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

FORM 10-K

[x] Annual report pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

For the fiscal year ended July 31, 2001

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: 0-27756

ALEXION PHARMACEUTICALS, INC.
(Exact Name of Registrant as Specified in Its Charter)

Delaware	13-3648318
(State or Other Jurisdiction of Incorporation or Organization)	(I.R.S. Employer Identification No.)

352 Knotter Drive, Cheshire Connecticut 06410
(Address of Principal Executive Offices) (Zip Code)

203-272-2596
(Registrant's telephone number, including area code)

Securities registered pursuant to Section 12(b) of the Act: None

Securities registered pursuant to Section 12(g) of the Act: Common Stock, par value $0.0001

Indicate by check mark whether the registrant: (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes [x] No [_]

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

The aggregate market value of the Common Stock held by non-affiliates of the registrant, based upon the last sale price of the Common Stock reported on the National Association of Securities Dealers Automated Quotation (NASDAQ) National Market System on October 24, 2001, was approximately $321,829,000.

The number of shares of Common Stock outstanding as of October 24, 2001 was 18,110,801.

PART I

THIS ANNUAL REPORT ON FORM 10-K CONTAINS FORWARD-LOOKING STATEMENTS THAT HAVE BEEN MADE PURSUANT TO THE PROVISIONS OF THE PRIVATE SECURITIES LITIGATION REFORM ACT OF 1995. SUCH FORWARD LOOKING STATEMENTS ARE BASED ON CURRENT EXPECTATIONS, ESTIMATES AND PROJECTIONS ABOUT THE COMPANY'S INDUSTRY, MANAGEMENT'S BELIEFS AND CERTAIN ASSUMPTIONS MADE BY THE COMPANY'S MANAGEMENT. WORDS SUCH AS "ANTICIPATES," "EXPECTS," "INTENDS," "PLANS," "BELIEVES," "SEEKS," "ESTIMATES," VARIATIONS OF SUCH WORDS AND SIMILAR EXPRESSIONS ARE INTENDED TO IDENTIFY SUCH FORWARD-LOOKING STATEMENTS. THESE STATEMENTS ARE NOT GUARANTEES OF FUTURE PERFORMANCE AND ARE SUBJECT TO CERTAIN RISKS, UNCERTAINTIES AND ASSUMPTIONS THAT ARE DIFFICULT TO PREDICT; THEREFORE, ACTUAL RESULTS MAY DIFFER MATERIALLY FROM THOSE EXPRESSED OR FORECASTED IN ANY SUCH FORWARD-LOOKING STATEMENTS. SUCH RISKS AND UNCERTAINTIES INCLUDE, BUT ARE NOT LIMITED TO, THOSE SET FORTH HEREIN UNDER "IMPORTANT FACTORS REGARDING FORWARD-LOOKING STATEMENTS," ATTACHED HERETO AS EXHIBIT 99, AS WELL AS THOSE NOTED IN THE DOCUMENTS INCORPORATED HEREIN BY REFERENCE. UNLESS REQUIRED BY LAW, THE COMPANY UNDERTAKES NO OBLIGATION TO UPDATE PUBLICLY ANY FORWARD-LOOKING STATEMENTS, WHETHER AS A RESULT OF NEW INFORMATION, FUTURE EVENTS OR OTHERWISE. HOWEVER, READERS SHOULD CAREFULLY REVIEW THE RISK FACTORS SET FORTH IN OTHER REPORTS OR DOCUMENTS THE COMPANY FILES FROM TIME TO TIME WITH THE SECURITIES AND EXCHANGE COMMISSION.

Item 1. BUSINESS.

Overview

We seek to develop pharmaceutical products for the treatment of heart disease, inflammation, diseases of the immune system and cancer in humans. During the fiscal years ended July 31, 2001, 2000, and 1999, we spent $38.8 million, $40.2 million, and $23.7 million, respectively, on research and development activities excluding acquisition related non-cash charges for in-process research and development and amortization of purchased intangibles. Our two lead product candidates are genetically altered antibodies that target specific diseases that arise when the human immune system induces undesired inflammation in the human body. Antibodies are proteins that bind to specific targets and are used by the immune system to protect the body.

Our two lead product candidates are designed to block components of the human immune system that cause undesired inflammation while allowing beneficial components of the immune system to remain functional. Our two lead product candidates are "humanized" antibodies, designed to block the inflammatory effects of the components of the immune system known as "complement." A humanized antibody is an antibody genetically altered to minimize or avoid an immune response in humans. Our two lead product candidates are:

- Pexelizumab. We completed a Phase IIb trial for the treatment of acute inflammation caused by the trauma of heart and lung bypass procedures during open heart surgery, and we are enrolling patients in two Phase II heart attack trials. We are developing pexelizumab in collaboration with Procter & Gamble Pharmaceuticals; and

- 5G1.1. We completed a Phase II trial for the chronic treatment of rheumatoid arthritis or RA and a Phase I pilot trial for psoriasis. We are enrolling patients in Phase II trials for the treatment of membranous nephritis and lupus nephritis, and in a Phase I pilot trial for bullous pemphigoid. We have completed enrollment in a Phase

I pilot trial in dermatomyositis patients. On-going 12-month extension studies in RA and membranous nephritis will help us assess long-term safety. We are developing 5G1.1 ourselves.

In September 2000, we acquired Prolifaron, Inc., through a merger with Alexion Antibody Technologies, Inc. (AAT), a newly created, wholly owned subsidiary of Alexion. Prolifaron was a biopharmaceutical company that possessed extensive research expertise and technologies in the area of creating fully human antibodies from libraries containing billions of human antibody genes.

In addition to our antibody product candidates which inhibit the inflammatory effects of complement and our technology programs focusing on human antibody discovery and development, we are developing products to block the harmful effects of a component of the immune system known as "T-cells" in pre-clinical studies. We call these products "apogens." We targeted our first apogen product candidate, known as MP4, for the treatment of patients with multiple sclerosis. We are also developing methods of blocking the human immune system to permit the use of cells and organs from non-human species in the treatment of diseases in humans. This product development program with genetically altered pig cells is initially targeting the treatment of patients with Parkinson's disease and patients with spinal cord injury.

The Immune System

The human immune system defends the body from attack or invasion by infectious agents or pathogens. This is accomplished through a complex system of proteins and cells, primarily complement proteins, antibodies and white blood cells, each with a specialized function. Under normal circumstances, complement proteins, together with antibodies and white blood cells, act to protect the body by removing:

- harmful microorganisms;
- cells containing foreign proteins known as antigens; and
- disease-causing combinations of antigens and antibodies known as immune complexes.

When activated by stimuli, the immune system triggers a series of enzymatic and biochemical reactions called the complement cascade that results in an inflammatory response. This inflammatory response is one of the immune system's weapons against foreign pathogens or otherwise diseased tissue. However, under certain circumstances, the complement cascade may be activated inappropriately to direct an inflammatory response at healthy tissue, which may result in acute and chronic inflammatory conditions.

Common heart diseases and procedures in which the complement cascade is activated include:

- cardiopulmonary bypass surgery;
- myocardial infarction or heart attack;
- unstable angina or painful chest pains associated with an insufficient blood supply to the heart;
- angioplasty or procedures for opening up narrowed or blocked arteries that supply the heart; and
- stroke and other peripheral vascular or blood circulatory diseases.

Common autoimmune diseases in which the complement cascade is activated include:

- rheumatoid arthritis;

- kidney diseases;
- lupus;
- inflammatory bowel diseases;
- inflammatory skin disorders; and
- multiple sclerosis.

T-cells, a type of white blood cell, play a critical role in the normal immune response by recognizing cells containing antigens and initiating the immune response. This response results in T-cells:

- attacking the antigen-containing tissue; and
- directing the production of antibodies by white blood cells to eliminate the antigen-bearing foreign organism.

In autoimmune diseases, T-cells may mistakenly attack healthy host tissue and may cause an inflammatory response resulting in tissue destruction. In the case of multiple sclerosis, this may cause paralysis due to destruction of nerve fibers in the brain.

Product Development Programs

We have focused our product development programs on anti-inflammatory therapeutics for diseases for which we believe current treatments are either non-existent or inadequate. Currently available drugs for certain autoimmune, cardiovascular and neurologic or nervous system diseases, in which the immune system attacks the patient's own tissue, broadly suppress the entire immune system, and may also cause potentially severe side effects. Our lead product candidates, which are genetically altered antibodies known as C5 Complement Inhibitors, are designed to selectively block the production of inflammation-causing proteins in the complement cascade. We believe that selective suppression of this immune response may provide a significant therapeutic advantage relative to existing therapies.

Additionally, we are developing selective T-cell inhibitors known as apogens and UniGraft xenotransplants or use of non-human cells and tissues for neurologic or nervous systems disorders.

Our product candidates are as follows:

Product candidate	Technology	Indication	Status
Pexelizumab	C5 Complement Inhibitor (single chain antibody)	Cardiopulmonary bypass	Phase IIb trial completed
		Myocardial infarction	
		(1) Thrombolysis -a)	Phase II trial ongoing
		(2) PTCA -b)	Phase II trial ongoing
5G1.1	C5 Complement Inhibitor (antibody)	Rheumatoid arthritis	Phase II trial completed
		Membranous nephritis	Phase II trial ongoing
		Lupus Nephritis	Phase II trial ongoing
		Psoriasis	Phase Ib trial completed

		Dermatomyositis	Phase Ib trial enrollment completed
		Bullous Pemphigoid	Phase Ib trial ongoing
MP4	Apogen	Multiple sclerosis	Pre-clinical
UniGraft-SCI	Cell replacement	Spinal cord injury	Pre-clinical
UniGraft-PD	Cell replacement	Parkinson's disease	Pre-clinical

(a- dissolving clots that block heart vessels
(b- percutaneous transluminal coronary angioplasty or PTCA, a procedure for opening up narrowed or blocked arteries that supply blood to the heart

C5 Complement Inhibitors

Complement proteins are a series of inactive proteins circulating in the blood. When activated by stimuli, including those associated with both acute and chronic inflammatory disorders, these inactive complement proteins are split by enzymes known as convertases into activated byproducts through the complement cascade.

Some of these byproducts, notably C3b, are helpful in fighting infections and inhibiting autoimmune disorders. However, the byproducts generated by the cleavage of C5, known as C5a and C5b-9, generally cause harmful inflammation. The inflammatory byproducts of C5 cause:

. activation of white blood cells;

. attraction of white blood cells;

. production of injurious hormones including tumor necrosis factor-alpha;

. activation of blood vessel-lining cells called endothelial cells, allowing leakage of white blood cells into tissue; and

. activation of blood-clotting cells called platelets.

The following diagram describes the complement cascade:

[diagram]

[GRAPHIC APPEARS HERE]

Because of the generally beneficial effects of the components of the complement cascade prior to C5 and the greater inflammatory disease-promoting effects of the cleavage products of C5, we have identified C5 as a potentially effective anti-inflammatory drug target. Our first two C5 Inhibitors specifically and tightly bind to C5 blocking its cleavage into harmful byproducts and are designed to inhibit subsequent damage from the inflammatory response.

In laboratory and animal models of human disease, we have shown that the administration of C5 Inhibitor, as compared to placebo, is effective in:

- preventing inflammation during cardiopulmonary bypass;
- reducing heart tissue damage during myocardial infarction;
- reducing brain damage in cerebral ischemia or reduced blood flow to brain tissue;
- enhancing survival in a model of lupus; and
- preserving kidney function in nephritis or inflammation of kidney tissue.

In addition, in human clinical trials, we have shown that C5 Inhibitors may be associated with reduction of:

- inflammation during cardiopulmonary bypass surgery;
- heart tissue damage during cardiopulmonary bypass surgery;
- new cognitive or mental faculty deficits after cardiopulmonary bypass surgery;
- an objective measure of disease activity in rheumatoid arthritis patients; and
- the incidence of proteinuria or abnormal loss of substantial amounts of protein in a patient's urine in lupus patients.

C5 Inhibitor Immunotherapeutic Product Candidates

We are developing one of our two lead C5 Inhibitor product candidates, pexelizumab, for the treatment of inflammation related to acute cardiovascular diseases and procedures. Our initial indications for pexelizumab are cardiopulmonary bypass surgery, myocardial infarction utilizing thrombolysis, and myocardial infarction utilizing PTCA. We are developing our other C5 Inhibitor product candidate, 5G1.1, for the treatment of inflammation related to chronic autoimmune disorders. The initial indications for which we have pursued clinical development activities for 5G1.1 are rheumatoid arthritis, membranous nephritis, lupus nephritis, dermatomyositis, bullous pemphigoid, and psoriasis. The selection of these eight indications is based upon our belief that each represents a clinical condition which is:

- closely tied to the production of activated complement byproducts;
- characterized by clear development pathways;
- inadequately treated by current therapies; and
- associated with substantial health care costs.

To date, pexelizumab and 5G1.1 have been observed to be safe and well tolerated in completed and ongoing clinical trials in which over 2,700 individuals were treated with either C5 Inhibitor or placebo.

Pexelizumab

Pexelizumab is a humanized, single chain antibody that has been shown to block complement activity for up to 20 hours after a single injection at the doses tested and is designed for the treatment of acute inflammatory conditions. In January 1999, we entered into a collaboration arrangement with Procter & Gamble to develop and commercialize pexelizumab. Under this collaboration, we expect to pursue the development of pexelizumab for the treatment of inflammation caused by various acute cardiovascular indications and procedures such as cardiopulmonary bypass surgery, myocardial infarction and angioplasty. Procter & Gamble has agreed to fund clinical development and manufacturing costs relating to pexelizumab for these indications.

Cardiopulmonary Bypass Surgery

In cardiopulmonary bypass surgery, blood is diverted from a patient's heart and lungs to a cardiopulmonary, heart-lung, bypass machine in the operating room. The machine adds oxygen to the blood and circulates the oxygenated blood to the organs in the patient's body. Significant side effects of CPB or cardiopulmonary bypass surgery include tissue damage and excessive bleeding during and after the procedure. We believe these side effects may result from the activation of the complement cascade when the patient's blood comes into contact with the plastic lining of the machine and when insufficient blood flows through the heart as a result of the procedure and after blood flow through the heart is reintroduced following completion of the procedure. Activated complement byproducts may be increased by over 1000% in patients undergoing CPB. The inflammation is also characterized by the activation of leukocytes, a type of white blood cell, and platelets, cells responsible for clotting. We believe that this leukocyte activation is associated with impaired lung, heart, brain and kidney function. We further believe that platelet activation and subsequent platelet dysfunction during the procedure impair a patient's ability to stop the bleeding that occurs after extensive surgery.

Pexelizumab is designed to rapidly penetrate the patient's tissues and to inhibit complement activation in patients immediately before, during and after cardiopulmonary bypass in order to reduce the cardiovascular and brain tissue damage and bleeding complications. We believe inhibition of the inflammatory response may reduce:

- the incidence of death;
- the incidence of heart tissue damage;

. the incidence of stroke or blockage of blood vessels in brain;

. post-operative or after surgery complications;

. the time spent by patients in the intensive care unit or ICU;

. the scope of required treatments associated with CPB; and

. the need for blood transfusions.

The American Heart Association estimates that in 1997, approximately 600,000 cardiopulmonary bypass operations were performed in the United States. Currently, products utilized in patients undergoing cardiopulmonary bypass are designed to enhance the coagulation of blood so as to reduce the need for blood transfusions. However, we believe these products have little beneficial effect on the heart and brain inflammatory complications associated with the surgery.

Our pre-clinical studies indicated that C5 Inhibitors can prevent activation of platelets and leukocytes and the subsequent inflammatory response that occurs during the circulation of human blood in a closed-loop cardiopulmonary bypass machine. These pre-clinical studies additionally indicated that administration of a C5 Inhibitor may reduce cardiac damage associated with reduced heart blood flow.

Clinical Trials - CPB

In January 1999, we commenced dosing in a Phase IIb clinical trial with pexelizumab in patients undergoing coronary artery bypass graft surgery with or without accompanying valve surgery during CPB. The objective of this multi-center, double-blinded, randomized, placebo-controlled study was to assess the safety and effectiveness of pexelizumab in these patients. After completion of this trial, preliminary results from this trial were released in January 2001 which suggested that pexelizumab blocked complement, reduced inflammation and appeared to be safe and well-tolerated. Some patients in the trial experienced serious adverse events which included irregular heartbeat, infection, right heart failure and internal bleeding. The most common adverse events were irregular heartbeat, nausea and anemia. The primary therapeutic, exploratory pre-set goal of the trial, referred to as the primary endpoint, was not achieved. However, in the pre-specified population that included approximately 90% of the patient population, the 800 patients who had coronary artery bypass graft surgery, or CABG, without valve surgery, those that received pexelizumab at the highest dose level experienced a significant reduction in larger post-surgical heart attacks.

We are currently evaluating the data from this trial. If our final analysis is positive, and if the FDA agrees with the analysis, we expect to initiate a Phase III trial of the safety and effectiveness of pexelizumab. We expect that this trial would focus on treating patients undergoing CABG along with CPB, and would be conducted with Procter & Gamble, our partner in the development and commercialization of pexelizumab.

Acute Myocardial Infarction

Myocardial infarction is an acute cardiovascular disorder in which the coronary arteries, the blood vessels that supply nutrients to the heart muscle, are blocked to such an extent that the flow of blood is insufficient to supply enough oxygen and nutrients to keep the heart muscle alive. With insufficient supply of blood, oxygen, and nutrients, the heart muscle may subsequently infarct or die. Upon the reduction in blood flow in the coronary artery, a complex cascade of inflammatory events involving complement proteins, platelets and leukocytes and their secreted factors, and endothelial cells, commences within the blood vessel. In patients suffering a myocardial infarction, activated complement byproducts are significantly elevated. This severe inflammatory response targeting the area of insufficient blood flow to cardiac muscle is associated with subsequent death of heart muscle. Restoration of blood flow is also associated with an additional inflammatory reaction with an accompanying production of activated complement byproducts. In addition to the high incidence of sudden cardiac death at the onset, severe complications associated with the initial survival of an acute myocardial

infarction include congestive heart failure, stroke, and death. The American Heart Association estimates that approximately 1.1 million people in the United States will have a heart attack in 2000.

We are developing pexelizumab to inhibit inflammation associated with complement activation in order to reduce the extent of death of heart muscle in patients suffering an acute myocardial infarction. In contrast, most drugs currently being developed or on the market to treat myocardial infarction are designed to improve blood flow through the heart, rather than treating the damaging effects of inflammation caused by myocardial infarction. We and our scientific collaborators have performed pre-clinical studies in rodents which have demonstrated that administration of a C5 Inhibitor during periods of insufficient supply of blood to the heart muscle and prior to restoration of normal flow to the heart muscle significantly reduced the extent of subsequent death of heart muscle compared to control animal studies. Additionally, administration of a C5 Inhibitor significantly reduced the extent of cardiac damage associated with reduced heart blood flow without subsequent restoration of blood flow.

Clinical Trials - Acute Myocardial Infarction

In October 1998, we commenced dosing subjects in a Phase I clinical trial in healthy individuals that was designed to evaluate dosing regimens for subsequent cardiopulmonary bypass and myocardial infarction clinical trials. We have used the results of this trial to select dosing regimens for subsequent clinical trials in acute myocardial infarction and CPB patients. The results of this trial indicated that pexelizumab was well tolerated at doses more than three times as high as had been previously administered. We are conducting two Phase II clinical trials, each one designed to enroll approximately 1,000 patients, with our collaborator Procter & Gamble, that test the safety and effectiveness of pexelizumab for the treatment of acute inflammation in patients suffering an acute myocardial infarction.

5G1.1

5G1.1 is a humanized antibody that blocks complement activity for one to two weeks at the doses tested and is designed for the chronic treatment of autoimmune diseases such as rheumatoid arthritis and nephritis. 5G1.1 is not included in the collaboration with Procter & Gamble, and we have retained full rights to 5G1.1.

Rheumatoid Arthritis

Rheumatoid arthritis is a chronic autoimmune disease directed at various organ and tissue linings, including the lining of the joints, causing inflammation and joint destruction. Clinical signs and symptoms of the disease include weight loss, joint pain, morning stiffness and fatigue. Further, the joint destruction can progress to redness, swelling and pain with frequent and severe joint deformity. Diagnostic procedures, which may include obtaining a sample of joint fluid, routinely demonstrate substantial elevations in the levels of activated complement byproducts in the joint fluid of affected rheumatoid arthritis patients. Rheumatoid arthritis is generally believed to be caused by different types of white blood cells, including T-cells, which both directly attack the patient's joints and also activate B-cells, another type of white blood cell, to produce antibodies which activate complement proteins in the joint leading to inflammation with subsequent tissue and joint destruction. It is estimated that more than 2.0 million people are currently affected by rheumatoid arthritis in the United States.

We are developing 5G1.1 for the treatment of patients with chronic inflammatory diseases, including rheumatoid arthritis. We have performed pre-clinical studies in rodent models of rheumatoid arthritis which have shown that C5 Inhibitor administration, as compared to placebo-treated subjects:

- reduced the swelling in joints;
- prevented the onset of erosion of joints;
- reduced the inflammatory white blood cell infiltration into the joints;

. prevented the spread of disease to additional joints;

. blocked the onset of clinical signs of rheumatoid arthritis; and

. reduced established disease.

Currently, there are a large number of anti-inflammatory drugs under development or on the market for the treatment of patients with rheumatoid arthritis. These drugs include non-steroidal anti-inflammatory drugs, and their more recent analog the COX-2 inhibitors, which generally treat the symptoms of the disease, but do not alter disease progression. There are also several currently available drugs that are disease-modifying agents, but these are associated with undesirable side effects. Recently, tumor necrosis factor, or TNF, inhibitors have been approved or are under development to reduce the inflammatory response. TNF is one of the many injurious substances that may be generated downstream of the complement cascade. In contrast to single agent inhibitors like TNF inhibitors, by acting at C5 of the complement cascade, we expect 5G1.1 both to block complement activation and reduce the production of many of these downstream harmful substances. Because of this dual action, we believe that 5G1.1 may provide a more potent anti-inflammatory effect.

Clinical Trials - Rheumatoid Arthritis

In December 1997, we filed an IND with the FDA for 5G1.1 in the treatment of rheumatoid arthritis patients. In our early clinical trials, single doses of 5G1.1 were safe and well tolerated in the study populations as compared to placebo, showed dose-dependent reduction in complement activity in the study subjects, and showed a reduction in C-reactive protein blood levels in the study subjects. C-reactive protein is considered by many physicians to be the most objective component of the American College of Rheumatology's definition of efficacy criteria for rheumatoid arthritis drug trials. Biological and clinical results from our Phase I/II trial demonstrated that 50% of rheumatoid arthritis patients receiving 8.0 mg/kg of 5G1.1 achieved an ACR 20 score, a measure of clinical benefit, as compared to 10% of placebo-treated patients.

