with rejection, we used oligonucleotide microarrays to define the intragraft transcriptional changes occuring during acute rejection in human pancreas allograft biopsies.

Methods: Total RNA was isolated from human pancreas allograft core tissue biopsies (n=2), which showed acute Maryland Grade II-III rejection histologically. RNA was then purified, reverse transcribed, labeled, and hybridized to Affymetrix Gene Chip® oligonucleotide microarrays containing probes to 12,000 full-length genes from the Unigene database. mRNA transcript levels were quantified and compared to those of biopsies from normal pancreata flushed with UW solution prior to transplantation (n=3). A comparison of the normalized data was based on log odds (lod) ratio; a score of ≥2 was considered significant. The sensitivity of detection is ≈1:100,000-300,000 transcripts or ≈1 copy/cell.

Results: A comparison of core tissue biopsies from acutely rejecting and normal pancreatic allografts revealed that rejection is associated with profound alterations in mRNA levels which may reflect changes in gene expression (range: 1-199 fold change). Of the 12,626 genes surveyed, 2287 (18%) were differentially expressed (lod score ≥2). 1132 (8.9%) mRNA transcripts were found to be more abundantly or newly expressed in rejecting pancreases, whereas the expression of 1155 (9.1%) genes was down-regulated in rejection samples compared to normal samples. The expression of a variety of cytokine regulatory pathway, adhesion molecule, MHC antigen, immunoglobulin, complement pathway, and apoptosis pathway genes were upregulated during pancreas rejection. In contrast, mRNA transcripts of exocrine enzyme genes (such as α-amylase, triglyceride lipase, RNAse), endocrine hormone genes (such as peptide YY), ductal genes (mucin), and regenerating protein (reg) genes were less abundant in rejection samples than in normal controls.

Conclusion: This study demonstrates that an RNA expression analysis using oligonucleotide-based microarrays is able to reveal a unique fingerprint of gene transcription for acute pancreas allograft rejection in humans. This methodology could be used to elucidate candidate markers for rejection or to potentially differentiate rejection from other intragraft pathology.

CONCURRENT SESSION 15:
TRANSPLANTATION: ALLOCATION

Abstract# 135

TRANSPLANTATION WITHOUT A PREOPERATIVE FINAL CROSSMATCH-IT CAN BE DONE IN SELECTED CIRCUMSTANCES. Arthur J. Matas,[1] Angelika Gruessner,[1] David E.R. Sutherland.[1] [1]*Surgery, University of Minnesota, Minneapolis, MN.*

Given the constant flux in caseload and the number of personnel available in the OR, waiting for a final XM often prolongs organ preservation time (a room available at the time a XM is started is not available when the XM is completed). Longer preservation is associated with increased DGF (and potentially the associated decreased graft survival). We have shown, in a retrospective analysis, that final XMs on 0% PRA recipients were always negative (Transplantation, 1999). We now describe a policy of: a) not doing screening XM and b) proceeding to the OR without a XM, in situations where the recipient's PRA has been documented to be 0% and when there have not been any interim transfusions (and the OR is ready before XM completion). Final XM is completed after the transplant.

All patients send sera every 6 weeks for PRA (antiglobulin technique). If ≥3 consecutive PRAs are 0%, no donor-specific screening XM is done prior to calling the patient in for tx (UNOS allocation algorithm used). If there have not been any interim transfusions (and an OR is available), we proceed to tx prior to completion of the final XM.

Between 1/1/98-12/31/99, we did 109 CAD kidney (K) and 79 simultaneous kidney pancreas (SPK) tx; 67 (61%) K and 56 (71%) SPK had 0% PRA. Of the 0% PRA, 25/67 (37%) K and 28/56 (50%) SPK had no pretx XM. For K with no XM, cold ischemia was shorter (13.2±.2 vs. 18±.9 hrs, p=.01) and DGF less (12% vs. 24%, p=.3); for SPK with no XM, cold ischemia was shorter (15±2 vs. 18±.9 hrs, p=.1); no diff in DGF. All post-XM were negative and there were no hyperacute rejections; there was no difference in posttransplant acute rejection episodes. Actuarial 1 yr graft survival. no pretransplant XM-K=87.5%, SKP=82%; Yes XM-K=88%, SKP=86% (NS). Our data suggests it is safe, in select circumstances, to proceed to the OR without a XM. Elimination of the screening XM for 0% PRA candidates saves money. Proceeding to the OR (if available) without a final XM shortens cold ischemia time.

Abstract# 136

THE ADVANTAGES OF SHARING ZERO HLA-MISMATCHED CADAVERIC KIDNEYS. Mark D. Stegall,[1] Patrick G. Dean,[1] Maureen A. McBride,[1] James J. Wynn.[1] [1]*The UNOS Combined Subcommittee on Kidney/Pancreas Allocation.*

Purpose. The beneficial effects of national sharing of zero antigen-mismatched (0-MM) cadaveric kidneys are controversial. The aim of this study was to analyze the effects of mandatory sharing on graft survival, cold ischemia time (CIT) and the transplantation of highly sensitized patients.

Methods. Using the UNOS/OPTN Transplant Database, we performed three analyses of the 0-MM sharing system. First, a multivariate analysis was performed on all solitary cadaveric kidney transplants between July 1, 1993 and June 30, 1996 (n=22,754) to determine the odds ratio of graft failure according to HLA mismatch and share type Second, during the same time period, we compared the survival of paired kidneys in which one was shared as either a 0-MM kidney (n=767) or as a payback kidney (n=520). Third, we analyzed the effect of 0-MM sharing on the transplantation of sensitized patients (PRA>60%) during the period from March 6, 1995 to December 31, 1998.

Results. During the latest time period, 33% of all cadaveric kidneys were shared nationally (16% for 0-MM and 17% as paybacks and other shares). The odds-ratio of 3-year graft failure for nationally shared 0-MM kidneys was significantly lower than less well-matched kidneys used locally. Sharing 0-MM kidneys increased the mean CIT versus other MM used locally (23.9+/-0.2 hrs vs 20.0+/-0.1 hrs). In the analysis of paired kidneys, 0-MM sharing increased 3-year graft survival over the paired kidney used locally (83 vs. 78%, respectively; p<0.01). Importantly, the "payback" kidney had the same three-year graft survival as the paired kidney used locally despite an increased CIT. 0-MM sharing significantly increased the chances of transplantation in patients with a PRA>60% comprising 40% of the transplants in this group compared to only 15% in less-sensitized patients. Interestingly, the graft survival of sensitized patients was not significantly improved using 0-MM kidneys (73% for 0-MM vs. 66% for non-0-MM at 3 years).

Conclusions. The current national system of sharing 0-MM kidneys improves graft survival without excessively prolonging CIT. Sharing does not decrease the graft survival of "payback" kidneys. 0-MM sharing provides almost half of the kidneys for highly sensitized patients without substantially improving graft survival. For these reasons, we believe that current national sharing system should be preserved.

Abstract# 137

ZERO-MISMATCHED KIDNEYS FROM EXPANDED DONORS: IS THE BENEFIT WORTH THE RISK? Maureen A. McBride,[1] Wida S. Cherikh,[1] James J. Wynn,[2] Kidney and Pancreas Transplantation Committee.[1] [1]*United Network for Organ Sharing, Richmond, VA;* [2]*Department of Surgery, Medical College of Georgia, Augusta, GA.*

Background: Recipients of zero antigen mismatched (0MM) cadaver kidneys continue to enjoy improved graft survival when compared to other cadaver kidney recipients. However, the extent to which adverse donor characteristics may abrogate the beneficial effects of excellent HLA matching has not been well studied. As a result, we assessed the impact of various donor factors on renal allograft outcomes, focusing on interactions of non-immunologic donor factors with the degree of HLA match and particularly on the effect of donor factors on 0MM transplants.

Methods: Using the UNOS/OPTN transplant database, we analyzed the outcomes of 33,266 cadaver donor renal transplants performed between 3/6/95 and 6/30/99. Multivariate analyses were performed and predicted outcomes estimated using Cox proportional hazards regression. Differences in proportions were analyzed using the Chi-square test.

Results: 5,209 (15.7%) kidneys were transplanted in 0MM recipients. Of those, 12.7% were recovered from donors ≥55 years old and 89% of 0MM kidneys were shared. Hypertension (HTN) occurred significantly more frequently in older (≥55) than younger (<55) donors of 0MM kidneys (42.1% vs 12.2%), as did death from cerebrovascular disese (78.3% vs 30.5%) and the combination thereof (36.1% vs 9.0%). Multivariate analysis of 0MM recipients revealed that donor age (relative risk [RR] 1.14/10 years), Black race (RR 1.62), and Hispanic ethnicity (RR 1.37) were highly significantly associated with increased risk of graft loss. There was a significant interaction of increased donor age, history of HTN and increased cold ischemia time (CIT): increased CIT had little impact in younger donors regardless of HTN but led to marked increases in the risk of graft failure in older donors, with the effect being more pronounced in those with HTN. When analyzing the entire group, kidneys from donors possessing adverse risk factors continue to achieve the greatest graft survival benefit when transplanted in very well matched recipients.

Conclusions: Kidneys from older hypertensive 0MM donors are at significantly increased risk of graft loss, especially in the presence of prolonged CIT. Although consideration should perhaps be given to sharing these kidneys only in circumstances of special benefit such as elevated recipient PRA, such deliberations should acknowledge the continued beneficial effect of HLA matching of kidneys from expanded donors.

Abstract# 138

WHAT ARE YOUR CHANCES OF GETTING A ZERO-ANTIGEN MISMATCHED KIDNEY? Harish D. Mahanty,[1] Calvin D. Lou,[1] George J. Chang,[1] John P. Roberts,[1] Lee Ann Baxter-Lowe.[1] [1]*Surgery, University of California, San Francisco, San Francisco, CA.*

Background. Six antigen matched kidney transplants afford the best long-term graft and patient survival. However, zero antigen mismatched (0 ag mm) kidney transplantation approaches graft and patient survival seen in 6 antigen matched transplants giving rise to the practice of nationally sharing 0 ag mm kidneys. The purpose of this study was to determine which patients on our cadaveric kidney transplant waiting list (CWL) have a high probability of receiving a 0 ag mm organ. **Methods.** The UNOS donor kidney census from 1989 to 1998 was used to determine the national frequency of donation practices by race. Haplotype frequencies categorized by race were obtained from Mori et. al. and, in combination with the UNOS data, were used to calculate a race-weighted probability for a patient on our CWL to receive a 0 ag mm organ. **Results.** 78% of the donated kidneys in the nation were of Caucasian(C), 10% of Black(B), 8% of Hispanic (H),1% of Asian (A) and 0.6% of Other (O) origin. Our CWL consisted of 2061 patients of which 42% were C, 18%B, 19%H, 17%A and 4%O. The probability of these patients receiving 0 ag mm kidneys ranged from 0.0% to 0.8%. Approximately 30% of the patients in the first quartile were heterozygous for the A1,B8,DR3 haplotype. Of the first quartile of candidates in our CWL predicted to receive a 0 ag mm organ 70% were C, 9% were B, 3% were A, 16% were H and 2% were O. Asians ranked highest in the fourth quartile (33%) followed by Blacks (30%). Hispanic candidates were most prevalent in the second quartile (28%). **Conclusion.** Patients who are heterozygous for the A1,B8,DR3 haplotype regardless of race had the highest probability of receiving a 0 ag mm organ. Predicting which patients will have a high probability of receiving 0 ag mm kidneys will allow for efficient management of kidney transplant waiting lists in terms of counseling patients for donor choices. An analogous analysis can be made to predict the probability that kidneys procured in a region will be shared. Finally, this research underscores the need for public awareness, especially in minority populations, for organ donation.

Abstract# 139

GRAFT SURVIVAL OF KIDNEYS FROM A /A B DONORS INTO B PATIENTS IS EQUIVALENT TO ABO COMPATIBLE (B AND O → B) TRANSPLANTATION. Nicolas A. Muruve, Bradley A. Warady, Mark I. Aeder, Daniel Murillo, Paul W. Nelson, Charles F. Shield, III, Christopher F. Bryan. [1]*Midwest Transplant Network, Westwood, KS.*

With the increased interest in transplantation of A and A B kidneys into B patients and the possible implementation of a national voluntary variance by which transplant centers in an OPO may allocate A and A B cadaveric kidneys to B patients, it is important to know that outcome is equivalent to that of ABO compatible transplantation within the B blood group. In the present study, we compared five-year graft survival outcome for 40 A /A B into B transplants with that of 80 B/O into B transplants. The majority of these patients were transplanted with a documented history of low anti-A IgG titers (<8) before transplantation. These transplants were done concurrently by the seven transplant centers in our OPO from 1994 through September, 2000 in accordance with our OPO's UNOS-approved variance to preferentially allocate A / A B kidneys to B patients. Graft survival data (patients who died with a functioning graft censored from further analysis) are shown in the following table.

		Graft Survival (Years)			
ABO Group	**Number**	**1**	**3**	**5**	**Log-rank**
A /A B → B	40	91%	84%	84%	
B/O → B	80	91%	83%	78%	0.44

These data show that the short (1 year) and more long-term (3 and 5 year) graft survival of A and A B cadaveric kidneys transplanted into B patients is equivalent to that of B patients who were transplanted with B or O kidneys. Finally, one-third of the 120 B candidates transplanted in this 6.75 year period received A or A B kidneys.

Conclusion: These data show that B recipients of A and A B cadaveric kidneys who have low anti-A titers, have graft survival that is equivalent to ABO compatible transplants. These data support the national application of A /A B → B cadaveric transplants in an effort to increase access of B waiting list candidates to kidneys and thereby their rate of transplantation.

Abstract# 140

THE FIRST TWO YEARS OF A REVISED SCHEME FOR ALLOCATING CADAVER KIDNEYS IN THE UK. R. J. Johnson, S. Armstrong, M. A. Belger, J. D. Briggs, P. J. Morris. [1]*on behalf of the UK Transplant Kidney and Pancreas Advisory Group, UK Transplant, Bristol, United Kingdom.*

A revised Kidney Allocation Scheme was introduced in the UK in July 1998 based on HLA matching at three levels: 000 mismatches, favourable matches (i.e. 100, 010 and 110 HLA-A, B, DR mismatches) and non-favourable matches (all other matches). Within these levels children and local patients receive priority and any ties are sorted on six points scoring factors: recipient age, donor-recipient age difference, matchability (based on HLA tissue type, unacceptable antigens and blood group), waiting time, sensitisation to HLA antigens and transplant centre import and export balance. To assess the effectiveness of the revised scheme, results of the first two years have been compared with those of the last 18 months of the previous scheme. There have been significant improvements in HLA matching for adults and children (p<0.0001 and

p<0.003, respectively), achieved through greater exchange of organs between centres. The proportion of 000 mismatched grafts has increased from 7% to 13% for adults and from 5% to 13% for children. There has also been a threefold increase in the number of 000 mismatched grafts for highly sensitised patients (HSP). For those kidneys allocated to adults through the national Scheme there have been some changes with regard to the points scoring factors. Firstly, transplanted recipients were significantly younger than previously (p<0.01). This was not an objective of the new Scheme and is a trend that will be carefully monitored. Secondly, the mean donor-recipient age difference has decreased by 2 years suggesting an effect over and above the trend of increased mean donor age. Also, patients who are moderately difficult to HLA match have received proportionally more transplants at the expense of those who are easiest to match (p<0.03). The median waiting time of adults receiving nationally allocated kidneys has continued to increase, namely from 336 days (IQ range, 135-669) to 439 days (IQ range, 173-910), (p<0.0001). It is not clear whether patients who have waited a long time have benefited through points scoring. In conclusion, the new UK Kidney Allocation Scheme has been associated with improved HLA matching for adults and children and for both first graft and regraft recipients; a threefold increase in the number of HSP 000 mismatched grafts; younger adults receiving kidneys and a decrease in donor-recipient age differences. Finally, matchability points scoring may have helped to achieve an increase in transplants for patients who are moderately difficult to HLA match.

Abstract# 141

DONOR/RECIPIENT AGE MATCHING IN RENAL TRANSPLANTATION. John S. Gill,[1] Dana Miskulin,[1] Brian J.G. Pereira,[1] David N. Landsberg.[2] *[1]William B. Schwartz Division of Nephrology, New England Medical Center, Boston, MA; [2]Nephrology, St. Paul's Hospital, Vancouver, BC, Canada.*

To examine the effect of donor/recipient age matching on actual and death censored graft survival.

METHODS: Retrospective cohort study of 816 first cadaveric renal allografts in British Columbia performed 1985-98. Donor age was categorized as young =18-50 years, and old > 50 years. Recipient age was categorized as young =18-55 years and old > 55 years. Four donor/recipient (D/R) groups were created: YD/YR n=439; OD/YR,n=115; YD/OR,n=207; OD/OR n=52. Survival estimates were stratified across D/R groups. We assessed the effect of D/R age groups on patient and graft survival (actual and death censored) using separate Cox Proportional Hazards models adjusting for HLA mismatches, cold ischemic time, cause of ESRD, and recipient race.

RESULTS: Median follow up was 89 months; 28% had graft loss, 27% died. In YR the time to 75% patient survival was > than the time to 75% graft survival: YD/YR 135 months(patient survival) vs. 98 months(graft survival); OD/YR 106 months(patient survival) vs. 31 months(graft survival). However, in OR the time to 75% patient survival was < graft survival: YD/OR 45 months (patient survival) vs. 66 months(graft survival); OD/OR 78 months(patient survival) vs. 130 months(graft survival). The multivariable models are shown (table). An interaction of donor and recipient age was not significant. Recipient age, but not donor age predicted patient survival.

CONCLUSIONS: Increased donor age, but not recipient age was independently associated with graft loss. OD/YR had the highest risk for graft loss in the death censored model; however, there was no interaction between donor and recipient age, thus this represents the effect of donor age. Donor age did not influence patient survival. In YR, patient survival was > graft survival; however, in OR, graft survival was > patient survival. Our results suggest consideration of age matching to maximize functional allograft survival of cadaveric organs.

		DEATH CENSORED GRAFT LOSS		
	YD/YR	OD/YR	YD/OR	OD/OR
HAZARD RATIO	1 2	2 8	Reference	1 7
95% CI	0 8-1 8	1 8-4 4		0 9-3 2
		ACTUAL GRAFT LOSS		
HAZARD RATIO	Reference	2 1	1 5	2 5
95% CI		1 5-2 8	1 2-2 0	1 7-3 7

Abstract# 142

KIDNEY ALLOGRAFT SURVIVAL AND HLA CLASS I MATCHING AT THE AMINO ACID TRIPLET LEVEL. Rene J. Duquesnoy,[1] Steve Takemoto.[2] *[1]University of Pittsburgh Medical Center, Pittsburgh, PA; [2]UCLA, Los Angeles, CA.*

HLAMatchmaker is a newly developed computer-based algorithm for identifying acceptable HLA mismatches for highly alloimmunized patients. This algorithm is based on the concept that immunogenic epitopes are represented by amino acid triplets on exposed parts of protein sequences of HLA-A, B, C chains accessible to alloantibodies. Donor HLA compatibility is determined by intralocus and interlocus comparisons of polymorphic triplets. For most patients, this algorithm can identify certain mismatched HLA antigens that are zero-triplet mismatches to the patient's HLA phenotypes and must therefore, be considered fully histocompatible at least at the humoral level of allosensitization. Moreover, antibody specificity analyses of high PRA sera have permitted a distinction of 55 polymorphic triplets with relatively high immunogenicity while the remaining 70 triplets have low immunogenicity.

The present study was designed to determine whether class I HLA matching at the triplet level affected kidney transplant outcome. The analysis was done with 87361 cadaveric kidney allograft recipients transplanted during 1987-1999 and reported to the UNOS Registry. Patients with none of the 125 triplets mismatched and 0-DR mismatches (N=196) had similar 3-year survival (79%) and half-life (11.9 years) as the 0-A,B,DR mismatched recipients (81%, 12.5 years), significantly higher than those with DR or triplet mismatches (71%, 8.7 years, P<0.001).

In an analysis of 10,690 cadaveric renal allograft recipients with PRA >50%, patients with 0-2 high immunogenicity triplet and 0-DR mismatches (N=375) had 72% 3-year survival and a 11.9 year half-life similar to sensitized 0-A,B,DR mismatched patients (77%, 11.6 years) and significantly higher than those with other HLA mismatches (62%, 8.0 years).

In conclusion, while HLAMatchmaker is designed to identify crossmatch-negative donors for highly sensitized patients, these findings suggest that HLA matching at the triplet level benefits kidney transplant outcome and increases the pool of histocompatible donors.

Abstract# 143

RENAL TRANSPLANTATION FOLLOWING BONE MARROW TRANSPLANTATION: PATIENT OUTCOMES AND NEED FOR IMMUNOSUPPRESSION. Khaled Hamawi,[1] J. Andrew Bertolatus.[1] *[1]Internal Medicine, University of Iowa, Iowa City, IA.*

Background: Bone marrow transplantation (BMT) is a rapidly expanding therapy for hematologic malignancies and other disorders.Currently over 1200 BMT are performed in the US each year.Chronic renal failure(CRF) secondary to BMT nephropathy(BMTN) is an increasingly recognized complication,which occurs in ≈ 25% of BMT survivors after 2 years.However few reports address the graft and patient outcomes after kidney transplantation(KT).In addition, there is some uncertainty concerning the need for immunosuppression when the kidney is obtained from the marrow donor.

Methods: From 1983-2000,9 patients with CRF after BMT underwent KT at our institution.Records were reviewed to determine patient demographics,clinical course and outcomes.Patients who received KT from the marrow donor were given no long-term immunosuppression therapy,while the others received standard institutional immunosuppression protocol.

Results: Etiology of renal failure was thought to be BMTN in all cases (biopsy proven in 1 case,clinical diagnosis in 8 cases).Five patients received kidney from the marrow donor,2 from a living unrelated donor and 2 from a cadaver donor.Age at the time of BMT ranged from 7-52 yr (median 39yr).Age at the time of KT ranged from 9-55yr(median 45yr).Follow up period(after KT) was 8 to 105 months(median 35).Of the 5 patients receiving KT from the marrow donor: two died after 35 and 38 months secondary to interstitial lung disease and sepsis respectively.Three patients are alive and on no immunosuppression therapy after 29-60 months of f/u.Two patients who received KT from unrelated donor are alive after 8and 30 months of f/u with standard immunosuppression.Two patients who received a cadaveric KT died after 10 and 105 months secondary to aspergillus infection and myocardial infarction.In the entire study no patient had clinically suspected or biopsy proven renal allograft rejection,regardless of the source of the kidney,including those receiving no long-term immunosuppression treatment.All allograft losses were due to death.Median SCr at the time of death or last f/u was 1.2 mg/dl.

Conclusions:KT after BMT is a viable treatment option for patients with BMTN with a median graft and patient survival of 38 months.Patient deaths (likely related to immunosuppression and chemotherapy) were the only cause of graft loss.Patients who receive a kidney from the marrow donor require no immunosuppression therapy.

CONCURRENT SESSION 16:
SIROLIMUS IN KIDNEY TRANSPLANTATION

Abstract# 144

TWO-YEAR SAFETY AND EFFICACY OF SIROLIMUS IN RENAL TRANSPLANTATION. Barry D. Kahan, for the Rapamune US and Global Study Groups. *[1]Division of Immunolgy & Organ Transplantation, University of Texas-Houston, Houston, TX.*

Purpose: Sirolimus (SRL) has been shown to significantly reduce the incidence of acute rejection episodes in renal allograft recipients at 6 months post-transplant. We report the long-term (24-month) results of two multicenter, randomized, double-blind studies comparing the safety and efficacy of SRL with azathioprine (AZA) and with placebo (PLA) in renal transplant recipients.

Methods: In two phase III clinical trials, 1295 patients were randomly assigned to receive either SRL 2 mg/day or 5 mg/day and AZA (US Study) or PLA (Global Study), in addition to cyclosporine and prednisone. Acute rejection, patient and graft survival rates, and safety parameters were evaluated at 24 months post-transplant.

Results: Intent-to-treat analysis of acute rejection (AR), patient survival (PS), graft survival (GS), creatinine, and Nankivell GFR at 24 months is described in the table below. SRL-treated patients experienced lower rates of acute rejection compared to those treated with AZA or PLA. Dose-related elevations of cholesterol and triglycerides were evident among treatment groups but improved over time and responded well to standard therapy. There was no difference in the rates of infection except for an increase of herpes simplex among patients treated with SRL 5 mg/day. Similarly, the rates of malignancy at 24 months were not significantly different between groups.

Conclusion: Sirolimus is safe and efficacious for long-term immunosuppression in renal transplant recipients. Sirolimus significantly reduces biopsy-confirmed acute rejection rates and provides comparable rates of patient and graft survival at 24 months.

	US Study			Global Study		
	SRL 2 mg/d	SRL 5 mg/d	AZA	SRL 2 mg/d	SRL 5 mg/d	PLA
AR (%)	24 6	17 9 *	32 9	30 0*	26 0***	43 8
PS (%)	94 6	94 8	96 9	93	94	92
GS (%)	86 4	88 6	90 0	85	87 5	83
Creatinine (mg/dL)	1 82*	2 11***#	1 52	1 78	2 17***##	1 60
GFR (mL/min)	62 35	54 61***##	66 88	59 71	51 75***##	64 01

*p<0 05, **p<0 01, ***p<0 001 vs AZA or PLA, #p<0 05, ##p<0 01 vs SRL 2 mg/d

Abstract# 145

OUTCOME OF 300 RENAL TRANSPLANT RECIPIENTS TREATED *DE NOVO* WITH A SIROLIMUS-CYCLOSPORINE REGIMEN AT A SINGLE CENTER. Barry D. Kahan,[1] Stephen M. Katz,[1] Richard J. Knight.[1] *[1]Division of Organ Transplant, University of Texas-Houston, Houston, TX.*

Purpose: We compared the outcomes of two overlapping cohorts of renal transplant recipients: 300 treated with sirolimus (SRL)-cyclosporine (CsA)±prednisone (Pred; with Pred=189, no Pred=111) *vs* 118 with CsA+Pred (n=118). **Methods:** The SRL regimen utilized doses of 1-10 mg/day to maintain trough concentrations at 5-15 ng/mL. The CsA regimen used area under the curve (AUC) concentrations to adjust drug doses such that the SRL-CsA group received 30-50% less CsA AUC exposure than the CsA-Pred group. **Results:** The two groups showed similar demographic features save for a significantly greater fraction of cadaveric recipients, a greater body weight, a higher number of HLA mismatches, and higher % PRA among the SRL-treated patients. Compared to the CsA-Pred regimen alone, patients treated with SRL displayed significantly reduced incidences of both acute rejection episodes (ARE) and biopsy-proven processes of chronic rejection (CR) at 2 years (Table 1). The respective reductions in ARE and CR were more evident for cadaver *vs* living donor transplants and for the 85% primary but not for the 15% repeat transplants (Table 1). Among the complications, SRL-CsA regimens were associated with decreased incidences of cytomegalovirus and headache adverse events, and increased incidences of lymphocele and wound infection. Over the course of the first 2 years, serum lipids were consistently elevated and formed cellular elements decreased in the SRL-CsA group, but the rejection-censored serum creatinine values were not significantly different (SRL-CsA±Pred 1.77, 1.70, 1.72, 1.78 *vs* CsA-Pred 1.69, 1.74, 1.83, 1.72 at 6, 12, 24, and 36 months). **Conclusions:** SRL conveys clear therapeutic benefits at 2 years to reduce the incidence of both acute and chronic rejection.

Demographic		ARE			CR	
Feature	CsA-Pred	SRL-CsA	"p"	CsA-Pred	SRL-CsA	"p"
Overall	29 5%	10 0%	0 0001	17 0%	05 4%	0 0001
Caucasians	22 0%	08 0%	0 02	07 3%	03 2%	NS
African-Americans	42 9%	15 5%	0 002	23 8%	09 9%	0 04
Cadaver	46 3%	11 5%	0 0001	22 0%	05 5%	0 002
Living Donor	20 8%	07 9%	0 009	14 3%	05 3%	0 030
Primary Transplants	29 7%	09 2%	0 0001	17 8%	04 8%	0 001
Re-transplants	29 4%	15 2%	0 18	11 8%	08 7%	0 52

Abstract# 146

SIROLIMUS AND CARDIOVASCULAR RISK FACTORS IN A CYCLOSPORINE ELIMINATION TRIAL. Francesco Paola Schena, the Rapamune Renal Function Study Group. *[1]Istituto di Nefrologia, Azienda Ospedaliera Policlinico Conzorziale, Bari, Italy.*

Background: Sirolimus (SRL) therapy is associated with moderate elevations in serum cholesterol and triglycerides that respond to standard lipid-lowering treatments. This study examined cardiovascular risk factors in patients receiving SRL-cyclosporine (CsA)-steroids continuously or SRL-steroids after CsA withdrawal at month 2.

Methods: In this open-label randomized study, 197 first or second cadaveric renal transplant recipients were randomized to full-dose CsA (microemulsion) plus fixed-dose SRL (2 mg/d) [Group A, n=97] or reduced-dose CsA plus concentration-controlled SRL (10-20 ng/mL) [Group B, n=100] at <7d after transplantation. All patients received standard corticosteroids. During the third month after transplantation, patients in Group B who did not have an acute rejection had CsA tapered and eliminated.

Results: Concentration control of SRL involves the use of significantly higher doses and higher trough blood levels. At 12 months, intent-to-treat analyses showed that graft survival (92.8% vs 94.0%), patient survival (96.9% vs 95.0%), and acute rejection (21.7% vs 21.0%) were not statistically different between Groups A and B, respectively. Of patients in Groups A and B, 2 (2.1%) and 2 (2.0%), respectively, experienced a fatal cardiac event. 5 (5.2%) patients in Group A and 8 (8.0%, p=NS) in Group B had treatment-emergent insulin-dependent diabetes. There were no significant differences in lipid values between groups (see Table). HDL-cholesterol levels were normal or elevated in 92% of assayed patients, notably, 23% and 31% of patients in Groups A and B, respectively, had elevated HDL-C levels. The use of lipid-lowering agents was similar in each group. The incidence of hypertension in Group B (31%) was significantly lower than in Group A (46%, p<0.05). Systolic blood pressure was significantly lower in Group B patients compared with Group A (p<0.05).

Conclusions: In patients who underwent CsA elimination, there were no adverse effects on hyperlipidemia, blood pressure, or diabetes, despite the use of higher SRL doses. HDL-C levels were normal or elevated in most patients. The effect of SRL maintenance therapy on long-term cardiovascular risk is therefore felt to be minimal in this relatively high-risk population.

Treatment	Total Cholesterol (mM)	Triglycerides (mM)	LDL-C (mM)	HDL-C (mM)	DBP (mm Hg)	SBP (mm Hg)
Group A	6 06±0 20	2 61±0 24	3 54±0 16	1 36±0 06	83 8±1 7	142 8±3 0
Group B	6 80±0 22	3 10±0 26	3 70±0 16	1 49±0 06	80 0±1 8	132 9±3 0

Abstract# 147

A PROSPECTIVE RANDOMIZED TRIAL OF SIROLIMUS VS. CYCLOSPORINE IN KIDNEY TRANSPLANTATION: IMPACT OF CALCINEURIN INHIBITOR ELIMINATION ON RENAL FUNCTION. Stuart M. Flechner,[1] David A. Goldfarb,[1] Charles Modlin,[1] Barbara Mastroianni,[1] Kathy Savas,[1] Venkatesh Krishnamurthy,[1] Daniel J. Cook,[1] Andrew C. Novick.[1] *[1]Urological Institute, Cleveland Clinic Foundation, Cleveland, OH.*

Purpose: Although nephrotoxic, calcineurin inhibitor drugs (CNI) have remained the primary immunosuppressive agents for kidney transplantation. The introduction of the antilymphocytic agent sirolimus provides the opportunity to develop protocols free of any CNI agents.

Methods: Thirty eight (of 60 planned) adult recipients of a primary HLA mismatched renal allograft were computer randomized to receive either concentration controlled sirolimus (Srl) or cyclosporine (CsA), and mycophenolate mofetil 1 gm bid, and steroids. Each recipient was given induction therapy with basiliximab 20mg on day 0 and 4. Trough blood level monitoring was used to keep CsA at 200-250 ng/ml and Srl at 10-12 ng/ml the first 6 months.

Results: The Srl group included 20 (12CD, 8LD) patients with a mean F/U 4 35 (1-8) mo. and the CsA group 18 (10CD, 8LD) mean F/U 4.27 (1-8) mo. There were no significant demographic differences in mean age (46.6, 46.2) years, gender M:F (10:10, 11:7), % diabetics (25, 33), or % black recipients (25, 22) between the groups, respectively. Among the CD recipients, 33% Srl and 36% CsA experienced delayed graft function. Patient and graft survival at 3 and 6 mo. has been 100% for each group. No Srl patients and one (6%) CsA patient experienced biopsy confirmed acute rejection. At 3 and 6 mo. the mean doses of Srl (mg/day) were (6 4, 6.25) and CsA (mg/kg/day) were (3.76, 3.66). Renal function was significantly (p<.001) better in the Srl patients; 3 and 6 mo. Scr mg/dl (1.24, 1.21) and Cockroft-Gault GFR ml/min (78.1, 81.8) vs. CsA Scr (1.82, 1.91) and GFR (66.9, 58.4). Mean total cholesterol (mg/dl) at 3 and 6 mo. (Srl 258, 265 vs CsA 260, 238) was not significantly different, while triglycerides mg/dl (Srl 335, 331 vs. CsA 211, 180) were greater (p<.01) in the Srl group.

Conclusion: Maintenance therapy with sirolimus, mycophenolate and steroids following basiliximab induction provides excellent rejection prophylaxis without the need for CNI for the first 6 months. Renal function is up to 30% better in kidneys spared exposure to any CNI agent. The significance of sirolimus toxicity profiles and dose adjustments await longer followup and analysis.

Abstract# 148

REMARKABLY LOW RATE OF ACUTE RENAL ALLOGRAFT REJECTION IN AFRICAN AMERICANS RECEIVING SIROLIMUS, LOW-DOSE TACROLIMUS, AND CORTICOSTEROIDS: PRELIMINARY RESULTS OFA PILOT STUDY. Hany H.S. Anton,[1] Thomas C. Knauss,[1] David Seaman,[2] Christopher Siegel,[2] James A. Schulak,[2] Donald E. Hricik.[1] *[1]Medicine, University Hospitals of Cleveland, Cleveland, OH; [2]Surgery, University Hospitals of Cleveland, Cleveland, OH.*

African American (AA) renal transplant recipients (RTRs) generally exhibit higher rates of rejection and lower rates of graft survival than non-AAs. We have enrolled 30 primary AA RTRs into a pilot study using immunosuppression consisting of sirolimus (15 mg load followed by doses targeted to trough levels of 10-20 ng/ml), tacrolimus (targeted to levels of 5-8 ng/ml), and steroids. The incidence of acute rejection and other outcomes were compared to those in a concurrent group of 29 primary non-AA RTRs treated with mycophenolate mofetil (2 gm/day), tacrolimus (targeted to levels of 8-12 ng/ml) and steroids. Induction antibodies were used in 3 of the 31 AAs and in 3 of 29 non-AAs. Comparing AAs to non-AAs, the groups were similar in age (46±15 vs 54±13 years), incidence of delayed graft function (11 vs 19%), frequency of living donors (30 vs 24%), and number of HLA mismatches (3.9±1.5 vs 3.8±1.8)(p=NS). There were more women in the AA group (48 vs 19%; p<0.01). After a mean follow-up of 25 weeks for each group, the incidence of acute rejection was 1/30 (3%) in the AA group and 5/29 (17%) in the non-AA group (p=0.001). Nonimmunologic graft losses occurred in 2 patients in each group. A single death occurred in a non-AA patient with ischemic colitis. The incidence of posttransplant diabetes mellitus (PTDM) was higher in AAs (18 vs 3%); p<0.001). There was a trend toward higher rates of hospital readmission in AAs (51 vs 40%; p=NS). The most common reason for readmission in the AA group was uncontrolled hyperglycemia. Serious infections (requiring readmission)occurred in 3 AAs and 1 non-AA (p=NS). 3 months posttransplant, trough tacrolimus levels were lower in AAs (5 0+2.1 vs 8.3±3.5 ng/ml, p<0.05) while serum creatinine concentrations were similar (1.4±.4 vs 1.3±.4 mg/dl; p=NS). Results from this pilot study demonstrate a remarkably low incidence of acute rejection in AA patients receiving no induction antibody in most cases, and maintenance therapy with sirolimus, tacrolimus, and steroids. Despite lower target tacrolimus levels, use of this regimen is associated with a higher incidence of PTDM in AAs than in non-AAs with higher levels Our results suggest that larger trials are warranted to asess the benefits and risks of sirolimus-based immunosuppression in high risk RTRs.

Abstract# 149

IMPROVED KIDNEY GRAFT FUNCTION IN PATIENTS RECEIVING SIROLIMUS, CYCLOSPORINE AND STEROIDS: TIIE ROLE OF CYCLOSPORINE BLOOD CONCENTRATION. Helio Tedesco,[1] Claudia R. Felipe,[1] Paula G. Machado,[1] Riberto Garcia,[1] Marcelo Franco,[1] Jose O. Medina.[1] *[1]Nephrology Division, Hospital do Rim e Hipertensao-UNIFESP, Sao Paulo, SP, Brazil.*

Phase III clinical trials comparing sirolimus (SRL) versus azathioprine (AZA) in combination with full doses of cyclosporine (CSA) and prednisone showed a dose dependent reduction of the incidence of acute rejection. Compared to AZA, patients receiving SRL showed higher mean creatinine levels at 6 months and this finding has been attributed to SRL-CSA pharmacokinetic interaction. **Objective**: In two trials, patients who received SRL-CSA combination had their CSA dose rapidly reduced after transplantation in attempt to minimize CSA toxicity and preserve graft function. This study evaluates graft functions, CSA doses and blood concentrations during the first 6 months of transplant in patients receiving SRL or AZA in combination with full doses of CSA and prednisone. **Methods**: Recipients of first living donor (non-HLA identical) received 2 mg/day fixed doses of SRL (n=82) or 2 mg/day of AZA (n=35). All patients received initial CSA doses of 8-10 mg/kg/day BID and prednisone 0.5 mg/kg/day CSA doses were adjusted to keep blood levels between 200-400 ng/mL (month 1), 150-300 ng/mL (month 2), and 100-200 ng/mL (month 3-6). **Results**: The incidence of biopsy proven acute rejection was 6.1% (5/82) for SRL and 14.6% (5/35) for AZA SRL trough blood concentrations were 6.7±3.3 (month 1), 6.9±2.5 (month 2), 7.8±3.0 (month 3), and 7.1±2.6 ng/mL (month 6) There were no differences comparing mean creatinine values of SRL and AZA patients at months 1, 2, 3 and 6 (1.54±0.66 vs. 1.62±1.27; 1.54±0.49 vs. 1.53±0.44; 1.56±0.57 vs. 1.45±0.38; 1.7±0.54 vs. 1.5±0 34 mg/dL, ns), respectively. SRL patients received lower CSA doses (351±104 vs 431±114 [month 1], 250±97 vs. 321±103[month 2], 211 ±75 vs. 296±94 [month 3], and 185±63 vs 269±75 mg/day [month 6], p<0.05). At months 2, 3 and 4 CSA trough concentrations were lower in SRL patients (224±93 vs. 279±112; 153±82 vs 192±71; 143±70 vs. 184±65, p<0.05). **Conclusion**: Early and rapid reduction in CSA dose to maintain blood concentrations at the lower limit of the target ranges is a safe and effective measure to preserve graft function without compromising the high efficacy of this immunosuppressive regimen. These findings suggest that decreased graft function observed in phase III SRL treated patients may be related to increased graft susceptibility to cyclosporine toxicity and that this effect can be prevented by early reduction of cyclosporine exposures.

Abstract# 150

CONVERSION TO SIROLIMUS: HOW TO DETERMINE THE OPTIMAL WINDOW OF OPPORTUNITY. A SINGLE CENTER EXPERIENCE. M. F. Egidi,[1] P. A. Cowan,[1] L. W. Gaber,[2] R. J. Stratta,[1] M. H. Shokouh-Amiri,[1] H. P. Grewal,[1] S. H. Vera,[1] M. R. Honaker,[1] A. O. Gaber.[1] *[1]Surgery, Health Science Center, Memphis, TN; [2]Pathology, UN of TN Health Science Center, Memphis, TN.*

Studies in transplant recipients (pts) have demonstrated good results with calcineurin inhibitor (CI) sparing regimens. Sirolimus (SLR) has been introduced in clinical trial in combinations with and without CI however reports of conversion from CI to SLR are limited. We report our experience in switching kidney (KT), kidney-pancreas (SPK), pancreas alone (PA), and liver (OLT) pts from CI, MMF and prednisone to SLR, MMF and prednisone because of nephrotoxicity (TOX)(KT=9, PA=2, OLT=2), hemolytic uremic syndrome (HUS) (KT=4, SPK=3), chronic allograft nephropathy (CAN) (KT=12), glucose intolerance (GLI) (KT=7, SPK=2, PA=1), persistent neurotoxicity (SPK=1, OLT=1) and miscellaneous reasons. SLR conversion was achieved by CI discontinuation, maintenance of the highest MMF dose tolerated and unchanged steroid dose. Contraindications for SLR conversion were rejection within last 3 months, elevated cholesterol (>300mg%)or triglycerides (>400mg%), thrombocytopenia (platelets <95,000), or advanced CAN. SLR was given as a loading dose (8 to 12 mg, PO) and maintained titrated to target trough levels (ng/ml) of 12-14 for KT and OLT and 14-16 for SPK and PA pts. RESULTS: 50 pts (64% males, 52% black, 35 ±10.6 yrs, 24.1 ±29.1 months post-transplant) with 1-12 month follow-up were switched to SLR. We report data on pts with at least 6 month follow-up. Resolution of HUS occurred in 7/7 pts (100%) with a drop in serum Cr (SCr) from 3.4 ± 1.3 to 2.0 ±0.9 mg/dL (p=.03). In combination analysis, SLR conversion due to TOX, HUS and CAN, improved SCr from 3.4 ± 9.6 to 2.2 ±1.4 mg/dL (p=NS). Seven of 10 pts (70%)completely resolved GLI and the other 3 improved blood sugar control. Only 1 SPK pt had a reversible kidney rejection due to noncompliance. SLR was discontinued in 2 SPK (untreatable nausea) and 1 KT (muscle weakness). Increases in cholesterol (186 ± 13.2 to 240 ±19.8 mg%, p=0.02)and triglycerides (190 ±35.9 to 290 ± 53.6 mg%, p=NS) and minimal reduction in platelet values (242 ± 13.5 to 196 ±24.9, p=NS) occurred. CONCLUSIONS: Our results indicate the safety of CI discontinuation and the efficacy to SLR conversion. These data suggest that a CI-free immunosuppressive regimen with SLR, MMF and steroids preserves graft function in pts with clinical indications warranting CI discontinuation. Further studies are mandatory to establish the role of SLR in preventing or reducing CAN progression.

