

(Mark One)

Annual report pursuant to Section 13 or 15(d) of the Securities
Exchange Act of 1934 for the Fiscal Year ended July 31, 1996

OR

Transition report pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934 for the transition period from
_____ to _____

Commission file number 0-20772

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

California
(State or other jurisdiction of incorporation or organization)

2714 Loker Avenue West, Carlsbad, California 92008
(Address of principal executive offices)

33-0476164
(I.R.S. Employer Identification No.)

Registrant's telephone number, including area code:
619) 929-9500

Securities registered pursuant to Section 12(b) of the Act: None
Securities registered pursuant to Section 12(g) of the Act: Unit
(Title of class) Common Stock, no par value
(Title of class)
Redeemable Class "A" Warrant (Title of class) Redeemable Class "B"
Warrant
(Title of class)

Indicate by mark whether the Registrant (1) has filed all reports
required to be filed by Section 13 or 15(d) of the Securities
Exchange Act 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 NO

As of October 21, 1996, the Registrant had 11,613,748 shares of Common
Stock, no par value, outstanding, and the aggregate market value of
the shares held by non-affiliates on that date was \$34,706,916 based
upon the last sales price of the Registrant's Common Stock reported
on the National Association of Securities Dealers, Inc. Automated
Quotation National Market System.*

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 the 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.

* Excludes 2,937,019 shares of Common Stock held by directors,
executive officers and shareholders whose beneficial ownership
exceeds ten percent of the shares outstanding on October 21, 1996.
Exclusion of shares held by any person should not be construed to
indicate that such person possesses the power, direct or indirect, to
direct or cause the direction of the management or policies of the
Registrant, or that such person is controlled by or under common
control with the Registrant.

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the Registrant's Proxy Statement for the 1997 Annual
Meeting of Shareholders to be filed on or before November 28, 1996
are incorporated by reference into Part III.

Item 1. Business.

Except for the historical information contained herein, the following discussion contains forward-looking statements that involve risks and uncertainties. The Company's actual results could differ materially from those discussed here. Factors that could cause or contribute to such differences include, but are not limited to, those discussed in the description of the Company's business below and the sections entitled "Licenses", "Manufacturing", "Sales and Marketing", "Competition", "Government Regulation", "Patents and Proprietary Rights" and "Management's Discussion and Analysis of Financial Condition and Results of Operations", those discussed in the S-3 Registration Statement File No. 333-3507 filed with U.S. Securities and Exchange Commission, as well as those discussed in any documents incorporated by reference herein or therein.

General

The Company is developing and marketing acute care drugs for the hospital market. On August 9, 1995, it acquired its first marketable products, Glofil and Inulin, two injectable drugs that assess kidney function by the measurement of glomerular filtration rate ("GFR"). It is also currently conducting six (6) Phase II clinical trials on CPC-111 and CPC-211, two drugs potentially indicated for acute care of a number of major disorders caused by impaired blood flow, known as "ischemia". These disorders include heart attack and ischemic heart disease, surgically-induced ischemia, sickle cell anemia crises, acute respiratory distress syndrome ("ARDS") and closed head injury and the drugs are covered by U.S. patents. During the year, the Company made two interim releases of statistically significant data from 55 patients in its Phase II clinical trial of CPC-111 in coronary artery bypass grafting surgery patients which showed that CPC-111 is acting as a cardioprotective drug. (See Clinical Programs on page 8).

Kidney Disease and the Company's Acquired Products. Kidney disease afflicts more than 2,000,000 Americans and results in over \$12 billion annually in healthcare costs in the United States. The Company believes that kidney disease can be reduced if identified at an early stage by the monitoring of renal function. Glofil and Inulin are products cleared by the U.S. Food and Drug Administration ("FDA") for monitoring renal function by determining GFR. Glofil is more accurate than its principal competitor, creatinine clearance, in the measurement of GFR for the estimated 500,000 patients with severe kidney disease and the Company is targeting this segment of the market. Nephrologists and nuclear medicine departments at major medical centers are the primary end users of these products. During the fiscal year ended July 31, 1996, the Company established a field sales force and customer service function, obtained the appropriate licenses for its own distribution facility in Carlsbad, California and Glofil and Inulin reached sales of \$1,275,000 after the Company acquired them.

Ischemia-Induced Cell Damage and the Company's Phase II Clinical Programs. Similarly, there are approximately 4,000,000 cases of ischemia-induced disorders annually in the United States, resulting in over 900,000 deaths and an estimated \$200 billion in annual costs for physical and mental rehabilitation and ongoing care, and yet there are currently no FDA-approved drugs to avoid or reverse the massive cell damage caused by ischemia (termed "cytoprotective drugs"). Currently approved drugs for treating cardiovascular ischemia, such as "clot busting" drugs, serve to re-establish blood flow but do not have direct cytoprotective benefits. The Company believes that the drugs it is developing, if cleared by the FDA and successfully marketed, should reduce significantly the number of fatalities and the rehabilitation and ongoing care costs associated with ischemic disorders.

Impairment of blood flow reduces the supply of oxygen to body cells, interrupting normal aerobic metabolism and causing depletion of adenosine triphosphate ("ATP"), the cells' primary energy source. Ischemia-induced depletion of ATP produces a myriad of increasingly destructive cellular events known as the "toxic ischemic cascade". The Company believes that all cytoprotective drugs under development by others for treatment of ischemia are focused on treating specific elements of the toxic ischemic cascade, leaving other elements free to cause cell, tissue and organ damage.

The Company's approach, based on preventing or reversing the toxic ischemic cascade, is comprehensive in nature and, the Company believes, potentially more effective. CPC-111 and CPC-211 are designed to act during and after ischemia by maintaining cellular ATP levels or accelerating their restoration. CPC-111 (a natural substance) and CPC-211 have very low orders of toxicity, making them more amenable to being used early in the patient management process, which is critical in acute care settings.

Further, CPC-111 and CPC-211 are small molecules, easily deliverable and inexpensive to produce. Extensive pre-clinical data shows desirable therapeutic effects in animal models of the targeted diseases. Significant human data, available from the Company's own studies and independent, physician-sponsored Investigational New Drug applications ("INDs"), show that each of these drugs is well tolerated when administered at clinically relevant doses to healthy subjects. The minimal side effects associated with CPC-111 and CPC-211 will greatly reduce their development risk and may permit their broad, early use in acute care settings, such as ambulances and emergency rooms, where rapid access to treatment is of utmost importance.

During the fiscal year ended July 31, 1994, the Company filed INDs with the FDA on CPC-111 to treat ischemic heart disease and on CPC-211 to treat acute stroke and began funding a Phase II trial of CPC-211 in closed head injury patients and continued a Phase II trial on CPC-211 in stroke patients. In ARDS patients under a physician-sponsored IND. During the year, the Company also began single-center Phase I clinical trial on CPC-211 in September 1994 and will begin a dose-ranging Phase II clinical trial on CPC-111 in sickle cell anemia crises patients and one in angioplasty patients and continued Phase II trials on CPC-111 in congestive heart failure patients and in cardiac bypass grafting surgery patients. The Company is also planning additional Phase II trials with CPC-111 in sickle cell crisis patients and in heart surgery patients. The Company's strategy is to reduce the risk associated with drug development by selecting for large-scale pivotal trials the clinical indication(s) showing the best Phase II results from the multiple small-scale Phase II trials on CPC-111 and CPC-211 that the Company has underway. The Company intends to begin Phase III trials of CPC-111 and CPC-211 during the fiscal year ending July 31, 1997.

Pre-clinical Programs. Further implementing its overall strategy of developing drugs that protect cells from ischemic damage, the Company is conducting pre-clinical studies on a number of additional drugs meant to reduce the neurodegeneration associated with stroke and traumatic head injury. The Company believes that these drugs will reduce "excitotoxicity", the excess release of excitatory amino acid ("EAA") neurotransmitters in the brain that stems from ischemically-caused ATP depletion in certain brain cells. Drugs being developed in these studies include: (i) a new class of neuronal calcium channel blockers which block excessive EAA neurotransmitter release; and (ii) a patented series of novel compounds which augment levels of adenosine (a naturally occurring substance which inhibits EAA release) in ischemic tissue by inhibiting its metabolism. During the year, the Company also received a \$100,000 Small Business Innovation Research Phase I grant to develop a prodrug for CPC-111. This program was recently initiated with the goal of producing CPC-111 prodrugs with improved pharmacokinetic properties.

Acquired Products for the Hospital Market

The Company's strategy includes building near-term sustainability with the cash flow from acquired acute care products for the hospital market with the goal of reducing its overall cash consumption rate and building its sales, marketing and distribution infrastructure in advance of FDA clearance of CPC-111 and CPC-211.

Glofil and Inulin

Kidney disease afflicts more than 2,000,000 persons in the United States and is increasing primarily due to the growth in diabetes and lupus cases. Kidney disease results in over \$12 billion annually in healthcare costs in the United States. The Company believes that more accurate diagnosis and monitoring of renal disease will aid in the management of the primary causes of end stage renal disease.

Glofil-125 and Inulin, two injectable products acquired by the Company on August 9, 1995, are FDA-cleared products for the measurement of renal function. Nephrologists and nuclear medicine departments at major medical centers are the primary users of these

products. During the fiscal year ended July 31, 1996, the Company recorded sales of \$1,275,000 from the sale of Glofil-125 and Inulin. Two customers using Glofil-125 for long-term research studies accounted for 39% and 15%, respectively, of net sales of these products.

Glofil-125 is an injectable radioactive diagnostic drug, which provides rapid information on GFRs with great accuracy. This is an established product that is covered by an FDA-cleared New Drug Application, and is currently sold by the Company in 4ml vials and in prefilled syringes through the 117 nationwide radiopharmacies of Syncor International pursuant to a distribution agreement entered into with the Company in February 1996. Inulin is an injectable diagnostic drug, which provides a measure of GFRs. Inulin is currently sold in 50 ml ampules with actual patient dosing correlated to patient weight.

