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

FORM 10-K

(Mark One)
[X] ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934
For the fiscal year ended December 31, 2002
OR
[ ] TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934
For the transition period from to
Commission file number 033-76414

ARIAD Pharmaceuticals, Inc.

(Exact name of registrant as specified in its charter)

Delaware	**22-3106987**
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification No.)

26 Landsdowne Street, Cambridge, Massachusetts 02139-4234
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: (617) 494-0400

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

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

Common Stock, $.001 Par Value

Rights to Purchase Series A Preferred Stock

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

Yes [X] No [ ]

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

Indicate by check mark whether the registrant is an accelerated filer (as defined in Rule 12b-2 of the Act).

Yes [X] No [ ]

The aggregate market value of the registrant's common stock held by nonaffiliates of the registrant (without admitting that any person whose shares are not included in such calculation is an affiliate) computed by reference to the price at which the common stock was last sold, or the average bid and asked price of the common stock, as of the last business day of the registrant's most recently completed second fiscal quarter was $121.8 million.

As of March 12, 2003, the registrant had 34,846,640 shares of common stock outstanding.

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PART I

ITEM 1: BUSINESS

The following Business Section contains forward-looking statements, which involve risks and uncertainties. Our actual results could differ materially from those anticipated in these forward-looking statements as a result of certain factors (see "Certain Factors That May Affect Future Results of Operations"). In this Annual Report on Form 10-K, we incorporate by reference certain information from parts of other documents filed with the Securities and Exchange Commission, or the SEC. The SEC allows us to disclose important information by referring to it in that manner. When reading this Annual Report, please refer to such information which is incorporated by reference in this document.

Our internet website address is http://www.ariad.com. Our annual reports on Form 10-K, quarterly reports on Form 10-Q and current reports on Form 8-K which have been filed with the SEC are available to you free of charge through a hyperlink on our internet website.

Corporate Overview

We are engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo,* that may be used to treat diseases in innovative ways. We are developing a comprehensive approach to the treatment of cancer and are primarily focused on a series of product candidates for targeted indications: (1) AP23573, which is in phase 1 development, to treat solid tumors and other malignancies; (2) AP23464 to block the spread of cancer, or metastases, and to treat certain forms of leukemia; and (3) AP23841 to treat cancer that has spread to bone, or bone metastases, and to treat primary bone cancers, such as osteogenic sarcomas. These small-molecule product candidates have novel mechanisms of action and have been demonstrated to provide highly potent targeted anti-cancer activity. The AP23573 and AP23841 class of drugs, known as mTOR inhibitors, starves cancer cells by inhibiting nutrient uptake (metabolic arrest) and growth factor stimulation. AP23464, a Src/Abl inhibitor, blocks the process by which certain solid tumors migrate from primary to distant sites; it also inhibits a key enzyme that is abnormally active in certain forms of leukemia.

AP23573 can also be used to block the migration and proliferation of vascular smooth muscle cells – the primary cause of narrowing and blockage of coronary and peripheral arteries. With the advent of a newly emerging medical technology – drug-delivery stents to reduce restenosis, or reblockage, of injured arteries following angioplasty and stenting – active discussions have been initiated with potential medical device partners that may open up this second clinical and commercial opportunity for AP23573 delivered in vascular stents.

We also are developing two regulated gene or cellular product candidates to treat diseases that occur commonly in patients with certain cancers and various blood diseases: (1) a regulated protein therapy product candidate to treat anemia in which the production of erythropoietin is controlled *in vivo* by an orally administered drug, AP22594; and (2) a T cell immunotherapy product candidate in which a non-immunosuppressive drug, AP1903, may be used to treat graft-vs-host disease following donor bone marrow and stem cell transplantation.

With respect to the development and commercialization of our lead product candidates, our goals are to: (1) enter into a partnership with a pharmaceutical or biotechnology company to develop and commercialize our lead product candidate, AP23573, to treat cancer; (2) enter into partnerships with medical device companies to develop and commercialize our lead product candidate, AP23573, in drug-delivery stents to decrease reblockage of arteries following angioplasty and stenting; (3) independently develop as many of

our product candidates as possible through at least phase 2 before partnering them; (4) establish the commercial infrastructure to market or co-market our anti-cancer product candidates in the United States; and (5) enter into partnerships for our other product candidates outside the United States.