We completed our first Phase II clinical trial testing the safety and effectiveness of repetitive dosing of 5G1.1 in patients with rheumatoid arthritis. Results showed that 5G1.1 appeared to be safe and well tolerated in patients in this trial. The most commonly observed adverse events were nausea and diarrhea. The results of this study suggested a significant three-month efficacy as measured by ACR 20 criteria for the active arm with a dosage regimen starting with five weekly loading doses followed by monthly intravenous or IV administration, compared to placebo. The primary endpoint, or therapeutic pre-set goal, for this trial was met by the group of patients who received this mid-level dosing regimen of 5G1.1. Patients who received higher or lower doses of 5G1.1 in the clinical trial, did not achieve the primary endpoint. Our six-month safety data from this clinical trial showed that 5G1.1 appeared to be safe and well tolerated in this study population. Our on-going 12-month open label extension studies in RA and membranous nephritis will help us assess long-term safety.

We are planning to initiate two Phase IIb RA studies, one of which we expect may serve as a pivotal study if we obtain strong efficacy and safety results.

Membranous Nephritis

The kidneys are responsible for filtering blood to remove toxic metabolites or breakdown by-products and maintaining the minerals and proteins in the blood that are required for normal metabolism. Each kidney consists of millions of individual filtering units, or glomeruli. When glomeruli are damaged, the kidney can no longer adequately maintain its normal filtering function. This may result in the build-up of toxins in the blood and the loss of valuable minerals and proteins in the urine. Clinically severe nephritis, or kidney inflammation, is found in many patients suffering from lupus and other autoimmune diseases. This condition occurs when more than 90% of the kidney is destroyed by disease. Kidney failure is frequently associated with:

. hypertension;

- strokes;
- infections;
- anemia;
- heart, lung and joint inflammation;
- coma; and
- death.

Many forms of damage to the glomeruli are mediated by the immune system, particularly by antibodies and activated complement proteins. Membranous nephritis is a form of kidney inflammation that is believed to be caused by a chronic autoimmune disorder that targets the kidney. We estimate that there are approximately 100,000 people currently afflicted with membranous nephritis in the United States.

Membranous nephritis is characterized by kidney inflammation and dysfunction that may eventually progress to kidney failure. Diagnostic criteria for membranous nephritis include kidney biopsies that may demonstrate the presence of antibodies and activated complement byproducts in the kidneys of affected patients. The subsequent kidney inflammation leads to the abnormal loss of substantial amounts of protein in the patient's urine; this condition is known as proteinuria and is recognized as an objective measurement of kidney disease. Loss of protein in the urine disturbs the normal control of water in the blood vessels and also is believed to directly further injure the kidney. Moreover, clinical studies by others have shown that the degree of proteinuria is associated with the incidence of subsequent kidney failure. Additional clinical signs associated with proteinuria may include:

- abnormally low levels of protein in the blood;
- a propensity for abnormal blood clotting;
- abnormal lipid or fat elevations; and
- substantial swelling in the abdomen, under the skin and in the legs.

Current therapies for membranous nephritis include potentially toxic drugs more frequently used in other indications such as cancer. These drugs generally act to suppress broadly the proliferation of many types of cells, including white blood cells. We believe that the use of such therapies is generally limited due to their unfavorable side effects. Even with current therapies, in such a severe disease population more than 30% of the patients are expected to progress to renal or kidney failure, which may require dialysis or transplantation. In contrast, 5G1.1 directly targets the inhibition of deleterious complement activation. We believe 5G1.1 may exert more selective and effective anti-inflammatory activity without the adverse effects associated with current therapies.

We have performed pre-clinical studies in rodent models of nephritis and observed that C5 Inhibitor administration, as compared to placebo-treated subjects, substantially reduced:

- scarring of the kidney;
- breakdown of kidney tissue into the urine;
- clogging of the kidney filtering units; and
- proteinuria.

Clinical Trials - Membranous Nephritis

We are developing 5G1.1 for a group of kidney and kidney-related chronic autoimmune disorders, which include membranous nephritis, lupus nephritis, and lupus. Our strategy is to develop 5G1.1 in kidney disease by initially obtaining safety data in the more readily available lupus patient population and then to commence efficacy trials in patients with a kidney disorder known as membranous nephritis. We are initially starting Phase II trials with 5G1.1 for the treatment of membranous nephritis patients because of the more uniform clinical presentations of membranous nephritis patients as compared to lupus patients. We then intend to expand our efforts to conduct advanced clinical trials in other kidney diseases and lupus.

In June 1999, we announce the results of our initial Phase I single-center clinical trial in 24 lupus patients receiving a single bolus administration of 5G1.1. This trial showed that a single dose of 5G1.1 was safe and well tolerated, reduced complement activity in a dose-dependent manner, and a single 8.0 mg/kg dose significantly lowered the incidence of proteinuria.

In August 1999, we commenced a Phase II multi-center, double-blinded, randomized, placebo-controlled clinical safety and efficacy trial with multiple doses of 5G1.1 at two to four week dosing intervals that is intended to enroll approximately 120 membranous nephritis patients.

In February 2000, we announced that the FDA designated Fast Track status for development of 5G1.1 for the treatment of patients with membranous nephritis. This designation provides for expedited development and application review for approval of a drug through the FDA.

Lupus

Lupus is an autoimmune disorder that damages the brain, lungs, heart, joints and especially the kidneys. In lupus, antibodies deposit within particular organs causing complement activation, inflammation and tissue destruction. For decades, clinical studies by others have demonstrated the presence of complement activation in lupus patients undergoing flares. Studies have further shown an abundant deposition of activated complement proteins with localized inflammation in tissue biopsies from kidney or other tissues in lupus patients. The Lupus Foundation estimates that approximately 1.4 million people in the United States have lupus. Further, up to one-half of individuals estimated to be afflicted with lupus have nephritis. Although lupus may affect people of either gender, women are 10 to 15 times more likely to suffer from the disease than men.

Patients with active lupus may have a broad range of symptoms related to the antibody and activated complement deposition and inflammation. Inflammation of the brain may cause seizures and other neurologic abnormalities. Inflammation of the heart may cause heart failure or sudden death. Lung inflammation causes shortness of breath. Lupus may also cause swollen joints and arthritis. One of the most common complications associated with lupus, however, is kidney disease, which often leads to kidney failure requiring dialysis or transplantation.

Current therapies generally act to suppress broadly the proliferation of many types of cells, including white blood cells. In contrast, 5G1.1 directly targets the inhibition of deleterious complement activation. We believe 5G1.1 may exert more selective and effective anti-inflammatory activity without the adverse effects associated with current therapies.

We are developing 5G1.1 for the prevention and treatment of inflammation in lupus patients. We have performed pre-clinical studies in a rodent model of lupus. In this chronic rodent model that spontaneously develops a disease similar to lupus, substantially more animals treated with a C5 Inhibitor survived as compared to untreated control animals.

Clinical Trials - Lupus

We filed an IND with the FDA in late December 1997 for 5G1.1 in the treatment of patients suffering from lupus and began a Phase I clinical trial in lupus patients in July 1998. As discussed above, in the Clinical Trials section of Membranous Nephritis, we announced results of a 24 patient, placebo-controlled clinical study in June 1999. This trial showed that a single dose of 5G1.1 was safe and well tolerated, reduced complement activity in a dose-dependent manner, and a single 8.0 mg/kg dose significantly lowered the incidence of proteinuria.

In June 2001, we announced the commencement of a Phase II 5G1.1 trial for lupus nephritis. This trial is expected to enroll approximately 40 lupus nephritis patients at several clinical sites in the United States and is designed to test the safety and biological efficacy of chronic administration of 5G1.1 for up to six months. The National Institute of Health has awarded an approximately $1.0 million grant to the University of Colorado Health Sciences Center to fund this multi-center Phase II study of 5G1.1 in patients with lupus nephritis.

Psoriasis, Dermatomyositis, and Pemphigoid

In addition to the above disease indications, we filed INDs for, and commenced Phase I pilot clinical trials with, 5G1.1 in patients afflicted with the chronic, autoimmune disorders psoriasis, bullous pemphigoid, and dermatomyositis. Psoriasis is life-long autoimmune disorder in which the immune system attacks the patient's skin, which may cause red, painful and disfiguring scaling in the affected areas. According to the National Centers for Health Statistics, psoriasis afflicts approximately 3 million peoples in the U.S. with an estimated 2 million physician visits a year. Bullous pemphigoid is an autoimmune disorder in which the immune system attacks the patient's skin, which may cause extensive and striking blistering and is associated with one-year mortality rates of between 20 - 40%. According to published reports, approximately 2,500 new cases of pemphigoid are expected to be diagnosed each year in the U.S., providing an estimated prevalence of approximately 25,000 patients. Dermatomyositis is an autoimmune disorder in which the immune system attacks the patients' muscles and skin, which may cause extensive rash and progressive and severe muscle weakness, pain and fatigue. According to the Muscular Dystrophy Association, approximately 2,000 - 3,000 new cases of dermatomyositis are diagnosed each year in the U.S., with an estimated prevalence of approximately 5,000 - 20,000 patients.

Clinical Trials - Psoriasis, Dermatomyositis, and Pemphigoid

We are testing 5G1.1 in two separate pilot Phase I clinical trials in patients suffering from dermatomyositis, a severe inflammatory muscle disorder, and bullous pemphigoid, a severe inflammatory skin disorder. We recently completed a Phase I clinical trial to investigate the safety of 5G1.1 in psoriasis patients. 5G1.1 appeared to be safe and well tolerated in this patient population. According to a standard measure of disease activity, 5G1.1 treatment did not influence the outcome of psoriasis in this trial.

In October 2000, we announced that the FDA granted Orphan Drug status for development of 5G1.1 for the treatment of patients with dermatomyositis. The Orphan Drug designation provides Alexion with market exclusivity for 5G1.1 for this indication for seven years from the drug's approval date.

Antibody Discovery Technology Platform

Combinatorial Human Antibody Library Technologies

In order to expand our pipeline of potential antibody therapeutics, in September 2000, we acquired Prolifaron, Inc., a privately held biopharmaceutical company, through a merger with our newly organized, wholly owned subsidiary, Alexion Antibody Technologies, Inc. (AAT). AAT possesses extensive research expertise and technologies in the area of creating fully human antibodies from libraries containing billions of human antibody genes.

AAT's goal is to develop new fully human therapeutic antibodies addressing multiple disease areas, including autoimmune and inflammatory disorders, cancer and infectious disease. AAT's technologies involve

the generation of diverse libraries of human antibodies derived from patients' blood samples, and the screening of these libraries against a wide array of potential drug targets. We believe that these technologies may be optimally suited to the rapid generation of novel, fully human, therapeutic antibodies directed at validated clinical targets. To date, we have focused on identifying antibodies which may be therapeutically effective in different autoimmune or inflammatory disorders, cancer, and infectious diseases. In addition, we believe that these technologies could permit the pre-clinical validation of new gene targets that are coming out of the international effort to sequence the human genome. We also believe that these technologies might provide new therapeutic antibodies when the libraries are screened against certain of these new gene targets.

In May 2001, we announced the results of a pre-clinical study of a new class of therapeutic antibodies that accelerated the return to normal platelet levels in an animal model of bone marrow toxicity commonly found in cancer patients. Antibodies in this new class function as agonists that stimulate their cell target, rather than blocking it, and were created using a rational design and selection process utilizing proprietary technology developed at AAT. This new class of antibody agonists is designed to selectively bind to the c-Mpl receptor on the surface of platelet precursors and then to stimulate platelet-specific proliferation and differentiation both in vitro and in vivo.

Pre-Clinical Programs

Apogen T-Cell Immunotherapeutic Technology and Product Candidates

MP4

MP4 is a recombinant or genetically-modified protein consisting of parts of two brain-derived proteins. These two proteins are believed to be major targets of disease-causing T-cells in patients with multiple sclerosis. MP4 is designed to bind specifically to, and eliminate, the small population of T-cells in multiple sclerosis patients which are responsible for attacking the patient's brain cells, while leaving the vast majority of uninvolved T-cells unaffected. In addition, MP4 is designed to induce other white blood cells to suppress other inflammatory cells.

In February 1998, we filed an IND with the FDA for MP4 for the treatment of patients suffering from multiple sclerosis. The FDA has accepted a plan for a Phase I clinical trial of MP4. We will determine whether we will conduct this trial or continue to develop MP4. We may seek to license our rights to MP4 or to otherwise collaborate with a partner in its development.

UniGraft Xenotransplantation Technologies Program

Most transplant procedures today are whole organ transplants. We believe that there is a far greater number of patients with medical disorders, such as Parkinson's disease and spinal cord injury, that are caused by the functional loss of highly specialized cells. The number of these patients is likely to grow due to both the aging of the population, with subsequent increase in the incidence of degenerative diseases, and with the increasing incidence of trauma. Therefore, cell transplantation could be an important benefit to a large number of previously untreated, or severely under-treated patients suffering from severe medical disorders. However, since there are no human donors of such specialized cells, there is currently no available supply of such cells for replacement therapy. Further, the immune system prevents the transplantation of cells from other species, known as xenografts, as they are recognized by the immune system as foreign and they are rejected. We are developing a portfolio of UniGraft anti-rejection technologies designed to permit the therapeutic transplantation of such cells without rejection.

Although approximately 21,000 people received whole organ transplants in the United States in 1999, there are many times that number of patients who have disorders that may be amenable to cell or tissue transplantation. It is estimated that this broader population includes approximately 200,000 patients suffering from spinal cord injury and 1.0 million individuals with Parkinson's disease. In particular, we believe that use of a safe and effective cell transplantation therapy for patients with spinal cord injury or Parkinson's disease would

represent major therapeutic advances. We are developing a portfolio of UniGraft anti-rejection technologies designed to permit the therapeutic transplantation of such cells without rejection.

Neurologic Cell Transplantation

We have developed methods of blocking the immune system which are designed to permit the replacement of damaged human brain cells and other neurologic cells with potentially highly therapeutic genetically modified porcine or pig cells.

Rejection of non-human tissue by patients is generally believed to occur in two stages:

- the hyperacute phase, which is very rapid, extending from minutes to hours; and

- the acute phase, which is somewhat less rapid, extending from days to months.

Hyperacute rejection is generally believed to be mediated by naturally-occurring antibodies in the patient, most of which target a sugar antigen uniquely present on the surface of non-human tissue but not on the patient's own tissue. After binding to the foreign tissue, these antibodies stimulate the activation of the recipient's inactive complement proteins on the surface of the donor tissue with subsequent destruction of the donor tissue. Subsequently, acute rejection of xenografts is generally believed to be mediated by white blood cells.

We are designing UniGraft cell products to resist complement/antibody-mediated hyperacute rejection. We have commenced pre-clinical studies employing the UniGraft technologies during transplantation of genetically modified and proprietary porcine cells that are resistant to destruction by human complement proteins. We are currently focusing our anti-rejection and molecular engineering technologies primarily on the development of UniGraft cells to treat Parkinson's disease and injuries to the spinal cord. We are currently performing pre-clinical studies in the spinal cord injury and Parkinson's disease programs and optimizing manufacturing methods of the genetically modified pig cells.

Grants from Advanced Technology Program and National Institute of Standards and Technology

In November 1997, both ourselves and United States Surgical Corporation ("US Surgical", a division of Tyco International, Ltd.) were awarded a three-year $2.0 million cooperative agreement from the National Institute of Standards and Technology or NIST under its Advanced Technology Program for funding a joint xenotransplantation project. In February 1999, this funding was amended to a single company award to us with our reacquisition of the rights to all aspects of our xenotransplantation program from US Surgical which had been acquired by Tyco International Ltd. As of July 31, 2001, this award has been completed.

In October 1998, we were granted our third award under this program, a three-year grant supporting product development within our neurologic disorder transplantation program. Through the program, we may receive up to approximately $2.0 million over three years to support our UniGraft program to develop a spinal cord injury product within our neurologic disorder xenotransplantation program. Through July 31, 2001, we had received approximately $1.2 million under this award.

In November 1999, we were granted our fourth award under this program, a three-year grant supporting product development within our UniGraft program. Through the program, we may receive up to approximately $2.0 million over three years to support our production of UniGraft products. Through July 31, 2001, we had received approximately $442,000 under this award.

Strategic Alliance with Procter & Gamble

In January 1999, we entered into an exclusive collaboration with Procter & Gamble to develop and commercialize pexelizumab. Under this collaboration, we expect to initially pursue the development of pexelizumab for the treatment of inflammation caused by cardiopulmonary bypass surgery, myocardial infarction

or heart attack, and angioplasty. Under the current agreement, Procter & Gamble is expected to fund clinical development and manufacturing costs relating to pexelizumab for these indications. Additionally, under this agreement, Procter & Gamble has contracted to pay us up to $95 million in payments, which included a one-time non-refundable up-front license fee, as well as milestone and research and development support payments. In addition, we will receive royalties on worldwide sales of pexelizumab for all indications. We also have a preferred position relative to third-party manufacturers to manufacture pexelizumab worldwide. We share co-promotion rights with Procter & Gamble to market pexelizumab in the United States, and have granted Procter & Gamble the exclusive rights to sell, market and distribute pexelizumab outside of the United States. Through July 31, 2001, we received proceeds of $44.8 million from Procter & Gamble, including a non-refundable up-front license fee of $10.0 million and $34.8 million in research and development support payments.

Manufacturing

We obtain drug product to meet our requirements for clinical studies using both internal and third-party contract manufacturing capabilities. We have a pilot manufacturing plant suitable for the fermentation and purification of certain of our recombinant compounds for clinical studies. Our pilot plant has the capacity to manufacture under cGMP or current good manufacturing practices regulations. We have also secured the production of clinical supplies of certain other recombinant products through third-party manufacturers. In each case, we have contracted product finishing, vial filling, and packaging through third parties. Procter & Gamble is responsible for securing commercial supplies of pexelizumab.

To date, we have not invested in the development of commercial manufacturing capabilities. Although we have established a pilot manufacturing facility for the production of material for clinical trials for some of our potential products, we do not have sufficient capacity to manufacture more than one drug candidate at a time or to manufacture our drug candidates for later stage clinical development or commercialization. In the longer term, we may contract the manufacture of our products for commercial sale or may develop large-scale manufacturing capabilities for the commercialization of some of our products. The key factors which will be given consideration when making the determination of which products will be manufactured internally and which through contractual arrangements will include the availability and expense of contracting this activity, control issues and the expertise and level of resources required for us to manufacture products. In addition, as our product development efforts progress, we expect that we will need to hire additional personnel skilled in product testing and regulatory compliance.

Sales and Marketing

We currently have no sales, marketing, or distribution capabilities. We will need to establish or contract these capabilities to commercialize successfully any of our drug candidates. We may promote our products in collaboration with marketing partners or rely on relationships with one or more companies with established distribution systems and direct sales forces. Under our current collaboration agreement, Procter & Gamble is obligated to sell, market and distribute pexelizumab for all approved indications worldwide. We share with Procter & Gamble co-promotion rights for pexelizumab in the United States. For other future drug products, as well as for pexelizumab in the United States, we may elect to establish our own specialized sales force and marketing organization to market our products.

Patents and Proprietary Rights

Patents and other proprietary rights are important to our business. Our policy is to file patent applications to protect technology, inventions and improvements to our technologies that are considered important to the development of our business. We also rely upon our trade secrets, know-how, and continuing technological innovations to develop and maintain our competitive position, as well as patents that we have licensed or may license from other parties.

We have filed several U.S. patent applications and international counterparts of certain of these applications. In addition, we have licensed several additional U.S. and international patents and patent

applications. Of our owned and licensed patents and patent applications as of July 31, 2001, 18 relate to technologies or products in the C5 Inhibitor program, 8 relate to the Apogen program, 33 relate to the UniGraft program, 11 relate to the recombinant human antibody program and 1 relates to the high throughput compound screening program. We will owe royalties and other fees to the licensors of some of those patents and patent applications in connection with the commercial manufacture and sale of our expected product candidates, including, pexelizumab and 5G1.1.

Our success will depend in part on our ability to obtain United States and international patent protection for our products and development programs, to preserve our trade secrets and proprietary rights, and to operate without infringing on the proprietary rights of third parties or having third parties circumvent our rights. Because of the length of time and expense associated with bringing new products through development and regulatory approval to the marketplace, the health care industry has traditionally placed considerable importance on obtaining patent and trade secret protection for significant new technologies, products and processes.

We are aware of broad patents owned by third parties relating to the manufacture, use, and sale of recombinant humanized antibodies, recombinant humanized single-chain antibodies, recombinant human antibodies, recombinant human single-chain antibodies, and genetically engineered animals. Many of our product candidates are genetically engineered antibodies, including recombinant humanized antibodies, recombinant humanized single chain antibodies, recombinant human antibodies, and recombinant human single chain antibodies, and other products are tissues from animals. We have received notices from the owners of some of these patents in which the owners claim that some of these patents may be relevant to the development and commercialization of some of our drug candidates. With respect to certain of these patents which we believe are relevant for the expeditious development and commercialization of certain of our products as currently contemplated, we have acquired licenses. With regard to certain other patents, we have either determined in our judgment that our products do not infringe the patents or we can license such patents on commercially reasonable terms or we have identified and are testing various approaches which we believe should not infringe the patents and which should permit commercialization of our products.

It is our policy to require our employees, consultants, members of our scientific advisory board, and parties to collaborative agreements to execute confidentiality agreements upon the commencement of employment or consulting relationships or collaborations with us. These agreements generally provide that all confidential information developed or made known during the course of relationship with us is to be kept confidential and not to be disclosed to third parties except in specific circumstances. In the case of employees, the agreements provide that all inventions resulting from work performed for us, utilizing our property or relating to our business and conceived or completed by the individual during employment shall be our exclusive property to the extent permitted by applicable law.

Government Regulation

The pre-clinical studies and clinical testing, manufacture, labeling, storage, record keeping, advertising, promotion, export, and marketing, among other things, of our proposed products are subject to extensive regulation by governmental authorities in the United States and other countries. In the United States, pharmaceutical products are regulated by the FDA under the Federal Food, Drug, and Cosmetic Act and other laws, including, in the case of biologics, the Public Health Service Act. At the present time, we believe that our currently anticipated products will be regulated by the FDA as biologics.

The steps required before a novel biologic may be approved for marketing in the United States generally include:

(1) pre-clinical laboratory tests and in vivo, or within a living organism, pre-clinical studies;

(2) the submission to the FDA of an IND for human clinical testing, which must become effective before human clinical trials may commence;

(3) adequate and well-controlled human clinical trials to establish the safety and efficacy of the product;

(4) the submission to the FDA of a biologics license application or BLA; and

(5) FDA review and approval of such application.

The testing and approval process requires substantial time, effort and financial resources. Prior to and following approval, if granted, the establishment or establishments where the product is manufactured are subject to inspection by the FDA and must comply with cGMP requirements enforced by the FDA through its facilities inspection program. Manufacturers of biological materials also may be subject to state regulation.

Pre-clinical studies include animal studies to evaluate the mechanism of action of the product, as well as animal studies to assess the potential safety and efficacy of the product. Pre-clinical safety tests must be conducted in compliance with FDA regulations regarding good laboratory practices and compounds used for clinical trials must be produced according to applicable cGMP requirements. The results of the pre-clinical tests, together with manufacturing information and analytical data, are submitted to the FDA as part of an IND, which must become effective before human clinical trials may be commenced. The IND will automatically become effective 30 days after receipt by the FDA, unless the FDA before that time requests an extension to review or raises concerns about the conduct of the trials as outlined in the application. In such latter case, the sponsor of the application and the FDA must resolve any outstanding concerns before clinical trials can proceed. We cannot assure you that submission of an IND will result in FDA authorization to commence clinical trials.