The Company believes there is substantial opportunity for increased utilization of Glofil-125. Present diagnostic procedures for measuring kidney function include serum creatinine and creatinine clearance tests. These two tests are the most commonly performed methods of measuring kidney function because they are cheaper tests, however both methods significantly overestimate kidney function in the estimated 500,000 patients severe renal disease. The use of Glofil-125 is a more direct, true measure of kidney function yielding accurate results. This improved accuracy can be essential to reliably monitoring disease progression and intervention, as well as assessing the immediate state of renal impairment. The biggest impediment to the continued growth in the sales of Glofil-125 would be a change in the ability of the end users to obtain reimbursement for the test or the unforeseen termination of the research studies being conducted by the Company's two largest customers.

Inulin, which is sold by the Company, and 99m Tc-DTPA (which is not sold by the Company) are alternative agents for GFR measurement, however the preparation and use of these two drugs is difficult and they do not provide the practical advantages of Glofil-125. There are no new diagnostic drugs being introduced or in development that the Company is aware of as a competitive threat to Glofil-125.

Cytoprotection Market Opportunities

Cytoprotective drugs for acute care settings that treat ischemic injury are not currently available and the market opportunities for the Company's drugs are large, totalling approximately 4,000,000 cases annually in the United States. The Company believes that its drugs, if approved, could substantially reduce not only the 900,000 fatalities associated with ischemia-related disorders but also reduce significantly the more than \$200 billion annual cost of rehabilitation and ongoing care in the United States.

The Company's drugs are designed to be administered intravenously in order to speed their delivery to the ischemic tissue. In order to ensure early interventions, they are intended to be standard components of "crash boxes" in ambulances, hospital emergency rooms, operating theater suites, endoscopy suites and radiology suites. Their lack of substantial toxicity should suit them for this purpose.

Circulatory System Ischemia

According to the American Heart Association and the National Institutes of Health, heart attack and stroke represent the number one (500,000 cases annually) and number three (150,000 cases annually) leading causes of death in the United States, respectively. Heart attack and stroke are the result of ischemia to the heart and brain, respectively.

Cardiovascular ischemia can result in a spectrum of clinically significant events ranging from angina (pain) to heart attack and sudden death. In addition to the numerous trauma or disease related causes of ischemia, there are a variety of voluntary surgical procedures which result in ischemia to vital organ systems. Procedures such as coronary artery bypass grafting surgery and coronary angioplasty, both of which are performed to improve blood flow to the heart, in themselves induce temporary ischemia which can result in tissue damage. Thus, CPC-111, if approved, could also be a part of the treatment regimen for these disorders. All of these conditions or procedures represent potential opportunities for use of the Company's drugs to reduce the tissue damage known to be associated with them.

Cerebrovascular ischemia (stroke) can result in temporary loss of consciousness, permanent behavioral and neurologic impairment, coma and death. Traumatic injury to the head is caused by accidents, near drownings and similar incidents. The resultant medical problems are, in large part, caused by ischemia to the brain. The biochemical processes associated with stroke and head trauma are thought to be very similar; thus, drugs developed for one indication are expected to be useful for the other.

Sickle Cell Anemia

Sickle cell anemia is an autosomal recessive genetic disease carried by about 8% of African-Americans. Approximately 60,000 African-Americans suffer from the most severe form (homozygous) of the disease, termed sickle cell "crisis", where the red blood cells form "sickle" shapes that can clog up capillaries and result in severe and disseminated ischemia, and undergo multiple crises each year. CPC-111 has been shown pre-clinically to prevent this "sickling" process and the Company is intends to evaluate it in a Phase II trial of sickle cell anemia crisis patients.

The Pathology of Ischemia

Metabolic Aspects (General, All Tissues)

All living animal cells require glucose and oxygen to survive, both of which are supplied to tissues by the blood. Glucose is transformed into carbon dioxide and water with the resultant formation of ATP. ATP is the universal fuel which is required to keep the cell alive. During and after ischemia, the decrease in cellular ATP levels damages the cell and, the Company believes, results in the toxic ischemic cascade, a myriad of cell-damaging processes discussed below which cause further cell damage.

ATP generation occurs in two phases. The first phase, called glycolysis or anaerobic metabolism does not require oxygen. The second phase called aerobic metabolism requires oxygen and occurs in mitochondria. Glycolysis is a means of producing cellular energy in ischemic conditions, and therefore, represents the body's natural defense against ischemic damage. For this reason, the facilitation of glycolysis is of interest therapeutically in the prevention of ischemic damage to tissues and organs. When pyruvic acid builds up during ischemia due to the inability of aerobic metabolism to utilize it, an enzyme converts it to lactic acid which blocks glycolysis. The therapeutic principle underlying CPC-111 and CPC-211 is to facilitate glycolysis during and after ischemia so the cell continues to produce ATP and the toxic ischemic cascade is pre-empted or reversed. Specifically, CPC-111 bypasses the lactic acid block and does not need to be energized by ATP to be metabolized. CPC-211 reduces ischemia induced lactic acid accumulation and therefore allows energy metabolism to continue.

Excitotoxicity (Nerve Tissue)

The destructive impact of ATP depletion in nerve tissue is further complicated by the over-production in nerve cells of various excitatory amino acids, chemicals that transmit nerve impulses from one nerve cell to another. The over-production and release of EAAs (predominately glutamate and aspartate) by nerve cells exposed to ischemia over-stimulates adjacent postsynaptic nerve cells, causing them in time to succumb to metabolic exhaustion and cell death. This ischemia-induced process, called delayed excitotoxicity, is associated with a number of acute (stroke and traumatic head injury) and chronic (Alzheimer's, Parkinson's Disease and Amyotrophic Lateral Sclerosis) neurologic disorders. Controlling delayed excitotoxicity by blocking the postsynaptic EAA receptors has recently attracted the attention of both academic and pharmaceutical scientists. The drugs in development that act by this mechanism to date have considerable side effects and only block selected receptor subtypes, therefore, only dealing with part of the problem since all receptor subtypes appear to cause damage.

Recent evidence has shown that specific presynaptic channels, neuronal calcium channels, regulate the release of neurotransmitters in nerve cells. The Company has shown that compounds which block excessive EAA neurotransmitter release from nerve cells greatly reduce excitotoxicity and post-ischemic tissue damage in animal models of stroke and head trauma. The Company is seeking to develop drugs that specifically block neuronal calcium channels and therefore, if successful, would block the excitotoxic process and

reduce the resultant cell damage. These drugs are expected to have a more comprehensive effect on excitotoxicity than the specific postsynaptic EAA receptor blockers, since they will reduce the stimulation of all and not just some EAA receptors. See "Neuronal Calcium Channel Blocker Program."

The Company has also shown that adenosine, a natural compound, has cytoprotective properties. The Company is seeking to develop a series of drugs, called adenosine metabolism inhibitors, which, if successful, would augment adenosine levels in ischemic tissue and have cytoprotective effects in both brain and heart tissue. See "Adenosine Metabolism Inhibitor Program."

The Toxic Ischemic Cascade

Ischemia-induced cell damage triggers a number of processes which cause further damage to each affected cell and its surrounding cells. This myriad of destructive processes is facilitated by reperfusion injury, which occurs after blood flow is re-established. The traumatized, ATP-depleted cell enters into the toxic ischemic cascade, resulting in the release of a host of toxic agents, including damaging reactive chemicals called free radicals, as well as other molecules that are products of cell membrane breakdown, all of which damage cells. Excessive intracellular calcium buildup is also an element of the toxic ischemic cascade and also triggers a host of other damaging processes such as activation of proteolytic enzymes (enzymes that break down proteins) which digest cells and activation of protein kinases which regulate cell metabolism. The traumatized cell also releases agents which stimulate the immune system, activating various blood cells, such as neutrophils and macrophages which actually eliminate the cell affected by ischemia. Rather than target each of these myriad events, the Company's drugs, CPC-111 and CPC-211, address the issue of ATP replenishment so that the cell can correct the ischemic cascade naturally.

There are currently no known FDA-approved cytoprotective drugs. Those under development are, to the Company's knowledge, primarily aimed at specific elements of the toxic ischemic cascade. The Company believes that its approach to cytoprotective drug development is unique in that it seeks to pre-empt or reverse the entire cascade by decreasing the initial metabolic trauma which triggers it (i.e., ATP depletion). The Company believes that this approach is preferable to treating specific elements of the cascade, since it more comprehensively addresses the underlying pathology and should therefore result in more efficacious therapy.

Ischemia Drugs in Development - The Metabolism Program

The Company is conducting four Phase II trials and has commenced clinical trials on CPC-111 and two Phase II trials on CPC-211. These drugs are designed to minimize the tissue damage associated with cardiovascular ischemia (e.g., congestive heart failure and angioplasty), surgically-induced ischemia, sickle cell anemia crises, ARDS, sepsis and acute cerebral ischemia (specifically, stroke and traumatic head injury). The Company expects to finish these trials during the fiscal year ending July 31, 1997 and also to begin Phase III trials with CPC-111 and CPC-211 in at least one indication each.

CPC-111. CPC-111 is a small non-peptide molecule that the Company believes (based on extensive pre-clinical and mechanistic data) stimulates and maintains glycolysis in cells undergoing ischemia by circumventing the ischemia-induced blockage of this process. The Company has licensed four issued U.S. patents which cover the use of CPC-111 for acute ischemic heart disease (such as acute myocardial infarction ("AMI"), congestive heart failure and unstable angina), surgically-induced ischemia (e.g., CABG surgery), ARDS, sepsis and other ischemic disorders and has several patents pending in the United States and Europe. Pre-clinical studies conducted by various investigators involving this compound in cardiovascular ischemia (including myocardial infarction), sickle cell anemia, septic and hemorrhagic shock and generalized trauma have shown therapeutic benefits as indicated by significant tissue preservation, improved organ function and survival in animal models.

There are published U.S. and foreign clinical studies with CPC-111 indicating that it is well tolerated in humans with little or no side effects. More than 500 patients have been tested with the drug worldwide. These studies indicate that the drug improves heart function in congestive heart failure patients as well as in other situations where the heart is injured.

