

ATHEROGENICS INC  
Form 10-K  
March 29, 2002

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**SECURITIES AND EXCHANGE COMMISSION**

**Washington, D.C. 20549**

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**Form 10-K**

(Mark One)

**ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d)  
OF THE SECURITIES EXCHANGE ACT OF 1934**

**For the fiscal year ended December 31, 2001**

or

**TRANSITION REPORT UNDER SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF  
1934**

**For the transition period from        to**

**Commission file number 0-31261**

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**AtheroGenics, Inc.**

*(Exact name of Registrant as specified in its charter)*

**Georgia**

*(State or other jurisdiction of  
incorporation or organization)*

**58-2108232**

*(I.R.S. Employer  
Identification Number)*

**8995 Westside Parkway,  
Alpharetta, Georgia 30004**

*(Address of principal executive offices,  
including zip code)*

**(678) 336-2500**

*(Registrant's telephone number,  
including area code)*

**Securities registered pursuant to Section 12(b) of the Exchange Act:**

**None**

**Securities registered pursuant to Section 12(g) of the Exchange Act:**

**Common Stock, No Par Value  
Common Stock Purchase Rights**

Indicate by check mark whether the registrant (1) 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  No

Indicate by check mark if disclosure of delinquent filers in response 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 to Part III of this Form 10-K or any amendment to this Form 10-K.

## Edgar Filing: ATHEROGENICS INC - Form 10-K

The aggregate market value of the voting stock held by non-affiliates of the registrant based on the last sale price for such stock on the Nasdaq National Market at March 20, 2002: \$95,404,999.

The number of shares outstanding of each of the registrant's classes of common stock, as of March 20, 2002: 27,898,873 (one class).

### **Documents Incorporated by Reference:**

**Portions of the proxy statement filed pursuant to Regulation 14A under the Securities Exchange Act of 1934 with respect to the 2002 Annual Meeting of Shareholders are incorporated herein by reference in Part III.**

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**PART I**

**Item 1. Business**

**Overview**

AtheroGenics is a research-based pharmaceutical company incorporated in the State of Georgia in 1993. We are focused on the discovery, development and commercialization of novel drugs for the treatment of chronic inflammatory diseases, such as atherosclerosis, rheumatoid arthritis, organ transplant rejection and asthma. We have developed a proprietary vascular protectant, or v-protectant, technology platform to discover drugs to treat these types of diseases. Based on our v-protectant technology platform, we have advanced three drug candidates into clinical development and are pursuing a number of other preclinical programs.

Our first v-protectants are drug candidates that block the production of proteins that are necessary to initiate and maintain inflammation. For example, one of these proteins, VCAM-1, binds to white blood cells that accumulate at the site of inflammation and directs these cells in their migration from the bloodstream into the tissue. We believe that v-protectants can suppress chronic inflammation by blocking production of VCAM-1 without undermining the body's ability to protect itself against infection.

AGI-1067 is our v-protectant candidate that is most advanced in clinical development. AGI-1067 is designed to benefit patients with coronary artery disease, which is atherosclerosis of the blood vessels of the heart. Atherosclerosis is a common disease that results from inflammation and the buildup of plaque in arterial blood vessel walls. We recently announced positive findings of a Phase II clinical trial, CART-1 (Canadian Antioxidant Restenosis Trial), that assessed in 305 patients the safety and effectiveness of AGI-1067 for the treatment of post-angioplasty restenosis. An analysis of the results indicated that six months after angioplasty, the blood vessels of patients who received AGI-1067 had larger openings, measured as luminal diameters of their coronary arteries, than those who received placebo. CART-1 data also suggested that AGI-1067 may actually reverse the progression of atherosclerosis. In December 2001, we initiated a CART-2 Phase IIb clinical trial for AGI-1067. CART-2 is a 500-patient clinical trial, which examines the effect of 12 months of AGI-1067 therapy on restenosis and atherosclerosis following angioplasty. An additional six-month Phase IIb trial, called DART-1 (Diabetes Atherosclerosis and Restenosis Trial), is being initiated to examine the effects of AGI-1067 on restenosis and atherosclerosis in Type 2 diabetics. Our Phase II clinical trial program follows the successful completion of seven Phase I clinical trials comprising more than 150 men and women.

AGIX-4207, our second v-protectant candidate, is a novel oral agent being developed for the treatment of the signs and symptoms of rheumatoid arthritis. In March 2001, we commenced a Phase I clinical trial to assess the safety and tolerability of AGIX-4207 in healthy volunteers. In February 2002, we received results from the Phase I clinical trial demonstrating that AGIX-4207 is well tolerated over the single and multiple dose ranges studied. Adverse events were generally mild and not considered clinically significant. In October 2001, we commenced a Phase I clinical trial to assess the safety and tolerability of AGIX-4207 I.V. in healthy volunteers. AGIX-4207 I.V. is an intravenous drug designed to treat rheumatoid arthritis patients in whom the rapid attainment of target drug levels in the blood is desirable.

Our third v-protectant candidate, AGI-1096, is a novel antioxidant and selective anti-inflammatory agent which is being developed to address the accelerated inflammation of the grafted blood vessels, known as arteritis, common in chronic organ transplant rejection. We commenced a Phase I clinical trial in February 2002 to assess the safety and tolerability of AGI-1096 in healthy volunteers.

We have identified additional potential v-protectant candidates to treat other chronic inflammatory diseases including, asthma. We are evaluating these v-protectant candidates to choose lead drug candidates for clinical development. We plan to develop these v-protectants rapidly and may seek regulatory fast track status, if available, to expedite development and commercialization. We will continue to expand upon our v-protectant technology platform using functional genomics to identify novel therapeutic gene targets. Functional genomics is the process by which one uses scientific models and techniques to discover and modify genes, measure the consequences of the modifications, and reliably determine the function of those genes.

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In June 2001, we entered into a worldwide exclusive license agreement with National Jewish Medical and Research Center of Denver Colorado to discover and develop novel therapeutics based on MEK kinases (MEKKs) and related technology for the treatment of inflammation. MEKKs are a family of intracellular signaling molecules that we believe play an important role in immuno-inflammatory diseases, such as asthma. Other licensed technology focuses on several naturally occurring substances and their application in the development of a potential treatment for asthma. We believe these new technologies will provide a second broad platform for the discovery and development of a new class of anti-inflammatory drug candidates.

**Inflammation and Disease**

Inflammation is a normal response of the body to protect tissues from infection, injury or disease. The inflammatory response begins with the production and release of chemical agents by cells in the infected, injured or diseased tissue. These agents cause redness, swelling, pain, heat and loss of function. Inflamed tissues generate additional signals that recruit white blood cells to the site of inflammation. White blood cells destroy any infective or injurious agent, and remove cellular debris from damaged tissue. This inflammatory response usually promotes healing but, if uncontrolled, may become harmful.

The inflammatory response can be either acute or chronic. Acute inflammation lasts at most only a few days. The treatment of acute inflammation, where therapy includes the administration of aspirin and other non-steroidal anti-inflammatory agents, provides relief of pain and fever for patients. In contrast, chronic inflammation lasts weeks, months or even indefinitely and causes tissue damage. In chronic inflammation, the inflammation becomes the problem rather than the solution to infection, injury or disease. Chronically inflamed tissues continue to generate signals that attract white blood cells from the bloodstream. When white blood cells migrate from the bloodstream into the tissue they amplify the inflammatory response. This chronic inflammatory response can break down healthy tissue in a misdirected attempt at repair and healing. Diseases characterized by chronic inflammation include, among others:

- atherosclerosis, including coronary artery disease;
- restenosis;
- rheumatoid arthritis;
- solid organ transplant rejection; and
- asthma.

Atherosclerosis is a common disease that results from inflammation and the buildup of plaque in arterial blood vessel walls. Plaque consists of inflammatory cells, cholesterol and cellular debris. Atherosclerosis, depending on the location of the artery it affects, may result in a heart attack or stroke. There are currently no medications available for physicians to treat directly the underlying chronic inflammation of atherosclerosis.

Atherosclerosis of the blood vessels of the heart is called coronary artery disease or heart disease. Treatment for coronary artery disease often progresses to therapeutic procedures, including angioplasty or bypass surgery, to re-establish an effective blood supply to the heart. Angioplasty corrects the blockage by the inflation of a balloon delivered by catheter, with or without the placement of a stent, a small cylindrical mesh device, at the site of the obstructing plaque. After angioplasty, the artery opened by the procedure often re-narrows. Significant re-narrowing may cause angina, a heart attack, or require a repeat angioplasty. Inflammation plays an important role in this re-narrowing called restenosis. There is currently no medical treatment for restenosis. Recently, several companies have announced results from clinical trials showing that drug coated stents may be effective in significantly reducing restenosis. These drug coated stents have not been given approval for use in the United States.

Rheumatoid arthritis is a chronic inflammatory disease of the joints. Rheumatoid arthritis is marked by stiffness, pain, limitations to activity and the destruction of joints, including knees and wrists. Present therapy for rheumatoid arthritis includes non-steroidal anti-inflammatory drugs, corticosteroids, and drugs designed to slow the progression of disease, termed disease modifying anti-rheumatic drugs (DMARDs). DMARDs include drugs that were originally designed to treat cancer, such as methotrexate. DMARDs have serious side

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effects. Recently, two new DMARDs developed by other companies, Enbrel(R) (etanercept) and Remicade(R) (infliximab), have been shown to improve the signs and symptoms of patients with rheumatoid arthritis. These drugs prove that blocking the activity of tumor necrosis factor, a molecule that stimulates a broad range of cellular activities implicated in the inflammation process, improves rheumatoid arthritis. However, both drugs must be injected and both increase the risk of severe infection.

Organ transplantation takes place when an organ from a donor is surgically removed and placed in a recipient patient whose own organ has failed because of disease or infection. Except for transplants between identical twins, all transplant donors and recipients are immunologically incompatible. This biological incompatibility is a barrier that causes the recipient's immune system to try to destroy or reject the new organ. The current treatment for prevention of organ transplant rejection focuses on the use of powerful immunosuppressive drugs such as cyclosporin A, tacrolimus and rapamycin (sirolimus). These drugs, which are initiated during the acute rejection phase, need to be taken continuously after the transplant, often cause side effects, and may still lead to long-term rejection of the transplant. Immunosuppressants may also impair the recipient's immune system in order to reduce the immune response against the transplant. The Scientific Registry of Transplant Recipients reports that even with the use of immunosuppressants, patients run the risk of losing a donated organ during the first three years following transplantation, and roughly 50 percent of patients have functioning organ transplants after approximately ten years.

Asthma is a common chronic inflammatory disease of the bronchial tubes, which are the airways in the lungs. Asthma is marked by episodic airway attacks that are caused by many stresses, including allergy, cold air, ozone or exercise. Asthma therapy has concentrated on the use of inhaled corticosteroids to reduce chronic inflammation and bronchodilators to provide symptomatic relief. Asthmatic patients, however, continue to experience flare-ups, or exacerbations, that are not prevented nor effectively treated by these medicines.

Many physicians are only now becoming aware of the key role of chronic inflammation in diverse diseases such as atherosclerosis and asthma for which existing anti-inflammatory treatments are incomplete and limited in use. As more physicians recognize that a wide range of chronic diseases are inflammatory in nature, we believe that these physicians will require safer and more effective anti-inflammatory treatments. We believe that one of these therapeutic approaches will be the administration of drugs designed to block the migration of white blood cells through blood vessel walls into inflamed tissues unless the inflammation is due to infection.

**V-Protectant Technology**

We have developed a proprietary v-protectant technology platform for the treatment of chronic inflammatory diseases. This platform is based on the work of our scientific co-founders R. Wayne Alexander, M.D., Ph.D. and Russell M. Medford, M.D., Ph.D. In 1993, Drs. Alexander and Medford discovered a novel mechanism within arterial blood vessel walls that could control the excessive accumulation of white blood cells without affecting the body's ability to fight infection. V-protectant technology exploits the observation that the endothelial cells that line the interior wall of the blood vessel play an active role in recruiting white blood cells from the blood to the site of chronic inflammation. V-protectants are drugs that block harmful effects of oxygen and other similar molecules, collectively called oxidants. Scientists have known for some time that some oxidants can damage cells, but have more recently determined that these same oxidants may also act as signals to modify gene activity inside cells. This change in gene activity leads to the production of proteins that initiate or maintain inflammation. The protein products of these cells, including VCAM-1, attract white blood cells to the site of chronic inflammation. We believe that an excess number of VCAM-1 molecules on the surface of cells is a disease state. We also believe that AGI-1067 and other v-protectants can act as antioxidants and can block the specific type of inflammation caused by oxidants acting as signals. We believe that v-protectants will provide this anti-inflammatory benefit without undermining the body's ability to protect itself against infection.



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**V-Protectants Block Activation of VCAM-1**

**In Cells That Line Blood Vessels**

**Activation of VCAM-1**

**Inhibition of VCAM-1**

- 1 Inflammatory agent attaches to cell surface receptor**
- 2 Receptor changes generate oxidant signals inside cell**
- 3 Oxidant signals stimulate gene to produce VCAM-1**
- 4 Cell produces VCAM-1 proteins**
- 5 VCAM-1 migrates to cell surface**
- 6 White blood cells attach to VCAM-1 on cell surface**

**Business Strategy**

Our objective is to become a leading pharmaceutical company focused on discovering, developing and commercializing novel drugs for the treatment of chronic inflammatory diseases. The key elements of our strategy include the following:

*Continue aggressive development program for AGI-1067.* We will seek initially to rapidly develop AGI-1067 for the treatment and prevention of restenosis and the progression of atherosclerosis in patients with coronary artery disease.

*Extend our v-protectant technology platform into additional therapeutic areas that address unmet medical needs.* We believe that our v-protectants have the potential for treating a wide variety of other chronic inflammatory diseases. These indications include: rheumatoid arthritis, asthma, solid organ transplant rejection and other diseases. We recently completed a Phase I clinical trial with a v-protectant compound, AGIX-4207, for the treatment of rheumatoid arthritis. We commenced Phase I clinical trials for AGIX-4207 I.V., an intravenously administered drug for the treatment of rheumatoid arthritis, and AGI-1096, a v-protectant developed for the prevention of solid organ transplant rejection.

*Expand our clinical product candidate portfolio.* In addition to our existing discovery programs, we intend to acquire rights to other product candidates and technologies that complement our existing product candidate lines or that enable us to capitalize on our scientific and clinical development expertise. We plan to expand our product candidate portfolio by in-licensing or acquiring product candidates, technologies or companies. For example, we expect our recent license agreement with National Jewish Medical and Research Center to provide us with a new, complementary platform for the discovery and development of drug candidates to treat inflammatory diseases.

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*Commercialize our products.* We plan to collaborate with large pharmaceutical companies to commercialize products that we develop to target patient or physician populations in broad markets. In contrast, we plan to develop a sales force to commercialize those of our products that we develop to target appropriate patient or physician populations in narrow markets.

**Products**

The table below summarizes our therapeutic programs, their target indication or disease and their development status.

Therapeutic Program	Disease/Indication	Development Status
<b>V-PROTECTANTS</b>		
AGI-1067	Restenosis, atherosclerosis	Phase IIb clinical trial
AGIX-4207	Rheumatoid arthritis	Phase I clinical trial
AGIX-4207 I.V	Rheumatoid arthritis	Phase I clinical trial
AGI-1096	Solid organ transplant rejection	Phase I clinical trial
Oral product candidate	Chronic asthma	Research
<b>DIAGNOSTICS</b>		
OXYKINE® assay	Atherosclerosis	Clinical testing
<b>FUNCTIONAL GENOMICS PROGRAM</b>		
	Inflammatory diseases	Research
<b>MEKK TECHNOLOGY PLATFORM</b>		
	Inflammatory diseases	Research

We have established therapeutic programs for product development using lead candidates we select from among our compound libraries. These programs seek to exploit the value of the products early and to expand their use broadly. We continue to test compounds to identify back-up and second-generation product candidates. We are also pursuing novel discovery targets in chronic inflammation.

**AGI-1067**

AGI-1067, our lead v-protectant product candidate, is a small molecular weight compound that patients take orally once per day. In preclinical testing, AGI-1067 demonstrated the following three biological properties that we believe will benefit patients with atherosclerosis:

*AGI-1067 blocks production of VCAM-1.* We believe that decreased VCAM-1 production will diminish atherosclerosis and restenosis.

*AGI-1067 is a potent antioxidant.* AGI-1067 protects LDL cholesterol from converting into a harmful inflammatory agent.

*AGI-1067 lowers LDL cholesterol.* LDL cholesterol lowering reduces the risk of developing atherosclerosis.