Clinical trials involve the administration of the investigational product to healthy volunteers or to patients, under the supervision of a qualified principal investigator. Clinical trials are conducted in accordance with protocols that detail many items, including:

. 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 at each clinical site must be reviewed and approved by an independent institutional review board, prior to the recruitment of subjects.

Clinical trials typically are conducted in three sequential phases, but the phases may overlap and different trials may be initiated with the same drug within the same phase of development in similar or differing patient populations. In Phase I, the initial introduction of the drug into human subjects, the drug is tested for safety and, as appropriate, for absorption, metabolism, distribution, excretion, pharmacodynamics and pharmacokinetics. Phase II usually involves studies in a limited patient population to:

. evaluate preliminarily the efficacy of the drug for specific, targeted indications;

. determine dosage tolerance and optimal dosage; and

. identify possible adverse effects and safety risks.

Phase III trials are undertaken to further evaluate clinical efficacy of a specific endpoint and to test further for safety within an expanded patient population at geographically dispersed clinical study sites. Phase I, Phase II or Phase III testing may not be completed successfully within any specific time period, if at all, with respect to any products being tested by a sponsor. Furthermore, the FDA may suspend clinical trials at any time

on various grounds, a finding that the subjects or patients are being exposed to an unacceptable health risk.

The results of the pre-clinical studies and clinical trials, together with detailed information on the manufacture and composition of the product, are submitted to the FDA as part of a BLA requesting approval for the marketing of the product. The FDA will occasionally convene an Advisory Panel of experts to review and recommend a non-binding course of action regarding a sponsor's BLA requests. The FDA may deny approval of the application if applicable regulatory criteria are not satisfied, or if additional testing or information is required. Post-marketing testing and surveillance to monitor the safety or efficacy of a product may be required. FDA approval of any application may include many delays or never be granted. Moreover, if regulatory approval of a product is granted, such approval may entail limitations on the indicated uses for which it may be marketed. Finally, product approvals may be withdrawn if compliance with regulatory standards is not maintained or if safety or manufacturing problems occur following initial marketing. Among the conditions for approval is the requirement that the prospective manufacturer's quality control and manufacturing procedures conform to cGMP requirements. These requirements must be followed at all times in the manufacture of the approved product. In complying with these requirements, manufacturers must continue to expend time, monies and effort in the area of production and quality control to ensure full compliance.

Both before and after the FDA approves a product, the manufacturer and the holder or holders of the BLA for the product are subject to comprehensive regulatory oversight. Violations of regulatory requirements at any stage, including the pre-clinical and clinical testing process, the review process, or at any time afterward, including after approval, may result in various adverse consequences, including the FDA's delay in approving or refusal to approve a product, withdrawal of an approved product from the market, and/or the imposition of criminal penalties against the manufacturer and/or the BLA holder. In addition, later discovery of previously unknown problems may result in restrictions on a product, its manufacturer, or the BLA holder, including withdrawal of the product from the market. Also, new government requirements may be established that could delay or prevent regulatory approval of our products under development.

For clinical investigation and marketing outside the United States, we are also subject to foreign regulatory requirements governing human clinical trials and marketing approval for drugs. The foreign regulatory approval process includes all of the risks associated with FDA approval set forth above as well as country-specific regulations.

No xenotransplantation-based therapeutic product has been approved for sale by the FDA. The FDA has not yet established definitive regulatory guidelines for xenotransplantation, but has proposed interim guidelines in an attempt to reduce the risk of contamination of transplanted organ and cellular products with infectious agents. Definitive guidelines in the United States may never be issued, if at all. Current companies involved in this field, including ourselves, may not be able to comply with any final and definitive federal guidelines that may be issued.

Competition

Currently, many companies, including major pharmaceutical and chemical companies as well as specialized biotechnology companies, are engaged in activities similar to our activities. Universities, governmental agencies and other public and private research organizations also conduct research and may market commercial products on their own or through joint ventures. These companies and organizations are in the United States, Europe and elsewhere. Many of these entities may have:

- substantially greater financial and other resources;
- larger research and development staffs;
- lower labor costs; and/or

. more extensive marketing and manufacturing organizations.

Many of these companies and organizations have significant experience in pre-clinical testing, human clinical trials, product manufacturing, marketing and distribution and other regulatory approval procedures. They may also have a greater number of significant patents and greater legal resources to seek remedies for cases of alleged infringement of their patents by us to block, delay, or compromise our own drug development process.

We compete with large pharmaceutical companies that produce and market synthetic compounds and with specialized biotechnology firms in the United States, Europe and elsewhere, as well as a growing number of large pharmaceutical companies that are applying biotechnology to their operations. Many biotechnology companies have focused their developmental efforts in the human therapeutics area, and many major pharmaceutical companies have developed or acquired internal biotechnology capabilities or have made commercial arrangements with other biotechnology companies. A number of biotechnology and pharmaceutical companies are developing new products for the treatment of the same diseases being targeted by us; in some instances these products have already entered clinical trials or are already being marketed. Other companies are engaged in research and development based on complement proteins, T-cell therapeutics, gene therapy and xenotransplantation.

Each of Avant Immunotherapeutics, Inc., Millennium Pharmaceuticals, Inc., Tanox, Inc., Abbott Laboratories, Baxter International Inc., Gliatech Inc., Neurogen Corporation, and Biocryst Pharmaceuticals Inc. has publicly announced intentions to develop complement inhibitors to treat diseases related to trauma, inflammation or certain brain or nervous system disorders. Avant has initiated clinical trials for a proposed complement inhibitor to treat acute respiratory distress syndrome, myocardial infarction, lung transplantation, and in infants and adults undergoing heart and/or lung bypass procedures. We are aware that Pfizer, Inc., GlaxoSmithKline plc, and, Merck & Co., Inc. are also attempting to develop complement inhibitor therapies. We believe that our potential C5 Inhibitors differ substantially from those of our competitors due to our compounds' demonstrated ability to specifically intervene in the complement cascade at what we believe to be the optimal point so that the disease-causing actions of complement proteins generally are inhibited while the normal disease-preventing functions of complement proteins generally remain intact as do other aspects of immune function.

We further believe that, under conditions of inflammation, a complement inhibitor compound which only indirectly addresses the harmful activity of complement may be bypassed by pathologic mechanisms present in the inflamed tissue. Each of Bayer AG, Immunex Corp., Pharmacia & Upjohn Inc. and Rhone-Poulenc SA sells a product which is used clinically to reduce surgical bleeding during cardiopulmonary bypass surgery, but has little beneficial effect on other significant inflammatory morbidities associated with cardiopulmonary bypass surgery. We believe that each of these drugs does not significantly prevent complement activation and subsequent inflammation that lead to organ damage and blood loss during cardiopulmonary bypass surgery, but instead each drug attempts to reduce blood loss by shifting the normal blood thinning/blood clotting balance in the blood towards enhanced blood clotting.

Nextran Inc., a subsidiary of Baxter International Inc., and Imutran Ltd., a wholly-owned subsidiary of Novartis Pharma AG, are seeking to develop pig cell xenograft technology. Novartis Pharma AG is also collaborating with Biotransplant Inc. to commercially develop xenograft organs in a joint venture known as Immerge, Inc. We are aware that Diacrin Inc. and Genzyme Tissue Repair, Inc. are working in this field.

Each of Cambridge Antibody Technology, PLC, MorphoSys AG, and Dyax Corporation have publicly announced intentions to develop therapeutic genetically altered human antibodies from libraries of human antibody genes. Additionally, each of Abgenix, Inc. and Medarex, Inc. have publicly announced intentions to develop therapeutic genetically altered human antibodies from mice that have been bred to include some human antibody genes.

Employees

As of October 1, 2001, we had 140 full-time employees, of which 123 were engaged in research, development, manufacturing, and clinical development, and 17 in administration and finance. Doctorates are held by 42 of our employees. Each of our employees has signed a confidentiality agreement.

Item 2. PROPERTIES.

Facilities

We lease our headquarters and research and development facilities in Cheshire, Connecticut, where we relocated in November 2000. The lease has a term of ten years and six months. At this site, we lease a total of approximately 82,000 square feet of space, which includes approximately 62,000 square feet related to research and laboratories. We have incurred initial leasehold improvements aggregating approximately $4.8 million. In addition, we are paying a pro rata percentage of real estate taxes and operating expenses. Our pilot manufacturing plant, which may be used for producing compounds for some of our current and anticipated clinical trials, is expected to remain in New Haven, Connecticut encompassing approximately 30,000 square feet of labs and offices. We are currently negotiating a longer-term arrangement for this facility. We believe our new facilities and our pilot manufacturing facility will be adequate for our ongoing current clinical activities. Alexion Antibody Technologies, Inc. leases approximately 7,500 square feet of labs and office space in San Diego, California.

Item 3. LEGAL PROCEEDINGS.

We are not a party to any material legal proceeding.

Item 4. SUBMISSION OF MATTERS TO A VOTE OF SECURITY HOLDERS.

None.

EXECUTIVE OFFICERS AND KEY EMPLOYEES OF THE COMPANY

The executive officers and key employees of the Company and their respective ages and positions with the Company as of October 1, 2001 were as follows:

Name	Age	Position with Alexion
Leonard Bell, M.D.	43	President, Chief Executive Officer, Secretary, Treasurer, Director
David W. Keiser	50	Executive Vice President, Chief Operating Officer
Stephen P. Squinto, Ph.D.	45	Executive Vice President and Head of Research
Katherine S. Bowdish, Ph.D.	44	Senior Vice President of Antibody Discovery, President of Alexion Antibody Technologies
Samuel S. Chu, Ph.D.	51	Vice President of Process Sciences and Manufacturing
Thomas I.H. Dubin, J.D.	39	Vice President and General Counsel
Barry P. Luke	43	Vice President of Finance and Administration, Assistant Secretary
Christopher F. Mojcik, M.D., Ph.D.	41	Vice President of Clinical Development
Nancy Motola, Ph.D.	48	Vice President of Regulatory Affairs and Quality Assurance
Scott A. Rollins, Ph.D.	38	Vice President of Drug Development and Project Management
Russell P. Rother, Ph.D.	40	Vice President of Discovery Research

Daniel N. Caron.....	38	Senior Director of Operations and Engineering
William Fodor, Ph.D.....	43	Senior Director of Xenotransplantation

Leonard Bell, M.D. is the principal founder of Alexion, and has been a director of Alexion since February 1992 and the Company's President and Chief Executive Officer, Secretary and Treasurer since January 1992. From 1991 to 1992, Dr. Bell was an Assistant Professor of Medicine and Pathology and co-Director of the program in Vascular Biology at the Yale University School of Medicine. From 1990 to 1992, Dr. Bell was an attending physician at the Yale-New Haven Hospital and an Assistant Professor in the Department of Internal Medicine at the Yale University School of Medicine. Dr. Bell was a recipient of the Physician Scientist Award from the National Institutes of Health and Grant-in-Aid from the American Heart Association as well as various honors and awards from academic and professional organizations. His work has resulted in more than 20 scientific publications and three patent applications. Dr. Bell is a director of The Medicines Company, and Connecticut United for Research Excellence, Inc. He also served as a director of the Biotechnology Research and Development Corporation from 1993 to 1997. Dr. Bell received his A.B. from Brown University and M.D. from Yale University School of Medicine. Dr. Bell is currently an Adjunct Assistant Professor of Medicine and Pathology at Yale University School of Medicine.

David W. Keiser has been Executive Vice President and Chief Operating Officer of Alexion since July 1992. From 1990 to 1992, Mr. Keiser was Senior Director of Asia Pacific Operations for G.D. Searle & Company Limited, a manufacturer of pharmaceutical products. From 1986 to 1990, Mr. Keiser was successively Licensing Manager, Director of Product Licensing and Senior Director of Product Licensing for Searle. From 1984 to 1985, Mr. Keiser was New Business Opportunities Manager for Mundipharma AG, a manufacturer of pharmaceutical products, in Basel, Switzerland where he headed pharmaceutical licensing and business development activities in Europe and the Far East. From 1978 to 1983, he was Area Manager for F. Hoffmann La Roche Ltd., a manufacturer of pharmaceutical products, in Basel, Switzerland. Mr. Keiser received his B.A. from Gettysburg College.

Stephen P. Squinto, Ph.D. is a founder of Alexion and has been Executive Vice President and Head of Research since August 2000. He has held the positions of Senior Vice President and Chief Technical Officer from March 1998 to July 2000, Vice President of Research, Molecular Sciences, from August 1994 to March 1998, Senior Director of Molecular Sciences from July 1993 to July 1994, and Director of Molecular Development from 1992 to July 1993. From 1989 to 1992 Dr. Squinto held various positions at Regeneron Pharmaceuticals, Inc. most recently serving as Senior Scientist and Assistant Head of the Discovery Group. From 1986 to 1989, Dr. Squinto was an Assistant Professor of Biochemistry and Molecular Biology at Louisiana State University Medical Center. Dr. Squinto's work has led to over 70 scientific papers in the fields of gene regulation, growth factor biology and gene transfer. Dr. Squinto's work is primarily in the fields of regulation of eukaryotic gene expression, mammalian gene expression systems and growth receptor and signal transduction biology. Dr. Squinto also serves as a Director of the BRDC since 1997. Dr. Squinto received his B.A. in Chemistry and Ph.D. in Biochemistry and Biophysics from Loyola University of Chicago.

Katherine S. Bowdish, Ph.D. has been Senior Vice President of Antibody Discovery since August 2001 and was Vice President of Antibody Discovery from September 2000 upon joining the Company. Dr. Bowdish has also been President of Alexion Antibody Technologies, Inc., a wholly-owned subsidiary of the Company, since September 2000. Dr. Bowdish was a co-founder and Chief Scientific Officer and Executive Vice President of Prolifaron, Inc. Prolifaron, a San Diego, California based antibody engineering company was merged into Alexion Antibody Technologies, Inc. in September 2000. From 1997 to 1998, and the Chief Executive Officer and Chief Scientific Officer of Prolifaron from 1998 to 2000. Dr. Bowdish previously held positions at The Scripps Research Institute, Monsanto, and Rockefeller University. Dr. Bowdish is an internationally recognized expert in the field of antibody engineering and has 19 years of experience in biotechnology research. Dr. Bowdish received her B.S. degree in biology from the College of William and Mary, M.A. degree in cell biology from Columbia University, and Ph.D. degree in genetics from Columbia University.

Samuel S. Chu, Ph.D. has been Vice President of Process Sciences and Manufacturing since September 2000. Before joining Alexion Dr. Chu was Director of the Biotech Development and Pilot Plant, Bio-Chemistry Division operations at Bristol-Meyers Squibb, Co from 1993 to 2000. From 1990 to 1993 Dr. Chu was an Associate Director of Product Development and Scale-up at Lederle-Praxis Biologicals, a division of American Cyanamid. From 1985 to 1990 Dr. Chu was the Associate Director of Product Development and Scale-up at Praxis Biologics. Dr. Chu received his B.S. from National Chung-Hsing University, M.S. from Illinois Institute of Technology, and Ph.D. degree from the University of Toronto.

Thomas I.H. Dubin, J.D. joined the Company in January 2001 as Vice President and General Counsel. From February 1999 to September 2000 he served as Vice President, General Counsel and Secretary for ChiRex Inc., a NASDAQ-traded international corporation providing advanced process development services and specialty manufacturing to the pharmaceutical industry, which in September 2000 was acquired by and merged into Rhodia. From 1992 to 1999, Mr. Dubin held various positions with Warner-Lambert Company, including Assistant General Counsel, Pharmaceuticals. Prior to his tenure with Warner-Lambert, Mr. Dubin was a corporate attorney for five years with Cravath, Swaine & Moore in New York. Mr. Dubin received his J.D. from New York University and his B.A., cum laude, from Amherst College.

Barry P. Luke has been Vice President of Finance and Administration since September 1998 and Senior Director of Finance and Administration of Alexion from August 1995 to September 1998 and prior thereto as Director of Finance and Accounting of the Company from May 1993. From 1989 to 1993, Mr. Luke was Chief Financial Officer, Secretary and Vice President-Finance and Administration at Comtex Scientific Corporation, a publicly held distributor of electronic news and business information. From 1985 to 1989, he was Controller and Treasurer of Softstrip, Inc., a manufacturer of computer peripherals and software. From 1980 to 1985, Mr. Luke was employed by General Electric Company where he held positions at GE's Corporate Audit Staff after completing GE's Financial Management Program. Mr. Luke received a B.A. in Economics from Yale University and an M.B.A. in management and marketing from the University of Connecticut.

Christopher F. Mojcik, M.D., Ph.D. has been Vice President of Clinical Development since August 2000. Since joining the Alexion in July 1998 to July 2000, Dr. Mojcik was Senior Director of Clinical Development. From 1996 until July 1998, he was an Associate Director in the Metabolics/Rheumatics Department at Bayer Corporation's Pharmaceuticals Division. Dr. Mojcik was responsible for Phase II and III development of certain arthritis programs and certain Phase IV programs in cardiopulmonary bypass. From 1993 to 1996, he was a Senior Staff Fellow in the Cellular Immunology Section of the Laboratory of Immunology in the NIAID at the NIH. From 1991 to 1993, he completed his Fellowship in Rheumatology in the National Institute of Arthritis and Musculoskeletal and Skin Diseases at the NIH. He received his B.A. from Washington University in St. Louis, Missouri, and his M.D. and Ph.D. from the University of Connecticut.

Nancy Motola, Ph.D. has been the Vice President of Regulatory Affairs and Quality Assurance since 1998. From 1991 to 1998, Dr. Motola served as Assistant, Associate and then Deputy Director, Regulatory affairs for the Bayer Corporation Pharmaceuticals Division where she was responsible for regulatory aspects of product development programs for cardiovascular, neuroscience, metabolic and oncology drugs and included drugs targeting arthritis, cardiac disorders, stroke and cognitive dysfunction. Dr. Motola has been responsible for the filing of numerous INDs, other regulatory submissions and has filed New Drug Applications for marketing approval resulting in three currently marketed drugs. Dr. Motola held regulatory affairs positions of increasing responsibility at Abbott Laboratories from 1989 to 1991 and at E.R. Squibb and Sons, Inc. from 1983 to 1989. She also served as past Chairperson of the Regulatory Affairs Section of the American Association of Pharmaceuticals Scientists. Dr. Motola received her B.A. from Central Connecticut State University and M.S. and Ph.D. degrees in medical chemistry from the University of Rhode Island.

Scott A. Rollins, Ph.D. is a co-founder of Alexion and has been Vice President of Drug Development and Project Management since August 2000. Dr. Rollins was Senior Director of Project Management and Drug Development from August 1999 to July 2000, Senior Director of Complement Biology from 1997 to 1999, Director of Complement Biology from 1996 to 1997, Principal Scientist from 1994 to 1996, and Staff Scientist from 1992 to 1994. Since 1994, Dr. Rollins has been responsible for the pre-clinical development of our anti-

inflammatory compound 5G1.1-SC. Since 1999, Dr. Rollins has been additionally responsible for the project management functions of 5G1.1-SC, currently under joint development with Procter & Gamble Pharmaceuticals. Prior to 1992, Dr. Rollins was a postdoctoral research fellow in the Department of Immunobiology at Yale University School of Medicine. Dr. Rollins' work has led to over 50 scientific papers and patents in the fields of complement biology. He received his B.S. in Cytotechnology and Ph.D. in Microbiology and Immunology from the University of Oklahoma Health Sciences Center.

Russell P. Rother, Ph.D. has been Vice President of Discovery Research since August 2001, Senior Director of Discovery Research from 1999 to 2001, Director of Gene Technologies from 1996 to 1999, Senior Staff Scientist from 1994 to 1996 and Staff Scientist from 1992 to 1994. As one of the original scientists at Alexion, Dr. Rother has played a critical role in the engineering and development of Alexion's current antibody therapeutics and continues to lead discovery efforts in the identification of new targets. Prior to 1992, Dr. Rother was a Postdoctoral Research Fellow in the Department of Immunobiology at Yale University School of Medicine. Dr. Rother's work has led to over 30 scientific papers and issued patents in the fields of gene therapy, autoimmunity and complement biology. Dr. Rother received a B.S. in Biology from Southwestern Oklahoma State University and a Ph.D. in Microbiology and Immunology from the University of Oklahoma Health Sciences Center in conjunction with the Oklahoma Medical Research Foundation.

Daniel N. Caron has been Senior Director of Operations and Engineering since 1998. After joining the Company in 1992, Mr. Caron was Operations Manager from 1992 to 1993, Senior Operations Manager from 1993 to 1996, and Director of Operations from 1996 to 1998. Mr. Caron has been responsible for managing the engineering, build-out, validation and maintenance of all of the Company's research, manufacturing, and administrative facilities. Prior to 1992, Mr. Caron was a research scientist at Imclone Systems, Inc., a biopharmaceutical firm. Mr. Caron received his B.A. in Biology from Adelphia University and M.S. in Biomedical Engineering from Polytechnic University of New York.

William Fodor, Ph.D. has been Senior Director of Xenotransplantation since 1997. After joining Alexion in 1992, Dr. Fodor was a Staff Scientist from 1992 to 1994, Principal Scientist from 1994 to 1996, and Director of Xenotransplantation from 1996 to 1997. Dr. Fodor has been responsible for managing the pre-clinical development and manufacturing of our xenotransplantation product candidates. Prior to 1992, Dr. Fodor was a postdoctoral research fellow in the Section of Immunobiology at Yale University School of Medicine and at Biogen, Inc., a biopharmaceutical firm. Dr. Fodor's work has led to over 30 scientific papers and patents in the fields of immunobiology and molecular biology. Dr. Fodor received his B.S. in Genetics and Ph.D. in Molecular Genetics from Ohio State University.

PART II

Item 5. MARKET FOR REGISTRANT'S COMMON EQUITY AND RELATED STOCKHOLDER MATTERS

Our common stock is quoted on The Nasdaq National Market under the symbol "ALXN." The following table sets forth the range of high and low sales prices for our common stock on The Nasdaq National Market for the periods indicated since August 1, 1998.

Fiscal 1999	High	Low
First Quarter (August 1, 1998 to October 31, 1998)	$ 10.25	$ 5.50
Second Quarter (November 1, 1998 to January 31, 1999)	$ 17.75	$ 8.38
Third Quarter (February 1, 1999 to April 30, 1999)	$ 14.25	$ 8.38
Fourth Quarter (May 1, 1999 to July 31, 1999)	$ 12.75	$ 8.75

Fiscal 2000	High	Low
First Quarter (August 1, 1999 to October 31, 1999)	$ 16.25	$10.00
Second Quarter (November 1, 1999 to January 31, 2000)	$ 50.13	$12.75
Third Quarter (February 1, 2000 to April 30, 2000)	$119.88	$34.81
Fourth Quarter (May 1, 2000 to July 31, 2000)	$ 84.50	$30.50

Fiscal 2001	High	Low
First Quarter (August 1, 2000 to October 31, 2001)	$118.63	$64.00
Second Quarter (November 1, 2000 to January 31, 2001)	$112.00	$42.75
Third Quarter (February 1, 2001 to April 30, 2001)	$ 54.50	$16.88
Fourth Quarter (May 1, 2001to July 31, 2001)	$ 29.99	$18.50

As of October 23, 2001, we had 141 stockholders of record of our common stock and an estimated 4,000 beneficial owners. The closing sale price of our common stock on October 23, 2001 was $17.83 per share.