We have an exclusive license to pioneering technology and patents related to the discovery, development and use of drugs to regulate NF-κB cell-signaling activity, which can be used to treat medically important disorders, including inflammation, cancer and osteoporosis. We permit broad use of our NF-κB intellectual property at no cost by investigators at academic and not-for-profit institutions to conduct non-commercial research. Our goal is to license our NF-κB technology to pharmaceutical and biotechnology companies conducting research on the discovery of drugs that modulate NF-κB cell signaling and/or marketing such drugs. Bristol-Myers Squibb Company entered into a research and development license for our NF-κB technology in November 2002.

We distribute our RegTech cell-signaling technologies at no cost to academic investigators in the form of our Regulation Kits. Over 600 academic investigators worldwide already are using this technology in diverse areas of research, and over 150 scientific papers describing their use have been published. Our goal is to license our RegTech cell-signaling technologies to pharmaceutical and biotechnology companies to accelerate their drug discovery. GPC Biotech AG entered into a research and development license for our ARGENT cell-signaling regulation technology in January 2003. In addition, we may jointly develop product candidates incorporating our ARGENT cell-signaling regulation technology, with companies that have proprietary therapeutic genes, cellular systems or gene-delivery vectors.

ARIAD was organized as a Delaware corporation in April 1991. Our principal executive offices are located at 26 Landsdowne Street, Cambridge, Massachusetts 02139-4234, and our telephone number is (617) 494-0400. We maintain an internet website at http://www.ariad.com, the contents of which are not incorporated into this Annual Report.

Our Primary Lead Product Candidates

We currently are focused primarily on developing three lead anti-cancer product candidates for targeted clinical indications – all of which are small-molecule drugs that regulate cell signaling. Many of the critical functions of cells, such as cell growth, differentiation, gene transcription, metabolism, motility and survival, take place through the processes of cell signaling**.** Disruption or over-stimulation of cell-signaling pathways has been implicated in many disease states. Our product candidates interfere with specific cellular proteins or pathways that have been well-characterized and validated as targets. All of these product candidates were developed in-house through the integration of structure-based drug design and chemo-informatics, or computational chemistry, functional genomics and proteomics. We believe that our product candidates, if successfully developed, will serve large, unmet medical needs.

Our primary lead product candidates are as follows:

Product Candidate	Target	Initial Clinical Indications	Development Status
AP23573	mTOR	Solid tumors/other malignancies	Phase 1
AP23464	Src/Abl	Cancer metastases	Pre-IND
AP23464	Src/Abl	Leukemia	Pre-IND
AP23841	mTOR	Bone metastases	Pre-IND
AP23841	mTOR	Primary bone cancers	Pre-IND
AP23573/Drug-delivery Stents	mTOR	Vascular disease (Restenosis)	Pre-IDE

In the case of pharmaceutical products, after a development candidate (and back-up candidates) have been designated, Pre-Investigational New Drug, or pre-IND, development is initiated and consists of longer-term investigational toxicology studies, as well as other studies required by the U.S. Food and Drug Administration, or FDA, or other regulatory authorities for inclusion in an IND or other regulatory filings to initiate human studies. Examples of these studies include toxicology, pharmacology, and metabolism studies conducted under the current Good Laboratory Practices, or cGLP, requirements, as well as *in vivo* efficacy studies in relevant animal models of disease. Clinical development requires manufacturing of clinical-grade material for use in humans produced under current Good Manufacturing Practices, or cGMP, requirements. Phase 1 development includes obtaining regulatory and institutional review board, or IRB, approvals for administering product candidates for the first time to healthy volunteers and select patients with disease (*e.g.,* refractory cancers) and conducting clinical trials that are designed to provide initial safety and pharmacokinetic data. Phase 2 development includes obtaining regulatory and IRB approvals for administering product candidates to patients with a broader spectrum of diseases and conducting clinical trials that are designed to provide further safety data and initial indications of a product candidate's clinical efficacy in its proposed use. In the case of medical devices such as drug-delivery stents, pre-Investigational Device Exemption, or pre-IDE, development consists of stent formulation development and other studies required by the FDA or other regulatory authorities for inclusion in an IDE or other regulatory filings to initiate human studies. Examples of these studies include kinetic experiments on the release of drug from drug-delivery stents, as well as safety and efficacy studies in relevant animal models of disease.