According to the American Heart Association, more than 12.6 million people in the United States have coronary artery disease, including approximately 1.1 million who have heart attacks every year. In order to make a definitive diagnosis in patients with suspected coronary artery disease, a specially trained cardiologist or radiologist performs a diagnostic procedure called angiography in which the cardiologist injects dye through an intravenous catheter to image the coronary arteries. Angiography can reveal coronary artery disease that may require an invasive procedure. Physicians perform this invasive procedure, called angioplasty, more than one million times annually worldwide. This procedure consists of placing a balloon-tipped catheter into the coronary artery and mechanically re-opening the blood vessel by expanding the balloon under very high

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pressure. In addition, cardiologists may opt to treat some of these coronary artery blockages by inserting a stent to keep the blood vessel open after the cardiologist removes the catheter.

Angioplasty does not cure coronary artery disease, nor does it treat the underlying chronic inflammation. In fact, angioplasty induces an inflammatory response that contributes to its failure in approximately 30 percent of patients who undergo the procedure. This process of re-narrowing, or post-angioplasty restenosis, is a major clinical problem that limits the effectiveness of the procedure. Restenosis following balloon angioplasty occurs due to local damage to the coronary artery. The development of stents and the ongoing research and development activities with respect to catheter improvement have not eradicated the problem of restenosis, but have introduced the new problem of in-stent restenosis which is particularly difficult to treat. In-stent restenosis occurs when the cells that surround the stent proliferate and fill the opening of the vessel. Drug coated stents are currently being tested in clinical trials and in limited trials have shown to be very effective in reducing restenosis in patients who can use these devices.

Our initial development targets are post-angioplasty restenosis and atherosclerosis. We believe that AGI-1067 may treat all areas of the coronary artery susceptible to atherosclerosis in a way that cannot be achieved with any existing therapy.

We have completed preclinical testing in multiple species to establish the therapeutic properties of AGI-1067. Our preclinical results indicated that, dosed orally, AGI-1067 blocked VCAM-1 production, blocked damage from oxidants and prevented atherosclerosis. In addition, AGI-1067 reduced LDL cholesterol comparably to and in combination with statins, which are widely used cholesterol-lowering drugs. In preclinical testing, AGI-1067 lowered bad cholesterol, increased good cholesterol and blocked atherosclerosis in a year-long preclinical model of progression of atherosclerosis.

Based upon our successful completion of preclinical testing, we studied AGI-1067 in seven Phase I clinical trials comprising more than 150 men and women, including healthy volunteers and patients up to the age of 85, to assess tolerability and potential for interaction with other drugs. In the course of these studies we gave AGI-1067 in combination with other drug classes commonly used in patients with atherosclerosis. In these seven clinical trials, six of which we conducted under the Investigational New Drug application for cholesterol lowering, some subjects reported mild nausea during the first few doses of AGI-1067, but the nausea abated while they continued to take the drug. Overall, the subjects tolerated AGI-1067 well, with no dose or use-limiting side effects. These clinical trial results, which showed that patients tolerated AGI-1067 well alone and in combination with other drugs, supported our progression to Phase II clinical trials.

In May 2001, we completed testing of our Phase II clinical trial, CART-1, that assessed in 305 patients the safety and effectiveness of AGI-1067 for the treatment of post-angioplasty restenosis. An analysis of the results indicated that six months after angioplasty, the blood vessels of patients who received AGI-1067 had greater luminal diameters of their coronary arteries than those who received placebo. This improvement showed a statistically significant dose response. At the highest dose of AGI-1067, the increase in the size of the target blood vessel was similar to that achieved with probucol, the active control drug in CART-1, which has been shown in previous clinical studies to reduce restenosis rates significantly following angioplasty without the use of a stent. CART-1 also suggested that AGI-1067 may actually reverse the progression of atherosclerosis. In November 2001 at the American Heart Association Scientific Sessions, CART-1 data were presented from a post-study intravascular ultrasound analysis of reference vessels (blood vessels of coronary arteries that were not targets of angioplasty procedures) which indicated lumen volumes increased for patients who received either of the top two doses of AGI-1067. In contrast, patients on placebo had decreased lumen volumes, consistent with the expected progression of atherosclerosis. In December 2001, we initiated a Phase IIb CART-2 clinical trial for AGI-1067. CART-2 is a 500-patient clinical trial, which examines the effect of 12 months of AGI-1067 therapy on restenosis and atherosclerosis following angioplasty. An additional six-month Phase IIb clinical trial, called DART-1, is being initiated to examine the effects of AGI-1067 on restenosis and atherosclerosis in Type 2 diabetics.

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***AGIX-4207***

Rheumatoid arthritis is a common auto-immune disease that affects joints and arterial blood vessels. According to the Arthritis Foundation, there are 2.1 million people with rheumatoid arthritis in the United States. Rheumatoid arthritis and related diseases cost the U.S. economy more than \$65 billion annually in direct and indirect costs. Approximately 70 percent of patients with rheumatoid arthritis are young and middle-aged women.

Physicians treat rheumatoid arthritis in a stepwise fashion, starting with the occasional to regular use of anti-inflammatory agents such as aspirin or ibuprofen, and proceeding to treatment with DMARDs, which can potentially be toxic. The newer DMARDs target the modulation of tumor necrosis factor (TNF), tissue repair and proliferation. The recent successful introduction of new drugs for rheumatoid arthritis has highlighted both the market potential and the size and scope of the unmet medical need of these patients. These drugs are partially effective and may cause serious side effects. AGIX-4207 is a selective modulator of TNF induced genes and is being tested as a medication that would be taken once a day. This selective nature of AGIX-4207 may decrease chronic inflammation in rheumatoid arthritis with fewer side effects. In March 2001, we commenced a Phase I clinical trial to assess the safety and tolerability of AGIX-4207 in healthy volunteers. In February 2002, we received results from the Phase I clinical trial demonstrating that AGIX-4207 is well tolerated over the single and multiple dose ranges studied. Adverse events were generally mild and not considered clinically significant.

Treatment of patients with rheumatoid arthritis progresses from pain relievers to increasingly toxic immunosuppressants, called disease modifiers. We will evaluate our v-protectant for the treatment of patients who are receiving moderate disease modifying therapy to determine whether AGIX-4207 will permit decreasing the use of toxic drugs while maintaining the patient's clinical status.

We have also developed an intravenously-dosed v-protectant drug candidate, AGIX-4207 I.V., to treat rheumatoid arthritis patients in whom the rapid attainment of target drug levels in the blood is desirable. These populations may include patients with flare-ups or exacerbations of the disease, patients who are intolerant of protein-based parenteral TNF inhibitors, hospitalized patients with rheumatoid arthritis who undergo elective or emergency surgical procedures and risks causing flare-ups, as well as patients who are unable to take oral medication. An exacerbation is a sudden worsening of the patient's arthritis or condition that usually requires hospitalization and intensive therapy. In October 2001, we began a Phase I clinical trial to assess the safety and tolerability of AGIX-4207 I.V. in healthy volunteers.

***AGI-1096***

Organ transplant rejection is caused when patients' immune systems recognize transplanted organs as foreign and, therefore, reject them. Acute rejection occurs soon after transplantation, while chronic rejection may take years. Recent industry sources report there are approximately 200,000 organ transplant recipients in the United States who are at risk of chronic transplant rejection. Chronic rejection is a major factor contributing to organ shortage.

Physicians treat these patients with powerful immunosuppressants to block all immune and inflammatory reactions that could cause solid organ transplant rejection. These therapies, however, may place patients at increased risk for infection. The vascular protection provided by our drug candidate may protect solid organs from rejection beyond the first year without increasing the risk of infection.

AGI-1096 is an anti-inflammatory agent designed to both diminish the organ transplant response to inflammation and directly protect the blood vessels to the transplanted organ through its v-protectant activity. AGI-1096 inhibits the expression of certain inflammatory proteins, including VCAM-1, in endothelial cells lining the inside surfaces of blood vessel walls. In February 2002, we commenced a Phase I clinical trial to assess the safety and tolerability of AGI-1096 in healthy volunteers.

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### ***AGI-Series for Respiratory Diseases***

According to the American Lung Association, approximately 24.7 million adults and children in the United States have been diagnosed with asthma in their lifetime. From 1980 to 1994, the prevalence of this disease increased by over 75%. Asthma morbidity and mortality continue to rise in spite of massive public health efforts. The American Lung Association reports that in 1999 the combined direct and indirect costs of asthma in the United States were approximately \$12.7 billion. Current therapies that target the underlying disease include corticosteroids and several classes of drugs that relieve symptoms but are not effective for chronic inflammation. None of these drugs, including inhaled corticosteroids, are particularly effective for treating exacerbations of asthma, which remain a major unmet medical problem. We believe that v-protectants may reduce the inflammation associated with chronic asthma and with the acute exacerbation of asthma, and may be useful in the treatment of up to 1.8 million patients annually who develop acute exacerbations of asthma and seek emergency room treatment in the United States.

We are evaluating classes of chemical compounds as potential treatments for asthma and other respiratory diseases. We will evaluate these components for regular treatment of chronic respiratory diseases or for exacerbations. We will test our compounds for delivery by the oral, intravenous or inhaled route of administration.

In June 2001, we entered into a worldwide exclusive license agreement with National Jewish Medical and Research Center to discover and develop novel therapeutics for the treatment of inflammation and asthma. We plan to use these new technologies to discover and develop additional drug candidates for the treatment of asthma.

### ***Diagnostic Assay Program***

Based on our v-protectant technology platform, we have designed a simple and proprietary blood test that measures a circulating blood marker for atherosclerosis. We plan to conduct tests on human blood samples to establish whether this new marker, called OXYKINE®, is an accurate and useful diagnostic tool. We believe OXYKINE® will allow physicians to determine whether a patient has active and progressive atherosclerosis and whether the disease is responding to medical therapy. We are not aware of any diagnostic tools that meet this profile.

## **Research Program**

We have built a robust research program using our demonstrated expertise in functional genomics, molecular biology, cell biology, physiology, pharmacology, biochemistry, and analytical and synthetic chemistry, bioengineering and medicine.

Our research program has four main objectives:

*To discover and develop v-protectants with enhanced potency and improved therapeutic properties.* We are synthesizing novel compounds and testing them in a variety of biochemical and cell-based assays to discover and develop new, small molecule v-protectants. We believe that these v-protectants will have improved therapeutic properties and applicability across a wide range of chronic inflammatory diseases. We have identified a novel series of highly potent v-protectants.

*To identify novel anti-inflammatory therapeutic targets utilizing functional genomics.* One part of our drug discovery platform is a set of techniques that connects our knowledge of genes, which code for proteins, to agents that modify gene activity. This collection of methods, called functional genomics, enables us to select targets efficiently. Our targets for therapy may be the gene, the protein, another substance in the body that links to the protein, or the agent that induces the change. For example, oxidants are agents that induce changes in gene activity. We believe our functional genomics program will enable us to identify novel genes and their protein products that are critical to the chronic inflammatory disease process. We plan to progress these genes and proteins into targets for novel classes of drugs.

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*To develop new classes of v-protectant drugs based on the novel therapeutic targets identified by our functional genomics program. We are identifying enzymes and other molecular targets that either control or are controlled by oxidant signals. We believe these discoveries will enable our chemists to synthesize the next generation of v- protectants. We intend to use these enzymes and other molecular targets for both internal efforts and as strategic collaboration assets.*

*To develop a second broad platform for the discovery and development of a new class of anti-inflammatory drug candidates. As a result of entering into the license agreement with National Jewish Medical and Research Center in June 2001, we plan to expand our research program in the future to include the discovery and development of new drug candidates through the exploitation of the licensed technology.*

### **Patents and Intellectual Property**

We have established a patent portfolio of owned and in-licensed patents that cover our lead compounds and their use. It is our goal to pursue both broad and specific patent protection in the key areas of our research and development both in the United States and internationally, and to identify value-added exclusive in-licensing opportunities.

#### ***V-Protectant Technology***

We have license agreements with Emory University and The Regents of the University of California covering aspects of our v-protectant technology. These agreements obligate us to make milestone payments upon attainment of agreed-upon goals and royalty payments on the sale of licensed products and technology. The licenses with Emory University and The Regents of the University of California also require us to be diligent in commercializing the licensed technologies within certain time periods.

Under our license agreement with Emory University, Emory University granted to us an exclusive license to make, use and sell methods and products covered by certain patents and patent applications owned by Emory University relating generally to the treatment and diagnosis of VCAM-1 related diseases. The license agreement requires us to make royalty payments to Emory University based on certain percentages of net revenue we derive from sales of products covered by the licensed patents or patent applications, and from sublicensing of the licensed patents or patent applications. The license agreement also requires us to make milestone payments to Emory University upon the occurrence of certain product development events. Milestone payments for AGI-1067 could total \$250,000 if all milestone objectives are met. We must indemnify Emory University for all claims and/or losses caused or contributed to by AtheroGenics arising out of our use of the license. We have procured commercial general liability insurance in specified amounts customary in the industry naming Emory University as an insured.

The Emory license agreement will terminate when all patent rights licensed under the agreement expire. Emory University may terminate the agreement if, after Emory gives notice to us, we fail to make a payment, we fail to render progress reports, we incur specified financial problems, we decide to no longer develop licensed products under the agreement, or we breach a material term of the agreement. We may terminate the agreement upon advance notice to Emory, or if Emory University violates certain material terms of the agreement.

Under our license agreement with The Regents of the University of California, we received a license to make, use and sell diagnostic and therapeutic methods and products using monoclonal antibodies in atherosclerosis and other diseases, which are claimed in applicable patent applications owned by The Regents of the University of California in the U.S. and Canada. We must make milestone payments to The Regents of the University of California upon occurrence of various product development events of up to \$45,000 for each therapeutic application and \$35,000 for each diagnostic application. In addition, we must pay to The Regents of the University of California a percentage of the net revenue we receive from the sale of products covered by the patents and patent applications and from our sublicensing the licensed patents and patent applications. The Regents of the University of California may terminate the agreement upon proper notice for violation of material terms of the agreement. The agreement expires in 2018, when the last patent covered by the license

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expires. We may terminate the agreement at any time upon prior notice to The Regents of the University of California. We must indemnify The Regents of the University of California for all losses and claims arising out of our use of the license. In addition, we have procured commercial liability insurance in specified amounts customary in the industry naming the University of California as an insured.

As part of our v-protectant technology patent portfolio, we also purchased U.S. Patent No. 5,262,439 under an agreement with Dr. Sampath Parthasarathy. We believe the cost of this agreement to us is immaterial.

***AGI-1067 Patent Portfolio***

Our patent coverage on AGI-1067 is based on patent filings that we own and patent filings exclusively licensed from Emory University. We own one issued patent, U.S. Patent No. 5,262,439, which expires in 2012, and related filings in Japan, Canada and Europe that generically cover the compound AGI-1067 as a member of a class of related compounds. We own another patent, U.S. Patent No. 6,147,250, that protects through 2018 the specific compound AGI-1067 and its use to treat VCAM-1 mediated diseases including, among others, atherosclerosis, post-angioplasty restenosis and coronary artery disease. We also own U.S. Patent No. 6,121,319, which covers the use of a class of compounds including AGI-1067 to treat VCAM-1 mediated diseases. Applications corresponding to U.S. Patent No. 6,147,250 and U.S. Patent No. 6,121,319 have also been filed in foreign patent offices. The patents that we have exclusively licensed from Emory University include the use of a substance that inhibits a class of oxidant signals to treat diseases mediated by VCAM-1.

***AGIX-4207 Patent Portfolio***

Our patent coverage on AGIX-4207 is based on patent filings that we own and patent filings exclusively licensed from Emory University. We own one U.S. patent application, and associated non-U.S. patent filings which describe AGIX-4207 and its use to treat rheumatoid arthritis, other inflammatory conditions and other disorders mediated by VCAM-1. Any patents issuing from this application will expire in 2018.

***AGI-1096 Patent Portfolio***

Our patent coverage on AGI-1096 is based on patent filings that we own and patent filings exclusively licensed from Emory University. We own one U.S. patent application, and associated non-U.S. patent filings which describe AGI-1096 and its use to treat disorders mediated by VCAM-1. Any patents issuing from the application will expire in 2018. We also own a provisional patent application claiming the use of AGI-1096 to treat solid organ transplant rejection.

***Other V-Protectant Compounds***

Certain patent applications in the United States and non-U.S. countries cover the use of a number of compounds identified in our research program to act as v-protectants, and specifically for use in treating cardiovascular and inflammatory disease. Some of these compounds are novel and some represent new uses for known compounds. In addition we have exclusively licensed patents from Emory University that cover the use of a class of compounds which act as v-protectants.