In March 2000, we completed a $120 million private placement of our 5.75% Convertible Subordinated Notes due March 15, 2007. The notes bear interest semi-annually on September 15 and March 15 of each year, beginning September 15, 2000. The holders of the notes may convert all or a portion of the notes into common stock at any time on or before March 15, 2007 at a conversion price of $106.425 per share. The notes were offered to qualified institutional buyers under the exemption from registration provided by Rule 144A under the Securities Act of 1933, as amended, and to persons outside the United States under Regulation S under the Securities Act. We incurred issuance costs related to this offering of approximately $4.0 million, including discounts to J.P. Morgan & Co., U.S. Bancorp Piper Jaffray, Chase H&Q and Warburg Dillon Read LLC, the

initial purchasers of the notes. The costs are being amortized into interest expense over the seven-year term of the notes.

In October 2000, we filed a shelf registration statement to offer up to $300 million of equity securities. On November 1, 2000, we sold 2.3 million shares of common stock at a price of $90.75 per share resulting in net proceeds of approximately $208.5 million, net of fees and other expenses of approximately $201,000 related to the transaction.

DIVIDEND POLICY

We have never paid cash dividends. We do not expect to declare or pay any dividends on our common stock in the foreseeable future. We intend to retain all earnings, if any, to invest in our operations. The payment of future dividends is within the discretion of our board of directors and will depend upon our future earnings, if any, our capital requirements, financial condition and other relevant factors.

Item 6. SELECTED CONSOLIDATED FINANCIAL DATA.

(in thousands, except per share data)

Consolidated Statements of Operations Data:	Fiscal Year Ended July 31,				
	2001	2000	1999	1998	1997
Contract research revenues	$ 11,805	$ 21,441	$ 18,754	$ 5,037	$ 3,811
Operating expenses:					
Research and development	38,871	40,187	23,710	12,323	9,079
General and administrative	7,135	4,175	2,953	2,666	2,827
In-process research and development (IPRD)	21,000	-	-	-	-
Amortization of goodwill (GW)	2,901	-	-	-	-
Total operating expenses	69,907	44,362	26,663	14,989	11,906
Operating loss	(58,102)	(22,921)	(7,909)	(9,952)	(8,095)
Other income, net	10,177	2,694	1,514	2,087	843
Loss before cumulative effect of SAB 101	(47,925)	(20,227)	(6,395)	(7,865)	(7,252)
Cumulative effect of adoption of SAB 101	(9,118)	-	-	-	-
Net Loss	$ (57,043)	$(20,227)	$ (6,395)	$(7,865)	$(7,252)
Preferred Stock dividends	-	-	-	(900)	-
Net loss applicable to common shareholders	$ (57,043)	$(20,227)	$ (6,395)	$(8,765)	$(7,252)
Basic and diluted net loss per common share	$ (3.28)	$ (1.45)	(0.57)	$ (0.87)	$ (0.97)
Shares used in computing net loss per common share	17,371	13,914	11,265	10,056	7,451

Consolidated Balance Sheet Data:	As of July 31,				
	2001	2000	1999	1998	1997
Cash, cash equivalents, and marketable securities	$355,274	$174,529	$28,328	$37,494	$22,749
Total current assets	362,747	180,080	35,662	37,840	22,981
Total assets	400,259	192,702	44,374	42,085	24,260
Notes payable, less current portion	3,920	3,920	4,383	832	-
Convertible subordinated notes	120,000	120,000	-	-	-
Total stockholders' equity	260,408	61,604	33,301	39,190	21,846

Item 7. MANAGEMENT'S DISCUSSION AND ANALYSIS OF FINANCIAL CONDITION AND RESULTS OF OPERATIONS.

This report contains forward-looking statements which involve risks and uncertainties. Such statements are subject to certain factors which may cause our plans and results to differ significantly from plans and results discussed in forward-looking statements. Factors that might cause or contribute to such differences include, but are not limited to, those discussed in "Important Factors Regarding Forward-Looking Statements" attached hereto as Exhibit 99.

Overview

We are engaged in the discovery and development of therapeutic products aimed at treating patients with a wide array of severe disease states, including cardiovascular and autoimmune disorders, inflammation and cancer. Since our inception in January 1992, we have devoted substantially all of our resources to drug discovery, research, product and clinical development. Additionally, through our wholly owned subsidiary, Alexion Antibody Technologies, Inc. or AAT, we are engaged in discovering and developing a portfolio of additional antibody therapeutics targeting severe unmet medical needs.

We are currently examining our two lead antibody product candidates in eight clinical development programs. One of our antibody product candidates, pexelizumab, is an antibody fragment being developed in collaboration with Procter & Gamble Pharmaceuticals, and has completed a Phase IIb study in CPB or cardiopulmonary bypass surgery patients undergoing CABG or coronary artery bypass graft surgery. Pending FDA or Food and Drug Administration discussions, we expect to initiate a Phase III efficacy trial with pexelizumab in CPB patients at the earliest possible opportunity. Also in collaboration with Procter & Gamble, we are currently conducting two additional large Phase II studies with pexelizumab in acute myocardial infarction or heart attack patients: one study in patients receiving thrombolytic therapy, a procedure for dissolving clots that block heart vessels, and the other in patients receiving angioplasty, a procedure for opening up narrowed or blocked arteries that supply blood to the heart. In September 2000, the FDA granted Fast Track status for the development of pexelizumab in CPB.

Our other lead antibody product candidate, 5G1.1, is in clinical development for the treatment of a variety of chronic autoimmune diseases. We initiated a Phase II study in lupus nephritis, a kidney disease, and a separate Phase II study in membranous nephritis, a kidney disease, is ongoing. We completed a Phase II clinical study in rheumatoid arthritis or RA patients and we are planning to initiate two Phase IIb RA studies, one of which we expect may serve as a pivotal study if we obtain strong efficacy and safety results.

In both rheumatoid arthritis and membranous nephritis, enrollment has commenced in additional 12-month open-label extension studies to test long-term safety. In addition, we have two separate on-going early stage clinical programs to study 5G1.1 in patients with dermatomyositis, a muscle disorder, and bullous pemphigoid, a severe inflammatory skin disorder. In October 2000, the FDA granted us Orphan Drug status for the development of 5G1.1 for the treatment of dermatomyositis. We recently completed a Phase I pilot safety trial of 5G1.1 in psoriasis patients.

To date, we have not received any revenues from the sale of products. We have incurred operating losses since our inception. As of July 31, 2001 we had an accumulated deficit of $124.3 million. We expect to incur substantial and increasing operating loses for the next several years due to expenses associated with:

- product research and development;
- pre-clinical studies and clinical testing;
- regulatory activities;
- manufacturing development, scale-up and commercial manufacturing; and

. developing a sales and marketing force.

In September 2000, we acquired Prolifaron, Inc. a privately held biopharmaceutical company located in San Diego, California, through the issuance of common stock and fully vested options having an aggregate fair value of approximately $43.9 million at the date of acquisition. Prolifaron was developing therapeutic antibodies addressing multiple diseases, including cancer. The acquisition was in the form of a merger between our new wholly owned subsidiary of Alexion to form Alexion Antibody Technologies, Inc., and Prolifaron. We accounted for the acquisition of Prolifaron using the purchase method of accounting. Through AAT0, we have developed important additional capabilities to discover and develop additional antibody product candidates for the treatment of inflammatory diseases and cancer.

In October 2000, we contributed technology to help form a new company, Arradial Inc., which is aimed at commercializing our novel, desktop silicon-based microarray assay technology. The technology is expected to have applications in genomics and drug discovery. We are a minority shareholder of Arradial, Inc.

We plan to develop and commercialize on our own those product candidates for which the clinical trials and marketing requirements can be funded by our own resources. For those products which require greater resources, our strategy is to form corporate partnerships with major pharmaceutical companies for product development and commercialization.

Results of Operations

Fiscal Years Ended July 31, 2001, 2000, and 1999

We earned contract research revenues of $11.8 million, $21.4 million, and $18.8 million for the fiscal years ended July 31, 2001, 2000, and 1999, respectively. The decrease in revenues in fiscal year 2001 as compared to 2000 was primarily due to lower contract research revenues resulting from the completion of the Phase II pexelizumab CPB study related to our collaborative research and development agreement with Procter & Gamble. The increase in revenues in fiscal year 2000 as compared to 1999 was primarily due to the increased contract revenues from our collaborative research and development agreement with Procter & Gamble.

During fiscal year 2001, we incurred research and development expenses of $38.9 million, excluding non-cash charges related to our acquisition of Prolifaron. These non-cash charges included the in-process research and development charge of $21 million and the amortization of goodwill or purchased intangibles of $2.9 million. For fiscal years 2000 and 1999, we incurred research and development expenses of $40.2 million and $23.7 million, respectively. The decrease in research and development expenses for fiscal 2001 as compared to 2000 was primarily attributable to lower clinical manufacturing and clinical trial costs associated with the completion of the Phase II pexelizumab CPB study. These lower costs were offset by increased costs from clinical trials, manufacturing development, and manufacturing of our other lead C5 Inhibitor product candidate, 5G1.1, and the consolidated on-going research and development costs of AAT which was formed through the September 2000 acquisition of Prolifaron. The increase in research and development costs for fiscal 2000 as compared to 1999 was primarily attributable to the on-going clinical trials of our lead C5 Inhibitor product candidates and the cost of manufacturing development and manufacturing of our C5 Inhibitors for our clinical trials.

Our general and administrative expenses were $7.1 million, $4.2 million and $3.0 million for fiscal years 2001, 2000, and 1999, respectively. The increase in general and administrative expenses in fiscal 2001 as compared to 2000 was principally due to increased personnel and professional fees as well as higher facilities expenses resulting from our relocation and expansion of our operations to support its growth, including our acquisition of Prolifaron. The increase in fiscal 2000 as compared to 1999 was principally due to higher payroll-related costs, as well as higher facilities expenses related to increased rent expense and professional fees related to public relations and patent/legal costs.

Total operating expenses were substantially higher in the twelve months ended July 31, 2001 due principally to the one-time non-cash in-process research and development charge of $21.0 million and the non-cash amortization of goodwill of $2.9 million resulting from the September 2000 acquisition of Prolifaron.

Other income, net, was $10.1 million, $2.7 million, and $1.5 million for fiscal years 2001, 2000, and 1999, respectively. The increase in fiscal year 2001 as compared to 2000 was due to increased interest income from higher cash balances resulting from the $208.5 million of net proceeds received from the sale of common stock in November 2000. The increase in other income, net, for fiscal 2000 as compare to 1999 was due to increased interest income from higher cash balances resulting from the net proceeds obtained from the issuance of $120 million of subordinated convertible notes in March 2000.

During fiscal year 2001, we recorded a $9.1 million non-cash charge that is related to the cumulative change in accounting principle per the adoption of Staff Accounting Bulletin No. 101 or SAB 101. We were required to adopt SAB 101 no later than July 31, 2001. We elected to adopt SAB 101 in the quarter ended April 30, 2001 and recognized the non-cash cumulative effect adjustment of $9.1 million as of August 1, 2000.

As a result of the above factors, we incurred net losses of $57.0 million, $20.2 million, and $6.4 million for fiscal years ended July 31, 2001, 2000, and 1999, respectively. Shown below are our statements of operations for fiscal years ended 2001, 2000, and 1999. Excluding the impact of the non-cash charges resulting from our acquisition of Prolifaron, we incurred a pro forma net loss of $24.0 million for the fiscal year ended July 31, 2001.

(amounts in 000s, except per share data)	Twelve months ended July 31,			
	2001 pro forma -a)	2001	2000	1999
Contract Research Revenues	$ 11,805	$ 11,805	$ 21,441	$18,754
Operating Expenses:				
Research and development	38,871	38,871	40,187	23,710
General and administrative	7,135	7,135	4,175	2,953
In-process research & development (IPRD)	-	21,000	-	-
Amortization of goodwill (GW)	-	2,901	-	-
Total operating expenses	46,006	69,907	44,362	26,663
Operating Loss	(34,201)	(58,102)	(22,921)	(7,909)
Other Income, net	10,177	10,177	2,694	1,514
Loss before cumulative effect of SAB 101	(24,024)	(47,925)	(20,227)	(6,395)
Cumulative effect of adoption of SAB 101	-	(9,118)	-	-
Net Loss	$(24,024)	$(57,043)	$(20,227)	$(6,395)
Net Loss per share	$ (1.38)	$ (3.28)	$ (1.45)	$ (0.57)

(a - excludes non-cash IPRD, Amortization of GW, and Cumulative effect of adoption of SAB 101

Liquidity and Capital Resources

Since our inception in January 1992, we have financed our operations and capital expenditures principally through private placements of our common and preferred stock, an initial public offering of our common stock and subsequent follow-on offerings, convertible subordinated notes, other debt financing, payments under corporate collaborations and grants, and equipment and leasehold improvements financing.

In October 2000, we filed a shelf registration statement to offer up to $300 million of equity securities. On November 1, 2000, we sold 2.3 million shares of our common stock at a price of $90.75 per share resulting in net proceeds to us of approximately $208.5 million, net of fees and other expenses of approximately $201,000 related to the transaction.

In March 2000, we completed a $120 million private placement of our 5.75% Convertible Subordinated Notes due March 15, 2007. The notes bear interest semi-annually on September 15 and March 15 of each year, beginning September 15, 2000. The holders may convert all or a portion of the notes into common stock at any time on or before March 15, 2007 at a conversion price of $106.425 per share. We incurred issuance costs related to this offering of approximately $4.0 million, which are being amortized into interest expense over the seven-year term of the notes. In May 2000, pursuant to a registration rights agreement, we filed a registration statement under the Securities Act of 1933 with the SEC to register resales of the notes and the shares of common stock into which the notes are convertible.

In November 1999, we sold 3.415 million shares of common stock at a price of $14.00 per share in a follow-on public offering, resulting in net proceeds to us of approximately $44.4 million.

In February 1999, we acquired the manufacturing assets, principally land, buildings and laboratory equipment, for the xenotransplantation program developed by US Surgical. We financed the purchase of the manufacturing assets through a $3.9 million term note payable to US Surgical. Interest is 6.0% per annum and is payable quarterly. The principal balance under the note is due in May 2005. Security for this term note is the manufacturing assets that we purchased.

As of July 31, 2001, our cash, cash equivalents, and marketable securities totaled $355.3 million. At July 31, 2001, our cash and cash equivalents consisted of $135.2 million that we hold in short-term highly liquid investments with original maturities of less than three months. The increase in cash, cash equivalents and marketable securities as compared to July 31, 2000 was due to the increase in available cash resulting from our follow-on public offering of our common stock in November 2000. As of July 31, 2001, we have invested $20.0 million in property and equipment to support our research and development efforts. We anticipate our research and development expense will increase significantly for the foreseeable future to support our clinical and manufacturing development of our product candidates.

We lease our headquarters and research and development facility in Cheshire, Connecticut that we relocated to in November 2000. The lease has a term of ten years and six months. At this site, we lease a total of 82,000 square feet of space, which includes approximately 62,000 square feet related to research and laboratories. We have incurred initial leasehold improvements aggregating approximately $4.8 million. In addition, we are paying a pro rata percentage of real estate taxes and operating expenses. Our pilot manufacturing plant, which may be used for producing compounds for some of our current and anticipated clinical trials, is expected to remain in New Haven, Connecticut and encompasses approximately 30,000 square feet of labs and offices. We are currently negotiating a longer-term arrangement for the facilities in New Haven, Connecticut. We believe our new space and our pilot manufacturing facility will be adequate for our current ongoing activities. Alexion Antibody Technologies, Inc., our wholly-owned subsidiary, leases approximately 7,500 square feet of labs and office space in San Diego, California.

Procter & Gamble has agreed to fund clinical development and manufacturing of pexelizumab, initially for use in cardiopulmonary bypass surgery, myocardial infarction and angioplasty. The Procter & Gamble collaboration does not involve any of our other product candidates.

We anticipate that our existing available capital resources together with the anticipated funding from our

collaboration agreement with Procter and Gamble, as well as the addition of our interest and investment income earned on available cash and marketable securities should provide us adequate resources to fund our operating expenses and capital requirements as currently planned for at least the next thirty-six months. Our future capital requirements will depend on many factors, including:

- progress of our research and development programs;
- progress and results of clinical trials;
- time and costs involved in obtaining regulatory approvals;
- costs involved in obtaining and enforcing patents and any necessary licenses;
- dependence on Procter & Gamble for performance of development and commercial matters related to pexelizumab;
- delays in development of or changes in status of commercial relationships;
- delays in developing or arranging satisfactory manufacturing capability;
- costs of manufacturing scale-up and commercial manufacturing;
- our ability to establish marketing and sales capabilities; and
- our ability to establish development and commercialization relationships.

We expect to incur substantial additional costs, for:

- research;
- pre-clinical studies and clinical testing;
- manufacturing process development;
- additional capital expenditures related to personnel, and facilities expansion;
- clinical and commercial manufacturing requirements, and;
- marketing and sales.

If and when we achieve contractual milestones related to product development and product license applications and approvals, additional payments would be required if we elect to continue and maintain our licenses with our licensors, aggregating up to $15.9 million. Furthermore, we will owe royalties to parties we have licensed intellectual property from in connection with the sale of our products. In addition to funds we may receive from our collaboration with Procter & Gamble, we will need to raise or generate substantial additional funding in order to complete the development and commercialization of our product candidates. Our additional financing may include public or private debt or equity offerings, equity line facilities, bank loans and/or collaborative research and development arrangements with corporate partners. There can be no assurance that funds will be available on terms acceptable by us, if at all, or that discussions with potential strategic or collaborative partners will results in any agreements on a timely basis, if at all. The unavailability of additional financing could require us to delay, scale back or eliminate certain research and product development programs or to license third parties to commercialize products or technologies that we would otherwise undertake ourselves, any of which could have a material adverse effect.

For tax reporting purposes, as of July 31, 2001, we had approximately $97.6 million of federal net operating loss carryforwards, which expire through 2021 and $10.0 million of tax credit carryforwards, which expire commencing in fiscal 2008. Provisions of the Tax Reform Act of 1986 may limit our ability to utilize net operating loss and tax credit carryforwards in any given year if certain events occur, including a provision relating to cumulative changes in ownership interests in excess of 50% over a three-year period. We believe that we have triggered these limitation provisions.

Recently issued accounting standards

In July 2001, the Financial Accounting Standards Board issued SFAS No. 141, "Business Combinations", and SFAS No. 142, "Goodwill and Other Intangible Assets", which together significantly change the accounting and disclosures required for these activities and related assets. The primary changes resulting from these standards consist of the cessation of the "pooling of interests" method of accounting and how goodwill and intangible assets will be segregated, amortized (or not amortized), reviewed for impairment (if any), and disclosed within the footnotes to financial statements. Our adoption of SFAS No. 141 will have no impact on the historical financial statements.

We elected to adopt SFAS No. 142 effective August 1, 2001. The adoption of SFAS No. 142 will cause the amortization as it relates to the $23.2 million of goodwill acquired in connection with acquisition of Prolifaron to cease at this date. Prior to the adoption of this standard, this annual amortization was expected to be approximately $3.3 million annually over a seven-year period. On a prospective basis, this goodwill is subject to annual impairment reviews, and, if conditions warrant, interim reviews based upon its estimated fair value. Impairment charges, if any, will be recorded as a component of operating expenses in the period in which the impairment is determined. We have not yet determined whether an impairment charge, if any, would need to be recorded as a result of the adoption of this standard.

In June 1998, the Financial Accounting Standards Board (FASB) issued SFAS No. 133, "Accounting for Derivative Financial Instruments and for Hedging Activities" which provides a comprehensive and consistent standard for the recognition and measurement of derivatives and hedging activities. SFAS No. 133 was effective for fiscal years beginning after June 15, 1999. In June 1999, the FASB issued SFAS No. 137, "Accounting for Derivative Financial Instruments and Hedging Activities - Deferral of the Effective Date of SFAS No. 133 - an Amendment of SFAS No. 133" for the sole purpose of updating the effective date of adoption of SFAS No. 133 to fiscal years beginning after June 15, 2000. The adoption of SFAS No. 133 did not have an impact on our results of operations or financial condition as we do not currently hold derivative financial instruments and do not engage in hedging activities.

Item 7A. QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK.

We account for our marketable securities in accordance with Statement of Financial Accounting Standards No. 115, "Accounting for Certain Investments in Debt and Equity Securities" ("SFAS 115"). All of the cash equivalents and marketable securities are treated as available-for-sale under SFAS 115.

Investments in fixed rate interest earning instruments carry a degree of interest rate risk. Fixed rate securities may have their fair market value adversely impacted due to a rise in interest rates. Due in part to these factors, our future investment income may fall short of expectations due to changes in interest rates or we may suffer losses in principal if forced to sell securities which have seen a decline in market value due to changes in interest rates. Our marketable securities are held for purposes other than trading and we believe that we currently have no material adverse market risk exposure. The marketable securities as of July 31, 2001, had maturities of less than two years. The weighted-average interest rate on marketable securities at July 31, 2001 and 2000 was 4.2% and 6.9%, respectively. The fair value of marketable securities held at July 31, 2001 was $220.1 million.

At July 31, 2001, we had aggregate fixed rate debt of approximately $124 million. If interest rates associated with this debt were increased 10%, a corresponding increase in our annual interest expense of approximately $700,000 would occur.

Item 8. FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA.

The consolidated financial statements and supplementary data of the Company required in this item are set forth at the pages indicated in Item 14(a)(1).

Item 9. CHANGES IN AND DISAGREEMENTS WITH ACCOUNTANTS ON ACCOUNTING AND FINANCIAL DISCLOSURE.

Not applicable.

PART III

Item 10. DIRECTORS, EXECUTIVE OFFICERS AND KEY EMPLOYEES.

Set forth below is certain information regarding our executive officers, directors and key employees:

Name	Age	Position with Alexion
John Fried, Ph.D.(1)(2)(3).....	71	Chairman of the board of directors
Leonard Bell, M.D.(3).....	43	President, Chief Executive Officer, Secretary, Treasurer, Director
David W. Keiser.....	50	Executive Vice President, Chief Operating Officer
Stephen P. Squinto, Ph.D.....	45	Executive Vice President and Head of Research
Katherine S. Bowdish, Ph.D.....	44	Vice president of Antibody Discovery, President of Alexion Antibody Technologies
Samuel S. Chu, Ph.D.....	51	Vice President of Process Sciences and Manufacturing
Thomas I.H. Dubin, J.D.....	39	Vice President and General Counsel
Barry P. Luke.....	43	Vice President of Finance and Administration, Assistant Secretary
Nancy Motola, Ph.D.....	48	Vice President of Regulatory Affairs and Quality Assurance
Christopher F. Mojcik, M.D., Ph.D.....	41	Vice President of Clinical Development
Scott A. Rollins Ph.D.....	38	Vice President of Drug Development Project Management
Russell P. Rother Ph.D.....	40	Vice President of Discovery Research
Daniel N. Caron (4).....	38	Senior Director of Operations and Engineering
William L. Fodor Ph.D. (4).....	43	Senior Director of Xenotransplantation
Jerry T. Jackson (2).....	60	Director
Max Link, Ph.D.(1)(2)(3).....	61	Director
Joseph A. Madri Ph.D.,M.D.....	55	Director
R. Douglas Norby (1).....	66	Director
Alvin S. Parven (2).....	61	Director

(1) Member of our Audit Committee of the Board of Directors.
(2) Member of our Compensation Committee of the Board of Directors.
(3) Member of our Nominating Committee of the Board of Directors.
(4) Key employee.