Solid Tumors and Other Malignancies

The Disease: Cancer, the second leading cause of death in the Western world, is a complex collection of hundreds of separate diseases characterized by the uncontrolled growth, proliferation and spread of abnormal cells. Great strides have been made in the past few decades in understanding the molecular basis for the transformation of normal cells into cancer cells by searching for genetic differences between them. These studies have revealed that cancer cells often harbor genetic mutations that alter the controlling mechanisms in cell signaling that constrain cell growth in healthy cells and induce cell death in abnormal cells. Two major classes of such genetic alteration have been identified. Genes that turn on cell growth and division and generate conditions conducive to the spread of cancer are often over-activated or over-expressed in tumors. These genes are called oncogenes, and they represent key targets for anti-cancer drug design, since drugs that inhibit their activity should re-establish normal regulation of cell growth and proliferation and prevent the spread of malignant cells. A second class of genes, called tumor suppressors,

plays an opposite role by preventing uncontrolled cell growth. Without treatment, these genes are often inactivated or deleted in tumors, leading to uncontrolled proliferation and potentially enhancing the effects of oncogenes.

Current Therapies: Several forms of medical therapy have evolved as adjuncts or alternatives to surgery, including the introduction of cytotoxic chemotherapy and radiation therapy over 50 years ago. Although these therapies have evolved and continue to be the mainstay of cancer treatment, especially after the cancer has spread from its site of origin, these therapies often are limited by lack of specificity and result in general toxicity by killing healthy cells, along with malignant cells. Recently, a number of alternative therapies have been introduced, including endocrine therapy to treat cancers of certain hormone-sensitive organs, recombinant biologics, and small molecules and monoclonal antibodies that target the molecular determinants of the transformation to malignancy. Several growth factor inhibitors and protein kinase inhibitors have been developed to block molecular pathways implicated in cancer.

Our Approach: Our lead anti-cancer product candidate, AP23573, is a potent small-molecule inhibitor of the cell-signaling protein mTOR – a well-defined target that plays a key role in orchestrating the progression of the cell cycle through the regulation of nutrient uptake (metabolic arrest) and growth factor stimulation. To sustain accelerated growth and proliferation, cancer cells require an ample supply of nutrients and stimulation by growth factors, and these key processes are blocked by AP23573. Our extensive *in vivo* studies demonstrate that AP23573 shrinks cancer cells and arrests tumor growth in animal models.

Other studies indicate that the effect of mTOR inhibitors may be even more pronounced in tumor cells with a mutated or inactivated form of a recently discovered tumor suppressor gene, known as PTEN. Several difficult-to-treat cancers, including prostate, uterine, pancreatic, and ovarian cancer, as well as melanoma, leukemia and gliomas, frequently are missing the PTEN protein due to such a mutation. Since cancer patients with PTEN-deficient tumor cells can be identified using readily available tests, it may be possible to select those who may benefit most from AP23573. This product candidate is currently in phase 1 development. Dosing of patients with refractory or advanced cancers is expected to begin in the second quarter 2003 in two clinical trials at major cancer centers in the United States.

Cancer Metastases

The Disease: Tumor cell migration, or metastases, is an early event in cancer progression. Cancer cells escape from the primary tumor, first invading the surrounding local tissue, then traveling through the blood or lymphatic circulation to more distant sites where they reestablish themselves and proliferate further. Only a small number of cancer cells, estimated to be less than 0.01%, survive this highly complex journey. The most common distant sites to which cancers migrate are bone, lung, liver, lymph nodes and brain.