***MEKK Technology***

In June 2001, we entered into a worldwide exclusive license agreement with National Jewish Medical and Research Center. Under the agreement, National Jewish granted us an exclusive license under several of its U.S. and foreign patents and patent applications and related technical information to make, use and sell diagnostics and therapeutics for the treatment of human diseases, including inflammation and asthma. Under the terms of the agreement with National Jewish, we may grant sublicenses of our rights to others.

Under the agreement with National Jewish, we have assumed responsibility for all future costs associated with research and development of products developed from the licensed technology. We have also assumed responsibility for the costs of filing, prosecuting and maintaining the licensed patent rights. We granted

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National Jewish a warrant to purchase up to 40,000 shares of our common stock at an exercise price of \$6.00 per share, subject to a vesting period. Under the agreement, we paid an upfront payment in connection with the execution of the agreement and will pay milestone payments to National Jewish upon the achievement of certain clinical and regulatory milestones. Upfront and milestone payments could aggregate up to approximately \$800,000. If we fail to meet various performance milestones by certain dates, some or all of the licensed technology will revert to National Jewish. We must also pay a royalty to National Jewish on net sales of licensed products. If we sublicense the licensed technology, we must pay to National Jewish a percentage of the amounts paid to us by the sublicensee, which, if the sublicense agreement is executed within one year following the date of the license agreement with National Jewish, could be a material percentage of the sublicense fee.

Our patent position, like that of many pharmaceutical companies, is uncertain and involves complex legal and factual questions for which important legal principles are unresolved or unclear. We may not develop or obtain rights to products or processes that are patentable. Even if we do obtain patents, they may not adequately protect the technology we own or in-license. In addition, others may challenge, seek to invalidate, infringe or circumvent any patents we own or in-license, and rights we receive under those patents may not provide competitive advantages to us.

We may terminate the license agreement with National Jewish at any time upon at least 90 days prior written notice. If we terminate the agreement in this manner, all licensed patent rights and related technology revert to National Jewish. Either party to the agreement may also terminate it upon a material, uncured breach by the other, or upon the bankruptcy or insolvency of the other. We must indemnify National Jewish for all losses and claims arising out of our use of the license. We will procure commercial liability insurance in amounts customary in the industry as required by the agreement.

Our commercial success will depend in part on our ability to manufacture, use, sell and offer to sell our product candidates and proposed product candidates without infringing patents or other proprietary rights of others. We may not be aware of all patents or patent applications that may impact our ability to make, use or sell any of our product candidates or proposed product candidates. For example, U.S. patent applications do not publish until 18 months from their effective filing date. Further, we may not be aware of published or granted conflicting patent rights. Any conflicts resulting from patent applications and patents of others could significantly reduce the coverage of our patents and limit our ability to obtain meaningful patent protection. If others obtain patents with conflicting claims, we may be required to obtain licenses to these patents or to develop or obtain alternative technology. We may not be able to obtain any licenses or other rights to patents, technology or know-how necessary to conduct our business as described in this report. Any failure to obtain such licenses or other rights could delay or prevent us from developing or commercializing our product candidates and proposed product candidates, which could materially affect our business.

Litigation or patent interference proceedings may be necessary to enforce any of our patents or other proprietary rights, or to determine the scope and validity or enforceability of the proprietary rights of others. The defense and prosecution of patent and intellectual property claims are both costly and time consuming, even if the outcome is favorable to us. Any adverse outcome could subject us to significant liabilities, require us to license disputed rights from others, or require us to cease selling our future products.

***Trademarks***

The U.S. Patent and Trademark Office issued a Certificate of Registration for the trademark OXYKINE on April 10, 2001. The Patent and Trademark Office issued a Certificate of Registration for the trademark AATHEROGENICS and DESIGN on November 7, 2000 and issued one for the trademark AGI on September 19, 2000.

On January 30, 2002, Applied Genetics Incorporated Dermatics filed with the United States Patent and Trademark Office a petition to cancel the trademark AGI. Applied Genetics has not requested any monetary damages. We filed an answer to the petition on March 11, 2002, and intend to vigorously defend our registration in this matter.



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### **Exclusive License Agreement with Schering-Plough**

In October 1999, we entered into a worldwide exclusive license agreement with Schering-Plough. This agreement consisted of contracts with two Schering-Plough affiliates. Under the agreement we granted to Schering-Plough an exclusive license under our patents and know-how to make, use and sell AGI-1067 and other specified compounds for the treatment of restenosis, coronary artery disease and atherosclerosis. Schering-Plough paid us an initial nonrefundable licensing fee of \$5,000,000 upon signing the agreement and, pursuant to the terms of the agreement, had assumed responsibility for all costs going forward associated with the development, manufacturing and commercialization of products containing AGI-1067 and any other licensed compound.

In October 2001, we reacquired the rights to AGI-1067 and related compounds and terminated the exclusive license agreement between us and Schering-Plough. The reacquisition of these rights will permit us to expedite the clinical development process for AGI-1067. In addition, Schering-Plough returned all licensed technology to us and returned all materials related to that technology. With the termination of this license agreement, Schering-Plough will have no further rights to the technology or financial obligations to us.

As a result of the reacquisition of the rights and termination of the license agreement, we will now be responsible for all research and development costs associated with AGI-1067, along with the manufacture and commercialization of products that we develop containing AGI-1067. We currently plan to undertake internally the development and commercialization of AGI-1067, although we may decide to enter into future collaborative agreements with third parties for all or part of that development and commercialization.

### **Manufacturing**

We have entered into an arrangement with a third party manufacturer for the supply of AGI-1067 bulk drug substance and another third party manufacturer for the formulated drug product. The supplier of the bulk drug substance for AGI-1067 operates under current Good Manufacturing Practice guidelines using cost-effective and readily available materials and reliable processes. The starting material used in the manufacturing process of AGI-1067 is probucol, which was once widely used in North America as a cholesterol-lowering agent, but has since been withdrawn from the North American market due to lack of efficacy. Under the terms of our contract, our bulk drug supplier is committed to manufacture sufficient quantities to support development activities for the foreseeable future.

After manufacture, a third party supplier formulates AGI-1067 into the drug product under current Good Manufacturing Practice guidelines. We anticipate that this supplier will be able to provide sufficient formulated drug product to complete our ongoing and currently planned clinical trials.

We plan to establish manufacturing agreements with third parties that comply with Good Manufacturing Practice guidelines for bulk drug substance and oral or intravenous formulations of our other v-protectant product candidates, including AGIX-4207, AGIX-4207 I.V. and AGI-1096.

### **Sales and Marketing**

We plan to collaborate with large pharmaceutical companies to commercialize product candidates which are for patient or physician populations in broad markets. We believe that collaborating with large companies that have significant marketing and sales capabilities provides for optimal penetration into broad markets, particularly those areas that are highly competitive. In contrast, we plan to develop a sales force to commercialize the products targeted at appropriate patient and physician populations in narrow markets. By using our own sales and marketing organization, we believe we can retain a higher percentage of the profits generated from the sale of our products.

### **Competition**

Developments by others may render our product candidates obsolete or noncompetitive. We face intense competition from other companies for collaborative arrangements with pharmaceutical, biotechnology and medical device companies for establishing relationships with academic and research institutes and for licenses

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to proprietary technology. These competitors, either alone or in collaboration, may succeed in developing technologies or products that are more effective than ours.

We believe pharmaceutical, biotechnology and medical device companies, as well as academic and research institutions and government agencies, have drug discovery and development programs related to our named therapeutic areas of interest. Many of these companies and institutions, including Aventis Pharmaceuticals, Inc., Genentech, Inc., Johnson & Johnson, and Novartis AG, have targeted indications that overlap significantly with our targets and have substantially greater resources than we do. They may, therefore, succeed in commercializing products before we do that compete with us on the basis of efficacy, safety and price.

Our ability to compete is predicated on three related factors:

First, our scientists and their collaborators have pioneered the basic discoveries and research methodologies linking oxidant signals to vascular cell inflammation. These discoveries and research methodologies form the foundation for our proprietary drug discovery programs relating to chronic inflammation.

Second, our scientific expertise, coupled with our expertise in clinical drug development, has enabled us to be the first company to conduct clinical trials of an orally-administered, small molecule v-protectant. We believe that our recently completed Phase II clinical trial demonstrates that we are maintaining this important first-to-clinic competitive advantage.

Third, we believe our scientific, development and licensing expertise strongly positions us to acquire promising technologies and products discovered outside AtheroGenics.

**Governmental Regulation**

We plan to develop prescription-only drugs for the foreseeable future. The U.S. Food and Drug Administration is the regulatory agency that is charged with the protection of people in the United States who take prescription medicines. Every country has a regulatory body with a similar mandate. In addition, the European Union has vested centralized authority in the European Medicines Evaluation Agency and Committee on Proprietary Medicinal Products to standardize review and approval across member nations.

Regulatory agencies have established guidelines and regulations for the drug development process. This process involves several steps. First, the drug company must generate sufficient preclinical data to support initial human testing. In the United States, the drug company must submit an Investigational New Drug application prior to human testing. The Investigational New Drug application contains adequate data on product candidate chemistry, toxicology and metabolism and, where appropriate, animal research testing to support initial safety evaluation in humans. In addition, the drug company provides to the FDA a clinical plan, including proposed use and testing in subjects comprising healthy volunteers and patients.

Clinical trials for a new product candidate usually proceed through four phases:

Phase I clinical trials explore safety, blood levels, metabolism and the potential for interaction with other drugs. Phase I typically proceeds from healthy volunteers into patients with the target disease and comprises up to approximately 200 total subjects.

Phase II clinical trials establish a dose for future testing and marketing in an adequate number of patients with the target disease. The clinical trials may include hundreds of patients who have the target disease and who are receiving a range of background medications. In addition, Phase II clinical trials verify the mechanisms of action proposed preclinically.

Phase III clinical trials usually include two adequate and well controlled studies in the target population. For most chronic diseases, drug companies study a few thousand patients to assure a broadly applicable assessment of safety and efficacy. At the successful conclusion of Phase III, drug companies may submit a product license application, called a New Drug Application in the United States. Upon accepting the submission, the FDA or non-

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U.S. regulatory authorities review the file for completeness, accuracy and adherence to regulations. These authorities may use internal and external consultants and may convene an expert committee to advise on the safety, effectiveness and usefulness of the proposed new product candidate prior to final regulatory judgment. The final step to registration is approval of the package insert or label that defines what the drug company may promote to physicians who use the new drug.

Phase IV clinical trials support marketing of the drug for its approved indication. Phase IV clinical trials generate data to allow promotion of the new drug in comparison with other approved drugs and to support healthcare economics claims. In addition, every pharmaceutical company is responsible for post-marketing surveillance for safety in the marketplace.

We must meet regulatory standards prior to exposing subjects to any candidate drug product. We remain responsible for any of these development activities whether we perform them internally or contract them to a third party. The FDA may audit us or our third party contractors at any time to ascertain compliance with standards. The FDA may halt all ongoing work if it determines that we or our contractors have deviated significantly from these standards. These standards include:

Good Manufacturing Practices, which govern process chemistry, formulation, labeling and handling of a drug throughout its life cycle;

Good Laboratory Practices, which govern the use of a drug in animal studies to support establishment of safety or the disposition and metabolism of the administered drug and handling of human or other biological samples for drug assays; and

Good Clinical Practices, which govern the exposure of human subjects under our protocols. Good Clinical Practices set standards for the constitution and activities of institutional review boards that are charged with assuring that the appropriate person gives informed consent prior to study participation and protect patients whether they receive an experimental drug, an approved drug, or an inactive look-alike called a placebo.

Advertising is subject to FDA approval in the United States and national review elsewhere. In addition, state and local governments and other federal agencies may control marketing if the drug substance, formulation, package, intended use or disposal is subject to local regulation.

The FDA has expanded its expedited review process in recognition that certain severe or life-threatening diseases and disorders have only limited treatment options. Fast track designation expedites the development process but places greater responsibility on a drug company during Phase IV clinical trials. The drug company may request fast track designation for one or more indications at any time during the Investigational New Drug application process, and the FDA must respond within 60 days. Fast track designation allows the drug company to develop product candidates and to request an accelerated or priority review of the New Drug Application based on clinical effectiveness in a smaller number of patients. If the FDA accepts the submission as a priority review, the time for New Drug Application review and approval is reduced from one year to six months. We plan to request fast track designation as appropriate for internal drug development programs.

**Research and Development**

Our research and development expenses in 2001, 2000 and 1999 were \$16.9 million, \$12.8 million and \$9.0 million, respectively.

**Employees**

We currently have 79 full-time employees, including 62 in research and development. The employee group includes 24 employees with Ph.D.s, six with M.D.s and 14 with Masters degrees. We believe that our employee relations are good.

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**Advisory Boards**

We have established advisory boards to provide guidance and counsel on aspects of our business. These boards are convened once a year and individual members are contacted as required. Members of these boards provide input on product research and development strategy, education and publication plans. The names and members of these boards are as follows:

**Scientific Advisory Board:**

R. Wayne Alexander, M.D., Ph.D., <i>Chairman</i>	Professor and Chairman of the Department of Medicine, Emory University School of Medicine
Victor J. Dzau, M.D.	Hershey Professor of the Theory and Practice of Medicine and Chairman, Department of Medicine, Harvard Medical School and Physician in Chief and Director of Research, Brigham and Women's Hospital
Erwin W. Gelfand, M.D.	Chairman, Department of Pediatrics, National Jewish Medical and Research Center
David Harrison, M.D.	Director, Cardiology and Bernard Marcus Professor of Medicine, Emory University
Gary L. Johnson, Ph.D.	Professor, Department of Pharmacology, University of Colorado Health Science Center
Dennis Liotta, Ph.D.	Samuel Candler Dobbs Professor of Chemistry, Emory University School of Medicine
Robert M. Nerem, Ph.D.	Director, Georgia Tech/ Emory Center (GTEC) for the Engineering of Living Tissues and Director, Parker H. Petit Institute for Bioengineering and Bioscience at Georgia Institute of Technology
Robert D. Rosenberg, M.D., Ph.D.	Whitehead Professor of Biology, Massachusetts Institute of Technology and William B. Castle Professor of Medicine, Harvard Medical School

**Clinical Advisory Board:**

William Virgil Brown, M.D.	Chief of Medicine and Primary Care, Veterans Affairs Medical Center, Emory University School of Medicine
Harvey M. Golomb, M.D.	Professor and Chairman, Department of Medicine, and Director, Section of Hematology/ Oncology, The University of Chicago
Joseph L. Witzum, M.D.	Professor of Medicine and Endocrinology, University of California at San Diego

**Forward-Looking Statements and Risks Related to Our Company and Business**

The Private Securities Litigation Reform Act of 1995 provides a safe harbor for forward-looking statements made by or on behalf of AtheroGenics. AtheroGenics and its representatives may from time to time make written or verbal forward-looking statements, including statements contained in this report and our other filings with the Securities and Exchange Commission and in our reports to our shareholders. Generally, the words, believe, expect, intend, estimate, anticipate, will and similar expressions identify forward-looking statements. All statements which address operating performance, events or developments that we expect or anticipate will occur in the future, including projections of our future results of operations or of our financial condition, research, development and commercialization of our product candidates, and

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anticipated trends in our business, are forward-looking statements within the meaning of the Reform Act. The forward-looking statements are and will be based on our then current views and assumptions regarding future events and operating performance, and speak only as of their dates. We undertake no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

The following are some of the factors that could affect our financial performance or could cause actual results to differ materially from those expressed or implied in our forward-looking statements:

***If AGI-1067 fails in clinical trials, we may not be able to generate future revenues or become profitable.***

AGI-1067 is our lead compound. This compound could fail in clinical trials if we show it is ineffective or causes unacceptable side effects in the patients we treated. Failure in clinical trials for AGI-1067 would have a material adverse effect on our ability to generate revenue or become profitable.

***We have a history of operating losses, and we may not generate revenue or achieve profitability in the future.***

Our ability to generate revenue and achieve profitability depends on our ability, alone or with collaborators, to complete successfully the development of our product candidates, conduct preclinical tests in animals and clinical trials in human beings, obtain the necessary regulatory approvals, and manufacture and market the resulting drugs. We have experienced operating losses since we began operations in 1994. As of December 31, 2001, we had an accumulated deficit of approximately \$61.3 million. We expect to incur additional operating losses over the next several years and expect cumulative losses to increase substantially as our research and development, preclinical, clinical, manufacturing and marketing efforts expand. Except for an initial licensing fee and research and development revenue that Schering-Plough paid to us, we have had no significant revenue to date.

