UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 10-K

(Mark One)

[X] Annual Report Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934
For the Fiscal Year Ended December 31, 1998

or

[ ] Transition Report Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934
Commission File Number: 0-19171

ICOS CORPORATION
(Exact name of registrant as specified in its charter)

Delaware	91-1463450
(State of incorporation)	(I.R.S. Employer Identification No.)

22021 - 20th Avenue S.E.
Bothell, Washington 98021
(425) 485-1900
(Address, including zip code, and telephone number, including area code, of principal executive offices)

Securities registered pursuant to Section 12(b) of the Act: None
Securities registered pursuant to Section 12(g) of the Act:
Common Stock, $.01 par value

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports) and (2) has been subject to such filing requirements for the past 90 days.

Yes X No

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. [X]

State the aggregate market value of voting and non-voting stock held by non-affiliates of the registrant as of March 26, 1999.

$1,489,109,168

Indicate the number of shares outstanding of each of the registrant's classes of Common Stock as of March 26, 1999.

Title of Class	Number of Shares
Common Stock, $.01 par value	42,692,350

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the Registrant's definitive Proxy Statement for the annual meeting of stockholders to be held on May 6, 1999 relating to "Election of Directors," "Continuing Directors (until 2000)," "Continuing Directors (until 2001)," "Other Executive Officers," "Compliance with Section 16(a) of the Securities Exchange Act of 1934," "Compensation of Directors," "Executive Compensation," "1998 Option Grants," "1998 Option Exercises and Year-end Option Values," "Compensation Committee Interlocks and Insider Participation," "Employment Contracts, Termination of Employment and Change of Control Arrangements," "Security Ownership of Certain Beneficial Owners and Management," and "Certain Relationships and Related Transactions" are incorporated by reference in Part III of this Form 10-K.

ICOS CORPORATION

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PART I

Item 1. Business

Overview

ICOS Corporation ("ICOS" or the "Company"), formed in 1989, is developing proprietary biopharmaceuticals and small molecule pharmaceuticals for the treatment of inflammatory diseases and other serious medical conditions.

The Company's fundamental strategy is to identify and develop a significant number of potential product candidates into breakthrough products with high commercial potential. By understanding the underlying biochemical and physiological mechanisms and identifying the cellular and molecular entities involved in the disease process, ICOS is developing biopharmaceutical products that address important opportunities in the treatment of chronic and acute diseases that have inflammatory components as well as certain cardiovascular diseases and cancer. Through this strategy, the Company believes it will be able to develop novel therapeutics that are more selective and efficacious than current therapeutics.

When used in this discussion, the words "believes," "intends," "anticipates," "plans to" and "expects" and similar expressions are intended to identify forward-looking statements. Such statements are subject to certain risks and uncertainties that could cause actual results to differ materially from those projected. See "Important Factors Regarding Forward-Looking Statements." Readers are cautioned not to place undue reliance on such forward-looking statements, which speak only as of the date hereof. The Company undertakes no obligation to publicly release the results of any revisions to such forward-looking statements that may be made to reflect events or circumstances after the date hereof or to reflect the occurrence of unanticipated events.

Long-Range Strategy

The Company continues to develop a broad portfolio of potential product candidates encompassing a wide variety of approaches to inflammatory conditions and other serious diseases. Human disease is a complex and complicated process involving many physiological and biological components. As such, the task of developing therapeutics to treat these diseases is difficult and time-consuming. A large number of potential product candidates are not successfully developed because of the inability to prove that they are either safe or efficacious. In order to compensate for this risk, the Company utilizes a strategy of developing a number of approaches to the treatment of inflammatory conditions which encompass a variety of mechanisms and approaches to the inhibition of inflammation and other disease processes. Presently, the Company and its affiliates have five product candidates in clinical trials including three monoclonal antibodies: LeukArrest(TM), ICM3 and IC14, Pafase(TM), a recombinant form of a naturally occurring human enzyme, and IC351, a small molecule product candidate. In addition, the Company has several product candidates in the research and preclinical phases of development. In 1998 the Company established commercial names for two of its product candidates: LeukArrest(TM) is the name presently given to the product candidate formerly known as Hu23F2G, a monoclonal antibody developed by ICOS to block leukocyte cell adhesion in humans, and Pafase(TM), formerly known as rPAF-AH, a potent proinflammatory mediator which is a naturally occurring human enzyme that destroys platelet-activating factor and eliminates its proinflammatory effects. Over the past few years the Company has established certain corporate collaborations to enhance and optimize the Company's development while maintaining substantial downstream product rights to potential products and offsetting a substantial portion of the financial risk of development of these product candidates. Most recently, during 1998 the Company established Lilly ICOS LLC ("Lilly ICOS"), a joint venture with Eli Lilly and Company ("Lilly"), to develop the small molecule product candidate known as IC351 for the treatment of sexual dysfunction. This is in addition to the Company's joint venture Suncos Corporation ("Suncos") established in 1997 with Suntory Limited of Japan ("Suntory") for the development of Pafase(TM) and a collaboration with Abbott Laboratories in the field of integrins and ICAMs. Each of these collaborations is described in more depth in the section entitled "Collaborations."

