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**ESC Congress 2004**  
**28 August – 1 September**  
**Munich, Germany**

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# ESC Congress 2004, Munich

*28 August – 1 September 2004*

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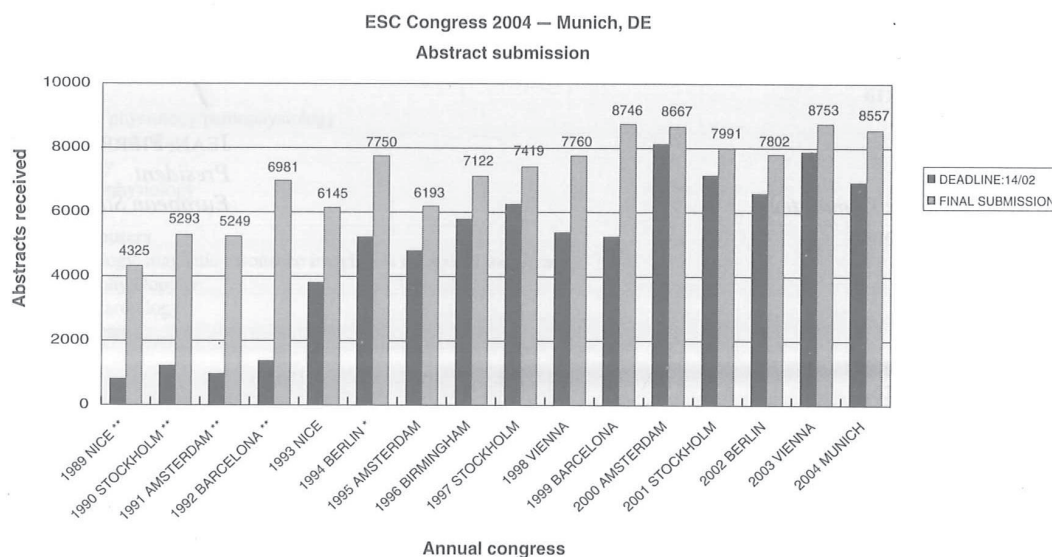
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## Abstracts selected for presentation at the European Society of Cardiology Congress 28 August – 1 September 2004, Munich – Germany

One of the main missions of the Congress of the European Society of Cardiology (ESC) is the presentation of innovative research. This year again, a large number of abstracts were submitted for review (see Figure 1). This high number of submissions underscores the strength of basic, epidemiological and clinical sciences in Europe and abroad, as well as the attractiveness of the European Congress as a whole. Out of 8557 submitted abstracts this year, 5.1% came from Japan, 3.9% from the USA and 2.4% from Brazil, amongst other non-European countries. The abstracts covered all aspects of cardiovascular medicine, but the three main areas accounting for 10% or more of all abstracts were coronary artery disease, heart failure and myocardial function, arrhythmias and pacing. Since abstracts are now submitted as either “Bench” or “Bedside”, the contribution of Basic Science is clearly identified and amounts to 16.3% of all submissions, an encouraging 2.3% increase compared to Vienna 2003.

**Figure 1. Abstracts submitted to the annual ESC Congress**



This year we will again be holding four Award sessions, in which the best science from young investigators under the age of 36 will be competitively presented. These sessions will all take place on Sunday, 29 August 2004, from 12:40 to 13:55 and we are looking forward to a large audience. Attractive prizes for the winning abstracts in Basic, Population, Clinical Sciences and Thrombosis are given by the ESC at the Awards Ceremony.

In line with the general theme of the Congress, “Diabetes & the Heart”, many abstract sessions will be devoted to basic and clinical aspects of this growing epidemic, not to mention the special EASD-ESC scholarship session, to be held on Tuesday, 31 August 2004, from 11:00 to 12:30. These outstanding contributions (half of which are selected by each organization) will be presented at both ESC and EASD meetings.