***If we do not successfully develop our other product candidates, we will have limited ability to generate revenue.***

All of our other programs are in early stages of development, and subject to the risks of failure inherent in developing drug products based on new technologies. We do not expect any of our potential product candidates to be commercially available until at least 2005. In addition, other than AGIX-4207, AGIX-4207 I.V. and AGI-1096, product candidates for which we are currently in Phase I clinical trials, our drug discovery efforts may not produce any other proprietary product candidates.

***We will not be able to commercialize our product candidates if we fail to demonstrate adequately their safety and efficacy.***

We cannot assure you that any product candidate we develop, alone or with others, will prove safe and effective in clinical trials and will meet all of the applicable regulatory requirements needed to receive regulatory approval. We will need to conduct significant research, preclinical testing and clinical trials before we can file product approval applications with the FDA and similar regulatory authorities in other countries. Preclinical testing and clinical trials are long, expensive and uncertain processes. We may spend several years completing our testing for any particular product candidate, and failure can occur at any stage.

The FDA or we may suspend our clinical trials at any time if either of us believes that we are exposing the subjects participating in these trials to unacceptable health risks. The FDA or institutional review boards at the medical institutions and healthcare facilities where we sponsor clinical trials may suspend any trial indefinitely if they find deficiencies in the conduct of these trials. We must conduct clinical trials in accordance with the FDA's Good Clinical Practices. The FDA and these institutional review boards have authority to oversee our clinical trials and the FDA may require large numbers of test subjects. In addition, we must manufacture the product candidates that we use in our clinical trials under the FDA's Good Manufacturing Practices.

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Even if we achieve positive results in early clinical trials, these results do not necessarily predict final results. A number of companies in the pharmaceutical industry have suffered significant setbacks in advanced clinical trials, even after achieving positive results in earlier trials. Negative or inconclusive results or adverse medical events during a clinical trial could cause the FDA or us to terminate a clinical trial or require that we repeat it.

Also, even if the FDA approves a New Drug Application for any of our product candidates, the resulting product may not be accepted in the marketplace. Physicians, patients, payors or the medical community in general may be unwilling to accept, utilize or recommend any of our products. In addition, after approval and use in an increasing number of patients, our products could show side effect profiles that limit their usefulness or require their withdrawal although the drugs did not show the side effect profile in Phase I through Phase III clinical trials.

***We may experience delays in our clinical trials that could adversely affect our financial results and our commercial prospects.***

We do not know whether planned clinical trials will begin on time or whether we will complete any of our clinical trials on schedule or at all. Product development costs to us and our collaborators will increase if we have delays in testing or approvals or if we need to perform more or larger clinical trials than planned. Significant delays may adversely affect our financial results and the commercial prospects for our products, and delay our ability to become profitable. We typically rely on third party clinical investigators at medical institutions and healthcare facilities to conduct our clinical trials and, as a result, we may face additional delaying factors outside our control.

***Because we cannot predict whether or when we will obtain regulatory approval to commercialize our product candidates, we cannot predict the timing of any future revenue from these product candidates.***

We cannot commercialize any of our product candidates, including AGI-1067, AGIX-4207, AGIX-4207 I.V. and AGI-1096, until the appropriate regulatory authorities have reviewed and approved the applications for the product candidates. We cannot assure you that the regulatory agencies will complete their review processes in a timely manner or that we will obtain regulatory approval for any product candidate we or our collaborators develop. Satisfaction of regulatory requirements typically takes many years, is dependent upon the type, complexity and novelty of the product and requires the expenditure of substantial resources. Regulatory approval processes outside the United States include all of the risks associated with the FDA approval process. In addition, we may experience delays or rejections based upon additional government regulation from future legislation or administrative action or changes in FDA policy during the period of product development, clinical trials and FDA regulatory review.

***If we do not comply with applicable regulatory requirements in the manufacture and distribution of our products, we may incur penalties that may inhibit our ability to commercialize our products and adversely affect our revenue.***

Our failure to comply with applicable FDA or other regulatory requirements including manufacturing, quality control, labeling, safety surveillance, promoting, and reporting may result in criminal prosecution, civil penalties, recall or seizure of our products, total or partial suspension of production or an injunction, as well as other regulatory action against our potential products or us. Discovery of previously unknown problems with a product, supplier, manufacturer or facility may result in restrictions on the sale of our products, including a withdrawal of such products from the market.

***We will incur additional expenses for research and development of AGI-1067 as a result of the termination of our license agreement with Schering-Plough, which could adversely impact our development of other product candidates and could materially adversely affect our financial liquidity.***

Because we have terminated our exclusive license agreement with Schering-Plough, we will now be responsible for all of the costs related to the continuing research and development of AGI-1067, which were

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previously reimbursed by Schering-Plough under the terms of the license agreement. These additional research and development expenses will result in the reduction of our available cash, which could adversely impact our ability to commence or continue other research and development projects, or could cause delays in the development of AGI-1067 or other projects.

***Our failure to protect adequately or enforce our intellectual property rights or secure rights to third party patents could materially adversely affect our proprietary position in the marketplace or prevent the commercialization of our products.***

Our patent position, like that of many pharmaceutical companies, is uncertain and involves complex legal and factual questions for which important legal principles are unresolved. In addition, we may not be able to obtain patent rights on products, treatment methods or manufacturing processes that we may develop or to which we may obtain license or other rights. Even if we do obtain patents, they may not adequately protect the technology we own or in-license. In addition, others may challenge, seek to invalidate, infringe or circumvent any patents we own or in-license, and rights we receive under those patents may not provide competitive advantages to us.

Our commercial success will depend in part on our ability to manufacture, use, sell and offer to sell our product candidates and proposed product candidates without infringing patents or other proprietary rights of others. We may not be aware of all patents or patent applications that may impact our ability to make, use or sell any of our product candidates or proposed product candidates. For example, U.S. patent applications do not publish until 18 months from their priority date. Further, we may not be aware of published or granted conflicting patent rights. Any conflicts resulting from patent applications and patents of others could significantly reduce the coverage of our patents and limit our ability to obtain meaningful patent protection. If others obtain patents with conflicting claims, we may need to obtain licenses to these patents or to develop or obtain alternative technology. We may not be able to obtain any licenses or other rights to patents, technology or know-how necessary to conduct our business as described in this report. Any failure to obtain such licenses could delay or prevent us from developing or commercializing our drug candidates or proposed product candidates, which would adversely affect our business.

Litigation or patent interference proceedings may be necessary to enforce any of our patents or other proprietary rights, or to determine the scope and validity or enforceability of the proprietary rights of others. The defense and prosecution of patent and intellectual property claims are both costly and time consuming, even if the outcome is favorable to us. Any adverse outcome could subject us to significant liabilities, require us to license disputed rights from others, or require us to cease selling our future products.

Our commercial success will also depend on our ability to manufacture, use, sell and offer to sell our product candidates and proposed product candidates without breaching our agreements with our patent licensees. We have obtained exclusive licenses to technologies from Emory University, covering aspects of our v-protectant technology; The Regents of the University of California, covering aspects of our diagnostic technology; and National Jewish, covering aspects of our new MEKK technology platform. Our exclusive license with Emory University requires us to take steps to commercialize the licensed technology in a timely manner. If we fail to meet these obligations, Emory University can convert our exclusive license to a non-exclusive license, can grant others non-exclusive rights in the licensed technology or can require us to sublicense aspects of the licensed technology. Our license agreement with The Regents of the University of California also includes a requirement that we develop the licensed technology within certain time limits. If we fail to meet these time limits, they can terminate our license. Further, The Regents of University of California are primarily responsible for patent prosecution of the technology we license from them, and we are required to reimburse them for the costs they incur in performing these activities. As a result, we do not have the ability to control these activities. Our license agreement with National Jewish requires us to develop the licensed technology in a timely manner. If we fail to meet these obligations, some or all of the licensed technology may revert to National Jewish.

We also rely upon trade secrets, proprietary know-how and technological advances which we seek to protect through agreements with our collaborators, employees and consultants. These persons and entities

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could breach our agreements, for which we may not have adequate remedies. In addition, others could become aware of our trade secrets or proprietary know-how through independent discovery or otherwise.

***If our competitors develop and market anti-inflammatory products that are more effective, have fewer side effects or are less expensive than our current or future product candidates, we may have limited commercial opportunities.***

Our competitors include large pharmaceutical and medical device companies and more established biotechnology companies. These competitors have significant resources and expertise in research and development, manufacturing, testing, obtaining regulatory approvals and marketing. Potential competitors also include academic institutions, government agencies, and other public and private research organizations that conduct research, seek patent protection and establish collaborative arrangements for research, development, manufacturing and commercialization. It is possible that any of these competitors could develop technologies or products that would render our technologies or product candidates obsolete or non-competitive, which could adversely affect our revenue potential.

***Third parties' failure to synthesize and manufacture our product candidates to our specifications could delay our clinical trials or hinder our commercialization prospects.***

We currently have no manufacturing facilities to synthesize or manufacture our product candidates, nor do we intend to develop these capabilities in the near future. Our reliance on third parties for these services exposes us to several risks that could delay our clinical trials or hinder our commercialization prospects. These risks include the following:

*A finding that a third party did not comply with applicable governmental regulations.* Manufacturers of pharmaceutical products are subject to continual review and periodic inspections by regulatory agencies. Failure of one of our third party manufacturers to comply with applicable regulatory requirements, whether or not related to our product candidates, could result in sanctions against our potential products, including recall or seizure, total or partial suspension of production or injunction.

*A failure to synthesize and manufacture our product candidates in accordance with our product specifications.* For example, a starting material used in the manufacturing process of AGI-1067 is probucol, which physicians previously prescribed as a cholesterol-lowering agent but which its manufacturer withdrew from the market for efficacy reasons. The occurrence of a rare side effect with chronic dosing of probucol requires that we maintain a very low maximal amount of probucol in the manufacture of AGI-1067.

*A failure to deliver product candidates in sufficient quantities or in a timely manner.* Any failure by our third party manufacturers to supply our requirements for clinical trial materials or supply these materials in a timely manner could jeopardize the scheduled initiation or completion of these clinical trials and could have a material adverse effect on our ability to generate revenue.

In addition, our continued dependence on third parties for the synthesis and manufacture of our future products may subject us to costs outside of our control, which could adversely affect our future profitability and our ability to commercialize products on a timely and competitive basis.

***If we are unable to create sales, marketing and distribution capabilities or enter into agreements with third parties to perform these functions, we will not be able to commercialize our future product candidates.***

We currently have no sales, marketing or distribution capabilities. Therefore, in order to commercialize our product candidates, we must either develop our own sales, marketing and distribution capabilities or collaborate with a third party to perform these functions. We have no experience in developing, training or managing a sales force and will incur substantial additional expenses in doing so. The cost of establishing and maintaining a sales force may exceed its cost effectiveness. In addition, we will compete with many companies



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that currently have extensive and well-funded marketing and sales operations. Our marketing and sales efforts may be unable to compete successfully against these companies.

To the extent we seek sales, marketing and distribution alliances for our future products, we face risks including the following:

we may not be able to find collaborators, enter into alliances on favorable terms or enter into alliances that will be commercially successful;

any collaborator might, at its discretion, limit the amount of resources and time it devotes to marketing our products; and

any collaborator may terminate its agreement with us and abandon our products at any time for any reason, regardless of the terms of the agreement.

***Our failure to attract, retain and motivate skilled personnel and cultivate key academic collaborations could materially adversely affect our research and development efforts.***

We are a small company with 79 full-time employees. If we are unable to continue to attract, retain and motivate highly qualified management and scientific personnel and to develop and maintain important relationships with leading academic institutions and scientists, we may not be able to achieve our research and development objectives. Competition for personnel and academic collaborations is intense. Loss of the services of any of our key scientific personnel and, in particular, Dr. Russell M. Medford, our President and Chief Executive Officer, could adversely affect progress of our research and development programs. Dr. Medford is the only employee with whom we have an employment agreement.

***If we need additional financing and cannot obtain it, we may not be able to develop or market our products.***

We may encounter increased costs due to unanticipated changes in our product development or commercialization plans. If these costs exceed our available funds, we will need to seek additional financing. If additional funds are not available, we may need to delay clinical studies, curtail operations or obtain funds through collaborative arrangements that may require us to relinquish rights to certain of our products or potential markets.

***Our failure to obtain an adequate level of reimbursement or acceptable prices for our products could diminish our revenues.***

Our ability to commercialize our future products successfully, alone or with collaborators, will depend in part on the extent to which reimbursement for the products will be available from:

government and health administration authorities;

private health insurers; and

other third party payors.

Government and other third party payors increasingly are attempting to contain healthcare costs by limiting both coverage and the level of reimbursement for new drugs. Third party private health insurance coverage may not be available to patients for any of our future products.

The continuing efforts of government and other third party payors to contain or reduce the costs of healthcare through various means may limit our commercial opportunity. For example, in some countries other than the United States, pricing and profitability of prescription pharmaceuticals are subject to government control. In the United States, we expect proposals to implement similar government control to continue. In addition, increasing emphasis on managed care in the United States will continue to put pressure on the pricing of pharmaceutical products. Cost control initiatives could decrease the price that we or any potential collaborators could receive for any of our future products and could adversely affect our profitability.

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*If plaintiffs bring product liability lawsuits against us, we may incur substantial financial loss or may be unable to obtain future product liability insurance at reasonable prices, if at all, either of which could diminish our ability to commercialize our future products.*

The testing and marketing of medicinal products entail an inherent risk of product liability. Clinical trial subjects, consumers, healthcare providers, or pharmaceutical companies or others selling our future products could bring product liability claims against us. We cannot assure you that we will be able to acquire or maintain insurance coverage at a reasonable cost or in sufficient amounts to protect us.

*Our quarterly operating results may fluctuate, causing volatility in our stock price.*

Our product candidates are now in research and various stages of development or clinical trials. Accordingly, we do not receive any revenues from sales of these product candidates. Our results of operations historically have fluctuated on a quarterly basis, which we expect to continue. Our results of operations at any given time will be based primarily on the following factors:

the status of development of our various product candidates;

whether we enter into collaboration agreements and the timing and accounting treatment of payments, if any, to us under those agreements;

whether and when we achieve specified development or commercialization milestones; and

the addition or termination of research programs or funding support.

We believe that quarterly comparisons of our financial results are not necessarily meaningful and should not be relied upon as an indication of future performance. These fluctuating results may cause the price of our stock to fluctuate, perhaps substantially.

**Item 2. *Properties***

Our scientific and administration facility encompasses approximately 27,000 square feet in Alpharetta, Georgia. We lease our facility pursuant to a long-term lease agreement that expires in 2009 and our aggregate commitment under this long-term, non-cancelable lease is approximately \$8 million. This lease may be extended at our option to 2019.

In November 2001, we leased a facility in Norcross, Georgia encompassing approximately 5,800 square feet. We lease this laboratory facility pursuant to a long-term lease agreement that expires in 2004 and our aggregate commitment under this long-term, non-cancelable lease is approximately \$346,000. We have the option to renew this lease under mutually agreeable terms.

**Item 3. *Legal Proceedings***

We are not currently a party to any legal proceedings.

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

None.

**Table of Contents****PART II****Item 5. Market for Registrant's Common Equity and Related Shareholder Matters****Common Stock Information**

Our common stock has been traded on the Nasdaq National Market under the symbol AGIX since August 9, 2000. Prior to that time, there was no public market for our common stock. The following table sets forth the range of high and low closing sale prices for our common stock as reported on the Nasdaq National Market during fiscal 2001 and the third and fourth quarters of fiscal 2000.

	<b>Common Stock</b>	
	<b>High</b>	<b>Low</b>
<b>Year ended December 31, 2000</b>		
Third quarter (commencing August 9, 2000)	\$ 10.75	\$ 8.00
Fourth quarter	8.94	4.63
<b>Year ended December 31, 2001</b>		
First quarter	7.13	5.25
Second quarter	7.25	4.53
Third quarter	6.76	3.95
Fourth quarter	6.10	2.71

As of March 1, 2002, there were approximately 3,600 holders of our common stock. This number includes beneficial owners of our common stock whose shares are held in the names of various dealers, clearing agencies, banks, brokers and other fiduciaries.

**Dividend Policy**

We have never declared or paid any dividends on our capital stock. We currently intend to retain all of our future earnings, if any, to finance our operations and do not anticipate paying any cash dividends on our capital stock in the foreseeable future.

**Changes in Securities and Use of Proceeds**

The Securities and Exchange Commission declared our Registration Statement on Form S-1 (File No. 333-31140) effective August 8, 2000. The net proceeds from the sale of the 6.9 million shares of common stock registered pursuant to the Registration Statement (including the exercise of the underwriters' over-allotment option) were \$49.4 million after deducting underwriting discounts of \$3.9 million and offering expenses of \$1.9 million.