Description of Programs

Development Pipeline - Overview

The clinical targets that are the subject of ICOS' discoveries include inflammation and other diseases whose pathology is a result of the dysfunction of the normal cellular mechanisms. The Company has discovered important molecules and mechanisms underlying directed cell adhesion, the inhibition of proinflammatory mediators and intracellular signal transduction. The chart below summarizes the programs with compounds currently in clinical development.

ICOS Clinical Development Projects
(Table 1)

Product Candidate	Indication	Status (1)
LeukArrest(TM)	Ischemic stroke	Phase 3 clinical trial
	Hemorrhagic shock	Phase 2 clinical trial
	Myocardial infarction	Phase 2 clinical trial
	Multiple sclerosis, acute exacerbation	Phase 2 clinical trial
ICM3	Severe psoriasis	Phase 1/2 clinical trial
IC14	Severe sepsis	Phase 1 clinical trial
Pafase(TM)	ARDS	Phase 2 clinical trial
	Acute pancreatitis	Phase 2 clinical trial
	Post-ERCP pancreatitis	Phase 2 clinical trial
IC351	Male erectile dysfunction	Phase 2 clinical trial

(1) Status as of March 31, 1999
Phase 1 clinical trial: safety and pharmacology, dose-determining drug regimen
Phase 2 clinical trial: determination of dose levels and potential efficacy of drug
Phase 3 clinical trial: efficacy and safety determination

LeukArrest(TM)

Background

The migration of circulating leukocytes into extravascular tissues in the course of inflammation involves a complex series of events. A critical step involves the firm attachment of circulating leukocytes to the endothelial wall. The CD11/CD18 family of cell adhesion molecules found on leukocytes mediate this adhesive interaction. It is believed that by intervening in the adhesion process, much of the inflammation-associated damage can be prevented. Monoclonal antibodies directed to CD11/CD18 adhesion molecules have been shown to protect against leukocyte-mediated tissue injury by blocking adherence in a variety of disease models.

LeukArrest(TM) is a recombinant humanized monoclonal antibody developed by ICOS to block CD11/CD18-mediated cell adhesion in humans. LeukArrest(TM) has been shown to bind to CD11/CD18 cell adhesion molecules on the surface of leukocytes and to block subsequent movement into the surrounding tissue. To date, approximately 850 subjects have been enrolled in clinical trials of LeukArrest(TM) investigating its use as a therapeutic for the treatment of ischemic stroke, hemorrhagic shock, myocardial infarction and multiple sclerosis. These trials are designed to gather safety, efficacy and pharmacological data to support further development of the program. ICOS is conducting clinical development of LeukArrest(TM) for the indications described below.