Each director will hold office until the next annual meeting of stockholders and until his or her successor is elected and qualified or until his or her earlier resignation or removal. Each officer serves at the discretion of the board of directors. Drs. Bell and Squinto and Mr. Keiser are each a party to an employment agreement with us.

John H. Fried, Ph.D. has been the Chairman of our board of directors of Alexion since April 1992. Since 1992, Dr. Fried has been President of Fried & Co., Inc., a health technology venture firm. Dr. Fried was a director of Syntex Corp., a life sciences and health care company, from 1982 to 1994 and he served as Vice Chairman of Syntex from 1985 to January 1993 and President of the Syntex Research Division from 1976 to

1992. Dr. Fried has originated more than 200 U.S. Patents and has authored more than 80 scientific publications. Dr. Fried received his B.S. in Chemistry and Ph.D. in Organic Chemistry from Cornell University.

Biographical details of the following persons are incorporated by reference herein to the section of this Report in Part I, Item 4. SUBMISSION OF MATTERS TO A VOTE OF SECURITY HOLDERS. entitled "EXECUTIVE OFFICERS AND KEY EMPLOYEES OF THE COMPANY": Leonard Bell, M.D, David W. Keiser, Stephen P. Squinto, Ph.D., Katherine S. Bowdish, Ph.D., Samuel S. Chu, Ph.D., Thomas I.H. Dubin, J.D., Barry P. Luke, Christopher F. Mojcik, M.D., Ph.D., Nancy Motola, Ph.D., Scott A. Rollins, Ph.D., Russell P. Rother, Ph.D., Daniel N. Caron, and William Fodor, Ph.D.

Jerry T. Jackson has been a director of Alexion since September 1999. He was employed by Merck & Co. Inc., a major pharmaceutical company, from 1965 until his retirement in 1995. During this time, he had extensive experience in sales, marketing and corporate management, including joint ventures. From 1993 until 1995, Mr. Jackson served as Executive Vice President of Merck with broad responsibilities for numerous operating groups including Merck's International Human Health, Worldwide Human Vaccines, the AgVet Division, Astra/Merck U.S. Operations, as well as worldwide marketing. During 1993, he was also President of the Worldwide Human Health Division. He served as Senior Vice President of Merck from 1991 to 1992 responsible for Merck's Specialty Chemicals and previously, he was President of Merck's Sharp & Dohme International. Mr. Jackson serves as a director of Cor Therapeutics, Inc. and M.D. Edge, Inc. Mr. Jackson received his B.A. from University of New Mexico.

Max Link, Ph.D. has been a director of Alexion since April 1992. From May 1993 to June 1994, Dr. Link was Chief Executive Officer of Corange (Bermuda), the parent company of Boehringer Mannheim Therapeutics, Boehringer Mannheim Diagnostics and DePuy Orthopedics. From 1992 to 1993, Dr. Link was Chairman of the Board of Sandoz Pharma, Ltd., a manufacturer of pharmaceutical products. From 1987 to 1992, Dr. Link was the Chief Executive Officer of Sandoz Pharma and a member of the Executive Board of Sandoz, Ltd., Basel. Prior to 1987, Dr. Link served in various capacities with the United States operations of Sandoz, including as President and Chief Executive Officer. Dr. Link is also a director of Access Pharmaceuticals, Inc., Discovery Labs, Inc., Protein Design Labs, Inc., Human Genome Sciences, Inc., and Cell Therapeutics, Inc., each a publicly held pharmaceutical company, as well as Celsion Corporation and Sulzer Medica, Ltd.

Joseph A. Madri, Ph.D., M.D. is a founder of Alexion and has been a director of Alexion since February 1992. Since 1980, Dr. Madri has been on the faculty of the Yale University School of Medicine and is currently a Professor of Pathology. Dr. Madri serves on the editorial boards of numerous scientific journals and he is the author of over 175 scientific publications. Dr. Madri works in the areas of regulation of angiogenesis, vascular cell-matrix interactions, cell-cell interactions, lymphocyte-endothelial cell interactions and endothelial and smooth muscle cell biology and has been awarded a Merit award from the National Institutes of Health. Dr. Madri received his B.S. and M.S. in Biology from St. John's University and M.D. and Ph.D. in Biological Chemistry from Indiana University.

R. Douglas Norby has been a director of Alexion since September 1999. Since January 2001, Mr. Norby has been Senior Vice President and Chief Financial Officer of Novalux, Inc. From 1996 until December 2000, Mr. Norby he served as Executive Vice President and Chief Financial Officer of LSI Logic Corporation, a semiconductor company, and he has also served on the Board of LSI. From September 1993 until November 1996, he served as Senior Vice President and Chief Financial Officer of Mentor Graphics Corporation, a software company. Mr. Norby served as President of Pharmetrix Corporation, a drug delivery company, from July 1992 to September 1993, and from 1985 to 1992, he was President and Chief Operating Officer of Lucasfilm, Ltd., an entertainment company. From 1979 to 1985, Mr. Norby was Senior Vice President and Chief Financial Officer of Syntex Corporation, a pharmaceutical company. Mr. Norby received a B.A. in Economics from Harvard University and an M.B.A. from Harvard Business School.

Alvin S. Parven has been a director of Alexion since May 1999. Since 1997, Mr. Parven has been President of ASP Associates, a management and strategic consulting firm. From 1994 to 1997, Mr. Parven was

Vice President at Aetna Business Consulting, reporting to the Office of the Chairman of Aetna. From 1987 to 1994, Mr. Parven was Vice President, Operations at Aetna Health Plans. Prior to 1987, he served in various capacities at Aetna including Vice President, Pension Services from 1983 to 1987. Mr. Parven received his B.A. from Northeastern University.

SECTION 16(a) BENEFICIAL OWNERSHIP COMPLIANCE

The information concerning our directors regarding compliance with Section 16(a) of the Securities Exchange Act of 1934 required by this Item will be set forth in our definitive Proxy Statement, to be filed within 120 days after the end of the fiscal year covered by this Annual Report on Form 10-K, and is incorporated by reference to our Proxy Statement.

Item 11. EXECUTIVE COMPENSATION.

The information required by this Item will be set forth in our definitive Proxy Statement, to be filed within 120 days after the end of the fiscal year covered by this Annual Report on Form 10-K, and is incorporated by reference to our Proxy Statement.

Item 12. SECURITY OWNERSCHIP OF CERTAIN BENEFICAL OWNERS AND MANAGEMENT.

The following table sets forth certain information with respect to the beneficial ownership of our common stock as of October 1, 2001, except as otherwise noted in the footnotes: (1) each person known by us to own beneficially more than 5% percent of our outstanding common stock; (2) each director and each named executive officer; and (3) all directors and executives of Alexion as a group.

Name and Address of Beneficial Owner (1)	Number of Shares Beneficially Owned (2)	Percentage of Outstanding Shares of Common Stock
OppenheimerFunds, Inc. Two World Trade Center, 34th Floor New York, NY 10048-0203 (3)	2,000,000	11.0%
Fidelity Management & Research Company 82 Devonshire Street Boston, MA 02109 (4)	1,833,269	10.1%
Scudder Kemper Investments, Inc. 345 Park Avenue New York, NY 10154-0010 (5)	1,373,400	7.6%
OrbiMed Advisors, LLC 41 Madison Avenue, 40/th/ Floor New York, NY 10010 (6)	950,000	5.2%
Leonard Bell, M.D. (7)	772,016	4.1%
Stephen P. Squinto, Ph.D. (8)	225,590	1.2%
David W. Keiser (9)	225,436	1.2%
John H. Fried, Ph.D. (10)	106,670	*
Joseph Madri, Ph.D., M.D. (11)	64,134	*
Max Link, Ph.D. (12)	32,157	*

Name and Address of Beneficial Owner (1)	Number of Shares Beneficially Owned (2)	Percentage of Outstanding Shares of Common Stock
Christopher F. Mojcik, M.D., Ph.D. (13)	30,076	*
Thomas I.H. Dubin, J.D. (14)	11,624	*
Jerry T. Jackson (15)	9,001	*
R. Douglas Norby (16)	9,001	*
Alvin S. Parven (17)	7,901	*
All Directors and Executive Officers as a group (17 persons) (18)	1,751,433	9.0%

_____

* Less than one percent.

(1) Unless otherwise indicated, the address of all persons is 352 Knotter Drive, Cheshire, Connecticut 06410.

(2) To our knowledge, except as set forth below, the persons named in the table have sole voting and investment power with respect to all shares of common stock shown as beneficially owned by them, subject to community property laws where applicable and the information contained in the footnotes in this table.

(3) This figure is based upon information set forth in Schedule 13G dated June 6, 2001.

(4) This figure is based upon information set forth in Schedule 13G dated May 10, 2001.

(5) This figure is based upon information set forth in Schedule 13G dated February 14, 2001.

(6) This figure is based upon information set forth in Schedule 13G dated August 8, 2001.

(7) Includes 611,916 shares of common stock that may be acquired upon the exercise of options within 60 days of October 1, 2001 and 300 shares, in aggregate, held in the names of Dr. Bell's three minor children. Excludes 148,084 shares obtainable through the exercise of options, granted to Dr. Bell, which are not exercisable within 60 days of October 1, 2001 and 90,000 shares held in trust for Dr. Bell's children of which Dr. Bell disclaims beneficial ownership. Dr. Bell disclaims beneficial ownership of the shares held in the name of his minor children.

(8) Includes 174,890 shares of common stock which may be acquired upon the exercise of options within 60 days of October 1, 2001; 5,200 shares held in trust for the benefit of Dr. Squinto's two minor children of which Dr. Squinto's spouse is the trustee; and 10,000 shares held in a charitable remainder trust of which Dr. Squinto and his spouse are the trustees and income beneficiaries. Excludes 61,610 shares obtainable through the exercise of options, granted to Dr. Squinto, which are not exercisable within 60 days of October 1, 2001. Dr. Squinto disclaims beneficial ownership of the shares held in the name of his minor children and the foregoing trusts.

(9) Includes 189,136 shares of common stock which may be acquired upon the exercise of options within 60 days of October 1, 2001 and 300 shares, in aggregate, held in the names of Mr. Keiser's three children. Excludes 74,864 shares obtainable through the exercise of options, granted to Mr. Keiser, which are not exercisable within 60 days of October 1, 2001. Mr. Keiser disclaims beneficial ownership of the shares held in the name of his minor children.

(10) Includes 4,001 shares of common stock, which may be acquired upon the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 16,166 obtainable through the exercise of options granted to Dr. Fried, which are not exercisable within 60 days of October 1, 2001.

(11) Includes 19,134 shares of common stock which may be acquired on the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 16,166 obtainable through the exercise of options granted to Dr. Madri, which are not exercisable within 60 days of October 1, 2001.

(12) Includes 4,001 shares of common stock which may be acquired upon the exercise of options within 60 days of October 1, 2001. Excludes 16,166 shares obtainable through the exercise of options, granted to Dr. Link, which are not exercisable within 60 days of October 1, 2001.

(13) Includes 30,076 shares of common stock, which may be acquired upon the exercise of options within 60 days of October 1, 2001. Excludes 52,924 shares obtainable through the exercise of options, granted to Dr. Mojcik, which are not exercisable within 60 days of October 1, 2001.

(14) Includes 11,624 shares of common stock which may be acquired on the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 59,376 shares obtainable through the exercise of options granted to Mr. Dubin, which are not exercisable within 60 days of October 1, 2001.

(15) Includes 9,001 shares of common stock which may be acquired on the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 17,999 shares obtainable through the exercise of options granted to Mr. Jackson, which are not exercisable within 60 days of October 1, 2001.

(16) Includes 9,001 shares of common stock which may be acquired on the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 17,999 shares obtainable through the exercise of options granted to Mr. Norby, which are not exercisable within 60 days of October 1, 2001.

(17) Includes 7,901 shares of common stock which may be acquired on the exercise of options that are exercisable within 60 days of October 1, 2001. Excludes 17,999 shares obtainable through the exercise of options granted to Mr. Parven, which are not exercisable within 60 days of October 1, 2001.

(18) Consists of shares beneficially owned by Drs. Bell, Fried, Link, Madri, Mojcik, and Squinto and Messrs. Dubin, Jackson, Keiser, Norby and Parven, and certain other officers. Includes 1,264,712 shares of common stock, which may be acquired upon the exercise of options within 60 days of October 1, 2001.

Item 13. CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS.

In June and October 1992, we entered into patent licensing agreements with Oklahoma Medical Research Foundation and Yale University. The agreements provide that we will pay to these institutions royalties based on sales of products incorporating technology licensed thereunder and also license initiation fees, including annual minimum royalties that increase in amount based on the status of product development and the passage of time. Under policies of OMRF and Yale, the individual inventors of patents are entitled to receive a percentage of the royalties and other license fees received by the licensing institution. Some of our founders and scientific advisors are inventors under patent and patent applications, including Dr. Bell, one of our directors and our President and Chief Executive Officer, Dr. Madri, one of our directors, Dr. Squinto, Executive Vice President and Head of Research, and Dr. Rollins, Vice President of Drug Development and Project Management with respect to patent applications licensed from Yale and therefore, entitled to receive a portion of royalties and other fees payable by us.

PART IV

Item 14. EXHIBITS, FINANCIAL STATEMENT SCHEDULES, AND REPORTS ON FORM 8-K.

(a) (1) Financial Statements

The financial statements required by this item are submitted in a separate section beginning on page F-1 of this report.

(2) Financial Statement Schedules

Schedules have been omitted because of the absence of conditions under which they are required or because the required information is included in the financial statements or notes thereto.

(3) Exhibits:

2.1 Agreement and Plan of Merger by and among Alexion Pharmaceuticals, Inc., PI Acquisition Company, Inc., and Prolifaron, Inc., dated September 22, 2000.*/(1)/

3.1 Certificate of Incorporation, as amended.*/(2)/

3.2 Bylaws.*/(2)/

4.1 Specimen Common Stock Certificate.*/(2)/

10.1 Employment Agreement, dated April 1, 2000, between the Company and Dr. Leonard Bell.*/(3)/

10.2 Employment Agreement, dated October 2, 2000, between the Company and David W. Keiser.*/(4)/

10.3 Employment Agreement, dated October 22, 1997, between the Company and Dr. Stephen P. Squinto.*/(5)/

10.4 Employment Agreement, dated September 21, 2000, between the Company and Dr. Katherine S. Bowdish.*/(1)/

10.5 Administrative, Research and Development Facility Lease, dated May 9, 2000, between the Company and WE Knotter L.L.C.*/(6)/

10.6 Option Agreement, dated April 1, 1992 between the Company and Dr. Leonard Bell.*/(2)/

10.7 Company's 1992 Stock Option Plan, as amended.*/(7)/

10.8 Company's 2000 Stock Option Plan.*/(8)/

10.9 Company's 1992 Outside Directors Stock Option Plan, as amended.*/(9)/

10.10 Exclusive License Agreement dated as of June 19, 1992 among the Company, Yale University and Oklahoma Medical Research Foundation.*/(3)/

10.11 License Agreement dated as of September 30, 1992 between the Company and Yale University, as

amended July 2, 1993.*/(2)/+

10.12 License Agreement dated as of August 1, 1993 between the Company and Biotechnology Research and Development Corporation ("BRDC"), as amended as of July 1, 1995.*/(2)/+

10.13 License Agreement dated January 25, 1994 between the Company and The Austin Research Institute.*/(2)/+

10.14 Exclusive Patent License Agreement dated April 21, 1994 between the Company and the National Institutes of Health.*/(2)/+

10.15 License Agreement dated July 22, 1994 between the Company and The Austin Research Institute.*/(2)/+

10.16 License Agreement dated as of January 10, 1995 between the Company and Yale University.*/(2)/+

10.17 License Agreement dated as of May 27, 1992 between the Company and Yale University, as amended September 23, 1992.*/(2)/+

10.18 Agreement to be Bound by Master Agreement dated as of August 1, 1993 between the Company and BRDC.*/(2)/

10.19 License Agreement dated March 27, 1996 between the Company and Medical Research Council.*/(10)/+

10.20 License Agreement dated May 8, 1996 between the Company and Enzon, Inc.*/(10)/+

10.21 Asset Purchase Agreement date as of February 9, 1999 between the Company and United States Surgical Corporation.*/(11)/

10.22 Collaboration Agreement dated January 25, 1999 between the Company and the Procter & Gamble Company, as amended.*/(11)/+

10.23 Letter agreement dated September 14, 1999 between the Company and Leonard Bell.*/(11)/

21.1 Subsidiaries of Alexion Pharmaceuticals, Inc.

23.1 Consent of Arthur Andersen LLP

99 Risk Factors

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* Previously filed

(1) Incorporated by reference to our report on Form 8-K, filed on October 3, 2000.

(2) Incorporated by reference to our Registration Statement on Form S-1 (Reg. No. 333-00202).

(3) Incorporated by reference to our Quarterly Report on Form 10-Q for the quarter ended April 30, 2000.

(4) Incorporated by reference to our Annual Report on Form 10-K for the fiscal year ended July 31, 2000.

(5) Incorporated by reference to our Annual Report on Form 10-K for the fiscal year ended July 31, 1997.

(6) Incorporated by reference to our Registration Statement on Form S-3 (Reg. No. 333-36738) filed on May 10, 2000.

(7) Incorporated by reference to our Registration Statement on Form S-8 (Reg. No. 333-71879) filed on February 5, 1999.

(8) Incorporated by reference to our Registration Statement on Form S-8 (Reg. No. 333-69478) filed on September 14, 2001.

(9) Incorporated by reference to our Registration Statement on Form S-8 (Reg. No. 333-71985) filed on February 8, 1999.

(10) Incorporated by reference to our Annual Report on Form 10-K for the fiscal year ended July 31, 1996.

(11) Incorporated by reference to our Annual Report on Form 10-K for the fiscal year ended July 31, 1999.

+ Confidential treatment was granted for portions of such document.

(b) Reports on Form 8-K

Current Report on Form 8-K filed on June 7, 2001 on the issuance of two press releases announcing the commencement of a Phase II lupus nephritis clinical trial and the completion of a Phase I Psoriasis pilot safety study.

(c) Exhibits

See (a) (3) above.

(d) Financial Statement Schedules

See (a) (2) above.

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.

Alexion Pharmaceuticals, Inc.

By:
/s/ LEONARD BELL

Leonard Bell, M.D.
President, Chief Executive Officer,
Secretary and Treasurer

By:
/s/ DAVID W. KEISER

David W. Keiser
Executive Vice President and Chief
Operating Officer

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

/s/ LEONARD BELL --- Leonard Bell, M.D.	President, Chief Executive Officer, Secretary, Treasurer and Director (principal executive officer)	October 26, 2001
/s/ DAVID W. KEISER --- David W. Keiser	Executive Vice President and Chief Operating Officer (principal financial officer)	October 26, 2001
/s/ BARRY P. LUKE --- Barry P. Luke	Vice President of Finance and Administration (principal accounting officer)	October 26, 2001
/s/ JOHN H. FRIED --- John H. Fried, Ph.D.	Chairman of the Board of Directors	October 26, 2001
/s/ JERRY T. JACKSON --- Jerry T. Jackson	Director	October 26, 2001
/s/ MAX LINK --- Max Link, Ph.D.	Director	October 26, 2001
/s/ JOSEPH A. MADRI --- Joseph A. Madri, Ph.D., M.D.	Director	October 26, 2001
/s/ R. DOUGLAS NORBY --- R. Douglas Norby	Director	October 26, 2001
/s/ ALVIN S. PARVEN --- Alvin S. Parven	Director	October 26, 2001

ALEXION PHARMACEUTICALS, INC.

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Report of Independent Public Accountants

To the Board of Directors and Stockholders of
Alexion Pharmaceuticals, Inc.:

We have audited the accompanying consolidated balance sheets of Alexion Pharmaceuticals, Inc. (a Delaware corporation) and subsidiaries (collectively, the Company) as of July 31, 2001 and 2000, and the related consolidated statements of operations, stockholders' equity and comprehensive loss, and cash flows for each of the three years in the period ended July 31, 2001. These consolidated financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these consolidated financial statements based on our audits.

We conducted our audits in accordance with auditing standards generally accepted in the United States. 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 accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, the consolidated financial statements referred to above present fairly, in all material respects, the consolidated financial position of Alexion Pharmaceuticals, Inc. and subsidiaries as of July 31, 2001 and 2000, and the consolidated results of their operations and their cash flows for each of the three years in the period ended July 31, 2001, in conformity with accounting principles generally accepted in the United States.

As further discussed in Note 2 to the consolidated financial statements, during the year ended July 31, 2001, the Company changed its method of revenue recognition relating to non-refundable upfront licensing fees in accordance with Staff Accounting Bulletin No. 101.

/s/ Arthur Andersen LLP

Hartford, Connecticut
August 31, 2001

ALEXION PHARMACEUTICALS, INC.

Consolidated Balance Sheets
(amounts in thousands)

ASSETS	July 31, 2001	July 31, 2000
CURRENT ASSETS:		
Cash and cash equivalents	$ 135,188	$ 91,858
Marketable securities	220,086	82,671
Reimbursable contract costs:		
Billed	2,974	3,660
Unbilled	4,006	1,435
Prepaid expenses	493	456
Total current assets	362,747	180,080
PROPERTY, PLANT, AND EQUIPMENT, net	13,731	8,213
GOODWILL, net of accumulated amortization of $2,901 at July 31, 2001	20,270	-
DEFERRED FINANCING COSTS, net of accumulated amortization of $751 and $185 at July 31, 2001 and 2000, respectively	3,265	3,752
OTHER ASSETS	246	657
Total assets	$ 400,259	$ 192,702
LIABILITIES AND STOCKHOLDERS' EQUITY		
CURRENT LIABILITIES:		
Current portion of notes payable	$ -	$ 369
Accounts payable	1,722	2,100
Accrued expenses	2,271	1,229
Accrued interest	2,646	2,730
Deferred revenue	1,351	750
Total current liabilities	7,990	7,178
DEFERRED REVENUE, less current portion included above	7,941	-
NOTES PAYABLE, less current portion included above	3,920	3,920
CONVERTIBLE SUBORDINATED NOTES	120,000	120,000
COMMITMENTS AND CONTINGENCIES (Notes 9, 11, and 14)		
STOCKHOLDERS' EQUITY:		
Preferred stock, $.0001 par value; 5,000 shares authorized; no shares issued or outstanding	-	-
Common stock, $.0001 par value; 145,000 shares authorized; 18,119 and 15,146 shares issued at July 31, 2001 and 2000, respectively	2	2
Additional paid-in capital	384,091	128,836
Accumulated deficit	(124,257)	(67,214)
Other comprehensive income (loss)	572	(20)
Treasury stock, at cost, 12 shares	-	-
Total stockholders' equity	260,408	61,604
Total liabilities and stockholders' equity	$ 400,259	$ 192,702

The accompanying notes are an integral part of these consolidated financial statements.