In December 1995 and in August 1996, the Company released data from its Phase II trial with CPC-111 in coronary artery bypass grafting surgery patients. Stage 1 data comprised 10 active- and 10 placebo-treated patients with the former receiving 250 mg/kg of CPC-111 prior to the surgery. Stage 3 data comprised 15 active- and 15 placebo-treated patients with the former receiving 250 mg/kg of CPC-111 prior to surgery and the cardioplegia solution was supplemented with 2.5 mM of the drug.

The Company monitored the patients at multiple time points during and after surgery to evaluate various hemodynamic and blood chemistry measures and post-operative inotropic and vasodilator therapy. Concerning the hemodynamic measures, the active-treated group in both stages showed higher cardiac output and cardiac index scores which were statistically significant at 12 hours and at the 0-12 hour area under the curve. Concerning the blood chemistry measure, creatinine kinase MB ("CKMB") isoenzyme tended to be lower in the treated group and reached statistical significance at 2-6 hours. CKMB is an isoenzyme that is released through the cell wall into the blood stream with the breakdown of the cellular wall by ischemia. Also, the active treated group required approximately 50% less inotrope support on the intensive care unit ("ICU") post-operatively and required less vasodilator therapy allowing for earlier patient discharges from the ICU.

The Company is continuing this study, comprising another 10 active- and 10 placebo-treated patients with the former receiving 125 mg/kg of CPC-111 pre-bypass, which is necessary to determine the dose-response curve. Also, the Company will determine whether response is improved by adding two post-bypass doses to the pre-bypass dose of the drug in an additional 15 active- and 15 placebo-treated patients.

filed anin NoConsistent with the Company's strategy to diversify drug development risk, and plans to conduct its own clinical trials of CPC-111 as a cytoprotective drug for the treatment of some or all of the ischemic disorders which have been covered by the "use" patents that have been licensed to the Company. In November 1994, during the fiscal year ended July 31, 1996, the Company will began a dose-ranging Phase II clinical trial with CPC-111 in sickle cell anemia crises patients and in angioplasty patients and continued Phase II trials in ischemic congestive heart failure patients and and is currently planning coronary artery bypass graftingsickle cell crisis pat surgery patients.

CPC-211. CPC-211 is also a small non-peptide molecule which acts on glycolysis at a different site from CPC-111. The Company has exclusive rights to an issued U.S. patent covering the use of CPC-211 in cerebral ischemia and in 1996 was issued a Notice of Allowance on a novel dosing regimen of CPC-211 based on data from the Company's Phase I trial. The Company believes that CPC-211 stimulates a specific enzyme which is present in the membrane of mitochondria that removes a precursor of lactic acid (pyruvic acid) from the cytoplasm of the cell by transporting it into the mitochondria, resulting in a reduction of cell acidity. Increased post-ischemia accumulation of lactic acid is a major causal factor in the cessation of glycolysis, the resultant decrease in cellular ATP levels and eventual cell death. Numerous studies have shown that CPC-211 reduces post-ischemia lactic acid levels in animal models, as well as in humans, subjected to various traumatic events which would otherwise have resulted in increased lactic acid (lactic acidosis). Pre-clinical animal studies involving central nervous system trauma (ischemia to both the brain and spinal cord) have documented the effectiveness of CPC-211 in normalizing cell function and preventing or reducing tissue damage.

CPC-211 has been employed by clinical investigators in patients on an experimental basis for the intravenous treatment of lactic acidosis. Published clinical studies have established that CPC-211 reduces serum lactic acid and exhibits no serious side effects. It has also been shown in human studies to permeate the blood-brain barrier and to reduce brain lactic acid levels in congenital lactic acidosis patients. The Company believes that the availability of human data on CPC-211 should facilitate and reduce the risk associated with its clinical development.

Further, filed an the Company's Phase I study in CPC-211 demonstrated that the drug promptly lowers serum lactate, and that this effect lasted for several hours. Using these data, this in January 1996 to treat stroke year the Company began a Phase II clinical trial on CPC-211 in closed head injury patients exploring similar properties of the drug in the human brain. In addition, a Phase II clinical trial on CPC-211

in stroke patients is continuing.

Ischemia Drugs in Pre-clinical Research-The Metabolism and Excitotoxicity Programs

The Company is also seeking to develop new drugs for the treatment of ischemia-related disorders involving neurological damage, such as stroke, traumatic head injury, epilepsy and chronic neurodegenerative disorders such as Alzheimer's and Parkinson's disease. These pre-clinical research programs are focused on either the metabolic or the excitotoxicity aspects of ischemia therapeutics, and involve the chemical modification of identified lead molecules that regulate adenosine metabolism and various calcium ion channels on neuronal cells.

Adenosine Metabolism Inhibitor Program. The Company is seeking to develop CPC-405 and certain of its derivatives, which are novel small molecules with demonstrated potency as inhibitors of adenosine metabolism. Adenosine is a natural cytoprotective agent which is generated in ischemic tissue and serves to protect cells from a variety of traumatic situations. Naturally generated adenosine is rapidly degraded by enzymes. The Company expects that CPC-405 will increase the level of adenosine in tissue traumatized by ischemia and thereby increase its cytoprotective effect. A U.S. patent has been issued on the composition of the CPC-400 series of drugs. The Company will seek to identify other lead compounds to take forward into clinical development.

Neuronal Calcium Channel Blocker Program. The Company believes that the therapeutic approach to excitotoxicity currently attracting the most commercial attention involves the development of specific EAA receptor blockers which inhibit the excessive postsynaptic EAA action that is triggered by ischemia. Although these EAA receptor blockers have neuroprotective properties in cell culture and animal models of ischemia, their usefulness is hampered by toxic side effects associated with the blockage of EAA receptors and by the fact that there are multiple EAA receptor subtypes, all of which appear to cause post-ischemic damage when they are excessively stimulated.

The Company is seeking to develop new classes of drugs that are designed to remedy excitotoxicity in a potentially more complete and effective manner by reducing EAA release from nerve cells, thereby reducing the over-stimulation of all EAA receptor subtypes. This pre-synaptic approach to neuroprotection is viewed by the Company as potentially more effective than blocking receptors post-synaptically.

Specifically, the Company is seeking to develop separate classes of small-molecule drugs that act as neuronal calcium channel blockers ("NCCB"), which it has labelled as the CPC-300, CPC-800 and CPC-8000 series; the Company has synthesized over 100 compounds in this series. If successful, these drugs would have the ability to normalize or decrease EAA release and thereby comprehensively reduce the over-stimulation of EAA receptors. Prototype agents such as CPC-8027 have shown the desired effect of acting at the neuronal calcium channels, which controls EAA release. The Company has demonstrated neuroprotection in several pre-clinical models with CPC-304, CPC-317, CPC-877 and CPC-8027 and intends to further modify them structurally with the goal of improved drug delivery to the central nervous system. These modifications will require additional pre-clinical testing.

The Company has devised a novel human cell assay which enables it to determine whether various NCCBs have the desired functional attributes. The assay involves using human cells which have the functional neuronal-type calcium channel within their membrane. Studies conducted by the Company indicate that the neuronal-type calcium channel on these cells is pharmacologically similar to that present on nerve cells, and the Company believes that possession of this assay technology confers a competitive advantage in the development of neuronal calcium antagonists. The channel is also coupled to a function that can be measured using conventional laboratory techniques. The Company has filed two U.S. patent applications covering certain compounds with activity at the neuronal calcium channel as well as the functional neuronal-type calcium channel assay in human cells.

In July 1994, t
Licenses

The Company believes its strategic objectives can best be met by combining its in-house research and development efforts with licenses and research collaborations with scientists at outside academic and

clinical research centers.

The principal sources of the Company's existing licenses are:

Angel K. Markov, M.D.

CPC-111. The Company has obtained an exclusive license from Dr. Markov to four U.S. patents covering the use of CPC-111 in a number of indications including acute ischemic heart disease (such as AMI, congestive heart failure and unstable angina), surgically-induced ischemia, sickle cell anemia, ARDS, sepsis and other ischemic disorders. The Company is funding clinical development in Dr. Markov's laboratories at the University of Mississippi Medical Center (and is currently funding a Phase II clinical trial there in ARDS patients under his IND). In this regard, the Company has undertaken certain development obligations which must be met in order to maintain this license in force. In the event the Company breaches the license agreement, such as by not meeting certain milestones within the specified time periods or by failing to expend certain amounts in connection with clinical trials within specified time periods, the license will automatically terminate and all rights under the license and information acquired by the Company concerning any products based on the licensed technology will revert to Dr. Markov. In the event of such termination, the Company will retain the rights to market products for which sales occurred within the calendar year prior to the termination, and all other products and information related thereto based on the licensed technology will revert to Dr. Markov. To date, the Company has met all milestones.

University of Cincinnati

CPC-211. The Company has an exclusive license from University of Cincinnati ("UC") to a U.S. patent covering the use of CPC-211 in cerebral ischemia. The Company has undertaken certain development obligations which must be met in order to maintain its rights in force. If certain milestones are not met by the Company within specified time periods, UC may, in its sole discretion, elect to continue the agreement, negotiate in good faith with the Company to modify the agreement or terminate the agreement upon 30 days' written notice in which event all rights under the license would revert to UEM. To date, the Company has met all milestones.

John W. Olney, M.D.

EAA Adjuvant. The Company has obtained a license to a U.S. patent held by Dr. Olney concerning a drug which the Company believes can be employed as an adjuvant to diminish the side effects of and enable broader use of neurologic drugs being developed by a variety of other companies. Composition of matter claims were recently allowed on a patent application also licensed to the Company by Dr. Olney. Elie Abushanab, Ph. D.

Adenosine Metabolism Inhibitor. The Company obtained a license to certain adenosine metabolism inhibiting compounds developed by Dr. Elie Abushanab, which the Company believes will enhance the levels of adenosine in cardiovascular ischemia situations. Composition of matter claims on a patent application covering certain of these compounds have been recently allowed. Under the license, the Company must pay Dr. Abushanab certain milestone payments relating to the development of any drug covered by the license. To date, the Company has met all milestones.