We have used and expect to continue to use the proceeds from our initial public offering for research and development activities, including clinical trials, process development and manufacturing support, and for general corporate purposes, including working capital. A portion of the proceeds may be used to acquire or invest in complementary businesses, products or technologies. As of December 31, 2001, the proceeds have been applied toward:

purchases of fixed assets and leasehold improvements, \$1.6 million;

operating activities, \$12.8 million; and

investments in highly liquid, interest bearing, investment grade securities, \$35.0 million.

**Table of Contents****Item 6. Selected Financial Data**

The selected financial data set forth below should be read in conjunction with our financial statements and the related notes and Management's Discussion and Analysis of Financial Condition and Results of Operations, included in this Form 10-K. The historical results are not necessarily indicative of the operating results to be expected in the future.

	Year Ended December 31,				
	2001	2000	1999	1998	1997
<b>Statement of Operations Data:</b>					
Revenues:					
License fees	\$ 1,111,111	\$ 3,333,333	\$ 555,556	\$	\$
Research and development	2,398,429	4,826,370	791,653		
Total revenues	3,509,540	8,159,703	1,347,209		
Operating expenses:					
Research and development*	16,884,027	12,815,788	9,041,345	8,954,904	4,656,478
General and administrative*	3,979,813	3,035,559	2,593,017	1,573,807	988,230
Amortization of deferred stock compensation	2,652,031	7,972,728	85,480		
Total operating expenses	23,515,871	23,824,075	11,719,842	10,528,711	5,644,708
Operating loss	(20,006,331)	(15,664,372)	(10,372,633)	(10,528,711)	(5,644,708)
Net interest income (expense)	2,366,748	1,714,850	(60,617)	(205,130)	485,392
Net loss	\$(17,639,583)	\$(13,949,522)	\$(10,433,250)	\$(10,733,841)	\$(5,159,316)
Basic and diluted net loss per share	\$ (0.68)	\$ (1.30)	\$ (4.27)	\$ (4.45)	\$ (2.25)
Shares used in computing basic and diluted net loss per share	26,010,347	10,747,773	2,443,237	2,409,948	2,292,966

\* Exclusive of amounts recorded as amortization of deferred stock compensation:

Research and development	\$ 940,053	\$ 1,856,932	\$ 23,649	\$	\$
General and administrative	\$ 1,711,978	\$ 6,115,796	\$ 61,831	\$	\$

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The following table contains a summary of our balance sheet data for the five years ending December 31, 2001.

	December 31,				
	2001	2000	1999	1998	1997
<b>Balance Sheet Data:</b>					
Cash and cash equivalents	\$ 28,682,050	\$ 26,463,070	\$ 13,409,450	\$ 3,686,423	\$ 6,925,364
Short-term investments	29,757,945	27,518,169			
Working capital (deficiency)	55,056,263	52,422,951	9,651,239	(4,259,366)	6,108,938
Total assets	62,255,278	57,598,951	15,717,214	5,341,816	7,612,796
Long-term obligations, less current portion		84,907	61,854	163,262	281,636
Deferred stock compensation	(2,975,314)	(5,930,880)	(1,809,680)		
Accumulated deficit	(61,277,987)	(43,638,404)	(29,688,882)	(19,255,632)	(8,521,791)
Total shareholders equity (deficit)	58,294,812	54,271,686	(29,288,600)	(18,973,881)	(8,240,444)

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

*The following discussion should be read in conjunction with our financial statements and related notes included in this Form 10-K.*

**Overview**

Since our operations began in 1994, we have focused on the discovery, development and commercialization of novel drugs for the treatment of chronic inflammatory diseases, such as atherosclerosis, rheumatoid arthritis and asthma. Based on our proprietary vascular protectant, or v-protectant, technology platform, we have advanced three drug candidates into development, AGI-1067, AGIX-4207 and AGI-1096, and are progressing on a number of other preclinical programs.

To date, we have devoted substantially all of our resources to research and development. We have not derived any commercial revenues from product sales and, excluding the effect of certain license fees of a non-recurring nature received in connection with entering into an exclusive license agreement, expect to incur significant losses in most years prior to deriving any such product revenue.

We have incurred significant losses since we began operations in 1994 and, as of December 31, 2001, had an accumulated deficit of \$61.3 million. We cannot assure you whether or when we will become profitable. We expect to continue to incur significant operating losses over the next several years as we continue to incur increasing research and development costs. We expect that losses will fluctuate from quarter to quarter and that such fluctuations may be substantial. Our ability to achieve profitability depends upon our ability, alone or with others, to complete the successful development of our product candidates, to obtain required regulatory clearances, and to manufacture and market our future products.

In October 1999, we entered into an exclusive licensing agreement with Schering-Plough Corporation covering our lead compound, AGI-1067. Under terms of the agreement, Schering-Plough obtained exclusive worldwide rights to AGI-1067 and related compounds. Schering-Plough was responsible for all costs of development and commercialization and paid us an initial licensing fee. In October 2001, we reacquired all rights to AGI-1067 and related compounds and terminated the license agreement. As a result, Schering-Plough returned all licensed technology and all materials related to that technology.

In June 2001, we entered into a worldwide exclusive license agreement with National Jewish Medical and Research Center of Denver, Colorado to discover and develop novel therapeutics based on MEK kinases, enzymes that participate in a broad range of cellular activities, and related technology for the treatment of inflammation. Other licensed technology focuses on the application of several naturally occurring substances in the development of a potential treatment for asthma. We expect these new technologies to provide a second broad platform for the discovery and development of a new class of anti-inflammatory drug candidates.



**Table of Contents****Critical Accounting Policies**

We have identified the following policies below as critical to our business operations and the understanding of our results of operations. The impact and any associated risks related to these policies on our business operations is discussed throughout Management's Discussion and Analysis of Financial Condition and Results of Operations.

***Revenue Recognition***

License fees, which are nonrefundable, are recognized when the related license agreements specify that no further efforts or obligations are required of AtheroGenics. AtheroGenics had committed to perform certain research and development activities as part of a license agreement, which has been terminated; accordingly, the upfront license payment was amortized over the anticipated time period to conduct such activities. Revenues under research and development arrangements were recognized as the research and development activities were performed pursuant to the terms of the related agreements. These revenues were billed quarterly and the related payments were not refundable. Revenues that had not been invoiced were reflected as unbilled receivables in the accompanying balance sheets.

***Stock-Based Compensation***

AtheroGenics has elected to follow Accounting Principles Board Opinion ( APB ) No. 25, *Accounting for Stock Issued to Employees* ( APB 25 ), in accounting for its stock-based employee compensation plans, rather than the alternative fair value accounting method provided for under SFAS No. 123, *Accounting for Stock-Based Compensation* ( SFAS 123 ), as SFAS 123 requires the use of option-valuation models that were not developed for use in valuing employee stock options. AtheroGenics accounts for transactions in which services are received in exchange for equity instruments based on the fair value of such services received from non-employees, in accordance with SFAS 123 and Emerging Issues Task Force (EITF) Issue No. 96-18, *Accounting for Equity Instruments that are Issued to Other than Employees for Acquiring, or in Conjunction with Selling, Goods or Services*.

**Results of Operations*****Comparison of Years Ended December 31, 2001 and 2000******Revenues***

Total revenues were \$3.5 million in 2001, compared to \$8.2 million in 2000. License fees of \$1.1 million and \$3.3 million during 2001 and 2000, respectively, were attributable to an exclusive license agreement signed in October 1999. These amounts represent the earned portion of the \$5.0 million initial license fee, which was amortized over 18 months. Amortization of the license fee was completed in April 2001. Research and development revenues related to the license agreement were \$2.4 million in 2001 and \$4.8 million in 2000. The lower research and development revenues reflect reduced activities due to the completion of the CART-1 Phase II clinical trial for AGI-1067. There will be no further revenues from this license agreement which was terminated in October 2001.

***Expenses***

***Research and Development.*** Research and development expenses, excluding amortization of deferred stock compensation, were \$16.9 million in 2001, compared to \$12.8 million in 2000. The increase of \$4.1 million, or 32%, is primarily due to increased preclinical and clinical studies including the Phase I studies for AGIX-4207 and AGIX-4207 I.V., compounds being developed for the treatment of rheumatoid arthritis, and AGI-1096, a compound being developed for the treatment of solid organ transplant rejection.

***General and Administrative.*** General and administrative expenses, excluding amortization of deferred stock compensation, were \$4.0 million in 2001, compared to \$3.0 million in 2000. The increase of \$1.0 million, or 33%, is primarily due to costs related to operating as a public company including legal fees and investor relations activities.

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*Amortization of Deferred Stock Compensation.* During 2001, we recorded non-cash deferred stock compensation of approximately \$1.1 million for warrants granted in connection with a licensing agreement with National Jewish and options granted for the addition of new members to our Scientific Advisory Board. The fair value of the warrants and options was determined by using the Black Scholes model. These amounts are included as a reduction of shareholders' equity and are being amortized over the vesting periods of individual warrants and options, generally five years, using the graded vesting method. The fair value of the options and warrants is re-measured at each measurement date. Amortization of deferred stock compensation was \$2.7 million in 2001, of which \$940,053 was attributable to research and development expenses and \$1.7 million was attributable to general and administrative expenses. In 2000, amortization of deferred stock compensation was \$8.0 million, of which \$1.9 million was attributable to research and development expenses and \$6.1 million was attributable to general and administrative expenses.

*Net Interest Income*

Net interest income was \$2.4 million in 2001, compared to \$1.7 million in 2000. This increase is primarily due to an increased level of investments with funds received from our initial public offering in August 2000 and our private placement financing in June 2001, offset by lower interest rates earned on invested funds.

*Income Taxes*

As of December 31, 2001, we had net operating loss carryforwards and research and development credit carryforwards of \$50.4 million and \$1.5 million, respectively, available to offset future regular and alternative taxable income. The net operating loss carryforwards and the research and development credit carryforwards will expire between 2010 and 2021. The maximum annual use of the net operating loss carryforwards is limited in situations where changes occur in our stock ownership. Because of our lack of earnings history, the resulting deferred tax assets have been fully offset by a valuation allowance. The utilization of the loss and credit carryforwards to reduce future income taxes will depend on our ability to generate sufficient taxable income prior to the expiration of the net operating loss carryforwards and research and development credit carryforwards. We have not yet completed full analysis of Internal Revenue Code Section 382 limitations on the cumulative net operating loss carryforward. However, the annual limitations are not expected to prevent utilization of the net operating loss carryforward due to significant increases in value indicated by the successive issuances of our stock. If a change in ownership has occurred, there will be an annual limitation; however, this limitation is not expected to result in a loss of the deferred tax benefit.

***Comparison of Years Ended December 31, 2000 and 1999***

*Revenues*

Total revenues were \$8.2 million for the twelve months ended December 31, 2000, compared to \$1.3 million in 1999. Revenues of \$3.3 million and \$555,556 in 2000 and 1999, respectively, were attributable to licensing fees from the exclusive license agreement signed in October 1999 with Schering-Plough. This amount represents the earned portion of the \$5.0 million initial licensing fee that was amortized over 18 months. Research and development revenues from our development activities on AGI-1067 were \$4.8 million and \$791,653 in 2000 and 1999, respectively.

*Expenses*

*Research and Development.* Research and development expenses, excluding amortization of deferred stock compensation, were \$12.8 million for the twelve months ended December 31, 2000, compared to \$9.0 million for the twelve months ended December 31, 1999. The increase of \$3.8 million, or 42%, reflects the continued expansion of our internal research and development capabilities, preclinical costs related to AGIX-4207, a novel compound being developed for the treatment of rheumatoid arthritis, and other product development programs.

*General and Administrative.* General and administrative expenses, excluding amortization of deferred stock compensation, were \$3.0 million for the twelve months ended December 31, 2000, compared to



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\$2.6 million for the twelve months ended December 31, 1999. The increase of \$442,542, or 17%, was primarily due to increases in facility costs, personnel costs in administration departments and professional fees.

*Amortization of Deferred Stock Compensation.* For the twelve months ended December 31, 2000, we recorded non-cash deferred stock compensation of approximately \$12.1 million for options granted with exercise prices below the deemed fair value for financial reporting purposes of our common stock on their respective grant dates. This deferred stock compensation is being amortized using the graded vesting method. Amortization of deferred stock compensation was \$8.0 million for the twelve months ended December 31, 2000, of which \$1.9 million was attributable to research and development expenses and \$6.1 million was attributable to general and administrative expenses. There was \$85,480 of amortization of deferred stock compensation for the twelve months ended December 31, 1999.

### *Net Interest Income*

Net interest income was \$1.7 million for the twelve months ended December 31, 2000 as compared to net interest expense of \$60,617 for the twelve months ended December 31, 1999. The increase in net interest income was due to an increased level of invested funds from the initial public offering proceeds, as well as the elimination of interest expense related to a bridge loan, which was converted to preferred stock in April 1999.

### *Income Taxes*

As of December 31, 2000, we had net operating loss carryforwards and research and development credit carryforwards of \$35.6 million and \$1.2 million, respectively, available to offset future regular and alternative taxable income.

## **Liquidity and Capital Resources**

Since inception, we have financed our operations primarily through private placements of preferred stock and our initial public offering of 6.9 million shares of our common stock that raised net proceeds of \$49.4 million. In June 2001, we completed a private placement of 3.6 million shares of our common stock that raised net proceeds of \$18.8 million. At December 31, 2001, we had cash, cash equivalents and short-term investments of \$58.4 million, compared with \$54.0 million at December 31, 2000. Working capital at December 31, 2001 was \$55.1 million, compared to \$52.4 million at December 31, 2000. The increase in cash, cash equivalents, short-term investments and working capital is primarily due to the funds received from the private placement of our common stock in June 2001.

Net cash used in operating activities was \$12.8 million in 2001, compared to \$8.8 million in 2000. The increase in the use of cash in operating activities is principally due to the funding of net losses, excluding non-cash charges. We expect an increase in net cash used in operating activities as a result of the termination of our license agreement with Schering-Plough.

Net cash used in investing activities was \$3.8 million in 2001, compared to \$28.2 million used in investing activities in 2000. Net cash used in investing activities during 2001 consisted primarily of net purchases of available-for-sale securities, and purchases of equipment and leasehold improvements. Net cash used in investing activities during 2000 consisted primarily of the purchases of short-term investments and purchases of equipment and leasehold improvements.

Net cash provided by financing activities was \$18.8 million in 2001, compared to \$50.1 million provided by financing activities in 2000. Net cash provided by financing activities in 2001 consisted primarily of \$18.8 million received from the private placement of our common stock in June 2001. Net cash provided by financing activities in 2000 consisted primarily of proceeds from our initial public offering in August 2000, and the exercise of preferred stock warrants and common stock options. We have entered into a non-formula revolving line of credit with Silicon Valley Bank in the amount \$5.0 million to be used for working capital requirements. Borrowings under this line of credit bear interest at the bank's prime rate plus 1.50% floating rate and will become due 30 months from closing. In addition, we have entered into an equipment financing facility with Silicon Valley Bank in the amount of \$2.5 million to be used to finance existing and new

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equipment purchases. Borrowings under the financing facility bear interest to be fixed at the time of each advance and each advance will mature in 33 months from the date of the advance.

Based upon the current status of our product development and commercialization plans, we believe that our existing cash and cash equivalents, along with our line of credit and equipment financing facility with Silicon Valley Bank, will be adequate to satisfy our capital needs for at least the next 12 months. However, our actual capital requirements will depend on many factors, including:

- the status of product development;
- the time and cost involved in conducting clinical trials and obtaining regulatory approvals;
- filing, prosecuting and enforcing patent and other intellectual property claims;
- competing technological and market developments; and
- our ability to market and distribute our future products and establish new licensing agreements.

**Recently Issued Accounting Standards**

In July 2001, the Financial Accounting Standards Board ( FASB ) issued SFAS Nos. 141, *Business Combinations* and 142, *Accounting for Goodwill and Other Intangibles* ( SFAS 141 and SFAS 142 ). SFAS 141 requires that the purchase method of accounting be used for all business combinations initiated after June 30, 2001, and provides new criteria for determining whether an acquired intangible asset should be recognized separately from goodwill. SFAS 142 requires that goodwill and intangible assets with indefinite useful lives no longer be amortized, but instead tested for impairment at least annually. Intangible assets that have finite lives will continue to be amortized over their useful lives. We do not expect the adoption of SFAS 141 and SFAS 142 to have an impact on our financial statements.