Abstract# 151

SIROLIMUS IS NOT NEPIIROTOXIC: EVIDENCE FROM CLINICAL TRIALS. Tim Mathew, the Sirolimus Clinical Trials Study Groups. *[1]Queen Elizabeth Hospital, Adelaide, Australia.*

Purpose: Studies in animal models have confirmed that sirolimus (SRL) has no deleterious effect on GFR. SRL does not inhibit calcineurin and thus would be expected to lack nephrotoxic properties. We report the results from clinical trials which provide evidence that SRL is not nephrotoxic.

Methods: Data were reviewed from 5 phase II trials in which SRL was administered either without cyclosporine (CsA) to renal transplant patients or as monotherapy to patients with psoriasis. Additional data were gleaned from 2 phase III trials of SRL (2 and 5 mg/day), CsA and azathioprine (AZA) or placebo (PLA), and from 2 trials of CsA elimination in SRL-treated renal transplant recipients.

Results: Renal function data are listed in the table. Patients treated with SRL and standard doses of CsA had moderately higher mean creatinine levels at 6 and 12 months compared with AZA and PLA cohorts. When SRL was given as monotherapy, psoriasis patients demonstrated normal renal function. However, as the primary therapy (6-9 mg/day) for renal transplantation, SRL treatment resulted in consistently lower creatinine and higher GFR values compared with a CsA-treated cohort. Renal function in the SRL arms of these trials was significantly better than seen in the CsA arms both at 1 and 2 years post-transplant. In a trial of CsA elimination at 3 months post-transplant, SRL-treated patients experienced a significant and durable improvement in renal function and improvement in graft function.

Conclusions: SRL is not nephrotoxic and does not have a deleterious effect on creatinine or GFR A sustained improvement in renal function is seen in patients who are maintained on SRL after CsA elimination.

Study	Treatment	Time	Creatinine (µmol/L)	GFR (mL/min)
Phase III SRL trials	SRL 2 mg/day	12 mo	158 0***	60 3***
	SRL 5 mg/day	12 mo	171 7***	55 2***
	AZA	12 mo	133 1	67 5
	PLA	12 mo	136 8	66 3
CsA Elimination trial	SRL + CsA elimination	12 mo	135 4**	68 3***
	CsA + SRL	12 mo	169 9	55 6
SRL Primary therapy	SRL (30 ng/mL)	24 mo	119 0*	69 3*
	CsA	24 mo	148 7	56 8
SRL monotherapy (psoriasis)	SRL (5 mg/day)	12 wk	85 7	NA

*p<0 05, **p<0 01 ***p<0 001 vs comparator; NA=not available

Abstract# 152

SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY PERMITS EARLY CYCLOSPORINE WITHDRAWAL RESULTING IN IMPROVED RENAL FUNCTION: 12-MONTH RESULTS OF THE TRI-CONTINENTAL TRIAL. Robert W.G. Johnson,[1] Henri Kreis, Rainer Oberbauer, Kerstin Claesson, Josette Eris, Manuel Arias, Leszek Paczek, Alfredo Mota, Francesco Schena, Felix Frey, Ahmed Shoker, Robert Koene, Anders Hartman, the Sirolimus Tri-continental Renal Transplant Study Group. [1]*The Renal Transplant Unit, Manchester Royal Infirmary, Manchester, United Kingdom.*

Purpose. The present study evaluated whether cyclosporine (CsA) could be withdrawn at 3 months from a sirolimus (SRL)-CsA-steroids regimen, thereby reducing CsA toxicities.

Methods. This was an open-label study conducted in 57 centers in Europe, Australia, and Canada. A total of 525, 1st (90%) or 2nd (10%) renal allograft recipients, with cadaveric (88%) or living donor (12%) allografts, received SRL 2 mg (tablets), CsA, and steroids when enrolled into the study. SRL blood levels were to be maintained above 5 ng/mL (immunoassay). At 3 months ± 2 weeks, eligible patients were randomized (1:1) to remain on triple-therapy (SRL-CsA-steroids), or to have CsA withdrawn over 6 weeks (SRL-steroids) and initiate concentration-controlled SRL therapy (blood trough levels 20 to 30 ng/mL, immunoassay).

Results (ITT-analysis at 12 months). 430 patients (82%) were randomized, including a majority of those who experienced an acute rejection during the pre-randomization period. Of the patients randomized to SRL-steroids, 92% were able to withdraw CsA. Graft survival (95.8 vs 97.7%), patient survival (97.2 vs 98.1%), acute rejection (13.5 vs 20.0%) and discontinuations (21 vs 28%) were not statistically different between SRL-CsA-steroids and SRL-steroids, respectively. Systolic and diastolic blood pressure as well as calculated GFR (56 vs 62 ml/min, p<0.001) were lower when CsA was eliminated. There was no difference in serum cholesterol. Investigators reported that hypertension, increased creatinine, hyperuricemia, tachycardia, *Herpes zoster*, and skin cancer occurred statistically more frequently in patients remaining on CsA, whereas thrombocytopenia, abnormal liver function tests and hypokalemia were reported more often in patients randomized to long-term SRL-steroids maintenance therapy.

Conclusion. This multicenter 525-patient study has demonstrated that initial therapy including CsA for 3 months followed by SRL-steroids maintenance therapy offers an important alternative to long-term calcineurin inhibitor therapy, permitting improved renal function in kidney transplantation.

CONCURRENT SESSION 17:
MECHANISMS OF ISCHEMIA/REPERFUSION INJURY I

Abstract# 153

ORGAN-SPECIFIC EFFECTS OF TARGETING NF-κB TO REDUCE ISCHEMIA/REPERFUSION INJURY. Liqing Wang,[1] Nida Shemmeri,[1] Kerrie L. Faia,[1] Wei Gao,[1] Vilmos Csizmadia,[1] Wayne W. Hancock.[1] [1]*Transplantation, Millennium Pharmaceuticals, Inc., Cambridge, MA.*

A central role for the NF-κB transcription factor pathway in induction of pro-inflammatory genes is well recognized, such that the development and application of inhibitors of this pathway are expected to be of therapeutic value in decreasing the effects of ischemia/reperfusion (I/R) injury. To test this hypothesis, we have studied the effects of I/R in mice genetically deficient in NFκB genes, beginning with p50-/- mice. We compared the effects of I/R on liver and kidney, given their importance in clinical transplantation, and capacity to provide biochemical evidence of injury. Northern and Western blot analyses of wild-type mice showed that liver and kidney contained comparable levels of p50 and p65, which form the classic p50/p65 NF-kB heterodimer, plus associated IκBα and the upstream kinases, IKKα and IKKβ. Animals (5/group/time-point) were subjected to 90' of 70% hepatic or 45' of left renal (with right renal resection) ischemia, and sacrificed at 4-48 h. Apart from oxidative stress (p>0.05), liver I/R was significantly worse in p50-/- vs. p50+/+ mice for each parameter (all p<0.001), including ALT (152 ± 20 vs. 19 ± 6 U/L); histology of widespread neutrophil recruitment and hepatic infarcts vs minor injury; TUNEL+ cells (>5/field vs. <1/field), and caspase-3 activation (3403 ± 121 vs. 1280 ± 240 U/mg). In stark contrast, renal I/R led to significantly less injury in p50-/- vs p50+/+ mice (p<0.01 for each parameter), including BUN (24 ± 3 vs. 38 ± 3 mg/dL), histology of negligible injury vs. neutrophil accumulation, TUNEL+ cells (<1/field vs. >5/field); caspase-3 activation (808 ± 60 vs. 1159 ± 406 U/mg); and oxidative stress (808 ± 60 vs. 1159 ± 406 μM/g). Comparable preliminary data have also been generated in control mice subjected to I/R and treated with small molecule inhibitors of NF-κB. Our findings (i) show that targeting of the NF-kB pathway is unlikely to provide a general mechanism for therapy of I/R; (ii) emphasize the critical importance of the liver's NF-κB-dependent capacity for anti-apoptotic gene induction and entry into the cell cycle; and (iii) indicate that targeting of NF-κB is a powerful approach for controlling renal I/R injury.

Abstract# 154

LIVER TRANSPLANT PRESERVATION INJURY ACTIVATES THE LPS/TOLL-LIKE RECEPTOR SIGNALING PATHWAY. George Tsoulfas, Yoshihito Takahashi, Raymond W. Ganster, Gautam Yagnik, Noriko Murase, David A. Geller.

Background: Endotoxin or LPS (lipopolysaccharide) initiates the significant complications of septic shock and multiple organ failure seen in gram-negative bacterial infections. LPS signaling is triggered by a receptor complex consisting of LPS, LBP (LPS-binding protein), CD14, and Toll-like receptor (TLR) 2 or 4. This signal transduction pathway activates NF-κB and other intracellular second messengers. **Objective**: To examine whether the LPS signaling pathway is activated in hepatic transplantation following clinically relevant graft reperfusion injury. **Methods**: Orthotopic syngeneic (Lew-Lew) rat liver transplantation was performed with 18 hours of cold preservation in UW solution. Rats were sacrificed at 1-48 hrs after transplantation. Northern blot analysis for hepatic CD14, LBP, and TLR-2 mRNA, liver enzyme analysis, and gel shift assay for NF-κB were performed. **Results**: AST and ALT peaked 12 hrs after transplant. NF-κB activity showed a biphasic peak at 1 and 12 hrs after reperfusion. mRNA for both CD14 and LBP were significantly upregulated with peak at 6-12 hours following transplant, corresponding to the second peak of NF-κB activation. TLR-2 mRNA was also increased. **Conclusion**: These data indicate for the first time that the LPS/TLR-2 signaling pathway is activated during liver graft reperfusion injury. These findings are especially important given that LPS levels are known to be elevated in the liver transplant setting. This study may also explain why liver transplant patients with severe preservation injury are markedly susceptible to a variety of bacterial infections early post-operatively.

Table (n = 3-4 rats per group, mean ± SE, mRNA is relative units, *p≤0.05 vs. Normal).

Groups	AST (U/L)	ALT(U/L)	NF-κB	CD14 mRNA	LBP mRNA
Normal Liver	118 ± 13	56 ± 2		116	18
Tx 6 hours	3052 ± 380*	2452 ± 375*	+	897*	117*
Tx 12 hours	4745 ± 667*	3528 ± 295*	+++	587	127*

Abstract# 155

HEME OXYGENASE-1 GENE TRANSFER INHIBITS INDUCIBLE NITRIC OXIDE SYNTHASE EXPRESSION AND PROTECTS GENETICALLY FAT ZUCKER RAT LIVERS FROM ISCHEMIA/REPERFUSION INJURY. Ana J. Coito,[1] Xiu-Da Shen,[1] Roland Buelow,[2] Farin Amersi,[1] Carolina Moore,[1] Ronald W. Busuttil,[1] Jerzy W. Kupiec-Weglinski.[1] [1]*Surgery, Dumont-UCLA Transplant Ctr., Los Angeles, CA;* [2]*SangStat Corp., Fremont, CA.*

Background: Ischemia/reperfusion (I/R) injury is a critical factor in the dysfunction of steatotic orthotopic liver transplants (OLT). We have shown that HO-1 induction ameliorates rat hepatic I/R injury. This study was designed to determine putative mechanisms of HO-1-mediated beneficial effects against I/R injury in a steatotic rat liver model.

Methods and Results: Genetically fat Zucker rats were injected i.v. with 2.4×10^9 pfu of either Ad-HO-1 or Ad-βGal. 24h later, livers were harvested and stored for 4h at 4C in UW solution. OLTs were then performed to syngeneic lean Zucker recipients. Ad-HO-1 therapy increased recipient survival rate to 82% (vs. 50% in Ad-βGal controls; n=10 rats/gr), and diminished hepatocyte injury (sGOT levels [IU/L] at day 1 = 1627 vs. 5721, p<0.04). Ad-HO-1 treated livers showed elevated levels of HO-1 enzymatic activity (3.4 nmol/mg/min) contrasting with undetectable levels in controls. These findings were correlated with higher densitometric levels of anti-apoptotic proteins, such as Bcl-2 (1.5-fold) and Bag-1 (3.4-fold) in the Ad-HO-1 treated livers. In addition, an active form of the pro-apoptotic caspase-3 (p20) was found 2.9-fold lower in Ad-HO-1 livers, as compared with controls. Next, we determined whether upregulation of HO-1 modulates inducible nitric oxide synthase (iNOS) expression. Both groups were characterized by elevated numbers of infiltrating macrophages (+++). Interestingly, while in the Ad-HO-1 group, macrophages were iNOS negative and dispersed throughout the liver, in the Ad-β Gal group, infiltrating macrophages localized preferentially in the portal areas and densely stained for iNOS. Unlike in control livers, iNOS was almost absent in the Ad-HO-1 treated livers, as determined by Western analysis. The expression of cellular fibronectin, an integral feature of allograft rejection cascade, was found depressed in the Ad-HO-1 group.

Conclusion. Ad-HO-1 therapy inhibits iNOS expression, prevents down-regulation of Bcl-2/Bag-1 proteins, reduces caspase-3 expression, and diminishes liver fibrosis. Whether the beneficial effect of Ad-HO-1 therapy is specifically dependent on iNOS inhibition requires further investigation. Hence, this work provides the rationale for the development of therapeutic approaches based on new mechanistic concepts of I/R injury in steatotic livers.

Abstract# 156

CARBON MONOXIDE PROVIDES PROTECTION AGAINST ISCHEMIA/REPERFUSION INJURY IN RAT LIVERS. Farin F. Amersi,[1] Xiu-Da Shen,[1] Dean Anselmo,[1] Judy Melinek,[1] Ronald W. Busuttil,[1] Roland Buelow,[2] Jerzy W. Kupiec-Weglinski.[1] *[1]Dumont-UCLA Liver Transplant Center, UCLA School of Medicine, Los Angeles, CA; [2]Sangstat Corp., Fremont, CA.*

Background: We have shown that upregulation of heme oxygenase-1 (HO-1), a stress-inducible antioxidant protein, ameliorates ischemia/reperfusion (I/R) injury in rat liver models. This cytoprotective effect has been attributed to the generation of the effector signalling molecule carbon monoxide (CO) during HO-1 catalysis of heme. A direct role of HO-1 derived CO in the protection against hepatic I/R injury needs to be established. This study was designed to determine the effects of CO on I/R injury in an *ex-vivo* isolated perfusion rat liver model.

Methods: Sprague Dawley rat livers were harvested and stored for 24 h at 4°C in UW solution (3-7 rats/gr). Livers were then perfused *ex-vivo* for 2 h on an isolated perfusion rat liver apparatus with rat whole blood supplemented with: (I) 0.03% CO + air (21% O_2); (II) air alone, (III) 95% O + 5% CO ; (IV) 0.03% CO +L-NAME (2mM), a nitric oxidase synthase inhibitor (iNOS); and (V) 0.03% CO + D-NAME, an inactive iNOS inhibitor. Portal vein blood flow, portal pressure, bile production and sGOT levels were assessed at 30 min intervals. Liver samples were collected for histology/HO-1 enzymatic activity.

Results: Livers perfused with 0.03% CO (300 ppm) had significantly higher portal venous blood flow, and increased total bile production compared with livers perfused with air ($p<0.01$) or 95% O_2 ($p<0.05$). Livers perfused with CO showed less hepatocyte injury compared with the groups perfused with air ($p<0.003$) or 95% O_2 ($p<0.01$) (sGOT [IU/L] at 2 h: 163,497, and 206, resp). HO-1 activity was increased in the CO treated livers compared with livers perfused with air or 95% O_2 (2.04 vs 0.87 vs 1.48 nmol/mg/min). This data correlated with histological Banff's criteria of I/R injury where CO perfused livers showed minimal lobular disarray/ballooning changes and no hepatocyte necrosis, vs moderate to severe changes in Gr II/III. Adjunctive use of L-NAME or D-NAME did not affect the benefical effects rendered by exogenous CO on hepatic I/R injury.

Conclusion: These intriguing results indicate that exogenous CO exerts potent cytoprotective effects against hepatic I/R injury. The effects of HO-1 - CO antioxidative pathway are independant of NO synthesis. Hence, regimens that employ exogenous CO should be revisited, as they may have potential therapeutic application in preventing hepatic I/R injury in liver transplant recipients.

Abstract# 157

HEME OXYGENASE-1 GENE TRANSFER PROTECTS AGAINST ISCHEMIA/REPERFUSION INJURY IN RAT RENAL ISOGRAFT MODEL. Tom D. Blydt-Hansen,[1,2] Masamichi Katori,[1] Charles Lassman,[3] Ana J. Coito,[1] Roland Buelow,[4] Robert Ettenger,[2] Ronald W. Busuttil,[1] Jerzy W. Kupiec-Weglinski.[1] *[1]Liver and Pancreas Transplantation, Dumont-UCLA Transplant Ctr, Los Angeles, CA; [2]Pediatric Nephrology, UCLA, Los Angeles, CA; [3]Pathology and Laboratory Medicine, UCLA, Los Angeles, CA; [4]Sangstat Corp., Freman, CA.*

Background: Ischemia/reperfusion (I/R) injury is an important problem in clinical transplantation. Gene transfer induced heme oxygenase-1 (HO-1) overexpression protects rat livers against I/R insult. This study was designed to evaluate the effects of Ad-HO-1 gene transfer in a rat renal isograft model.

Methods: Donor LEW kidneys were perfused with Ad-HO-1 ($2.5x10^9$pfu) or Ad-β-gal ($2.5x10^9$pfu) or saline, stored at 4°C for 24h and transplanted orthotopically into LEW recipients. Contralateral native nephrectomy was performed at day 5. Serum creatinine and urine protein/creatinine ratio were analyzed at day 7 and 30. The kidneys were analyzed for severity of acute tubular necrosis (ATN), % glomerulosclerosis, HO-1 and β-gal expression patterns.

Results: Ad-HO-1 based gene transfer conferred a survival advantage when compared with both saline or Ad-β-gal treated controls ($p<0.05$).

(Table)

Serum creatinine levels (mg/dl) were elevated at day 7 in all 3 groups (range 3.5-4.3), but normalized to 0.8 by day 30 selectively in the HO-1 group. Urine protein/creatinine ratio at day 7 was elevated in the β-gal compared to the HO-1 group (9.4 vs 4.6, $p<0.05$) suggesting a more severe glomerular injury. Histologically, ATN was graded as more severe in the β-gal group at all time points. The extent of glomerulosclerosis was increased in the β-gal group compared to the HO-1 group at day 3 (18.5% vs. 11%) and day 7 (30% vs. 4%, $p<0.01$). The β-gal expression was readily detectable after 24h preservation in the β-gal group in a glomerular distribution. HO-1 expression was discernible by immunohistochemistry in some glomeruli and tubules at 3h post-transplant in the HO-1 group, whereas it was absent in the β-gal group.

Conclusion: Our findings are consistent with protective effects of HO-1 overexpression using a gene transfer approach against severe renal I/R injury, with reduced mortality and attenuation of tissue injury.

Treatment	(n)	Survival (days)	Median
Ad-HO-1	9	6,7,9,>22,>22,>32,>32,>71,>76	>22
Ad-β-gal	4	5,5,8,8	8
Saline	8	6,6,7,7,7,8,9,>44	7

Abstract# 158

COLD ISCHEMIA/REPERFUSION (CIR) INJURY IN THE MURINE ORTHOTOPIC LIVER TRANSPLANT (mOLT) MODEL: CRITICAL ROLE OF IL-6 IN RECOVERY. Xingyi Que, Fotini Debonera, Xavier Aldeguer, Andrew E. Gelman, Gideon A. Zamir, Kim M. Olthoff.

This study examines the use of the murine IL-6 -/- strain in a mOLT model to determine pathways involved in the recovery of grafts from CIR injury. The aims were: 1) demonstrate feasibility of mOLT with prolonged cold storage and determine maximum length of cold ischemia, 2) correlate injury and regenerative response with increasing preservation times, 3) determine role of IL-6 in recovery from CIR injury.

Methods: mOLT was performed using syngeneic wild-type C57BL/6 → C57BL/6 (Group A) and knockout C57BL/6 IL-6 -/- → IL-6 -/- (Group B) combinations. Grafts were preserved at 4°C in UW solution for 4 periods: 1 (A1), 4 (A2), 8 (A3) and 16 (A4) hours, and transplanted. Survival was monitored up to 7 days (n=6/group). Injury was determined by histology, and DNA replication quantified by BrdU uptake at 48 hours. The role of IL-6 in the recovery from CIR injury was determined with the use of human recombinant IL-6 (1mg/kg) given to the recipient 30 minutes prior to OLT.

Results: Survival in the wild-type group was 95%, 83%, 75% and 0% for Groups A1, A2, A3 and A4 respectively. Group A1 and A2 grafts demonstrated normal histology, however, group A3 grafts showed hepatocyte vacuolization and swelling with zone 3 hepatocyte dropout. BrdU uptake in A3 significantly exceeded that of both A1 and A2, indicating an intense regenerative response. IL-6-/- recipients of IL-6-/- livers exposed to minimal ischemia survived only for 2-4 days post mOLT. Death was associated with the development of liver failure, and histology showed extensive patchy necrosis. IL-6 -/- recipients were able to be rescued by a single subcutaneous dose of IL-6, with improved histology and survival (> 7 days).

Conclusions: This study demonstrates the clear advantage of the ability to utilize murine OLT with KO strains in the study of mechanisms associated with recovery from CIR injury. Increasing lengths of cold ischemia correlate with progressive tissue damage, and recovery is associated with a regenerative response corresponding to the severity of injury. Severe irreversible injury occurs within 16 hours. More interesting is the inability of the liver to recover in the absence of IL-6. Previous studies indicate the critical role of IL-6 in the regenerative response after hepatectomy, however, this study demonstrates the need for this growth factor in the recovery of hepatocytes even prior to the initiation of regeneration. It is still to be determined whether overexpression of IL-6 will enhance recovery.

Abstract# 159

AN NOVEL INHIBITOR OF RHO-ASSOCIATED PROTEIN KINASE, Y-27632, AMELIORATES HEPATIC ISCHEMIA AND REPERFUSION INJURY. Keisa Takeda,[1] Maeng Bong Jin,[1] Tsunenori Sakurai,[1] Tsuyoshi Shimamura,[1] Hiroyuki Furukawa,[1] Miri Fujita,[2] Satoru Todo.[1] *[1]First Department of Surgery, Hokkaidou University, Sapporo, Hokkaidou, Japan; [2]Second Department of Pathologyy, Hokkaidou University, Sapporo, Hokkaidou, Japan.*

BACKGROUND: Hepatic ischemia and reperfusion (I/R) injury is augmented by production of vasoconstrictive peptides and activation of neutrophils. Rho family of small GTPases is known to play a key role in vasoconstriction and neutrophil adhesion by activating a serine/threonine kinase, p160ROCK. In the present study, we examined our hypothesis whether inhibition of ROCK kinase by a novel ROCK inhibitor, Y-27632(Y), protects the liver against I/R injury. **MATERIALS AND METHODS:** Male Sprague-Dawley rats (180-280g) were subjected to 70% partial hepatic warm ischemia by clamping the vessels of the left and middle lobes. After 120min, the vascular clip was released and non-ischemic right lateral and caudate lobes were excised. Rats were divided into 4 groups (n=6); non-treated control, Y-10, -20 and -30mg/Kg. The agent was given orally 1 hour before ischemia. One week animal survival, blood pressure (BP), heart rate, hepatic tissue blood flow (HTBF), liver fuction tests, and plasma lactate and ammonia level were evaluated. Histological findings were analyzed by H.E. staining, and the number of neutrophils infiltrated into liver tissue was counted. **RESULTS:** Animal survival was remarkably improved by the treatment. BP in Y-10 during ischemia was almost the same as that of control and kept significantly higher for first 30min. after reperfusion. The recovery of HTBF in Y-10 after reperfusion was the best, although HTBFs of Y-20 and -30 were better than that of control. Serum liver enzymes were significantly improved in the treated groups. Histological findings were also attenuated. The number of infiltrated neutrophils were significantly reduced by Y-27632 treatments. **CONCLUSION:** Inhibition of ROCK kinase by Y-27632 has protective effect on hepatic I/R injury. Manipulation of the Rho-ROCK system may pave a novel approach to investigate I/R injury of the liver and related problems.

treatment	n	7-day survival	LDH	ALT	% HTBF at 25min. after reperfusion	average BP during ischemia	average BP after reperfusion
1 control	6	1/6	89356 0± 19293 3	10829 8± 2497 2	43 8±10 2	117 7±8 3	73 1±14 8
2 Y 10mg/Kg	6	6/6	56672 0± 5411 7*	6805 3± 1916 1*	63 6±5 3*	115 3±4 6	87 1±7 7*
3 Y 20mg/Kg	6	3/6	60269 0± 30245 7*	7018.5± 2936 5	55 3±3 1	99 8±9 1*	77 8±4 0
4 Y 30g/Kg	6	4/6	56500 0± 7526 0*	9097± 4463 9	54 7±2 8	102.3±3 7*	72 6±4 2

value, mean±S D, *P<0 05 relative to control

Abstract# 160

P38 MITOGEN-ACTIVATED PROTEIN KINASE INHIBITOR AS AN ADDITIVE TO UW SOLUTION AMELIORATES REPERFUSION INJURY IN LIVER TRANSPLANTATION. Daisuke Yoshinari,[1] Izumi Takeyoshi,[1] Mitsunobu Kobayashi,[1] Susumu Ohwada,[1] Yoshihiro Yabata,[1] Koshi Matsumoto,[2] Yasuo Morishita.[1] *[1]Second Department of Surgery, Gunma University School of Medicine, Maebashi, Gunma, Japan; [2]Department of Pathology, Nippon Medical School Second Hospital, Kawasaki, Kanagawa, Japan.*

Purpose: We evaluated the effects of FR167653, a novel p38 mitogen-activated protein kinase (MAPK) inhibitor, as an additive to UW solution on ischemia/reperfusion injury in liver transplantation.

Methods: Male Lewis rats weighing 200 to 260 g were used. In the FR group, the donor liver was perfused with 100 ml/kg body weight of cold UW solution containing FR167653 (60 mg/L) and stored in the same solution for 30 hr at 4°C. In the control group, UW solution without FR167653 was used. The grafts were then reflushed with lactated Ringer's solution and transplanted orthotopically into the recipient. Ten-day survivors were recorded (each n=14 or 15). Liver tissue blood flow (LTBF), serum ALT and LDH levels, and histological findings were evaluated after reperfusion (each n=6 to 8). The activities of p38 MAPK and p46/p54 c-jun N-terminal kinase (JNK) in the liver graft were also determined during cold storage and after reperfusion (each n=4). Data are expressed as the mean±SEM.

Results: The ten-day survival rate was significantly ($p<0.01$) better in the FR group (7/14) than in the control group (1/15). Both serum ALT (IU/L) and LDH (IU/L) levels 1 hr after reperfusion in the FR group (547±38 and 12652±638, respectively) were significantly ($p<0.05$) lower than in the control group (792±82 and 15948±1107, respectively). The LTBF (a percentage of pre-transplantation) 6 hr after reperfusion in the FR group (62±9.2) was significantly ($p<0.05$) higher than in the control group (30±8.1). The FR group showed less histologic damage compared to the control group. p38 MAPK and p46/p54 JNKs were not activated during cold ischemic storage. Both p38 MAPK and p46/54 JNKs in the control group and only p46/p54 JNKs in FR group were markedly activated after reperfusion, however, p38 MAPK in the FR group was less activated than in the control group. The activities of p38 MAPK (the ratios to the mean for the control group) 0.5 and 1 hr after reperfusion in the FR group (0.64±0.15 and 0.46±0.087, respectively) were significantly ($p<0.01$) lower than in the control group (1.0±0.089 and 1.0±0.097, respectively).

Conclusion: The addition of FR167653 to UW solution attenuated cold-ischemia / reperfusion injury in liver transplantation associated with the inhibiton of p38 MAPK activation.

Abstract# 161

ELEVATED CYCLIC AMP AMELIORATES ISCHEMIA-REPERFUSION INJURY IN RAT CARDIAC ALLOGRAFTS: A NOVEL APPLICATION OF WATER-SOLUBLE FORSKOLIN DERIVATIVE. Seiichiro Murata, Douglas N. Miniati, Murray Kown, Mark L. Koransky, Robert C. Robbins. *[1]Department of Cardiothoracic Surgery, Stanford University, Stanford, CA.*

Objective: The oxidative stress following ischemia-reperfusion (IR) of cardiac allografts leads to activation of cardiomyocytes, which produce injurious cytokines such as tumor necrosis factor alpha (TNF-α). Cyclic adenosine monophosphate (cAMP) is an important second messenger that decreases during organ preservation. NKH477 is a recently developed water-soluble forskolin derivative that directly activates adenylate cyclase and increases intracellular cAMP. In previous *in vitro* studies, increased intracellular cAMP induces a protein kinase activity that reduces the cytokine signal for vascular cell adhesion molecule (VCAM-1) expression. We hypothesized that augmenting donor heart cAMP levels could reduce allograft tissue inflammation and improve the response to IR injury **Methods:** PVG to ACI rat heterotopic cardiac allografts were reperfused for 4 hours after 60 minutes of ischemia. Donor hearts were procured and assigned to two groups: intracoronary perfusion and incubatution in NKH477 (10μ g/ml) solution (n=6), intracoronary perfusion and incubation in phosphate buffered saline prior to transplantation (n=6). Grafts were analyzed by RT-PCR for TNF-α and VCAM-1 mRNA expression, and for myeloperoxidase (MPO) activity. **Results:** Grafts in the NKH477 treated group had lower levels of MPO activity compared to control (0.55±0.21 vs. 0.96±0.20 U/mg total protein, $p<0.05$) TNF-α and VCAM-1 mRNA expression were also significantly downregulated in the NKH477 treated group (TNF-α/G3PDH ratio=0.44±0.21 vs. 0.99±0.31, $P<0.01$, VCAM-1/G3PDH ratio=0.54±0.31 vs. 1.07 ±0.28, $p<0.05$). **Conclusions:** This study shows for the first time that water-soluble forskolin derivative can ameliorate IR injury following experimental cardiac transplantation in a rodent model The beneficial effects of increased intracellular cAMP were associated with downregulation of TNF-α and VCAM-1 expression. Future studies will correlate this strategy for reducing IR injury with the development of graft coronary artery disease

CONCURRENT SESSION 18:
THORACIC ORGAN DONOR SHORTAGE: BETTER MANAGEMENT/
ALTERNATIVE STRATAGIES

Abstract# 162

PRE-OPERATIVE HLA-DR MATCHING AND EXTENDED LUNG PRESERVATION WITH UW SOLUTION COULD SIGNIFICANTLY IMPROVE GRAFT OUTCOME. Daniel S. Woolley,[1] William J. Burlingham,[1] Lynn D. DeVito-Haynes,[1] Glenn E. Leverson,[1] Wim van der Bij,[1] W. J. de Boer,[1] Bouke G. Hepkema,[1] Keith C. Meyer,[2] Richard D. Cornwell,[2] Robert B. Love.[1] *[1]Cardiothoracic Surgery, University of Wisconsin, Madison, WI; [2]Pulmonary Medicine, University of Wisconsin, [1]Surgery, University Hospital Groningen, Groningen, The Netherlands.*

INTRODUCTION: In orthotopic lung transplantation (OLT), 6 hours is generally regarded as the extent of total ischemic time. This is the limiting factor in the ability to perform pre-operative human leukocyte (HLA) matching. HLA-DR matching's role in lung transplantation has not been well defined.

At UW, our practice has been to use organs that have sustained greater than 6 hours of cold ischemic time for transplantation. Standard preservation techniques include 1mg of PGE, infusion of 4L UW solution (4C), and retrograde pulmonary vein flushing of 1L UW solution per lung.

METHODS: A cohort of 237 recipients of OLT's (112 from UW Hospital and 125 from University Hospital Groningen) were retrospectively analyzed for outcome, specifically time to poor function. Poor function was defined as time to bronchiolitis obliterans syndrome (BOS, >20% decrease in FEV1 from post-op baseline) or graft loss. Recipients were divided into two groups; Group 1, HLA-DR 0-mismatch (with respect to recipient, most of whom were HLA-DR 1-matched, DR-homozyogous donors (n=20)), Group 2, HLA-DR 1-or more mismatch (n=217). UW patients were further subdivided into groups based on preservation time (PT, cross clamp to pulmonary re-perfusion, < or > 8 hours), bilateral OLT recipients PT were time to second lung re-perfusion. Inter group significance ($p<0.05$) was determined using chi-square and life table analysis.

RESULTS: At 8 years post-operatively, Group 1, HLA-DR 0-mismatch had a 55.6% freedom from BOS or graft loss vs. 23.0% in Group 2, HLA-DR 1-or more mismatch($p=0.0285$). There were no significant differences in freedom from poor function in UW patients with HLA-DR 0-mismatches when compared against preservation times of < or > 8 hours, 50.0% vs 66.6% at 3 years($p=0.3980$).

CONCLUSIONS: Patients with an HLA-DR 0-mismatch have a significant advantage over those who have a 1-or more mismatch with regard to freedom from BOS or graft loss. Extended lung preservation with UW solution beyond 8 hours could potentially open the door for this information to be exploited. We propose that HLA-DR typing to accomplish a 0-mismatch in orthotopic lung transplant recipients could significantly improve long term outcomes without effecting early mortality.

Abstract# 163

FLOW PRA DETECTED HLA ANTIBODIES CAN IDENTIFY CLINICALLY RELEVANT FLOW CYTOMETRIC POSITIVE CROSSMATCHES AMONG CARDIAC ALLOGRAFT RECIPIENTS. Piotr Przybylowski,[1] Howard Gebel,[2] Robert Bray,[3] Branislav Radovancevic,[4] O.Howard Frazier,[4] Chris Garcia,[1] Stephanie Rasmussen,[1] Shauna Garner,[1] Barry Kahan,[1] Ronald Kerman.[1] *[1]Surgery, Univ. of Texas Medical School, Houston, TX; [2]Surgery, Louisiana State Univ, Shreveport, LA; [3]Pathology, Emory Univ Hospital, Atlanta, GA; [4]Surgery, Texas Heart Institute, Houston, TX.*

We previously reported that cardiac allograft recipients (recips) transplanted (Tx) following an IgG anti-human globulin (AHG) negative crossmatch (XM) but an IgG flow cytometric positive crossmatch (FCXM) had a significantly poorer one year graft survival of 68% (15/22) compared to 86% (49/57) for FCXM negative recips ($p<0.02$). Testing included the historically highest PRA and immediate preTx sera. It was not readily apparent why 32% of FCXM positive recips lost their grafts while 68% of FCXM postive recips experienced successful graft survival beyond one year. Since the FCXM assay measures antibodies bound to any cell membrane antigen it is possible that the FCXM detected reactivity was not anti-HLA related. With the introduction of the Flow PRA assay to detect IgG HLA class I and/or class II antibodies a clarification of the problem was possible. The Flow PRA assay measures antibodies bound to a solid phase matrix to which soluble HLA class I or class II antigens are attached and provides an HLA specific alternative to membrane dependent assays. We hypothesized that only HLA specific antibodies would be associated with failed grafts. Therefore, we retrospectively Flow PRA tested 5 of the preTX sera from FCXM-positive failed graft recips and 11 of the preTx sera from FCXM positive successful recips. All 5 of the FCXM positive sera from failed grafts tested positive for Flow PRA HLA class I (but not class II) antibodies, whereas all 11 sera from FCXM positive successful recips were Flow PRA non-reactive for both HLA class I and class II antibodies. Thus, in this retrospective study we observed that in the absence of HLA antibody FCXM positive sera was likely non-HLA related and recips could be expected to enjoy successful graft survival In contrast, FCXM positive sera that did contain Flow PRA detected IgG HLA class I antibodies were associated with graft loss. Our data supports the concept that the Flow PRA assay is a sensitive and clinically relevant test that identifies IgG HLA antibodies and cardiac allograft recips at risk for graft loss.

Abstract# 164

DONOR CARDIAC TROPONIN T AND PROCALCITONIN ARE INDEPENDENT PREDICTORS OF EARLY GRAFT FAILURE AFTER HEART TRANSPLANTATION. Evgenij V. Potapov,[1] Frank D. Wagner,[1] Matthias Loebe,[1] Britta Jonitz,[1] Ekaterina A. Ivanitskaia,[1] Julia Stein,[1] Ralf Sodian,[1] Martin Moeckel,[2] Christian Mueller,[3] Roland Hetzer.[1] *[1]Cardiothoracic and Vascular Surgery, Deutsches Herzzentrum Berlin, Berlin, Germany; [2]Internal Medicine, Humboldt University of Berlin, Virchow Klinikum, Berlin, Germany; [3]Clinical Chemistry, Humboldt University of Berlin, Virchow Klinikum, Berlin, Germany.*

Background: Recently we demonstrated that cardiac troponin T (cTnT) >0.1 µg/L and procalcitonin (PCT) >2 ng/mL might be predictors of early graft failure. However, it is not clear whether one of them is a surrogate marker. We evaluated the relationship between these markers as predictors of early graft function after heart transplantation (HTx).

Methods: cTnT and PCT serum concentrations were measured in samples retrieved immediately before pericardium opening from 92 consecutive brain dead donors accepted for HTx. The donors were retrospectively divided into two groups: group I (n=78) - grafts with good function, group II (n=14) - early graft failure after HTx.

Results: No differences in donor and recipient characteristics were found between groups. There were no correlations between cTnT and PCT values (r=0.12, p=0.27). In group II cTnT values >0.1 µg/L were found in 9 (64%) and PCT values >2 ng/mL in 5 donors (36%). At least one of both markers was elevated in 12 donors (86%). In two donors both markers were above the cut-off levels and in further two below. There was no significant interaction between both markers when using logistic a regression model (p=0.28). The specificity, sensitivity and unadjusted odds ratios for single parameters and for the combination (at least one of both markers was elevated) are given in the table.

Conclusions: cTnT and PCT levels in heart donors at the time of explantation were independent prognostic markers of early graft failure. This fact may suggest two different mechanisms of the impairment in the donor hearts: primary myocardial damage and damage related to systemic inflammatory response. The combination of the cTnT value >0.1 µg/L or PCT level of >2 ng/mL had a higher sensitivity than the single parameters alone Both markers can be used as additional parameters in heart donor selection.

Parameter	Sensitivity (%)	Specificity (%)	Unadjusted Odds Ratio
TnT >0.1 µg/L	64	97	68.4 (11.5-405.4, p<0.0001)
PCT >2 ng/mL	36	91	5.6 (1.5-21.5, p=0.017)
At least one marker is elevated	86	90	52.5 (9.9-277.8, p<0.0001)

Abstract# 165

RETROSPECTIVE ASSESSMENT OF CARDIAC CATHETERIZATION AND ANGIOGRAPHY AS A SCREENING TOOL IN CADAVERIC HEART DONORS. Robert S. D. Higgins,[1] Leah E. Bennett,[2] Abelardo DeAnda.[1] *[1]Surgery, Medical College of Virginia, Richmond, VA; [2]UNOS, Richmond, VA.*

Recent efforts to optimize the use of available organ donors stimulated our assessment of cardiac catheterization data and coronary angiography as a screening intervention in cadaveric donors. Between November 1, 1999 and August 31, 2000, there were 5037 cadaveric organ donors recovered in the United States. Of these, 3995 had a validated Cadaveric Donor Registration (CDR) form as of November 2000, providing the necessary data for analysis.

Information regarding coronary angiography (CA) and right heart catheterization (RHC) was requested only on donors for whom the heart was recovered, whether for transplant or for another purpose (e.g., for heart valves). The impact of CA or RHC on utilization of the donor heart was analyzed with a chi-squared test. For the CA data, Fishers exact test was used to determine if an abnormal test excluded the heart from being transplanted. The mean age for donors who had RHC was 36.3 years and for those without was 30.4 years.

		Coronary Angiogram			
Heart Disposition	**Number of Donors**	**Unknown**	**No**	**Yes, Normal Results**	**Yes, Abnormal Results**
Recovered for reason other than TX	564	112(-)	413 (91.4%)	24(5.3%)	15(3.3%)
Recovered for TX but not transplanted	24	7(-)	14(82.4%)	2(11.8%)	1(5.9%)
Transplanted	1618	137(-)	1238(83.6%)	236(15.9%)	7(0.5%)
		Right Heart Catheterization			
Recovered for reason other than TX	564	24(-)	482(89%)	58(10.7%)	
Recovered for TX but not transplanted	24	0	22(91.7%)	2(8.3%)	
Transplanted	1618	6(-)	1395(86.5%)	217(13.5%)	

There was a significant relationship between performing an angiogram and whether the heart was transplanted (p=0.0001). In donors whom had an angiogram, there was a significant relationship between the angiogram result and whether the heart was transplanted (p<0.0001). There was no significant relationship between performing a right heart catheterization and whether the heart was transplanted (p=0.09).**Conclusion:** RHC has limited value in determining whether a donor organ is suitable for heart transplantation. Coronary angiography is a sensitive screening tool in a small percentage of these donors. However, further analysis will be necessary to evaluate the impact of donor age and echocardiographic findings to determine the most appropriate screening tools to optimize donor heart utilization.

Abstract# 166

THE EFFECTS OF TEMPERATURE ON EARLY CARDIAC GRAFT FUNCTION. John W. Fehrenbacher, Carlos A. Labarrere, Nicholas L. Finley, Daniel J. Beckman, David A. Hormuth.