Clinical Application - Ischemic Stroke

During an ischemic stroke a blood vessel in the brain becomes blocked and blood flow to a region of the brain is reduced. This ischemia results in injury and death of the affected tissue. Although the stroke event arises from the blockage of one or more cerebral blood vessels by a blood clot, a significant portion of the tissue injury and death is thought to be caused by neutrophil-mediated inflammatory mechanisms. Restoring blood flow, oxygen and leukocytes, in particular neutrophils, to these tissues results in activation and adhesion of neutrophils to the endothelium. Once attached to the endothelial lining

these activated neutrophils release toxins, such as free radicals and proteases, that damage the endothelium and the surrounding tissue. Data from preclinical studies has indicated that LeukArrest(TM) inhibits neutrophil functions shown to be important for neutrophil-induced damage. The Company believes that treating patients who have suffered a stroke with LeukArrest(TM) may limit the degree of inflammatory tissue damage and protect significant amounts of brain and CNS tissue and, thus, may decrease the extent of brain damage for these patients.

This molecule showed safety and efficacy in a randomized double-blind placebo controlled parallel study. Data from this study suggests that high dosages of LeukArrest(TM) improved neurological outcomes and exhibited a higher treatment response rate than did placebo. LeukArrest(TM) is currently being evaluating in a Phase 3 trial for ischemic stroke. This trial is designed to treat approximately 800 subjects in a randomized double-blind placebo controlled parallel study to evaluate its efficacy.

Clinical Application - Hemorrhagic Shock

Each year, approximately 150,000 Americans suffer major trauma and associated blood loss, leading to shock. Approximately 125,000 of these victims are at risk for the development of hemorrhagic shock. A major cause of morbidity and mortality in those who survive the initial injury is multiple organ dysfunction, for which there is no specific treatment. The intensive care necessary for the support of these patients is extremely expensive.

Based on in vitro and in vivo data, it has been hypothesized that multiple organ dysfunction is the result of neutrophil-mediated tissue injury. Resuscitation of the trauma patient by medical staff administering intravenous fluids and blood products leads to the re-establishment of circulation in the affected tissues. Restoring blood flow, oxygen and leukocytes, in particular neutrophils, to these tissues results in activation and adhesion of neutrophils to the endothelium. Once attached to the endothelial lining these activated neutrophils release toxins, such as free radicals and proteases, that damage the endothelium and the surrounding tissue. The consequences of this include edema, hemorrhage and thrombosis that can often result in organ dysfunction and ultimately organ failure.

Since the adhesion of neutrophils to endothelial cells is inhibited by LeukArrest(TM), ICOS believes that treatment of trauma-induced hemorrhagic shock patients with LeukArrest(TM) may prevent the development of multiple organ dysfunction and improve overall mortality rates.

LeukArrest(TM) is being evaluated in a randomized double-blind placebo controlled parallel group Phase 2 study to evaluate LeukArrest(TM) for the treatment of severe tissue damage related to severe trauma. The trial is designed to evaluate the molecule's efficacy in reducing fluid requirements and organ failure in patients that have suffered massive blood loss due to traumatic injury. ICOS believes that blockage of leukocyte movement into tissues after re-establishment of fluid levels may prevent resulting tissue damage and resulting organ failure.

Clinical Application - Myocardial Infarction ("MI")

Each year, approximately 1.5 million myocardial infarctions occur in the United States, resulting in significant morbidity and mortality. During an MI, a coronary artery becomes blocked, impairing blood flow to a region of the heart and damaging surrounding tissue. A significant portion of the tissue injury and death is thought to be caused by neutrophil-mediated inflammatory mechanisms. Current treatment of MI is unable to protect at-risk tissue from this neutrophil-mediated damage.

A common and serious complication of MI is the failure of the heart to pump blood adequately. Generally, the larger the amount of tissue damage, the less able the heart is to pump blood, resulting in congestive heart failure, which is the major cause of in-hospital mortality and disability following MI.

Preclinical studies have provided evidence that LeukArrest(TM) inhibits neutrophil functions shown to be important for neutrophil damage in models of MI. ICOS believes that treating patients with LeukArrest(TM) during an MI may limit the degree of inflammatory tissue damage and protect significant amounts of heart tissue. In turn, this tissue preservation should help maintain the pumping capacity of the heart, thereby reducing mortality and disability.

LeukArrest(TM) is currently being evaluated for its use in preventing tissue damage after myocardial infarction or heart attack, in an acute setting. Initially, the molecule's safety and pharmacology profile was tested in a randomized double-blind placebo controlled parallel Phase 2 study involving 60 patients at 18 sites. Results of this initial study indicated that both

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