ALEXION PHARMACEUTICALS, INC.

Consolidated Statements of Operations
(amounts in thousands, except per share amounts)

	For the Years Ended July 31,		
	2001	2000	1999
CONTRACT RESEARCH REVENUES	$ 11,805	$ 21,441	$ 18,754
OPERATING EXPENSES:			
Research and development	38,871	40,187	23,710
General and administrative	7,135	4,175	2,953
In-process research and development (Note 4)	21,000	-	-
Amortization of goodwill (Note 4)	2,901	-	-
Total operating expenses	69,907	44,362	26,663
Operating loss	(58,102)	(22,921)	(7,909)
OTHER INCOME AND EXPENSE:			
Interest income	17,975	5,833	1,702
Interest expense	(7,798)	(3,139)	(188)
Loss before cumulative effect of Staff Accounting Bulletin No. 101 (SAB 101)	(47,925)	(20,227)	(6,395)
CUMULATIVE EFFECT OF ADOPTION OF SAB 101 (Note 2)	(9,118)	-	-
Net loss	$ (57,043)	$ (20,227)	$ (6,395)
BASIC AND DILUTED PER SHARE DATA:			
Loss before cumulative effect of adoption of SAB 101	$ (2.76)	$ (1.45)	$ (0.57)
Cumulative effect of adoption of SAB 101	(.52)	-	-
Net loss	$ (3.28)	$ (1.45)	$ (0.57)
PRO FORMA AMOUNTS ASSUMING ADOPTION OF SAB 101 APPLIED RETROACTIVELY:			
Pro forma operating loss	$ (58,102)	$ (22,333)	$ (17,615)
Pro forma net loss	$ (47,925)	$ (19,639)	$ (16,101)
Pro forma basic and diluted net loss per common share	$ (2.76)	$ (1.41)	$ (1.43)
SHARES USED IN COMPUTING BASIC AND DILUTED NET LOSS PER COMMON SHARE AND PRO FORMA NET LOSS PER COMMON SHARE	17,371	13,914	11,265

The accompanying notes are an integral part of these consolidated financial statements.

ALEXION PHARMACEUTICALS, INC.

Consolidated Statements of Stockholders' Equity and Comprehensive Loss
(amounts in thousands)

	Common Stock Shares	Common Stock Amount	Additional Paid-In Capital	Accumulated Deficit
BALANCE, July 31, 1998	11,237	$ 1	$ 79,776	$ (40,592)
Issuance of common stock from exercise of stock options	67	-	383	-
Non-cash compensation expense related to grant of stock options	-	-	132	-
Net change in unrealized losses on marketable securities	-	-	-	-
Net loss	-	-	-	(6,395)
Comprehensive loss				
BALANCE, July 31, 1999	11,304	1	80,291	(46,987)
Issuance of common stock from exercise of options	225	-	1,697	-
Non-cash compensation expense related to grant of stock options	-	-	430	-
Issuance of common stock from exercise of warrants	202	-	1,998	-
Issuance of common stock, net of issuance costs of $3,391	3,415	1	44,420	-
Net change in unrealized losses on marketable securities	-	-	-	-
Net loss	-	-	-	(20,227)
Comprehensive loss				
BALANCE, July 31, 2000	15,146	$ 2	$ 128,836	$ (67,214)

	Other Comprehensive Income (Loss)	Treasury Stock, at cost Shares	Treasury Stock, at cost Amount	Total Stockholders' Equity	Total Comprehensive Loss
BALANCE, July 31, 1998	$ 5	12	$ -	$ 39,190	
Issuance of common stock from exercise of Stock options	-	-	-	383	
Non-cash compensation expense related to grant of stock options	-	-	-	132	
Net change in unrealized losses on marketable securities	(9)	-	-	(9)	$ (9)
Net loss	-	-	-	(6,395)	(6,395)
Comprehensive loss					$ (6,404)
BALANCE, July 31, 1999	(4)	12	-	33,301	
Issuance of common stock from exercise of options	-	-	-	1,697	
Non-cash compensation expense related to grant of stock options	-	-	-	430	
Issuance of common stock from exercise of warrants	-	-	-	1,998	
Issuance of common stock, net of issuance costs of $3,391	-	-	-	44,421	
Net change in unrealized losses on marketable securities	(16)	-	-	(16)	$ (16)
Net loss	-	-	-	(20,227)	(20,227)
Comprehensive loss					$ (20,243)

BALANCE, July 31, 2000	$	(20)	12	$ -	$ 61,604

The accompanying notes are an integral part of these consolidated financial statements.

ALEXION PHARMACEUTICALS, INC.

Consolidated Statements of Stockholders' Equity and Comprehensive Loss (Continued)
(amounts in thousands)

	Common Stock Shares	Common Stock Amount	Additional Paid-In Capital	Accumulated Deficit
BALANCE, July 31, 2000	15,146	$ 2	$ 128,836	$ (67,214)
Issuance of common stock from exercise of options	299	-	2,199	-
Non-cash compensation expense related to grant of stock options	-	-	408	-
Issuance of common stock from exercise of warrants	18	-	179	-
Issuance of common stock, net of issuance costs of $201	2,300	-	208,524	-
Issuance of common stock and stock options to acquire all outstanding equity of Prolifaron	356	-	43,945	-
Net change in unrealized gains (losses) on marketable securities	-	-	-	-
Net loss	-	-	-	(57,043)
Comprehensive loss				
BALANCE, July 31, 2001	18,119	$ 2	$ 384,091	$ (124,257)

	Other Comprehensive Income (Loss)	Treasury Stock, at cost Shares	Treasury Stock, at cost Amount	Total Stockholders' Equity	Total Comprehensive Loss
BALANCE, July 31, 2000	$ (20)	12	$ -	$ 61,604	
Issuance of common stock from exercise of options	-	-	-	2,199	
Non-cash compensation expense related to grant of stock options	-	-	-	408	
Issuance of common stock from exercise of warrants	-	-	-	179	
Issuance of common stock, net of issuance costs of $201	-	-	-	208,524	
Issuance of common stock and stock options to acquire all outstanding equity of Prolifaron	-	-	-	43,945	
Net change in unrealized gains (losses) on marketable securities	592	-	-	592	$ 592
Net loss	-	-	-	(57,043)	(57,043)
Comprehensive loss					$ (56,451)
BALANCE, July 31, 2001	$ 572	12	$ -	$ 260,408	

The accompanying notes are an integral part of these consolidated financial statements.

ALEXION PHARMACEUTICALS, INC.

Consolidated Statements of Cash Flows
(amounts in thousands)

	For the Years Ended July 31,		
	2001	2000	1999
CASH FLOWS FROM OPERATING ACTIVITIES:			
Net loss	$ (57,043)	$ (20,227)	$ (6,395)
Adjustments to reconcile net loss to net cash used in operating activities:			
In-process research and development	21,000	-	-
Cumulative effect of adopting SAB 101	9,118	-	-
Amortization of goodwill	2,901	-	-
Depreciation and amortization	2,620	1,769	889
Compensation expense related to grant of stock options	408	430	132
Changes in assets and liabilities -			
Reimbursable contract costs	(1,842)	1,767	(6,725)
Prepaid expenses	270	16	(263)
Accounts payable	(789)	(1,444)	2,734
Accrued expenses	845	(1,026)	1,459
Accrued interest	(84)	2,657	51
Deferred revenue	(576)	300	383
Net cash used in operating activities	(23,172)	(15,758)	(7,735)
CASH FLOWS FROM INVESTING ACTIVITIES:			
Purchases of marketable securities	(561,940)	(93,065)	(88,987)
Proceeds from marketable securities	425,117	14,468	90,882
Purchases of property, plant, and equipment	(7,021)	(2,229)	(1,912)
Investment in patents and licensed technology	(65)	(40)	-
Cash paid for transaction costs, net of cash received for acquisition of Prolifaron	(464)	-	-
Net cash used in investing activities	(144,373)	(80,866)	(17)
CASH FLOWS FROM FINANCING ACTIVITIES:			
Net proceeds from issuance of common stock	210,902	48,116	383
Net proceeds from issuance of convertible subordinated notes	-	116,063	-
Repayments of notes payable	(369)	(462)	(369)
Other	342	527	467
Net cash provided by financing activities	210,875	164,244	481
NET INCREASE (DECREASE) IN CASH AND CASH EQUIVALENTS	43,330	67,620	(7,271)
CASH AND CASH EQUIVALENTS, beginning of year	91,858	24,238	31,509
CASH AND CASH EQUIVALENTS, end of year	$ 135,188	$ 91,858	$ 24,238
SUPPLEMENTAL DISCLOSURE OF CASH FLOW INFORMATION:			
Cash paid for interest expense	$ 7,316	$ 296	$ 188
SUPPLEMENTAL DISCLOSURE OF NON-CASH FINANCING ACTIVITIES:			
Fixed assets acquired pursuant to seller financing	$ -	$ -	$ 3,920
Acquisition of Prolifaron through issuance of common stock and stock options	$ 43,945	$ -	$ -

The accompanying notes are an integral part of these consolidated financial statements.

(1) ORGANIZATION AND OPERATIONS

Alexion Pharmaceuticals, Inc. ("Alexion" or the "Company") was organized in 1992 and is engaged in the development of therapeutic products for the treatment of a wide array of severe diseases, including cardiovascular and autoimmune disorders and cancer. The Company's two lead product candidates are antibodies that address specific diseases that arise when the human immune system attacks the human body itself and produces undesired inflammation. Antibodies are proteins that bind specifically to selected targets in the body. After the antibody binds to its target, it may activate the body's immune system against the target, block activities of the target or stimulate activities of the target. For one of its lead antibody product candidates, pexelizumab, a large Phase IIb clinical study in cardiopulmonary bypass ("CPB") patients was completed and two additional large Phase II studies in myocardial infarction (heart attack) patients are in progress. For the Company's other lead antibody product candidate, 5G1.1, a large Phase II clinical study in rheumatoid arthritis patients was completed and clinical programs are on-going in four additional diseases, including a Phase II study in membranous nephritis patients, as well as open label extension trials in rheumatoid arthritis and membranous nephritis patients. The Company is also developing Apogen immunotherapeutic products to target T-cell related disorders and is developing therapies employing the transplantation of cells from other species into humans, known as xenotransplantation.

In September 2000, the Company acquired Prolifaron, Inc. ("Prolifaron"), a privately held biopharmaceutical company with extensive combinatorial human antibody library technologies and expertise (the Prolifaron Acquisition) (see Note 4).

In October 2000, the Company filed a shelf registration statement to offer up to $300 million of equity securities (see Note 12).

The Company has incurred consolidated losses since inception and has made no product sales to date. The Company will continue to seek financing to obtain regulatory approvals for its product candidates, fund operations losses, and if deemed appropriate, establish manufacturing, sales, marketing and distribution capabilities. The Company expects to incur substantial expenditures in the foreseeable future for the research and development and commercialization of its products. The Company will seek to raise necessary funds through public or private equity or debt financings, bank loans, collaborative or other arrangements with corporate sources, or through other sources of financing.

(2) CUMULATIVE EFFECT OF ACCOUNTING CHANGE

In December 1999, the Securities and Exchange Commission staff issued Staff Accounting Bulletin No. 101, "Revenue Recognition in Financial Statements" (SAB 101). SAB 101 summarizes certain of the staff's views in applying generally accepted accounting principles to revenue recognition in financial statements and specifically addresses revenue recognition in the biotechnology industry for non-refundable up-front fees. Prior to the implementation of SAB 101, non-refundable license fees received upon execution of license agreements were recognized as revenue immediately. The Company adopted SAB 101 in fiscal 2001 and has therefore changed its revenue recognition policy for up-front non-refundable payments from immediate recognition to deferral of the revenue with the up-front fee amortized into revenue over the life of the agreement.

In 1999, the Company recognized $10 million of revenue from a non-refundable up-front licensing fee received from Proctor & Gamble (see Note 10). With the adoption of SAB 101, the Company is now required to recognize this $10 million license fee as revenue over the average of the remaining patent lives of the underlying technologies (17 years) as the agreement with Proctor & Gamble provides for ongoing collaborative services and the funding of specified clinical development and manufacturing costs of the Company's pexelizumab product candidate. The license is being recognized over the lives of the patents, as the agreement does not have a specified contractual term. As part of the change to the accounting method, the Company has recognized a non-cash cumulative effect adjustment of $9.1 million as of August 1, 2000. There are no income tax effects related to this accounting change.

The Company has provided pro forma operating loss, net loss and net loss per share information as if the Company had adopted SAB 101 for all periods presented.

(3) SUMMARY OF SIGNIFICANT ACCOUNTING POLICIES

Principles of consolidation

The accompanying consolidated financial statements include Alexion Pharmaceuticals, Inc. and its wholly-owned subsidiaries, Alexion Antibody Technologies ("AAT") and Columbus Farming Corporation ("Columbus"). Results of operations of AAT are included in the Company's consolidated statements of operations since September 23, 2000, the effective date of the Prolifaron acquisition (see Note 4). Columbus was formed on February 9, 1999 to acquire certain manufacturing assets from United States Surgical Corporation ("US Surgical"). All significant inter-company balances and transactions have been eliminated in consolidation.

Cash and cash equivalents

Cash and cash equivalents are stated at cost, which approximates market, and includes short-term highly liquid investments with original maturities of less than 90 days.

Marketable securities

The Company invests in marketable debt securities of highly rated financial institutions and investment-grade debt instruments and limits the amount of credit exposure with any one entity.

The Company has classified its marketable securities as "available for sale" and, accordingly, carries such securities at aggregate fair value. Unrealized gains or losses are included in other comprehensive income (loss) as a component of stockholders' equity. No significant realized gains or losses were recorded during the years ended July 31, 2001, 2000 and 1999. At July 31, 2001, the Company's marketable securities had a maximum maturity of less than two years with an average of approximately twelve months. The weighted average interest rate associated with these marketable debt securities was 4.2% and 6.9% as of July 31, 2001 and 2000, respectively.

The following is a summary of marketable securities at July 31, 2001 and 2000 (amounts in thousands):

	Amortized Cost	Unrealized Gains (Losses)	Fair Value
Federal agency obligations	$ 123,022	$ 387	$ 123,409
Corporate bonds	96,492	185	96,677
Total marketable securities at July 31, 2001	$ 219,514	$ 572	$ 220,086
Federal agency obligations	$ 43,435	$ (14)	$ 43,421
Corporate bonds	39,256	(6)	39,250
Total marketable securities at July 31, 2000	$ 82,691	$ (20)	$ 82,671

Long-lived assets

The Company accounts for its investments in long-lived assets in accordance with Statement of Financial Accounting Standard (SFAS) No. 121, "Accounting for the Impairment of Long-Lived Assets and Long-Lived Assets to be Disposed of". SFAS No. 121 requires a company to review long-lived assets for impairment whenever events or changes in circumstances indicate that the carrying amount of an asset may not be recoverable. The Company has reviewed its long-lived assets and determined that no impairments exist.

Revenue recognition

Contract research revenues recorded by the Company consist of research and development support payments and license fees under collaborations with third parties and amounts received under various government grants.

As a result of the Company's adoption of SAB 101 (see Note 2), up-front, non-refundable license fees received in connection with a collaboration are deferred and amortized into revenue over the life of the underlying technologies.

Revenues derived from the achievement of milestones are recognized when the milestone is achieved, provided that the milestone is substantive and a culmination of the earnings process has occurred. Research and development support revenues are recognized as the related work is performed and expenses are incurred under the terms of the contracts for development activities.

Unbilled reimbursable contract costs as shown on the accompanying consolidated balance sheets represent reimbursable costs incurred in connection with research contracts which have not yet been billed. The Company bills these costs and recognizes the costs and related revenues in accordance with the terms of the contracts.

Deferred revenue results from cash received or amounts receivable in advance of revenue recognition under research and development contracts (see Notes 2 and 10).

Research and development expenses

Research and development expenses are expensed when incurred unless recoverable under contract. Research and development expenses include the following major types of costs: salaries and benefit costs, research license fees and various contractor costs, depreciation and amortization of lab facilities and leasehold improvements, building and utilities costs related to research space and lab supplies.

Use of estimates in the preparation of financial statements

The preparation of financial statements in conformity with accounting principles generally accepted in the United States requires management to make estimates and assumptions that affect the reported amounts of assets and liabilities and disclosure of contingent assets and liabilities at the date of the financial statements and the reported amounts of revenues and expenses during the reporting period. Actual results could differ from those estimates.

Comprehensive income (loss)

The Company reports and presents comprehensive income (loss) in accordance with SFAS No. 130 "Reporting Comprehensive Income," which establishes standards for reporting and display of comprehensive income (loss) and its components in a full set of general purpose financial statements. The objective of the statement is to report a measure of all changes in equity of an enterprise that result from transactions and other economic events of the period other than transactions with owners (comprehensive income (loss)). The Company's other comprehensive income (loss) arises from net unrealized gains (losses) on marketable securities. The Company has elected to display comprehensive income (loss) as a component of the statements of stockholders' equity and comprehensive loss.

Stock options

The Company accounts for stock options granted to employees in accordance with Accounting Principles Board Opinion No. 25. The Company accounts for stock options granted to consultants in accordance with Emerging Issues Task Force ("EITF") 96-18, "Accounting for Equity Instruments that are Issued to Other Than Employees for Acquiring or in Conjunction with Selling, Goods or Services". The Company recognizes compensation cost associated with stock option grants, if any, on a pro-rata basis over the applicable vesting term.

Net loss per common share

The Company computes and presents net loss per common share in accordance with SFAS No. 128, "Earnings Per Share". Basic net loss per common share is based on the weighted average shares of common stock outstanding during the period. Diluted net loss per common share assumes in addition to the above, the dilutive effect of common shares equivalents outstanding during the period. Common share equivalents represent dilutive stock options, warrants, and convertible subordinated debt. These outstanding stock options, warrants and convertible subordinated debt entitled holders to acquire 4,689,075, 3,829,887 and 2,568,587 shares of common stock at July 31, 2001, 2000 and 1999, respectively. There is no difference in basic and diluted net loss per common share as the effect of common share equivalents is anti-dilutive for all periods presented.

The pro forma net loss per share as reported in the accompanying statements of operations for the years ended July 31, 2001, 2000 and 1999, assumes the retroactive adoption of SAB 101 (see Notes 2 and 17).

Segment reporting

SFAS No. 131, "Disclosure about Segments of an Enterprise and Related Information", establishes annual and interim reporting standards for an enterprise's operating segments and related disclosures about its products, services, geographic areas and major customers. The Company has determined that it operates in only one segment. In addition, all revenues are generated from U.S. entities, and all long-lived assets are maintained in the U.S.

Fair value of financial instruments

Financial instruments, include cash and cash equivalents, marketable securities, notes payable and convertible subordinated notes. Cash and cash equivalents and marketable securities are carried at fair value. Notes payable and convertible subordinated notes are carried at cost. Management believes notes payable approximates fair value based upon recent borrowing rates. The carrying value of convertible subordinated notes exceeded fair value by approximately $46.3 million based upon trading values reported at July 31, 2001.

Recently issued accounting standards

In July 2001, the Financial Accounting Standards Board issued SFAS No. 141, "Business Combinations", and SFAS No. 142, "Goodwill and Other Intangible Assets", which together significantly change the accounting and disclosures required for these activities and related assets. The primary changes resulting from these standards consist of the cessation of the "pooling of interests" method of accounting and how goodwill and intangible assets will be segregated, amortized (or not amortized), reviewed for impairment (if any), and disclosed within the footnotes to financial statements. The Company's adoption of SFAS No. 141 will have no impact on the historical financial statements.

The Company is electing to adopt SFAS No. 142 effective August 1, 2001. The adoption of SFAS No. 142 will cause the amortization as it relates to the $23.2 million of goodwill acquired in connection with acquisition of Prolifaron (see Note 4) to cease at this date. Prior to the adoption of this standard, this annual amortization was expected to be approximately $3.3 million annually over a seven year period. On a prospective basis, this goodwill is subject to annual impairment reviews, and, if conditions warrant, interim reviews based upon its estimated fair value. Impairment charges, if any, will be recorded as a component of operating expenses in the period in which the impairment is determined. The Company has not yet determined whether an impairment charge, if any, would need to be recorded as a result of the adoption of this standard.

In June 1998, the Financial Accounting Standards Board (FASB) issued SFAS No. 133, "Accounting for Derivative Financial Instruments and for Hedging Activities" which provides a comprehensive and consistent standard for the recognition and measurement of derivatives and hedging activities. SFAS No. 133 was effective for fiscal years beginning after June 15, 1999. In June 1999, the FASB issued SFAS No. 137, "Accounting for Derivative Financial Instruments and Hedging Activities - Deferral of the Effective Date of SFAS No. 133 - an Amendment of SFAS No. 133" for the sole purpose of updating the effective date of adoption of SFAS No. 133 to fiscal years beginning after June 15, 2000. The adoption of SFAS No. 133 did not have an impact on the Company's results of operations or financial condition as the Company does not currently hold derivative financial instruments and does not engage in hedging activities.

(4) PROLIFARON ACQUISITION

On September 23, 2000, the Company acquired Prolifaron, Inc. ("Prolifaron"), a privately-held biopharmaceutical company with extensive combinatorial human antibody library technologies and expertise. The acquisition was accomplished when Prolifaron was merged with a wholly owned subsidiary of Alexion and renamed Alexion Antibody Technologies, Inc. In consideration thereof, the Company issued 355,594 shares of the Company's common stock and fully vested options to purchase 44,364 shares of the Company's common stock at a weighted average exercise price of $44.35 per share, in exchange for all of the outstanding equity of Prolifaron including fully vested options under their stock option plan. The fair value of the Company's common stock and stock options issued at the date of the acquisition was approximately $43.9 million.

The Prolifaron acquisition has been accounted for as a purchase and, accordingly, the purchase price has been allocated to the assets acquired and liabilities assumed based on their estimated fair values at the date of the acquisition. The Company allocated $21.0 million of the purchase price to in-process research and development projects. This allocation represented the estimated fair value based on risk-adjusted cash flows related to the incomplete research and development projects. At the date of the acquisition, development of these projects had not yet reached technological feasibility and the research and development in progress has no alternative future uses. Accordingly, these costs were expensed as of the acquisition date. At the merger date, Prolifaron was conducting pre-clinical development and testing activities with a goal to develop technologies for antibody discovery and engineering and identify new fully human therapeutic antibodies addressing multiple disease areas. The drug candidates under development represent innovative technologies addressing autoimmune and inflammatory disorders, and cancer.

As of the acquisition date, Prolifaron had incurred approximately $5.7 million of expenses on development projects since its inception in 1998, and expected to spend approximately $8.5 million over the next seven years to complete animal testing of the developmental drug candidates. Management anticipates the in-process projects would, if successful, be marketed in the U.S. in five to nine years.