Objective. The temperature of the preservation solution for cardiac transport is associated with subsequent ischemia, reperfusion, and tissue damage. We investigated whether preservation at warmer temperatures was associated with better outcome. Methods. Outcomes of cardiac transplant recipients of donor hearts transported at 8°-12°C (n=13) and at or below 4°C (n=53) were compared. Fisher's exact, Student t-test, or repeated measures analysis were used for data analysis. Results. Hearts transported at 8°-12°C had significantly shorter recovery time (47 min) than those transported at or below 4°C (69.9 min) (p=0.012). Critical care stay was also significantly shortened (71.1 hr vs. 132.1 hr) (p=0.007). The 4°C group required over twice the time on post-operative ventilation assistance (16.2 hr vs. 6.5 hr, p=0.034). The average concentrations utilized for dobutrex (p<0.001) and epinephrine (p=0.001) were significantly decreased in the 8°-12°C group. Thirty-six percent of the 4°C group had myocardial fibrin within microvasculature and cardiomyocytes during the first post-operative month as opposed to 0% of the 8°-12°C group (p=0.025). Operational mortality decreased from 13.5% in the 4°C group to 0% in the 8°-12°C group. There were no significant differences between groups with respect to heart rate, systolic pressure, or SvO2 at 0, 4, and 12 hr after reaching critical care; total hospital stay; average dopamine; and isuprel drip dosage. Conclusions. These results show that increased temperatures in the transporting solution significantly improve cardiac graft function and patient recovery shortly after surgery; this is probably associated with reduced tissue damage.

Abstract# 167

FIRST CLINICAL USE OF THE JARVIK 2000 LEFT VENTRICULAR ASSIST SYSTEM AS A BRIDGE TO TRANSPLANTATION: HEMODYNAMIC AND ECHOCARDIOGRAPHIC ASSESSMENT. Reynolds M. Delgado,[1] Timothy J. Myers,[1] Branislav Radovancevic,[1] Igor D. Gregoric,[1] Kathy Miller,[1] Tehreen Khan,[1] Romeo Majano,[1] Biswajit Kar,[1] Robert K. Jarvik,[2] O. H. Frazier.[1] *[1]Transplant Service, Texas Heart Institute, Houston, TX; [2]Jarvik Heart Inc, New York, NY.*

We studied the Jarvik 2000 axial-flow left ventricular assist system (LVAS) as a bridge to transplantation in 4 patients with end-stage heart failure. Invasive hemodynamic studies and echocardiography were performed preoperatively, during the early postoperative period, and monthly thereafter. Preoperatively, all patients had markedly deranged hemodynamic values (unless otherwise stated, mean values are given): cardiac index (CI), 1.81 L/min/m^2; pulmonary capillary wedge pressure (PCWP), 19.8 mmHg; mean blood pressure (mBP) 66 mmHg. The left ventricular ejection fraction (LVEF) was <20%, and the left ventricular diastolic dimension (LVDD) was 7.6 mm. Forty-eight hours after the implant, the CI was 3.53 L/min/m^{2}the PCWP was 10 mmHg, and the BP was 73.5 mmHg. During the support period, the typical pump speed was 9,000 rpm. The hemodynamic parameters changed markedly as the pump speed increased (Table I). During short pump-off tests, the patients showed signs of heart failure but remained alert and in stable condition. When the pump was reactivated, the CI and PCWP normalized, the pulmonary artery pressure (PAP) decreased, and the pulse pressure (PP) decreased significantly. At high pump speeds, echocardiography showed a decreasing LVDD and an increasing pump flow. Left ventricular unloading was documented by closure of the aortic valve, in most instances, at pump speeds of ≥11,000 rpm. All patients remained hemodynamically stable during the support period and returned to a normal physical status. We conclude that the Jarvik 2000's axial flow results in effective hemodynamic support and left ventricular unloading.

Pump Speed	**CI (L/min/m²)**	**mBP (mmHg)**	**PP (mmHg)**	**PAP (mmHg)**	**PCWP (mmHg)**
0	2.46	58.67	37.00	30.25	15.33
8,000	3.12	68.00	28.00	28.00	13.00
9,000	3.20	71.40	24.71	28.25	12.67
10,000	3.26	73.44	21.00	28.25	11.67
11,000	3.37	74.67	16.60	27.00	11.00
12,000	3.86	81.60	12.40	27.00	10.00

Abstract# 168

HLA ALLOSENSITIZATION DURING VENTRICULAR ASSIST DEVICE SUPPORT CAN BE PREDICTED AND IS RELATED TO DECREASED TRANSPLANT SURVIVAL. Ganesh S. Kumpati,[1] Daniel J. Cook,[2] Eugene H. Blackstone,[1] Jennifer White,[4] Ashraf S. Abdo,[2] James B. Young,[3] Randall C. Starling,[1] Nicholas G. Smedira,[1] Patrick M. McCarthy.[1] *[1]Thoracic and Cardiovascular Surgery, Cleveland Clinic Foundation, Cleveland, OH; [2]Allogen Lab, Cleveland Clinic Foundation, Cleveland, OH; [3]Cardiology, Cleveland Clinic Foundation, Cleveland, OH; [4]Biostatistics and Epidemiology, Cleveland Clinic Foundation, Cleveland, OH.*

Objectives: (1) Identify pretransplant (preTx) predictors of HLA allosensitization during ventricular assist device (VAD) support, as measured by flow cytometry crossmatch (FCXM). (2) Determine if FCXM class I and II positives are associated with decreased transplant (Tx) survival.

Methods: Of 234 patients who had VADs placed between 1991 and July 2000, 155 reached Tx, and 7 are on current support. PreTx variables included age, gender, preVAD ECMO, etiology (ischemic cardiomyopathy [ICM] and all others [nonICM]), transfusions (total and non leukocyte reduced [non LKRD] red blood cells [PRBC] and platelets [Plat]), length of support, and serial T cell cytotoxic PRA (TPRA > 10% at pre VAD, 2 weeks post, highest after 2wk [peak], and immediately preTx) FCXM recipient IgG reactivity against donor T and B lymphocytes was available in 132 cases and was divided into three groups (HLA class 0=T-B-, I=T+B+, and II=T-B+). Correlates of FCXM and TPRA were identified by logistic regression. Survival was estimated by the Kaplan-Meier method.

Results: Female gender (P=.02), earlier VAD date (P=.0003), and younger age (P=.06) were associated with peak TPRA >10%. Peak TPRA >10%, nonICM, and increased preTx nonLKRD PRBC were associated with positive FCXM class I and II reactions (table). FCXM class I and II positives had decreased Tx survival (P=.002, table).

Conclusions: (1) Using FCXM as a measure of allosensitization, preTx risk factors have be identified. (2) HLA allosensitization during VAD support is associated with decreased Tx survival, and warrants possible pre and post Tx treatment changes.

Factor/FCXM class	0 (n=40)	I (n=37)	II (n=55)	P
Peak TPRA	16%	67%	18%	< 0001
NonICM (%)	30	51	38	003
PreTx nonLKRD PRBC (%>0 units)	32	43	44	03
1 yr survival (%)	100	73	86	
3 yr survival (%)	100	65	80	
5 yr survival (%)	100	53	64	

Abstract# 169

NOSOCOMIAL BLOODSTREAM INFECTIONS IN PATIENTS WITH IMPLANTABLE LEFT VENTRICULAR ASSIST DEVICES [LVAD]. Steven M. Gordon, Steven K. Schmitt, Micah Jacobs, Goormastic Marlene, Nicolas Smedira, Mike Yeager, Janet M. Serkey, Katherine Hoercher, Patrick M. McCarthy.

Implantable LVADs are used as a bridge to transplantation but are associated with a high risk of infection including BSI. The primary objectives of our study were to determine the attack rate and incidence of BSI in patients with LVADs. We reviewed the medical records of all patients with implantable LVADs at the Cleveland Clinic with lesser than or equal to 72 hours of support from December 1991 through June 2000. Nosocomial BSI was defined using CDC definitions. An LVAD-related BSI was defined as one where the same pathogen is cultured from the device and the blood with no other obvious source. 214 patients were included in the study (17,831 LVAD days). 140 BSI were identified in 104 patients for an attack rate of 49% and incidence of 7.9 BSI per 1000-LVAD-days 38% of the BSI were LVAD-associated and the most common pathogens causing BSI were Coagulase-negative-staphylococci [n=33], S. aureus and Candida spp. [19 each], and Pseudomonas aeruginosa [16]. A Cox proportional hazard model found BSI in patients with LVADs to be significantly associated with death (Hazard ratio = 4.02, P < 0.001). Fungemia had the highest hazard ratio (10.9) followed by gram-negative (5.1) and gram-positive bacteremias (2.2). We conclude patients with implantable LVADs have a high incidence of BSIs, which are associated with a significantly increased mortality. Strategies to reduce the risk of LVAD-associated BSIs should be a high priority

Abstract# 170

SUCCESSFUL TRANSITION OF LVAD PATIENTS FROM HOSPITAL TO HOME: ARE WE APPROACHING THE GOAL OF CHRONIC SUPPORT? Ashley Sims, Tiffany Buda, Katherine Hoercher, Phyllis Colosimo, Nicholas Smedira, Michael Banbury, Patrick McCarthy.

As confidence in the reliability of left ventricular assist devices (LVAD) has grown among health care providers and waiting time for a suitable organ donor is increasing, more patients are continuing to be discharged to an outpatient setting on mechanical support. We review our experience with discharge of this population. Methods: Between December 1991 and October 2000, 240 LVADS have been implanted (Novocor = 56, Heartmate =184). Since the FDA permitted discharge of these patients in September of 1995, 100 patients have been discharged to an outpatient setting (home/hospital housing=58; extended care facility= 42) to await transplantation (mean length of support = 91 days). In anticipation of hospital discharge, the patient, family, and various community resources require extensive training to ensure a safe and successful transition to the outpatient setting. Readmission was required for 39 of these patients. The reason for readmission is listed in the table below. This experience has demonstrated that 1) there is a high risk of cerebral vascular accident associated with Novacor use; 2) there is a high risk of device malfunction or failure with earlier versions of Heartmate, and 3) there remains a constant risk of infection in both devices. Although we are closer to approaching the goal of chronic support as an alternative to transplantation, the major clinical problem with these devices continues to be infection. As the infection problems are addressed in the current or second generation pumps, we may see results that are equivalent to transplantation

Hospital Readmissions	
Infection	42%
Pump Malfunction/Failure	28%
Neuro Event	18%
Other	22%

Concurrent Session 19: Liver Transplantation: Hepatitis C II

Abstract# 171

CHANGES IN HEPATITIS C VIRUS (HCV) POPULATION IN SERUM AND PERIPHERAL BLOOD MONONUCLEAR CELLS IN CHRONICALLY HCV-INFECTED PATIENTS RECEIVING LIVER GRAFT FROM HCV-INFECTED DONORS. H. Vargas,[2] L.F. Wang,[2] J. Wilkinson,[1] M. Radkowski,[1] T. Laskus,[1] J. Rakela.[1] *[1]Mayo Clinic Scottsdale, Scottsdale, AZ; [2]University of Pittsburgh Medical Center, Pittsburgh, PA.*

We have previously reported that among HCV-infected recipients who receive hepatic allograft from an HCV-infected donor, either the donor's or the recipient's HCV strain predominates in serum (Gastroenterology 117:149-153, 1999). **Purpose:** To determine whether, 1) the takeover of one HCV strain is exclusionary and long-lasting and 2) the takeover of one HCV strain can also be observed in an extrahepatic compartment permissive to HCV replication (PBMCs) in HCV-infected recipients who received a graft from an HCV-infected donor. **Patient & Methods:** We have studied HCV isolates from sequential serum and PBMCs samples from 11 and 8 patients, respectively. We developed strain-specific RT/PCR for HCV isolates from serum and PBMC origin; these assays detected minor HCV sequences at a concentration $1:10^4$ - 10^6 below that of a major sequence. **Results:** Five of 11 patients retained their original (recipient) infecting HCV strain and the donor HCV strain was detected only transiently. In the remaining 6 patients, the recipient HCV strains was transiently detected in the first 2-3 weeks after which only the donor HCV strain was consistently present. It was noteworthy that in 1 of 6 patients, the original recipient strain was transiently detected 8 months after transplantation when the donor HCV strain was predominantly present. HCV isolates from PBMCs that were studied in 8 of the 11 patients, paralleled closely in terms of their origins (donor vs. recipient) with those obtained in serum samples. **Conclusion:** The takeover of one strain by another is complete and long-lasting; however, the minor strain can be found intermittently by sensitive, strain-specific assays. Viral population in PBMCs evolve synchronously with those present in serum that are liver-derived We postulate that the outcome of HCV superinfection is determined by direct competition between infecting strains and, one competing strain, in the absence of niche differentiation, will eventually eliminate or exclude the other.

Abstract# 172

DISTINCTION OF ACUTE CELLULAR REJECTION FROM RECURRENCE OF HCV THROUGH INTRAGRAFT GENE EXPRESSION PATTERNS. R. Sreekumar,[1] R. Wiesner,[1] C. Rosen,[1] M. Charlton.[1] *[1]Transplant Center, Mayo Clinic and Foundation, Rochester, MN.*

Introduction: Treatment of acute cellular rejection (ACR) is associated with increased viral load, more severe histological recurrence and attenuated patient and graft survival following liver transplantation for HCV. Recurrence of HCV may be difficult to distinguish histologically from ACR. Because the immunological mechanisms of ACR and HCV recurrence differ, we hypothesized that ACR is associated with the expression of a specific subset of immune activation genes that may serve as a diagnostic indicator of ACR. **Aim** To study intragraft gene expression patterns in ACR and during recurrence of HCV in HCV-infected recipients. **Methods**: High density synthetic oligonucleotide microarrays were utilized to determine relative intragraft gene expression profiles in four HCV-infected recipients with steroid responsive ACR by Banff criteria and four HCV-infected recipients with recurrence of HCV. Total RNA was extracted from snap frozen liver biopsies **Results**. A summary of genes relatively overexpressed during ACR is shown below. Values indicate the fold difference in abundance of intragraft RNA for individual genes during ACR:

Relative Ingragraft Expression in Recipients With ACR vs. HCV Recurrence

MHC expression	T-cell Activation/Function
↑1.6 MCH class I	↑4.6 TNF-α converting enzyme
↑3.7 MHC class-II	↑3.2 TNF-α
Complement Activation	↑1.9 T-cell activation protein
↑1.6 Ser/Thr Protein Kinase	↑2.9 IL-2
↑1.8 Complement Component 1q	↑2.7 Aff-1
B-cell Activation	↑2.9 Granzyme B
↑2.1 IL 10	↑2.4 TGF-β
↑2.1 P-Selectin	↑3.9 TNF related apoptosis inducing ligand

Conclusion: In HCV-infected liver transplant recipients, ACR is associated with an intragraft gene expression profile that is distinct from that seen during recurrence of HCV. It may be possible, using intragraft gene expression profiles, to differentiate ACR from recurrence of HCV following liver transplantation. Whether peripheral T-cells yield similar results is the subject of further investigation.

Abstract# 173

STEROID-FREE INDUCTION WITH TACROLIMUDS AND DACLIZUMAB IN LIVER TRANSPLANT RECIPIENTS WITH HEPATITIS C - A PRELIMINARY REPORT OF A PROSPECTIVE RANDOMIZED TRIAL.

Tomoaki Kato,[1] Guy Neff,[2] Marzia Montalbano,[2] Olivia M. Hung,[1] Reynel Lavandera,[1] Deborah Weppler,[1] David M. Levi,[1] Jose R. Nery,[1] Seigo Nishida,[1] Antonio D. Pinna,[1] Rajender Reddy,[2] Christopher B. O'Brien,[2] Philip Ruiz,[1] Andreas G. Tzakis.[1]
[1]Division of Liver and GI Transplant, University of Miami, Miami, FL; [2]Division of Hepatology, University of Miami, Miami, FL; [3]Division of Immunopathology, University of Miami, Miami, FL.

Purpose Recurrence of Hepatitis C virus (HCV) infection continues to be a major problem following orthotopic liver transplantation (OLT). Immunosuppression (ISP) therapy undoubtedly plays a role in the process of recurrence. We report the safety and efficacy of steroid-free induction in patients receiving OLT with HCV. **Methods** OLT recipients with HCV were prospectively randomized to receive either tacrolimus/daclizumab (study arm) or tacrolimus/corticosteroid (control arm) for induction ISP. Daclizumab was given at a dose of 2mg/kg intraoperatively for patients in the study arm, then 1mg/kg at weeks 1, 2, 4, 6, 8 and 10. Patients in the control arm received regular doses of corticosteroid, and tapered with discontinuation by week 12. Treatment of mild to moderate rejection consisted of a corticosteroid bolus with or without 5 days course of tapering doses, and OKT 3 for resistant or severe rejection. Treatment with ribavirin and interferon was started for HCVRNA >10mil in b-DNA assay. **Results** Twenty-eight patients (14 in each arm) were enrolled as of 11/2000. In the study arm, 4 patients were excluded from the analysis; 2 due to rapamycin conversion for tacrolimus toxicity and two due to retransplant. Of the remaining 10 patients in the study arm, 9 were steroid-free at 1-12 months follow-up and one patient required steroid for the treatment of rejection. In the control arm, 3 patients were excluded; one due to early death and two converted to rapamycin therapy. In the remaining 11 patients in the control arm, acute rejection occurred in four patients. Total dose of corticosteroid in the control arm ranged from 2.56GM to 5.76GM (mean 3.7 GM) in the first three months. Biopsy proven recurrent HCV occurred in 3 patients in the control arm (27%) while none of the study group developed recurrence at a short-term follow-up. **Conclusions** Although the results are preliminary, it appears that steroid-free induction for liver transplant recipients with HCV infection is safe and well-tolerated. In addition, recurrence of HCV infection may be reduced in patients with steroid-free induction.

Abstract# 174

INTRAHEPATIC CYTOKINE EXPRESSION ASSOCIATED WITH HEPATITIS C VIRUS (HCV) RECURRENCE POST LIVER TRANSPLANTATION FOR IICV INFECTION.

Amany Zekry,[1] Alex Bishop,[1] Geoffrey W. McCaughan.[1] *[1]AW Morrow Gastroenterology & Liver Centre, Sydney University, Sydney, NSW, Australia.*

Background: Post liver transplantation, acute rejection associated with HCV, chronic hepatitis and cholestatic hepatitis are well-recognized clinical sequelae of HCV graft reinfection. Distinguishing the two former groups may represent a clinical challenge.In addition, liver injury in cholestatic hepatitis has been suggested to be due to direct cytopathic effects of the virus. **Aim:** To characterize the intrahepatic cytokine response associated with reinfection of the graft with HCV. **Methods:** We examined intrahepatic expression of Th1 cytokines: IL2, IFN-γ, TNF-α and Th2 cytokines: IL4 and IL10 in 47 liver specimens. These included: A) 3 groups in the post transplant setting; 15 acute rejection with HCV (AR-HCV), 6 cholestatic hepatitis, and 13 recurrent chronic hepatitis C (CHI). B) 2 non-transplanted groups; 8 chronic hepatitis C infection (CH), and 5 normal livers. Total RNA was prepared from liver biopsies and reverse transcribed. Intrahepatic cytokines were quantified by real-time PCR. Intrahepatic viral loads were measured using an Amplicor monitor. **Results**: Compared to CHI, the AR-HCV expressed more IL10 and IL4 ($P = 0.02$). The CHI and CH groups had a similar cytokine profile with upregulation of IFN-γ and IL2. There was also increased expression of IL10 and IL4 in the cholestatic group compared to the CHI ($P = 0.011$) and CH ($P = 0.005$) groups. In addition, at the time of the liver biopsies, intrahepatic viral loads were highest in the cholestatic group compared to the AR-HCV, CHI and CH groups ($P = 0.03$). There was no difference in the intrahepatic viral loads between the AR-HCV and CHI groups. **Conclusions:** 1) The cholestatic group demonstrated upregulation of Th2 cytokines IL10 and IL4 in addition to high viral load. This suggests that in the cholestatic group, a Th2 immune response may favor viral replication and graft damage. 2) Similar intrahepatic cytokine profiles existed in the immunosuppressed and non-immunosuppressed chronic HCV patients with upregulation of Th1 cytokines.3) Acute rejection associated with HCV recurrence is characterized by increased expression of Th2 cytokines IL10 and IL4 compared to immunosuppressed chronic hepatitis. The intrahepatic viral load was however similar in both groups

Abstract# 175

TRANSMISSION AND EVOLUTION OF VIRAL QUASISPECIES IN HEPATITIS C VIRUS POSITIVE LIVER TRANSPLANT RECEIPIENTS.

J. Rakela,[1] T. Laskus,[1] J. K. Wilkinson,[1] M. Radkowski,[1] H. Vargas,[2] V. Balan,[1] D. D. Douglas,[1] M. E. Harrison,[1] A. Moss,[1] D. C. Mulligan.[1] *[1]Mayo Clinic Scottsdale, Scottsdale, AZ; [2]University of Pittsburgh Medical Center, Pittsburgh, PA.*

Hepatitis C virus (HCV) infection is the leading cause of chronic liver disease requiring liver transplantation(LTx). **Purpose:** 1) To elucidate the patterns of HCV quasispecies transmission and early evolution following LTx and 2) to determine whether they have predictive value with respect to disease recurrence. **Patients and Methods:** We have analyzed 17 HCV-positive patients who underwent LTx. Fifteen patients had end-stage HCV-related liver disease while in the remaining 2 subjects the reason for LTx was hepatocellular carcinoma and hepatic hemangioendothelioma, respectively. Serum samples were drawn before LTx and then 1-2 times a week for the first 1-2 months. For analysis of viral quasispecies, fragments of E2 region encompassing hypervariable region 1 were amplified by nested RT-PCR and compared by single strand conformational polymorphism (SSCP). E2 region quasispecies was analyzed in all 17 patients.**Results:** In 12 out of 17 (71%) subjects the SSCP band patterns before and early after transplantation were identical suggesting that all variants within the pretransplant quasispecies established infection in the new liver. Five of 17 patients showed "bottleneck" phenomenon; these 5 patients had minimal liver test abnormalities and did well clinically. In 4 subjects (24%) no changes with respect to the number or position of SSCP bands were observed during follow-up; 2 of those 4 patients died of recurrent HCV and multiple organ failure, respectively. In the remaining 13 patients (76%) either additional viral variants developed or both the position and number of viral variants changed. In 8 out of 13 patients (62%) showing SSCP evolution, quasispecies composition stabilized after the first 2-3 weeks and remained unchanged thereafter. The number of SSCP bands observed in pretransplant sample was 7(median) with a range of 4 to 13. **Conclusions:** In the majority of HCV-infected transplant recipients, E2 quasispecies composition before and early after LTx are identical suggesting that all viral variants are competent to infect the graft. Changes within E2 are common after LTx but quasispecies composition often stabilizes after the first 2-3 weeks. Those patients with "bottleneck" pattern tended to have a mild clinical course with minimal liver test abnormalities. Those patients with no "evolution" of quasispecies had a tendency to worse clinical outcome.

Abstract# 176

IS MYCOPHENOLATE BENEFICIAL IN THE TREATMENT OF RECURRENT HEPATITIS C FOLLOWING LIVER TRASNPLANTATION?

Gregory A. Smallwood,[1] Michael E. de Vera,[2] Laurel Davis,[2] Enrique Martinez,[3] Andrei C. Steiber,[2] Thomas G. Heffron.[2]
[1]Pharmacy, Emory University Hospital, Atlanta, GA; [2]Surgery, Emory University School of Medicine, Atlanta, GA; [3]Medicine, Emory University School of Medicine, Atlanta, GA.

Background: Ribavirin, a competitive inosine monophosphate dehydrogenase (IMPDH) inhibitor, has been shown to have antiviral activity and is useful in treating hepatitis C (HCV) infection in combination with a -interferon. Mycophenolate mofetil (MMF) is a prodrug of mycophenolic acid, which is a non-competitive IMPDH inhibitor and is useful as an immunosuppressive agent. **Objective**: The aim of this review is to determine if MMF is beneficial in patients being treated with a -interferon and ribavirin for recurrent HCV following liver transplantation. **Methods:** From November 1997 through May 2000, 64 patients, all serum RNA positive for hepatitis C, were transplanted for complications of chronic hepatitis C. The control group (n = 33) was given dual immunosuppression without mycophenolate and the treatment group (n = 31) received triple immunosuppression with mycophenolate. Recurrent HCV was defined as an increase in liver enzymes with chronic hepatitis found on liver biopsy in the absence of features of cellular rejection. Patients with recurrence HCV as defined by liver biopsy were begun on a -interferon and ribavirin. Patients were compared on the basis of biopsy scores, days to recurrence, viral genotype, hospitalizations, patient and graft survival, and response to alpha-interferon and ribavirin. **Results in table 1.** There were no differences between responders vs. non-responders in relation to patient demographics or viral genotype in reference to mycopholate usage. **Conclusion:** Mycophenolate incorporated into the liver transplant immunosuppressive regimen for HCV patients did not delay recurrence or improve response to ribavirin/interferon. Clinical outcomes and histologic features of recurrent hepatitis C were similar, regardless of the use of mycophenolate.

	MMF Group	Control Group	p value
Number	31	33	
Recurrent HCV	15 (48.4%)	17 (51.5%)	NS
Days to recurrence	254 (± 302)	237 (± 325)	NS
Biopsy Score	4.2 (± 0.8)	4.4 (± 0.4)	NS
Biochemical Response	12 (80.0%)	15 (88.2%)	NS
Viral Clearance	0	3	NS
2 Year Survival	82.5 (± .06)	78.6 (± .06)	NS

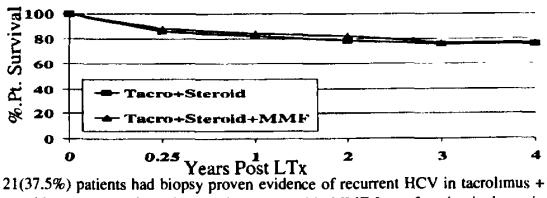
Table #1 - Results from treatment outcomes with interferon/ribavirin for liver transplant recepients with recurrent HCV

Abstract# 177

ROLE OF MYCOPHENOLATE MOFETIL (MMF) IN LIVER TRANSPLANTATION (LTX) FOR HCV RELATED END STAGE LIVER FAILURE (A PROSPECTIVE RANDOMISED TRIAL).

Ashok B. Jain,[1] Randeep S. Kashyap,[1] John J. Fung.[1] *[1]Surgery, T.E. Starzl Transplantation Institute, Pittsburgh, PA.*

Introduction: MMF has been shown to have antiviral effects. Its role in primary LTx for HCV related end stage liver failure is not known. The aim of the present study is to compare the outcome of patients who received liver transplant for HCV infection with MMF and compare with those who did not receive MMF. Study population: Between 8/17/95 to 5/16/98, 106 patients underwent liver transplant for HCV related disease. 56 patients were randomized to tacrolimus + steroid and 50 were randomized to tacrolimus + steroid + MMF. All patients were followed until 1/15/2000. Mean follow up 3.1±0.8 years. Patient survival, graft survival, and rate of recurrence of HCV were studied prospectively. Results: During the study 30(67%) patients (who were randomized to tacro+steroid+MMF) discontinued MMF after 80± 110 days, median 36 (range2 to 434 days) after LTx for infection 13(43.3%), hematological disorders 8(26.6%), gastrointestinal complications 5 (16.7%) and other miscellaneous reasons 4(13.3%). Nine patients received MMF (who were randomized to tacro+steroid), 6 for reiection and 3 for neurotoxicitv. Patient survival for both groups is shown below.



21(37.5%) patients had biopsy proven evidence of recurrent HCV in tacrolimus + steroid group Vs 17(34%) in tacrolimus + steroid +MMF. Liver function is shown in the table below. Conclusion: No difference in patient survival or graft survival was observed with use of MMF. Similarly the rate of recurrence for HCV and LFT were also comparable in both groups. Either MMF has no inherint anti-HCV effect or initially high doses of tacrolimus and steriods reduces any beneficial effect of MMF.

Abstract# 178

COMBINATION OF INTERFERON-ALPHA AND RIBAVIRIN FOR RECURRENT HEPATITIS C AFTER ORTHOTOPIC LIVER TRANSPLANTATION—IMPACT ON HISTOPATHOLOGY, VIREMIA AND GRAFT FUNCTION.

Arno Kornberg, Merten Homman, Andrea Tannapfel, Rigo Voigt, Thomas Grube, Johannes Scheele.

Introduction: Hepatitis C recurrence after liver transplantation (OLT) is a serious problem. Aim of this study was the assessment of feasibility and efficacy of a combined long-term therapy with Interferon-alpha (IFN) and Ribavirin (Rb) in patients with recurrent Hepatitis C (HCV) after OLT. Patients and methods: Between 1995 and 2000, 15 patients with bioptically and serologically confirmed recurrence of HCV were treated with IFN (3 MU/3 times a week) and Rb (3x200mg/day) for a minimum of 12 months. Therapy was continued if serological HCV-RNA clearance could not be achieved, otherwise Rb-monotherapy began. Every three months, biochemical and virological response was evaluated. 6 months after start of therapy, routine biopsy of the graft for determination of the hitological activity index (HAI) was performed.

Results: Therapy started between 3 and 128 weeks after OLT. Actual follow-up after beginning of therapy varies between 6 and 45 months. In 14 cases, genotype was 1b (93%). Mean HAI dropped from 6,8 to 4,0 after 6 months of therapy 58% of patients with a follow-up of 12 months (7/12) and 66% of patients with a follow-up of 36 months (2/3) revealed serological HCV-RNA-clearance. Therapy lead to significant reduction of aminotransferases during the first month of therapy (ASAT 0,94/0,20; ALAT 1,5/0,30)(p=0,004). Virological responders showed lower aminotransferases than partial responders after 12 months of therapy (ASAT 0,19/0,22)(p=0,34). In addition, graft function (prothrombin time, factor V) improved slightly. 93% of patients had leucopenia that reqired therapy. 33% of patients developed anemia with need for transfusion. In 4 patients (26,6%) dosage of IFN or/and Rb had to be reduced between 1 and 12 months after beginning of therapy. In two further patients therapy had to be stopped because of severe leucopenia and development of depressive syndrom.

Conclusion: Long-term combination therapy of IFN and Rb is feasible and leads to complete biochemical response in more than 50% of patients. But also partial responders profit from improved graft function. Therapy can be complicated by severe side effects that are reversible after reduction or discontinuation. The impact on long-term patient and graft survival remains speculative.

Abstract# 179

EFFECT OF INTERFERON-α AND RIBAVIRIN ON LIVER HISTOLOGY IN LIVER TRANSPLANT PATIENTS WITH RECURRENT HEPATITIS C.

Aejaz Nasir,[2] Michael E. de Vera,[1] Gregory A. Smallwood,[1] Andrei C. Stieber,[1] Thomas G. Heffron.[1] *[1]Division of Liver Transplantation, Emory University Hospital, Atlanta, GA; [2]Dept. of Pathology, Emory University Hospital, Atlanta, GA.*

We have questioned the efficacy of interferon-α and ribavirin (I/R) for the treatment of recurrent hepatitis C virus (HCV) infection after liver transplantation (LT). Here we evaluate the histologic outcomes of patients treated with this regimen. *Methods* : LT patients with recurrent HCV infection (elevated ALT, positive serum HCV RNA, and biopsy-proven hepatitis without rejection) have been treated with interferon-α (1.5-3 million units SQ tiw) and ribavirin (400-1,000 mg po daily). LFTs and HCV RNA were monitored. A liver biopsy was performed when clinically indicated and after 12 months of treatment Hepatitis activity index (HAI) and fibrosis scores were determined. *Results* . Thirty-two patients have been treated for at least 3 months, including 13 who have been on 12 or more months of therapy. Eighteen patients have had follow-up biopsies and comprise the study group (age, 53 ± 8 years; 13 males). Ten patients (Group I) had histologic evidence of progression (mean HAI score from 3.4 ± 1.8 to 4.5 ± 1.4) despite I/R therapy. None had fibrosis prior to I/R but 8 were found to have fibrosis in follow-up biopsies (mean fibrosis score, 2.4, range 1-5). Eight patients (Group II) had stable or improved HAI scores (mean, 4.6 ± 1.6 to 3.6 ± 1.4). Only 1 patient in this group had pre-treatment fibrosis and this improved with therapy (mean fibrosis score, 5 to 3). The mean duration of I/R therapy for patients in both groups were similar as were the number of patients in each group who had a biochemical or virological response to I/R. *Conclusions* : Despite interferon-α and ribavirin therapy, a significant number of patients showed histologic progression of recurrent hepatitis C in liver allografts. Biochemical response, virologic response, or pre-treatment HAI scores were not predictive of histologic outcome. A more efficacious treatment regimen for recurrent HCV after liver transplantation is needed.

	Group I	Group II
Mean duration of treatment	11 ± 3 months	12 ± 5 months
Pre-treatment HCV RNA (I U)	4,598,366 ± 1,428,271	3,766,012 ± 1,654,667
Biochemical response	7/10 patients	5/8 patients
Complete virological response	1/10 patients	1/8 patients

Concurrent Session 20:
Islet Transplantation and Long-Term Results of Pancreas Transplantation

Abstract# 180

INSULIN INDEPENDENCE AFTER SINGLE-DONOR ISLET TRANSPLANTATION IN TYPE 1 DIABETES WITH hOKT3γ-1 (ala-ala), SIROLIMUS, AND TACROLIMUS THERAPY.

Bernhard J. Hering,[1] Raja Kandaswamy,[1] James V. Harmon,[1] Jeffrey Ansite,[1] Sue Clemmings,[1] Tetsuya Sakai,[1] Julianne Zabloski,[1] Kathy Duderstadt,[1] Kathy Jacobsen,[1] Carrie Gibson,[1] Steve Paraskevas,[1] Peter Eckman,[1] Masahiko Nakano,[1] Toshiya Sawada,[1] Hui J. Zhang,[1] David E.R. Sutherland,[1] Jeffrey A. Bluestone.[2] *[1]Dept of Surgery, University of Minnesota, Minneapolis, MN; [2]Diabetes Center, University of California, San Francisco, San Francisco, CA.*

Background . The Edmonton group's report of consistent diabetes reversal after sequential islet transplants from ≥ 2 donors marks a turning point in islet transplantation. We sought to determine whether optimization of pancreas preservation, islet processing, and induction immunotherapy enables diabetes reversal after single-donor islet transplantation.

Methods: The new features of the protocol are: 1. Two-layer (PFC/UW) pancreas preservation; 2. isopycnic islet separation on iodixanol gradients, 3. islet culture for 2 days prior to transplant; 4. prospective islet potency testing; and 5. induction immunosuppression with hOKT3γ-1 (ala-ala), a new generation anti-CD3 antibody proposed to selectively inhibit and tolerize the inflammatory .the inflammatory subset of auto and alloreactive Th1 cells while sparing the suppressive Th2 T cell subset, thus mitigating inflammation and restoring peripheral self-tolerance. The protocol used in this study is otherwise identical to the Edmonton protocol.

Results: To date, 3 C-peptide negative, non-uremic, Type 1 diabetic patients with hypoglycemia unawareness have received transplants. Current follow-up is 96, 95, and 33 days without evidence of rejection. Cold ischemia time was 6, 8, and 8 hrs, the number of islets transplanted was 8.2, 15.2, and 11.4 KIE/kg body weight with a purity of 40, 50, and 80%, a viability of 98, 98, and 98%, and an in vitro insulin stimulation index of 17, 14, and 7, respectively. All 3 patients achieved and sustained insulin independence and freedom from hypoglycemia with mean HbA1c levels (%) of 6.1 at 1, 5.5 at 2, and 5.1 at 3 months. Exogenous insulin was gradually tapered in patient #1, reduced to ≤5 U/d starting on day 2 in patients #2 and #3, and discontinued on days 37, 33, and 26 posttransplant. Serious adverse events have been limited to a transient rash in 1 patient and temporary, clinically inconsequential neutropenia in 3 patients

Conclusion: The preliminary results suggest the feasibility of successful single-donor islet transplantation in selected patients with currently available tools.

Abstract# 181

SOLITARY ISLET CELL TRANSPLANTATION FROM A SINGLE DONOR FOR PATIENTS WITH TYPE 1 DIABETES AND HYPOGLYCEMIA UNAWARENESS. Rodolfo Alejandro, Aileen M. Caulfield, Tatiana Froud, Jacqueline V. Ferreira, Lisa C. Rothenberg, Ismail H. Al-Abdullah, Andres Boker, David A. Baidal, Topaz J. Kirlew, Norma S. Kenyon, Camillo Ricordi.

Six consecutive adult patients with type 1 diabetes and severe hypoglycemia unawareness received islet cells from a single donor, as well as 2 infusions of CD34+ enriched donor stem cell infusions from the same donor. In an attempt to limit inflammation subsequent to intrahepatic islet infusion, thus enhancing engraftment, patients were treated with a single dose of infliximab, a TNFα specific monoclonal antibody at the time of transplant. Immunosuppression consisted of induction therapy with daclizumab and maintenance treatment with tacrolimus and sirolimus. Results are summarized in the table below

Patients received a mean of 8629+/-2102 IEQ/kg body weight Three patients achieved insulin independence for 5, 17 and 50 days respectively, after which a small dose of insulin was required. Overall, mean daily insulin dose decreased from 43.5+/-19.8 to 17.2+/-14.1 U (p=0.0025). Mean HbA1c decreased from 7.5+/-1.1 to 5.9+/-0.8 % (p=0.005). Mean amplitude of glycemic excursions (MAGE) decreased from 150+/-62 to 59+/-18 mg/dl (p=0 009).

C-peptide is detectable in the serum of all 6 patients after a median follow-up of 4 months (range 2-7), no rejection episodes or recurrence of autoimmune disease have occurred Severe hypoglycemic episodes or comas have not occurred since transplant in any of the recipients, and 4/6 patients claim improved hypoglycemia awareness.

These data show that single donor islet cell transplantation can consistently improve metabolic control even in the absence of insulin independence. A further aim of this protocol is to examine the possibility of discontinuing immunosuppression subsequent to confirmation of donor-specific unresponsiveness induced through CD34+ enriched donor stem-cell infusions under the cover of the tolerance promoting effect of sirolimus.

Patient	IEQ/kg	Insulin/kg/d-PRE	Insulin/kg/d-POST	HbA1c(%)-PRE	HbA1c(%)-POST
1	7045	0 8	0 42	6 4	5 6
2	12484	0 69	0 12	7 7	5 9
3	8012	0 58	0 11	9 5	6 4
4	7039	0 48	0 07	7 2	4 6
5	7521	0 46	0 34	6 9	6 1
6	9547	0 73	0 45	7 5	6 3

Abstract# 182

EVALUATION OF THE LIVER PRE AND POST PERCUTANEOUS TRANSHEPATIC INTRAPORTAL ISLET CELL TRANSPLANTATION. Tatiana Froud, Aileen M. Caulfield, Jacqueline V. Ferreira, Andres Boker, David A. Baidal, Lisa C. Rothenberg, Robin Harbach, Yrizarry Jose, Camillo Ricordi, Rodolfo Alejandro. *[1]Diabetes Research Institute, University of Miami Hospitals and Clinics, Miami, FL.*

Current techniques in islet cell transplantation (ICT) use the percutaneous transhepatic route to access the hepatic portal vein into which the islets are infused. Engrafting islets lodge in distal portal radicals thereby causing acute elevation of the portal pressure and liver ischemia, manifested as an elevation of liver enzymes. Current improved isolation techniques have abrogated those rare complications previously associated with ICT such as portal vein thrombosis, acute portal hypertension, DIC and death. Islet infusion, however, can result in hepatic inflammation, which this paper seeks to further evaluate

Fourteen transplant recipients received a mean of 8.2 ml total tissue volume (TTV) (range 3-12); in thirteen (93%) the portal pressure rose during infusion. Mean opening pressure was 10.3 mm Hg (range 5-19), mean peak pressure was 18.1mm Hg (range 5-32) and mean rise was 7.9 mm Hg (range 0-20). Delayed portal pressure was obtained in 7 patients, in this group the mean peak pressure was 21.9 mm Hg (range 6-20), which decreased to 18.1 mm Hg (range 14-31) after 15 minutes. Rise in portal pressure correlated directly with amount of tissue infused (p<0.03).

Liver enzymes (ALT, AST and alkaline phosphatase) rose in 12 of 13 patients. Initial rise commenced 4 days post procedure (range 2-10), peak rise occurred at 8 days (range 4-22) and enzymes had normalized in all but 1 patient by 42 days (mean 29) without intervention. Elevation of liver enzymes did not correlate with TTV. In 5 patients there was a second elevation at a mean of 39 days (range 22-64) post transplant The etiology of this is unknown; possibilities include a side effect from adjuvant medication or subacute inflammatory hepatitis, which could have implications on islet function/survival. The percutaneous procedure could contribute to initial elevation of enzymes, particularly if multiple needle passes are required to access the portal vein. Current technique utilizes ultrasound guidance thereby reducing the number of needle passes required.

In conclusion, to prevent hepatic complications from intrahepatic ICT, ultrasound guidance is recommended, total tissue volume of transplanted islets should be <10 ml, portal pressure should be maintained below 25 mm hg and liver function should be closely monitored for at least the first three months post transplant.

Abstract# 183

TWO-LAYER (UNIVERSITY OF WISCONSIN SOLUTION/ PERFLUOROCARBON) METHOD OF HUMAN PANCREAS PRESERVATION PRIOR TO ISLET ISOLATION IMPROVED THE YIELD AND VIABILITY. Shinichi Matsumoto,[1,2] Theodor H. Rigery,[1] Sabrina A. Qually,[1] Ian Sweet,[1] Yoshikazu Kuroda,[4] R. Brian Stevens.[2] *[1]Puget Sound Blood Center, Seattle, WA; [2]Surgery, University of Washington, Seattle, WA; [3]Diabetes Endocrinology Research Center, University of Washington, Seattle, WA; [4]First Department of Surgery, Kobe University, Kobe, Hyogo, Japan.*

Purpose: The purpose of this study is to compare the results of human islet isolation from pancreata preserved by TLM or UW storage prior to islet isolation. In addition, we examined whether the TLM is useful for the Ricordi method compared to our open pan method.

Methods: Human pancreata (n=8) from organ donors were removed from UW and divided in half. Each half was preserved in either UW or the TLM for approximately 24 hours. Islets were isolated from each half by enzymatic digestion using the pan method followed by purification. We isolated an additional 4 TLM preserved human pancreata using the Ricordi method. We measured islet yield, viability (AO/PI staining) and glucose stimulated insulin release (stimulation index). Isolated islets from the TLM group were perifused to verify dynamic response to glucose. Results: Islet yields from TLM-preserved pancreata were significantly better than yields from UW-preserved pancreata, irrespective of isolation method[UW(Pan) vs TLM (Pan) [P<0.001]; UW (Pan) vs TLM (Ricordi)[P<0.05]]. Islet viability in TLM groups was significantly improved compared to UW group irrespective of isolation method [UW (Pan) vs TLM (Pan) [P=0.06], UW (Pan) vs TLM (Ricordi) [P<0.01]). Glucose stimulated insulin release or stimulation index was improved with islets obtained from TLM preserved pancreata irrespective of the isolation method (Pan or Ricordi). Although the pan method generates more islets, the Ricordi method generates more viable islets (Table). Islets obtained from TLM-preserved pancreata show a biphasic response to high glucose during the perifusion analysis. Conclusion: The TLM of pancreas preservation prior to islet isolation improves the yield and viability of isolated islets from human pancreata heretofore deemed unusable due to prolonged preservation, thus expanding the donor pool for islets.