In October 2001, FASB issued SFAS No. 144, *Accounting for the Impairment or Disposal of Long-lived Assets*, ( SFAS 144 ) which is applicable to financial statements issued for fiscal years beginning after December 15, 2001. SFAS 144 establishes a new method of accounting and reporting for the impairment of long-lived assets other than goodwill and intangible assets. The statement provides a single accounting model for long-lived assets to be disposed of and changes the criteria required to classify an asset as held-for-sale. We do not expect the adoption of SFAS 144 to have a material impact on our financial statements.

**Item 7A. Quantitative and Qualitative Disclosures on Market Risk**

The primary objective of our investment activities is to preserve principal while at the same time maximizing the income we receive from our investments without significantly increasing risk. Some of the securities that we invest in may have market risk. This means that a change in prevailing interest rates may cause the fair value of the principal amount of the investment to fluctuate. For example, if we hold a security that was issued with a fixed interest rate at the then-prevailing rate and the prevailing interest rate later rises, the fair value of the principal amount of our investment will probably decline. To minimize this risk in the future, we intend to continue to maintain our portfolio of cash equivalents and short-term investments in a variety of securities, including commercial paper, money market funds, and government and non-government debt securities. The average duration of all of our investments has generally been less than one year. Due to the short-term nature of these investments, we believe we have no material exposure to interest rate risk arising from our investments.

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**Item 8. *Financial Statements and Supplementary Data***

**ATHEROGENICS, INC.**

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**REPORT OF INDEPENDENT AUDITORS**

The Board of Directors and Shareholders

AtheroGenics, Inc.

We have audited the accompanying balance sheets of AtheroGenics, Inc. as of December 31, 2001 and 2000, and the related statements of operations, shareholders' equity (deficit) and cash flows for each of the three years in the period ended December 31, 2001. These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these financial statements based on our audits.

We conducted our audits in accordance with 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 financial statements referred to above present fairly, in all material respects, the financial position of AtheroGenics, Inc. at December 31, 2001 and 2000, and the results of its operations and its cash flows for each of the three years in the period ended December 31, 2001, in conformity with accounting principles generally accepted in the United States.

/S/ ERNST & YOUNG LLP

Atlanta, Georgia  
February 8, 2002

**Table of Contents****ATHEROGENICS, INC.****BALANCE SHEETS**

	December 31,	
	2001	2000
<b>ASSETS</b>		
Current assets:		
Cash and cash equivalents	\$ 28,682,050	\$ 26,463,070
Short-term investments	29,757,945	27,518,169
Accounts receivable		1,138,244
Prepaid expenses, note receivable and other current assets	576,734	545,826
	<u>59,016,729</u>	<u>55,665,309</u>
Equipment and leasehold improvements:		
Laboratory equipment	1,861,221	1,352,692
Leasehold improvements	1,420,579	966,869
Computer and office equipment	968,329	476,276
Construction in progress	309,384	131,185
	<u>4,559,513</u>	<u>2,927,022</u>
Less accumulated depreciation and amortization	(1,644,001)	(1,152,028)
	<u>2,915,512</u>	<u>1,774,994</u>
Long-term notes receivable	323,037	158,648
	<u>323,037</u>	<u>158,648</u>
<b>Total assets</b>	<b>\$ 62,255,278</b>	<b>\$ 57,598,951</b>
<b>LIABILITIES AND SHAREHOLDERS EQUITY</b>		
Current liabilities:		
Accounts payable	\$ 1,121,550	\$ 504,991
Accrued research and development costs	1,307,435	342,210
Accrued compensation	902,571	640,975
Accrued liabilities	541,809	517,312
Current portion of capitalized lease obligation	87,101	125,759
Deferred revenues		1,111,111
	<u>3,960,466</u>	<u>3,242,358</u>
Long-term portion of capitalized lease obligations		84,907
Shareholders' equity		
Preferred stock, no par value:		
Authorized 5,000,000 shares		
Common stock, no par value:		
Authorized 100,000,000 shares; issued and outstanding 27,834,773 and 23,909,295 shares at December 31, 2001 and 2000, respectively	121,723,102	103,608,655
Warrants	771,713	225,713
Deferred stock compensation	(2,975,314)	(5,930,880)
Accumulated deficit	(61,277,987)	(43,638,404)
Accumulated other comprehensive income	53,298	6,602
	<u>58,294,812</u>	<u>54,271,686</u>
<b>Total shareholders' equity</b>	<b>58,294,812</b>	<b>54,271,686</b>

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Total liabilities and shareholders' equity	<u>\$ 62,255,278</u>	<u>\$ 57,598,951</u>
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The accompanying notes are an integral part of these financial statements.

**Table of Contents****ATHEROGENICS, INC.****STATEMENTS OF OPERATIONS**

	Year Ended December 31,		
	2001	2000	1999
<b>Revenues:</b>			
License fees	\$ 1,111,111	\$ 3,333,333	\$ 555,556
Research and development	2,398,429	4,826,370	791,653
<b>Total revenues</b>	<b>3,509,540</b>	<b>8,159,703</b>	<b>1,347,209</b>
<b>Operating expenses:</b>			
Research and development*	16,884,027	12,815,788	9,041,345
General and administrative*	3,979,813	3,035,559	2,593,017
Amortization of deferred stock compensation	2,652,031	7,972,728	85,480
<b>Total operating expenses</b>	<b>23,515,871</b>	<b>23,824,075</b>	<b>11,719,842</b>
Operating loss	(20,006,331)	(15,664,372)	(10,372,633)
Net interest income (expense)	2,366,748	1,714,850	(60,617)
Net loss	\$(17,639,583)	\$(13,949,522)	\$(10,433,250)
Net loss per share basic and diluted	\$ (0.68)	\$ (1.30)	\$ (4.27)
Weighted average shares outstanding basic and diluted	26,010,347	10,747,773	2,443,237

\* Exclusive of amounts recorded as amortization of deferred stock compensation:

Research and development	\$ 940,053	\$ 1,856,932	\$ 23,649
General and administrative	\$ 1,711,978	\$ 6,115,796	\$ 61,831

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

**Table of Contents****ATHEROGENICS, INC.****STATEMENTS OF SHAREHOLDERS EQUITY (DEFICIT)**

	Common Stock		Warrants	Deferred Stock Compensation	Accumulated Deficit	Accumulated Other Comprehensive Income	Total Shareholders Equity (Deficit)
	Shares	Amount					
<b>Balance at January 1, 1999</b>	2,410,375	\$ 281,751	\$	\$	\$(19,255,632)	\$	\$(18,973,881)
Issuance of stock for exercise of stock options at \$.10 to \$.30 per share	126,168	33,051					33,051
Deferred stock compensation related to stock option grants		1,895,160		(1,895,160)			
Amortization of deferred stock compensation				85,480			85,480
Net loss					(10,433,250)		(10,433,250)
<b>Balance at December 31, 1999</b>	2,536,543	2,209,962		(1,809,680)	(29,688,882)		(29,288,600)
Issuance of stock for exercise of stock options at \$.30 to \$.38 per share	602,650	185,788					185,788
Issuance of stock for services	11,000	85,438					85,438
Issuance of common stock, net of issuance cost of \$5,770,749	6,900,000	49,429,251					49,429,251
Deferred stock compensation related to stock option grants		12,093,928		(12,093,928)			
Amortization of deferred stock compensation				7,972,728			7,972,728
Preferred stock conversion	13,859,102	39,604,288					39,604,288
Preferred stock warrant conversion			225,713				225,713
Net loss					(13,949,522)		(13,949,522)
Unrealized gain on available-for-sale securities						6,602	6,602
Comprehensive loss							(13,942,920)
<b>Balance at December 31, 2000</b>	23,909,295	103,608,655	225,713	(5,930,880)	(43,638,404)	6,602	54,271,686
Issuance of stock for exercise of stock options at \$.30 to \$.38 per share	335,478	108,764					108,764
Issuance of stock for services	5,000	29,778					29,778
Issuance of common stock, net of issuance cost of \$1,788,310	3,585,000	18,825,440					18,825,440
Deferred stock compensation for issuance of stock options and warrants related to a technology license agreement		546,200	546,000	(1,092,200)			
Amortization of deferred stock compensation				2,652,031			2,652,031
Adjustment to deferred stock compensation for forfeited stock options		(1,395,735)		1,395,735			
Net loss					(17,639,583)		(17,639,583)
Unrealized gain on available-for-sale securities						46,696	46,696



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Comprehensive loss							(17,592,887)
<b>Balance at December 31, 2001</b>	<b>27,834,773</b>	<b>\$ 121,723,102</b>	<b>\$ 771,713</b>	<b>\$ (2,975,314)</b>	<b>\$ (61,277,987)</b>	<b>\$ 53,298</b>	<b>\$ 58,294,812</b>

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

**Table of Contents****ATHEROGENICS, INC.****STATEMENTS OF CASH FLOWS**

	Year Ended December 31,		
	2001	2000	1999
<b>Operating activities</b>			
Net loss	\$(17,639,583)	\$(13,949,522)	\$(10,433,250)
Adjustments to reconcile net loss to net cash used in operating activities:			
Depreciation and amortization	491,973	420,192	279,823
Amortization of deferred stock compensation	2,652,031	7,972,728	85,480
Amortization of debt discount			235,750
Stock issued for services	29,778	85,438	49,998
Preferred stock issued for interest			271,071
Changes in operating assets and liabilities:			
Accounts receivable	1,138,244	(346,591)	(791,653)
Prepaid expenses, note receivable and other assets	(195,297)	(422,996)	977,544
Accounts payable	616,559	(174,151)	(770,411)
Accrued liabilities	1,251,318	974,897	(1,028,422)
Deferred revenues	(1,111,111)	(3,333,333)	4,444,444
Net cash used in operating activities	(12,766,088)	(8,773,338)	(6,679,626)
<b>Investing activities</b>			
Purchases of equipment and leasehold improvements	(1,632,491)	(738,053)	(1,115,085)
Purchases of short-term investments	(2,193,080)	(27,511,567)	
Net cash used in investing activities	(3,825,571)	(28,249,620)	(1,115,085)
<b>Financing activities</b>			
Payments on capital lease	(123,565)	(175,096)	(198,236)
Proceeds from the issuance of preferred stock, Series C			17,535,923
Proceeds from the issuance and exercise of preferred stock warrants		636,635	
Proceeds from the issuance of common stock	18,825,440	49,429,251	
Proceeds from the exercise of common stock options	108,764	185,788	33,051
Proceeds from bridge loan financing, net of warrants			150,000
Net cash provided by financing activities	18,810,639	50,076,578	17,520,738
Increase in cash and cash equivalents	2,218,980	13,053,620	9,726,027
Cash and cash equivalents at beginning of period	26,463,070	13,409,450	3,683,423
Cash and cash equivalents at end of period	\$ 28,682,050	\$ 26,463,070	\$ 13,409,450
<b>Supplemental disclosures of cash flow information</b>			
Interest paid	\$ 21,536	\$ 30,524	\$ 28,317
Equipment purchases under capitalized lease obligation		222,500	

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Conversion of bridge loan and accrued interest to preferred stock		6,421,071
Warrants issued for extension of bridge loan		235,750
Option and warrants issued for technology license agreement	1,092,200	

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

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**NOTES TO FINANCIAL STATEMENTS**

**1. Description of Business and Significant Accounting Policies**

***Description of Business***

AtheroGenics, Inc. ( AtheroGenics ) was incorporated on November 23, 1993 (date of inception) in the State of Georgia to focus on the discovery, development and commercialization of novel therapeutics for the treatment of chronic inflammatory diseases, such as heart disease (atherosclerosis), rheumatoid arthritis and asthma.

***Use of Estimates***

The preparation of the financial statements in conformity with generally accepted accounting principles requires management to make estimates and assumptions that affect the amounts reported in the financial statements and accompanying notes. Actual results could differ from those estimates.

***Cash and Cash Equivalents***

AtheroGenics considers all highly liquid investments with a maturity of three months or less when purchased to be cash equivalents. AtheroGenics cash equivalents consist primarily of money market accounts, commercial paper, government agency notes and corporate notes on deposit with several financial institutions and the carrying amounts reported in the balance sheets approximate their fair value.

***Short-Term Investments***

Management determines the appropriate classification of debt securities at the time of purchase and reevaluates such designation as of each balance sheet date. These investments are accounted for in accordance with Statement of Financial Accounting Standards ( SFAS ) No. 115, *Accounting for Certain Investments in Debt and Equity Securities* ( SFAS 115 ). AtheroGenics has classified all investments as available-for-sale. Available-for-sale securities are carried at fair value, with the unrealized gains and losses, net of tax, reported in a separate component of shareholders equity. Realized gains and losses are included in investment income and are determined on a specific identification basis.

Short-term investments consist of certificates of deposit, commercial paper, government agency notes and corporate notes that will mature between four and twelve months.

***Fair Value of Financial Instruments and Concentration of Credit Risk***

Financial instruments that subject AtheroGenics to concentration of credit risk consist primarily of cash, cash equivalents and short-term investments. These assets are maintained by reputable third party financial institution custodians. The carrying values reported in the balance sheets for cash, cash equivalents and short-term investments approximate their fair values.

***Accounts Receivable***

Accounts receivable consisted of accounts receivable and unbilled receivables from Schering-Plough. As of December 31, 2001, there were no accounts receivable or unbilled receivables as a result of the termination of the license agreement with Schering-Plough (see Note 2 License Agreement ).

***Equipment and Leasehold Improvements***

Equipment and leasehold improvements are stated at cost. Depreciation of computer and lab equipment is computed using the straight-line method over the estimated useful lives of three and five years, respectively. Amortization of leasehold improvements is recorded over the shorter of: (a) the estimated useful lives of the related assets; or (b) the lease term.



**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)*****Revenue Recognition***

License fees, which are nonrefundable, are recognized when the related license agreements specify that no further efforts or obligations are required of AtheroGenics. AtheroGenics had committed to perform certain research and development activities as part of the license agreement with Schering-Plough; accordingly, the upfront license payment was amortized over the anticipated time period to conduct such activities. Revenues under research and development arrangements were recognized as the research and development activities were performed pursuant to the terms of the related agreements (see Note 2 License Agreement ). These revenues were billed quarterly and the related payments were not refundable. Revenues that had not been invoiced were reflected as unbilled receivables as described in the accounts receivable note above.

***Research and Development and Patent Costs***

Research and development costs, including all clinical trial expenses and expenditures related to obtaining patents, are charged to expense when incurred.

***Stock-Based Compensation***

AtheroGenics has elected to follow Accounting Principles Board Opinion ( APB ) No. 25, *Accounting for Stock Issued to Employees* ( APB 25 ), in accounting for its stock-based employee compensation plans, rather than the alternative fair value accounting method provided for under SFAS No. 123, *Accounting for Stock-Based Compensation* ( SFAS 123 ), as SFAS 123 requires the use of option-valuation models that were not developed for use in valuing employee stock options. AtheroGenics accounts for transactions in which services are received in exchange for equity instruments based on the fair value of such services received from non-employees, in accordance with SFAS 123 and Emerging Issues Task Force (EITF) Issue No. 96-18, *Accounting for Equity Instruments that are Issued to Other than Employees for Acquiring, or in Conjunction with Selling, Goods or Services*.

***Income Taxes***

The liability method is used in accounting for income taxes; deferred income tax assets and liabilities are determined based on differences between financial reporting and tax bases of assets and liabilities and are measured using the enacted tax rates and laws that are expected to be in effect when the differences are anticipated to reverse.

***Comprehensive Income***

AtheroGenics computes comprehensive income in accordance with SFAS No. 130, *Reporting Comprehensive Income* ( SFAS 130 ). SFAS 130 establishes standards for the reporting and display of comprehensive income and its components in the financial statements. Comprehensive income, as defined, includes all changes in equity during a period from non-owner sources, such as unrealized gains and losses on available-for-sale securities. Comprehensive loss was \$17,592,887 and \$13,942,920 for the years ended December 31, 2001 and 2000, respectively, as AtheroGenics reported an accumulated unrealized gain from available-for-sale securities of \$53,298. Comprehensive loss was equal to net loss for the year ended December 31, 1999.

***Recently Issued Accounting Standards***

In July 2001, the Financial Accounting Standards Board ( FASB ) issued SFAS Nos. 141, *Business Combinations* and 142, *Accounting for Goodwill and Other Intangibles* ( SFAS 141 and SFAS 142 ). SFAS 141 requires that the purchase method of accounting be used for all business combinations initiated after June 30, 2001, and provides new criteria for determining whether an acquired intangible asset should be recognized separately from goodwill. SFAS 142 requires that goodwill and intangible assets with indefinite useful lives no longer be amortized, but instead tested for impairment at least annually. Intangible assets that

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

have finite lives will continue to be amortized over their useful lives. AtheroGenics does not expect the adoption of SFAS 141 and SFAS 142 to have an impact on its financial statements.