Based on estimates for 2003, approximately 1.2 million new cases of invasive cancer will be diagnosed annually in the United States. In one-third of these newly diagnosed cases, patients are likely to have overt spread of the primary cancer at initial presentation. Another third are likely to have microscopic metastases which are undetectable with routine diagnostic tests other than tumor biopsy. As soon as the diagnosis of cancer is established, the first priority is to determine whether, and to what extent, the disease has spread beyond the primary site(s). This process is referred to as staging, and there is a high correlation between the extent of metastases and patient morbidity and mortality.

Current Therapies: Chemotherapy is often administered as an adjunct to surgery in an attempt to eliminate disseminated tumor cells. Because of the general toxicity of chemotherapy, the side-effects can be severe, and the cure rate is low. Sometimes, very high doses of chemotherapy are prescribed despite the risk that potentially life-threatening complications may result, including compromise of the patient's bone marrow and stem cells, which may necessitate a bone-marrow or stem-cell transplant.

Alternative therapies that may be used to treat cancer metastases include hormonal therapy, radiotherapy, analgesics for pain and anti-resorptive drugs for bone metastases; these treatments may decrease morbidity and mortality but rarely eradicate the patient's cancer after it has spread widely.

The urgent need for safer and more effective alternatives to treating the spread of cancer has prompted widespread research to decipher the multi-step genetic regulation of the metastatic process. Numerous studies demonstrate that migrating tumor cells generally exhibit either a loss or an augmentation of the function of a particular gene or set of genes – in some cases, both loss and augmentation. Two approaches are being considered: those that target factors outside the cell that stimulate gene expression and those that target gene-regulated factors inside the cell which have been implicated in tumor progression and metastases.

Our Approach: We have pioneered the discovery and development of small-molecule drugs to inhibit proteins, such as Src, that are believed to orchestrate the progression and spread of cancer. Recent research has elucidated the critical role that enhanced expression of the Src protein plays in the dissemination of colon cancer – a finding that has attracted widespread attention in the scientific community. Based on this work, the Src protein is now known to be involved in loosening the structure of tissue surrounding a tumor, opening the way for cancer cells to spread. AP23464 is a potent inhibitor of the Src protein (and the Abl protein – see next section on leukemia) and is being developed to treat the spread of solid tumors, such as colon and breast cancers. AP23464 is in pre-IND development.

Leukemia

The Disease*:* Leukemia is cancer that occurs in the bone marrow resulting in excessive production of dysfunctional blood cells, usually white blood cells, and, as a result, insufficient production of red blood cells and platelets. Based on estimates for 2003, approximately 30,000 patients will be diagnosed with leukemia in the United States annually, and over 20,000 are expected to die from this disease. While ten times as many adults as children develop leukemia, this blood malignancy is the number one cause of death from cancer in children and young adults under age 20.

Multiple genetic defects have been implicated in the development of leukemia, of which there are four main types based on how fast the abnormal cells are produced – acute, or quickly, and chronic, or slowly – and on the type of white blood cell that has become malignant. Acute myeloid leukemia, or AML, is the most common type, occurring in about one-third of newly diagnosed leukemia patients. Chronic myelogenous leukemia, or CML, was the first cancer to be linked to an identified genetic defect. This genetic abnormality leads to the over-activation of a protein known as Abl, which plays a pivotal role in regulating cell growth and maturation. While CML is diagnosed in fewer than 5,000 patients in the United States each year, it is frequently associated with a poor prognosis. A closely related genetic defect is detectable in approximately 25 to 30% of patients with acute lymphoid leukemia, or ALL.

Current Therapies: There are five major approaches to the treatment of leukemia: (1) cytotoxic chemotherapy, often given in combination, to kill leukemia cells; (2) interferon therapy to slow the reproduction of leukemia cells and promote the immune system's anti-cancer activity; (3) radiation therapy to kill leukemic cells in localized areas such as the brain; (4) bone-marrow or stem-cell transplantation to enable treatment with high doses of chemotherapy and radiation therapy; and (5) surgery to remove an enlarged spleen. Recently, a new drug called imatinib mesylate (known as Gleevec®) that inhibits the activity of the Abl protein has been added to the treatment options for CML patients. However, not all patients are equally responsive to this drug, and many who are initially responsive become resistant to the drug after an initially favorable response. Investigation of the mechanism of this resistance suggests that over-activation of another protein, the Src protein – also known to be a key regulator of cell-growth – may play an important role in the development of resistance to imatinib mesylate therapy.