Group	Storage Time (h)	Yield(Digest) (IE/g)	Yield(Pure) (IE/g)	Viability (%)	Stimulation Index
UW(Pan)	37±5	2794±467	1959±360	64 0±7 5	1 8±0 5
TL(Pan)	35±5	6864±854	5080±943	83 6±5 3	2 7±0 7
TL(Ricordi)	27±4	6120±1914	2944±882	94 6±2 3	3 3±1 2

Abstract# 184

PRE AND PERI-OPERATIVE MANAGEMENT INFLUENCES THE CLINICAL OUTCOME OF ISLET TRANSPLANTATION. Paola Maffi, Federico Bertuzzi, Daniela Guiducci, Carlo Socci, Luca Aldrighetti, Rita Nano, Marco Salvioni, Antonio Secchi, Valerio Di Carlo. *[1]Scientific Institute San Raffaele, Milan, Italy.*

The aim of our study was to characterized the main factors affecting the long-term function in human islet transplantation. In 1989-2000,38 patients underwent islet transplantation. In all cases the isolation method was the modified automated and the seat of infusion was the portal vein.The study follow-up was divided between 2 Eras (1st Era 1989-1996:21 cases; 2nd Era 1998-2000:17 cases) according to different pre/perioperative management.The pre-operative management of 2nd Era was characterized by in vitro measurement of cytosolic calcium concentration (Ca2+)i,an early indicator of islet viability, and the peri operative management by the introduction of adjuvant therapy (association of metformin, nicotinamide, pentoxifylline).During the 2nd Era mycofenolate mofetyl instead of azathiopnne and low doses and/or steroids withdrawal were used; while ATG for induction and cyclosporin for chronic treatment were used in both groups. The following parameters were considered during the whole follow-up:fasting and after stimulus C-peptide secretion (C-Pep),glycaeted hemoglobin (HbA1c), Exogenous Insulin Requirement (EIR),insulin independence. In all cases C-Pep >1 ng/ml was observed after the procedure, without any case of primary non function. The fasting C-Pep considered during the 2 weeks after the transplant was significantly different between the 2 Eras (1st Era 1.9±0.9 ng/ml; 2nd Era 3.1±0.8 ng/ml, p<0.001). The rate of islet rejection was: 3 cases (14%) in the 1st Era, 2 cases (17%)in the 2nd Era, without any difference. The success of transplant in terms of HbA1c values (%) was not different at 1 month (1st Era: 5.0 ±2.9; 2nd Era: 7±1.1) but it resulted significant different at 1 year (1st Era: 7.9±1; 2nd Era: 6.7±1.4, p<0.05). The EIR was reduced < 50% of the pre-transplant dose in 10 cases (48%) and in 15 cases (88%) respectively during the 1st and the 2nd Era. The insulin independence occured in 7 cases (33%) in the 1st Era and in 10 cases (59%) in the 2nd Era;2/7 patients maintained the insulin independence for more than 1 year during the 1st Era, while 3/3 (the other 7 had a follow-up < 1 year) during the 2nd Era . In conclusion: the pre/peri-operative management seems to influence the clinical outcome of the islet transplant in terms of function survival and of improvement in metabolic control; the use of micofenolate mofetyl and low doses or steroids free therapy do not affect the rejection rate.

Abstract# 185

SUSTAINED IMPROVEMENT IN CARDIOMYOPATHY FOLLOWING PANCREAS-KIDNEY TRANSPLANATION: A FOUR YEAR FOLLOW-UP. Patricia A. Cowan,[1,2] Donna K. Hathaway,[1] Mona N. Wicks,[1] Robert J. Stratta,[2] M. H. Shokouh-Amiri,[2] A. Osama Gaber.[2] [1]*Nursing, UN of TN Health Science Center, Memphis, TN;* [2]*Surgery, UN of TN Health Science Center, Memphis, TN.*

The purpose of this study was to determine if early improvements in cardiomyopathy, associated with diabetes and uremic, are sustained in pancreas-kidney (SPK) transplant recipients with extended graft survival. Transthoracic echocardiographic exams using 2-dimensional, M-mode, and Doppler interrogation in parasternal and apical views were performed prior to and yearly following transplantation in SPK and diabetic kidney alone (KA) recipients. Thirty patients with functioning grafts (26 SPK vs 4 KA) with data prior to transplantation and at 1 year and 4 years post-transplant were included in the analysis. SPK and KA groups were similar in age (36 vs 36 yrs), race (100% vs 96% Caucasian), and years of diabetes (21 vs 26), while the SPK group had more males (75% vs 25%).

RESULTS: SPK recipients exhibited continued improvement in cardiac systolic (shortening fraction, SF) and diastolic (EA peak velocity ratio, EA ratio) function at 48 months post-transplant, as well as improved left ventricular cardiac geometry (posterior wall thickness, PWT) while KA recipients had deterioration in cardiac geometry (PWT and interventricular septal thickness) and diastolic function (EA ratio) with improved SF.

	SF		EA Ratio		PWT	
Month	KA	SPK	KA	SPK	KA	SPK
0	30 5 ± 3 3	28 4 ± 1 3	1 09 ± 0 16	1 15 ± 0 06	11 0 ± 0 7	11 7 ± 0 3
12	31 7 ±3 3	33 7 ± 1 3*	0 82 ± 0 16^	1 19 ± 0 09^	12 8 ± 0 7	11 7 ± 0 3
48	33 3 ± 5 0	36 7 ± 1 4*	0 91 ± 0 20^	1 36 ± 0 08*^	13 3 ± 0 7 *^	10 9 ± 0 3*^

NOTE values reported as means ±SE

*p<0.05 from baseline within same group;^p<0.05 between groups at like times.

CONCLUSIONS: These findings are consistent with our initial observations of early (1 yr post-transplant) improvements in cardiac function and geometry following SPK transplantation. Restoration of euglycemia by SPK transplantation results in progressive sustained improvement in systolic and diastolic function and cardiac geometry supporting the long-term benefit of SPK transplantation on cardiac function.

Abstract# 186

IMPROVEMENT IN HYPERTENSION AFTER SIMULTANEOUS PANCREAS-KIDNEY (SPK) TRANSPLANTATION. Micheal D. Elliott, Mihai Gheorghiade, Michele A. Parker, Lorenzo G. Gallon, Dixon B. Kaufman.

Hypertension invariably complicates the course of type 1 diabetics with diabetic nephropathy, and is causally related to the markedly elevated risk of cardiovascular events. Recent data suggests that hypertension management may improve following successful SPK transplantation. Accordingly, we investigated the utilization of antihypertensive medications before and following kidney-alone and SPK transplantation. Table 1 displays the percentage of patients requiring 0, 1, 2, 3, or ≥4 antihypertensive medications before and following transplantation for the following groups: kidney-alone in type I diabetics (n=28), SPK-bladder drainage (n=45), and SPK-enteric drainage (n=45). All SPK patients received MMF + tacrolimus + prednisone. Kidney-alone patients were treated with aza/MMF + CsA + prednisone. The minimum follow-up for all groups was ≥ 1 year.

Number of	KIDNEY	KIDNEY	SPK-Bladder	SPK-Bladder	SPK-Enteric	SPK-Enteric
HTN Meds	Pre-Tx	Post-Tx	Pre-Tx	Post-Tx	Pre-Tx	Post-Tx
0	4%	4%	16%	69%	2%	67%
1	19%	30%	29%	29%	20%	18%
2	37%	40%	33%	2%	32%	9%
3	33%	15%	16%	0%	26%	4%
≥4	7%	11%	6%	0%	20%	2%

The mean number of antihypertensive medications per patient prior to transplantation in the kidney-alone, SPK-bladder and SPK-enteric groups were: 2.3 ± 1.1, 2.5 ± 1.1, and 2.5 ± 1.2, respectively. At 1 year follow-up, the number of antihypertensive medications per patient for these groups were now 2.0 ± 1.3, 0.8 ± 1.0 and 0.6 ± 1.0, respectively. The reduction in antihypertensive medication utilization for SPK recipients occurred in association with a concomitant improvement in blood pressure. No significant difference in the percentage of kidney-alone patients determined to be normotensive (BP£130/85) was observed following transplantation(14% pre-Tx vs. 22% post-Tx,p=ns). In contrast, the percentage of patients determined to be normotensive in both the bladder and enteric SPK groups increased significantly following transplantation(19% pre-Tx vs. 57% post-Tx). Conclusions: A marked improvement in hypertension was observed following successful SPK. This improvement was dependent on the pancreas transplant but independent of the surgical method of exocrine drainage.

Abstract# 187

HUMAN ISLET TRANSPLANTATION NETWORK FOR THE TREATMENT OF TYPE I DIABETES: FIRST (1999-2000) DATA FROM THE SWISS-FRENCH GRAGIL CONSORTIUM. José Oberholzer,[1] Pierre-Yves Benhamou,[2] Christian Toso,[1] Laurance Kessler,[3] Alfred Penformis,[4] François Bayle,[2] Charles Thivolet,[5] Xavier Martin,[5] Lionel Badet,[5] Cyrille Colin,[5] Philippe Morel.[1] [1]*Surgery, University Hospital, Geneva, Switzerland;* [2]*Endocrinology and Nephrology, University Hospital, Grenoble, France;* [3]*Endocrinology, University Hospital, Strasbourg, France;* [4]*Endocrinology, University Hospital, Besançon, France;* [5]*Endocrinology and Urology, Hospices Civils, Lyon, France.*

Aims/hypothesis. Improvement of islet transplantation requires clinical series large enough to implement controlled new strategies. The goal of this study was to demonstrate the feasibility of a multicentric network for islet transplantation in type 1 diabetic patients.

Methods. Gathering five centers (Besançon, Geneva, Grenoble, Lyon, Strasbourg), the GRAGIL network allows pancreas procurement, recipient recruitment, transplantation procedure and follow up while islet isolation is performed in one single laboratory (Geneva). Pancreata were procured in each of the 5 centers and transported with an ischemia time inferior to 8 hours to Geneva. Islets were isolated by a standard automated method and cultured until transplantation. If the islet number was too low for a graft (< 6000 Islet-equivalent /kg), culture period was extended up to 12 days until an other isolation became available. Islets were transplanted by percutaneous transhepatic intraportal injection. Immunosuppression was performed with cyclosporine, mycophenolate mofetil, steroids and an anti-interleukin 2 receptor antibody (Basiliximab, Simulect, Novartis).

Results. From March 1999 to June 2000, 56 pancreas procurements were performed, average yield was 234,500 islet-equivalent, with 32 preparations > 200,000 islet-equivalent. Ten C-peptide negative type 1 diabetic patients (5 men and 5 women, median age 44 years, median diabetes duration 29 years) with an established kidney graft (> 6 months) received 9,030±1,090 islet-equivalent/kg with a median purity of 63 %. The number of pancreata required for each graft was 1 (n=5) or 2 (n=5). With a median follow-up of 12 months, we observed 0% primary nonfunction, 50% graft survival and 20% insulin-independence (achieved with one pancreas per recipient).

Conclusions. This study demonstrates the interest and the feasibility of a multicentric collaboration in human islet transplantation.Insulin-independence in uremic type I diabetic patients can be achieved with an islet graft isolated from a single pancreas.

POSTER SESSION I:
KIDNEY - ACUTE/CHRONIC REJECTION I

Abstract# 188 — Poster Board #-Session: P1-I

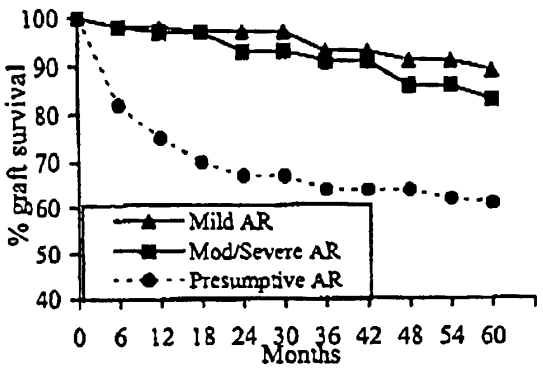
OUTCOME AFTER TREATMENT OF BIOPSY PROVEN VS. PRESUMPTIVE ACUTE REJECTION (AR) IN KIDNEY TRANSPLANT RECIPIENTS. Abhi Humar,[1] Raja Kandaswamy,[1] Roger Denis,[1] Steven Paraskevas,[1] Kristen Gillingham,[1] Arthur Matas.[1] [1]*Surgery, University of Minnesota, Minneapolis, MN.*

Background: Kidney biopsy remains the gold standard for the diagnosis of acute rejection. Sometimes, however, AR is diagnosed and treated without the benefit of a biopsy (presumptive AR). We sought to determine what effect this had on outcome.

Results: Of over 2000 kidney transplants performed between 1984-1999, we identified 336 recipients treated for a single episode of AR. Recipients with multiple AR episodes were excluded. Of these 113 (33.6%) were treated for biopsy proven mild AR, 174 (51.7%) for moderate or severe AR, and 49(14.6%) for presumptive AR (no biopsy). Demographics for the 3 groups are shown. Graft survival after treatment of AR, short & long-term, was significantly worse in the presumptive AR group (graph). The cause of the lower graft survival was multifactorial with a higher incidence of chronic rejection, infections, and recipient deaths (usually due to sepsis).

Conclusion: Biopsy remains an essential part of the evaluation for graft dysfunction, and should always be obtained if possible before the initiation of high dose anti rejection medications. Treatment for presumptive AR may lead to overdiagnosis, or underestimate the severity of rejection; in either case this may lead to improper treatment and poorer outcomes.

	Mild AR	Mod/Severe AR	Presumptive AR
Total#	113	174	49
Recipient Age	44 1	40 9	44 9
Recipient sex (%male)	56 6%	70 7%	46 9%
# of mismatches	2 76	3 15	2 79
High PRA (>75%)	8 5%	1 8%	2 4%
% AR treated with antibody	25 6%	40 8%	32 6%



Abstract# 189 **Poster Board #-Session: P2-I**

POOR RENAL ALLOGRAFT FUNCTION AFTER TRANSPLANT (TX) PREDICTS POOR OUTCOME AT 1 YEAR. Arthur J. Matas,[1] William D. Irish ,[2] the DGF Study Group. [1]*Fairview University Medical Center, Minneapolis, MN;* [2]*SangStat Medical Corporation, Fremont, CA.*

Introduction: Delayed graft function (DGF, dialysis in first 7d post-tx) increases the risk of a poor outcome after renal tx. A single center study (Clin.Trans.1997.11:623) showed that slow graft function (SGF; sCr>3.0mg/dl on POD5 with no need for dialysis) is associated with worse outcome when compared with recipients that have immediate graft function (IGF). Is worse GS the only penalty for poor graft function in the early post-tx period? Data from a large prospective study of DGF was reviewed to determine if early graft function had an impact on renal function (RF) in surviving grafts.**Methods:**492 adult recipients (US & Europe) of grafts (11/97-3/99) from high-risk CAD donors (CIT≥24h or donor age≥50y) that survived 7d post-tx were analyzed. Follow-up was 1y. Recipients were divided into 3 groups:IGF, SGF and DGF. Graft loss (GL) was patient death or return to dialysis. Acute rejection (AR) was biopsy proven.RF shown is sCr of functioning grafts at 1y.**Results:**71% and 55% of allografts were from donor ≥50y or CIT≥24h, respectively, 27% had both. CSA and MMF were used in >80% of recipients. IGF, SGF, DGF occurred in 32%, 27% and 41% of recipients, respectively. SGF and DGF more then doubled the risk of AR (p<0.05) and tripled the risk of GL (p=0.001) compared to recipients with IGF. sCr was 46% and 53% higher in patients who suffered SGF and DGF, respectively. sCr >1.8mg/dl was 2 and 3 times more common in SGF and DGF patients, respectively. The number of SGF and DGF patients with sCr>2.4mg/dl was 5 times that of IGF patients. The interaction between CIT>24h and donor age >50y and DGF/SGF and IGF was not significant(p=0.2881).

	GS	1y Rej-free GS	1y sCr (mg/dl)	sCr>1.8 mg/dl	sCr>2.4 mg/dl
IGF	98%	87%	1.5	17%	5%
SGF	92%	71%	2.2	38%	24%
DGF	86%	59%	2.3	59%	27%
p-value	0.0004	0.0001	<0.0001	<0.0001	<0.0001

Conclusions:1)SGF, like DGF, results in more AR and worse GS at 1y. 2)RF in grafts surviving 1y is worse in recipients with both DGF and SGF. 3) SGF is a clearly defined risk factor for a worse outcome. These data suggest two penalties for DGF or SGF. One is immunologic (worse GS and more AR). The second is worse RF in surviving grafts. Strategies to decrease the incidence of DGF and SGF may improve both short-term and long-term renal tx results.

Abstract# 190 **Poster Board #-Session: P3-I**

AN ANALYSIS OF TISSUE REPAIR GENES IN NEPHROTOXICITY AND ACUTE REJECTION IN RENAL TRANSPLANT PATIENTS. Ashwani K. Khanna, Matthew S. Plummer, Barbara Bresnahan, Sundaram Hariharan. [1]*Medicine, Medical College of Wisconsin, Milwaukee, WI;* [2]*Medicine, Medical College of Wisconsin, Milwaukee, WI;* [3]*Medicine, Medical College of Wisconsin, Milwaukee, WI;* [4]*Medicine, Medical College of Wisconsin, Milwaukee, WI.*

Despite a decrease in the incidence of acute rejection, nephrotoxicity remains a significant problem in transplant patients treated with either cyclosporine (CsA) or tacrolimus (Tac). Both experimental and clinical studies have demonstrated that the side effects of CsA and Tac are indistinguishable from one another. This study was designed to study the intra-renal expression of tissue repair genes: TGF-b, collagen, fibronectin and osteopontin, MMP-2, MMP-9 and TIMP-2 in biopsies obtained from renal transplant patients treated with either CsA or Tac. Renal biopsies from 47 patients who received either TAC or CsA as a maintenance immunosuppression were obtained for this study after an informed consent (approved by IRB). The biopsies were performed for elevated serum creatinine. The levels of creatinine were 2.4 ± 0.3 and 2.1 ± 0.2 mg/dl for TAC and CsA treated patients, respectively. We compared the expression of tissue repair genes in patients diagnosed with nephrotoxicity and rejection treated with TAC (n=17) or CsA (n=15). The mean ± SEM of the ratio of these genes for all histologic diagnosis is shown in the Table. The expression of TGF-beta, Collagen, fibronectin and osteopontin was significantly more in patients with tacrolimus nephrotoxicity compared to either cyclosporine nephrotoxicity or acute rejection. The expression of Matrix Metalloproteinase-9 (MMP-9) was significantly less in patients with tacrolimus nephrotoxicity than with CsA nephrotoxicity. The mRNA expression of tissue inhibitor of Matrix Metalloproteinase-2 (TIMP-2) was not different among these patients.The expression of MMP-9 and TIMP-2 were not detectable in patients with acute rejection. The ratio of MMP-9/TIMP-2 was more in patients with CsA nephrotoxicity than with tacrolimus nephrotoxicity. These results show that decreased MMP-9 expression in tacrolimus treated patients may cause abnormal accumulation of ECM and result in nephrotoxicity. The results from this study demonstrate that the expression of tissue repair genes significantly differ in patients with acute rejection and nephrotoxicity and can potentially be used as diagnostic markers for renal dysfunction.

Gene	CsA Toxicity	TAC Toxicity	Acute Rejection
TGF-Beta	0.04± 0.03*	0.2± 0.1**	0.007± 0.003
MMP-9	0.9± 0.09	0.6± 0.09	Not detected
TIMP-2	0.9± 0.2	0.9± 0.3	Not detected
Fibronectin	0.1± 0.04	0.4± 0.1***	0.1± 0.07***
Osteopontin	1.5 ± 0.2	2.1 ± 0.15***	1.6 ± 0.2***
Collagen	0.1± 0.08	0.3± 0.2**	0.16± 0.05**

* = Gene/β-actin ratio;

Two tailed t test: Tacrolimus vs CsA and acute rejection ** = p<0.05; *** = p<0.02

Abstract# 191 **Poster Board #-Session: P4-I**

47 CONSECUTIVE KIDNEY TRANSPLANTS WITHOUT REJECTION-THE IMPORTANCE OF DIURNAL CSA DOSING. Barry J. Browne,[1] Cynthia Op't Holt,[1] Osemwegie E. Emovon.[1] [1]*Surgery and Medicine, University of South Alabama, Mobile, AL.*

Acute rejection(AR) following transplantation may be due to episodic subtherapeutic cyclosporine (CsA) levels related to diurnal variation of hepatic drug2 metabolism. Hypothesis: We postulated that asymmetrical dosing of CsA based on individualized pharmacokinetic profiles would optimize drug exposure and decrease the risk of AR. Methods: Beginning in December 1998, all patients undergoing kidney transplantation at our institution were prospectively treated with a diurnally split dose of CsA microemulsion given q12hrs(3.5mg/kg qAM, 3.0 mg/kg qPM). AM doses were adjusted to reach a day-time area under the concentration curve (AUC) of 7800 nghr/ml(utilizing 2hr and 6hr levels) and PM doses were adjusted to an AM trough of 300 ng/ml. Patients received high-dose steroids tapered to 20 mg prednisone by day 6 and to 10mg by 6 months. CsA was started within 48 hrs and Mycophenolate Mofetil (1000mg q12hr) was added on day 3 in most patients and continued for 3 months. Only 1 patient received antibody induction. Results: Patients required an average of 9 3-point profiles during the first 3 months to maintain target CsA levels. Pharmacokinetic and functional data are summarized in the table as mean ± SEM.

	Week 1	Month 1	Month 2	Month 3	Month 6
AM Dose (mg/kg)	3.7 ± 1.8	3.2 ± 1.4	3.1 ± 1.3	2.7 ± 1.2	2.4 ± 1.2
PM dose (mg/kg)	2.8 ± 2.1	2.7 ± 1.8	2.4 ± 1.4	2.1 ± 1.3	1.9 ± 1.2
Trough (ng/ml)	299 ± 10	301 ± 8	305 ± 12	288 ± 7	275 ± 10
AUC (nghr/ml)	7913 ± 252	8252 ± 287	7790 ± 373	7238 ± 331	6602 ± 311
Creatinine (mg/ml)	4.5 ± 0.8	1.7 ± 0.1	1.5 ± 0.1	1.5 ± 0.1	1.5 ± 0.1

47 kidneys(77% cadaveric) were transplanted into 44 adult patients(3 double grafts). Half of recipients were African-American, 64% were men, and 20% had Hepatitis C. 60% had ³3 HLA mismatches and 16% had PRA>20%. At 10 months mean follow-up, no patient has had a documented episode of AR. 18 patients required biopsy for acute allograft dysfunction, however no histologic evidence of AR or CsA-toxicity was identified and the creatinine normalized in each case without altering immunosuppression. Patients continued to require increased CsA doses in the AM compared to the PM (p<0.05) throughout the study to maintain target levels. Conclusion: Diurnal dosing of CsA based on individual pharmacokinetic profiles optimizes CsA exposure and reduces the risk of AR after kidney transplantation.

Abstract# 192 **Poster Board #-Session: P5-I**

SIROLIMUS THERAPY FOR HIGH-RISK RENAL TRANSPLANT PATIENTS. Barry D. Kahan,[1] the Rapamune Renal Function Study Group.
[1]Division of Immunology & Organ Transplantation, University of Texas Health Science Center Medical School, Houston, TX.

Background: Renal transplant recipients with delayed graft function (DGF) are known to be at increased risk for acute rejection and graft loss. We report the outcomes of patients with DGF treated with sirolimus who were enrolled in a cyclosporine (CsA) elimination study.

Methods: Two-hundred forty six (246) first cadaveric renal allograft recipients were enrolled in a Phase II, open-label, randomized study at 17 centers in the United States and Europe to examine the effect of CsA elimination in patients initially treated with full-dose CsA (microemulsion) plus fixed-dose sirolimus (2 mg/day) and corticosteroids. Patients [n=49] with DGF which resolved later than day 7 post-transplant were not randomized into the CsA elimination arms of the trial and were assigned to a rescue group. Patients in the rescue group received up to 5 mg/day sirolimus together with standard CsA and corticosteroids and were evaluated at 12 months for rates of acute rejection, patient and graft survival, and safety.

Results: The rescue group contained a higher proportion of black recipients and had a higher incidence of diabetes mellitus as primary etiology of renal failure than those without DGF. Compared with randomized patients, patients with DGF tended to be slightly older, received organs with slightly longer total ischemia time, and had a lower incidence of receiving organs from CMV-positive donors. Intent-to-treat analysis at 12 months showed that the rate of biopsy-confirmed acute rejection in the rescue group was 18.4% (9/49). Graft survival and patient survival were 77.6% and 89.8%, respectively. Serum creatinine was measured in 23/49patients and was 219.2 ± 22.6 mmol/L (mean ± SEM); GFR in these patients was 49.1 ± 3.3 mL/min. Fasting total cholesterol, HDL cholesterol, and triglycerides were 6.2 ± 1.3 mmol/L (n=13), 1.3 ± 0.4 mmol/L (n=12), and 3.4 ± 2.3 mmol/L (n=13), respectively. In this group, 4 patients died and there were no malignancies. Treatment-emergent adverse events were comparable to those in randomized patients.

Conclusion: Sirolimus-treated renal transplant recipients with DGF experienced acceptable rates of acute rejection and patient and graft survival. As expected in patients with DGF, renal function was moderately impaired Adverse events were similar to those experienced by low and moderate risk patients receiving sirolimus therapy.

Abstract# 193 **Poster Board #-Session: P6-I**

SMADS DEFINE A CHRONIC ALLOGRAFT NEPHROPATHY (CAN) GENOTYPE AND PHENOTYPE: IMPLICATIONS FOR INTRAGRAFT MONITORING IN CAN. Bryan N. Becker,[1] Lynn M. Jacobson,[2] Gretchen J. Malin,[1] Jacquelyn K. Aschenbrenner,[2] Yolanda T. Becker,[2] Debra A. Hullett.[2] *[1]Medicine, University of Wisconsin, Madison, WI; [2]Surgery, University of Wisconsin, Madison, WI.*

Transforming growth factor-beta (TGF-β) is an important mediator of chronic allograft nephropathy (CAN). Yet, there is little data examining how TGF-β exerts its effects in CAN. Normally, TGF-β initiates signaling and changes in gene expression through Smads. These proteins are intracellular transcriptional comodulators. R-Smads, e.g. Smad2, alter TGF-β's fibrotic effects. I-Smads, e.g. Smad7, act as transcriptional co-repressors of TGF-β-affected genes, including TGF-β itself. Relative changes in Smad levels influence biological activity. We examined biopsy (bx) samples from normal human kidney obtained from donors (NL:n=4), CAN tissue (n=5) and normally functioning renal transplants (RTx.n=2) for Smad2, Smad7, and TGF-β mRNA expression, using semi-quantitative reverse transcription polymerase chain reaction (RT-PCR) with Vistra green. The CAN patients had an average creatinine of 3.8 mg/dl, off calcineurin inhibitors at time of bx. RT-PCR products were normalized to S26 mRNA expression in the bx samples. In bx samples with adequate tissue (n=3 for NL + RTx; n=3 for CAN), we examined Smad2 and Smad7 protein expression by immunoblot (normalizing the results to tubulin expression in the bx) Smad2 and TGF-β mRNA expression were actually similar in NL, RTx, and CAN tissue However, Smad7 mRNA expression was reduced in CAN tissue ($p < 0.04$ vs others) Surprisingly, Smad2 protein levels were significantly greater in NL and RTx tissue (6.9 fold increase, p=0.05 vs. CAN). In contrast, Smad7 protein levels were greater in CAN tissue (4.4 fold increase; p=0.05 vs. NL). These data suggest that Smad2 and Smad7 undergo a phenotypic switch that defines the development of CAN, despite relatively constant TGF-β mRNA expression. It appears that under conditions of good graft function, Smad7 could be sequestered in a nuclear pool bound to transcription factors or target genes. The converse situation may exist in CAN with Smad2. It is also possible that the rate of transcription for Smad2 and Smad7 is significantly altered in NL vs. CAN tissue. Finally, the disparity between mRNA and protein levels in this small study suggests that proteomics may be an important tool for intragraft monitoring as an adjunct to gene expression analysis, especially in the evolution of CAN.

Abstract# 194 **Poster Board #-Session: P7-I**

A MULTICENTRE RANDOMISED CONTROLLED STUDY INVESTIGATING THE EFFECT OF MYCOPHENOLATE MOFETIL (MMF) ON RENAL FUNCTION AFTER CYCLOSPORINE A (CsA) WITHDRAWAL IN RENAL TRANSPLANT PATIENTS WITH A "CREEPING CREATININE": AN INTERIM ANALYSIS OF AN ONGOING STUDY. Christopher R.K. Dudley, the Mycophenolate Mofetil Creeping Creatinine Study Group.
[1]Richard Bright Renal Unit, Southmead Hospital, Bristol, United Kingdom.

Chronic allograft dysfunction (CAD) is the commonest cause of late graft loss. Incompletely defined immunological and non-immunological mechanisms contribute to this condition. The aim of this multicentre randomised controlled study is to investigate the effect of MMF after CsA withdrawal in renal transplant recipients with CAD. Patients receiving a cyclopsorine-based immunosuppressive regimen with progressively deteriorating graft function, defined by a significantly negative slope of reciprocal creatinine plotted against time, have been identified. A baseline allograft biopsy has been performed to exclude recurrent or de novo renal disease. To date, 93 patients have been randomised to treatment with MMF (1g bd) and CsA withdrawal (Group A, n=50) or to continue treatment with CsA according to the centre's normal practice (Group B, n=43). The slope of the reciprocal creatinine plot against time has been calculated for 55 patients over a 6 month follow up after complete CsA withdrawal. Patients with a slope greater than or equal to zero are defined as responders.

Responder	Group A (MMF)	Group B (CsA)
Yes	19 (65%)	6 (23%)
No	10 (35%)	20 (77%)

At the time of this interim analysis, one patient in each group has experienced an acute rejection episode. There have been two patient deaths in Group A (sepsis, liver failure) and two graft losses in Group B.

These data suggest that the substitution of CsA with MMF in patients with chronic allograft dysfunction is safe and results in improved graft function.

Abstract# 195 **Poster Board #-Session: P8-I**

DURATION OF DECLARED BRAIN DEATH IN CADAVERIC DONORS. Darin J. Treleaven,[1] Jacqueline Gough,[1] Christian G. Rabbat,[1] Scott Brimble,[1] David Ludwin,[1] Dianne Arlen,[1] David Russell.[1] *[1]Division of Nephrology, McMaster University, Hamilton, ON, Canada.*

Renal injury due to events during brain death has been proposed as a mechanism to explain the difference in delayed graft function (DGF) rates between live and cadaveric allografts (Halloran, JASN, 1999). We hypothesized that increasing length of time from declaration of brain death to organ retrieval and total time in the ICU may be surrogates for increasing tissue damage resulting in DGF, particularly in older donors. Data on the former has not previously been reported.

151 consecutive donors provided kidneys transplanted into 158 recipients at our center between 1990 and 1999. ICU and demographic data (donor height and weight, donor cause of death, total ICU time, and time between retrieval and declaration of brain death), donor past medical history (smoking, vascular disease, hypertension) and cold ischemic time (CIT) were determined. The effect of specific ICU and demographic factors on DGF were compared in older (age greater than 50 years) and younger donors. DGF was defined as the need for dialysis within the first week post-transplantation.

The mean donor age was 39 years (SD 14.6), the mean CIT was 19.5 hours (5.5), the mean total time in the ICU was 51.0 hours (33.8), the mean length of brain death before organ retrieval was 12.4 hours (8.8). The rate of DGF was 27.6%. Multivariate regression analysis of ICU and demographic factors showed that only age and cold ischemic time were significantly associated with DGF

Time since brain death and total time in the ICU were not significantly associated with the development of DGF, even when adjusted for dono, age.

FACTOR	ODDS RATIO	95 % C I	p value
Donor Age > 50 yrs	2 5	1 2-5 3	017
C I T > 24 hrs	3 2	1 3-8 0	014
Brain Death > 24 hrs	1 3	2-7 5	N/S
Total ICU Time > 48 hrs	7	3-1 6	N/S

Conclusion: We have demonstrated in our sample of 151 consecutive donors that well-established factors such as donor age and CIT were strongly associated with DGF but we failed to show any relationship between duration of brain death and delay in allograft function. To confirm our hypothesis, other markers of injury will have to be sought.

Abstract# 196 **Poster Board #-Session: P9-I**

A RANDOMIZED AND PROSPECTIVE STUDY COMPARING TREATMENT WITH HIGH-DOSE INTRAVENOUS IMMUNOGLOBULIN WITH MONOCLONAL ANTIBODIES FOR RESCUE OF KIDNEY GRAFTS WITH STEROID-RESISTANT REJECTION. Domingo H. Casadei,[1] Maria Del Carmen Rial,[1] Gerhard Opelz,[2] Julio C. Goldberg,[1] Jorge A. Argento,[1] Gabriela F. Greco,[1] Olga E. Guardia,[1] Emilio Haas,[1] Eduardo Raimondi.[3] *[1]Instituto de Nefrologia, Buenos Aires, Argentina, [2]Transplantation Immunology University of Heidelberg, Heidelberg, Germany, [3]Fundacion Favaloro, Buenos Aires, Argentina.*

To compare the effectiveness of IVIg vs OKT3 as a treatment of steroid resistant rejections. From 1/95 to 6/97 30 patients were divided into two groups. Resistant rejections were diagnosed by core biopsy. Group A received 500 mg/kg/d IVIg (Sandoglobulin™) during 7 consecutive days while B received 5 mg/d of OKT3 during two weeks. Cyclosporine was stopped in both groups. Data included: Demography, missmatch HLA, pre-post creatinine levels and rejections rate in one month post treatment.

Fisher's exact test and Kaplan-Meyer were used. There was no significant differences in any of the data. Success was achieved for A 11/15 (73.3%) and B 13/15 (86.6%) p=0.79 Pre-post creatinine levels were for A 2.99 ± 1.30 mg/dl and 2.1 ± 0.70 mg/dl vs B 3.1 ± 1.1 mg/dl and 2.5 ± 0.8 mg/dl. Besides, there was no differences in the creatinine of the first month (A 2.35 ± 0.78 mg/dl B 2.51 ± 1.10 mg/dl p=0.66) or in the third ones (A 1.83 ± 0.58 mg/dl B 2.30 ± 0.89 mg/dl p=0.24). Rejections rate post treatment was for A 5/11(46%) and B 9/12(75%) p=0.4. Two-year patient survival was 87 y 92% for A and B groups respectively. Graft survival was 80% in both groups. If this results were confirmed in a larger number of patients, IVIg could become the better choice of rejection treatment for steroid resistant rejection because of a complete absence of the unwanted side effects commonly associated with OKT3.

Abstract# 197 **Poster Board #-Session: P10-I**

RECIPIENTS OF HIGH-RISK CADAVERIC DONORS THAT SUFFER DELAYED GRAFT FUNCTION (DGF) HAVE WORSE GRAFT SURVIVAL (GS), IMPAIRED RENAL FUNCTION AND MORE ACUTE REJECTION (AR). Gabriel M. Danovitch,[1] William D. Irish,[2] The DGF Study Group. *[1]Nephrology, UCLA, Los Angeles, CA; [2]SangStat Medical Corporation, Fremont, CA.*

The impact of DGF after renal transplant has not previously been studied in a prospective fashion. In a multicenter, prospective study patients received grafts from high risk cadaver donors (CIT≥24h or donor age≥50y). 12 month (m) data were analyzed to determine impact of DGF on GS and renal function. **Methods**: A validated database of 492 adults transplanted between 11/97-3/99 in the US and EU was analyzed. Patients were part of a randomized study using ANTILFA antiCD11a antibody to prevent reperfusion injury; the therapy was equivalent to placebo. DGF was defined as the need for dialysis in the first 7d after transplant. Graft loss was death or return to dialysis. Rejection was confirmed by biopsy in >90% of the cases. Renal function was measured by serum creatinine (sCr) levels. Note: 71% and 55% of allografts were from donor age≥50 or CIT≥24h, respectively, and 27% had both. CSA and MMF were used in >80% of recipients. **Results**: DGF and AR occurred in 41% and 26% of the patients, respectively. The risk of AR was two times greater with DGF [Odds Ratio(OR)=2] and the risk of graft loss was almost 3 times greater with DGF [Hazard Rate Ratio=2.8]. Rejection free GS was greater without DGF compared to with DGF (p=0.0001). Mean sCr was significantly greater in the DGF group (p=0.004) and more then twice the number of patients with surviving grafts showing DGF had sCr>1.8mg/dl at 12m vs those without DGF (p=0.001). The interaction between CIT≥24h and donor age≥50y was also studied. In this analysis, both have significant but independent effects (CIT≥24h. OR=2.1,p=0.0026; donor age≥50y: OR=2.0,p=0.0025).

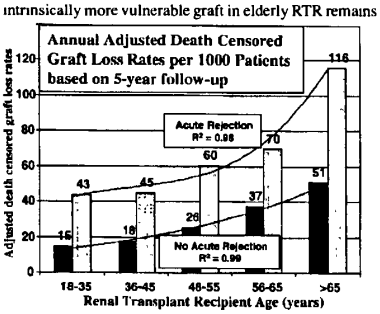
Kidney Status	N	12m sCr (mg/dl)	sCr>1.8 mg/dl	12m AR-free GS
DGF	201	2.2	59%	59%
No DGF	289	1.8	26%	80%

Conclusions: 1) These data confirm the negative impact of DGF on 12m graft survival in a prospective multicenter trial with standard immunosuppression. 2) DGF increases the risk of AR and the combination of AR and DGF appears to be additive 3) Renal function is worse in recipients who experience DGF at all time points 4) 12m sCr may be a surrogate of long-term graft survival. 5) Advanced donor age and prolonged CIT both contribute to the risk of DGF. These results predict that prevention of DGF will improve both graft survival and renal function.

Abstract# 198 **Poster Board #-Session: P11-I**

INTERACTION BETWEEN ACUTE REJECTION AND RECIPIENT AGE ON LONG TERM RENAL ALLOGRAFT SURVIVAL. Herwig-Ulf Meier-Kriesche,[1] Otto Leiti,[2] Gary S. Friedman,[2] Bruce Kaplan.[1] *[1]Department of Internal Medicine, University of Michigan, Ann Arbor, MI; [2]Department of Transplantation, Saint Barnabas Medical Center, Livingston, NJ.*

Introduction: Elderly renal transplant recipients (RTR) have been shown to have less frequent acute rejection (AR) episodes as compared to younger RTR, but on the other hand poorer death censored graft survival. We hypothesized that AR might have a different prognostic impact in elderly as compared to younger renal transplant recipients. Methods: We analyzed USRDS data from 48,821 primary, solitary, RTR transplanted between 1988 and 1997. We estimated death censored graft loss (GL) by Kaplan-Meier and Cox-proportional hazard models for the interaction between categorized age groups and acute vs no acute rejection. Cox proportional hazard models were adjusted for 13 pertinent covariates, and annual adjusted GL rates were calculated based on actual and estimated 5-year follow-up. Results: Annual adjusted GL rates increased with increasing recipient age exponentially, but with a steeper slope in patients with rejection as compared to patients who had not suffered an AR episode during the first 6 month post-transplant. Kaplan-Meier estimated 5-year death censored graft survival was 91.5% vs 82.1% in the youngest recipient group and 75.0% vs 59.9% in the oldest recipient group when comparing no AR versus AR. Conclusions: AR has a strong negative impact on death censored graft survival in older renal transplant recipients. Despite of the lower incidence of AR in the elderly renal transplant recipients, AR is a primary prognostic indicator for long term graft survival in this patient population. Whether this effect is due to more violent AR episodes or because of an intrinsically more vulnerable graft in elderly RTR remains to be investigated.



Poster Session I:
Kidney - GVH, Complications, Infections I

Abstract# 199 **Poster Board #-Session: P12-I**

UROLOGIC CANCERS IN KIDNEY TRANSPLANT RECIPIENTS. Christopher Gran,[1] John Hulbert,[1] Ken Roberts,[1] Sid Jain,[1] Arthur Matas,[2] Abhi Humar.[2] *[1]Urology, University of Minnesota, Minneapolis, MN; [2]Surgery, University of Minnesota, Minneapolis, MN.*

Background: Urologic cancers in kidney transplant (tx) recipients are uncommon. We examined a large group of recipients at a single center to determine the incidence, risk factors, management, and outcome with neoplasms in this area.

Results: Between 1963 to 1999, 5491 kidney transplants were performed. We identified 31 recipients (22 males, 9 females) with 33 urologic cancers (incidence= 0.6%). There were 24 kidney tx, 6 kidney/pancreas tx, and 1 liver/kidney tx. 29 of the 31 recipients were primary tx— 13 living donor and 18 cadaver. A significant smoking history was present in 16 of the 31 recipients. Three had been treated for a pretx bladder transitional cell carcinoma (TCC), with a mean cancer-free period of 70 months (range= 27- 186) prior to tx. Mean age at tx was 51.6 yrs (range= 4-75), with a mean time to diagnosis of 51 months posttx (range= 1- 243). Tumors were as follows: 9 renal cell carcinomas (RCC) (incidence= 0.16%), 8 adenocarcinomas of the prostate (incidence= 0.15%), 8 TCC of the bladder (incidence= 0.15%), 5 lymphomas involving the kidney (incidence= 0.09%), 2 seminomas (incidence= 0.06%), and 1 squamous cell ca. (SCC) of the penis (incidence= 0.03%) Treatment was as follows: 14 with surgery alone; 3 with surgery/ chemotherapy, 3 with surgery/chemotherapy/radiation; 1 with chemotherapy alone; 4 with radiation alone, 3 with no treatment. Outcomes by tumor type were as follows: 6 of 9 with RCC deceased, 3 from malignancy; 4 of 8 with TCC deceased, 2 from malignancy; 3 of 8 with prostate cancer deceased, 2 from malignancy from some other source; 5 of 5 with lymphoma deceased, 5 from malignancy; 1 of 2 with seminoma dead, none from malignancy; 1 of 1 with SCC of the penis dead, due to a coexistent rectal malignancy. With mean follow up of 6.8 years, 18 (58%) of the 31 recipients are deceased, 11 of malignancy. Of these 8 (25%) were from their urologic tumor; the other 3 from some other malignancy. Mean survival after diagnosis for those deceased was 33 months (range= 0- 124). Thirteen recipients (42%) are still alive, with no evidence of tumor.