In October 2001, FASB issued SFAS No. 144, *Accounting for the Impairment or Disposal of Long-lived Assets*, ( SFAS 144 ) which is applicable to financial statements issued for fiscal years beginning after December 15, 2001. SFAS 144 establishes a new method of accounting and reporting for the impairment of long-lived assets other than goodwill and intangible assets. The statement provides a single accounting model for long-lived assets to be disposed of and changes the criteria required to classify an asset as held-for-sale. AtheroGenics does not expect the adoption of SFAS 144 to have a material impact on its financial statements.

***Reclassifications***

Certain prior year balances have been reclassified to conform with the current year presentation. These reclassifications had no effect on previously reported net loss or shareholders' equity (deficit).

**2. License Agreement**

On October 22, 1999, AtheroGenics entered into an exclusive license agreement (the Agreement ), consisting of contracts with each of Schering Corporation and Schering-Plough Ltd. (collectively, Schering-Plough ). The Agreement provided for license fees and milestone payments to be made by Schering-Plough to AtheroGenics.

In November 1999, under the terms of the Agreement, AtheroGenics received a \$5,000,000 non-refundable license fee for the exclusive worldwide license to patent rights and licensor know-how held by AtheroGenics. AtheroGenics amortized the fee over 18 months, which represents the period AtheroGenics conducted development activities pursuant to the Agreement. Under the Agreement, AtheroGenics granted to Schering-Plough rights to develop, make, have made, import, export, use, distribute, market, promote, offer for sale and sell AGI-1067, AtheroGenics' lead product candidate, and specified compounds.

To the extent that AtheroGenics performed additional research and development at Schering-Plough's request, Schering-Plough paid AtheroGenics for such research and development. AtheroGenics recognized research and development revenues of \$2,398,429, \$4,826,370 and \$791,653 during 2001, 2000 and 1999, respectively, in relation to such requests.

In October 2001, AtheroGenics reacquired all rights to AGI-1067 and related compounds and terminated the license agreement. As a result, Schering-Plough returned all licensed technology and all materials related to that technology.

**3. Net Loss Per Share**

Net loss per share has been computed according to SFAS No. 128, *Earnings Per Share* ( SFAS 128 ), which requires disclosure of basic and diluted earnings per share. Basic earnings per share excludes any dilutive effects of options, shares subject to repurchase, warrants, and convertible securities. Diluted earnings per share includes the impact of potentially dilutive securities. AtheroGenics' potentially dilutive securities are antidilutive and, therefore, are not included in the computation of weighted average shares used in computing diluted loss per share. Following the guidance given by the Securities and Exchange Commission, common stock and preferred stock that has been issued or granted for nominal consideration prior to the anticipated effective date of the initial public offering must be included in the calculation of basic and diluted net loss per common share as if these shares had been outstanding for all periods presented. AtheroGenics has not issued or granted shares for nominal consideration since its formation.

Basic and diluted pro forma net loss per share for 2000 and 1999 was computed by dividing the net loss by the weighted average number of shares of common stock outstanding plus the conversion of all outstanding convertible preferred stock into common stock, which occurred upon consummation of AtheroGenics' initial

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

public offering, retroactive to the date of issuance. This information is included in the following table for comparative purposes.

The following is a reconciliation of the numerator and denominator of basic and diluted net loss per share amounts:

	Year Ended December 31,		
	2001	2000	1999
<b>Basic and diluted:</b>			
Net loss	\$(17,639,583)	\$(13,949,522)	\$(10,433,250)
Weighted average shares used in computing basic and diluted net loss per share	26,010,347	10,747,773	2,443,237
Basic and diluted net loss per share	\$ (0.68)	\$ (1.30)	\$ (4.27)
<b>Pro forma basic and diluted:</b>			
Shares used above		10,747,773	2,443,237
Pro forma adjustment to reflect weighted average effect of assumed conversion of preferred stock		8,595,672	10,268,792
Pro forma weighted average shares of common stock outstanding		19,343,445	12,712,029
Basic and diluted pro forma loss per share		\$ (0.72)	\$ (0.82)

During all periods presented, AtheroGenics had securities outstanding which could potentially dilute basic earnings per share in the future, but were excluded from the computation of diluted net loss per share, as their effect would have been antidilutive. These outstanding securities consist of the following at the dates indicated:

	Year Ended December 31,		
	2001	2000	1999
Convertible (at one share for one share) preferred stock			13,643,837
Options	3,360,660	2,858,175	1,785,325
Warrants	350,290	250,290	467,503
Total	3,710,950	3,108,465	15,896,665
Weighted average exercise price of options	\$ 2.99	\$ 1.49	\$ 0.28
Weighted average exercise price of warrants	\$ 4.14	\$ 3.40	\$ 3.21



**4. Redeemable Convertible Preferred Stock**

AtheroGenics sold shares of Series A, Series B, Series B-1 and Series C Redeemable Convertible Preferred Stock at various offerings between 1994 and 2000. These shares were convertible into common stock, at a conversion rate of one-to-one, upon certain qualifying conditions which included the completion of an underwritten public offering of AtheroGenics common stock.

On August 8, 2000, AtheroGenics Registration Statement on Form S-1 was declared effective by the Securities and Exchange Commission. Immediately prior to the closing of AtheroGenics initial public offering on August 14, 2000, all of the outstanding shares of convertible preferred stock automatically

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

converted into 13,859,102 shares of common stock. Immediately following the automatic conversion of preferred stock, an amended and restated certificate of incorporation was filed. Under the amended and restated certificate of incorporation, AtheroGenics is authorized to issue 100,000,000 shares of common stock and 5,000,000 shares of preferred stock.

The following table summarizes AtheroGenics' outstanding shares and value of Series A, Series B, Series B-1 and Series C Redeemable Convertible Preferred Stock at December 31, 2000 and 1999:

	December 31, 2000		December 31, 1999	
	Shares Outstanding	Carrying Value	Shares Outstanding	Carrying Value
Series A		\$	1,000,000	\$ 1,000,000
Series B			4,586,815	13,704,499
Series B-1				
Series C			8,057,022	24,006,992
	<u>—</u>	<u>—</u>	<u>13,643,837</u>	<u>\$38,711,491</u>

**5. Common Stock**

On August 14, 2000, AtheroGenics completed an initial public offering of 6,900,000 shares of common stock (including the exercise of underwriters' over-allotment option) that raised gross proceeds of approximately \$55,200,000 and net proceeds of approximately \$49,400,000.

On June 19, 2001, AtheroGenics completed a private placement of 3,585,000 shares of common stock that raised gross proceeds of approximately \$20,600,000 and net proceeds of approximately \$18,800,000. Both new and existing investors participated in the transaction.

On November 9, 2001, AtheroGenics' Board of Directors adopted a Shareholder Rights Plan declaring a dividend distribution of one common stock purchase right on each outstanding share of its common stock. Until the rights become exercisable, the rights will trade automatically with the common stock of AtheroGenics and separate rights certificates will not be issued. Under the rights plan, each right consists of an initial right and subsequent rights. Initial rights will be exercisable only if a person or group acquires 15% or more of AtheroGenics' common stock, whether through open market or private purchases or consummation of a tender or exchange offer. Any shareholders who owned, as of November 9, 2001, in excess of 15% of AtheroGenics' common stock will be permitted to acquire up to an aggregate of 20% of AtheroGenics' outstanding common stock without triggering the rights plan. If, following the exercise of initial rights, a person or group again acquires 15% or more of AtheroGenics' common stock, or a person or group who had previously acquired 15% or more of AtheroGenics' common stock acquires an additional 10% or more of the common stock, the subsequent rights become exercisable. Each right will initially entitle shareholders to buy eight shares of common stock at an exercise price equal to 20% of the then current market value of the common stock, calculated and adjusted according to the terms of the rights plan. The number of shares that can be purchased upon exercise will increase as the number of shares held by the bidder increases.

If AtheroGenics is acquired in a merger or other business combination, each right will entitle its holder to purchase, at the right's then-current exercise price, a number of the acquiring company's shares equal value to those obtainable if the rights were exercisable in AtheroGenics' stock.

The rights are intended to enable all shareholders to realize the long-term value of their investment in AtheroGenics. They will not prevent a takeover, but should encourage anyone seeking to acquire AtheroGenics to negotiate with the Board prior to attempting a takeover. The Board of Directors may redeem any non-exercisable rights at any time at its option at a redemption price of \$.0001 per right. The rights plan expires at the close of business on November 8, 2011.

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)****6. Stock Options and Warrants**

During 1995, AtheroGenics established a stock option plan (the 1995 Plan) which, as amended, provides that options to purchase AtheroGenics common stock may be granted to employees, directors, consultants or contractors with exercise prices not less than 75% of the fair values of the shares on the dates of grant.

The 1995 Plan, as amended, authorizes the grant of options for up to 1,264,084 shares of AtheroGenics common stock, and as of December 31, 2001, AtheroGenics had reserved 267,800 shares of common stock for future issuance under the 1995 Plan. Options granted under the 1995 Plan vest over periods ranging from the date of grant to five years from that date. A summary of stock option activity under the 1995 Plan follows:

	Number of Shares	Price Range	Weighted Average Price
Outstanding at January 1, 1999	457,000	\$.10-.30	\$.20
Exercised	(24,000)	.10	.10
Canceled	(17,800)	.30	.30
Outstanding at December 31, 1999	415,200	.10-.30	.20
Exercised	(165,200)	.10-.30	.29
Outstanding at December 31, 2000 and 2001	250,000	.10-.30	.14

The following table summarizes information concerning outstanding and exercisable options under the 1995 Plan as of December 31, 2001:

Exercise Price	Options Outstanding			Options Exercisable	
	Number Outstanding	Weighted Average Remaining Years	Weighted Average Exercise Price	Number Exercisable	Weighted Average Exercise Price
\$.10	200,000	3.62	\$.10	200,000	\$.10
.30	50,000	4.61	.30	49,000	.30
	250,000	3.82	.14	249,000	.14

Effective July 30, 1997, AtheroGenics established an equity ownership plan (the 1997 Plan) whereby options to purchase AtheroGenics common stock may be granted to employees, directors, consultants or contractors with exercise prices not less than the fair values of the shares on the dates of grant. The 1997 Plan authorizes the grant of options for up to 1,474,416 shares of AtheroGenics common stock. On January 28, 2000, AtheroGenics Board of Directors authorized an additional 2,250,000 shares to be issued under the 1997 Plan. As of December 31, 2001, AtheroGenics had reserved 2,831,975 shares of common stock for issuance under the 1997 Plan. The 1997 Plan allows for grants of non-qualified options, incentive stock options and shares of restricted stock. Non-qualified options granted under the 1997 Plan may vest immediately for non-employees, but vest over a four-year period for employees. Incentive stock options generally vest over four years. The majority of the stock options granted under the 1997 Plan are incentive stock options.

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

A summary of stock option activity under the 1997 Plan follows:

	Number of Shares	Price Range	Weighted Average Price
Outstanding at January 1, 1999	778,875	\$ .30	\$ .30
Granted	748,000	.30-.31	.30
Exercised	(102,168)	.30	.30
Canceled	(54,582)	.30	.30
Outstanding at December 31, 1999	1,370,125	.30-.31	.30
Granted	1,797,850	.38-9.88	2.28
Exercised	(448,450)	.30-9.88	.50
Canceled	(111,350)	.30-8.25	.67
Outstanding at December 31, 2000	2,608,175	.30-9.88	1.62
Granted	791,450	4.37-6.85	6.01
Exercised	(340,478)	.30-6.56	.41
Canceled	(228,487)	.30-8.25	2.31
Outstanding at December 31, 2001	2,830,660	.30-9.88	2.93

The following table summarizes information concerning currently outstanding and exercisable options granted under the 1997 Plan as of December 31, 2001.

Options Outstanding				Options Exercisable	
Exercise Price	Number Outstanding	Weighted Average Remaining Years	Weighted Average Exercise Price	Number Exercisable	Weighted Average Exercise Price
\$ .30-.31	644,595	7.16	\$ .30	424,385	\$ .30
.38-4.36	913,915	8.08	.38	481,413	.38
4.37-6.03	637,150	9.28	5.38	169,503	5.50
6.04-9.88	635,000	9.26	6.83	107,883	7.77
	2,830,660	8.41	2.93	1,183,184	1.76

Effective April 18, 2001, AtheroGenics established an equity ownership plan (the 2001 Plan) whereby options to purchase AtheroGenics common stock may be granted to employees, directors, consultants or contractors with exercise prices not less than the fair values of the shares on the dates of grant. The 2001 Plan authorizes the grant of options for up to 2,000,000 shares of AtheroGenics common stock. As of December 31, 2001, AtheroGenics had reserved 2,000,000 shares of common stock for issuance under the 2001 Plan. The terms of the 2001 Plan are substantially similar to the terms of the 1997 Plan.

A summary of stock option activity under the 2001 Plan follows:

Number of	Weighted Average
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	<u>Shares</u>	<u>Price Range</u>	<u>Price</u>
Outstanding at January 1, 2001		\$	\$
Granted	280,000	6.05	6.05
Outstanding at December 31, 2001	280,000	6.05	6.05

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

The following table summarizes information concerning currently outstanding and exercisable options granted under the 2001 Plan as of December 31, 2001.

Options Outstanding				Options Exercisable	
Exercise Price	Number Outstanding	Weighted Average Remaining Years	Weighted Average Exercise Price	Number Exercisable	Weighted Average Exercise Price
\$6.05	280,000	10.0	\$6.05	—	\$

During 2000 and 1999, in connection with the grant of certain options to employees, AtheroGenics recorded non-cash deferred stock compensation of \$12,093,928 and \$1,895,160, respectively, representing the difference between the exercise price and the deemed fair value of AtheroGenics common stock on the dates these stock options were granted. Deferred stock compensation is included as a reduction of shareholders equity and is being amortized to expense using the graded vesting method. The graded vesting method provides for vesting of each portion of the overall award over its respective vesting period, and results in higher vesting in earlier years than straight-line vesting. During 2001, 2000 and 1999, AtheroGenics recorded amortization of deferred stock compensation of \$2,316,141, \$7,972,728 and \$85,480, respectively.

In June 2001, in connection with the grant of certain warrants as part of a licensing agreement with National Jewish Medical and Research Center and options granted for the addition of new members to the Scientific Advisory Board, AtheroGenics recorded non-cash deferred stock compensation of \$1,092,200. The fair value of the warrants and options for purposes of this calculation was determined by using the Black Scholes model. These amounts are included as a reduction of shareholders equity and are being amortized over the vesting periods of the individual warrants and options, generally five years, using the graded vesting method. During 2001, AtheroGenics recorded a total of \$335,890 of amortization of deferred stock compensation for these warrants and options. The fair value of the options and warrants is re-measured at each measurement date. Accordingly, at December 31, 2001, 100,000 shares of common stock were reserved for issuance upon the exercise of outstanding warrants.

At December 31, 2001, AtheroGenics had a total of \$2,975,314 remaining to be amortized over the corresponding vesting period of each respective option. Such amortization will approximate \$1,923,000 in 2002, \$837,000 in 2003, \$133,000 in 2004, \$63,000 in 2005 and \$19,000 in 2006. During 2001, 165,500 shares were forfeited and deferred stock compensation was decreased by \$1,395,735.

Pro forma information regarding net income is required by SFAS 123, which also requires that the information be determined as if AtheroGenics had accounted for the employee stock options granted subsequent to December 31, 1994 under the fair value method. The fair value for these options (which are granted with an exercise price equal to fair market value as determined by the board of directors on the grant date) was estimated at the date of grant using the minimum value method with the following weighted average assumptions for 2001, 2000 and 1999: risk-free interest rates of 4.51%, 6.36% and 5.75%, respectively; no dividend yield; and a weighted average expected life of the options of five years. For the period following AtheroGenics initial public offering, the Black-Scholes option valuation model was used to calculate the fair value of options granted. This method includes the above assumptions as well as the estimated volatility (99.79% and 20.85% for 2001 and 2000, respectively) of the common stock.

For purposes of pro forma disclosures, the estimated fair values of the options are amortized to expense over the options vesting periods. The weighted average fair values of options granted during 2001, 2000 and 1999 equal \$4.60, \$1.16 and \$2.54, respectively. Pro forma net loss and net loss per share are as follows:

	Year Ended December 31,		
	2001	2000	1999
Net loss	\$(18,694,195)	\$(14,151,546)	\$(10,503,993)
Net loss per share (basic and diluted)	(0.72)	(1.32)	(4.30)



**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

In August 1998, in connection with a bridge loan agreement, AtheroGenics issued to lenders warrants for 205,002 shares of Series B Redeemable Convertible Preferred Stock. These warrants became exercisable on January 1, 1999 for \$3.00 per share and expire on August 19, 2008.