Manufacturing

The Company does not currently intend to undertake the manufacture of any drugs which may derive from its technologies. Glofil is manufactured for the Company by the former owner of the drug and Inulin is manufactured for the Company by one of its existing contract manufacturers. In the case of CPC-111 and CPC-211, there are alternative sources of supply for the bulk drug in existence, and the Company has entered into arrangements with third parties to manufacture and formulate CPC-111 and CPC-211 for clinical trials. There can be no assurance that any of the Company's contract manufacturers will continue to meet the Company's requirements for quality, quantity and timeliness or the FDA's current Good Manufacturing Practice ("cGMP") requirements or that the Company would be able to find a substitute manufacturer for Glofil, Inulin, CPC-111, CPC-211 or any other of its drugs which would meet these requirements or that lots will not have to be recalled with the attendant financial consequences to the Company. The Company's dependence upon others for the manufacture of its drugs may adversely

affect the future profit margin, if any, on the sale of those drugs and the Company's ability to develop and deliver products on a timely and competitive basis. In the event the Company is unable to obtain or retain contract manufacturers or to obtain manufacturing on commercially acceptable terms, it may not be able to commercialize its drugs as planned.

Sales and Marketing

The commercialization of drug products is an expensive and time-consuming enterprise. The Company currently has a customer service representative and three field sales representatives for Glofil and Inulin and is hiring additional sales representatives. To market any of its drugs directly, the Company expects to develop a marketing and sales force with technical expertise and supporting distribution capability. The Company believes that it will be able to serve the hospital market in North America do so with a 50 to 70 person sales and marketing staff. Since its drugs will be sold primarily to and administered in acute care facilities rather than sold directly to physicians' offices or retail drug stores. There can be no assurance that the Company will be able to establish sales and distribution capabilities or be successful in gaining market acceptance for its drugs.

Competition

The pharmaceutical industry in general is highly competitive. Competition in the areas of the Company's activities is substantial and expected to increase. The Company faces competition from specialized biotechnology companies, large pharmaceutical companies, academic institutions, government agencies and public and private research organizations, many of which have extensive resources and experience in research and development, clinical testing, manufacturing, regulatory affairs, distribution and marketing. Some of these entities have significant research activities in areas upon which the Company's programs focus. Many of the Company's competitors possess substantially greater research and development, financial, technical, marketing and human resources than the Company and may be in a better position to develop, manufacture and market drugs. These entities may discover and develop drugs competitive with or superior to those developed by the Company.

Government Regulation

The manufacture and sale of drugs are subject to extensive and arduous regulation by United States and foreign governmental authorities prior to commercialization. In particular, drugs are subject to rigorous preclinical and clinical testing and other approval requirements by the FDA and comparable foreign regulatory authorities. The process for obtaining the required regulatory approvals from the FDA and other regulatory authorities takes many years and is very expensive. There can be no assurance that any drug developed by the Company will prove to meet all of the applicable standards to receive marketing approval in the United States or abroad. There can be no assurance that any such approvals will be granted on a timely basis, if at all. Delays and costs in obtaining these approvals and the subsequent compliance with applicable federal and state statutes and regulations could materially adversely affect the Company's ability to commercialize its drugs and its ability to receive sales revenues.

The research activities required by the FDA before a drug can be approved for marketing begin with extensive preclinical animal and laboratory testing. The tests include laboratory evaluation of product chemistry and animal studies for the safety and efficacy of the drug. The results of these studies are submitted to the FDA as part of an IND which is reviewed by the FDA prior to beginning clinical trials, first in normal volunteers and then in patients with the disease.

Clinical trials involve the administration of the investigational new drug to healthy volunteers or to patients, under the supervision of a qualified physician-principal investigator. Clinical trials are conducted in accordance with government-established statutes, regulations and guidelines and under protocols that detail the objectives of the study, the parameters to be used to monitor safety and the efficacy criteria to be evaluated. Each protocol must be submitted to the FDA as part of the IND. Further, each clinical study must be evaluated by an independent Institutional Review Board ("IRB") at the institution at which the study will be conducted. The IRB considers, among other things, ethical factors, the safety of

human subjects and the possible liability of the institution, and approves the informed consent to be obtained from all subjects and patients in the clinical trials. The Company will have to monitor the conduct of clinical investigators in performing clinical trials and their compliance with FDA requirements.

Clinical trials are typically conducted in three sequential phases (Phase I, Phase II and Phase III), but the phases may overlap. There can be no assurance that Phase I, Phase II or Phase III testing will be completed successfully within any specified time period, if at all, with respect to any of the Company's drugs. Furthermore, the Company or the FDA may suspend clinical trials at any time if it is felt that the subjects or patients are being exposed to an unacceptable health risk or that the investigational product lacks any demonstrable efficacy.

The results of the pharmaceutical development, preclinical studies and clinical studies are submitted to the FDA in the form of a New Drug Application ("NDA") for approval of the marketing and commercial shipment of the drug. The testing and approval process is likely to require substantial time (frequently five to eight years or more) and expense and there can be no assurance that any approval will be granted on a timely basis, if at all. The FDA may deny an NDA if applicable regulatory criteria are not satisfied, require additional testing or information, or require post-marketing testing and surveillance to monitor the safety of the Company's drugs. Notwithstanding the submission of the NDA and any additional testing data or information, the FDA may ultimately decide that the application does not satisfy its regulatory criteria for approval. Finally, drug approvals may be withdrawn if compliance with labeling and cGMP regulatory standards is not maintained or if unexpected safety problems occur following initial marketing.

Among the conditions for clinical studies and NDA approval is the requirement that the prospective manufacturer's quality control and manufacturing procedures conform to CGMP, which must be followed at all times. In complying with standards set forth in these regulations, manufacturers must continue to expend time, monies and effort in the area of production and quality control to ensure full technical compliance.

The U.S. Congress has recently enacted the Prescription Drug Act of 1992 requiring companies engaged in pharmaceutical development, such as the Company, to pay user fees in the amount of at least \$100,000 upon submission of an NDA. In addition to regulations enforced by the FDA, the Company also is subject to regulation under the Occupational Safety and Health Act, the Environmental Protection Act, the Toxic Substances Control Act, the Resource Conservation and Recovery Act and other present and potential future federal, state or local regulations. For marketing outside the United States, the Company is subject to foreign regulatory requirements governing human clinical trials and marketing approval for drugs. The requirements governing the conduct of clinical trials, product licensing, pricing and reimbursement vary widely from country to country.

Patents and Proprietary Rights

The Company's success may depend in large measure upon its ability to obtain patent protection for its drugs, maintain confidentiality and operate without infringing upon the proprietary rights of third parties. The Company has obtained patent coverage, either directly or through licenses from third parties, for certain of its drugs. It has licensed (i) four U.S. patents on CPC-111 which have expiration dates ranging from 2002 to 2008 and (ii) one U.S. patent on CPC-211 which has an expiration date of 2003. During the fiscal year ended July 31, 1996, the Company also received a notice of allowance on a U.S. patent on a novel dosing regimen for CPC-211.

The Company has filed patent applications with respect to other programs and expects to file additional applications in the future. There can be no assurance that any of these patent applications will be approved, except where claims have already been examined and allowed, or that the Company will develop additional proprietary products that are patentable. Nor can there be any assurance that any patents issued to the Company or its licensors will provide the Company with any competitive advantages or will not be challenged by third parties or that patents issued to others will not have an adverse effect on the ability of the Company to conduct its business. Furthermore, because patent applications in the United States are maintained in secrecy until issue, and because publication of discoveries in the scientific and patent literature often lag behind

actual discoveries, the Company cannot be certain that it was the first chronologically to make the inventions covered by each of its pending U.S. patent applications, or that it was the first to file patent applications for such inventions. In the event that a third party has also filed a U.S. patent application for any of its inventions, the Company may have to participate in interference proceedings declared by the United States Patent and Trademark Office to determine priority of the invention, which could result in substantial cost to the Company, even if the eventual outcome is favorable to the Company. In addition, there can be no assurance that the Company's U.S. patents, including those of its licensors, would be held valid by a court of law of competent jurisdiction. If patents are issued to other companies that contain competitive or conflicting claims which ultimately may be determined to be valid, there can be no assurance that the Company would be able to obtain a license to any of these patents.

Under Title 35 of the United States Code, as amended by the General Agreement on Tariffs and Trade implementing the Uruguay Round Agreement Act of 1994 ("GATT"), patents that issue from patent applications filed prior to June 8, 1995, will enjoy a 17-year period of enforceability as measured from the date of patent issue while those that issue from applications filed on or after June 8, 1995 will enjoy a 20-year period of enforceability as measured from the date the patent application was filed or the first claimed priority date, whichever is earlier. Patents that issue from applications filed on or after June 8, 1995, may be extended under the term extension provisions of GATT for a period up to five years to compensate for any period of enforceability lost due to interference proceedings, government secrecy orders or appeals to the Board of Patent Appeals or the Federal Circuit.

Under the Drug Price Competition and Patent Term Restoration Act of 1984, including amendments implemented under GATT (the "Patent Term Restoration Act"), the period of enforceability of a first or basic product patent or use patent covering a drug may be extended for up to five years to compensate the patent holder for the time required for FDA regulatory review of the product. This law also establishes a period of time following FDA approval of certain drug applications during which the FDA may not accept or approve applications for similar or identical drugs from other sponsors. Any extension under the Patent Term Restoration Act and any extension under GATT are cumulative. There can be no assurance that the Company will be able to take advantage of such patent term extensions or marketing exclusivity provisions of these laws. While the Company cannot predict the effect that such changes will have on its business, the adoption of such changes could have a material adverse effect on the Company's ability to protect its proprietary information and sustain the commercial viability of its products. Furthermore, the possibility of shorter terms of patent protection, combined with the lengthy FDA review process and possibility of extensive delays in such process, could effectively further reduce the term during which a marketed product could be protected by patents.