Conclusions: Urologic cancers in renal tx recipients are rare. The tumors most frequently diagnosed were RCC, TCC of the bladder, and adenocarcinoma of the prostate. Primary urologic tumors generally had a good outcome with surgical therapy-mortality in the majority of cases was not related o these tumors. Lymphomas, however, had a fatal outcome in all of these patients.

Abstract# 200 **Poster Board #-Session: P13-I**

RELATIONSHIPS BETWEEN CYTOMEGALOVIRUS SHEDDING AND EBV VIRAL LOAD AT THE EARLY PHASE OF RENAL TRANSPLANTATION. Claire Presne, Andre S. Pruna, Philippe Bidet, Veronique Auguste, Gilles Duverlie, Hakim Mazouz, Pierre F. Westeel, Jacques Petit, Albert Fournier, Antoine Garbarg-Chenon. [1]*Renal Transplantation, CHU AMIENS-Sud, Amiens, France;* [2]*Virology Laboratory, Hôpital Trousseau, Paris, France.*

Cytomegalovirus affects up to 20% renal transplant recipients during the 2nd month. Reactivation of other viruses at the same time might be a factor of clinical severity. We assumed that co-infection with an early emergence of EBV might trigger more severe CMV infection. We studied 41 consecutive renal transplant patients over 1 year. CMV antigenemia by our routine test(pp65 in white blood cells)usually required Ganciclovir when positive in >30 neutrophils/200,000. EBV viral load was counted at day 0,15,30 by PCR on blood lymphocytes(Ly),amplifying the gene of p140 protein. Results were given as genome equivalents(GE)on 100,000 Ly Immunosuppression was quantified in mg/kg/month for each drug. Ly subsets were checked by FACS at day 0,15,30 for CD2,19,3,4,8 and NK markers. CMV-IgG were present in 23 patients, 13 of them received a CMV positive kidney;18 recipients were CMV IgG-negative and 8 got a positive kidney.CMV infection affected 14 patients, with systemic symptoms in 3. There was no difference in age,gender,creatinine clearance at day 30, CMV-IgG status and Ly subsets when comparing the 14 patients to the 27 others. Both groups were similar as regards to the use and dosage of all the immunosuppressors,except for steroids which were higher in patients who did not suffer CMV infection (30.8+/-12 vs24+/-3.9 mg/kg/mo, p=0.04). EBV-GE at days 15/30 were 756/9651 in CMV non-infected and 2113/17425 in CMV infected patients (NS). Using a ROC-curve,a threshold of 2000 GE was defined to discriminate patients at high risk of CMV infection, but there was no link between EBV-GE>2000 and the occurrence of CMV. By contrast, there was a strong association between GE at day 15 and the number of pp65-positive nuclei at day 30-40 (37 in EBV-GE<2000 vs 425 in GE>2000, p=0.02).This was relevant since the highest pp65 was associated with clinical severity. Highest steroid dosage was also associated with EBV-GE>2000.

In summary, there was a relationship between EBV viral load at day 15 and the severity of CMV infection at day 30-40, but not with the risk of CMV outbreak 15 to 25 days later. EBV-GE>2000 at day 15 might however be a deciding factor for an early start of Ganciclovir. The highest steroid dosage associated with the highest EBV viral load and the absence of CMV flaring was not explained in this study.

Abstract# 201 **Poster Board #-Session: P14-I**

CYCLOSPORINE A USE IS NOT INDEPENDENTLY ASSOCIATED WITH INCREASED PLASMA TOTAL HOMOCYSTEINE LEVELS IN AUSTRIAN OR UNITED STATES CHRONIC RENAL TRANSPLANT RECIPIENTS. Andrew G. Bostom,[1] Gere Sunder-Plassmann,[2] Manuela Fodinger,[2] Reginald Y. Gohh,[3] Jacob Selhub.[4] [1]*Internal Medicine, Memorial Hospital of Rhode Island, Pawtucket, RI;* [2]*Department of Laboratory Medicine, University of Vienna, Vienna, Austria;* [3]*Internal Medicine, Rhode Island Hospital, Providence, RI;* [4]*Vitamin Bioavailability Laboratory, Tufts Jean Mayer USDA-HNRCA, Boston, MA.*

It has been contended, based on a single uncontrolled observation (Transplantation 1996; 61: 509-12.), that cyclosporine A (CsA) use "independently" raises plasma total homocysteine (tHcy) levels in renal transplant recipients (RTR). We examined this contention in a large number of chronic, stable (i.e., at least 6-months post-transplantation with a serum creatinine of 4.2 mg/dL or less) Austrian [n=637] and United States [US; n=86] RTR. We controlled for the major established determinants of tHcy levels readily available in the clinical setting, including renal function (as serum creatinine), B-vitamin status (of plasma folate and B-12), and age. Linear modeling with analysis of covariance adjusted for age, serum creatinine, and plasma folate and B12, revealed that geometric mean tHcy levels (nmol/mL) did not differ comparing either the Austrian or US RTR receiving CsA, relative to those untreated with CsA: [Austrian CsA users, n=591, tHcy = 15.5; Austrian CsA non-users, n=46, tHcy=14.9; p=0.311]; [US CsA users, n=69, tHcy = 15.1; US CsA non-users, n=17, tHcy=14.6; p=0.673].

We conclude that CsA use, independent of the known determinants of tHcy levels readily available in the clinical setting, has neither a statistically significant nor clinically relevant impact on tHcy levels among chronic, stable RTR.

Abstract# 202 **Poster Board #-Session: P15-I**

A COMPARATIVE STUDY OF PROPHYLACTIC ORAL GANCYCLOVIR AND VALACYCLOVIR IN HIGH-RISK KIDNEY TRANSPLANT RECIPIENTS. Angelito Yango,[1] Abdulrahman Zanabli,[1] Paul Morrisey,[1] Abinash Roy,[1] Reginald Gohh.[1] [1]*Rhode Island Hospital, Providence.*

Cytomegalovirus (CMV) infection is a signifiant cause of morbidity and mortality in organ transplant patients and occurs in approximately 20-60% of all renal transplant recipients. Disease manifestations range in severity from mild febrile syndrome to multi-organ involvement and usually occur within 6 months after transplantation. In organ transplant recipients, prophylactic treatment with oral ganciclovir significantly reduces the incidence of CMV infection. Recently, valacyclovir, a prodrug of acyclovir, has been shown to be safe and effective in preventing CMV disease after transplantation. Furthermore, a three month course of valacyclovir is $1,500.00 less than ganciclovir. We compared the efficacy of oral ganciclovir with valacyclovir in preventing CMV infection after kidney transplantation. A retrospective analysis was done in two groups of patients. Group 1 (n=70) consisted of patients prophylactically treated with PO ganciclovir 1 gram TID for 12 weeks post transplant and Group 2 (n=60) consisted of patients treated prophylactically with valcacyclovir 2 grams QID. All patients had a minimum follow up of 6 months after transplant with no statistical differences in age, gender, race, cause of end stage renal failure, type of transplant, induction rate and CMV D/R status. Both drugs were well tolerated with only 1 patient in Group 1 discontinuing therapy due to side effects.

The incidence of acute rejection during the first six months of follow up was similar in both groups (13.8% vs 9.0%). Likewise, the incidence of CMV infection in the two groups was not statistically significant (6.9% vs 5.4%). In both groups, all CMV infections occured in high risk patients (D+/R+ and D+/R-).

We conclude that prophylactic use of valacyclovir is as effective as oral ganciclovir in reducing CMV infection after kidney transplantation and that treatment with valacyclovir offers and advantage over ganciclovir in terms of cost reduction.

Abstract# 203 **Poster Board #-Session: P16-I**

THE EFFECT OF MYCOPHENOLATE MOFETIL ON HEPATITIS B VIRAL LOAD IN STABLE RENAL TRANSPLANT RECIPIENTS WITH CHRONIC HEPATITIS B. Bart D. Maes,[1] Jos F. van Pelt,[2] Peeters Jacques,[1] Nevens Frederik,[2] Evenepoel Pieter,[1] Kuypers Dirk,[1] Messiaen Thierry,[1] Fevery Johan,[2] Vanrenterghem F. Yves.[1] [1]*Nephrology, University Hospital Gasthuisberg, Leuven, Belgium;* [2]*Hepatology, University Hosital Gasthuisberg, Leuven, Belgium.*

Background : Mycophenolate mofetil (MMF) is widely used in the prevention of acute renal graft rejection. It was also shown to inhibit hepatitis B virus (HBV) replication in cultures of human hepatocytes. The aim of the present study was to investigate the effect of MMF on HBV replication in renal transplant patients with chronic hepatitis B. Methods : Twelve stable HBV(+) renal transplant patients with a maintenance immunosuppressive regimen based on corticosteroids (CS) ± cyclosporine (CsA) ± azathioprine (Aza), were given MMF therapy either in stead of Aza or in addition to CS ± CsA. HBV concentrations were measured by the Amplicor HBV-Monitor test on at least three separate occasions before and at regular interval until 1 year after introduction of MMF. Results . Baseline HBV viremia was 2.8 ± 3.1 x 109 copies/mL Introduction of MMF at a mean dose of 2 g/day for a year was not followed by significant changes in HBV viremia (2.5 ± 3.3 x 109). In the individual patient, HBV DNA changed >1 log copies/mL in only 3 patients (2 decrease, 1 increase). Also no significant changes were noted in liver tests and in hematological cell counts. Conclusion : The introduction of MMF does not affect HBV replication in stable HBV(+) renal transplant recipients with high viremia on maintenance therapy containing CS. Whether MMF is able to suppress HBV replication in vivo in patients not taking immunosuppressive therapy or in combination with nucleoside analogues remains to be investigated.

Abstract# 204 **Poster Board #-Session: P17-I**

INCIDENCE OF POSTTRANSPLANT GLYCEMIC DISORDERS IN RENAL TRANSPLANT RECIPIENTS TREATED WITH LOW AND STANDARD DOSE TACROLIMUS. Bart D. Maes,[1] Dirk Kuypers,[1] Pieter Evenepoel,[1] Thierry Messiaen,[1] Chantal Mathieu,[2] Willy Coosemans,[3] Jacques Pirenne,[3] Yves Vanrenterghem.[1] [1]*Department of Nephrology, Univeristy Hospital Gasthuisberg, Leuven, Belgium;* [2]*Department of Endocrinology, Univeristy Hospital Gasthuisberg, Leuven, Belgium,* [3]*Department of Transplant Surgery, Univeristy Hospital Gasthuisberg, Leuven, Belgium.*

Background : Because diabetes mellitus (DM) has a major impact on long-term outcome of renal allografts, attempts are made to reduce the risk the development of posttransplant glycemic disorders. High trough levels of FK506 after renal transplantation are known to predispose for posttransplant DM. Aim · The aim of this study was to evaluate the incidence of posttransplant glycemic disorders in renal transplant recipients, treated with a low versus a standard dose of FK-506

Methods : 72 patients without history of glucose metabolism abnormalities and treated with an induction immunosuppressive regimen consisting of FK-506, methylprednisolone (MP) and mycophenolate mofetil (MMF) were analysed. In the first group (n=33), the aimed trough level of FK506 during the first 3 months after transplantation was 5 to 10 ng/mL, while in the other (n=39) it was aimed at 10 to 15 ng/mL. In the former group, additional induction therapy was given using Dachzumab. All subject were evaluated for glucose metabolism disorders at 3 months using an oral glucose tolerance test. According tot the new WHO criteria, impaired glucose tolerance (IGT) was defined as a plasma glucose level > 140 mg/dl and DM as a plasma glucose level > 200 mg/dL at 2 hours.

Results . The mean dose of steroids during the first three months was 0.17 ± 0.32 and 0.14 ± 0.03 mg/kg/day in the low versus standard dose FK506 group; the mean trough levels of FK506 were 10.7 ± 1.2 and 15.7 ± 2.3 ng/mL, resp., at 1 month, and 9.9 ± 0.9 and 14.8 ± 1.6 ng/mL, resp., at three months. The mean number of trough levels of FK506 > 15 ng/mL was 1.9 ± 1.7 and 8.3 ± 3.7, resp, in the low and standard dose

FK506 group In the group receiving low doses of FK506, the incidence of IGT was 30 %, and of DM 6 %. In the group on standard dose of FK506, there was a 26 % incidence of IGT and a 15 % incidence of DM. Conclusion : Induction with an FK-506 - MP - MMF based immunosuppressive regimen, resulted in a high incidence of glucose metabolism disorders in renal transplant recipients. Using lower doses of FK506 in combination with monoclonal antibodies, the incidence of DM was reduced by more than 50%. Nevertheless, the incidence of IGT remained unaltered.

Abstract# 205 Poster Board #-Session: P18-I

ERYTHROPOIETIN THERAPY IMPROVES POST-TRANSPLANT ANEMIA AND CHRONIC RENAL TRANSPLANT DYSFUNCTION. Bryan N. Becker,[1] Yolanda T. Becker,[2] Glen E. Leverson,[2] Dennis M. Heisey.[2] *[1]Medicine, University of Wisconsin, Madison, WI; [2]Surgery, University of Wisconsin, Madison, WI.*

Hypoxia is an important stimulus for renal fibrosis and thus likely contributes to chronic allograft nephropathy. One strategy to offset renal hypoxia is to treat any co-existing anemia to improve renal oxygen delivery Erythropoietin (epo) is an anemia therapy with proven efficacy. Moreover, human renal tissue expresses epo receptors involved in renal repair responses and the regulation of hypoxia genes including NADPH oxidase. We evaluated outcomes in 116 renal transplant (RTx) patients (pts) who received epo post-RTx. Two separate pt groups were evaluated: (1) pts who initiated epo within 4-18 days post-RTx (n=59)(avg. dose 9670 ± 2500 U SQ q week)(mean ± S.E.); and (2) pts who initiated epo > 293 days after RTx (n=57)(avg. dose 9788 ± 5754 U SQ q week). Group 1 pts remained on epo for 122 ± 164 days (d). Their avg. hematocrit (HCT) increased from 34%(d^0) to 35.8% (d^{100}) to 36.9% (d^{200}). Group 2 pts were on epo for 205 ± 197d with an avg. initial HCT of 31.5% (d^0). HCT increased in Group 2 pts to 32.7% (d^{100};p=0.002 vs. d^0) and 33.4% (d^{300}; N.S. vs. d^{100}). Avg. Scr increased slightly in Group 1 pts from 1.51 mg/dl (d^0) to 1.57 mg/dl on d^{300}falling to 1.53 mg/dl by d^{600}. Group 2 pts showed a similar trend with a small increase in Scr after starting epo (d^0:2.86 mg/dl; d^{100}:2.9 mg/dl; n.s. vs. d^0). Thereafter, avg. Scr decreased to 2.6 mg/dl (d^{300};n.s.;p=0.07 vs. d^0) and 2.1 mg/dl (d^{600+}; p=0.05 vs. day 0). More than 95% of Group 1 and 2 pts remained on calcineurin inhibitors during epo therapy. Despite their poor graft function at the outset of therapy, 61% of grafts in Group 2 pts continued to function during the follow-up period. Epo also improved VO max 29 ± 6% in a subgroup of Group 2 pts (n=10) as determined by the STEP test on d^0 and d^{200}(p=0.024 vs. n=10 untreated pts with avg. HCT 30.6%). Thus, epo was effective in improving HCT when initiated early or late after RTx. It also led to improved exercise tolerance in pts with graft dysfunction. Interestingly, epo also was associated with a biphasic response in RTx function: an early increase in Scr followed by a significant decline in Scr. This pattern was evident in a majority of pts with chronic graft dysfunction. This may be due to the increased HCT with a gradual increase in renal oxygen delivery or, given epo's potential intrarenal effects, could result from a novel anti-inflammatory effect associated with epo itself.

Abstract# 206 Poster Board #-Session: P19-I

SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY: AN OVERVIEW OF THE LIPID PROFILE AND CARDIOVASCULAR RISK FACTORS. Christophe Legendre,[1] Neville Jamieson, José Colon, João Pena, Javier Martinez, Heide Sperschneider, Alfredo Mota, the Sirolimus Tri-continental Renal Transplant Study Group. *[1]Service de Néphrologie, Hôpital Saint-Louis, Paris, France.*

Purpose. Sirolimus (SRL)-induced hyperlipidemia would be acceptable if lipid elevations were treatable and if SRL had favorable effects on other cardiovascular risk factors. This paper examines cardiovascular risk factors in patients given an initial regimen consisting of SRL-cyclosporine (CsA)-steroids followed by CsA withdrawal. **Methods** 525 patients in Europe, Australia, and Canada, averaging 46 years old, received SRL 2 mg (tablets), CsA, and steroids when enrolled. SRL blood levels were to be maintained above 5 ng/mL (immunoassay). At 3 months ± 2 weeks, the 430 eligible patients were randomized (1:1) to remain on triple therapy, or to have CsA withdrawn and SRL troughs increased to 20 to 30 ng/mL.

Results. At 12 months, graft survival (95.8 vs 97.7%), patient survival (97.2 vs 98.1%) an primary acute rejection (13.5 vs 20.0%) were not statistically different between patients randomized to SRL-CsA-steroids and SRL-steroids, respectively. Of the 525 enrolled patients, 13 (2.5%) experienced a myocardial infarction or cardiac arrest, 7 (1.3%) of which were fatal. Nine events occurred during the 3 months preceding randomization, 3 were observed in the SRL-CsA-steroids group, and 1 occurred while on SRL-steroids maintenance therapy. Only 3.8% of all patients had treatment-emergent insulin-dependent diabetes There was no difference in lipid parameters between groups. In both groups, approximately 60% and 20% of patients received statin and fibrate treatment, respectively. HDL-cholesterol was normal or elevated in over 95% of patients. Diastolic and systolic blood pressure were lower when CsA was withdrawn, though significantly fewer hypertensive treatments were employed.

Results at 12 months (mean ±SE)

Regimen	T-Chol (mM)	Trig (mM)	LDL-C (mM)	HDL-C (mM)	LDL/HDL	D-BP(mmHg)	S-BP (mmHg)
SRL+CsA	6 0 ±0 1	2 2 ±0 1	3 5 ±0 1	1 5 ±0 04	2 5 ±0 1	81 2 ±0 8	140 ±1 4
SRL	6.3 ±0 1	2 5 ±0 1	3 5 ±0 1	1 6 ±0 05	2 4 ±0 1	78 7 ±0 8	134 ±1 4
p	0 31	0 07	0 64	0 84	0 61	0 027	0 007

Conclusion. SRL-CsA-steroid therapy followed by SRL-steroid maintenance therapy was associated with manageable lipid elevations, normal LDL/HDL cholesterol ratios and lower blood pressure. The incidences of cardiovascular events and diabetes were well within those reported for other therapies.

Abstract# 207 Poster Board #-Session: P20-I

SOLE RELIANCE ON RANDOM / CLINIC BP VALUES MIS-CLASSIFIES HYPERTENSION IN 20% OF RENAL TRANSPLANT RECIPIENTS (RTx) - AN AMBULATORY BP MONITORING (ABPM) ANALYSIS. S. Jayawardene, R. S. Bakri, H. O'Sullivan, D. J.A. Goldsmith. *[1]Nephrology and Transplantation Unit, Guy's Hospital, London, United Kingdom.*

Hypertension (H) remains a major problem after successful engraftment, and a significant cardiovascular risk factor. Most studies concentrate on daytime BP values, but it is well-known that night-time BP is abnormal in most renal patients (lack of normal diurnal fall in BP with sleep ie < 10% fall in systolic BP with sleep compared to waking BP). It is this nocturnal BP elevation that most closely correlates with end-organ damage. We wished to see how the diagnosis of H. was affected by the use of random daytime BP readings (clinic values) compared to 24 hour ambulatory BP monitoring (ABPM) data.

We performed 178 24 hour ABPM readings (30 minutes by day, 60 minutes by night) in 169 RTx patients (77 women, 92 men, mean age 45+/-13 years) over the period April 1998 to June 2000. Time from engraftment was 1 - 251 months (mean 42 months). 90% of patients were taking regular anti-BP treatment.

For each patient we compared the following BP measures - "clinic BP" (mean of 2 - 6 clinic BP readings over a 3 month period), "daytime ABPM" (mean of daytime BP values during awake period of 24 hrs ABPM), "nightime ABPM" (mean of night BP values during sleep period of 24 hrs ABPM).

	Systolic BP	Diastolic BP	% Normal BP	Normal BP
Clinic BP	141 7	83 5	30	<135/85
Daytime BP	139 2	83.9	37	<135/85
NightimeBP	135 6	79 7	21	<125/75
Diurnal BP fall	3 4%	5 6%		>10%

We found discordance in diagnosis of H. between "clinic BP" and "daytime BP" or "nightime BP" ABPM readings in 20% of the patients - in 7 cases (4%) there was pure "office / white coat H.", while in 29 cases (16%) the ABPM derived values showed raised BP, in contrast to normal clinic BP. In 17 cases, only the nightime BP values were elevated.

We have found in this large ABPM series first that there is a low incidence (4%) of "white-coat" hypertension in RTx (resulting in BP treatment where none may be needed), and more worryingly a 16% incidence of ABPM-hypertension not reflected in clinic values (leading to undertreatment of H. in these patients). ABPM is a very valuable, perhaps indispensible, tool in assessment of BP in RTx.

Abstract# 208 Poster Board #-Session: P21-I

MARKERS OF ACUTE INFLAMMATION ARE RAISED IN RENAL TRANSPLANTATION (RTx) PATIENTS, ESPECIALLY IN THE PRESENCE OF CARDIOVASCULAR DISEASE (CVD). Rashed S. Bakri,[1] Ben Afzali,[1] Peter J. Lumb,[2] George Chik,[2] Anthony S. Wierzbicki,[2] Martin Crook,[2] David Goldsmith.[1] *[1]Nephrology and Transplantation, Guy's Hospital, London, United Kingdom; [2]Chemical Pathology, Guy's Hospital, London, United Kingdom.*

Cardiovascular disease (CVD) remains a major challenge to the transplant patient and physician alike; premature death with a functional graft is too common, and chronic allograft deterioration may share many of the same risk factors as CVD. However, atherosclerosis is mechanistically complex, involving both dyslipidemia and also inflammation of the arterial wall.

78 RTx patients in good health were screened in outpatient clinic for cardiovascular disease (history, examination, investigations), and blood was drawn for plasma fibrinogen and high-sensitivity C reactive protein hs-CRP, and for lipid fractions. There were 44 men (M) and 34 women (W) (age 45.8 +/- 14.2 years). 58 patients had no evidence of CVD (29 M, 34 W; age 42.6 +/- 13.7 yrs) and 20 had evidence of CVD (15 M, 6 W; age 54.3 +/1 12.0 yrs, p < 0.01). There were 7 / 20 with diabetes (DM), and 6 / 20 smokers (S) in the CVD+ group (compared to 10 / 58 DM; 6 / 58 S). Mean creatinine was 167 +/- 77 umol/l (1.9 +/- 0.88 mg/%). Mean time post-engraftment was 72 +/- 79 (range 2 - 252 months).

	CVD+	CVD-	p
Age (years)	54 3 (12 0)	42 6 (13 7)	<0 01
BP (mmHg)	137 2 / 78 7	131 6 / 77 9	NS
Total C / HDL-C	4 27	3 93	NS
Creatinine (umol/l and mg%)	179 1 2 03	162 3 1 84	NS
Hs-CRP (mcg/l)	20 8	4 2	<0 05
Albumin (g/l)	37 4	40 4	NS
Fibrinogen (mmol/l)	5 17 (1 82)	4 0 (1 26)	<0 001

In the absence of intercurrent illness, both fibrinogen and CRP values were raised in RTx patients, especially in those with overt CVD. This supports the growing body of evidence that inflammatory mechanisms are important in the increased atherogenesis seen in R

Abstract# 209 **Poster Board #-Session: P22-I**

PLASMA TOTAL SIALIC ACID (SA) LEVELS ARE RAISED IN RENAL TRANSPLANT RECIPIENTS (RTx) AND CORRELATE WITH THE PRESENCE OF CARDIOVASCULAR DISEASE (CVD).

Rashed S. Bakri,[1] David J.A. Goldsmith,[1] Ben Afzali,[1] Peter J. Lumb,[2] George Chik,[2] Anthony S. Wierzbicki,[2] Martin Crook.[2] *[1]Nephrology and Transplantation, Guy's Hospital, London, United Kingdom; [2]Chemical Pathology, Guy's Hospital, London, United Kingdom.*

Cardiovascular disease (CVD) remains a major challenge to the transplant patient and physician alike; premature death with a functional graft is too common, and chronic allograft nephropathy shares many CVD-risk factors. However, atherosclerosis is mechanistically complex, involving both dyslipidemia and also arterial wall inflammation. Sialic acid (SA) is a family of > 30 negatively charged sugars (eg N-acetylated neuraminic acid) associated with the protein and lipid portions of lipoproteins, with diverse biological roles, and has been shown to correlate with the presence and severity of CVD in the general population, and in diabetics. We report on the plasma levels of total SA in RTx with and without CVD.

78 RTx patients in good health were screened in clinic for CVD (history, examination, investigaitons), and blood was drawn for plasma total SA, and lipid fractions. SA was measured using a novel specific enzymatic colorimetric assay (intra-assay variability ~ 3.5%).

There were 44 men (M) and 34 women (W) (age 45.8 +/- 14.2 yrs). 58 patients had no evidene of CVD (29 M, 34 W; age 42.6 +/- 13 7 yrs) and 20 had evidence of CVD (15 M, 6 W; age 54.3 +/- 12.0 yrs, p < 0.01). There were 7 / 20 with diabetes (DM), and 6 / 20 smokers (S) in the CVD+ group (compared to 10 / 58 DM; 6 / 58 S). Mean creatinine was 167 +/- 77 umol/l (1.9 +/- 0.88 mg/%). Mean time post-engraftment was 72 +/- 79 (range 2 - 252 months).

	CVD+	CVD-	P
Age (years)	54 3 (12 0)	42 6 (13 7)	<0 01
BP (mm Hg)	137 2 / 78 7	131 6/77 9	NS
Total C / HDL C	4 27	3 93	NS
Creatinine (umol/l and mg%)	179 1 2 03	162 3 1 84	NS
Sialic acid (mg/dl)	90 7 (23 8)	78 7 (15 0)	0 01

Reference range for plasma total SA in normal healthy populations is about 62 mg/dl. This study is the largest and only the second to report on SA in RTx. Plasma total SA levels are greatly increased in RTx, and that SA levels (unlike plasma lipids) are raised even further in RTx with CVD. Prospective work is now needed to investigate how successful plasma total SA will be as an independent marker for CVD.

Abstract# 210 **Poster Board #-Session: P23-I**

WHICH IS THE BETTER METHOD FOR CONTROL OF POST-RENAL TRANSPLANTATION (RTx) HYPERPARATHYROIDISM (HPT) - TOTAL OR SUBTOTAL PARATHYROIDECTOMY (PTX)?

Satishkumar A. Jayawardene,[1] William J. Owen,[2] David J.A. Goldsmith.[1] *[1]Nephrology and Transplantation, Guy's Hospital, London, United Kingdom; [2]Surgery, Guy's Hospital, London, United Kingdom.*

HPT after RTx is uncommon but can have serious consequences both for the allograft and the patient. Which surgical PTX procedure to perform is controversial for patients on dialysis and after RTx.

Since 1967, 1780 patients have undergone RTx at this unit with long-term follow-up. In this period 21 RTx patients have undergone 26 sugical PTX. Operations performed included 14 total PTXs - with significant enlargement / hyperplasia of all glands - (9 primary total PTX and 5 effective total-PTX after removal of remnant tissue left behind after previous subtotal PTX), versus 12 (deliverate) subtotal PTX. Of the 5 patients who had undergone redo PTX for recurrent HPT, 4 had previously undergone subtotal PTX when on dialysis, and one after Tx

Mean patient age was 51 years (range 19 to 77 years), with 8 mean and 13 women. Mean follow-up was 52 months (range 1 to 280 months) from PTX to death or present day. Only one patient died (four years later from cancer), all patients have functional allografts. There were no important post-operative surgical complications. Vitamin D therapy was needed long-term in the majority of patients post-PTX - 15/21 (71%) - in 11 / 12 (92%) of total-PTX compared with only 4 / 13 (31%) who had undergone subtotal-PTX ($p < 0.05$ by Chi-squared). One patient who underwent a subtotal-PTX post RTx required excision of the remnant gland for recurrent hyperparathyroidism three years later (1 / 12 or 8.25 % recurrence rate, cf. 0% for total-PTX group).

Biochemistry at the latest follow-up is tabulated

Calcium	Meq/l	Patients	intact PTH	Nmol/l	Patients
LOW	<8 8	1 (total PTX)	LOW	<10	12 (11 total PTX)
NORMAL	8 8 - 10 4	22	NORMAL	10 - 65	3
HIGH	>10 4	2	HIGH	>65	6

Our results show that long-term post-PTX hypocalcaemia was rare, but that vitamin-D dependency was present in 70% of patients (and in >90% of patients after total-PTX). Long-term low-PTH (ie functional hypoparathyroidism) was present in 50% of patients, nearly all after total-PTX. This was of no clinical significance however. Total-PTX is the definitive procedure for HPT (ie no recurrences) but at the expense of long-term vitamin-D dependency.

Abstract# 211 **Poster Board #-Session: P24-I**

DELAYED FUNCTION OF RENAL ALLOGRAFTS: INCIDENCE, OUTCOME AND RISK FACTORS. Graeme R. Russ.[1] *[1]ANZDATA Registry, Queen Elizabeth Hospital, Adelaide, SA, Australia.*

The databases of the Australian and New Zealand Dialysis and Transplant Registry (ANZDATA) and the Australian and New Zealand Organ Donation Registry (ANZOD) have been examined to determine risk factors and outcome measures for renal allografts that experience post-transplant delayed graft function (DGF). Primary cadaveric grafts performed from 1990 to 1999 have been studied. DGF was determined to have occurred if dialysis was required beyond day 1 post-transplant and immediate function (ImmF) if dialysis was not required beyond day 1. Grafts that never functioned have been excluded. Of 2944 evaluable grafts, 569 (19.3%) experienced DGF. 12 month graft survival was 91% for ImmF and 90% for DGF but after the first year grafts with ImmF had a superior survival such that there was a 9% difference at 9 years (p=0.0001, log rank). However, serum creatinine at 12 months was significantly higher in grafts with DGF (178 versus 141 umol/L, p<0.001, ANOVA). Survival of grafts with DGF of <7 days (n=206) was not different from grafts with ImmF, but DGF lasting >7 days was associated with a worse graft survival (p<0.0001 with ImmF and p=0.02 with DGF<7 days). Serum creatinine at 12 months was also significantly worse with DGF>7 days (188 umol/L). Rejection episodes occurring in the first month post-transplant were seen in 32% of grafts with DGF and 26% of grafts with ImmF (p=0.08). Biopsy-proven vascular rejection occurred more commonly in grafts with DGF>7 days compared to grafts with ImmF (16.2% v 8.9%, p=0.003) but not grafts with DGF<7 days (4%).

Donor factors which contributed significantly to DGF were high terminal serum creatinine, older age, death by cerebrovascular disease, history of chronic hypertension, the need for cardiopulmonary resuscitation, ischaemia time over 18 hours, use of dopamine/dobutamine as maintenance treatment, but not terminal oliguria, the use of adrenalin/noradrenalin, pitressin or prostacyclin as maintenance treatment, the use of mannitol, chlorpromazine or methylprednisolone as terminal treatment. Recipient factors which protected against DGF were pretransplant treatment with peritoneal dialysis, administration of diltiazem in the first 24 hours, as well as perfusion of the kidney with UW solution. Recipients with a high PRA had a higher incidence of DGF.

In summary, DGF after primary cadaveric renal transplantation, especially beyond 7 days post-transplant, is associated with poorer long term graft function and a greater incidence of vascular rejection. Modification of factors to reduce DGF may improve long term outcome.

POSTER SESSION I:
KIDNEY - IMMUNOSUPPRESSION A I

Abstract# 212 **Poster Board #-Session: P25-I**

HUMAN GENE EXPRESSION BY ALLOACTIVATED LYMPHOCYTES (MLR)IN SEVERE COMBINED IMMUNE DEFICIENT MICE (SCID). Ahmed S. Shoker, Zhao-Rong Lun, Rezvan Choudry.

The SCID mouse provides a neutral environment to study human immune responses. We therefore tested human cytokine gene expression (IL-2, IL-4,Interferon Gamma (IFNγ), Transforming Growth Factor Beta 1 (TGFβ1); and CD40 ligand (CD40L) in splenic extracts of SCID mice after intraperitoneal injection of PBL's from two persons (direct MLR) or allopeptides (indirect MLR) in presence or absence of Cyclosporin A (CSA) or FK506 given for seven days.

METHODS:

Cytokine gene expression in SCID splenic extracts was detected by Rt and quantitative (Qrt, for IFNγ, TGFβ1 and CD40L) PCR. All cells, allopeptides, CSA (25 mg/kg) or FK506 (0 5 mg/kg) were administered intraperitoenally.

RESULTS:

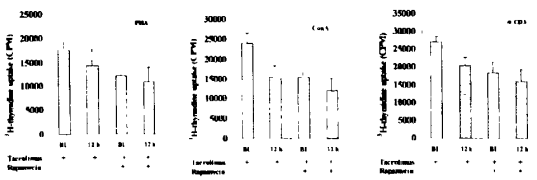
The numbers of SCID mice expressing the human cytokine genes were as follow: In both direct and indirect MLR IL-4 gene was present in 50% at three and seven days. IL-2, IL-10, IFNγ, TGFβ were all expressed in more than 75% of the control mice. All gene expressions were decreased fourteen days after engraftment to 0%-50%. Qrt results showed significant variation in gene expression levels between different PBL combinations. Concomitant Cyclosporin or FK506 administration with human lymphocytes or allopeptides for seven days did not block early or late (one week after seven day administration of CSA or FK506) cytokine gene expression in either the direct or indirect MLR but paradoxically enhanced levels of IFNγ, TGFβ and CD40L gene expression by more than two times in some experiments.

CONCLUSIONS:

Similar to studies in humans and at variance with in vitro studies, Cyclosporin or FK506 did not abrogate cytokine gene expression. The results explain late transplant rejections after rapid calcineurin inhibitor withdrawal or reduction and that our model may be used as a surrogate model to predict human transplant alloresponse.

Abstract# 213 **Poster Board #-Session: P26-I**

EFFECT OF CONCOMITANT TACROLIMUS AND RAPAMYCIN THERAPY ON IMMUNE FUNCTION IN RENAL TRANSPLANT RECIPIENTS. Ashwani K. Khanna, Matthew S. Plummer, Cathy M. Bromberek, Sundaram Hariharan. *[1]Medicine, Medical College of Wisconsin, Milwaukee, WI, [2]Medicine, Medical College of Wisconsin, Milwaukee, WI; [3]Surgery, Medical College of Wisconsin, Milwaukee, WI; [4]Medicine, Medical College of Wisconsin, Milwaukee, WI.*



We have demonstrated an efficient inhibition of lymphocyte proliferation with a combination of sub-clinical doses of rapamycin with tacrolimus or cyclosporine.In this study, we explored the effect of a combination therapy of tacrolimus and Rapamycin on human subjects after approval from the institute review board (IRB).A total of 10 stable renal transplants recipients were selected to participate in a pharmacokinetic (PK) study with a combination of Tacrolimus and Rapamycin. These patients were receiving tacrolimus (0.05-1.0 mg/Kg BID), with MMF/azathioprine and prednisone. Blood from these patients were drawn at time zero and 12 h interval for lymphocytes and serum samples. MMF/azathioprine from these patients were discontinued and replaced with Rapamycin (0.5 to 3 mg/Kg), patients were monitored for 14 days and blood was drawn at time zero and 12 h interval for lymphocytes and serum samples Lymphocyte proliferation was quantified by 3H-thymidine uptake assay (results expressed as counts per minute). The mRNA expression of TGF-beta, TNF-alpha, IL-10, IL-6, IFN-gamma and was studied by RT-PCR assay (expressed as ratio of gene to b-actin, a housekeeping gene) and the circulating levels of TGF-beta, IL-2, IFN-gamma and TNF-alpha was studied by specific ELISA kits. The combination of tacrolimus with rapamycin resulted in a significant inhibition of lymphocyte proliferation (Mean ± SEM, n=10) compared to tacrolimus alone (PHA: 17510 ± 4094 vs 10963 ± 3008 p<0.05; ConA: 23995 ± 2449 vs 12124 ± 2946 p<0.006; and anti-CD3: 27089 ± 1557 vs 16093 ± 3097 p<0.005).These results shown in figure demonstrate the efficacy of the combination therapy with tacrolimus and rapamycin.More significantly, the mRNA expression of proinflammatory cytokines TNF-alpha, Interferon-gamma and cyclins G1, D3 and E were significantly decreased in patients treated with combination of tacrolimus and rapamycin. In conclusion, these novel findings provide a mechanism and rationale for a concomitant therapy with tacrolimus and rapamycin in renal transplant patients.

Abstract# 214 **Poster Board #-Session: P27-I**

USE OF MICROEMULSION FORMULATIONS OF CYCLOSPORINE IS ASSOCIATED WITH AN INCREASED INCIDENCE OF HISTOLOGIC EVIDENCE OF CHRONIC CYCLOSPORINE TOXICITY. Bernard Benedetto,[1] Robert Madden,[1] Alexander Kurbanov,[1] Michael Germain,[2] George Lipkowitz.[1] *[1]Department of Surgery, Transplant Division, Tufts University School of Medicine, Springfield, MA; [2]Department of Medicine, Renal Division, Tufts University School of Medicine, Springfield, MA.*

The purpose of this study was to examine the impact of the microemulsion formulation of cyclosporine on the long-term (>1 year) incidence of chronic cyclosporine toxicity in renal allografts. This formulation is known to have increased bioavailibility compared to the oil-based formulation and may result in increased nephrotoxic exposure to the allograft. We performed a retrospective review of all renal allograft biopsies performed more than 1 year post transplant for declining allograft function between 1992 and 2000(n=173). Biopsy results were separated into two groups. Group one (n=82)consisted of biopsies from 1992-1996 when oil-based oral formulation cyclosporine was used. Group two (n=91) consisted of biopsies from 1996-2000 when the new microemulsion-based formulation was used. Histologic evaluation of renal allograft biopsies was performed by one of two pathologists with extensive experience in renal histopathology. There was a significant increase in the incidence of histologically diagnosed chronic cyclosporine toxicity in the microemulsion group (14.3% vs. 1.2%, p<.001) There was no difference in the incidence of chronic rejection in the two groups (14.6% vs. 21.9%, p=ns) There were no statistically significant differences in age, sex, race, cyclosporine trough levels, number of prior renal allografts or cause of renal failure between the two groups In conclusion, although the improved pharmacokinetics of the microemulsion formulation of cyclosporine may lead to decreased intra-patient variability and more consistent serum levels, long term (>1 year)use may be associated with an increased incidence of histologic evidence of chronic cyclosporine toxicity. The clinical relevance of prolonged renal allograft exposure to the microemulsion formulations of cyclosporine requires further evaluation.

Abstract# 215 **Poster Board #-Session: P28-I**

TACROLIMUS PHARMACOKINETICS AFTER KIDNEY TRANSPLANTATION. Felix Braun,[1] Beatrice Peters,[1] Ekkehard Schütz,[2] Thomas Lorf,[1] Ruben Canelo,[1] Michael Oellerich,[2] Burckhardt Ringe.[1] *[1]Georg-August-Universität, Klinik für Transplantationschirurgie, Göttingen, Germany; [2]Georg-August-Universität, Abteilung Klinische Chemie, Göttingen, Germany.*

Tacrolimus (Tac) trough level adjusted dosing is recommended due to its highly variable inter- and intraindividual bioavailability. Therefore, we studied tacrolimus pharmacokinetics (PK) to evaluate the use of tacrolimus trough level adjusted dosing especially in the early period after kidney transplantation (KTx).

Twenty patients (9 female, 11 male) with a median (range) age of 40 (32-69) years underwent KTx for end-stage renal disease. Tacrolimus was given orally at a starting dose of 2x0.075 mg/kg/d. Tacrolimus dosages were adjusted to a target range of 10-15 µg/l. Tac-PKs (T 0, 0.5, 1, 2, 3, 4, 6, 8, 9, 10, 11, 12 h) were taken after the first dose (PK1), during the second week (PK2), and at three months after KTx (PK3). Tacrolimus concentrations were measured by MEIA II.

Tac pharmacokinetik data are depicted in the table below. Correlation of tacrolimus trough levels (c12h) and Tac-area under the curve concentrations (AUC) were r=0.82 PK1, r=0.9 PK2, and r=0.75 PK3. From PK1 to PK3, there was a trend to shorter times in reaching maximal tacrolimus concentrations (tmax), while the maximal tacrolimus concentrations (cmax) did not differ.

Tacrolimus trough levels had a good correlation to Tac-AUCs after first dose (PK1), early (PK2) and late steady state (PK3). This finding suggests that the first trough levels are good indicators of systemic exposure. The first tacrolimus trough levels might already be used to optimize the trough level adjusted dosing.

PK	n	Tac (mg/kg)	AUC (mg/l*h)	tmax (h)	cmax (mg/l	c12h (mg/l)
1	20	0.07±0.01	132.9±85.9	2.5 (0.5-11)	22.4±11.7	8.2±7.4
2	20	0.09±0.02	159.3±47.8	2 (0.5-9)	22.5±8.8	9.3±2.7
3	16	0.07±0.03	175.8±33.8	1 (1-4)	24.2±7.1	10.6±1.9

Abstract# 216 **Poster Board #-Session: P29-I**

LATE CORTICOSTEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS ON TRIPLE IMMUNOSUPPRESSIVE THERAPY. RANDOMISED CONTROLLED TRIAL. Christopher K.T. Farmer, Ian C. Abbs, Rachel M. Hilton, Geoff Koffman, Jane Watkins, Steven H. Sacks. *[1]Department of Nephrology and Transplantation, Guy's Hospital, London, United Kingdom.*

Corticosteroids have been the mainstay of immunosuppression in clinical transplantation for 30 years but cause significant morbidity. Over 70% of renal transplant recipients remain on long-term steroid therapy.