In February 1999, in connection with an amendment to the bridge loan agreement, AtheroGenics issued the lenders additional warrants to purchase 200,001 shares of Series C Redeemable Convertible Preferred Stock. The warrants became exercisable on April 13, 1999 for \$3.00 per share and expire on December 31, 2008.

The Series B and Series C Redeemable Convertible Preferred Stock were subsequently converted into common stock at a conversion rate of one-to-one upon the completion of AtheroGenics' initial public offering in August 2000. At such time, the warrants became exercisable for common stock. Accordingly, at December 31, 2001, 250,290 shares of common stock were reserved for issuance upon the exercise of outstanding warrants.

**7. Short-Term Investments**

Short-term investments consist of debt securities classified as available-for-sale and have maturities greater than 90 days and less than twelve months from the date of acquisition. AtheroGenics has invested primarily in corporate notes and commercial paper, all of which have a minimum investment rating of A1/P1, and government agency notes. AtheroGenics had no realized gains or losses from the sale of investments for the period ended December 31, 2001. The following table summarizes unrealized gains and losses on AtheroGenics' short-term investments:

	Available-for-Sale Securities			Estimated Fair Value
	Amortized Cost	Gross Unrealized Loss	Gross Unrealized Gain	
Government agency notes	\$ 16,081,537	\$	\$ 15,212	\$ 16,096,749
Corporate notes	7,084,617		38,086	7,122,703
Commercial paper	6,500,000			6,500,000
Certificate of deposit	38,493			38,493
Balance at December 31, 2001	\$ 29,704,647	\$	\$ 53,298	\$ 29,757,945

All available-for-sale securities held at December 31, 2001, will mature during 2002.



**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)****8. Income Taxes**

At December 31, 2001, AtheroGenics had net operating loss carryforwards and research and development credit carryforwards of \$50,389,176 and \$1,544,330, respectively, for income tax purposes, which both begin to expire in 2010. The significant components of the deferred tax assets are:

	December 31,	
	2001	2000
Net operating loss carryforwards	\$ 19,147,887	\$ 12,763,242
Deferred revenue		422,222
Research credits	1,544,330	1,241,809
Deferred stock compensation	3,284,001	698,197
Other	259,746	392,092
	<hr/>	<hr/>
Total deferred tax assets	24,235,964	15,517,562
Valuation allowance	(24,235,964)	(15,517,562)
	<hr/>	<hr/>
Net deferred tax assets	\$	\$
	<hr/>	<hr/>

Because of AtheroGenics' lack of earnings history, the deferred tax assets have been fully offset by a valuation allowance. The valuation allowance increased \$8,718,402 and \$3,271,774 in 2001 and 2000, respectively.

AtheroGenics' net operating loss carryforwards may be subject to certain Internal Revenue Code Section 382 limitations on annual utilization in the event of changes in ownership. These limitations could significantly reduce the amount of the net operating loss carryforwards available in the future. AtheroGenics has not yet completed a full analysis of IRC Section 382 on the cumulative net operating loss carryforward. However, the annual limitations are not expected to prevent utilization of the net operating loss carryforward due to the significant increases in value indicated by the successive issues of our stock. If a change in ownership has occurred, there will be an annual accrual limitation; however, this limitation is not expected to result in a loss of the deferred tax benefit.

**9. Leases**

On June 19, 1998, AtheroGenics entered into a ten-year operating lease for office and laboratory space through March 1, 2009. Monthly lease payments of approximately \$89,400 began March 2, 1999, the date occupancy commenced. These payments are subject to increases during each successive twelve-month period based on changes in the Consumer Price Index (CPI). Future increases in monthly lease payments due to increases in the CPI are considered to be contingent rentals, and, therefore, will be charged to expense over the lease term as they become payable. AtheroGenics may extend the lease term for two successive five-year periods. AtheroGenics' other operating lease obligations are not significant.

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

At December 31, 2001, AtheroGenics' minimum aggregate commitments (net of sublease income) under long-term, non-cancelable operating leases are as follows:

	<u>Gross</u>	<u>Sublease Income</u>	<u>Net</u>
2002	\$ 1,247,663	\$ 323,508	\$ 924,155
2003	1,247,663	230,811	1,016,852
2004	1,228,416	184,975	1,043,441
2005	1,126,100	184,975	941,125
2006	1,117,583		1,117,583
Thereafter	2,421,432		2,421,432
	<u>\$ 8,388,857</u>	<u>\$ 924,269</u>	<u>\$ 7,464,588</u>

Rent expense under operating leases amounted to \$835,608, \$786,452 and \$639,934 in 2001, 2000 and 1999, respectively.

Equipment and leasehold improvements include the following amounts for leases that have been capitalized at December 31, 2001 and 2000:

	<u>2001</u>	<u>2000</u>
Lab equipment	\$ 972,500	\$ 972,500
Less accumulated amortization	(837,162)	(742,205)
	<u>\$ 135,338</u>	<u>\$ 230,295</u>

Amortization of leased assets is included in depreciation and amortization expense. The equipment leases provide for one-year extensions at the end of the lease terms.

Future minimum lease payments under capital leases consist of the following at December 31, 2001:

2002	\$92,125
2003	
	<u>92,125</u>
Total minimum lease payments	92,125
Less amounts representing interest and warrants	(5,024)
	<u>87,101</u>
Present value of net minimum lease payments	87,101
Less current portion	<u>87,101</u>
	<u>\$</u>

The amounts recorded as capital lease obligations approximate the estimated fair market values.

**10. Related Party Transactions**

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During the year ended December 31, 2001, AtheroGenics made a secured loan in the amount of \$200,000 to one of its executive officers, who is also a shareholder. The loan bears interest at a rate of 2.48% per annum, the applicable Federal rate at the time of the loan, and is due on December 26, 2004. The loan is secured by 41,000 shares of AtheroGenics common stock.

AtheroGenics has a sublease agreement for a portion of its office and laboratory space with Inhibitex, Inc. The monthly lease payments are approximately \$20,000. The lease term ends on December 31, 2005. Dr. Medford, the President and Chief Executive Officer of AtheroGenics, and Mr. Henos, the Chairman of AtheroGenics Board of Directors, are both on the Inhibitex Board of Directors.

**Table of Contents****NOTES TO FINANCIAL STATEMENTS (Continued)**

AtheroGenics has a sublease agreement for a portion of its office space with ATV Management Corp. Monthly lease payments are approximately \$3,400. The lease term ends on July 31, 2003. The Chairman of the Board of Directors of AtheroGenics is the President and sole shareholder of ATV Management Corp.

**11. Employee Benefit Plan**

AtheroGenics has a defined contribution plan covering eligible employees, which is qualified under Section 401(k) of the Internal Revenue Code. Under the provisions of the plan, eligible participating employees may elect to contribute up to 15% of their salary (up to the maximum amount of tax deferred contribution allowed by the Internal Revenue Code). AtheroGenics may make a discretionary contribution. During 2001, AtheroGenics matched 50% of employees' contributions, up to a maximum of 6% of the employees' annual base compensation. AtheroGenics contribution to the plan for 2001, 2000 and 1999 aggregated \$91,852, \$62,093 and \$37,703, respectively. AtheroGenics' stock is not an eligible investment under this plan.

**12. Quarterly Results of Operations (Unaudited)**

The following is a summary of the unaudited quarterly results of operations:

	<b>Year Ended December 31, 2001</b>			
	<b>1st Quarter</b>	<b>2nd Quarter</b>	<b>3rd Quarter</b>	<b>4th Quarter</b>
Net revenues	\$ 1,430,422	\$ 481,245	\$ 538,511	\$ 1,059,362
Operating loss	(3,884,934)	(4,537,847)	(5,456,025)	(6,127,525)
Net loss	(3,100,628)	(3,949,277)	(4,862,219)	(5,727,459)
Net loss per share data:				
Basic and diluted	(0.13)	(0.16)	(0.18)	(0.21)

	<b>Year Ended December 31, 2000</b>			
	<b>1st Quarter</b>	<b>2nd Quarter</b>	<b>3rd Quarter</b>	<b>4th Quarter</b>
Net revenues	\$ 2,091,280	\$ 2,064,050	\$ 1,905,155	\$ 2,099,218
Operating loss	(3,552,560)	(3,219,745)	(4,475,752)	(4,416,315)
Net loss	(3,394,793)	(3,083,213)	(3,963,531)	(3,507,985)
Net loss per share data:				
Basic and diluted	(1.29)	(1.05)	(0.30)	(0.15)

Because of the method used in calculating per share data, the quarterly per share data will not necessarily add to the per share data as computed for the year.

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

None.

**PART III**

**Item 10. *Directors and Executive Officers of the Registrant***

We have set forth information relating to the directors and executive officers and compliance with Section 16(a) of the Securities Exchange Act of 1934 under the captions Executive Officers and Directors and Section 16(a) Beneficial Ownership Reporting Compliance, respectively, in our proxy statement for our 2002 annual meeting of shareholders to be held on April 24, 2002. We are incorporating this information by reference in this Form 10-K. Our definitive proxy statement was filed with the Securities and Exchange Commission on March 11, 2002.

**Item 11. *Executive Compensation***

We have set forth information relating to executive compensation under the caption Executive Compensation in the proxy statement referred to in Item 10 above. We are incorporating this information by reference in this Form 10-K.

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

We have set forth information relating to ownership of our common stock by certain persons under the caption Security Ownership of Certain Beneficial Owners and Management in the proxy statement referred to in Item 10 above. We are incorporating this information by reference in this Form 10-K.

**Item 13. *Certain Relationships and Related Transactions***

We have set forth information relating to existing or proposed relationships or transactions between us and certain of our affiliates under the caption Certain Relationships and Related Transactions in the proxy statement referred to in Item 10 above. We are incorporating this information by reference in this Form 10-K.

**PART IV**

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

(a)(1) Financial Statements, filed as part of this report

Financial Statements (indexed as pages 30-47)

Report of Independent Auditors

Balance Sheets as of December 31, 2001 and 2000

Statements of Operations for the years ended December 31, 2001, 2000 and 1999

Statements Shareholders' Equity (Deficit) for the years ended December 31, 2001, 2000 and 1999

Statements of Cash Flows for the years ended December 31, 2001, 2000 and 1999

Notes to Financial Statements

(2) Financial Statement Schedules

No financial statement schedules are provided, because the information called for is not required or is shown either in the financial statements or the notes thereto.

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(3) Listing of Exhibits The response to this portion of Item 14 is submitted below as Item 14(c) of this report.

(b) Reports on Form 8-K filed in the fourth quarter of 2001:

AtheroGenics filed a report on Form 8-K on October 9, 2001 under Item 5 to report the reacquisition of full rights to AGI-1067 from Schering-Plough.

AtheroGenics filed a report on Form 8-K on November 19, 2001 under Item 5 to report the adoption of a shareholders' right plan.

(c) Exhibits

Exhibit No.	Description
3.01**	Form of Fourth Amended and Restated Articles of Incorporation of AtheroGenics, Inc.
3.02*	Form of Third Amended and Restated Bylaws of AtheroGenics, Inc., as amended
4.01**	Form of Common Stock Certificate.
4.02**	Amended and Restated Master Rights Agreement dated October 31, 1995, as amended by First Amendment dated November 1, 1995; Second Amendment dated July 30, 1996; Third Amendment dated April 13, 1999; Fourth Amendment dated May 11, 1999; and Fifth Amendment dated August 30, 1999.
4.03**	Applicable provisions of Fourth Amended and Restated Articles of Incorporation and Third Amended and Restated Bylaws of AtheroGenics, Inc. (incorporated by reference to Exhibits 3.01 and 3.02).
4.04	Rights Agreement dated as of November 9, 2001 between AtheroGenics, Inc. and American Stock Transfer & Trust Company, as Rights Agent (filed as an exhibit of the same number with AtheroGenics' Form 8-K on October 9, 2001 and incorporated herein by reference).
10.02**+	Exclusive License Agreement dated July 17, 1998 between The Regents of the University of California and AtheroGenics, Inc.
10.03**+	License Agreement dated January 11, 1995 between Emory University and AtheroGenics, Inc.
10.04**+	Patent Purchase Agreement dated April 26, 1995 between AtheroGenics, Inc. and Sampath Parthasarathy, together with Services Agreement dated April 26, 1995 between AtheroGenics, Inc. and Sampath Parthasarathy.
10.05**+	Sponsored Research Agreement dated October 14, 1996 between Emory University and AtheroGenics, Inc.
10.07**	AtheroGenics, Inc. 1995 Stock Option Plan, together with form of nonqualified stock option agreement.
10.08**	AtheroGenics, Inc. 1997 Equity Ownership Plan, as amended by Amendment No. 1 and Amendment No. 2.
10.09**	Preferred Shares Purchase Warrant dated August 24, 1998 between AtheroGenics, Inc. and certain Lenders named therein.
10.10**	Series C Convertible Preferred Stock Purchase Warrants of AtheroGenics, Inc.
10.11**	Promissory Note dated April 1, 1999 between Inhibitex, Inc. and AtheroGenics, Inc.
10.12**++	Lease Agreement dated June 19, 1998 between Cousins Properties, Inc. and AtheroGenics, Inc.
10.13**++	Master Equipment Lease dated November 1, 1995 between Phoenix Leasing Incorporated and AtheroGenics, Inc.
10.14***	Employment Agreement dated March 1, 2001 between AtheroGenics, Inc. and Russell M. Medford.
10.15***	Amendment dated January 1, 2001 to Promissory Note dated April 1, 1999 between Inhibitex, Inc. and AtheroGenics, Inc.

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Exhibit No.	Description
10.16	Form of Common Stock Purchase Agreement dated as of June 19, 2001 between AtheroGenics, Inc. and the Purchasers named therein (filed as an exhibit of the same number with AtheroGenics Registration Statement on Form S-1, Registration No. 333-64228, on July 23, 2001 and incorporated herein by reference).
10.17+	Exclusive License Agreement dated as of June 29, 2001 between AtheroGenics, Inc. and National Jewish Medical and Research Center (filed as an exhibit with the same number with Amendment No. 1 to AtheroGenics Registration Statement on Form S-1, Registration No. 333-64228, on June 29, 2001 and incorporated herein by reference).
10.18	AtheroGenics, Inc. 2001 Equity Ownership Plan (filed as Appendix B to the proxy statement for AtheroGenics 2001 Annual Shareholders Meeting as filed on March 22, 2001 and incorporated herein by reference).
10.19*	Promissory Note and Stock Pledge Agreement dated as of December 26, 2001 between AtheroGenics, Inc. and Mitchell Glass.
23.01*	Consent of Ernst & Young LLP.
24.01*	Powers of Attorney.

\* Filed herewith.

\*\* Filed as the exhibit of the same number with AtheroGenics registration statement on Form S-1, Registration No. 333-31140, declared effective by the SEC on August 8, 2000, and incorporated herein by reference.

\*\*\* Filed as an exhibit of the same number with AtheroGenics Annual Report on Form 10-K for the year ended December 31, 2001, and incorporated herein by reference.

+ Certain confidential information contained in this document has been omitted and filed separately with the Commission pursuant to a request for confidential treatment under Rule 406 of the Securities Act of 1933, as amended.

++ We agree to furnish supplementally to the Commission a copy of any omitted schedule or exhibit to this agreement upon request by the Commission.



**Table of Contents****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, on March 29, 2002.

ATHEROGENICS, INC.

By: /s/ RUSSELL M. MEDFORD, M.D., PH.D.

Russell M. Medford, M.D., Ph.D.  
*President and Chief Executive Officer*

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

Name	Title	Date
<b>Principal Executive Officer:</b>		
/s/ RUSSELL M. MEDFORD	President and Chief Executive Officer, Director	March 29, 2002
Russell M. Medford		
<b>Principal Financial and Principal Accounting Officer:</b>		
/s/ MARK P. COLONNESE	Senior Vice President of Finance and Administration and Chief Financial Officer	March 29, 2002
Mark P. Colonnese		
<b>Additional Directors:</b>		
*	Director	March 29, 2002
Michael A. Henos		
*	Director	March 29, 2002
R. Wayne Alexander		
*	Director	March 29, 2002
Vaughn D. Bryson		
*	Director	March 29, 2002
T. Forcht Dagi		
*	Director	March 29, 2002
Arthur M. Pappas		
*	Director	March 29, 2002
William A. Scott		

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\*

Director

March 29,  
2002

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Stephen G. Sudovar

\*By: /s/ MARK P. COLONNESE

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Mark P. Colonnese  
Attorney-in-fact