The Company also relies on trade secrets and proprietary know-how. The Company has been and will continue to be required to disclose its trade secrets and proprietary know-how to employees and consultants, potential corporate partners, collaborators and contract manufacturers. Although the Company seeks to protect its trade secrets and proprietary know-how, in part by entering into confidentiality agreements with such persons, there can be no assurance that these agreements will not be breached, that the Company would have adequate remedies for any breach or that the Company's trade secrets will not otherwise become known or be independently discovered by competitors.

Scientific Advisory and Clinical Trials Advisory Boards

Scientific Advisory Board

The Company currently has a Scientific Advisory Board ("SAB") whose members periodically advise the Company with respect to the Company's scientific research and development programs. The SAB does not meet as a group; rather individual member(s) are contacted for advice on an as-needed basis. The members are compensated through the grant of stock options and, if meetings are held, will receive fees for attending meetings as well as reimbursement for expenses. The Company has hired certain SAB members to perform services for the Company such as assay development and compound preparation.

The members of the Company's SAB are:

Name and Affiliation / Area of Expertise

Chung Hsu, M.D., Ph.D.
Director of Stroke Clinical Trials,
Washington University, St. Louis
School of Medicine
Animal models of stroke/clinical trials

Ronald Hayes, Ph.D.
Professor of Neurosurgery,
University of Texas, Houston
Animal models of head trauma

Bruce P. Bean, Ph.D.
Professor of Neurobiology,
Harvard University
Neuronal ion channels

Robert Parks, M.D., Ph.D.
Professor of Pharmacology,
Brown University
Biochemical pharmacology

John Olney, M.D.
Professor of Psychiatry and Neuropathology,
Washington University, St. Louis
Excitotoxicity; animal models of stroke

Edward J. Cragoe, Ph.D.
Former Senior Director of Medicinal Chemistry,
Merck Sharp & Dohme Research Laboratories
Medicinal chemistry/ion channels

K.C. Nicolaou, Ph.D.
Head of Chemistry,
The Scripps Research Institute
Professor of Chemistry,
University of California, San Diego
Medicinal chemistry

Harold Kimelberg, Ph.D.
Professor, Division of Neurology,
Albany Medical College
Glial cell release of glutamate

Elie Abushanab, Ph.D.
Professor of Medicinal Chemistry
and Chemistry
College of Pharmacy
University of Rhode Island
Medicinal chemistry/adenosine

Thomas J. Maloney
President
The Iso-Tex Companies
Friendswood, Texas
Radioisotopes/Nuclear Medicine

Claude Wasterlain, M.D.
Chief of Neurology Services
Sepulveda VA Medical Center/
Professor of Neurology
University of California Los Angeles
Stroke and epilepsy

Clinical Trials Advisory Boards

The Company has assembled two Clinical Trials Advisory Boards ("CTABs"), composed of physician "thought leaders" in the cardiology and neurology area, to assist in the planning, design and execution of the Company's clinical trials involving CPC-111 and CPC-211. The individuals who constitute each of the CTABs are paid consultants to the Company and are listed below:

Cardiovascular Clinical Trials Advisory Board

Eric J. Topol, M.D. (Chair)
Chairman, Department of Cardiology,
Director, Center for Thrombosis and Vascular Biology,
Cleveland Clinic and Foundation

Robert M. Califf, M.D.
Associate Professor of Medicine,
Duke University Medical Center

David R. Holmes, Jr., M.D.
Associate Professor of Medicine,
Mayo Clinic Medical School

Cerebrovascular Clinical Trials Advisory Board

William G. Barsan, M.D. (Chair)
Director of Emergency Medicine,
University of Michigan Medical School

Charles G. Brown, M.D.
Associate Professor & Research Director,
Department of Emergency Medicine
Ohio State University

Randall M. Chestnut, M.D.
Oregon Health Sciences Center
School of Medicine, Division of Neurosurgery
Portland, Oregon

Patrick D. Lyden, M.D.
Chief, Stroke Clinic
University of California, San Diego

Anthony Marmarou, Ph.D.
Medical College of Virginia
Division of Neurosurgery
Richmond, Virginia

The members of the SAB and the CTABs may be employed by or have consulting agreements with entities other than the Company, some of which may compete with the Company. These other obligations may limit the availability of the members to the Company. Most are not expected to participate actively in the Company's development. Certain of the institutions with which the members are affiliated may have regulations or policies which are unclear with respect to the ability of such persons to act as part-time consultants or in other capacities for a commercial enterprise. Regulations or policies now in effect or adopted in the future may limit the ability of the members to consult with the Company. The loss of the services of certain of the members could adversely affect the Company.

Furthermore, inventions or processes discovered by the SAB and CTAB members will not, unless otherwise agreed, become the property of the Company but will remain the property of such persons or of their full-time employers. In addition, the institutions with which the members are primarily affiliated may make available the research services of their scientific and other skilled personnel, including the members, to entities other than the Company. In rendering such services, such institutions may be obligated to assign or license to a competitor of the Company patents and other proprietary information which may result from such services, including research performed by a member for a competitor of the Company.

Scientific and Other Personnel

As of September 30, 1994, the Company had 3121 full-time employees, five of whom hold Ph.D. degrees, one of whom also holds an M.D. degree and one of whom holds a J.D. degree. Twelve of the full-time employees are employed in finance and general administration, seven in clinical and regulatory affairs and quality assurance, seven in research and development, and five in sales and marketing, customer service and business development. and the remainder of the employees are engaged in research, development and clinical activities. The Company believes that it maintains good relations with its employees.

Executive Officers of Registrant

Set forth below is certain information with respect to the executive officers of the Company at September 30, 1994:

Name	Age	Position
Paul J. Marangos, Ph.D.	49	Chairman of the Board, President and Chief Executive Officer
Stephen C. Eisold	50	Executive Vice President of Commercial Development and Chief Operating Officer
Anthony W. Fox, M.D., Ph.D.	40	Vice President, Drug Development
David W. Nassif, J.D.	40	Vice President, Chief Financial Officer and Secretary

Paul J. Marangos, Ph.D., has been President and Chairman of the Board since he founded the Company in November 1990. In February 1993, he became Chief Executive Officer. From April 1988 to November 1990, he was Senior Director of Research at Gensia Pharmaceuticals, Inc., a biotechnology company. From 1980 to 1988, he was Chief of Neurochemistry in the Biological Psychiatry Branch, National Institute of Mental Health. Dr. Marangos obtained his doctorate in biochemistry from the University of Rhode Island and did his post-doctoral work at the Roche Institute of Molecular Biology. He has published 250 research papers and four books in the field of biochemistry and pharmacology. Dr. Marangos' most recent book, published in July 1992, is entitled Emerging Strategies in Neuroprotection. He is a member of the Society for Neuroscience and the American Academy for the Advancement of Science. Dr. Marangos is the founding editor of the Journal of Molecular Neuroscience published by Humana Press.

Stephen C. Eisold joined the Company in May 1996 as the Executive Vice President of Commercial Development and Chief Operating Officer. From February 1990 to May 1996, he held various executive positions at Gensia Inc., most recently as Vice President and General Manager of the North American Pharmaceuticals Division. Prior thereto, Mr. Eisold held various sales, marketing and commercial development positions in the pharmaceutical industry since 1973. He received his bachelor of science from Springfield College and his masters in business administration from Rockhurst College.

Anthony W. Fox, M.D., Ph.D., joined the Company in April 1994 as the Vice President of Drug Development. From March 1990 to April 1994, he was employed by Glaxo, Inc., a pharmaceutical company, the last three years as Director, Cardiovascular and Anesthesiology Clinical Research. Dr. Fox was Group Leader, Division of Clinical Affairs, Norwich Division, of Procter & Gamble, a consumer products company, from May 1987 to March 1990. He received his bachelor of science in pharmacology with honors, his bachelor of medicine, bachelor of surgery and doctor of medicine degrees from the University of London, is a member of the Royal Colleges of Physicians and is a fully registered medical practitioner in the United Kingdom.

David W. Nassif, J.D., joined the Company in August 1993 as Vice President, Chief Financial Officer and Secretary. From January 1993 to August 1993, he was a consultant to various public and private companies in the areas of capital raising, investor relations and securities compliance. From July 1992 to January 1993, he was the Vice President, Chief Financial Officer and Assistant Secretary of 999, Inc., a diversified manufacturing and environmental services company. From December 1987 to July 1992, he was the Vice President and Assistant Secretary of Showscan Corporation, a technology company. Mr. Nassif holds honors finance, management information systems and law degrees from the University of Virginia.

Item 2. Properties.

The Company leases and occupies two buildings in Carlsbad, California at a total monthly rental of \$30,000. The Company's

executive offices and distribution facility are located in 14,595 square feet of space located at 2714 Loker Avenue West (the "2714 Space") and the Company's pre-clinical research group and laboratories are located in 8,547 square feet at 2732 Loker Avenue West (the "2732 Space"). The lease on the 2714 Space commenced April 1996 and has a term of 69 months. The lease on the 2732 Space commenced in December 1993 and has a term of 81 months. Both leases have clauses providing for rent increases at various points in time during the term of the leases.

Item 3. Legal Proceedings.

The Company is not a party to any legal proceedings.

Item 4. Submission of Matters to a Vote of Security Holders.

No matters were submitted to a vote of the Company's security holders during the fourth quarter of the fiscal year ended July 31, 1996.

PART II.

Item 5. Market for Registrant's Common Equity and Related Shareholder Matters.

The Common Stock of the Company was quoted on the Nasdaq SmallCap Market under the symbol "CYPR" since from November 3, 1992, the date of the Company's initial public offering, on November 3, 1992 until April 11, 1995 when it commenced trading on the Nasdaq National Market System under the same trading symbol. Prior to that date, there was no public market for the Common Stock. The Redeemable Class A and Class B Warrants are also quoted on the Nasdaq SmallCap Market National Market System under the symbols "CYPRW" and "CYPRZ", respectively. The following table sets forth for the calendar quarters indicated, the high and low sales prices of the Common Stock on the Nasdaq SmallCap Market or the Nasdaq National Market System, as the case may be, as reported in published financial sources. On May 8, 1995, the Company split its stock on a 2.5:1 basis. All of the prices shown in the table have been adjusted for the split.