The aims of this study were to assess in a randomised controlled trial the safety and benefits of late steroid withdrawal in renal transplant recipients with Creatinine(Cr)<200µmol/l) on triple immunosuppression (prednisolone, azathioprine and Neoral). Primary end-points were acute cellular rejection, graft and patient survival. Secondary end-points included serum Cr, change in cystatin C, cholesterol, blood pressure, body weight, bone mineral density (BMD) and rate of change of Cr.

In 1997 all 608 patients in our centre were screened, 209 were eligible. 92 were randomised (44 withdrawal, 48 control); all other patients were prospectively followed. 57 withdrew steroids outside the trial and 60 declined entry. Prednisolone dose was reduced by 1 mg/month. A rise in serum Cr of >15% resulted in a renal biopsy. BMD (DEXA) was measured at baseline and at 12 months.

The results of this study illustrate:

1. Late prednisolone withdrawal improves bone density.
2. One-year post withdrawal there is no significant change in mean serum Cr in the withdrawal limb compared to controls.
3. Rate of change of serum creatinine (ΔCr) appears to be worst in those patients who withdrew steroids (in RCT).
4. Highly selected patients who withdrew steroids outside the trial experienced no detrimental effect on serum creatinine and rate of change of creatinine was near zero.

	Declined	Withdrawn	Control (Randomised)	Withdrawal (Randomised)
n	60	50	48	44
Males (%)	41(68%)	25 (50%)	29 (59%)	30 (70%)
Age (Mean)	40.8 (15.0)	44.2 (13.7)	44.6 (12.7)	41.5 (17.4)
Follow up years (SD)	1.9 (0.3)	1.9 (0.8)	1.3 (0.3)	1.4 (0.3)
Acute Rejection	2	0	1	1
Chronic Rejection	2	1	1	3
Start mean Cr(µmol/l)	125	112	131	131
End mean Cr(µmol/l) (p)	127 (ns)	113 (ns)	137 (ns)	142 (ns)
ΔCr µmol/l/year (p)	4.6	2.8 (ns)	2.0	7.9 (p=0.02)
Femoral Neck BMD	-	-	-0.1%	+3.9%

Abstract# 217 **Poster Board #-Session: P30-I**

WHAT IS THE VALUE OF SHORT SYNACTHEN TESTS IN PREDICTING EASE OF STEROID WITHDRAWAL IN RENAL TRANSPLANT RECIPIENTS? RANDOMISED CONTROLLED TRIAL. Christopher K.T. Farmer, Ian C. Abbs, Rachel M. Hilton, Geoff Koffman, Jane Watkins, Steven H. Sacks. [1]*Department of Nephrology and Transplantation, Guy's Hospital, London, United Kingdom.*

Withdrawal of steroids from renal transplant recipients may not be without risk of acute or chronic rejection. Some individuals develop a syndrome of malaise, polyarthralgia, muscle aches, depression and occasionally erythematous rashes. This has been termed steroid withdrawal syndrome (SWS).

We proposed that individuals who develop biochemical hypoadrenalism following steroid withdrawal would be more at risk of rejection and development of steroid withdrawal syndrome.

The aim of our study was to investigate the predictive value of short Synacthen tests on individuals withdrawing steroids in a randomised controlled trial. 92 patients were randomised to either withdraw steroids gradually (1mg/month, 44 patients) or to remain on steroids (48 patients). Patients withdrawing prednisolone had standard dose Synacthen tests (250mcg synthetic ACTH, serum cortisol at T0, T30mins, T60mins) performed at baseline, on completion of steroid withdrawal and 3 months post withdrawal. Two Synacthen tests were performed in the control group (19 patients) 6 months apart to confirm no change in adrenal function. All Synacthen tests were performed in the morning and, in those patients on steroids, prednisolone levels were measured to confirm that patients had omitted the last dose. Three patients in the withdrawal group had biopsy proven chronic allograft nephropathy. There was no correlation between hypoadrenalism and the development of rejection (acute or chronic) or the rate of decline in renal function. 8 patients developed SWS in the withdrawal group, none in the control group. Those patients who developed steroid withdrawal syndrome had abnormal Synacthen tests at the time of steroid withdrawal (mean),see table. However, of the patients who developed SWS, two developed chronic allograft nephropathy ($p<0.041$, Fishers Exact Test).

This study illustrates:

1. Late withdrawal of corticosteroids is associated with a significant incidence of symptoms (9%).
2. Hypoadrenalism is predictive of the development of symptoms following steroid withdrawal.
3. Patients with symptoms on steroid withdrawal may be more likely to develop chronic allograft nephropathy.

Synacthen test at the time of withdrawal	Baseline	30 mins	60 mins
Mean Cortisol (SEM) Symptomatic patients	261 (4 9)	496 (9 6)	546 (9 9)
Mean Cortisol (SEM) Asymptomatic patients	340 (6 9)	573 (10.5)	637 (13 8)

Abstract# 218 **Poster Board #-Session: P31-I**

SUSTAINED LOW REJECTION RATES WITH TACROLIMUS THERAPY: TWO YEAR FOLLOW-UP OF A LARGE, MULTICENTRE EUROPEAN TRIAL IN RENAL TRANSPLANTATION. Domingo Del Castillo Caba, the Spanish-Italian Tacrolimus Renal Transplantation Study Group. [1]*Servicio de Nefrologia, Hospital Reina Sofia, Cordoba, Spain.*

This study compared the efficacy and safety of tacrolimus-based dual therapy (tacrolimus/steroids) with triple therapy (tacrolimus/steroids/azathioprine) in renal transplant recipients. **Methods**: In this 3-month (with a 21-month follow-up), prospective, open, parallel-group study, 25 centers in Spain and 11 centers in Italy randomized 475 adult patients to either dual (n=236) or triple (n=239) therapy. The two study groups had similar baseline characteristics. **Results**: During the first 3 months, the incidence of acute rejection was 28.8% (dual) and 29.7% (triple). Biopsy-proven rejection ocurred in 16.5% (dual) and 15.5% (triple) of patients; steroid-resistant rejections were experienced by 5.1% (dual) and 3.8% (triple) of patients. Between Months 4-24, new acute rejections occurred in 5 (dual) and 8 (triple) patients; new steroid-resistant rejections were reported in 2 (dual) and in 1 (triple) patients. At Month 24, 10 patients (4.2%) in the dual group and 14 patients (5.9%) in the triple group had chronic rejection Two years post transplant, patient survival was 97.8% (dual) and 96.0% (triple), graft survival was 91.9% and 91.6%, respectively (Kaplan Meier method). Serum creatinine at Month 24 was 150.1 µmol/L (dual) and 154.1 µmol/L (triple). The incidence of leukopenia between Months 4-24 was 2.1% (dual) and 10.5% (triple), $p<0.001$. The 3-month incidence of de-novo IDDM was 5.5% (dual) and 4.4% (triple). During the follow-up, de-novo IDDM occurred in 1 patient of the triple therapy group. In the first year post transplant, 7 patients (dual: 2, triple: 5) developed malignancies; in the second year, 4 additional malignancies were reported (dual: 1, triple: 3). At Month 24, 64.0% (dual) and 60.3% (triple) of patients received antihypertensive medications; 14.4% (dual) and 12.6% (triple) of patients received antihyperlipidaemics. The mean oral daily tacrolimus dose at Month 24 was 0.1 mg/kg in both groups, the corresponding mean whole blood trough levels were 10.04 ng/mL (dual) and 10.23 ng/mL (triple). The initial treatment was adhered to by 67.4% (dual) and 46.4% (triple) of patients. **Conclusions**: Both dual and triple tacrolimus regimens were efficacious and safe over a 2-year period. Both recurrent and new onset late rejections (after Month 3) are rare events with tacrolimus treatment. High patient and graft survival was sustained up to 24 months post transplantation.

Abstract# 219 **Poster Board #-Session: P32-I**

KINETICS OF EARLY PERIPHERAL BLOOD LYMPHOCYTE RECONSTITUTION FOLLOWING INDUCTION TREATMENT WITH ANTITHYMOCYTE GLOBULIN (THYMOGLOBULIN®) IN KIDNEY TRANSPLANTATION. E. Renoult,[1] M. Ladrière,[1] M. C. Béné,[2] M. N. Kolopp-Sarda,[2] G. Faure,[2] M. Kessler.[1] [1]*Nephrology;* [2]*Immunology, CHU, Nancy, France.*

Whereas polyclonal antilymphocyte antibodies have been shown to be extremely immunosuppressive in organ transplantation via depletive and nondepletive mechanisms on immunocompetent T cells, little is known about the kinetics of T-cell regeneration after induction therapy with these agents.

The aim of the present study was to assess peripheral blood lymphocyte (PBL) dynamics following the use of rabbit antithymocyte globulin ATG and the clinical issues

The reconstitution of PBL was prospectively studied in 51 patients (age 18-69 yrs, 31 males and 20 females) who received induction with ATG (Thymoglobulin®, IMTIX-SangStat) and methylprednisolone (40 mg/d) during the 9.8 ±1.5 first days after their first kidney transplantation.ATG was used according to the CD3 subset level(1-5%). All the patients had maintenance immunosuppression with microemulsion cyclosporine (CsA) and prednisone. CsA was introduced 4.2 ± 1.4 days post-transplant Sequential follow-up of PBL subsets was performed during the first trimester (daily during ATG therapy, bi-weekly until the second month and then bi-monthly). PBL subsets were enumerated in flow cytometry using antibodies to CD3, CD4, CD8. Results were expressed as percentages of labeled PBL.Data concerning acute rejection and (viral, bacterial, fungal) infections were collected.

The levels of PBL were 69.8 ± 12.4% for CD3, 46.8 ±11.6% for CD4 and 15.9 ± 12.4% for CD8 cells before transplantation and 57.1+ 16.7% for CD3, 21.5 + 11.4% for CD4 and 27.1 + 12.3 % for CD8 cells at the end of the third month. The kinetics of PBL reconstitution after ATG therapy was extremely variable : the percentage of CD3 subset reached 20% after 1 to 67 days (mean 14 ± 12.1 days) and 50 % after 3 to 99 days (mean 39.2 ± 26.7 days). Only a tendency but no significant relation could be shown between slow cell regeneration and increased number of viral infections. On the hand, the incidence of acute rejection was higher (p=0,03) in the patients who had faster recovery of CD3 (>20% before day 20).

In conclusion, there is a wide individual variability in the kinetics of PBL repopulation, after induction with ATG, in spite of a similar immunosuppressive treatment. Analysis of immune reconstitution early after ATG treatment can be useful considering the trend towards greater incidence of viral infections if slow T cell regeneration and the higher risk of acute rejection in recipients with rapid recovery of PBL.

Abstract# 220 **Poster Board #-Session: P33-I**

USE OF C-2 MONITORING TO OPTIMIZE CYCLOSPORINE MICROEMULSION DOSING IN DE NOVO RENAL TRANSPLANT RECIPIENTS: A SAFETY ANALYSIS. Edward Cole,[1] Aziz Walele,[1] Daniel Cattran,[1] Stanley Fenton,[1] Catherine O'Grady,[1] Carl Cardella.[1] [1]*Multi Organ Transplant Programme, University of Toronto, Toronto, ON, Canada.*

Use of C-2 (2 hour post cyclosporine dose) target levels for cyclosporine monitoring has been validated in clinical studies in de novo liver transplantation, but requires further testing in renal transplantation. In this study,cyclosporine dose adjustments in de novo renal transplant recipients were made according to C-2 levels (with blinded C-0) and targeted to achieve levels of 1.7 ug/ml (early graft function) and 1.3 ug/ml (delayed graft function) within 3 to 5 days of drug initiation. In patients with delayed graft function, cyclosporine was initiated a mean of 5 days post transplant. Renal function and rejection rates were compared with a historical control group (n=15 in both) matched for gender, age, PRA, donor source (7 living donors and 8 cadaveric in both), and delayed graft function, but monitored with trough cyclosporine levels.

In the C-2 group, mean C-2 levels were 1.45+/-0 40 ug/ml(Day 3), 1.74+/-0.43ug/ml(Day 5-6) and 1.97+/-0.39 ug/ml(Day 30). Four patients did not reach their C-2 target by day 5 but only 1 failed to reach target by day 7. When unblinded, the mean C-0 for the C-2 group on day 5 was 355+/-121 vs 301+/-124 ug/ml for the C-0 group. There was 1 rejection episode in the C-2 group in a patient who failed to reach target, and none in the C-0 group.

Mean serum creatinine at 30 days was similar (145+/-35umoles/L for C-2 vs 142+/-41 umoles/L for C-0). Patients with early graft function had a mean day 5 C-2 of 2.09+/-0.433ug/ml and mean 30 day serum creatinine of 123+/-14 uM. Those with delayed graft function had a mean day 5 C-2 of 1.42+/-0.148ug/ml and a 30 day serum creatinine of 160+/-40 umoles/L (P=0.038). There was no correlation between C-2 at 3,5 or 30 days and serum creatinine at day 30. When the 8 patients in each group with early graft function were compared, the mean serum creatinine at day 30 was 123+/-27 (C-2) vs 145+/-28 umoles/L(C-0)(p=ns).

This pilot study shows that, over the short term, cyclosporine dose adjustment utilizing C-2 monitoring results in no adverse impact on renal function in de novo renal transplants despite a probable increase in early drug exposure.

Abstract# 221 **Poster Board #-Session: P34-I**

A RANDOMIZED, OPEN-LABEL, PROSPECTIVE STUDY COMPARING TWO DIFFERENT CYCLOSPORIN A AREAS UNDER THE TIME-CONCENTRATION CURVE FOR THE PREVENTION OF REJECTION IN RENAL TRANSPLANTATION.

Zita M.L. Britto,[1] Cristiane F. Alves,[1] Flavio J. Paula,[1] Luis S. Azevedo,[1] Maria Cristina R. Castro,[1] Francine C. Lemos,[1] Eduardo Mazzucchi,[1] Pedro R. Chocair,[1] Joao A. Fonseca,[1] William C. Nahas,[1] Luis E. Ianhez,[1] Elias David Neto.[1] *[1]Unidade de Transplante Renal, Universidade de Sao Paulo, Sao Paulo, Sao Paulo, Brazil.*

High CyA 2-hour level(C2)is said as necessary to prevent rejection. However, no other study either confirmed this or find levels for patients (pts) with induction therapy. Studies using calcineurin activity indicate a much lower CyA concentration as necessary for maximum inhibition. In this study, pts were randomized to either Low (2400-3400) or High (4300-5300 ng.ml/hr) CyA-AUC0-4. CyA blood levels were measured at 0,1,2 and 4 hr after morning dose. Measurements were performed at 4, 7, 14, 21 and 28 days and then every 2 weeks up to the 90th day. 27 pts were enrolled to the High and 33 to the Low group. No differences were found between Low vs High regarding to demographics, PRA, DGF, HLA and donor type and other immunosuppressants. Basiliximab, ATG or OKT3 were used in 50% of pts. Biopsy-proven AR up to the 90th day was the end-point. Mean follow-up was 213±110 days. AR occurred at a median of 12(8-19) days in 6(24%)of the High and in 10(30%)of the Low group(NS). Because 60% of the pts only, remained within their target AUC level, the AUCs at the time of AR episodes were compared among pts who rejected versus those who did not. By step-wise regression, C2 ($p<0.001$)and AUC0-4 ($p<0.001$)predicted the presence of AR. By logistic regression a AUC0-4 >4587ng.ml/hr or C2>1817ng/ml had a 78% probability of rejection-free. For induction, lower C2 (>1040ng/ml)and AUC0-4 (>3100 ng.ml/hr) were necessary. Of these 29 pts, 17 used OKT3/ATG and 12 Basiliximab. No differences in PRA, MMF and DGF occurred but the number of cadaver($p<0.03$) and re-Tx($p<0.001$) was higher in the OKT3/ATG group. C2 and AUC0-4 levels to prevent AR were similar in these two types of induction therapy (1072±527 vs 1002±527 ng/ml (NS) and 3211±1161 vs 3054±1555ng.ml/hr(NS, respectively. Abbreviated AUC0-4 (calculated with C2 only) showed similar results than trapezoidal AUC0-4. These data show that C2 should be used to monitor CyA after renal transplantation. C2 level >1800 ng/ml is necessary during the first 90 days following RTX to avoid rejection. The data also indicate, for the first time, that a lower C2 level (>1100 ng/ml) can be used for patients with induction therapy regardless of whether ATG/OKT3 or Basiliximab is chosen.

Abstract# 222 **Poster Board #-Session: P35-I**

SIROLIMUS (SIR) PLUS CYCLOSPORINE (CSA) TO WITHDRAW STEROID (SW) IN LONG TERM KIDNEY TRANSPLANT RECIPIENTS (KTX).

Franco Citterio,[1] Vittorio Alfieri,[2] Paolo Altieri,[3] Pasquale Berloco,[4] Marco Castagneto,[1] Francesco Marchini,[5] Gavina Murgia,[3] Luca Poli,[4] Paolo Rigotti,[5] Giuseppe Segoloni.[2] *[1]Surgery, Catholic University, Rome, Italy; [2]Nephrology, University of Torino, Torino, Italy; [3]Nephrology, Transplant Unit, Cagliari, Italy; [4]Surgery, University La Sapienza, Rome, Italy; [5]Surgery, University of Padova, Padova, Italy.*

Prospective randomized trials have shown a significative reduced rate of acute rejection (AR) in rapamycin-treated KTX. We speculated that the increased protection from AR of the Rapamacyn-Cyclosporine umbrella could allow successful Steroid-Withdrawal (SW) in long term KTX with steroids complications. A multicenter, prospective phase 2 pilot study of SW was initiated with the following entry criteria: KTX at least 12 months after TX, stable renal function with creatinine less than 2.5 mg/dl, adult, no AR by 180 days, presence of at least one of : post-tx diabetes, body overweight, osteoporosis, hypertension, skin/ocular complications. The study consisted of 3 phases A, B, C. In phase A (one month), 42 long term (132+/-75 months after Tx) KTX were converted to a triple immunosuppressive regimen with SIR (5-10 ng/ml) plus CSA (50-150 ng/ml) and steroids. In phase B (two months) steroids were tapered and stopped. In phase C KTX were maintained without steroids with SIR (8 - 20 ng/ml) plus CSA (50-150 ng/ml). Primary endpoints of the study were incidence of acute rejection and safety. RESULTS: Phase A was completed by 41/42 pts. In phase B 3 pts were treated for non biopsy-proven rejection, 1 patient died for causes unrelated to the study, 3 patients were removed from the study because of toxicity. In phase C (3 months follow-up), 1 pt was treated for non biopsy-proven rejection, 6 patients were removed from the study because of toxicity. In 38/42 pts SW, by intention to treat analysis, was successful without AR. Cumulative AR rate at 3 months after SW is 9.5%. 12/42 pts experienced increased creatinine and arthralgia, reversible with the reduction/suspension of SIR/CSA. The introduction of Sirolimus and SW did not significantly changed platelets counts, Cholesterol, and triglycerides respect to the screening evaluation (screening vs phase C 3 months: PTL 216+/-61 vs 224+/-58; Chol 222+/-56 vs 178+/-63; trigl 184+/-107 vs 206+/-139). These preliminary data indicate that Sirolimus together with Cyclosporine allow safe Steroid Withdrawal in long term renal transplant recipients. The 6 months follow up will be presented.

POSTER SESSION I:
KIDNEY - PEDIATRICS, RECURRENT DISEASE I

Abstract# 223 **Poster Board #-Session: P36-I**

PRELIMINARY RESULTS OF THE USE OF HUMANIZED ANTI-CD154 IN HUMAN RENAL ALLOTRANSPLANTATION.

Allan D. Kirk,[1] Stuart J. Knechtle,[2] Hans W. Sollinger,[2] Flavio G. Vincenti,[3] Scott Stecher,[4] Kari Nadeau.[4] *[1]NN-TAB, NMRC, Bethesda, MD; [2]Univ. of Wisconsin, Madison, WI; [3]UCSF, San Francisco, CA; [4]Biogen, Inc, Cambridge, MA.*

The humanized CD154-specific Mab hu5c8 has been shown to prevent allograft rejection in non-human primates. This report documents the initial experience with hu5c8 in human renal transplantation.

The initial trial involved ten patients enrolled at the time of cadaveric transplantation randomized to standard immunosuppression (CsA, MMF, and steroids; n=5) or hu5c8 plus MMF and a 16 day steroid taper (n=5). Hu5c8 was given IV: 20 mg/kg pre- and post-operatively, and on days 3, 10, 18, 28 and monthly for 6 months. Follow-up is greater than 1 year. All control patients remain well with a mean creat. of 1.6 at 6 months. Four of the hu5c8 patients remain well with a mean creat. of 0.9 at 6 months. Histology at 6 months in the control subjects showed CsA toxicity in 2, borderline changes in 1, and a nonspecific lymphocyte infiltrate in the remaining 2. Routine histology in the hu5c8 patients at 6 months showed Banff 1A rejection in 1, borderline changes in 2, and a nonspecific lymphocyte infiltrate in the remaining 2.

Two additional subjects were subsequently treated with hu5c8 at the time of living donor transplantation. Both received 70 mg/kg pre-op, 30 mg/kg post-op, and 30 mg/kg on days 3 and 10 and were prophylaxed against thromboembolism with low molecular weight heparin plus aspirin and converted to low dose coumadin One was treated with hu5c8 alone and also received 30 mg/kg on days 18 and 28. This patient had a steroid sensitive rejection on day 4, was started on steroids and MMF, and remains well with a creat. of 1.0. The other patient was treated from the onset with prednisone and MMF and remains well with a creat. of 1.4. Histology at 1 month showed in both patients an interstitial predominantly CD4 lymphocyte infiltrate similar to that reported from non-human primate studies.

No patients have had changes in their CD4 counts. The study drug was well tolerated during the infusion. Due to thrombo-embolic events in these patients and patients in hu5c8 trials for non-transplant indications, all trials involving hu5c8 have been halted pending additional study.

In summary, 7 patients have been transplanted using hu5c8. Most have experienced mild rejection episodes early in their post-operative course that have, in general, been easily reversed with steroids. Long term renal function has remained good. It is likely that the optimal use of this agent is yet to be defined.

Abstract# 224 **Poster Board #-Session: P37-I**

LONG-TERM CYCLOSPORINE IMMUNOSUPPRESSION: IS THERE NEPHROTOXICITY IN KIDNEY TRANSPLANT RECIPIENTS?

Steven Paraskevas,[1] Roger Denny,[1] Thiagarajan Ramcharan,[1] Kristen Gillingham,[1] Raja Kandaswamy,[1] Abhinav Humar,[1] Arthur Matas.[1] *[1]Surgery, University of Minnesota, Minneapolis, MN.*

Calcineurin inhibitors (CI) have been associated with significant side effects including hypertension, cosmetic changes, hyperlipidemia, and nephrotoxicity. ESRD has developed in up to 10% extrarenal tx recips after 5-10 yrs of CI immunosuppression. Yet, it is unclear if similar ESRD occurs after kidney transplantation. We studied serum Cr levels vs. time in 609 primary adult (technically successful) kidney only tx recips (1/84-12/89). Maintenance immunosuppression was CSA/AZA/P. CSA levels were maintained at 75-100 mg/ml after 1 year (by HPLC). Long-term recips have only occasional level monitoring. Study A - of the 609, 323 recipients never had an AR episode. Serum Cr level from 1-15 yrs is shown in the Table. There is no significant change in serum Cr vs. time. Graft loss in this cohort was due to: CR(n=5), DC meds (n=3), recurrent disease (n=5), CSA toxicity (n=1), death (n=103), other(n=10).

Study B - 407 recips had function ≥4 yrs. Graft loss in those with <4 yrs function was due to: AR(n=20), CR (n=34), DC meds (n=8), death 9 (n=52), other (n=13). The Cr level from 1-15 yrs (for those with ≥4 yr function) is shown in the table. There is no significant change in Cr level vs. time. Graft loss after 4 yrs was due to: AR (n=3), CR (n=39), DC meds (n=11), recurrent disease (n=10), death (n=95), unknown (n=2), other (n=16). We conclude that, in kidney transplant recipients with 15 yr follow-up, there is no evidence of significant CI-related deterioration of renal function.

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
A-Mean Cr	1.5	1.4	1.4	1.4	1.4	1.4	1.3	1.3	1.4	1.4	1.4	1.4	1.4	1.5	1.6
±SD	.4	.4	.4	.4	.5	.5	.4	.4	.5	.6	.5	.5	.7	1.3	.8
B-Mean Cr	1.7	1.7	1.5	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.5	1.5
±SD	.7	.6	.7	.4	.4	.4	.4	.4	.5	.5	.6	.5	.6	1.1	.7

Abstract# 225 **Poster Board #-Session: P38-I**

CORRELATION BETWEEN CYCLOSPORINE (CsA) PHARMACOKINETIC PARAMETERS AND ACUTE HISTOLOGICAL FINDINGS IN SURVEILLANCE BIOPSIES.

Azemi A. Barama, James Gough, Rachel McKenna, Mauricio Monroy, Serdar Yilmaz, Gerard Murphy, Farshad Sepandj, Halgrimur Benediktsson.

Introduction: Recent reports in therapeutic drug monitoring indicate that pharmacokinetic studies are useful in optimizing immunosuppressive effect of CsA early after kidney transplantation. The impact of this approach on long-term graft function however is not clear Histological changes, found in surveillance biopsies done 6 mo. after transplantation, are a good surrogate for long-term graft function. We explored the potential relationship between CsA pharmacokinetic parameters & the presence of clinically unsuspected acute rejection (sub-clinical rejection -ScRj). Methods: 88 kidney transplant recipients on Neoral, Cellcept & prednisone maintenance therapy were reviewed. Neoral dose was adjusted based on trough level (C0). Clinical acute rejections (AR) between Transplant and biopsy(Bx)-time were recorded. ScRj was defined,using the Banff schema, as tubulitis with marked interstitial infiltrate (>1i+t1) in protocol biopsies. Pharmacokinetic parameters examined included: C0(ug/L), & dose/weight corrected C0 (DwcC0)(ug/L/mg/kg) at 1,3 mo & Bx-time, & C2 (2hrs post dose level) (ug/L),& DwcC2 (ug/L/mg/kg)at Bx-time. Results: ScRj was diagnosed in 31% of patients with AR & 18% in patients without AR. The AR-/ScRj+ patients had significantly higher C2 than the AR-/ScRj+ patients($p<0.05$). The AR+/ScRj+ patients had higher C2 but lower DwcC0 levels than AR+/ScRj- patients at each time point($p<0.05$)& Lower DwcC2. Conclusion: These data suggest that patients with C2 levels lower than 1000ug/L are at increased risk for ScRj & that optimal C2 levels for rejection free regimen are between 1100 & 1200 ug/L at 6-9 mo post-transplant. DwcC0 as an indicator of absorption efficiency for a given patient, may aid in the identification of poor absorber of Neoral who require appropriate dose adjustment or alternative strategies to provide effective immunosuppression.

Group (n)	DwcC0-3mo	DwcC0-Bx	C0-Bx	C2-Bx
AR-/ScRj-	124+/-54	142+/-72	287+/-65	1187+/-337*
AR-/ScRj+	136+/-61	146+/-4	256+/-58	1002+/-239*
AR+/ScRj-	124+/-41	164+/-60*	291+/-58	1245+/-275
AR+/ScRj+	92+/-33	95+/-27*	241+/-84	1467+/-413
p	0.05	0.001	ns	<0.05

Abstract# 226 **Poster Board #-Session: P39-I**

LOW INCIDENCE OF ADVERSE EVENTS IN A PILOT STUDY IN KIDNEY TRANSPLANTS USING A STEROID FREE, CALCINEURIN SPARING, MYCOPHENOLATE & SIROLIMUS BASED REGIMEN.

C. Marsh,[1] R. Wilburn,[2] L. Wrenshall,[1] R. Stevens,[1] C. Davis.[3] *[1]Surgery, University of Washington, Seattle, WA, [2]Medicine, Virginia Mason Medical Center, Seattle, WA; [3]Medicine, University of Washington, Seattle, WA.*

A unique steroid free, low dose mycophenolate (MMF) & sirolimus based regimen, avoiding calcineurins (in the first 2 weeks) was tested in kidney transplants alone (and is applicable to kidney/islet transplantation). Induction therapy (ATG 1.5 mg/kg x 4 doses or Zenepax® 1 mg/kg x 5 doses), sirolimus (2 mg/day), & MMF (1 gm/day) are instituted on day 0 TNFR:Fc (Enbrel®) 25 mg. is given SQ on day 0, 4, & 8. Low dose cyclosporine (CsA) or tacrolimus is started on POD 14 and adjusted to "low" trough levels (CsA~50 ng/dl or tacrolimus~5 ng/dl). Protocol biopsies were obtained on POD 30. 13 pts. (11 Caucasians, 2 Asians) were enrolled (age, 43+/-8.3), with primary renal disease diagnosis of PCKD (3), IGA (2), IDDM (3), Congenital (2), HTN (1), FSGS (1), & Unknown (1). 8 patients received induction with ATG. 3 patients had significant delayed graft function

Male/Female 7/6	FK level Day 30	8 3±8 2	Hct Day 30	28 1±5 9
CAD/LRD 9/4	Sirolimus level Day 30	10 3±7 5	Plt # Day 30	261±137
Cr Day 30 2 3±3 2	MPA level Day 30	1 8±1 8	WBC Day 30	4 3±4 04

One mild rejection was found on a protocol biopsy and treated with a pulse of methylprednisolone. The patient is now off steroids. Adverse events consisted of leukopenia (5), 3 requiring the temporary withholding of MMF, anemia (3), oral ulcer (1), mild triglyceridemia (4), and 4 infections (yeast UTI, CMV pneumonia, wound infection, CMV hepatitis). Interestingly, many patients have been below target trough levels for sirolimus & mycophenolic acid, nevertheless, there has not been a "clinical" rejection.

A steroid free, calcineurin sparing regimen based on MMF and sirolimus can be employed without significant adverse events or rejections. Enrollment & follow up continues.

Abstract# 227 **Poster Board #-Session: P40-I**

A PROSPECTIVE MULTICENTER STUDY DEMONSTRATES SAFETY AND EFFICACY OF PERIPHERAL VEIN ADMINISTRATION OF THYMOGLOBULIN FOR INDUCTION IMMUNOSUPPRESSION.

Robert Steiner,[1] Douglas Norman,[2] David Cohen.[3] *[1]UC San Diego, San Diego, CA; [2]Oregon Health Science University, OR; [3]New York Presbyterian Hospital.*

Current practice for the administration of polyclonal antibodies has been via a high flow or central vein to avoid local irritation or inflammation. However, local symptoms at the site of peripheral vein (PV) infusion of medications or fluid are common, affecting 25-70% of all hospitalized patients In order to assess the safety and efficacy of PV administration of Thymoglobulin (Thymo), a rabbit, polyclonal anti-thymocyte globulin, a prospective study was performed at 3 centers with a minimum patient follow-up of 6 months using standard maintenance immunosuppression. Thymo 1.5mg/kg IV was mixed with 1000u of heparin, and 20mg of hydrocortisone in 500cc of standard diluent solution (0.9% saline or 0.45% saline were preferred diluents, as heparin, hydrocortisone and D5W were noted to occasionally precipitate) administered at 83cc/hr to 125cc/hr for 5-7 doses through a 22 micron filter via PV (16-22 gauge) catheters (no PICC lines were used). 37 patients enrolled in the study (median age 45y±10; 64% males, 36% females, 58% CAD, 42% LRD) 3 transplants were technical failures and were subsequently excluded from the analysis. **Results:** The per patient infusion related local PV incidents were closely examined including redness (21%), pain (30%), swelling (30%), clotting during infusion (6%) and need for PV catheter change (2.8, including elective changes). Only 3 patients (1 elective, 2 after PV infiltration) required 1 dose administered via central line to complete the desired course of drug. T-cell subsets (median numbers) were obtained on 22 patients, showing effective T-cell depletion.

	Baseline	Day 10	Day 30
CD4 cells (#/µl)	45	6	19
CD8 cells (#/µl)	29	7	19

7 biopsies were obtained for renal dysfunction (1 Normal, 2 Borderline, 2 Banff I, 1 Banff II, 1 CYA toxicity). One patient died of infection 84 days post transplant **Conclusions:** 1) Nearly all patients completed their entire Thymo course via PV infusion 2) Local PV infusion related side effects are similar to that seen with other IV fluids 3) Effective T-cell depletion was observed with low rejection and excellent graft survival. Results in our series of patients show that Thymo can be administered safely, conveniently and economically by peripheral vein infusion.

Abstract# 228 **Poster Board #-Session: P41-I**

LONG TERM BENEFITS AND SIDE EFFECTS OF CICLOSPORIN (CYA) TO MYCOFENOLATE MOFETIL (MMF) CONVERSION IN RENAL TRANSPLANT PATIENTS.

H. François,[1] M. Ammor,[1] R. Djeffal,[1] V. Paradis,[1] F. Kriaa,[1] A. Durrbach,[1] B. Charpentier.[1] *[1]94270, Bicetre Hospital, Le Kremlin Bicetre, France.*

Background . To reduce renal toxicity of CyA, the conversion from CyA to MMF has been proposed recently. Nevertheless, long-term side effects and renal outcomes have not been reported yet.

Aim : To evaluate benefits and adverse effects of CyA to MMF switch.

Methods : A comparative and retrospective analysis of 40 renal transplant patients disclosing CyA renal toxicity (n=30), chronic rejection (n=4), symptomatic hyperuricemia requiring allopurinol (n=5) or azathioprine induced hepatitis (n=1). Their treatment was switched step-wisely from CyA (25mg per month) to MMF (2g per day)(group 1). They were compared to 40 renal transplanted patients (group 2) matched for their date of birth (+/- 5 years), their transplant follow up before and after the switch, and their initial immunosuppressive treatment.

Results : The mean follow up of both group was similar before the switch 72 +/- 41 months and after the switch 27+/-12. The mean creatinemia was similar in both groups. In group 1, it decreased from 191mumol/l before the switch to 180mumol/l at 3 months ($p=0.01$) and 154 mumol/l at 2 years ($p<0.01$), whereas it remained unchanged in group 2. The systolic blood pressure decreased from 153mmHg before the switch to 143 and 140 at 6 ($p<0.01$) and 24 months ($p<0.005$) respectively. The lipid profile were not significantly improved after the switch. None of the patient developed acute rejection after the switch Three grafts were lost because of chronic rejection in the first group compared to five (ns) in the second group. Thirteen patients (32.5%) of the group 1 developed a severe disease (life-threatening infection (n=5, 12.5%), malignancy (n=5, 12.5%) including lymphoma (n=3, 7.5%) or severe MMF side effects (n=3; 7.5%)) In contrast, only 3 patients in the second group had similar events (7.5%) ($p<0,01$). In addition, 55% patients had mild side effects of MMF (diarrhea, myelotoxicity, non life-threatening infection or benign tumors) requiring a reduction of MMF for 47 5% of patients:(15% had a 25% reduction of the MMF treatment and 32.5% a reduction of more than 50%).

Conclusions : In renal transplant recipients, the CyA to MMF conversion seems to minimally improve renal function but is associated with a high rate of malignancies and life-threatening infections. Other regimens of CyA to MMF conversion should be evaluated to avoid effects of the over-immunosuppression.

Abstract# 229 **Poster Board #-Session: P42-I**

CYCLOSPORIN-A BLOOD CONCENTRATION AT 2 HOURS IS THE BEST PARAMETER TO CALCULATE AREA UNDER THE TIME-CONCENTRATION CURVE (0 T0 4 HOURS).

Elias David-Neto,[1] Zita M.L. Britto,[1] Cristiane F. Alves,[1] Francine C. Lemos,[1] William C. Nahas,[1] Luis E. Ianhez.[1] *[1]Renal Transplantation Unit, University of Sao Paulo, Sao Paulo, Sao Paulo, Brazil.*

In a previous study, we have demonstrated that CyA-AUC0-4 0 to 4 hours after the oral Neoral dose represents the best parameter to avoid acute cellular rejection. Patients with a AUC0-4 > 4400-5500ng.ml/h have the least chance for rejection and CyA nephrotoxicity. However, AUC0-4 is not feasible in clinical practice and therefore there is a need for another parameter to estimate it. We evaluated which parameters of the complete CyA-pharmacokinetics (PK) better correlates with the CyA-AUC0-4. 73 adult (>18y) patients performed 259 full AUC0-4 with CyA blood levels obtained at 0, 1,2 and 4 hours after an oral Neoral dose (5.18±2.54mg/k/d). All of these studies were performed at least after 14 days (35±17) following renal Tx,in order to allow CyA metabolism to stabilize Average C0, C1, C2, C4 and trapezoidal AUC0-4 were

250±122, 964±614, 1044±501, 644±327ng/ml and 3301±1468ng.ml/h, respectively. In these 259 curves, Cmax (1220±584ng/ml) occurred at C1 in 43%, at C2 in 47% and at C4 in 10% of the curves. By multiple linear regression analysis, C2 wshowed to be the best predictor of the AUC0-4 (R=0.93, p<0.001) where: AUC0-4 = 451+(2.729xC2). Trough level (C0) had only a moderate correlation to the AUC0-4 (R= 0.72). To certify whether the equation predicts correctly the AUC0-4 ,we then tested this equation in another 33 adult patients who had not participated in its development. In these patients, calculated AUC (AUC0-4calc) correlated extremely well with trapezoidal AUC0-4 (R=0.90,p<0.001) and all values were within the 95% prediction interval. In only 3 (9%) times of these 33 instances a wrong decision would have been taken considering AUC0-4calc only. In 1 instance it would be slightly (+82 ng.ml.h) increased, in 1 case it would have been unnecessarily decreased by -100 ng.ml/h and in the other it would be maintained when it had to be increased by +133 ng/ml/hr. All small variations of the target values. We have demonstrated, in this very large CyA-Pk study, that C2 can be safely used to estimate AUC0-4calc and that calculated AUC0-4 is a true estimate of the trapezoidal AUC0-4. A C2 between 1500 and 1800 ng/ml is within the Cya therapeutic range. However, this equation is valid only after 14 days after transplant. During the first 14 days, a complete AUC0-4 should be performed.

Abstract# 230 Poster Board #-Session: P43-I

EFFECT OF LIVING RELATED DONOR BONE MARROW INFUSION ON CHIMERISM IN KIDNEY TRANSPLANT PATIENTS. Gaetano Ciancio,[1] Joshua Miller,[1] Rolando Garcia-Morales,[1] George W. Burke,[1] Camillo Ricordi,[1] Andreas Tzakis,[1] Violet Esquenazi.[1] *[1]Surgery/Division of Transplantation, University of Miami, Miami, FL.*

We have recently demonstrated, in 5.1/2 year follow-up, the clinical efficacy of donor bone marrow infusions (DBMC) in preventing long-term cadaver renal allograft (CAD) losses, primarily due to the prevention of chronic rejection. In the present study, between November of 1996 and May of 2000, 110 living related donor (LRD) kidney transplants were performed of which 45 received donor iliac crest marrow ($1.8X10^8$± $7.5X10^7$ cells/kg body weight ±SD) in a single infusion 4 days postoperatively. OKT3 (n=24) and Zenapax, (n=21) were used for induction, with maintenance tacrolimus, mycophenolate and methylprednisolone (MP) immunosuppression. Follow-up has ranged from 6 months to 4 years. In entire group of the 110 patients, there were 2 mortalities (unrelated to rejection) and no graft losses to date. In the (more long-term) follow-up of CAD DBMC recipients, donor mononuclear cell chimerism in recipient iliac crest marrow measured by PCR-flow followed yearly is averaging 1.3% at 4 years, i.e., chimerism has tripled from 1 year postoperatively. In the LRD DBMC group, chimerism levels in the peripheral blood were 2 fold higher at 1 and 2 years postoperatively than were seen in the CAD group at these intervals.By 2 years postoperatively, in the LRD DBMC group iliac crest chimerism levels have reached 0.85% i.e., an increase approaching that seen in CAD DBMC recipients at the 2 year interval who were infused with 4 times the number of DBMC (and with the virtual absence of acute rejection episodes in the LRD group). In this (newer) LRD group, dosaging, thus far, of tacrolimus, mycophenolate and MP at 1 and 2 years is no different between the DBMC and control patients, nor are serum creatinine concentrations different at these time intervals. Conclusion: Although DBMC infusion has not yet been shown to be more clinically efficacious in this LRD group, there is certainly reason to believe that it is safe. Also, since the growth of chimerism is accelerated over that seen in the CAD DBMC group, long-term survival is expected to be improved over the controls (as in the CAD group only found after 5 years). This should allow (monitored) withdrawal immunosuppression to be performed with more certainty (now underway in CAD recipients).

Abstract# 231 Poster Board #-Session: P44-I

EFFICACY AND SAFETY OF DACLIZUMAB INDUCTION FOR PRIMARY KIDNEY TRANSPLANT RECIPIENTS IN COMBINATION WITH TACROLIMUS, MYCOPHENOLATE MOFETIL AND STEROIDS AS MAINTENANCE IMMUNOSUPPRESSION. Gaetano Ciancio,[1] George W. Burke,[1] Audrey Miller,[1] Kiliana Suzart,[1] Jose Figueiro,[1] Anne Rosen,[1] David Roth,[1] Warren Kupin,[1] Joshua Miller.[1] *[1]Surgery/Division of Transplantation, University of Miami, Miami, FL.*

The efficacy of IL2-receptor blockers in lowering the incidence of early acute rejection (AR) in cyclosporine-treated kidney recipients is well known. We evaluated the efficacy and safety of Daclizumab (DAC) as induction therapy in combination with tacrolimus (FK) and mycophenolate mofetil (MMF). From March 1998 to November 2000, we analyzed 241 primary kidney transplant recipients (Adult=226, Pediatric=15) who received tacrolimus, MMF, methylprednisolone and DAC induction. DAC (1mg/kg) was given on the day of surgery, and every other week for a total of 5 doses. MMF was started on day 1 (1 gm bid) and adjusted for side effects and WBC count. FK was withheld until the serum creatinine was < 4 mg/dl. Mean recipient age was 44.5 years (range 3-72). Among patients with a mean follow-up of 18.2 months (15-999 days), there have been 4 early (< 6 months) biopsy-proven AR episodes (1.6%) and 9 late (>6 months) biopsy-proven AR episodes (3.7%). Few complications have occurred. Specifically, there were no lymphoproliferative disorders, 1 Kaposi's sarcoma, 2 tissue-invasive CMV infections, 2 symptomatic CMV viremias, 3 UTI, 2 pneumonias and 1 worsening of hepatitis C. During the period of study, 10 patients lost their grafts including 5 deaths with functioning graft (1 myocardial infarct, 2 pneumonia, 1 sepsis and 1 liver failure) and 5 patients returned to dialysis (rejection related graft lost). The latest mean serum creatinine was 1.4±0.9 mg/dl. The mean hospital length of stay (LOS) was 11.5±6.1 days (range 5-58), which was significantly shorter than when compared to 15.6±9.8 days (range 6-122) of an immediate historical control group of 219 primary kidney recipients who received FK, MMF, steroids and OKT3 (5mg/day x 10-14 days) (p=0.0002). The actuarial 2.5-year patient and graft survival was 97.7% and 95.6% respectively. In summary, the combination of FK, MMF, steroids and DAC is safe (no side effects) and effective for primary kidney recipients, resulting in a low incidence of early and late acute rejection and shortening of the initial hospitalization when compared to our previous protocol.