Abstract selection is based on expert and unbiased peer review, resulting in the final selection of 2680 abstracts to be included in one of the 124 oral sessions or 1449 poster presentations. Expert colleagues from Europe and abroad grade the abstracts on-line, with the help of customized web-based tools. The abstracts are blinded for the author’s names, city and country of origin. The average grade given by these experts was used to select the best abstracts for further evaluation by members of the Congress Programme Committee. We would like to extend our sincere thanks to all the members of the

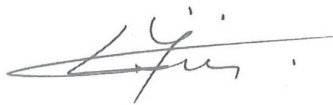


Congress Programme Committee and to the 543 abstract graders for their dedication and enthusiastic work in reviewing the submissions.

It is important to note that selection of an abstract for oral or poster presentation does not reflect the grades this abstract received. Oral sessions are constructed by the Programme Committee to present related work covering one particular topic. Thus, the poster sessions feature some of the highest graded abstracts of the congress, in particular the Moderated Poster sessions. These posters will be presented and discussed in a specially designed and highlighted area of the poster hall. Using microphone support, scientists will have the opportunity to present and defend their findings in front of some of the most prestigious experts of our discipline.

In addition, this year we will introduce an entirely new concept, namely the e-Poster sessions, whereby the 464 posters selected for the topics Basic Science & Cardiac Imaging will be available in electronic format for the entire duration of the congress through a specially dedicated computer system (e-Poster lounge of 40 computers located in the Poster hall close to the FOCUS rooms). This technology has a lot to offer to stimulate interaction and maximize exposure of the data. Not only can you discuss with the poster presenter during his allotted timeslot, but any participant in the Congress can drop in whenever convenient and visualize any of the e-Posters, alone or in company. Facilities for presentation to small groups via projection on larger plasma screens can be made available. Computer facilities allow for the inclusion of video clips, movies or other animations in the material to be presented, and there are many other useful features to be discovered . . . Look for all necessary information about this exciting new approach to scientific exchange on the ESC website, [www.escardio.org](http://www.escardio.org).

To all organizers, participants and accompanying persons, we extend a warm welcome. We hope that you will enjoy the conference, both scientifically and socially.



WILLIAM WIJNS  
*Chairman*  
*ESC Congress Programme Committee*



JEAN-PIERRE BASSAND  
*President*  
*European Society of Cardiology*



### 218 Inhaled treprostinil is a potent pulmonary vasodilator in severe pulmonary hypertension



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**Background:** Treprostinil has been approved for therapy of PAH (US and Canada) as continuous subcutaneous infusion. However, local pain at the infusion site is a major drawback. Inhaled therapy with another stable prostacyclin analogue (iloprost) has been approved for PPH (EMA). In this study we investigated the acute hemodynamic response to inhaled treprostinil.

**Methods:** Open-label, single blind placebo-controlled clinical study. After placement of a Swan-Ganz catheter and a femoral artery line, patients inhaled solvent solution (placebo) (n=8) or treprostinil for 6 min (OptiNeb ultrasound nebulizer, Nebu-tec, Germany) in concentrations of 16, 32, 48, and 64 µg/ml (n=6, 6, 6, and 3 patients). Measurement was performed before and after 0, 15, 30, 60, 90, 120, 150 and 180 min. The mean area between the placebo and the treprostinil curves (ABC186) was calculated (baseline=100%).

**Results:** We investigated idiopathic PAH (n=10), collagen vascular disease (n=5), chronic thromboembolic disease (n=9), and pulmonary fibrosis (n=5), f/m = 19/10, age 56 ± 3 years, PAP, PAWP, and CVP 51.3 ± 2.2, 9.2 ± 0.8, and 6.6 ± 0.6 mmHg, CO 4.4 ± 0.3 l/min, SvO<sub>2</sub> 62.3 ± 1.2%, PVR 885 ± 72 dyn cm s<sup>-5</sup>. At 16 µg/ml there were no significant adverse events. Headache, cough or bronchoconstriction were observed in 2, 1, and 2 patients at 32, 48, and 64 µg/ml. These were mild and transient in all patients but one (64 µg/ml) who complained of major headache for 1 hour. Placebo inhalation was followed by slowly increasing PVR. Compared to this, the maximum treprostinil effect was reached after about 50 min and half-maximal effects at about 110 min. The ABC186 for PVR was -24.7 ± 4.4, -28.7 ± 4.9, and -29.0 ± 4.7%; PAP -14.4 ± 3.3, -13.5 ± 5.2, -13.1 ± 2.6%; SAP -5.1 ± 3.0, -6.0 ± 3.1, -3.8 ± 2.1% at 16, 32 and 48 µg/ml.