Year ended July 31, 1996	High	Low
First Quarter	\$7.759.13	\$35.00.00
Second Quarter	\$12.506.13	\$3.13
Third Quarter	\$12.006.19	\$10.3754.56
Fourth Quarter	\$6.13	\$11.3753.63
Year ended July 31, 1995	High	Low
First Quarter	\$5.50	\$5.00
Second Quarter	\$5.90	\$4.60
Third Quarter	\$6.70	\$3.20
Fourth Quarter	\$9.50	\$6.88
3		
Second Quarter (from November 3, 1992)		\$11.25
\$ 7.25		
Third Quarter		\$10.75
9.25		\$
Fourth Quarter	\$ 9.75	\$ 1.50

Year ended July 31, 1994

The last sales price of the Common Stock on October 21, 1994 was \$4.0013.50.

According to a survey as of September 23, 1996, there were 1,562 beneficial owners of the Common Stock. 3

The Company has not paid any dividends since its inception and does not intend to pay any dividends on its Common Stock in the foreseeable future.

Item 6. Selected Financial Data.

The following table sets forth certain financial data with respect to the Company. The selected financial data should be read in conjunction with the Company's Financial Statements (including the Notes thereto) and "Management's Discussion and Analysis of Financial Condition and Results of Operations" appearing elsewhere in this

Report.

	Years ended July 31,				
	1992	1993	1994	1995	1996
			<		
				C	
Statement of Operations Data:					
Net sales	\$ -	\$ -	\$ -	\$ -	\$ 1,275
Gross Profit	-	-	-	-	870
Total operating expenses	359	1,715	2,565	3,910	4,988
Loss from operations	(359)	(1,715)	(2,565)	(3,910)	(4,118)
Other income, net	51	121	190	797	1,028
Net loss	(308)	(1,594)	(2,375)	(3,113)	(3,090)
Net loss per share	(0.07)	(0.28)	(0.32)	(0.32)	(0.27)
Shares used in computing net loss per share	4,350	5,637	7,357	9,860	11,518

	July 31,				
	1992	1993	1994	1995	1996
Balance Sheet Data:					
Cash, cash equivalents and short-term investments	\$ 220	\$ 4,444	\$ 5,666	\$ 13,442	\$ 15,997
Working capital	27	4,311	5,284	12,934	15,384
Total assets	303	4,900	6,206	14,175	19,747
Long-term debt	-	160	240	195	228
Common stock	257	6,748	9,927	20,945	21,838
Accumulated deficit	(310)	(1,904)	(4,279)	(7,392)	(10,482)
Total shareholders' equity	110	4,578	5,476	13,366	18,510

Item 7. Management's Discussion and Analysis of Financial Condition and Results of Operations

Except for the historical information contained herein, the following discussion contains forward-looking statements that involve risks and uncertainties, including statements regarding the period of time during which the Company's existing capital resources and income from various sources will be adequate to satisfy its capital requirements. The Company's actual results could differ materially from those discussed herein. Factors that could cause or contribute to such differences include but are not limited to, those discussed in this section, as well as in the sections entitled "Business", "Licenses", "Manufacturing", "Sales and Marketing", "Competition", "Government Regulation", "Patents and Proprietary Rights", those discussed in the S-3 Registration Statement File No. 333-3507 filed with U.S. Securities and Exchange Commission, as well as those discussed in any documents incorporated by reference herein or therein..

The Company was founded in 1990, commenced its research and development activities in 1991, completed an initial public offering

(the "IPO") in November 1992, commenced clinical trials in December 1994 and acquired two FDA-cleared products, Glofil and Inulin, (the "Acquisitions") in August 1995. The Company has sustained an accumulated deficit of \$10,480,000 from inception through July 31, 1996. As the Company will not have significant positive net operating cash flow for the next few years and the Company's research and development, clinical testing and regulatory, sales and marketing and general and administrative expenses during these years will be substantial and increasing, the Company expects to incur increasing losses for the foreseeable future.

will continue to be in the development stage for the next few years and its

Results of Operations

Year ended July 31, 1996 compared to year ended July 31, 1995

During the fiscal year ended July 31, 1996, the Company sustained a loss of \$3,090,000 (or \$.27 per share) compared to a loss of \$3,113,000 (or \$.32 per share) for the prior fiscal year. The gross profit of \$870,000 on sales of Glofil and Inulin and other income of \$1,028,000 (principally interest income) during the current fiscal year was offset by \$4,988,000 in expenses in the sales and marketing, general and administrative, clinical testing and regulatory and research and development areas. During the prior fiscal year, there were no product sales and other income of \$797,000 (principally interest income) was offset by \$3,910,000 in expenses in the above areas.

During the current year, the Company spent \$343,000 on sales and marketing, principally in the hiring of a field sales force and a customer service function and in various marketing and promotional programs. No such expense was recorded in the prior fiscal year as the Company was still in the development stage and had not yet acquired Glofil and Inulin.

General and administrative expense increased \$671,000 during the current year, principally due to \$438,000 in amortization expense of the purchased technology related to the acquisition of Glofil and Inulin. The Company also recorded a \$196,000 increase in the amortization of deferred compensation related to the issuance of stock, stock options and warrants at prices below market value and a \$178,000 increase in salary expense due to increased hiring of staff.

Clinical testing and regulatory expense decreased by \$149,000 during the current year, principally due to a decrease of \$209,000 in contract research organization costs because of a lower accrual for the Company's Phase II congestive heart failure trial on CPC-111 and a decrease of \$177,000 in licensing milestone expenses, offsetting increases in other areas, including a \$180,000 increase in salary expense due to additional hiring. During the prior year, the Company recorded a one-time milestone expense from the issuance of a non-qualified stock option grant to the licensor of CPC-211 as a milestone payment for the completion of that drug's Phase I trial.

Research and development increased by \$214,000 during the current year due to increases in salary expense, the use of outside collaborators and expense related to the Phase II Small Business Innovation Research Grant for the neuronal calcium channel blocker program (which grant was completed during the year) and the six-month Phase I Small Business Innovation Research Grant for the CPC-111 pro-drug program (which was awarded to the Company during the current year).

In addition, net interest and other income for the current year increased by \$231,000 principally due to (i) the interest income from a larger investment portfolio as a result of the various private placements during the year (described below in Liquidity and Capital Resources) and the Class A Warrant Program (also described below in Liquidity and Capital Resources), which was not available for all of the prior-year period because the program began in November 1994 and was completed in February 1995 and (ii) fees and interest earned on a loan that the Company made during the current year to a financial advisor.

Year ended July 31, 1995 compared to year ended July 31, 1994

During the fiscal year ended July 31, 1995 (the "1995 Year"), the Company sustained a loss of \$3,113,000 (or \$.32 per share) compared to a loss of \$2,375,000 (or \$.32 per share) for the prior fiscal year. Increased payroll caused increases in research and development, clinical testing and regulatory and general and administrative

expenses. Clinical testing and regulatory expenses during the 1995 Year also reflected the commencement and completion of the Phase I trial on CPC-211 (and the expense related to a milestone payment to the licensor of CPC-211 to the Company in the form of a common stock purchase warrant), the commencement of the Phase II trial on CPC-111 in congestive heart failure patients and the commencement of the Phase II trial on CPC-111 in cardiac surgery patients. Financial advisory and investor relations costs accounted for significant increases in general and administrative expenses during the 1995 Year, including \$315,000 of expense recognized in connection with the issuance (and subsequent repricing) of a warrant as compensation for services performed through July 31, 1995.

During the 1995 Year, the Company received \$250,000 of income from the Phase II Small Business Innovation Research Grant awarded to the Company by the National Institutes of Health in July 1994 (the "Grant"). Research and development expense for the quarter includes expenses incurred in connection with the Grant.

In addition, net interest and other income for the 1995 Year increased 269% to \$590,000 due principally to the interest income from a larger investment portfolio and the investment of the proceeds of the Special Class A Warrant Program at higher rates than the Company's then-existing investment portfolio for similar securities and maturities.

Year ended July 31, 1993 compared to year ended July 31, 1992

During the fiscal year ended July 31, 1993, the Company sustained a loss of \$1,593,602 compared to a loss of \$308,440 for the prior fiscal year. Following the IPO, the Company for the first time had sufficient capital to expand its research and development activities, begin preparation of the INDs and conduct related clinical activities. In addition, the Company incurred substantial costs associated with being a public company. Accordingly, research and development, clinical and regulatory and general and administrative costs increased substantially over the prior fiscal year. Liquidity and Capital Resources

The Company has principally funded its activities to date through its initial public offering ("IPO") in November 1992, in which it raised net proceeds of \$5,951,000, subsequent exercises of its Redeemable Class A Warrants in 1994 and early 1995, which raised \$10,497,000, exercises by the underwriter of the IPO of its unit purchase options (and the Redeemable Class A Warrants within such options), which raised \$1,681,000, that it had received as part of its compensation for the IPO and a private placement of mandatorily convertible notes during July 1996, which raised \$7,000,000. through the issuance of 1,150,000 units, each comprising a share of Common Stock, a Redeemable Class A Warrant ("Class A Warrant") and a Redeemable Class B Warrant ("Class B Warrant"). In October 1993, the Company received proceeds of \$725,625 from the exercise of "bridge" warrants for 537,500 shares of Common Stock (the "Bridge Warrant Exercise") which had been issued in connection with a bridge loan to the Company prior to the IPO.

From December 1993 to March 1994, the Company conducted a special program designed to encourage holders of Class A Warrants to exercise their warrants immediately (the "Class A Warrant Program") in order to provide the Company with additional working capital prior to commencing clinical testing. The holders of the Class A Warrants were offered one-half of a Class B Warrant and 1.175 shares of Common Stock upon exercise of each Class A Warrant at an adjusted price of \$9.253 (equal to 117.5% of the original exercise price of \$7.875). This program resulted in the Company's receipt of net proceeds of \$2,355,123 from the exercise of 258,831 Class A Warrants and the Company's issuance of 304,123 shares of Common Stock and 129,414 Class B Warrants.