In making its purchase price allocation, management considered present value calculations of income, an analysis of project accomplishments and remaining outstanding items, an assessment of overall contributions, as well as technological and regulatory risks. The value assigned to purchased in-process technology was determined by estimating the costs to develop the acquired technology into commercially viable products, estimating the resulting net cash flows from the projects, and discounting the net cash flows to their present value. The revenue projection used to value the in-process research and development was based on estimates of relevant market sizes and growth factors, expected trends in technology, and nature and expected timing of new product introductions by Prolifaron and its competitors.

The rates utilized to discount the net cash flows to their present value were based on estimated cost of capital calculations. Due to the risks associated with the projected cash flow forecast, a discount rate of 40 percent was considered appropriate for the in-process research and development. The selected rate reflects the inherent uncertainties surrounding the successful development of the purchased in-process technology, the useful life of such technology, and the uncertainty of technological advances that are unknown at this time.

If these projects are not successfully developed, the sales and profitability of the combined companies may by adversely affected in future periods. Additionally, the value of other acquired intangible assets may become impaired. The research and development projects acquired in connection with the acquisition of Prolifaron are expected to continue in line with the estimates described above.

The excess cost over the fair value of the net assets acquired, which amounted to approximately $23.2 million, is reflected as goodwill and is being amortized over approximately 7 years (see Recently Issued Accounting Standards, Note 3). The following table summarizes the allocation of the purchase price to the net assets acquired (amounts in thousands):

Cash and cash equivalents acquired	$ 771
Reimbursable contract costs	43
Prepaid expenses and other current assets	307
Property, plant and equipment	493
Other	3
Goodwill	23,171
In-process research and development	21,000
Accounts payable and accrued expenses	(540)
Accrued transaction costs	(1,303)
Total fair value of equities issued	$ 43,945

The following unaudited pro forma condensed consolidated information has been prepared to give effect to the acquisition as if such transaction had occurred at the beginning of the periods presented. The historical results have been adjusted to reflect: i) elimination of the one-time charge to operations for the purchase of acquired in-process research and development, ii) amortization of goodwill arising from the transaction, and iii) elimination of income tax benefits or expenses that would not have been realized on a combined basis (amounts in thousands, except per share amounts).

	Years Ended July 31	
	2001 Pro forma	2000 Pro forma
Contract research revenues	$ 12,926	$ 24,019
Net loss before cumulative effect of adoption of SAB 101	(27,724)	(23,955)
Net loss	(36,842)	(23,955)
Basic and diluted net loss per common share	(2.11)	(1.68)
Shares used in computing basic and diluted net loss per common share	17,423	14,270

Had SAB 101 been retroactively applied to the pro forma information for the year ended July 31, 2000, contract revenues would increase and net loss would decrease by $588,000. Basic and diluted net loss per share would be reduced to $(1.64) per share for that year.

The unaudited pro forma condensed consolidated financial information is not necessarily indicative of what actual results would have been had the transaction occurred on the dates indicated and do not purport to indicate the results of future operations.

(5) PROPERTY, PLANT, AND EQUIPMENT

Property, plant, and equipment is recorded at cost and is depreciated over the estimated useful lives of the assets involved. Depreciation and amortization commences at the time the assets are placed in service and is computed using the straight-line method over the estimated useful lives of the assets. Maintenance and repairs are charged to expense when incurred. Depreciation and amortization of fixed assets, was approximately $2,095,000, $1,429,000, and $764,000 for the years ended July 31, 2001, 2000, and 1999, respectively.

Asset	Estimated Useful Life
Building and building improvements	15 years
Leasehold improvements	Life of lease
Laboratory equipment	5 years
Furniture and office equipment	3 years

A summary of property, plant, and equipment is as follows (amounts in thousand):

	July 31, 2001	July 31, 2000
Land	$ 364	$ 364
Building, building improvements and leasehold improvements	9,088	4,070
Laboratory and support equipment	9,338	7,378
Furniture and office equipment	1,852	1,217
	20,642	13,029
Less - Accumulated depreciation and amortization	(6,911)	(4,816)
	$ 13,731	$ 8,213

(6) ACCRUED EXPENSES

A summary of accrued expenses is as follows (amounts in thousands):

	July 31, 2001	July 31, 2000
Payroll and employee benefits	$ 1,121	$ 809
Accrued rent	452	-
Research and development expenses	398	176
Other	300	244
	$ 2,271	$ 1,229

(7) NOTES PAYABLE

A summary of notes payable is as follows (amounts in thousands):

	July 31, 2001	July 31, 2000
Term note payable to US Surgical bearing interest at 6% per annum, payable quarterly. The principal balance under the note matures in May 2005. The note payable is secured by certain manufacturing assets of Columbus.	$ 3,920	$ 3,920
Term loan payable to a bank repaid in fiscal 2001.	-	369
	3,920	4,289
Less - current portion	-	369
Total long-term	$ 3,920	$ 3,920

(8) CONVERTIBLE SUBORDINATED NOTES

In March 2000, the Company completed a $120 million private placement of 5.75% Convertible Subordinated Notes due March 15, 2007. The notes bear interest payable semi-annually on September 15 and March 15 of each year, beginning September 15, 2000. The holders may convert all or a portion of the notes into common stock at any time on or before March 15, 2007 at a conversion price of $106.425 per share.

The notes are subordinated to all the Company's existing and future senior indebtedness and are effectively subordinated to all of the indebtedness and other liabilities (including trade and other payables) of the Company and its subsidiaries. The indenture governing the notes does not limit the amount of indebtedness, including senior indebtedness, which the Company may incur.

Noteholders may require the Company to repurchase their notes upon a repurchase event as defined by the loan agreement in cash, or, at the option of the Company, in common stock, at 105% of the principal amount of the notes, plus accrued and unpaid interest.

The notes are not entitled to any sinking fund. At any time or from time to time on or after March 20, 2003 and ending on March 14, 2007, the Company may redeem some or all the notes on at least 30 days notice as a whole or, from time to time, in part at certain premiums over the principal amount plus accrued interest.

The Company incurred deferred financing costs related to this offering of approximately $4.0 million which are recorded in the consolidated balance sheet and are being amortized as a component of interest expense over the seven-year term of the notes. Amortization expense associated with the financing costs was $566,000 and $185,000 for the years ended July 31, 2001 and 2000, respectively.

(9) LICENSE AND RESEARCH & DEVELOPMENT AGREEMENTS

The Company has entered into a number of license and research and development agreements since its inception. These agreements have been made with various research institutions, universities, and government agencies in order to advance and obtain technologies management believes important to the Company's overall business strategy.

License agreements generally provide for an initial fee followed by annual minimum royalty payments. Additionally, certain agreements call for future payments upon the attainment of agreed to milestones, such as, but not limited to, Investigational New Drug (IND) application or Product License Approval (PLA). These agreements require minimum royalty payments based upon sales developed from the applicable technologies, if any.

Research and development agreements generally provide for the Company to fund future project research and clinical trials. Based upon these agreements, the Company may obtain exclusive and non-exclusive rights and options to the applicable technologies developed as a result of the applicable research.

The minimum fixed payments (assuming non-termination of the above agreements) as of July 31, 2001, for each of the next five years are as follows (amounts in thousands):

Years Ending July 31,	License Agreements	Research and Development Agreements
2002	$ 487	$ 4,680
2003	367	6,504
2004	347	-
2005	402	-
2006	425	-

Should the Company achieve certain milestones related to product development and product license applications and approvals, additional payments would be required. As of July 31, 2001, these agreements contain milestone payment provisions aggregating approximately $15.9 million. The agreements also require the Company to fund certain costs associated with the filing of patent applications.

(10) CONTRACT RESEARCH REVENUES

During the three years ended July 31, 2001 the Company recorded contract research revenues from research and development support payments and license fees under collaboration with third parties and amounts received from various government grants.

In January 1999, the Company and Procter & Gamble Pharmaceuticals Inc. ("P&G") entered into an exclusive collaboration to develop and commercialize pexelizumab, one of the Company's lead product candidates. Under this collaboration, the Company is pursuing the development of pexelizumab for the treatment of inflammation caused by cardiopulmonary bypass surgery, heart attack, and angioplasty. The Company has granted P&G an exclusive license to the Company's intellectual property related to pexelizumab, with the right to sublicense. P&G has agreed to fund clinical development and manufacturing costs relating to pexelizumab for these indications. Additionally, P&G has agreed to pay the Company up to $95 million in payments, which include a non-refundable up-front license fee, milestone payments, and research and development support payments. The Company will also receive royalties on worldwide sales of pexelizumab, if any, for all indications. The Company also has a preferred position relative to third-party manufacturers to manufacture

pexelizumab worldwide. The Company shares co-promotion rights with P&G to sell, market and distribute pexelizumab in the United States, and has granted P&G the exclusive rights to sell, market and distribute pexelizumab outside of the United States. Through July 31, 2001, the Company received proceeds of approximately $44.8 million from P&G, including receiving a non-refundable up-front license fee of $10 million in fiscal 1999 (see Note 2) and $34.8 million for research and development support expenses. Approximately 91% and 87% of reimbursable contract costs relate to the P&G collaboration agreement as of July 31, 2001 and 2000, respectively.

The Company has been awarded various grants by agencies of the U.S. government to fund specific research projects. Based upon costs incurred under these projects as of July 31, 2001, the Company has up to approximately $1.5 million of additional funding available under these grants.

A summary of revenues generated from contract research collaboration and grant awards is as follows (amounts in thousands):

Collaboration/Grant Awards	Years Ended July 31, 2001	2000	1999
P&G	$ 9,728	$ 19,708	$ 17,753
U.S. government grants	1,677	1,733	834
Other	400	-	167
	11,805	21,441	18,754
Pro forma revenue as if SAB 101 was retroactively adopted (see Note 2)	-	588	(9,706)
Total pro forma revenues	$ 11,805	$ 22,029	$ 9,048

(11) COMMITMENTS

The Company has entered into two, three and five year employment agreements with certain of its executives. These agreements provide that these individuals will receive aggregate annual base salaries of approximately $928,000 as of July 31, 2001. These individuals may also receive discretionary bonus awards, as determined by the Board of Directors.

As of July 31, 2001, the Company leases its headquarters and research and development facilities. The lease commenced in August 2000 and has a term of ten years and six months. The Company is required to pay a pro rata percentage of real estate taxes and operating expenses. Monthly fixed rent starts at approximately $80,000, increasing to approximately $96,000 over the term of this lease. The Company has issued a $200,000 open letter of credit to secure the lease.

The pilot manufacturing plant, which is used for producing compounds for clinical trials, has a monthly fixed rent of approximately $16,500. The Company is currently leasing the facility on a month to month basis. The Company leases additional research facilities at a monthly fixed rent of approximately $14,200. This lease expires in August 2002.

Lease expense for the Company's facilities was $1,536,000, $694,000, and $420,000 for the years ended July 31, 2001, 2000 and 1999, respectively. Lease expense is being recorded on a straight-line basis over the applicable rental terms.

Aggregate future minimum annual rental payments for the next five years under noncancellable operating leases (including facilities and equipment) are as follows (amounts in thousands):

Years Ended July 31,	
2002	$ 1,178
2003	1,146
2004	1,131
2005	1,095
2006	1,085
2007 and thereafter	5,127

(12) COMMON STOCK

Fiscal 2001 Common Stock Sale

In October 2000, the Company filed a shelf registration statement to offer up to $300 million of equity securities. In November 2000, the Company sold 2.3 million shares of common stock at a price of $90.75 per share resulting in proceeds of approximately $208.5 million, net of fees and other expenses of approximately $201,000 related to the transaction.

Fiscal 2000 Common Stock Sale

In November 1999, the Company sold 3.415 million shares of common stock at a price of $14 per share in a follow-on public offering resulting in net proceeds of approximately $44.4 million net of fees and other expenses of $3.4 million related to the transaction.

(13) STOCK OPTIONS AND WARRANTS

Stock Options

During fiscal 2001, the stockholders of the Company approved the adoption of the 2000 Stock Option Plan and elected to terminate the previous 1992 Plan. Under the 2000 Plan, incentive and nonqualified stock options may be granted for up to a maximum of 1,500,000 shares of common stock to directors, officers, key employees and consultants of the Company. As July 31, 2001, approximately 376,700 options were available for grant under the 2000 Plan. During fiscal 2001, the stockholders of the Company approved an amendment to the 1992 Stock Option Plan for Outside Directors. This amendment increases the number of stock options granted initially to qualifying directors as well as upon annual re-election to the board of directors. Options generally become exercisable in equal proportions over three to four years and remain exercisable for up to ten years after the grant date, subject to certain conditions.

SFAS No. 123, "Accounting for Stock-Based Compensation" requires the measurement of the fair value of stock options or warrants to be disclosed in the notes to financial statements. The Company has computed the pro forma disclosure required under SFAS No. 123 for options granted using the Black-Scholes option pricing model prescribed by SFAS No. 123. The assumptions used are as follows:

	2001	2000	1999
Risk free interest rate	4.7%	6.0%	5.0%
Expected dividend yield	0%	0%	0%
Expected lives	5 years	5 years	5 years
Expected volatility	101%	85%	65%

Had compensation cost for the Company's stock option plans been determined based on the fair value at the grant dates of awards under these plans consistent with the method of SFAS No. 123, the Company's net loss and pro forma net loss per common share would have been increased to the pro forma amounts indicated below (amounts in thousands, except per share amounts):

	2001	2000	1999
Net loss:			
As reported	$ (57,043)	$ (20,227)	$ (6,395)
Pro forma	(69,470)	(22,887)	(8,419)
Net loss per common share:			
As reported	$ (3.28)	$ (1.45)	$ (0.57)
Pro forma	(4.00)	(1.64)	(0.74)

A summary of the status of the Company's stock option plans at July 31, 2001, 2000 and 1999 and changes during the years then ended is presented in the table and narrative below:

	2001		2000		1999	
	Options	Weighted Average Exercise Price	Options	Weighted Average Exercise Price	Options	Weighted Average Exercise Price
Outstanding at August 1	2,684,215	$ 21.09	2,348,587	$ 8.10	1,727,986	$ 7.40
Granted	1,275,164	30.05	644,800	63.18	780,750	9.64
Exercised	(299,525)	7.34	(225,083)	7.56	(66,587)	5.75
Cancelled	(98,334)	33.20	(84,089)	17.34	(93,562)	9.73
Outstanding at July 31	3,561,520	$ 25.12	2,684,215	$ 21.09	2,348,587	$ 8.10
Options exercisable at July 31	1,622,164	$ 14.65	1,443,554	$ 7.26	1,238,398	$ 6.46
Weighted-average fair value of options granted during the year		$ 25.70		$ 45.02		$ 6.52

During fiscal 2001, options to purchase 1,220,800 shares of common stock were granted to employees at exercise prices equal to the fair value of the stock at the date of grant. The weighted average exercise price of these options was $29.16 per share. The weighted average fair value of these options at the date of grant was $22.40 per option. In addition, options to purchase 10,000 shares of common stock were granted to employees at exercise prices which were less then the fair value of the common stock at the date of grant. Accordingly, the Company is recording compensation expense based upon this difference over the vesting period associated with these options. Compensation expense associated with these options is $121,000 for the year ended July 31, 2001. Aggregate compensation expense of approximately $203,000 associated with these option grants is expected to be recognized over the next three years. The weighted average exercise price of these options was $75.51 per share. The weighted average fair value of these options at the date of grant was $92.27 per option.

In connection with acquisition of Prolifaron (see Note 4), the Company issued fully vested options to purchase 44,364 shares of common stock at a weighted average exercise price of $44.35 per share. The weighted average fair value of these options at the date of grant was $101.46 per option. The value of these options were included as a component of the purchase price of Prolifaron at the date of acquisition.

During fiscal 2000, options to purchase 644,800 shares of common stock were granted to employees and consultants of the Company at an exercise price equal to the fair value of the stock at the date of grant. The Company is recording compensation expense based upon the fair value of the options granted to consultants over the vesting term. Compensation expense related to these options was $93,000 and $239,000 for the years ended July 31, 2001 and 2000, respectively.

During fiscal 1999, options to purchase 513,500 shares of common stock were granted to employees at exercise prices equal to the fair value of the stock at the date of grant. The weighted average exercise price of these options was $9.98 per share. The weighted average fair value of these options at the date of grant was $5.89 per option. In addition, options to purchase 267,250 shares of common stock were granted to employees subject to shareholders' approving an increase in total shares available to be granted under the plan. These options were granted at an exercise price of $9.00 per share which was equal to the fair value of the common stock at the date of grant. However, the exercise price of these options was less than the fair value of the stock at the date of shareholder approval. Accordingly, the Company is recording compensation expense based upon this difference over the vesting period associated with these options. Compensation expense associated with these options is $194,000, $191,000 and $132,000 for the years ended July 31, 2001, 2000 and 1999, respectively. Aggregate compensation expense of approximately $166,000 associated with these option grants is expected to be recognized over the next two years. The weighted average fair value of these options at the date of shareholder approval was $7.73 per option.

The following table presents weighted average price and life information about significant option groups outstanding at July 31, 2001:

Range of Exercise Prices	Number Outstanding	Weighted Average Remaining Contractual Life (Yrs.)	Weighted Average Exercise Price	Number Exercisable	Weighted Average Exercise Price
$ 2.37 - $ 9.00	752,850	4.5	$ 5.55	687,412	$ 5.25
$ 9.01 - $ 20.99	975,785	6.4	10.49	725,161	10.40
$ 21.00 - $ 22.50	955,300	9.7	21.00	-	-
$ 32.00 - $ 54.00	182,085	8.9	37.46	48,585	43.21
$ 61.00 - $ 87.00	655,500	8.8	66.91	161,006	65.32
$ 106.00 - $ 108.00	40,000	9.1	107.88	-	-
	3,561,520	7.5	$ 25.12	1,622,164	$ 14.65

Warrants

In connection with the Company's initial public offering in 1996, the Company sold to its underwriter, for nominal consideration, warrants to purchase 220,000 shares of common stock which have been fully exercised as of July 31, 2001. These warrants were exercisable at a price of $9.90 per share for a period of forty-two (42) months commencing on August 27, 1997. During fiscal 2000, warrants to purchase 201,883 shares were exercised resulting in proceeds of $2.0 million to the Company. During fiscal 2001, the remaining warrants to purchase 18,117 shares were exercised resulting in proceeds of approximately $179,000 to the Company.

(14) RIGHTS TO PURCHASE PREFERRED STOCK

In February 1997, the Board of Directors of the Company declared a dividend of one preferred stock purchase right for each outstanding share of common stock (including all future issuances of common stock). Under certain conditions, each right may be exercised to purchase one one-hundredth of a share of a new series of preferred stock at an exercise price of $75.00 (see below), subject to adjustment. The rights may be exercised only after a public announcement that a party acquired 20% or more of the Company's common stock or after commencement or public announcement to make a tender offer for 20% or more of the Company's common stock. The rights, which do not have voting rights, expire on March 6, 2003, and may be redeemed by the Company at a price of $.01 per right at any time prior to their expiration or the acquisition of 20% or more of the Company's stock. The preferred stock purchasable upon exercise of the rights will have a minimum preferential dividend of $10.00 per year, but will be entitled to receive, in the aggregate, a dividend of 100 times the dividend declared on a share of common stock. In the event of a liquidation, the holders of the shares of preferred stock will be entitled to receive a minimum liquidation payment of $100 per share, but will be entitled to receive an aggregate liquidation payment equal to 100 times the payment to be made per share of common stock.

On September 18, 2000, the Board of Directors of the Company amended the purchase price under the preferred stock purchase rights. Such purchase price, for each one one-hundredth of a share of preferred stock to be issued upon the exercise of each preferred stock purchase right was increased from $75.00 to $725.00. Except for the increase in the purchase price, the terms and conditions of the rights remain unchanged.

In the event that the Company is acquired in a merger, other business combination transaction, or 50% or more of its assets, cashflow, or earning power are sold, proper provision shall be made so that each holder of a right shall have the right to receive, upon exercise thereof at the then current exercise price, that number of shares of common stock of the surviving company which at the time of such transaction would have a market value of two times the exercise price of the right.

(15) 401(k) PLAN

The Company has a 401(k) plan. Under the plan, employees may contribute up to 15 percent of their compensation with a maximum of $10,500 per employee in calendar year 2000. The Company matches contributions at a rate of $0.50 for each dollar deferred up to the first 6% of compensation. The Company made matching contributions of approximately $177,000, $127,000, and $85,000 for the years ended July 31, 2001, 2000 and 1999, respectively.

(16) INCOME TAXES

At July 31, 2001, the Company has available for federal tax reporting purposes, net operating loss carryforwards of approximately $97.6 million which expire through 2021 (of which approximately $11.2 million resulted from the exercise of nonqualified stock options as discussed below). The Company also has federal and state research and development credit carryforwards of approximately $10.0 million which begin to expire commencing in fiscal 2008. The Tax Reform Act of 1986 contains certain provisions that limits the Company's ability to utilize net operating loss and tax credit carryforwards in any given year resulting from cumulative changes in ownership interests in excess of 50% over a three-year period. The Company believes it has triggered these limitation provisions.

Recently enacted state tax legislation in Connecticut may allow the Company to exchange certain research and development tax credit carryforwards generated in fiscal 2001 for a cash payment. If the Company elects to exchange such credits for cash, the Company will recognize the value associated with these tax credit carryforwards when receivable from the applicable taxing authority.

The Company follows SFAS No. 109, "Accounting for Income Taxes." This statement requires that deferred income tax assets and liabilities reflect the impact of "temporary differences" between the amount of assets and liabilities for financial reporting purposes and such amounts as measured by tax laws and regulations.

The components of deferred income tax assets are as follows (amounts in thousands):

	July 31, 2001	July 31, 2000
Deferred tax assets:		
Net operating loss carryforwards, federal and state	$ 36,870	$ 24,250
Tax credit carryforwards	9,990	5,580
Deferred revenues	3,350	-
Other	-	230
Total deferred tax assets	50,210	30,060
Less: Valuation allowance for deferred tax assets	50,210	30,060
Net deferred tax assets	$ -	$ -

The exercise price of nonqualified stock options gives rise to compensation which is included in the taxable income of the applicable employees and deducted by the Company for federal and state income tax purposes. As a result of the exercise of nonqualified stock options, the Company has related net operating loss carryforwards of approximately $11.2 million which can be used to offset future taxable income, if any. When realized, the related tax benefits of these net operating loss carryforwards will be credited directly to paid in capital.

The reconciliation of the statutory Federal income tax rate to the Company's effective income tax rate is as follows:

	Years ended July 31, 2001	2000	1999
Statutory rate	(34)%	(34)%	(34)%
State tax benefit, net of Federal taxes	(5)	(5)	(5)
In-process research and development	14	-	-
Amortization of goodwill	2	-	-
Research & development credits	(5)	(11)	-
Increase in deferred tax valuation allowance	28	50	39
Effective rate	-%	-%	-%

The Company has not yet achieved profitable operations. Accordingly, management believes the tax benefits as of July 31, 2001 do not satisfy the realization criteria set forth in SFAS No. 109 and has recorded a valuation allowance for the entire deferred tax assets.