Our Approach: Our scientists have discovered a small-molecule product candidate that potently inhibits both the Abl protein and the Src protein. This dual inhibitor, AP23464, may address the need for an alternative treatment for CML patients who have become resistant to imatinib mesylate and for certain patients with leukemia as first-line therapy. In addition, AP23464 may be administered in combination with drugs that target other proteins implicated in the uncontrolled cell growth and proliferation that are characteristic of most cancers, including leukemia. AP23464 is in pre-IND development.

Bone Metastases

The Disease: The spread of malignant cells from the primary tumor to the skeleton and the subsequent destruction of bone is a frequent and debilitating complication of many cancers, especially breast, prostate, colon, and lung cancer and multiple myeloma. An estimated 750,000 patients worldwide die each year with bone metastases. In those patients originally diagnosed with breast and prostate cancers, most of the tumor burden at the time of death is in bone.

Bone is a very attractive target for migrating cancer cells. Once they arrive, cancer cells begin stimulating the breakdown of bone by cells known as osteoclasts. The breakdown of bone, in turn, releases bone-derived growth factors that attract more cancer cells and facilitate their growth and proliferation, creating a vicious cycle of malignant growth within bone.

Bone metastases are a common cause of cancer-associated morbidity. Like osteoporosis, bone metastases result in severe bone loss but also are associated with local pain, fractures, vertebral instability and compression, and elevations in blood calcium often to life-threatening levels. These clinical findings can be devastating to a cancer patient. Bone metastases are a major problem in cancer management, both in terms of cost and morbidity.

Current Therapies: There is no known cure for bone metastases. However, several therapies are used to treat bone metastases and skeletal-related events, including chemotherapy, hormonal therapy, radiotherapy, analgesics for pain and anti-resorptive drugs. Most recently, newer-generation bisphosphonates have been advanced for the treatment of bone metastases and have already achieved substantial clinical use for this indication.

Our Approach: Recent research demonstrates that most skeletal destruction due to bone metastases is caused by cancer cell stimulation of osteoclasts, the bone cells responsible for bone breakdown, and the tumor burden in bones, especially the long bones, spine, and hips. Advances in our understanding of the biology of bone metastases has led to a new preventive and therapeutic strategy: designing a small-molecule product candidate, AP23841, that both inhibits bone breakdown – interrupting the vicious cycle that makes bone such an attractive environment for tumor growth – and blocks the growth of cancer cells that have spread to bone. AP23841 is a potent inhibitor of mTOR, a protein known to play a key role in orchestrating nutrient uptake and growth factor stimulation in tumors, and has chemical properties that target it selectively to bone. AP23841 is in pre-IND development.

Primary Bone Cancers

The Disease: Primary bone cancer is relatively rare with approximately 2,400 new cases diagnosed each year in the United States. The most common type of primary bone cancer is osteogenic sarcoma which develops in new tissue in growing bones, followed by Ewing's sarcoma which begins in immature nerve tissue in the bone marrow. Both cancers almost always occur in children and adolescents, particularly those who have had radiation and chemotherapy treatments for other diseases. Approximately 1,300 patients with primary bone cancer will succumb to their disease this year.

Current Therapies: Primary bone cancers are extremely difficult to treat. Pre-operative chemotherapy followed by limb-sparing surgery, if possible, is the standard regimen. Multiple chemotherapeutic agents are used in combination to treat these aggressive tumors. Unfortunately, many patients require limb amputation and/or radiation therapy, and five-year survival is poor, especially if the primary tumor is located in the pelvis or the cancer has already spread outside of bone.