At July 31, 1996, the Company had cash, cash equivalents and short-term investments of \$13,442,000, compared to \$13,442,000, compared to \$4,444,259 at July 31, 1995. The period-to-period increase was principally due to the private placement of convertible notes, Bridge Warrant Exercise and the. This factor also contributed to the increase in working capital at July 31, 1996 to \$15,384,000, compared to \$12,934,000, compared to \$4,311,350 at July 31, 1995.

The Company expects that its cash needs will increase significantly in future periods due to expansion of its research and development programs, increased clinical testing activity, growth of administrative, clinical and laboratory staff and their related

equipment and space needs. The Company Management believes that the Company's working capital will be sufficient to fund the operations of the Company for approximately 36 months dependent, in part, on the timing of the commencement of each phase of the clinical trials on CPC-111 and CPC-211 and the funding priorities that it gives its various research programs, the results of clinical tests and research programs; competing technological and market developments; the time and costs involved in obtaining regulatory approvals and in obtaining, maintaining and enforcing patents; the cost of product acquisitions and their resulting cash flows and other factors. estimates that its existing cash, cash equivalents and short-term investments and the income from the Phase II SBIR Grant will be sufficient to meet the Company's cash needs for at least the next 12 months. Accordingly, the Company will need to raise substantial additional funds to continue development and commercialization of its products. The Company's future cash requirements will be affected by results of research and development programs and clinical testing; competing technological and market developments; the time and costs involved in obtaining regulatory approvals and in obtaining, maintaining and enforcing patents; success in obtaining additional grants and other factors.

The Company expects to seek additional funds through exercises of its currently outstanding options and warrants, public or private equity financings, and collaborations or from other sources with corporate partners. There can be no assurance that funds can be obtained on desirable terms or at all. The Company may seek to raise additional capital whenever conditions in the financial markets are favorable, even if the Company does not have an immediate need for additional cash at that time.

Item 8. Financial Statements and Supplementary Data.

The Financial Statements of the Company and Report of Independent Auditors are filed as exhibits hereto, listed under Item 14 of this Report and incorporated herein by reference.

Item 9. Changes in and Disagreements with Accountants on Accounting and Financial Disclosure.

None.

PART III.

Item 10. Directors and Executive Officers of the Registrant.

The information regarding directors is hereby incorporated by reference to the section entitled "Election of Directors" in the Company's definitive Proxy Statement to be filed with the Securities and Exchange Commission in connection with the Company's 1997 Annual Meeting of Shareholders (the "Proxy Statement").

The information regarding executive officers appears under the section entitled "Executive Officers of Registrant" appearing in Item 1 of Part I of this Report.

Item 11. Executive Compensation.

The information required by this item is hereby incorporated by reference to the section entitled "Executive Compensation" in the Proxy Statement.

Item 12. Security Ownership of Certain Beneficial Owners and Management.

The information required by this item is hereby incorporated by reference to the section entitled "Security Ownership of Certain Beneficial Owners and Management" in the Proxy Statement.

Item 13. Certain Relationships and Related Transactions.

The information required by this item is hereby incorporated by reference to the section entitled "Transactions with Related Parties" in the Proxy Statement.

Item 14. Exhibits, Financial Statement Schedules and Reports on Form 8-K.

(a) (1) (2) Financial Statements and Schedules.

The financial statements are incorporated herein by reference from Exhibit 99.1, which begins with the Table of Contents on Page F- 1.

(a) (3) Exhibits.

See Exhibit Index on page 29. The following exhibits are referenced or included in this report.

The following management compensation plans and arrangements are required to be filed as exhibits pursuant to Item 14(c) of this report.

Exhibit

Number Description

3.1* Restated Articles of Incorporation of the Registrant, as amended.

3.2* Bylaws, as amended.

4.1* Specimen stock certificate.

4.2* Specimen Redeemable Class A Warrant.

4.3* Specimen Redeemable Class B Warrant.

4.4* Unit Purchase Option.

4.5* Form of Warrant Agreement.

4.6 Reference is made to Exhibits 3.1 and 3.2.

10.1* 1992 Stock Option Plan and related fForms of Incentive Stock Option and Nonstatutory Stock Option. ****

10.2 Amended 1992 Stock Option Plan.**

10.33* Form of Stock Purchase and Right of First Refusal Agreement, with related schedule.*

10.4* Employment Agreement, dated July 10, 1991 as amended and restated September, 1992, between the Registrant and Paul J. Marangos, Ph.D. ***

10.55 Amendment No. 1 to Employment Agreement, dated May 9, 1994, between the Registrant and Paul J. Marangos, Ph.D.*****

10.6 Amendment No. 2 to Employment Agreement, dated March 9, 1995, between the Registrant and Paul J. Marangos, Ph.D.***

10.7 Amendment No. 3 to Employment Agreement, dated October 1, 1996, between the Registrant and Paul J. Marangos, Ph.D.

10.8 1993 Non-Employee Directors Stock Option Plan and related form of Nonstatutory Stock Option. ****

Lenders' Warrants, dated July 31, 1992, among the Registrant and J. Morton Davis, Rosalind Davidowitz and Bernard B. Levine.

10.9* License Agreement, dated as of August 20, 1992, between the Registrant and Angel K. Markov, M.D. (with certain confidential information in brackets deleted). **

10.10* License Agreement, dated as of August 27, 1992, between the Registrant and University E.M., Inc. (with certain confidential information in brackets deleted). **

10.11 * License Agreement, dated September 3, 1992, between the Registrant and John Olney, M. D. (with certain confidential information in brackets deleted). **

10.14 License and Support Agreement, dated as of February 18, 1993, between the Registrant and Elie Abushanab, Ph.D. (with certain confidential information in brackets deleted).****

10.15 1993 Non-Employee Directors Stock Option Plan and related form of Nonstatutory Stock Option. ****

23.1 Consent of Ernst & Young LLP, Independent Auditors.

24.1 Power of Attorney. Reference is made to page 26.

99.1 Financial Statements and Schedule* Filed as an exhibit to the Registrant's Registration Statement on Form S-1, Registration No. 33-51682, and incorporated herein by reference.

** Filed as an exhibit to the Registrant's Form 10-Q for the period ended January 31, 1995, and incorporated herein by reference.

*** Filed as an exhibit to the Registrant's Form 10-K for the fiscal year ended July 31, 1994.

** Certain confidential portions deleted pursuant to Order Granting Application Under the Securities Act of 1933 and Rule 406 Thereunder Respecting Confidential Treatment, dated November 3, 1992.*** This

exhibit is a management contract or compensatory plan or arrangement required to be filed pursuant to Item 14(c) of this re**** Filed as an exhibit to the Registrant's Form 10-K for the fiscal year ended July 31, 1993.

Certain confidential portions deleted pursuant to Order Granting

Application Pursuant to Rule 24B-2 Under the Securities Exchange Act

of 1934 Respecting Confidential Treatment, dated December 20,

1993.(b) Reports on Form 8-K.

There were no reports on Form 8-K filed during the fourth quarter of 19964.

(c) Exhibits.

The exhibits required by this Item are listed under Item 14 (a)

(3).

(d) Financial Statement Schedules.

The financial statement schedule required by this Item is incorporated herein by reference from Exhibit 99.1, which begins with the Table of Contents to the Financial Statements on Page F-1.

SIGNATURES

Pursuant to the requirements of Section 13 or 15(d) of the Securities Exchange Act of 1934, the Registrant has duly caused this Report to be signed on its behalf by the undersigned, thereunto duly authorized, in the City and County of San Diego, State of California, on the 24th day of October, 1996.

CYPROS PHARMACEUTICAL CORPORATION

(Signature)

Paul J. Marangos

Chairman of the Board, President and Chief Executive Officer

POWER OF ATTORNEY

KNOW ALL MEN BY THESE PRESENTS, that each person whose signature appears below constitutes and appoints Paul J. Marangos, and David W. Nassif, and each of them, his attorney-in-fact, each with the power of substitution, for him in any and all capacities, to sign any amendments to this report, and to file the same, with exhibits thereto and other documents in connection therewith, with the Securities and Exchange Commission, hereby ratifying and confirming all that each of said attorneys-in-fact, or his substitute or substitutes, may do or cause to be done by virtue hereof.

Pursuant to the requirements of the Securities Exchange Act of 1934, this report has been signed below by the following persons on behalf of the Registrant and in the capacities and on the dates indicated.

Signature	Title	Date
Paul J. Marangos (Signature)	Chairman of the Board President and Chief Executive Officer and Director4	October 24, 1996

David W. Nassif (Signature)	Vice President, Chief Financial Officer and Secretary (Principal Financial and Accounting Officer)	
Digby W. Barrios (Signature)	Director	October 24, 19946
Bernard B. Levine	Director	October 24, 19946
Virgil Thompson (Signature)	Director	October 24, 19946
Robert A. Vukovich (Signature)	Director	October 24, 19946

Exhibit Index

Exhibit Number	Description	Page No.
2.1 (1)	Pharmaceutical Products Purchase and Distribution Support Agreement as of August 9, 1995 by and among Iso-Tex Diagnostics, Inc., Cypros Pharmaceutical Corporation and Thomas J. Maloney. (2)	
2.2 (1)	Glofil Contract Manufacturing and Royalty Agreement as of August 9, 1995 by and among Iso-Tex Diagnostics, Inc., Cypros Pharmaceutical Corporation and Thomas J. Maloney. 2)	
2.3 (1)	Merger Agreement as of August 9, 1995 among Cypros Pharmaceutical Corporation, Iso-Tex Diagnostics "B", Inc. and Jean and Thomas Maloney. (2)	
3.1 (3)	Restated Articles of Incorporation of the Registrant.	
3.2 (4)	Amendment to Restated Articles of Incorporation.	
3.3 (3)	Bylaws, as amended.	
4.1 (3)	Specimen stock certificate.	
4.3 (3)	Specimen Redeemable Class B Warrant.	
4.5 (3)	Form of Warrant Agreement.	
4.6	Reference is made to Exhibits 3.1 and 3.2.	
10.1 (3)	Forms of Incentive Stock Option and Nonstatutory Stock Option.	
10.2 (4)	Amended 1992 Stock Option Plan.	
10.3 (3)	Form of Restricted Stock Purchase Agreement, with related schedule.	
10.4 (3)	Employment Agreement, dated July 10, 1991 as amended and restated September 1, 1992, between the Registrant and Paul J. Marangos, Ph.D	
10.5 (5)	Amendment No. 1 to Employment Agreement, dated May 9, 1994, between the Registrant and Paul J. Marangos, Ph.D.	
10.6 (6)	Amendment No. 2 to Employment Agreement, dated March 9, 1995, between the Registrant and Paul J. Marangos, Ph.D.	
10.7 (7)	1993 Non-Employee Directors Stock Option Plan and related form of Nonstatutory Stock Option.	
10.8	Amendment No. 3 to Employment Agreement, dated October 1, 1996, between the Registrant and Paul J. Marangos, Ph.D.	30
10.9 (3)	License Agreement, dated as of August 20, 1992, between the Registrant and Angel K. Markov, M.D. (with certain confidential information in brackets deleted). (7)	

10.11 (3) License Agreement, dated as of August 27, 1992, between the Registrant and University E.M., Inc. (with certain confidential information in brackets deleted). (7)

10.12 (4) Assignment of and Amendment to License Agreement by and between University E.M., Inc., University of Cincinnati and the Registrant.