(17) UNAUDITED QUARTERLY FINANCIAL INFORMATION

The following is condensed quarterly financial information (amounts in thousands, except per share amounts):

	Fiscal 2001			
	First quarter	Second quarter	Third quarter	Fourth quarter
Contract research revenues	$ 3,399	$ 3,174	$ 1,960	$ 3,272
Operating expenses	33,650	12,248	11,125	12,884
Operating loss	(30,251)	(9,074)	(9,165)	(9,612)
Net loss before cumulative effect of adopting SAB 101	(29,441)	(4,486)	(6,462)	(7,536)
Cumulative effect of adopting SAB 101	(9,118)	-	-	-
Net loss applicable to common shareholders	(38,559)	(4,486)	(6,462)	(7,536)
Net loss per common share: Basic and diluted	(2.52)	(0.25)	(0.36)	(0.42)

	Fiscal 2000			
	First quarter	Second quarter	Third quarter	Fourth quarter
Contract research revenues	$ 6,288	$ 6,679	$ 4,483	$ 3,991
Operating expenses	11,755	10,990	11,634	9,983
Operating loss	(5,467)	(4,311)	(7,151)	(5,992)
Net loss applicable to common shareholders	(5,177)	(3,639)	(6,319)	(5,092)
Net loss per common share: Basic and diluted	(0.46)	(0.26)	(0.42)	(0.34)

The Company was required to adopt SAB 101 no later than July 31, 2001. The Company elected to adopt SAB 101 during the quarter ended April 30, 2001, retroactive to August 1, 2000. Accordingly, the following quarterly information for the fiscal quarters ended October 31, 2000 and January 31, 2001, reflects the quarters as previously reported prior to adoption and as restated for the retroactive adoption of SAB 101 to August 1, 2000, as noted in the column headings. The impact of the change resulted in an increase in total revenues and corresponding decrease in loss before cumulative effect of a change in accounting principle of $147,000 for each of the quarters ended October 31, 2000 and January 31, 2001 as compared to amounts previously reported in Form 10-Q's filed with the SEC (amounts in thousands, except per share amounts).

	First Quarter Ended October 31, 2000		Second Quarter Ended January 31, 2001	
	As previously reported	Restated	As previously reported	Restated
Contract research revenues	$ 3,252	$ 3,399	$ 3,027	$ 3,174
Operating expenses	33,650	33,650	12,248	12,248
Operating loss	(30,398)	(30,251)	(9,221)	(9,074)
Total other income, net	810	810	4,588	4,588
Net loss before cumulative effect of adopting SAB 101	(29,588)	(29,441)	(4,633)	(4,486)
Cumulative effect of adopting SAB 101	-	(9,118)	-	-
Net loss	$ (29,588)	$ (38,559)	$ (4,633)	$ (4,486)
Basic and diluted net loss per common share	$ (1.93)	$ (2.52)	$ (0.26)	$ (0.25)
Shares used in computing basic and diluted net loss per common share	15,323	15,323	17,999	17,999

Exhibit 21.1

SUBSIDIARIES OF ALEXION PHARMACEUTICALS, INC.

Columbus Farming Corporation (New York)
100% owned by Registrant

Alexion Antibody Technologies, Inc. (California)
100% owned by Registrant

EXHIBIT 23.1

Consent of Independent Public Accountants

As independent public accountants, we hereby consent to the incorporation of our report included in this Form 10-K, into the Company's previously filed Registration Statements File numbers 333-19905, 333-24863, 333-29617, 333-41397, 333-47645, 333-71879, 333-71985, 333-36738, 333-69478, 333-47594, 333-52856, 333-52886 and 333-59702.

/s/ ARTHUR ANDERSEN LLP

Hartford, Connecticut
October 25, 2001

RISK FACTORS

The following risk factors should be carefully considered in evaluating our Company and our business because these risk factors have a significant impact on our business, operating results, financial condition, and cash flows. If any of these risks actually occurs, our business, financial condition, operating results and/or cash flows could be harmed.

If we continue to incur operating losses, we may be unable to continue our operations.

We have incurred losses since we started our company in January 1992. As of July 31, 2001, we had an accumulated deficit of approximately $124.3 million. If we continue to incur operating losses and fail to become a profitable company, we may be unable to continue our operations. Since we began our business, we have focused on research and development of product candidates. We have no products that are available for sale and do not know when we will have products available for sale, if ever. We expect to continue to operate at a net loss for at least the next several years as we continue our research and development efforts, continue to conduct clinical trials and develop manufacturing, sales, marketing and distribution capabilities. Our future profitability depends on our receiving regulatory approval of our product candidates and our ability to successfully manufacture and market approved drugs. The extent of our future losses and the timing of our profitability, if we are ever profitable, are highly uncertain.

If we do not obtain regulatory approval for our drug products, we will not be able to sell our drug products.

We cannot sell or market our drugs without regulatory approval. If we do not obtain regulatory approval for our products, the value of our company and our results of operations will be harmed. In the United States, we must obtain approval from the U.S. Food and Drug Administration, or FDA, for each drug that we intend to sell. Obtaining FDA approval is typically a lengthy and expensive process, and approval is highly uncertain. Foreign governments also regulate drugs distributed outside the United States, whose approval can also be lengthy, expensive and highly uncertain. None of our product candidates has received regulatory approval to be marketed and sold in the United States or any other country. We do not anticipate receiving regulatory approval of any of our product candidates, if ever, for at least the next several years.

If our drug trials are delayed or achieve unfavorable results, we will have to delay or may be unable to obtain regulatory approval for our products.

We must conduct extensive testing of our product candidates before we can obtain regulatory approval for our products. We need to conduct both preclinical animal testing and clinical human trials. These tests and trials may not achieve favorable results. We would need to reevaluate any drug that did not test favorably and either alter the study, the drug or the dose and perform additional or repeat tests, or abandon the drug development project. In those circumstances, we would not be able to obtain regulatory approval on a timely basis, if ever. Even if approval is granted it may entail limitations on the indicated uses for which the drug may be marketed.

We have announced the completion of a Phase IIb trial of pexelizumab for the treatment of complications in patients after cardiopulmonary bypass surgery, including the reduction of the frequency and severity of myocardial infarctions and frequency of death, and completion of a Phase II trial of 5G1.1 for the treatment of rheumatoid arthritis. Completion of these and other trials does not guarantee that we will initiate additional trials for our product candidates, that if the trials are initiated what the scope and phase of the trial will be or that they will be completed, or that if the trials are completed, the results will provide a sufficient basis to proceed with further trials or to apply for or receive regulatory approvals or to commercialize products. Results of these trials could be inconclusive, necessitating additional or repeat trials.

There are many reasons why drug testing could be delayed or terminated. For human trials, patients must be recruited and each product candidate must be tested at various doses and formulations for each clinical indication. Also, to ensure safety and effectiveness, the effect of drugs often must be studied over a long period of time,

especially for the chronic diseases that we are studying. Unfavorable results or insufficient patient enrollment in our clinical trials could delay or cause us to abandon a product development program.

Additional factors that can cause delay or termination of our clinical trials include:

- slow patient enrollment;
- long treatment time required to demonstrate effectiveness;
- lack of sufficient supplies of the product candidate;
- adverse medical events or side effects in treated patients;
- lack of effectiveness of the product candidate being tested; and
- lack of sufficient funds.

We may expand our business through new acquisitions that could disrupt our business and harm our financial condition.

Our business strategy includes expanding our products and capabilities, and we may seek acquisitions to do so. Acquisitions involve numerous risks, including:

- substantial cash expenditures;
- potentially dilutive issuance of equity securities;
- incurrence of debt and contingent liabilities;
- difficulties in assimilating the operations of the acquired companies;
- diverting our management's attention away from other business concerns;
- risks of entering markets in which we have limited or no direct experience; and
- the potential loss of our key employees or key employees of the acquired companies.

We cannot assure you that any acquisition will result in long-term benefits to us. We may incorrectly judge the value or worth of an acquired company or business. In addition, our future success would depend in part on our ability to manage the rapid growth associated with some of these acquisitions. We cannot assure you that we will be able to make the combination of our business with that of acquired businesses or companies work or be successful. Furthermore, the development or expansion of our business or any acquired business or companies may require a substantial capital investment by us. We may not have these necessary funds nor may they be readily available to us on acceptable terms or at all. We may also seek to raise funds by selling shares of our stock, which could dilute your ownership interest in our company.

On September 22, 2000, we purchased all of the capital stock and other outstanding securities of Prolifaron, Inc., a privately held biopharmaceutical company that is developing therapeutic antibodies addressing multiple disease, including cancer, for approximately 400,000 shares of our outstanding capital stock. We cannot assure you that the integration of our business with the businesses of Prolifaron will be successful.

If we fail to obtain the capital necessary to fund our operations, we will be unable to continue or complete our product development.

We believe we have sufficient capital to fund our operations and product development for at least thirty-six months. We may need to raise additional capital after that time to complete the development and commercialization of our product candidates. Additional financing could take the form of public or private debt or equity offerings, equity line facilities, bank loans and/or collaborative research and development arrangements with corporate partners. The amount of capital we may need depends on many factors, including:

- the progress, timing and scope of our research and development programs;
- the progress, timing and scope of our preclinical studies and clinical trials;

. the time and cost necessary to obtain regulatory approvals;

. the time and cost necessary to further develop manufacturing processes, arrange for contract manufacturing or build manufacturing facilities and obtain the necessary regulatory approvals for those facilities;

. the time and cost necessary to develop sales, marketing and distribution capabilities;

. changes in applicable governmental regulatory policies; and

. any new collaborative, licensing and other commercial relationships that we may establish.

We may not get funding when we need it or funding may only be available on unfavorable terms. If we cannot raise adequate funds to satisfy our capital requirements, we may have to delay, scale-back or eliminate our research and development activities or future operations. We might have to license our technology to others. This could result in sharing revenues that we might otherwise retain for ourselves. Any of these actions may harm our business.

If our collaboration with Procter & Gamble is terminated or Procter & Gamble reduces its commitment to our collaboration, our ability to commercialize pexelizumab in the time expected, or at all, and our business would be harmed.

We rely exclusively on Procter & Gamble to perform development, obtain commercial manufacturing, and provide sales and marketing for pexelizumab. While we cannot assure you that pexelizumab will ever be successfully developed and commercialized if Procter & Gamble does not perform its obligations in a timely manner, or at all, our ability to commercialize pexelizumab in a timely manner or at all, will be significantly adversely affected. We rely exclusively on Procter & Gamble to provide funding and additional resources for the development and commercialization of pexelizumab. These include funds and resources for:

. clinical development and clinical and commercial manufacturing;

. obtaining regulatory approvals; and

. sales, marketing and distribution efforts worldwide.

We cannot guarantee that Procter & Gamble will devote the resources necessary to successfully develop and commercialize pexelizumab in a timely manner, if at all. Furthermore, Procter & Gamble may devote the necessary resources, but we may still not successfully develop and commercialize pexelizumab. Either party may terminate our collaboration agreement for specified reasons, including a material breach.

Termination of our agreement with Procter & Gamble would cause significant delays in the development of pexelizumab and result in additional development costs. We would need to fund the development and commercialization of pexelizumab on our own or identify a new development partner. We might also have to repeat testing already completed with Procter & Gamble.

If we are unable to engage and retain third-party collaborators, our research and development efforts may be delayed.

We depend upon third-party collaborators to assist us in the development of our product candidates. If any of our existing collaborators breaches or terminates its agreement with us or does not perform its development work under an agreement in a timely manner or at all, we would experience significant delays in the development or commercialization of our product candidates. We would also experience significant delays if we could not engage additional collaborators when required. In either event, we would be required to devote additional funds or other resources to these activities or to terminate them. This would divert funds or other resources from other parts of our business.

We cannot assure you that:

. current collaboration arrangements will be continued in their current form;

. we will be able to negotiate acceptable collaborative agreements to develop or commercialize our products;

. any arrangements with third parties will be successful; or

. current or potential collaborators will not pursue treatments for other diseases or seek other ways of developing treatments for our disease targets.

If the trading price of our common stock continues to fluctuate in a wide range, our stockholders will suffer considerable uncertainty with respect to an investment in our stock.

The trading price of our common stock has been volatile and may continue to be volatile in the future. Factors such as announcements of fluctuations in our or our competitors' operating results, fluctuations in the trading prices or business prospects of our competitors and collaborators, including, but not limited to Procter & Gamble, changes in our prospects, and market conditions for biotechnology stocks in general could have a significant impact on the future trading prices of our common stock and our outstanding notes. In particular, the trading price of the common stock of many biotechnology companies, including ours, has experienced extreme price and volume fluctuations, which have at times been unrelated to the operating performance of the companies whose stocks were affected. This is due to several factors, including general market conditions, the announcement of the results of our clinical trials or product development and the results of our attempts to obtain FDA approval for our products. In particular, since August 1, 1999, the sales price of our common stock has ranged from a low of $10.00 per share to a high of $119.88 per share. While we cannot predict our future performance, if our stock continues to fluctuate in a wide range, an investment in our stock or our outstanding notes may result in considerable uncertainty for an investor.

If we cannot protect the confidentiality and proprietary nature of our trade secrets, our business and competitive position will be harmed.

Our business requires using sensitive technology, techniques and proprietary compounds that we protect as trade secrets. However, since we are a small company, we also rely heavily on collaboration with suppliers, outside scientists and other drug companies. Collaboration presents a strong risk of exposing our trade secrets. If our trade secrets were exposed, it would help our competitors and adversely affect our business prospects.

In order to protect our drugs and technology more effectively, we need to obtain patents covering the drugs and technologies we develop. Our drugs are expensive and time-consuming to test and develop. Without patent protection, competitors may copy our methods, or the chemical structure or other aspects of our drug. Even if we obtain patents, the patents may not be broad enough to protect our drugs from copycat products.

If we are found to be infringing on patents owned by others, we may be forced to obtain a license to continue the sale or development of our drugs and/or pay damages.

Parts of our technology, techniques and proprietary compounds and potential drug candidates may conflict with patents owned by or granted to others. If we cannot resolve these conflicts, we may be liable for damages, be required to obtain costly licenses or be stopped from manufacturing, using or selling our products or conducting other activities. For example, we are aware of broad patents owned by others relating to the manufacture, use and sale of recombinant humanized antibodies, recombinant humanized single chain antibodies, recombinant human antibodies, recombinant human single chain antibodies, and genetically engineered animals. Many of our products are genetically engineered antibodies, including recombinant humanized antibodies, recombinant humanized single chain antibodies, recombinant human antibodies, and recombinant human single chain antibodies, and other products are tissues from genetically engineered animals.

We have received notices from the owners of some of these patents claiming that their patents may be relevant to the development of some of our drug candidates. In response to some of these notices, we have obtained licenses. However, with regard to other patents, we have either determined in our judgment that:

. our products do not infringe the patents;

. we do not believe the patents are valid; or

. we have identified and are testing various modifications that we believe should not infringe the patents and which should permit commercialization of our product candidates.

Any patent holders could sue us for damages and seek to prevent us from manufacturing, selling or developing our drugs. Legal disputes can be costly and time consuming to defend. If any of these actions are successful, we could be required to pay damages or to obtain a license to sell or develop our drugs. A required license may not be available on acceptable terms, if at all.

If the testing or use of our products harms people, we could be subject to costly and damaging product liability claims.

The testing, manufacturing, marketing and sale of drugs for use in humans exposes us to product liability risks. Side effects and other problems from using our products could give rise to product liability claims against us. We might have to recall our products, if any, from the marketplace. Some of these risks are unknown at this time. For example, little is known about the potential long-term health risks of transplanting pig tissue into humans, a goal of our UniGraft product development program. Use of C5 Complement Inhibitors, such as 5G1.1 and pexelizumab, is associated with an increased risk for infection with Neisseria bacteria. Serious cases of Neisseria infection can result in brain damage, loss of limbs or parts of limbs, kidney failure, or death.

In addition, we may be sued by people who participate in our clinical trials. A number of patients who participate in such trials are already very ill when they enter a study. Any informed consents or waivers obtained from people who sign up for our trials may not protect us from liability or litigation. Our product liability insurance may not cover all potential liabilities or may not completely cover any covered liabilities. Moreover, we may not be able to maintain our insurance on acceptable terms. In addition, negative publicity relating to a product liability claim may make it more difficult, or impossible, for us to recruit patients for our clinical trials or to market and sell our products. As a result of these factors, a product liability claim, even if successfully defended, could have a material adverse effect on our business, financial condition or results of operations.

If we cannot manufacture our drug candidates in sufficient amounts at acceptable costs and on a timely basis, we may be unable to have the necessary materials for product testing, and later for potential sale in the market. Either event would harm our business.

For our drug trials, we need to produce sufficient amounts of product for testing. Our small manufacturing plant cannot manufacture enough of our product candidates for later stage clinical development. In addition, we do not have the capacity to produce more than one product candidate at a time. We depend on a few outside suppliers for manufacturing. If we experience interruptions in the manufacture of our products for testing, our drug development efforts will be delayed. If any of our outside manufacturers stops manufacturing our products or reduces the amount manufactured, we will need to find other alternatives. If we are unable to find an acceptable outside manufacturer on reasonable terms, we will have to divert our own resources to manufacturing which may not be sufficient to produce the necessary quantity or quality of product. As a result, our ability to conduct testing would be materially adversely affected. Submission of products and new development programs for regulatory approval would be delayed. Our competitive position and our prospects for achieving profitability could be materially and adversely affected.

Manufacture of drug products is highly regulated by the FDA and other domestic and foreign authorities. We cannot assure you that we or our third-party collaborators will successfully comply with all of those regulations, which would have a materially adverse effect on our business.

We have no experience or capacity for manufacturing drug products in volumes that would be necessary to support commercial sales. If we are unable to establish and maintain commercial scale manufacturing within our planned time and cost parameters, sales of our products and our financial performance would be adversely affected.

Other than our agreement with Procter & Gamble to develop and commercialize pexelizumab, we have no arrangements to manufacture our products on a commercial basis. Currently, we are relying on Procter & Gamble to retain appropriate commercial manufacturing for pexelizumab through one or more third-party manufacturers. The failure of Procter & Gamble to obtain appropriate commercial manufacturing for pexelizumab on a timely basis, or at all, may prevent or impede the commercialization of pexelizumab. We have not proceeded far enough in negotiations with any other potential partner to predict the cost of a commercial manufacturing arrangement for our

other potential products nor have we explored the cost or time required to establish our own commercial manufacturing facility.

If we are unable to establish sales, marketing and distribution capabilities, or to enter into agreements with third parties to do so, we will be unable to successfully market and sell future drug products.

We have no sales, marketing or distribution personnel or capabilities. If we are unable to establish those capabilities, either by developing our own capabilities or entering into agreements with others, we will not be able to successfully sell our products. In that event, we will not be able to generate significant revenues. We cannot guarantee that we will be able to hire the qualified sales and marketing personnel we need. We may not be able to enter into any marketing or distribution agreements with third-party providers on acceptable terms, if at all. Currently, we are relying on Procter & Gamble for sales, marketing and distribution of pexelizumab. Procter & Gamble, or any future third-party collaborators, may not succeed at selling, marketing or distributing any of our future drug products.

If we are unable to obtain reimbursement from government health administration authorities, private health insurers and other organizations for our future products, our products may be too costly for regular use and our ability to generate revenues would be harmed.

Our products, once commercialized, like similar products in the market place, may be significantly more expensive than traditional drug treatments. Our future revenues and profitability will be adversely affected if we cannot depend on governmental and private third-party payors to defray the cost of our products to the consumer. If these entities refuse to provide reimbursement with respect to our products or determine to provide a low level of reimbursement, our products may be too costly for general use. Our profitability may be adversely impacted if we choose to offer our products at a reduced price. Any limitation on the use of our products or any decrease on the price of our products without a corresponding decrease in expenses will have a material adverse effect on our ability to achieve profitability.

Even if we successfully develop our products for transplanting animal cells into humans, this technology may not be accepted by the market due to medical concerns or unanticipated regulation.

Our program for the development of animal cells for transplantation into humans may never result in any therapeutic products. This technology is subject to extensive clinical testing and we are not aware of any such technology that has been approved for sale by the FDA or comparable foreign regulatory authorities. Even if we succeed in developing these products, our products may not be widely accepted by the medical community or third-party payors until more facts are established and ethical consensus is reached regarding the use of animal cells. In addition, concerns relating to the risk of introducing animal viruses to infect the human species through the transplantation process may also create additional regulatory hurdles for FDA approval. If accepted, the degree of acceptance may limit the size of the market for our products. Moreover, due to the controversial nature of transplantation of animal cells into humans generally, market prices for our securities may be subject to increased volatility.

If our competitors get to the marketplace before we do with better or cheaper drugs, our drugs may not be profitable to sell or to continue to develop.

Each of Avant Immunotherapeutics, Inc, Millennium Pharmaceuticals, Inc., Tanox, Inc., Abbott Laboratories, Baxter International Inc., Gliatech Inc., Neurogen Corporation, and Biocryst Pharmaceuticals have publicly announced their intentions to develop drugs which target the inflammatory effects of complement in the immune system. We are also aware that Pfizer, Inc., GlaxoSmithKline Plc and Merck & Co., Inc. are also attempting to develop complement inhibitor therapies. Each of Cambridge Antibody Technology Group plc, MorphoSys AG and Dyax Corporation have publicly announced intentions to develop therapeutic human antibodies from libraries of human antibody genes. Additionally, each of Abgenix Inc. and Medarex, Inc. have publicly announced intentions to develop therapeutic human antibodies from mice that have been bred to include some human antibody genes. These and other pharmaceutical companies, many of which have significantly greater resources than we, may develop, manufacture and market better or cheaper drugs than our product candidates. They may establish themselves in the marketplace before we are able to even finish our clinical trials. Other pharmaceutical companies also compete with

us to attract academic research institutions as drug development partners, including for licensing these institutions' proprietary technology. If our competitors successfully enter into such arrangements with academic institutions, we will be precluded from pursuing those specific unique opportunities and may not be able to find equivalent opportunities elsewhere.

If we fail to recruit and retain personnel, our research and product development programs may be delayed.

We are highly dependent upon the efforts of our senior management and scientific personnel, particularly, Leonard Bell, M.D., our president, chief executive officer and director, David W. Keiser, our executive vice president and chief operating officer and Stephen P. Squinto, Ph.D., our executive vice president and head of research. There is intense competition in the biotechnology industry for qualified scientific and technical personnel. Since our business is very science-oriented and specialized, we need to continue to attract and retain such people. We may not be able to continue to attract and retain the qualified personnel necessary for developing our business. We have a key man insurance policy for Dr. Bell and we have employment agreements with each of Dr. Bell, Mr. Keiser and Dr. Squinto. To our knowledge, none of our key personnel is planning to retire or is nearing retirement age. Further, to our knowledge, there is no tension between any of our key personnel and the Board. If we lose the services of our management and scientific personnel or fail to recruit other scientific and technical personnel, our research, and product development programs would be significantly and detrimentally affected.

In particular, we highly value the services of Dr. Leonard Bell, our President and Chief Executive Officer. The loss of his services could materially and adversely affect our ability to achieve our development objectives.