Our Approach: Like all cancers, bone tumors require an ample supply of nutrients and the stimulation of growth factors to sustain accelerated growth and proliferation. Our small-molecule product candidate, AP23841, has chemical properties that selectively target the compound to bone where it can inhibit the mTOR protein. The AP23841 class of compounds (including its analog, AP23675) inhibits the growth and proliferation of bone cancer cells making it well suited for the intended clinical application. AP23841 is in pre-IND development.

Vascular Disease (Restenosis After Angioplasty and Stenting)

The Disease: The primary cause of death in the Western world is cardiovascular disease, and the common form is the narrowing and blockage of arteries due to atherosclerosis. Over one million coronary angioplasty procedures are performed annually in the United States to open narrowed or occluded arteries, followed in approximately half of the cases by the insertion of a wire-mesh scaffold, or stent, to prevent or minimize the likelihood of restenosis, or reblockage. A similar procedure is also commonly used to treat blocked peripheral arteries. Without stents, the incidence of vascular reblockage within the first few months runs as high as 40%, depending on the configuration and location of the vascular lesion and other clinical factors. With the use of stents to help keep dilated coronary arteries open, the incidence of reblockage is reduced substantially but still unacceptably high.

Current Therapies: Numerous drugs, including many antiplatelet agents, anticoagulants, ACE inhibitors, and cytotoxic agents, administered to patients following coronary angioplasty and stenting have failed to significantly reduce the overall incidence of vascular reblockage. Novel therapeutic interventions are needed. Recent clinical studies have found extremely low reblockage rates in patients treated with stents that are coated with and deliver small-molecule drugs, such as sirolimus, an mTOR inhibitor, or paclitaxel, a cytotoxic agent. Such stents may become the standard-of-care for patients undergoing interventional procedures to open narrowed coronary or peripheral arteries.

Our Approach: We have pioneered the discovery and development of sirolimus analogs, including AP23573, our lead product candidate for cancer. The highly potent activity of AP23573 in arresting the growth and proliferation of cancer cells can be duplicated in vascular smooth muscle cells – the primary cause of narrowing and reblockage of arteries. We are currently in active discussions with potential medical device partners to develop and commercialize next-generation stents that deliver AP23573 locally to the site of vascular injury.

Our Other Product Development Programs

We also are developing two regulated gene or cellular therapy product candidates to treat diseases that occur commonly in patients with certain cancer and various blood diseases: (1) a regulated protein therapy product candidate to treat anemia in which the production of erythropoietin, or Epo, is controlled *in vivo* by an orally administered drug, AP22594; and (2) a T cell immunotherapy product candidate in which a non-immunosuppressive drug, AP1903, may be used to treat graft-vs-host disease, or GvHD, following allogeneic, or donor, bone marrow and stem cell transplantation. These product candidates utilize our ARGENT cell-signaling technology and are summarized as follows:

Product Candidate	Target	Initial Clinical Indications	Development Status
AP22594	Epo	Anemia	Pre-IND
AP1903	Fas	GvHD after T Cell Immunotherapy (Leukemia and other blood cancers)	Phase 2

The development timelines for our GvHD and anemia product candidates have been extended so that we may address vector manufacturing challenges and obtain additional funding or partners for these programs. Future clinical studies of these product candidates can only be conducted after we have successfully addressed these issues.

Anemia

The Disease: Red blood cells, produced in the bone marrow, transport oxygen from the lungs to the cells of the body and carbon dioxide to the lungs. Erythropoietin, or Epo, is a naturally occurring protein made primarily in the kidney that stimulates the manufacture of more red blood cells, when needed. If the body loses its ability to manufacture sufficient quantities of Epo, the optimal number of red blood cells (and their oxygen carrying component, hemoglobin) can no longer be maintained in the circulation – a condition known as anemia. This is usually the case with patients being treated with current highly cytotoxic anti-cancer therapies and with patients suffering from severe renal disease.

Current Therapies: Recombinant Epo (including a newer-generation version) is presently being used for the treatment of anemia caused by cancer chemotherapy, chronic renal failure (including end-stage renal disease) and zidovudine treatment of HIV-infected individuals. Recombinant Epo is routinely injected on a recurring schedule into a vein or under the skin.