10.13 (6) License and Support Agreement, dated as of February 18, 1993, between the Registrant and Elie Abushanab, Ph.D. (with certain confidential information in brackets deleted). (8)

10.14 (9) Note Purchase Agreement dated July 11, 1996 by and among Cypros Pharmaceutical Corporation and Paresco, Inc.

10.15 (9) Note Purchase Agreement dated July 31, 1996 by and among Cypros Pharmaceutical Corporation and Cameron Capital Ltd.

23.1 Consent of Ernst & Young LLP, Independent Auditors. 31
24.1 Power of Attorney. Reference is made to page 27.

27. Financial Data Schedule - as filed electronically by Registrant (for SEC use only)

99.1 Financial Statements. 32

(1) Filed as an exhibit to the Registrant's Form 8-K dated August 10, 1995 and incorporated herein by reference.

(2) Certain confidential portions deleted pursuant to an application for Order Granting Confidential Treatment Under the Securities Exchange Act of 1934 and Rule 24b-2 Thereunder filed concurrently with the Form 8-K.

(3) Filed as an exhibit to the Registrant's Registration Statement on Form S-1, Registration No. 33-51682, and incorporated herein by reference.

(4) Filed as an exhibit to the Registrant's Form 10-Q for the period ended January 31, 1995, and incorporated herein by reference.

(5) Filed as an exhibit to the Registrant's Form 10-K for the fiscal year ended July 31, 1994.

(6) Filed as an exhibit to the Registrant's Form 10-K for the fiscal year ended July 31, 1993.

(7) Certain confidential portions deleted pursuant to Order Granting Application Under the Securities Act of 1933 and Rule 406 Thereunder Respecting Confidential Treatment, dated November 3, 1992.

(8) Certain confidential portions deleted pursuant to Order Granting Application Pursuant to Rule 24B-2 Under the Securities Exchange Act of 1934 Respecting Confidential Treatment, dated December 20, 1993.

(9) Filed as an exhibit to the Registrant's Form 8-K dated September 20, 1996 and incorporated herein by reference.

Form 10-K Items 14(a) (1) and (2)

Cypros Pharmaceutical Corporation

Years ended July 31, 1996, 1995 and 1994
with Report of Independent Auditors

Cypros Pharmaceutical Corporation

Form 10-K Items 14(a) (1) and (2)

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Report of Ernst & Young LLP, Independent Auditors F-2

Financial Statements (Item 14(a) (1)):

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Financial Statement Schedules (Item 14(a) (2)):

All financial statement schedules are omitted because the information described therein is not applicable, not required or is furnished in the financial statements or notes thereto.

Report of Independent Auditors

The Board of Directors and Shareholders
Cypros Pharmaceutical Corporation

We have audited the accompanying balance sheets of Cypros Pharmaceutical Corporation as of July 31, 1996 and 1995, and the related statements of operations, shareholders' equity, and cash flows for each of the three years in the period ended July 31, 1996. These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these financial statements based on our audits.

We conducted our audits in accordance with generally accepted auditing standards. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, the financial statements referred to above present fairly, in all material respects, the financial position of Cypros Pharmaceutical Corporation at July 31, 1996 and 1995, and the results of its operations and its cash flows for each of the three years in the period ended July 31, 1996 in conformity with generally accepted accounting principles.

ERNST & YOUNG
(Signature)

August 26, 1996

Cypros Pharmaceutical Corporation
Balance Sheets

	July 31, 1996	1995
Assets		
Current assets:		
Cash and cash equivalents	\$8,306,752	\$5,026,745
Short-term investments, held to maturity (Notes 1 and 2)	7,690,297	8,415,250
Accounts receivable	149,626	-
Inventory	63,386	-
Prepaid expenses and other current assets	61,409	25,910
 Total current assets	 16,271,470	 13,467,905
Property, equipment and leasehold improvements, net (Note 2)	608,206	411,651
Purchased technology, net of		

accumulated amortization of \$438,238 at July 31, 1996 (Note 1)	2,629,427	
Licenses and patents, net of accumulated amortization of \$87,277 and \$61,418 at July 31, 1996 and 1995, respectively (Note 4)	111,231	99,591
Deposits and other assets	126,180	195,692
Total assets	\$ 19,746,514	\$ 14,174,839

Liabilities and shareholders' equity

Current liabilities:		
Accounts payable	\$	\$ 138,537
	119,092	
Accrued compensation	155,748	83,594
Other accrued liabilities	231,864	189,713
Purchased asset obligation (Note 1)	200,000	-
Current portion of long-term debt (Note 3)	99,282	99,282
Current portion of capital lease obligation (Note 4)	81,035	22,517
Total current liabilities	887,021	533,643
Long-term debt (Note 3)	41,367	140,650
Capital lease obligations (Note 4)	187,265	54,149
Deferred rent (Note 4)	120,411	80,519
Commitments (Note 4)		
Shareholders' equity: (Note 5)		
Common stock, 30,000,000 shares authorized, 11,613,748 and 11,352,017 shares issued and outstanding as of July 31, 1996 and 1995, respectively	21,838,493	20,944,995
Mandatorily convertible notes		-
	7,458,498	
Deferred compensation		(186,993)
	(304,309)	
Accumulated deficit	(10,482,232)	(7,392,124)
Total shareholders' equity	18,510,450	13,365,878
Total liabilities and shareholder's equity	\$ 19,746,514	\$ 14,174,839

See accompanying notes.

Cypros Pharmaceutical
Corporation

Statements of Operations

	Years ended July 31,		
	1996	1995	1994
Net sales	\$ 1,275,240	-	-
Cost of sales	405,142	-	-
Gross profit	870,098	-	-
Operating expenses:			
Sales and marketing	343,054	-	-
General and			

administrative	2,254,000	1,583,420	1,015,666
Clinical testing and regulatory	1,389,128	1,537,964	1,022,134
Research and development	1,002,226	788,424	526,835
Total operating expenses	4,988,408	3,909,808	2,564,635
Loss from operations	(4,118,310)	(3,909,808)	(2,564,635)
Research grant income	270,510	250,000	-
Interest income, net (Note 3)	757,692	547,107	189,372
Net loss	\$ (3,090,108)	\$ (3,112,701)	\$ (2,375,263)
Net loss per share	\$ (0.27)	\$ (0.32)	\$ (0.32)
Shares used in computing net loss per share	11,518,169	9,859,857	7,357,960

See accompanying notes.

THIS AMENDMENT NO. 3 TO AMENDED AND RESTATED EMPLOYMENT AGREEMENT (the "Agreement") is made and entered into effective as of October 1, 1996 (this "Amendment") by and between Cypros Pharmaceutical Corporation, a California corporation (the "Company") and Paul J. Marangos ("Executive"). The Company and Executive are hereinafter collectively referred to as the "Parties," and individually referred to as "Party."

For good and valuable consideration, receipt of which is hereby acknowledged by the Company and Executive, the Parties, intending to be legally bound, agree that Section 3 and Section 4 of the Agreement is hereby amended and restated in its entirety as follows:

"3. Term of Employment.

3.1. Subject to earlier termination as provided in this Agreement, Executive shall be employed pursuant to the terms of this Agreement for a term beginning September 1, 1992 and expiring at midnight on August 31, 1999. After the expiration of such term, this Agreement shall continue from month to month in the absence of written notice to the contrary from either Party to the other."

"4. Compensation of Executive.

4.1. During the term of this Agreement, the Company shall pay Executive a salary (the "Base Salary") of Two Hundred Sixteen Thousand Five Hundred Dollars (\$216,500) per year, payable in regular periodic payments in accordance with Company policy. Such salary shall be prorated for any partial year of employment on the basis of a 365-day year."

IN WITNESS WHEREOF, the Parties have executed this Amendment No. 3 as of the date first above written.

The Company:
Cypros Pharmaceutical Corporation
a California corporation
(Signature)
David W. Nassif
Vice President and Chief Financial Officer

Executive:
(Signature)
Paul J. Marangos

CONSENT OF ERNST & YOUNG LLP, INDEPENDENT AUDITORS

We consent to the incorporation by reference in the Registration Statements (Form S-3 and S-8) of our report dated August 26, 1996, with respect to the financial statements of Cypros Pharmaceutical Corporation included in the Annual Report (Form 10-K) for the year ended July 31, 1996.

ERNST & YOUNG LLP
(Signature)
San Diego, California
October 24, 1996

YEAR
JUL-31-1996
JUL-31-1996
8,306,752
7,690,297
149,626
0
63,386
61,409
885,314
(277,108)
19,746,514
887,021
0
0
21,838,493
(3,328,043)
19,746,514
1,275,240
1,275,240
405,142
405,142
4,988,408
0
47,953
(3,090,108)
0
(3,090,108)
0
0
0
(3,090,108)
0
(0.27)
0