Abstract# 232 Poster Board #-Session: P45-I

A COMPARISON OF FIBROGENIC GENE mRNA LEVELS IN RENAL TRANSPLANT BIOPSIES TAKEN FROM PATIENTS ON A RANDOMISED TRIAL OF AZATHIOPRIN VERSUS MYCOPHENOLATE MOFETIL. Gareth R. Bicknell, Sunjay Jain, Michael L. Nicholson.

Background: We have previously shown that glomerular expression of collagen III and tissue inhibitor of metalloproteinases (TIMP)-1 mRNA is greater in cyclosporin-treated transplant patients than in tacrolimus-treated patients. In this study, we investigate the mRNA levels in glomerular and interstitial tissue taken from patients taking part in a randomised trial of azathioprin versus mycophenolate mofetil (MMF).

Methods: Patients with cadaveric and asystolic renal transplants were given a 40% reduced dose of micro-emulsion cyclosporin and randomised either to MMF (1 g bd; n = 15) or azathioprin (75 mg od; n = 15). Tissue core transplant biopsies were performed before entry into the trial and at 6 months after entry. Glomerular and interstitial samples were isolated, and the mRNA was extracted using oligo-dT paramagnetic beads. Complementary DNA was synthesised, amplified and detected by RT-PCR and an enzyme-linked immunosorbent assay. Correction for cellularity was made by comparison with the housekeeping gene, glyceraldehyde phosphate dehydrogenase. Samples with genomic DNA contamination were not used.

Results: Glomeruli from all patients exhibited a general decrease over time in the expression of TIMP-1 ($P < 0.001$), inducible nitric oxide synthase (iNOS; $P = 0.020$), and possibly TIMP-2 ($P = 0.084$) mRNA. The decrease in TIMP-1 and iNOS mRNA was primarily due to patients on azathioprin (TIMP-1, $P < 0.001$; iNOS, $P = 0.067$). However, in spite of randomisation, azathioprin patients entered the trial with higher TIMP-1 ($P = 0.048$). Similarly, they entered with a lower level of transforming growth factor (TGF)-β mRNA ($P = 0.049$). At 6 months TGFβ was still lower in these patients ($P = 0.048$), even though their levels had risen since entry ($P = 0.005$). The expression of collagen III mRNA was not detected in a sufficient number of samples to warrant statistical analysis. Similarly, in the interstitial samples, collagen III, TGFβ, and TIMP-1 were not detected sufficiently to warrant statistical analysis. The expression of TIMP-2 and iNOS mRNA was not significantly different between drug therapies.

Conclusions: TIMP-1 and iNOS mRNA levels were likely due to cyclosporin dose reduction. Increased TGFβ mRNA expression indicated a potential for future rejection, although TGF mRNA levels do not necessarily equate with active TGF protein. The results agree with those of the parallel clinical study, but the long-term impact of the different immunosuppressive therapies has yet to be assessed.

Abstract# 233 **Poster Board #-Session: P46-I**

DACLIZUMAB AND MYCOPHENOLATE MOFETIL REDUCE THE NEED FOR CYCLOSPORINE WITHOUT INCREASING RISK FOR ACUTE REJECTION IN RENAL TRANSPLANTATION. Gordon R. Ingle,[1] Asha Moudgil,[1] Ashley Vo,[1] Stanley C. Jordan.[1] [1]*Renal Transplantation and Transplant Immunology, Cedars-Sinai Medical Center, Los Angeles, CA.*

PURPOSE: Cyclosporine (CSA) adverse effects (AEs) can compromise outcomes in renal transplant (RTx). This prospective, pilot study assessed the efficacy and safety of daclizumab (DAC), mycophenolate mofetil (MMF), corticosteroids (CS), and reduced dosed CSA in RTx. METHODS: 30 patients at low risk for acute rejection (AR) (first RTx, PRA<40%, non-black) were evaluated. 15 patients (12 cadaveric, 2 LR, 1 LUR) were prospectively enrolled to receive DAC 1mg/kg/dose every 14 days for 5 doses, MMF 1gm BID, CS per routine protocol and half the usual CSA (Neoral) dose at our institution. Endpoints included incidence of AR, mean serum creatinine (SCr), AE profile, infectious complications, and incidence of death and/or graft failure. A control group of 15 patients were matched for age, gender, race, type of RTx, mean antigen mismatch, and cold ischemia time. Follow-up was one year. RESULTS: Study and control groups were similar at baseline except mean PRA was significantly lower in the control group (0.13% vs 5.2%, p=0.01).

Endpoint	Study Group	Control Group	p-value
Mean CSA level @ 1, 6, and 12 months	202, 198, 187	320, 302, 303	<0.0001 at all points
AR at 1 year	0	0	1.0
Mean SCr @ 1, 6, and 12 months	1.47, 1.25, 1.24	1.65, 1.36, 1.3	0.29, 0.35, 0.63
Number of Infections	19	29	0.39
Patients requiring a biopsy/FNA	20%	53%	0.13
Patients experiencing a CSA-associated AE	47%	100%	0.0022

Controlling for PRA differences did not alter the above outcome variables. The need for FNAs/renal biopsies was reduced by 63%. Patient and graft survival were 93% in the treatment group versus 100% in the control group (p=0.30). One patient died of a MI with a functioning graft. CONCLUSIONS: For patients at low risk of AR, DAC induction along with MMF allows for significant reduction in CSA concentrations without increasing risk of AR. This regimen was also better tolerated with significantly fewer CSA-associated AEs in the study group. Clinical significance with positive statistical trends were noted in the need for renal biopsies (and the costs and complications associated with this procedure) and infection rates. Multicenter studies with more patients to enhance statistical power and to support the findings in this pilot study are warranted.

Abstract# 234 **Poster Board #-Session: P47-I**

WHICH PATIENTS BENEFIT FROM CYCLOSPORINE WITHDRAWAL FOLLOWED BY SIROLIMUS (RAPAMUNE®) MAINTENANCE THERAPY? Henri Kreis,[1] José M. Morales, Peter Morris, Antonio Henriques, Pierre Daloze, Giuseppe Segolini, Uwe Heemann, Eric Nègre, the Sirolimus Tri-continental Renal Transplant Study Group. [1]*Service de Réanimation et de Transplantation, Hôpital Necker, Paris, France.*

Purpose. The present paper examines which renal transplant recipients, based on renal function, benefit from cyclosporine (CsA) withdrawal after 3 months of a sirolimus (SRL)-CsA-steroids regimen.

Methods. This was an open-label study conducted in 57 centers in Europe, Australia, and Canada. A total of 525 renal allograft recipients received SRL 2 mg (tablets), CsA, and steroids when enrolled into the study. SRL blood levels were to be maintained above 5 ng/mL (immunoassay). At 3 months ± 2 weeks, eligible patients (n=430) were randomized to remain on triple-therapy (SRL-CsA-steroids, n=215), or to have CsA withdrawn over 6 weeks (SRL-steroids, n=215) and initiate concentration-controlled SRL therapy.

Results. At 1 year post-transplant, renal function was significantly better for the SRL-steroids patients versus the SRL-CsA-steroids patients as measured by mean creatinine (159 vs 142 µmol/L, p<0.001) and calculated GFR (56 vs 62 ml/min, p<0.001). As seen in the table, significantly more patients in the SRL-steroids group had an improvement in renal function (p<0.001, Cochran-Mantel-Haenszel test).

% of Patients with Improvement or Decrease in Creatinine or Calculated GFR

Group	Renal function	>10% improvement	0-10% improvement	0-10% decrease	>10% decrease
SRL-CsA-steroids	Creatinine	22.8	16.8	19.0	41.3
	Calculated GFR	20.5	19.9	21.0	38.6
SRL-steroids	Creatinine	47.2	17.2	17.2	18.4
	Calculated GFR	51.3	20.9	10.8	17.9

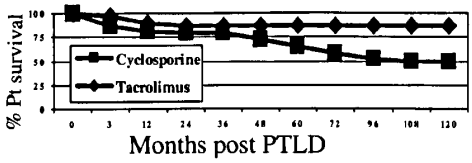
In addition, mean creatinine difference between the 2 groups at 12 months was analyzed by quartiles according to their creatinine values at 3 months (baseline before randomization). For all quartiles the renal function was better in the SRL-steroid patients. This difference was significant for the first 3 quartiles (<115, [115; 146]; [146-176] mmol/L; p<0.006).

Conclusions. This study has shown that 1) 70% of patients will have an improvement in their renal function when CsA is eliminated in favor of SRL maintenance therapy; 2) renal function is significantly better for those patients who eliminate CsA; and 3) all patients potentially benefit, irrespective of their serum creatinine at 3 months.

Abstract# 235 **Poster Board #-Session: P48-I**

COMPARISON OF POST TRANSPLANT LYMPHO PROLIFERATIVE DISORDERS (PTLD) IN CHILDREN UNDER CYCLOSPORINE AND TACROLIMUS, 766 CONSECUTIVE RECEIPIENTS: 15 YEARS EXPERIENCE. Ashok B. Jain,[1] George Mazariegos,[1] Randeep S. Kashyap,[1] Cataldo Doria,[1] Mike Nalesnik,[1] Jorge Reyes.[1]

PTLD has remained a major concern after liver transplantation (LTx) particularly in children. Aim : To examine the rate of PTLD, site of PTLD, interval after LTx to PTLD development and survival after PTLD and CyA and Tacrolimus. Material: 766 children (age < 18 years 386 boys & 380 girls) received LTx between 1981 and 1996. 483 children commenced on Cyclosporine (CsA) and 283 on Tacrolimus. They all were followed until Nov.1999. The mean follow up for CsA was 156 ± 25 months and for tacrolimus was 79 ± 22 months. Results: 48 (9.9%) children under CsA and 38 (13.5%) children under tacrolimus developed PTLD 33.3 ± 30 (range 0.66 - 107) and 8±11 (range 1.6 - 30.8) months after LTx respectively. The difference in rate of PTLD was not significant (P=0.14). The site of PTLD were lymph node, gastointestinal, liver, lung, multiple adenoid and tonsils, miscellaneous and unknown. The frequency under CsA was 12, 3, 3, 3, 3, 0, and 16 (total 48; 4.9%) and that under tacrolimus was16, 7, 6, 0, 1, 2, 2, 2 and 49 (total 38; 73.5%) respectively. Kaplan Meier actuarial percentage of survival in months post PTLD is shown in the figure.



Post PTLD survival under tacrolimus was significantly better compared to CsA (P= 0.0013) Conclusion: Although the rate of PTLD after LTx in children under tacrolimus was not statistically higher for CsA, actuarial survival was better in the tacrolimus group as compared to CsA group This may reflect in part the improvement in PTLD diagnosis and treatment

Abstract# 236 **Poster Board #-Session: P49-I**

ANALYSIS OF HYPERLIPIDEMIA IN CHILDREN WITH KIDNEY TRANSPLANTS. Maria Hardstedt, Kristen Gillingham, Blanche M. Chavers. *Depts. of Pediatrics and Surgery, University of Minnesota, Minneapolis, MN.*

Hyperlipidemia is a known risk factor for atherosclerotic cardiovascular disease in adult kidney transplant (Tx) recipients. **Purpose:** The aim of this study was to evaluate the incidence of hyperlipidemia and associated variables in pediatric kidney Tx recipients at a single center. **Methods:** We studied 64 children transplanted between January 1991 and December 1993 (38 M, 26 F, 52 living donor, 12 cadaver; 55 primary, 9 reTx; 54 Caucasian, 10 non-Caucasian). The mean age at Tx was 8 ± 6 yrs (X±SD, range 0.7 to 17 yrs) Immunosuppression included prednisone, azathioprine, cyclosporine (CsA), and antibody therapy. Total cholesterol (TC) and triglyceride (TG) levels were measured preTx and at 1, and 5 yrs postTx.

Results: The mean preTx TC level was 193 mg/dl (range 85-415 mg/dl). Elevated preTx TC levels (>200 mg/dl) were present in 22 patients (34%) and were associated with Black race (p=0.014), primary Tx (p=0.04), and preTx peritoneal dialysis (p=0.0001) The mean preTx TG level was 332 mg/dl (range 46-2400 mg/dl). Elevated preTx TG levels (>200 mg/dl) were present in 39 patients (64%) and were associated with Tx age <6 yrs (p=0.0001) and preTx dialysis (p=0.04).

Hypercholesterolemia persisted in 10/18 (56%) patients at 1-yr postTx; hypertriglyceridemia persisted in 8/27 (31%). At 5-yrs postTx, TC levels were elevated in 19/38 (50%) patients measured and were associated with an increased incidence of acute rejection (79% vs 47%, p=0.03) and a higher mean CsA level at 1-yr (99.7 vs 73.1 ng/ml, p=0.05). TG levels were elevated in 7/26 (27%) patients measured and were associated with female sex (p=0.03), chronic rejection (43% vs 16%, p=0.02), decreased graft survival (83% vs 100%, p=0.02) and elevated serum creatinine (2.0 vs 1.1 mg/dl, p=0.02). Hyperlipidemia at 5-yrs postTx was not associated with Tx age, donor source, Tx number, race, primary disease, or steroid dosage.

Conclusions: We conclude that hyperlipidemia is present pre and postTx in a significant percentage of children with end-stage kidney disease. It persists at least 5 yrs postTx, predisposing these children to atherosclerosis in later life. The identification and treatment of hyperlipidemia should be included in pediatric kidney Tx protocols.

Abstract# 237 **Poster Board #-Session: P50-I**

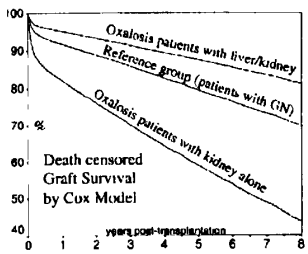
SUPERIOR DEATH-CENSORED RENAL ALLOGRAFT SURVIVAL IN OXALOSIS PATIENTS WITH A LIVER TRANSPLANT. Diane M. Cibrik,[1] Bruce Kaplan,[1] Julie A. Arndorfer,[1] Akinlolu Ojo,[1] Alan B. Leichtman,[1] Herwig-Ulf Meier-Kriesche.[1] *[1]Internal Medicine, University of Michigan, Ann Arbor, MI.*

Previous studies have suggested that patients with oxalosis have poor renal allograft survival. To evaluate renal allograft survival for oxalosis patients with or without a liver transplant as compared to patients with other renal diseases, we analyzed 20,759 renal transplant recipients registered in the USRDS database between 1988 and 1997. Of these patients, 105 patients had oxalosis as their primary diagnosis as compared to 20,654 patients with glomerulonephritis (GN). Among the 105 patients with oxalosis, 35 patients had a liver transplant followed by a kidney transplant (LKTx) and 70 patients had a kidney transplant alone (KTA).

Methods: A Cox proportional hazard model was used to estimate the adjusted death-censored graft survival among oxalosis patients who received a KTA or a LKTx. The Cox model was adjusted for 16 covariates relevant to death-censored graft survival. Patients with GN were the designated reference group.

Results: Oxalosis patients receiving a KTA had a significantly worse adjusted death-censored graft survival (44%) compared to patients with GN (70%) at 8 years post-transplantation ($p<0.001$). In contrast, oxalosis patients who received a LKTx had the significantly higher death-censored graft survival (81%) as compared to oxalosis patients who received a KTA (44%, $p<0.001$) but not statistically different from patients with GN. Patient survival for the three groups was not significantly different.

Conclusion: Patients with oxalosis that receive a LKTx have superior death-censored graft survival as compared to oxalosis patients that receive a KTA and equivalent death-censored graft survival as compared to patients with GN.



Abstract# 238 **Poster Board #-Session: P51-I**

EFFECTIVENESS OF TACROLIMUS IN PREVENTING THE RECURRENCE OF IgA NEPHROPATHY AFTER RENAL TRANSPLANTATION. Yoshihiko Watanabe,[1] Kazunari Tanabe,[2] Tadahiko Tokumoto,[2] Hiroaki Shimmura,[2] Hiroshi Nihei,[1] Hiroshi Toma.[2] *[1]Department of Medicine, Kidney Center, Tokyo Womens Medical University, Tokyo; [2]Department of Urology, Kidney Center, Tokyo Womens Medical University, Tokyo, Japan.*

INTRODUCTION: Our recent review showed that the recurrence of the original renal disease, such as IgA nephropathy (IgAN) is the most important cause of graft loss after renal transplantation under cyclosporine (CyA) immunosuppression. Since IgAN is one of the leading causes of chronic renal failure in Japan, prevention of the recurrence of IgAN after renal transplantation plays an important role in improving long-term renal allograft survival. Tacrolimus (TAC) is a newly developed potent immunosuppressive drug which is reported to be effective in treating glomerulonephritis. We reviewed our data to certain whether TAC is more useful than CyA in preventing the recurrence of IgAN after renal transplantation.

MATERIALS AND METHODS: Between 1983 and 1999, 1360 patients underwent renal transplantation at our institute. In 71 patients their original renal disease was confirmed to be IgA nephropathy by renal biopsy. Of these patients 52 were treated with CyA (CyA group) and 19 were treated with tacrolimus (TAC group). Thirty-five patients had undergone living renal transplantation and 25 cadaveric renal transplantation. Graft biopsy was performed whenever a patient showed graft dysfunction, such as proteinuria, elevation of serum creatinine levels, or hematuria.

RESULTS: No significant difference was noted in patient survival between the two groups. Graft survival in the CyA group was 96.2%, 65.3%, and 35.5% at 1, 5, and 10 years, respectively after renal transplantation. In contrast, in the TAC group, it was 94% and 94% at 1 and 4 years, respectively after renal transplantation. Since TAC was first used clinically in 1996 in Japan, we examined the incidence of recurrence of IgAN and graft loss due to IgAN within 4 years after renal transplantation. Recurrence of IgAN within 4 years was noted in 11 patients (20%) in the CyA group and in only 2 patients (10%) in the TAC group. Although graft loss due to recurring IgAN within 4 years was noted in 4 patients (9%) in the CyA group, no recipients treated with TAC (TAC group) lost their graft due to recurring IgAN

CONCLUSION: The incidence of recurring IgAN and graft loss due to recurring IgAN were much more frequent in the CyA group than in the TAC group. TAC seemed to be much more effective than CyA in preventing the recurrence of IgAN after renal transplantaion.

Abstract# 239 **Poster Board #-Session: P52-I**

CYCLOSPORINE PHARMACOKINETICS UNALTERED BY BASILIXIMAB IN PEDIATRIC RENAL TRANSPLANT RECIPIENTS. J. M. Kovarik,[1] L. Chodoff,[1] A. Korn.[1] *[1]Novartis Pharmaceuticals, Basel, Switzerland, East Hanover, NJ.*

A recent retrospective report suggested that when pediatric renal allograft recipients received a basiliximab-cyclosporine-prednisone regimen, cyclosporine (CsA) exposure was increased compared with historical controls thereby necessitating lower CsA doses. This report contrasts with prospectively collected CsA troughs (Cmin) in the two double-blinded, placebo-controlled phase 3 trails with basiliximab in adult renal transplantation which demonstrated that basiliximab did not influence CsA disposition. **Pediatric data and evaluation:** The recently completed international multicenter pediatric trial (n=39 patients, 1-16 yrs old) used basiliximab with a CsA-prednisone regimen and prospectively collected both CsA and basiliximab pharmacokinetic data weekly in the first 3 months posttransplant. CsA target ranges were 150-450 ng/ml in month 1 and 100-300 ng/ml thereafter using a monoclonal-specific assay. For each patient, the CsA Cmin, dose, and Cmin/Dose was identified from the last 3 weeks of CD25 saturation (ie, when basiliximab conc >0.2 mcg/ml) and the 3 weeks after CD25 desaturation. Comparison of these time periods allowed each patient to act as his/her own control. Median CsA data are tabulated below; basiliximab was present at CD25-saturating concentrations in weeks -3 to -1 and absent in weeks 1 to 3.

CsA parameter	Week -1	Week -2	Week -3	Week 1	Week 2	Week 3
Dose (mg/day)	242	319	248	236	243	241
Cmin (ng/ml)	217	247	231	207	216	186
Cmin/Dose (ng/ml per mg/day)	0 96	0 98	0 88	0 92	0 99	0 86

CsA doses: There was a wide range of CsA mg-doses, but on average, they were relatively stable in the presence and absence of basiliximab; specifically, they did not differ in week -1 vs 1 ($p=0.32$). **CsA Cmin:** Cmins decreased over time as is conventional in the early posttransplant months. Between week -1 and 1, 23 patients (59%) had a numerical decrease in Cmin and 16 (41%) had an increase. Median Cmins in week -1 and 1 did not differ ($p=0.22$). **Dose-normalized Cmins:** Cmin/Dose remained relatively stable over the 6-week period of evaluation; medians did not differ before (week -1) or after (week 1) CD25 desaturation ($p=0.51$). **Conclusions:** There was no evidence from prospectively collected data that the systemic presence of basiliximab at pharmacologically active concentrations had an influence on cyclosporine disposition in pediatric de novo renal allograft recipients.

POSTER SESSION I:

KIDNEY - PRESERVATION, DONATION/ALLOCATION, ECONOMICS/PUBLIC POLICY, SURGICAL TECHNIQUES, AND OTHER I

Abstract# 240 **Poster Board #-Session: P53-I**

HAND-ASSISTED LAPAROSCOPIC LIVE DONOR NEPHRECTOMY: THE OHIO STATE UNIVERSITY EXPERIENCE. Aamer Ar'Rajab, Ronald P. Pelletier, Mitchell L. Henry, Elmahdi A. Elkhammas, Ginny L. Bumgardner, Elizabeth A. Davies, Ronald M. Ferguson.

The expanding use of live donor kidneys has been employed to overcome the national organ shortage. However, the donor nephrectomy procedure is a major operation with significant morbidity. The laparoscopic approach has emerged as an attractive alternative. We have performed a total of 53 laparoscopic donor nephrectomies since March 2000. All the laparoscopic procedures were performed hand-assisted. The procedure consisted of a midline infra-umbilical incision for hand insertion as well as two 12 mm ports for the endoscope and one working instrument.

The following is our current experience with laparoscopic donor nephrectomy. The donors consisted of 32 females and 21 males. There were 46 Caucasians, 6 African Americans, and 1 Asian American. The left kidney was removed in 52 donors, and the right kidney in one donor. Forty-four kidneys had one artery, seven had two, one had three and one had four arteries Two kidneys had double ureters. The warm ischemia time (defined from cross clamping the first renal artery until starting the cold perfusion on the back table) was 186 ± 40 seconds (mean ± SD). The time from cross clamping the first renal artery until kidney removal was 112 ± 24 seconds. The corresponding times for 10 open donor nephrectomies performed during the same period were 149 ± 47 seconds and 63 ± 19 seconds, respectively. The average length of stay was 3.1± 0.9 days. Four complications occurred in 4 patients (wound cellulitis, superficial thrombophlihitis, ileus and retroperitoneal hematoma). The wound cellulitis required readmission on POD#17 for I.V. antibiotics. The superficial thrombophlibitis occurred on POD # 16 and required no treatment. The ileus occurred on POD#9 and required readmission, NGT decompression and I.V. fluids. One patient developed a large non-expanding retroperitoneal hematoma diagnosed on CT scan and necessitating a blood transfusion. None of these procedures required conversion to open donor nephrectomy. The transplanted kidneys demonstrated excellent function immediately in 50 recipients, while 3 patients had delayed graft function with ATN required dialysis. Serum creatinine decreased from 6.3±2.4 mg/dl preoperatively to 1.8±1.2 mg/dl on POD#4.

In conclusion, the hand-assisted laparoscopic procedure for live donor nephrectomy is a safe procedure for the donor and results in a good quality organ for the recipient.

Abstract# 241 **Poster Board #-Session: P54-I**

WHO BECOMES A NON-DIRECTED KIDNEY DONOR? Cheryl Jacobs,[1] Deborah Roman,[1] Catherine Garvey,[1] Abhi Humar,[1] Arthur Matas.[1] [1]*Surgery, University of Minnesota, Minneapolis, MN.*

Background: Use of non-directed kidney donors (volunteers who donate to an anonymous recipient) remains an unconventional and controversial practice. We studied demographics, personality profiles, and motives of our non-directed donors.

Results: Of the 118 non-directed donor inquiries made to our center between 10/98 and 11/00, 7 such donations have occurred. The average time from first contact to actual donation was 9 months (range 6-11 months). Of the 7, 2 were females, 5 males. Mean age was 43 (range 21-63); 6 were white, 1 was Native American. Two had Master's degree, 2 had college degrees, and 3 were high school graduates. Five were single and 2 were married; 2 had biological children. Four lived alone. Five donors were employed, one was a homemaker, and one was in job transition. All donors were out-of-state residents; 5 applied for grants to assist with travel. Five of the 7 had support persons with them at the time of surgery. Postoperatively there were no surgical complications in the donors and none reported changes in their mental health or well-being; all were glad they donated. All recipients have functioning grafts.

Psychological testing revealed all donors were of normal, or above intelligence, with no aberrant personality traits or psychopathology, as indicated by MMPI results. None had been treated for alcohol or substance abuse; 2 had some intervention for depression remotely in the past. Psychological testing revealed the majority of individuals displayed an outgoing and social personality. All donors reported they were aware of the organ shortage and need for kidney donors. In all, motivation was to help another less fortunate so that they might improve another life. Four had a history of community service (volunteering, blood, platelet, or marrow donation.) Two had previously served in the military. One wanted to "give something back" to the community for having been able to live a fortunate life. Four reported they were motivated by religious, or spiritual beliefs. Only one had known someone special in his life that was affected by kidney disease.

Conclusions: We found that non-directed donors did not exhibit any major psychopathology and were able to make informed choices, without coercion. They were motivated by either a history of service to others, or a desire to do something good in their life. Our experience indicates that after careful evaluation, altruistic stranger donors should be considered and not be ruled out a priori.

Abstract# 242 **Poster Board #-Session: P55-I**

KIDNEY CANCERS IN RENAL TRANSPLANT RECIPIENTS. Christopher Gran,[1] John Hulbert,[1] Ken Roberts,[1] Sid Jain,[1] Arthur Matas,[2] Abhi Humar.[2] [1]*Urology, University of Minnesota, Minneapolis, MN;* [2]*Surgery, University of Minnesota, Minneapolis, MN.*

Background: Kidney cancers occurring after renal transplant (tx) may present in the native or tx kidney. We studied a large group of tx performed at a single center to determine incidence, risk factors, management, and outcome.

Results: Between 1963-1999, 5491 kidney tx were performed. We identified 13 recipients (7 females, 6 males) with 14 kidney tumors (incidence=0.25%). All were primary tx in the following distribution: kidney alone (n=8), kidney/pancreas (n=4), and kidney/liver (n=1). Nine of 14 tumors were renal cell carcinomas (RCC), 5 in the native kidney and 4 in the tx kidney. Mean age was 55.1 yrs (range=26-75); mean time to diagnosis 48 mths posttx (range=1- 81) in a native kidney and 89 mths (range=0-243) in the tx kidney. Tumor grade was as follows: Grade I (well-differentiated, n=4), Grade III (marked atypia, n=2), and unspecified grade (n=3). Of the 5 recipients with native kidney RCC, 1 had metastases at the time of diagnosis, 1 had a coexistent bladder cancer, and 1 had a coexistent lymphoma of the transplant kidney Treatment of RCC in the native kidney was with nephrectomy. One recipient received no treatment, as the tumor was discovered at autopsy. Additional treatment with chemotherapy and radiation were given to the recipients with metastatic disease and coexistent bladder cancer. All 4 maintained normal function in their transplant kidney after treatment. Treatment of RCC in the transplant kidney was with tx nephrectomy (n=3) or local excision (n=1). The 1 recipient treated with local excision continues to maintain good graft function. Six of the 9 recipients are deceased, 3 as a result of malignancy (only 1 as a direct result of their RCC, 2 attributed to some other malignancy).

Five tumors were Non-Hodgkin's lymphomas (NHL), all involving the tx kidney. Mean age was 38.7 yrs (range=3-61); mean time to diagnosis was 9.2 mths (range=2-24) posttx. In all cases the tumor was intermediate or high grade; metastatic disease was identified in 4 of the recipients. Treatment was varied and included nephrectomy, radiation, and alteration of immunosuppression. However, prognosis was poor; all 5 of recipients are deceased, all as a result of their malignancy (mean survival=3 mths, range=0-13 mths).

Conclusions. RCC generally occurs late after transplant and has a favorable prognosis. Lymphomas in the transplant kidney tend to occur earlier, are predominantly high-grade, and are associated with a very poor outcome.

Abstract# 243 **Poster Board #-Session: P56-I**

CV EVENTS AND DEATH ON PROVINCIAL RENAL TRANSPLANT WAITING LIST (RTXWL): THE BC EXPERIENCE. A. Levin,[1] D. Landsberg,[1] L. Siosan,[1] L. Venables,[1] L. Liu,[1] J. Gill,[1] W. Gourlay.[1] [1]*Nephrology, St. Paul's Hospital, Vancouver, BC, Canada;* [2]*BC Transplant Society, Vancouver, BC, Canada;* [3]*University of British Columbia, Vancouver, BC, Canada.*

The impact of increase in waiting time for pts on the RTxWL is not well described. This provincial cohort study was designed to track pts in BC on RTxWL. Pts already on the RTxWL prior to 06/98 (N=392, prevalent cohort) and those assessed during the following year until 06/99 (N=243, incident cohort) had clinical, laboratory and history data entered into the data base. Data from initial assessment and 6 mo follow-up (Fup) were collected We report here the results of 473 pts awaiting 1st RTx from initial and 822 follow-up visits.

Of the 473 pts, 58% were male, X age was 48 yrs (20-74 yrs). No differences in age, gender or diabetic status b/n prevalent and incident cohorts were found. 23% of pts had DM, 51% were current smokers, and 12.7% had a Hx of CVD. During the current Fup period, 19 (4%) were removed from the RTxWL, 23 (5.1%) were put on hold, and 89 (19%) received RTx: 32% within 1 yr and 44% after >2 yrs (X waiting time = 26 mo, median 22 mo). Those who received Tx had increases in Hgb (+20 vs +8 g/L in dialysis cases p<.01, and lowering of BP 20/12 mm/Hg vs 5/1 mmHg in dialysis pts p<.01). Sixteen pts died during Fup. 13 (3.4%) prior to Tx, and 3 post Tx. Of those who died, 25% had a Hx of CV event vs 8.5% of survivors (p<.05). Those who died pre Tx were predominantly male (61%), DM (61%), and had a fall in Hgb (122 g/L to 94 g/L)and in BP (150/80 to 107/60)b/n initial assessment and prior to death. Of the 3 who died post Tx, 2 were female, 66% were DM, and they had a similar fall in Hgb and BP (114 to 104 g/L and 120/80 to 100/70) for assessment prior to death; no differences in age were seen (57 vs 56 yrs). CV events occurred in 23 pts during time on the RTxWL; 15/23 (63%) were from the historical cohort. 5/23 had a Hx of CV event. Mean time b/n activation and event was 21.5 mo.

Pts at risk for death prior to or post Tx are older, male and diabetic. CV events occur during Tx waiting period even though pts are screened for CV disease. CV risk education should be the focus of pt care prior to and while on the active TxWL. Ongoing data collection may allow us to develop models to identify high risk pts and to identify important targets for intervention.

Abstract# 244 **Poster Board #-Session: P57-I**

LONG-TERM OUTCOME OF RENAL TRANSPLANTATION IN RECIPIENTS OLDER THAN 65 YEARS. Amado Andres,[1] Juan C. Herrero,[1] Jose M. Morales,[1] Teresa Ortuño,[1] Beatriz Domíguez,[1] Eduardo Hernandez,[1] Manuel Praga.[1] [1]*Nephrology, H. 12 de Octubre, Madrid, Spain.*

In the last years the indication of dialysis and renal transplant has been expanded to older people. From January 1991 to May 2000 we have performed in our center 1016 cadaveric renal transplants. 104 of them were performed in recipients ≥65 years (26 older than 70 years). Mean follow up was 33±26 months (range 3-116). Donor s age was 66±11 years (range 15-85) and donor mean serum creatinine 0.9±0.3 mg/dl. 14 out of 104 older recipients were transplanted with dual renal transplant. The recipients were 34 females and 70 males with a mean age of 68±3 years (range 65-76). Cold ischemia time was 23±5 hours 57% of them presented acute tubular necrosis and 14% acute rejection. Eight recipients (7.6%) had primary non-function due to death of the recipient (3 cases), surgical complications (4 cases) and acute rejection (1 case). Thirty-two recipient lost their renal graft function during the evolution due to patient death with functioning graft (13 cases), chronic rejection (7 case), graft thrombosis (3 cases), acute rejection (1 case)and primary non-function (8 cases). Sixteen patients died during the follow up due to coronary artery disease (7 cases), cancer (3 cases), infections (5 cases), gastrointestinal hemorrhage (1 case). Others complications were 11 lymphoceles, 12 ureteral problems (7 stenosis, 5 fistulas), 10 complication of surgical incision (dehiscences or infections), 4 renal artery stenosis (that needed angioplasty), 10 CMV infections and 3 varicela infections. Actuarial graft survival (censored death with functioning graft) was 88%, 80% and 72% at one, three and five years respectively. One, three and five years actuarial patient survival was 93%, 86% and 68% respectively.Mean serum creatinine at the end of the follow up was 1.7±0.6 mg/dl.In conclusion renal transplant in patients older than 65 years is a satisfactory treatment that show excellent long-term results with morbi-mortality no higher than dialysis.

Abstract# 245 **Poster Board #-Session: P58-I**

PROSPECTIVE INCEPTION COHORT ANALYSIS OF OPEN DONOR NEPHRECTOMY WITH HANDOSCOPIC AND LAPAROSCOPIC DONOR NEPHRECTOMY. Amy D. Lu,[1] Lynt B. Johnson,[1] Jeff S. Plotkin,[1] Joseph Buell,[2] James F. Whiting,[3] William H. Marks,[4] Phil Chapman,[4] Kenneth A. Newell,[5] Paul C. Kuo.[1] *[1]Surgery, Georgetown University, Washington, DC, [2]Surgery, University of Cincinnati, Cincinnati, OH; [3]Surgery, Maine Medical Center, Portland, ME, [4]Surgery, Swedish Medical Center, Seattle, WA; [5]Surgery, University of Chicago, Chicago, IL.*

Hypothesis. To evaluate any differences among the three approaches of donor nephrectomies.

Methods. We analyzed data in a prospective inception cohort of four institutions. The laparoscopic method utilizes four 1 cm. port incisions with an additional infraumbilical incision to extract the kidney. The handoscopic method utilized a pneumosleeve device to allow for direct hand manipulation of the kidney. 251 consecutive patients were divided into three groups based on the method of donor nephrectomy. Analysis of 9 donor and outcome variables were performed. Significance was defined at $p<0.05$. Chi-square and T-test were performed where appropriate

Results. There were 109 laparoscopic cases, compared with 66 open and 76 handoscopic cases. A significant difference in operative times was seen between the handoscopic and laparoscopic cases; however there was also an increase in the conversion rate in the handoscopic cases compared to the laparoscopic (9.4% vs.4%). There was no difference in the incidence of complications among the three groups as well as no significant difference in the incidence of delayed graft function (DGF).

Cases	Number (N)	Age (yrs)	OR time (hrs:min)	Conversions	DGF
Handoscopic	76	40.5±0.8	2.5±5.5*	9%*	3.4%
Laparoscopic	109	39±1.0	3.59±5.2*	4.6%*	3.7%
Open	66	31.2±1.5	2.3±0.3*		4.5%

*p<0.05

Conclusions. Handoscopic donor nephrectomy shortens the operative time and may quicken the learning curve. The laparoscopic and handoscopic methods are effective operations compared to the traditional approach.

Abstract# 246 **Poster Board #-Session: P59-I**

PERCENT IMPROVEMENT IN HEART RATE VARIABILITY IN DIABETIC AND NONDIABETIC KIDNEY AND KIDNEY PANCREAS RECIPIENTS. Ann K. Cashion ,[1] Rebecca P. Winsett,[1] Patricia F. Joplin,[1] Robert J Stratta,[2] Osama Gaber,[2] Donna K. Hathaway.[1] *[1]College of Nursing, University of Tennesee Health Science Center, Memphis, TN; [2]Dept. of Surgery, University of Tennessee Health Science Center, Memphis, TN.*

Purpose. Improvement in autonomic function as indicated by changes in heart rate variability (HRV) from pre to post transplant, regardless of transplant type, has been documented. The purpose of this study was to describe percent improvement in HRV indices from pre to 12-month posttransplant by transplant type.

Methods: Twenty-four hour HRV indices in frequency (low, LF; and high, HF) and time (standard deviation of R-R intervals, SDNN) domains were obtained from nondiabetic kidney (NonDM kidney, n=75), kidney pancreas (KP, n=26), and diabetic kidney (DM kidney, n=14) recipients with matched pre and 12 months posttransplant (postTX) data

Results: The sample was 69% men, 44% African-Am., mean aged 44 ± 11 yrs. Subgroups were similar in age and gender. PostTx adverse events did not differ by groups. As expected, overall improvement was seen from pre to posttransplant (see table). KP recipients had the lowest pre and postTx HRV indices, but improved by 21% in LF and 23% in SDNN at 12 mo postTx. While NonDM kidney recipients had the highest HRV indices at both time points, they also had the lowest percent improvement for all indices Data showed an improvement of 12% for HF and 34% for SDNN in DM kidney recipients. The SDNN increased ($p< 0.05$) for all groups.

	NonDiabetic Kidney			Kidney Pancreas			Diabetic Kidney		
	LF	HF	SDNN	LF	HF	SDNN	LF	HF	SDNN
PreTx	4.81	3.66	91	2.49	2.26	56	3.54	2.94	65
12 mo PostTx	5.07*	3.82	111*	3.16*	2.45	72*	3.88	3.36	98*
Improvement	5%	4%	18%	21%	7%	23%	9%	12%	34%

*p≤0.05 from pre to post

Conclusions: Patients with diabetes and renal failure have more compromised autonomic function than patients with renal failure alone and they show the greatest percent improvement at 12 mo. postTx, regardless of transplant type. Further study is needed to determine if this trend continues. While improvement is reported in sympathetic and parasympathetic (LF, HF) modulation, it is important to note that for all transplant types significant improvement is seen in SDNN, a marker for increased risk for sudden cardiac death in other populations.

Abstract# 247 **Poster Board #-Session: P60-I**

ENHANCED CHARACTERIZATION OF PRE-SENSITIZATION STATUS IN KIDNEY TRANSPLANT CANDIDATES USING SENSITIVE TECHNIQUES. Antonina Piazza, Elvira Poggi, Giuseppina Ozzella, Palmina I. Monaco, Simona Servetti, Carlo U. Casciani, Domenico Adorno. *[1]CNR - Inst. Tissue Typing, Rome, Italy.*

A careful characterization of HLA antibodies (Abs) in potential transplant recipients avoids risk of early rejection and improves graft survival in kidney transplantation. This study aimed at investigating the sensitization status due to different sources of immunization in transplants candidates using the sensitive and specific FlowPRA beads (One Lambda Inc, CA) technique.

Among 838 kidney transplant recipients, periodically screened for alloantibody production using FlowPRA class I and class II Screening method (a pool of 30 beads coated with different purified HLA class I or class II antigens), only the 221 patients (pts) exposed to a single kind of immunizing event were enrolled in the study. Our patient population was divided into three groups according to the type of immunizing event: Ts-group (107 transfused pts); Tx-group (39 previously transplanted pts), Pg-group (75 pts who had pregnancies/abortions). In order to define the HLA specificity of the detected Abs, FlowPRA class I and class II Specific assays (four groups of eight beads coated with different purified HLA class I or II antigens) were used.

FlowPRA Screening results showed a significantly higher incidence of sensitized pts in the Tx-group (38.5%, $p<0.00001$) and in the Pg-group (25.3%, $p<0.00001$) than in the Ts-group (2.8%). We moreover highlighted a great incidence of HLA class II Abs not only in the Tx-group (73.3%), but also in the Pg-group (52.6%).

Analysis of HLA class I Abs specificity showed a high incidence of CREG Abs (mainly against CREG 1C) both in the Pg-group (93.7%) and the Tx-group (85.7.5%). Remarkably 8 of the 20 (40%) CREG-specific Abs evidenced in the sera of the Pg pts and 6 of the 14 (42.8%) found in the Tx pts were intra-CREG Abs. As regards HLA class II specificity, our study evidenced that Abs directed towards a public antigen (DR51, DR52, DR53) were present in 40% of the Pg pts and 18% of the Tx pts.

In conclusion our data demonstrated that transplants and pregnancies had a similar strong immunogenicity as regards incidence and intensity of sensitization and Abs specificity. Moreover, the finding of an elevated production of HLA class I intra-CREG Abs and HLA class II Abs directed toward public-antigens in patients sensitized only by pregnancies, indicates that this kind of immunizing event has a high immunogenic capacity, which must be thoroughly investigated before transplantation using sensitive and specific techniques.

Abstract# 248 **Poster Board #-Session: P61-I**

QUALITY OF LIFE IN RENAL TRANSPLANT PATIENTS WITH FUNCTIONING GRAFTS; THE STORY UNDERNEATH. Argiris Asderakis,[1] Christopher Brown,[2] Phil Dyer,[2] Robert W.G. Johnson.[2] *[1]Transplant Unit, University Hospital of Wales, Cardiff, United Kingdom, [2]Renal Transplant Unit, Manchester Royal Infirmary, Manchester, United Kingdom.*

Aim: To measure the subjective QOL(Quality of Life)of patients with functioning kidney grafts and associate it with risk factors including the use of maintenance steroids.