**Conclusion:** Treprostinil inhalation results in a significant long-lasting pulmonary vasodilatation. With the applied technology, at a concentration of 16 µg/ml, near maximal pulmonary vasodilatation is achieved without adverse effects. At higher doses, local and systemic side effects may occur, whereas pulmonary selectivity is preserved.

This study was supported by Lung Rx.

### 219 The endothelin-receptor antagonist bosentan for the treatment of pulmonary arterial hypertension associated with congenital heart defects



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**Background:** Treatment with the oral dual endothelin-receptor antagonist bosentan has been shown to be an effective alternative option to intravenous epoprostenol in functional class (FC) III idiopathic pulmonary arterial hypertension (PAH) patients. In patients with PAH associated with congenital heart defects (CHD), an improvement of exercise capacity and hemodynamics has been demonstrated with epoprostenol in one uncontrolled study (Rosenzweig et al. *Circulation* 1999; 99: 1858-65).

The aim of this retrospective study was to evaluate the efficacy and safety of bosentan in FC III-IV CHD-PAH patients.

Study population consisted in 24 patients (22 females, mean age 35 ± 15 years [8-68]) with CHD-PAH: atrial septal defect (ASD: 13), ventricular septal defect (VSD: 4), partial abnormal pulmonary venous return (3, associated with ASD in 2 and repaired common atrium in 1), patent ductus arteriosus (PDA: 2), VSD associated with PDA (1), aortopulmonary window (1). Four patients had undergone previous cardiac surgery.

Patients had deteriorated despite conventional therapy (including oral anticoagulants, oxygen, diuretics) and were treated with chronic oral bosentan.

**Results:** Before starting bosentan, 22 patients were in FC III and 2 in FC IV, with a resting O<sub>2</sub> saturation (SaO<sub>2</sub>) of 89 ± 9%. Mean 6-min walk distance (6MWD) was 288 ± 94 m and mean Borg index 3.0 ± 1.9. At last evaluation performed after 10 ± 9 months of bosentan treatment, 1 patient was in FC I, 8 were in FC II, 13 remained in FC III and 2 in FC IV. The mean 6MWD improved by 49 m (349 ± 85 m, p = 0.008) with no change in Borg index (3.0 ± 1.8) and resting SaO<sub>2</sub> (89 ± 6%). There were no differences between pre and post-tricuspid shunt subgroups in terms of baseline characteristics and response to bosentan therapy. After 13 ± 9 months of follow-up, all patients are alive on bosentan, but 3 (1 ASD, 1 VSD, 1 aortopulmonary window) required combination therapy with intravenous epoprostenol after 5, 7 and 9 months on bosentan.

**Conclusion:** Chronic oral bosentan treatment improves exercise capacity in patients with PAH associated with CHD who deteriorated despite conventional therapy. Bosentan had no adverse effect on arterial oxygen saturation. As previously demonstrated in patients with idiopathic PAH, long term bosentan may be an important therapeutic option for patients with PAH associated with CHD.

### 220 Sildenafil in the treatment of primary pulmonary hypertension



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**Background:** The role of sildenafil as a pulmonary vasodilator is being extensively evaluated in the treatment of pulmonary hypertension.

**Aim:** This was a prospective study to assess the benefit of adding sildenafil in patients with high pulmonary artery pressures secondary to atrial septal defect (ASD), already receiving the conventional therapy.