Patients and methods: 103 renal transplant patients with functioning grafts at least 1year post-transplant were interviewed by using 3 instruments of measurement of QOL: Kidney Transplant Questionnaire (KTQ) - a disease specific questionnaire, SF36 - a generic health questionnaire for chronic illness, and EORTC health thermometer. Patients with an acute infection, acute rejection or cardiac event in the last 4 weeks were excluded.

Results:54.4% of patients were receiving maintenance steroids. The total KTQ score was 138.8(±24.5). A worse total KTQ, was associated with a creatinine over 200mm/l ($p=0.02$), treatment with steroids ($p=0.03$) but not with sex, age group and mode of dialysis treatment pre-transplant. A worse appearance score in KTQ was more common in females ($p=0.04$), patients on steroids ($p=0.1$), and patients with a creatinine over 200μm/l ($p=0.05$). A worse score in the physical dimension of the SF-36 was associated only with an age over 55 years ($p=0.006$), but not with other variables. The use of steroids was associated with a worse score in the emotional dimension of both KTQ ($p=0.08$) and the SF-36 ($p=0.08$). The perception about their health measured by SF-36 was worse in patients on steroids ($p=0.01$). Patients with a creatinine over 200μm/l had less vitality ($p=0.04$) than the rest. The mean Health Thermometer score was 69.85 (±18.4) and was significantly reduced with increased age ($p=0.04$), creatinine over 200μm/l ($p=0.07$), CAPD use before the transplant as opposed to haemodialysis or no dialysis ($p=0.04$), and the use of maintenance steroids ($p=0.04$).

Conclusion: Kidney transplant patients have a good subjective QOL as measured by various scoring systems. Their age seems to be affecting only the physical dimension of those scores. In contrast the use of maintenance steroids seem to be adversely affecting the quality of life in multiple dimensions (physical, emotional, appearance) as well as the overall perception of the patients about their health prospects. Patients with a creatinine over 200μm/l seem to have more physical problems, less vitality and worse overall health. Good kidney function on a steroid free regimen seems to be the ideal solution for a good quality of life following renal transplant.

Abstract# 249 **Poster Board #-Session: P62-I**

LAPAROSCOPIC DONOR NEPHRECTOMY: A POSITIVE COMPARISON WITH OPEN PROCEDURES. Brian M. Gogel, L. Michael Goldstein, Robert C. Schoenvogel, Howard C. Derrick, III, Matthew V. Westmoreland, Michael Seiba, Edmund Q. Sanchez, Shigeru Marubashi, Robert M. Goldstein, Marlon F. Levy, Ernesto P. Molmenti, Carlos G. Fasola, Thomas A. Gonwa, Laura L. Christensen, Goran B. Klintmalm. [1]*Baylor University Medical Center, Dallas, TX.*

PURPOSE: An initial laparoscopic donor nephrectomy (LDN) experience was reviewed to determine the safety of this new procedure and to evaluate the quality of harvested organs.

METHODS: Between September 22, 1999, and July 12, 2000, the initial 31 LDN were performed. These are compared to 124 open nephrectomies (ODN) performed between 1989 and 2000. Since 1996, all patients had preoperative assessment of the renal vasculature with magnetic resonance angiography (MRA). Potential donors were candidates for LDN if MRA identified standard left renal vasculature. LDN were performed by surgeons with extensive experience with advanced laparoscopic surgery. In the LDN group, 30 kidneys had a single artery and only 1 had two arteries. In the ODN group, there were kidneys with one (n=115), two (n=7), or three (n=2) arteries. In all LDN the left kidney was harvested via a transperitoneal approach. In the ODN group, 93 left kidneys and 31 right kidneys were harvested via a flank approach. Male donors made up 51% and 33% of the LDN and ODN groups, respectively. The median age of donors was 40.1 years (range 18-65) and 41.1 years (range 16-64) in the LDN and ODN groups, respectively.

RESULTS: The mean operative time for LDN was 234 minutes (range 135-460) and ODN was 145 minutes (range 105-220) ($p<0.0001$). The mean operative time in the first 15 LDN was 271 minutes (range 203-460) and the second 15 LDN was 198 minutes (range 135-317) ($p<0.002$). The mean blood loss was 40cc (range 0-100) for LDN and 134cc (range 25-400) for ODN ($p<0.0001$). There were no intraoperative complications or conversions to open nephrectomy in the LDN group. The mean length of stay (2.8 days vs 5.4) ($p<0.0001$), narcotic use (54 mg vs 116) ($p<0.0001$), and time to regular diet (1.6 days vs 2.6) ($p<0.0001$) were less in the LDN group. There were no mortalities in the LDN group and the only morbidity was fever (n=1, 3%). In the ODN group there were 38 complications including fever (n=13, 10%), wound infection (n=9, 7%), pneumothorax (n=1, 1%), others (n=15, 12%). In the recipients, ureteral complications were observed in 2 (6%) LDN and 9 (7%) ODN.

CONCLUSIONS: With experience LDN have surgical times equivalent to the average ODN and shorter hospitalization, thus indicating true resource saving with this highly technical procedure.

Abstract# 250 **Poster Board #-Session: P63-I**

FUNCTIONAL PERFORMANCE AND HEALTH-RELATED QUALITY OF LIFE AFTER KIDNEY TRANSPLANTATION ARE ADVERSELY AFFECTED BY DIABETES AND CADAVERIC ORGANS. David H. VanBuren,[1] William Nylander,[1] Irene Feurer,[1] Theodore Speroff,[1] Robert E. Richie,[1] Simin Goral,[1] Keith Johnson,[2] Harold Helderman,[1] Christina Ynares,[2] Denise VanBuren,[2] Jeannie Hopkins,[1] Sarah Swanson,[1] Jackie Ray,[1] Paul E. Wise,[1] C. Wright Pinson.[1] [1]*Vanderbilt University, Nashville, TN,* [2]*Renal Consultants, Nashville, TN;* [3]*Nashville VA Medical Center, Nashville, TN.*

Background: We recently reported objective functional performance (FP) and subjective health-related quality of life (HRQOL) following liver, heart, lung, and kidney transplantation (Tx). The combined organ groups improved, but kidney recipients started from a relatively superior point and demonstrated less improvement in FP and no significant change in HRQOL. The current study was undertaken to identify factors specific to renal Tx that are associated with FP and HRQOL. **Methods:** Karnofsky FP status was assessed at baseline, 3 and 6 months, and annually thereafter (to 4 years maximum) in 86 adult patients. Patients reported HRQOL using the Short Form-36 (SF-36) and Psychosocial Adjustment to Illness Scale (PAIS). Clinical data included primary diagnosis, whether diabetic prior to Tx, time on dialysis prior to Tx, and donor organ type (cadaveric or living). Data were analyzed via analysis of variance methods and path analysis. A HRQOL composite comprising the SF-36 and PAIS domains was derived via factor analysis. Summary data are mean ± standard error of the mean. **Results:** FP improved ($p < .001$) following renal Tx (from 75 ± 1 to 77 ± 1, 81 ± 1, and 82 ± 1 at 0, 3, 6, and 12 months, respectively). Patients receiving organs from a living donor showed continued improvement, while those receiving a cadaveric (CADV) organ stabilized between 6 and 12 months (interaction contrast $p < .05$). Patients requiring dialysis prior to Tx demonstrated trends towards less improvement in FP over the first year ($p = .09$) and lower SF-36 physical component scores overall (39 ± 1 vs 44 ± 2, $p = .08$). Path analysis confirmed improvement in FP with time post Tx ($\beta = .23$) but negative effects on FP of diabetes ($\beta = -.22$) and CADV organs ($\beta = -.23$, all $p \leq .05$). In turn, FP had a direct effect on HRQOL ($\beta = .40$, $p < .001$). **Conclusions:** Functional performance shows modest improvement with time following renal Tx and is negatively impacted by diabetes and receiving a CADV organ. The effects of time post Tx, donor type, and diabetes on FP in turn affect HRQOL.

Abstract# 251 **Poster Board #-Session: P64-I**

LAPAROSCOPIC LIVE DONOR NEPHRECTOMY (LapNx) RESULTS IN INCREASED UTILIZATION OF LEFT KIDNEY GRAFTS WITH MULTIPLE RENAL ARTERIES - A SURGICAL AND NONSURGICAL OUTCOME ANALYSIS OF 79 CONSECUTIVE CASES. Christoph Troppmann,[1] K. Wiesmann,[1] B. Wolfe,[1] J. P. McVicar,[1] R. V. Perez.[1] [1]*Department of Surgery, University of California Davis, Sacramento, CA.*

Operative-technical limitations of LapNx have led to the more frequent choice of ***left*** kidneys for nephrectomy (in >90% of all LapNxs in the U.S.) - irrespective of their number of renal arteries (RAs). But the implications for donors and transplant (tx) recipients of this dramatic change in surgical practice are unclear.

Methods: We studied retrospectively in an intent-to-treat analysis 79 consecutive left LapNxs done at a single center 04/97-10/00 using standard LapNx and arterial reconstruction techniques. In all, 58 kidneys (73%, Group A) had single, and 21 (27%, Group B) multiple RAs. Median posttx follow-up was 3.0 yr. Demographics did not differ significantly between both groups. Immunosuppression included a triple protocol.

Results: Length of stay was similar for donors and recipients in both groups. We noted 6 (8%) intraoperative conversions to open nephrectomy (5 [9%], Grp A vs 1 [5%], Grp B; p=n.s.). Mean total (donor+recipient) operating time was significantly longer for kidneys with multiple RAs (610 min for Grp B vs 553 min for Grp A, p=0.01). No graft was lost from technical complications. Early and late graft outcome was (Grp A vs. B: all p-values >0.05):

	Median Posttx Serum Creatinine [mg/dl.]			≥1 Acute Rejection	2-yr Graft Survival
	1 week	1 month	2 yrs		
Group A (n=58)	1.7	1.4	1.4	20 (35%)	95%
Group B (n=21)	1.9	1.4	1.4	7 (33%)	100%

4 grafts (5%; all in Grp A, p=n.s.) had delayed function (defined as dialysis during the first week posttx).

Conclusions: While the procurement rate of grafts with multiple RAs in our series was 69% higher compared to an open historical control cohort ([16%], *Ann Surg 221:406, 1995*), LapNx and tx of those kidneys was safe for both donor and recipient. However, total operating time was significantly longer for txs with multiple RAs. Multiple RAs are thus not a contraindication to LapNx, but longer follow-up will be necessary to assess for late (>5 yr) complications (eg, RA stenosis, ureteral strictures) that may result from this shift of kidney selection strategy in live donors. Optimization of operative techniques for ***right*** LapNx will be necessary to minimize the increased resource utilization associated with the procurement of left kidneys with multiple RAs.

Abstract# 252 **Poster Board #-Session: P65-I**

INCREASED SENSITIVITY OF FLOW CLASS I PRA BEADS IN DETECTING HLA CLASS I ANTIBODY LEVELS (PRA) COMPARED WITH ANTIGLOBULIN (AHG) IN CANDIDATES FOR CADAVERIC RENAL RETRANSPLANTATION. Christopher F. Bryan, Scott B. McDonald, Karen A. Baier, Alan M. Luger, Mark I. Aeder, Daniel Murillo, Nicolas A. Muruve, Paul W. Nelson, Charles F. Shield, III, Bradley A. Warady. [1]*Midwest Transplant Network, Westwood, KS.*

Our centers' graft survival for cadaveric renal retransplantation has improved over the years in large part due to use of flow cytometry crossmatching to eliminate the transplantation of kidneys to patients with donor-specific HLA Class I antibodies that are undetectable by anti-human globulin (AHG) crossmatch methodology. It has recently become possible to screen sera of regraft candidates for routine HLA Class I by flow cytometry using a mixture of 30 distinct bead populations each coated with the Class I antigen phenotype derived from different cell lines. In this study we compared the efficacy of Class I antibody screens done by flow cytometry beads with that of our routine AHG method for patients currently awaiting cadaveric renal retransplantation. Serum samples were chosen from 21 regraft candidates on our current (May-June, 2000) OPO-wide kidney waiting list whose current AHG PRA indicated that they were unsensitized (< 3%) and 5 regrafts who had an intermediate Class I PRA (11 - 79%). The results of that comparison are shown in the following table.

	Current Class I PRA (%) Level			
History of Positive AHG PRA (≥10%)	AHG	Flow Beads	Peak AHG PRA	Percent Regrafts with Positive Flow Class I PRA (>10%) & Neg AHG PRA
No (n=16)	0.5 ± 0.8	11.6 ± 14.8	0.6 ± 1.3	43% (7/16)
Yes (n=5)	0.4 ± 0.8	40.4 ± 19.4	83.0 ± 18.6	100% (5/5)
Intermediate (n=5)	33.2 ± 13	72.4 ± 10.2	51.2 ± 32.2	

Regraft candidates whose current AHG Class I PRA was negative segregated into two groups by flow Class I PRA based on the patient's historical AHG sensitization. Of those regrafts whose AHG Class I PRA history was negative, 43% had a positive flow Class I PRA. Their AHG PRA (0.5% ± 0.8) was significantly lower than the flow PRA (11% ± 14) ($p<0.01$). In regrafts with a history of AHG sensitization, 100% of the patients had a positive flow Class I PRA and had a higher flow PRA (40% ± 19) than AHG (0.4% ± 0.8) ($p<0.01$). Finally, the intermediate AHG Class I PRA group had a higher flow Class I PRA (72% ± 10) than the AHG PRA (33% ± 13) ($p<0.001$). Based on these results, we now do all Class I antibody screens for regraft candidates by flow beads and note a higher correlation with final regraft flow crossmatch results.

Abstract# 253 **Poster Board #-Session: P66-I**

OXIDANT STRESS AND ANTIOXIDANT CAPACITY IN URINE OF RENAL TRANSPLANT RECIPIENTS PREDICTS EARLY GRAFT FUNCTION. Daniel A. Shoskes,[1] Asha R. Shahed,[2] Sun Kim,[2] Hans A. Gritsch,[3] Gabriel Danovitch,[3] Alan Wilkinson.[3] *[1]Urology, Cleveland Clinic Florida, Fort Lauderdale, FL; [2]Urology, Harbor-UCLA Medical Center, Torrance, FL; [3]Urology, UCLA, Los Angeles, CA.*

Non-immune injury is a major deterrent to renal allograft survival. Oxidative stress can reflect inflammatory and ischemic injury in several diseases, including ischemia-reperfusion injury. We have reported that higher urinary levels of the oxidant stress marker 8-isoprostane F2α (IsoP) and lower total antioxidant capacity (TEAC) levels in urine of cadaveric (CAD) kidney donors correlated with delayed graft function (DGF) We wished to study if these markers correlate with early graft function inrecipients.

Methods: Urine was collected from 29 recipients on days 1-5 post transplant. There were 12 living related recipients (LRD), 9 CAD with early graft function (EF) and 8 CAD with DGF. Urine was centrifuged and pellet and supernatant frozen. IsoP levels were measured using an EIA kit and TEAC levels were measured as Trolox equivalent.

Results: A significant increase in TEAC levels was seen in cadaveric recipients with DGF (1.88 ± 0.21) compared to either cadaveric recipients with EF (0.71 ± 0.12) or LRD recipients (0.69 ± 0.12) (p=0.0006). No changes were detected in the IsoP levels between patient groups.

Discussion: Total antioxidant capacity is significantly higher in urine from renal transplant recipients with DGF. Renal antioxidant enzymes may be induced in response to oxidant stress. IsoP levels are not predictive of early graft function. Urinary antioxidant capacity is a simple and rapid test that can predict early graft function in recipients and guide immunosuppression selection.

Abstract# 254 **Poster Board #-Session: P67-I**

MICROINVASIVE DONOR NEPHRECTOMY (MDN). Deepak Mital,[1] Weislaw Podlasek,[1] Stephen C. Jensik.[1] *[1]General Surgery, Section of Transplantation, Rush Medical College, Chicago, IL.*

PURPOSE: In living donor nephrectomy, at least a 6-7 cm. incision has to be made to extract the intact kidney. MDN with an anterior, retroperitoneal approach allows the entire open procedure to be performed through a single incision of this small size in the majority of donors.We have reviewed our results with MDN to determine how this open technique compares to Laparoscopic Donor Nephrectomy (LDN) in terms of morbidity for the living kidney donor.

METHODS: MDN is performed with an incision as small as 6.5cm., from the tip of the 10th rib to the edge of the rectus abdominis muscle.No rib is resected.The peritoneum is retracted medially with Deaver retractors.This direct approach provides excellent access to the renal artery and vein. They are divided after application of clips/staples, providing long vessels for transplantation. We have compared our results in 40 patients who had MDN from Jan.1999 to Oct.2000 with the University of Maryland LDN experience in 320 donors published recently (J.Urol.,Vol.164, 1494-99, Nov.2000)

RESULTS: (Means ±S.D.)

	No of Donors	Age (years)	Creatinine Clearance (ml /min)	Operative Time (min)	Morphine Requirement (mg)	Clear Liquid Diet (hrs)	Regular Diet (hrs)	Hospital Stay (hrs)
MDN	40	39 02±11 59	121 6±26 22	145 30±32 12	68 36±45 85	19 04±6 9	35 27±9 85	54 81±14 12
LDN	320	38 7±10 9	116 8±30 9	214 5±49 0	86 8±80 9	26 1±9 0	50 1±4 7	66 6±22

Renal allograft survival with MDN was 100%, with no technical losses.

CONCLUSION: MDN is associated with minimum morbidity for the donor. It is less invasive than LDN, being entirely retroperitoneal. This approach avoids injuries to the spleen and other intra-abdominal organs reported with LDN. The procedure allows rapid resumption of diet and discharge from the hospital. Our results compare quite favorably to those achieved with LDN in the largest single center experience published to date. MDN utilizes a minimum of medical resources by reducing operative time, avoiding use of expensive laparoscopic instruments and allowing early return to normal activities, driving and work.

Abstract# 255 **Poster Board #-Session: P68-I**

COMPARISON OF CREATININE CLEARANCE AND SERUM CREATININE SIX MONTHS AFTER RENAL TRANSPLANTATION AS PREDICTORS OF LONGTERM GRAFT SURVIVAL. Colin Geddes,[2] Carl Cardella,[1] Daniel Cattran,[1] Stanley Fenton,[1] Edward Cole.[1] *[1]Multi Organ Transplant Programme, University of Toronto, Toronto, ON, Canada; [2]Department of Medicine, North Glasgow University NHS Trust, Glasgow, United Kingdom.*

Several studies, including our own, have shown that renal function within the first year post transplant is an important predictor of longterm graft survival. The aim of this study was to assess whether calculated creatinine clearance is a superior predictor compared to serum creatinine and to determine whether there is a critical value of renal function associated with better outcome or whether the relationship is continuous.

Serum creatinine,weight, and age at 6 months were used to calculate creatinine clearance using the Cockcroft-Gault equation in 375 primary cadaveric renal transplants performed in a single centre between 1985 and 1997. Patients received similar cyclosporine based immunosuppressive protocols. Graft survival was analysed with and without censoring death. Serum creatinine and creatinine clearance at 6 months were compared as predictors of 5 year graft survival by measuring the area under the receiver operator characteristic (ROC) curve, which plots the sensitivity of a variable in predicting an ordinal outcome (in this case 5 year graft survival) against 1 minus the specificity at different cutoff values. A variable with poor predictive characteristics will have an area under the ROC curve of 0.5.

Eighty-two grafts failed in the first 5 years, 45 because of death with a functioning graft. The area under the ROC curves for 6 month serum creatinine and creatinine clearance as predictors of uncensored graft survival were 0.65 and 0.66 respectively and both were significantly greater than 0.5 (p<0.001). The values for censored graft survival were 0.73 and 0.68 (p<0.001 for both). All of the ROC curves were smooth, with no critical cutoff level of renal function for success or failure.

In conclusion, this study confirms that renal function at 6 months is a good predictor of 5 year graft survival with both serum creatinine or calculated creatinine clearance functioning equally well. Measurement of renal function at 6 months is therefore a useful surrogate marker for interventional trials in renal transplantation. The failure to find a specific level of renal function with an adverse prognostic effect suggests that the transplanted kidney behaves similarily to the partially damaged native kidney.

Abstract# 256 **Poster Board #-Session: P69-I**

A COMPARISON OF CLINICAL AND HUMANISTIC OUTCOMES IN RENAL TRANSPLANT RECIPIENTS AT ONE AND TEN YEARS. Elizabeth A. Davies,[1] Lisa R. Raiz,[2] Ronald M. Ferguson.[1] *[1]Surgery, The Ohio State University, Columbus, OH; [2]Social Work, Ohio University, Athens, OH.*

Purpose: This study examines differences in clinical and humanistic outcomes in successful renal recipients at one and ten years post transplant.

Methods: Kidney Transplant Outcomes Management System (KTOMS) was developed to integrate clinical outcomes and quality of life measurements in patients. A written survey is administered to patients during outpatient visits to the transplant center. Examined variables included gender, race, serum creatinine, employment status, receipt of disability check, smoking status, sexual functioning, patient perception of functioning and well-being(SF-36)and patient satisfaction with transplant physicians. T-tests and ANOVA were used to evaluate the variables.

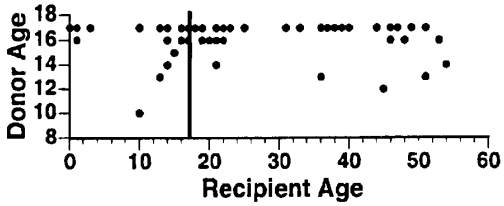
Results: 297 patients completed the survey:149 respondents 1 yr (1YR) and 148 10 yrs post transplant(10YR). 60% of 1YR and 55% of 10YR are male(p = NS). 22% of 1YR were African American and 9% of 10YR were African American (p = 0.004). Mean serum creatine for 1YR is 1.82 ± 1.03 and for 10YR is 1.68 ± 0.8 (p=NS). For 1YR, 37% were employed and 63% were not employed. For 10YR, 45% were employed and 55% were not employed (p=NS). 53% of patients 1YR received disability check while only 34% of patients 10Y received one (p < 0.002). For 1YR, 43% never smoked, 44% quit smoking and 13% currently smoke. For 10YR, 49% never smoked, 34% quit smoking and 17% currently smoke (p=NS). Although only 50% of patients denied alteration in their sexual functioning, there is no differences between groups. Patient perception of general health is significantly higher in 1YR than 10YR (62.3 ± 20.4 v 54.5 ± 24.9, p <0.01). Patient satisfaction with transplant physician care provided is significantly higher in 1YR (26.5 ± 3.7 vs 23.9 ± 4.6 (p < 0.001)).

Conclusions: 1. No differences between 1YR and 10YR are identified in gender, serum creatinine, employment status, smoking status, sexual functioning,and measures of patient perception of functioning and well being (SF-36). 2. African Americans are significantly under-represented in the 10 YR group potentially due to higher incidence of graft loss in the first 10 yrs. 3. Patient perception of their general health is significantly lower at 10 years. 4. Patient satisfaction with physician care is significantly lower at 10 years. 5. Decreases in the two subjective measures (general health and physician care satisfaction) suggest continuing assessment and intervention are warranted throughout the post-transplant period.

Abstract# 257 **Poster Board #-Session: P70-I**

THE USE OF MINORS AS LIVE KIDNEY DONORS. Francis L. Delmonico, William E. Harmon. [1]*Surgery, Harvard Medical School, Boston, MA;* [2]*Pediatrics, Harvard Medical School, Boston, MA.*

Background: A recent consensus conference on live organ donation concluded that informed consent requires the donor be competent, willing to donate, free of coercion, medically and psychosocially suitable, and fully informed of the risks and benefits. Organ donation from a minor strains the concept of voluntarism and it undermines the ability to provide valid consent. Nevertheless, the conference suggested that there may be exceptional circumstances to permit the ethical use of a minor as an organ donor, as in the case of identical twins, when the the potential donor and recipient are both highly likely to benefit. **Objective:** We analyzed the UNOS data base of kidney transplants to determine the frequency, donor/recipient pair demographics, and the transplant outcome of all live donors <18 years age. **Results:** Of approximately 40,000 live kidney donors between 1987 and 2000, 60 were <18 years of age (0.15%). There were only 7 pediatric recipients (12%) who appeared to be an identical twin by age, gender, blood type, and HLA match. 36 of the recipients (60%) were adults, and 21 of these were > 30 years of age. The largest age discrepancy was 14 year old donor for a 54 year old recipient. Overall, there was a O HLA mm in only 22 recipients.



The 5 year allograft survival was 77%; 2 of the 12 failures (16%) were due to acute graft thombosis. **Summary:** These data reveal: 1) that a minor donor kidney was transplanted more frequently to an adult than a pediatric recipient; 2) that only 12 % of all recipients were identical twins; and 3) that the use of a minor donor provided no better outcome than expected from an adult donor. **Conclusions:** These observations suggest a practice pattern that uses minor donors in clinical circumstances not anticipated by the consensus conditions of the live donor conference. Live organ donation from a minor should only be considered when there is no potential adult living donor available and all other opportunities for transplantation have been exhausted.

Abstract# 258 **Poster Board #-Session: P71-I**

BILATERAL PHARMACOKINETIC INTERACTION BETWEEN CYCLOSPORINE A AND ATORVASTATIN IN RENAL TRANSPLANT RECIPIENTS. Hallvard Holdaas ,[1] Anders Aasberg,[1] Anders Hartmann,[1] Ellen Fjeldsaa,[1] Stein Bergan.[2] [1]*Medical Department, National Hospital, Oslo, Norway;* [2]*Institute of Clinical Biochemistry, National Hospital, Oslo, Norway.*

Atorvastatin is increasingly used as a cholesterol-lowering agent, also in solid organ transplant recipients receiving cyclosporine A (CsA). However, the potential bilateral pharmacokinetic interaction between atorvastatin and CsA in transplant recipients has not previously been examined.

Methods: Baseline 12-hour CsA pharmacokinetic investigation was performed in 21 renal transplant recipients and repeated after 4 weeks of atorvastatin treatment (10 mg per day). At week four 24-hour pharmacokinetics of atorvastatin was also performed. All patients received a cyclosporine A and prednisolone based immunosuppressive regimen

Results: Treatment with 10 mg atorvastatin resulted in a 6-fold increase in plasma HMG-CoA reductase inhibitory activity compared with historic controls (P<0.001). Atorvastatin had a moderate effect on the pharmacokinetics of cyclosporine by reducing the AUC0-12 (area under curve,0-12 hours) by 9.5 + 18% (p<0.05) and Cmax (maximal concentration) by 13.5 + 24 % (p<0.01), while C12 (trough level) was unchanged. Total cholesterol and LDL-cholesterol decreased by 26.8 + 8.4% (p<0.0001) and 41.5 + 11.0% (p<0.0001), respectively, following 4 weeks of atorvastatin treatment.

Conclusion: Bilateral interaction between atorvastatin and CsA results in a 6-fold increase in plasma HMG-CoA reductase inhibitory activity and a small decrease in systemic exposure of CsA. Based on the findings in this study we recommend a starting dose of atorvastatin of 10 mg in renal transplant recipients receiving CsA

Abstract# 259 **Poster Board #-Session: P72-I**

LAPAROSCOPIC DONOR NEPHRECTOMY IN THE PRESENCE OF MULTIPLE RENAL ARTERIES. Hazem I. Abou El Fettouh, Inderbir S. Gill, Anoop M. Meraney, Brian Herts, Andrew C. Novick, David A. Goldfarb. [1]*Urology, Cleveland Clinic Urologic Institute, Cleveland, OH.*

Material and methods: Since January 1994, 180 patients underwent live donor nephrectomy (LDN) at our institution (open =130 and laparoscopic=50). Multiple renal arteries in the donor kidney were present in 10 (20%) donors in the laparoscopic group and in 11 (8%) donors in the open group. These data were retrospectively compared to 40 patients undergoing laparoscopic LDN wherein the donor kidney had a solitary renal artery

Results: see Table

	Open LDN with Multiple arteries (n=11)	Laparoscopic LDN Multiple arteries =10)	Laparoscopic LDN with single artery (n=40)	
Artey Length (cm)	-	3 8	3 5	
Vein Length (cm)	-	3 9	4 1	
OR time *(min)	194	219	222	
Blood Loss (ml)	158	116	159	
Warm ischemia (min)	-	3 9	4 1	
Analgesia** (mg MS04)	69 1	30 5	27 5	
Donor Serum Creatinine Pre-OP (mg/dl)		1 1	0 9	0 9
Donor serum creatinine post-OP (mg/dl)		1 4	1 2	1 3
Recipient creatinine at discharge (mg/dl)		1 7	1 6	1 4
Graft loss	1	1	-	
Hospital stay*** (day)	3 8	2 1	2	

*p=009 **p=0 03 ***p<0 001

Conclusion: The presence of multiple renal arteries is not a contraindication for laparoscopic donor nephrectomy. Similar to donor kidneys with a single renal artery, multiple-artery donor kidneys can be efficiently harvested laparoscopically and transplanted with good allograft outcome. One primary modification in the laparoscopic technique is to minimize individual dissection, and harvest the multiple arteries enbloc with an endo-GIA staples.

Abstract# 260 **Poster Board #-Session: P73-I**

THE FUNCTIONAL WEIGHT - A NEW SCORE FOR EVALUATION OF OLD DONORS. Heiner H. Wolters,[1] Thorsten Vowinkel,[1] Jens G. Brockmann,[1] Norbert J. Senninger,[1] Karl-Heinz Dietl.[1] [1]*Klinik und Poliklinik fuer Allg Chirurgie - Transplantation Westf. Wilhelms-Universitaet Muenster, Muenster, Germany.*

Background: The use of renal transplants from old donors has been tested in different transplantation programs with controverse results. A convincing score for evaluating marginal organs whether they are suitable for transplantation (Tx) or not is still missing. In our centre a new score has been developed and analyzed.

Methods: From July 1996 to July 2000 kidneys from 37 marginal donors (53-84y) were available in our centre. In these cases we decided to perform dual (D-NTX), single (S-NTX) or no kidney Tx by using our new score, the functional weigth (fcw). This score represents the mass of functioning nephrons and is calculated by using the following formula after having taken a pre-TX biopsy:

fcw = weight of kidney x (100 minus grade of glomerulosclerosis in histology (%) / 100). Every recipient was meant to gain >150 g functioning nephron mass. Thus a single Tx-was done with fcw > 150 g per organ (n=14 organs). A dual transplantation was done with fcw < 150 g per organ (n=30 recipients). If the sum of fcw of both kidneys was less than 150g no Tx was done.There was no significant difference neither in cold ischemia time (20 4 ± 2 3h) nor in HLA-mismatch or immunosuppressive regimen (CyA/MMF/Prednisolon) between single or dual transplantation.

Results. In 7 donors functional weight of single kidneys were > 150 g, in 30 donors functional weight of single kidney was < 150 g. Thus 14 S-NTX and 30 D-NTX were performed. One year patient and graft survival for D-NTX is 94 %, for S-NTX 100%. 3 D-NTX patients died with functioning grafts (mon:h 4,25,27, infection(2)/lung-cancer(1)). Rate of delayed onset of graft function (30% D-NTX vs 31% S-NTX) was comparable as well as incidence of acute rejection. 14% vs. 20 % respectively. The mean creatinine value 12 month post transplant was significantly higher in S-NTX (1 6±0 5 vs 2.3±0 7; p<0.05).

Conclusion: The rate of patient and transplant survival clearly shows that the new score is helpful to decide whether to perform S-NTX or D-NTX in marginal organs. With this new score more marginal organs can be used which are now refused for TX either for single or dual transplantation.

Abstract# 261 **Poster Board #-Session: P74-I**

EXCELLENT SHORT-TERM OUTCOME OF ABO-INCOMPATIBLE RENAL TRANSPLANTATION UNDER TACROLIMUS AND MYCOPHENOLATE MOFETIL IMMUNOSUPPRESSION. Hiroaki Shimmura,[1] Kazunari Tanabe,[1] Tadahiko Tokumoto,[1] Fusako Toda,[1] Shohei Fuchinoue,[2] Satoshi Teraoka,[2] Hiroshi Toma.[1] [1]*Department of Urology, Kidney Center, Tokyo Womens Medical University, Tokyo,* [2]*Department of Surgery, Kidney Center, Tokyo Womens Medical University, Tokyo, Japan.*

PURPOSE. ABO-incompatible living kidney transplantation (LKT) has been performed for widening of indications for kidney transplantation. Both tacrolimus

and mycophenolate mofetil (MMF) are newly developed potent immunosuppressive drugs which we have been using in ABO-incompatible LKT. We reviewed the recent results of ABO-imcompatible LKT under tacrolimus and MMF immunosuppression.

PATIENTS AMD METHODS: Thirteen patients with end-stage renal failure underwent ABO-incompatible living kidney transplantation at our institute between January 2000, and September 2000 There were 9 males and 4 females with mean age of 34.0 years. Incompatibilty in ABO blood group antigens was as follows: A1→O, 5 patients; B→O, 1 patient; A1B→A1, 1 patient; B→A1, 2 patients; A1→B, 2 patients; A1B→B, 2 patients. Plasmatheresis was carried out to remove the anti-AB antibodies prior to the kidney transplantation. In the induction phase, methylprednisolone, tacrolimus, mycophenolate mofetil, anti-lymphocyte globulin, and deoxyspergualin were used for immunosuppression. Local irradiation of the graft was performed at a dose of 1.5 Gy, on the first, third, and fifth days after transplantation. Splenectomy was done at the time of kidney transplantation in all patients.

RESULTS: During observation, only one patient lost graft due to sever pancreatitis, however the patient did not experience any rejection episode. Patient survival was 100% at one year. Furthermore, graft survival in tacrolimus and MMF-treated ABO-incompatible recipients was 92% at one year, which was much better than early results in cyclosporine-treated ABO-incompatible recipients (one year graft survival, 75%). Of 13 ABO-incompatible LKT recipients, only five experienced the acute rejection (38.5%).

CONCLUSION: ABO-incompatible LKT under tacrolimus and MMF immunosuppression offers an excellent short-term outcome.

Abstract# 262 Poster Board #-Session: P75-I

PROSPECTIVE NON RANDOMIZED STUDY COMPARING LAPAROSCOPIC DONOR NEPHRECTOMY (LDN), MINIMALLY INVASIVE OPEN DONOR NEPHRECTOMY (MIODN) AND OPEN DONOR NEPHRECTOMY (ODN).

Ignacio T. Castillon-Vela, Arturo Martinez, Evan Vapnek, Vladimir Ayvazyan, Sergey Ayvazyan, Giouziele Nabieva, Robert Naraghi, Roberto Mendez, Rafael G. Mendez, Hamid Shidban. [1]*National Institute of Transplantation, Los Angeles, CA.*

At our Center donor nephrectomy (DN) is performed through three different techniques: LDN, ODN and MIODN. We report a prospective comparative study focusing on recipient results.

METHODS: Since 6/99 65 ODN, 32 LDN and 51 MIODN (anterior subcostal retroperitoneal) were performed at our center.

Donor demographics:

	AGE (ty)	HEIGHT(cm)	WEIGHT(kg)	BMI	SEX(F%)	LNRD
ODN (n 65)	36 ±11	166 ±8	74 ±12	26 7 ±3	52	17(62%)
MIODN (n 45)	40 ±19	158 ±26 p1	70 ±26	25 6 ±6	63	12(24%)
LDN (n 32)	36 ±11	160 ±11	73 ±16	26 2 ±4	63	7(22%)

p1 p<0.05 comparing with ODN; p2: p<0.05 comparing with MIODN

RESULTS: Both LDN and MIODN reduced postoperative morbidity and pain; narcotic requirements were 51% and 22% lower than ODNrespectively . Warm ischemia time (WIT) and operation time (OT) were significantly longer in the LDN group: ODN: 2±1min and 2.1±0.4 hrs; MIODN: 2.1±0.7min and 2.6±0.58hrs p1; LDN 6.2±0.9min p1p2 and 4.1±0.9 hrs p1p2.

Incidence of delayed graft function in the LDN group (12.5%) was higher than MIODN (1.96%, p=0.04) but no different than ODN (4.6%); One primary graft failure occurred in each group (LDN and ODN: vein thrombosis; MIODN: recipient morbid obesity). Postransplant urological complications incidence was 12.5% for LDN(4 urine leaks), 5.9% for MIODN (2 leaks,1 obstruction) and 12.3% for ODN(6 leaks, 2 obstruction).

CONCLUSIONS: LDN and MIDN are both valuable techniques to reduce living donor postoperative morbidity and pain. Longer warm ischemia and operation times in LDN do not affect postransplant function or incidence of complications, although a learning curve is associated with the operation.

POSTER SESSION I:
LIVER - IMMUNOSUPPRESSION, ACUTE/CHRONIC REJECTION, GVH, PEDIATRICS I

Abstract# 263 Poster Board #-Session: P76-I

REDUCTION OF IMMUNOSUPPRESSION FOLLOWING BONE MARROW INFUSIONS IN LIVER TRANSPLANTATION.

A. G. Tzakis,[1] D. Weppler,[1] C. Ricordi,[2] R. Garcia-Morales,[1] R. Cirrocco,[1] N. Kenyon,[2] C. Nery,[1] M. R. Carreno,[1] P. Ruiz,[3] D. Levi,[1] V. Esquenazi,[4] A. D. Pinna,[1] J. Miller.[4] [1]*Transplantation, University of Miami, Miami, FL;* [2]*Diabetes Research Institute, University of Miami, Miami, FL;* [3]*Pathology, University of Miami, Miami, FL;* [4]*Transplantation, University of Miami and Veteran's Administration Medical Center, Miami, FL.*

Introduction: Unmodified cadaveric donor bone marrow infusions (DBMI) were administered perioperatively to liver transplant recipients in order to enhance chimerism (Group A). We studied the tolerogenic effect of DBMI by attempting to incrementally withdraw immunosuppression after 3+ years of therapy and compare them to a group that did not receive DBMI (Group B).

Materials and Methods: Stable liver recipients at least 3 years after transplantation, without episodes of rejection for at least one year, and no history of autoimmune disease were invited to participate. Immunosuppression was withdrawn incrementally; 1/3 at enrollment, 1/3 at 12 months (Phase 1), and completely by the end of year 2 (Phase 2). Routine blood work was obtained monthly. Additional surveillance was carried out at enrollment, 18 months, and 30 months by liver biopsy, iliac crest bone marrow aspirate (BM) and peripheral blood (PBL) analysis. All episodes of rejection were histologically confirmed.

Results: Ninety-nine patients were studied, 44 in Group A and 55 in Group B. Demographics and diagnosis at time of liver transplant were similar for both groups. Incidence of prior rejection was somewhat higher in Group B than Group A (35% vs 30%), as were baseline tacrolimus (0.073 vs 0.055 mg/kg/day) and cyclosporine (2.355 vs 1.753 mg/kg/day) doses. Twenty-one Group A and 26 Group B patients have completed Phase 1. No patients have completed Phase 2. To date, 3 Group A (6.8%) and 12 Group B (22.2%) patients developed rejection during Phase 1; 3 Group A (14.3%) vs 11 Group B (42.3%) have developed rejection during Phase 2. These differences were significant (p<0.05). There were two mortalities, both in Group A, from uncontrolled sepsis following biliary reconstruction (n=1) and coronary artery disease (n=1). Chimeric levels were studied in 19 Group A patients and 28 Group B patients. Group A patients had a BM chimeric mix level six times that of Group B (1.017% vs 0.168%). The PBL chimeric level was four times higher in Group A vs B (0.141% vs 0.036%).

Conclusion: DBMI may have a tolerogenic effect and allow withdrawal of immunosuppression in a significant number of liver transplant recipients.

Abstract# 264 Poster Board #-Session: P77-I

HYPERLIPIDEMIA IN PEDIATRIC LIVER TRANSPLANT PATIENTS ON CYCLOSPORINE-BASED IMMUNOSUPPRESSION.

Hansa M. Gupta,[1] Mohamed Abdolell,[1] Annie H. Fecteau.[1] [1]*Pediatric Surgery, Hospital for Sick Children, Toronto, ON, Canada.*

Purpose: The aim of this study is to determine the frequency of post-transplant hyperlipidemia in pediatric liver transplant pts receiving cyclosporine (CYA) and prednisone (pred) immunosuppression, to further evaluate the need for post-transplant lipid-lowering therapy.

Methods: The study group consists of 70 pediatric liver recipients transplanted between 1990-99, who were treated with CYA-based triple immunosuppression and survived at least 1 year. The mean age of the pts is 57.3 months, the mean follow-up is 46.4 months, and the male to female ratio is 1:1. Serum cholesterol (chol) and triglyceride (tg) levels were determined at 0, 3, 6, 9, 12, 24, 36, 48, and 60 months. Age and gender adjusted serum chol and tg values, published in the NCEP Report on blood cholesterol levels in children were used to calculate individual percentiles (perc.) at each time periods.

Results: The serum chol and tg levels are elevated (greater than 75th and 95th perc.) in a significant number of the pts, both pre- and post-transplant (Table). The number of pts with serum chol levels elevated above the 75th perc. is 43% pre-transplant and then gradually declines to 15% by 1 year post-transplant. In contrast, the percent of pts with a serum tg level above the 75th perc. rises from 77% pre-transplant to 100% of the pts in the first year, declines to 80% by 24 months, and then remains at approximately that level for the next 4 years. No statistically significant associations were found between mean CYA level/time period or total pred/kg/time period and serum tg or chol levels by ANOVA.

Conclusion: Serum chol and tg levels are elevated in a significant number of pediatric liver transplant pts receiving CYA and pred immunosuppression. The number of pts with serum chol levels above the 75th perc. gradually decreases over the first year. In contrast, serum tg levels remain above the 75th perc. in more than 3/4 of pts. The results of this study suggest a need for routine serial lipid analysis, diet modification, exercise, and lipid-lowering agents.

Variable	0 mos	3 mos	6 mos	9 mos	12 mos	24 mos	36 mos	48 mos	60 mos
Mean Serum CYA Level/Time Period	—	332 0	280 7	268 2	246 5	224 2	184 3	159 6	133 7
Percent Pts with chol Value > 75th Percentile	42 9	38 0	21 3	10 4	14 3	23 5	20 0	16 1	8 0
Percent Pts with chol Value > 95th Percentile	13 9	12 0	14 9	6 3	3 6	9 8	5 0	3 2	4 0
Percent Pts with tg Value > 75th Percentile	76 7	98 0	95 8	93 5	100	76 9	83 3	83 9	82 61
Percent Pts with tg Value > 95th Percentile	50 0	81 6	64 6	46 7	94 8	51 9	50 0	45 2	39 1