**Methods:** Thirty consecutive patients with moderate to severe primary pulmonary hypertension were included in this study. All the patients were diagnosed previously and were receiving the conventional therapy with digoxin, diuretic and a calcium channel blocker. Sildenafil was added in the dose of 50 mg twice a day without changing the previous regimens. Changes in the New York Heart Association (NYHA) symptom class, distance covered during the six minute walk test and modified Borg dyspnea score were evaluated monthly. Acceptance of the new drug was assessed every week in the first month and then at the monthly follow up. Echocardiography and Doppler study was undertaken at baseline and every month for a period of six months. The parameters studied were the pulmonary artery systolic pressure (PASP) by tricuspid regurgitation (TR) jet and pulmonary artery diastolic pressure (PADP) by pulmonary regurgitation (PR) jet.

**Results:** Mean age of the subjects was 42.6 ± 9.3 years. Twenty seven (90%) were females and 3 (10%) were males. Sildenafil was well tolerated and there was no dropout because of undesirable effects of the drug. Changes in the heart rate and systemic blood pressure were not significant enough to warrant withdrawal of the drug. Two patients died during the follow-up period. At the beginning of the therapy, 22 (73.3%) patients were in NYHA Class III or IV while at the end of six months, only 8 patients remained in either of these classes (p < 0.05). By the 6 min walk test, functional capacity improved from 181.5 ± 122.4 meters to 302.7 ± 150.3 meters p < 0.05. Modified Borg dyspnea score improved from 5.6 ± 1.2 to 3.3 ± 1.1 (p < 0.05). PASP by TR jet (mmHg) was down from 81.3 ± 14.7 to 51.4 ± 11.7 (p < 0.05). PADP by PR jet (mmHg) reduced from 56.2 ± 11.7 to 33.4 ± 9.1 (p < 0.05).

**Conclusion:** Sildenafil is well tolerated, improves symptoms, and reduces the systolic and diastolic pulmonary artery pressures in patients with moderate to severe primary pulmonary arterial hypertension.

### REVISITING THE ELECTROCARDIOGRAM AND ELECTROPHYSIOLOGIC MARKERS OF ARRHYTHMIC EVENTS

#### 221 Prevalence of brugada-type ecg in an apparently healthy european population



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**Background:** The Brugada Syndrome ECG is characterized by ST-segment elevation in right precordial leads and elevated risk of lethal arrhythmias in absence of identifiable structural heart disease. Few data are available on the Brugada type ECG, especially in Europeans. No epidemiological study has applied the diagnostic criteria recently proposed by the Study Group of the Molecular Basis of Arrhythmias of the ESC.

**Methods:** We analysed the ECG and clinical data of apparently healthy European adults undergoing routine medical examinations for occupational reasons. At each examination subjects underwent a medical interview, physical examination, blood pressure measurement and 12-lead ECG. Enrolment was confined to persons without a history of heart disease at the time of first attendance in whom at least one 12-lead ECG of good quality was recorded. The ECG records of all 7483 subjects (89.6% male, age 29.5 ± 10.8 years at first attendance) were reviewed by three cardiologists. We reviewed 1,97 ± 2,1 ECGs for each subject. We considered a patient having a Brugada ECG pattern if 2 or more of the cardiologists judged that at least one of that persons ECGs fulfilled the criteria of the ESC Study Group.

**Results:** The Brugada pattern was present in 26 patients (0.35%), all male (table). In 17 cases (65.4%), information was available about the progress of the subject subsequent to the ECG on which the Brugada pattern was first recorded. No sudden death or cardiac arrhythmia was recorded among these patients in a follow-up of 5.2 ± 4.6 years (total follow-up 87.8 patient-years).

	Total	Pattern 1	Pattern 2	Pattern 3	Tot. Brugada
Pts (n)	7383	2	21	3	26
Male	6618	2	21	3	26
Female	765	0	0	0	0
Male prevalence (*10000)	-	3,02	31,73	4,53	39,29
Total Prevalence (*10000)	-	2,71	28,44	4,06	35,22