Our Approach: We are developing an alternative approach to deliver and regulate therapeutic proteins such as Epo based on our ARGENT cell-signaling regulation technology. Rather than relying on repetitive injections of Epo to provide the therapeutic benefit, our approach is designed to involve a single, or infrequent, injection(s) of the Epo gene using a gene-transfer vector into the patient's muscle or other tissue in an inactive form, with Epo only being produced when the patient takes our small-molecule drug, AP22594. We have demonstrated production of therapeutically effective amounts of Epo for almost five years after one-time injection in monkeys of our proprietary gene complex. We anticipate that the patient will take AP22594 on a regular schedule (e.g., weekly, biweekly or monthly) to activate the Epo gene and manufacture Epo in the target tissue (i.e., skeletal muscle). The production of Epo would only occur in response to the patient taking the drug, and the amount of Epo manufactured *in vivo* would depend on the amount of AP22594 the patient takes.

We believe that the potential competitive advantages of our product candidate for anemia include:

- Replacement of a frequently injected recombinant product largely by a small pill taken every one to four weeks;
- Epo production precisely controlled within a therapeutic window as opposed to the oscillating blood levels that frequently occur by injectable routes of administration;
- The ability to cost-effectively deliver the high doses of Epo needed to treat certain types of refractory anemias.

Our AP22594 product candidate for anemia is currently in pre-IND development. One of the principal challenges being addressed in this program is the manufacture of cGMP gene-transfer vector for use in pharmacology and toxicology studies and for human trials.

Graft-vs-Host Disease

The Disease: Bone-marrow transplantation, or BMT, followed by T-cell immunotherapy, has become a well-established medical procedure to treat leukemias and other advanced-staged cancers, as well as non-malignant diseases, such as hemoglobinopathies and autoimmune diseases. The procedure permits patients to receive high doses of cytotoxic chemotherapy and/or radiation therapy that not only eliminates the unwanted (malignant) cells but also destroys healthy cells within the bone marrow. Therefore, a patient's bone marrow must be replaced by infusion of bone marrow or peripheral stem cells, generally followed by an infusion of donor T cells that provides temporary immune function that combines potent anti-cancer, anti-viral and anti-bacterial activity until their own immune system has reconstituted. In approximately one third of the procedures, the bone marrow or stem cells and T cells must be provided by a donor, often unrelated to the BMT recipient.

In approximately half of the recipients of donor BMT and donor T cells, the T cells attack the patients' own tissues, causing a disease known as graft-vs-host disease, or GvHD. If there is not a good genetic match between the donor and patient, the recipient has an even higher risk (as high as 90% depending on the degree of mismatch) of developing GvHD – a life-threatening complication. The major organs attacked are the patient's skin, mucosa, liver and gastrointestinal tract.

Current Therapies: The incidence and severity of GvHD can be reduced by withholding T-cell immunotherapy following BMT. Unfortunately, this also eliminates the beneficial effects of those T cells. Highly effective treatments for GvHD are currently not available. In fact, clinical experience indicates that approximately 50% of GvHD patients fail to respond fully to the current standard treatment which generally consists of immunosuppressive agents. Such drugs also put the patient at greater risk of infection, since they compromise an already weakened immune system.

Our Approach: We are developing a non-immunosuppressive treatment for GvHD that we believe will target and eliminate only the T cells that cause the disease (*i.e.,* the donor's T cells), if those T cells attack the patient's own tissues, while preserving the immune cells that are being produced by the BMT. In our GvHD product candidate, donor T cells are modified using a gene-transfer vector to make them susceptible to our small-molecule drug candidate, AP1903. This drug candidate may be administered if GvHD occurs, potentially killing the disease-causing donor T cells and leaving other immune cells unaffected.

We believe that our AP1903 T-cell immunotherapy product candidate may have a favorable impact on patient outcome and increase the number of patients who could benefit from donor BMT and T-cell immunotherapy by improving the risk-to-benefit ratio of the underlying treatment. AP1903 was found to be safe and well tolerated in a Phase 1 clinical study. In addition, this study showed that AP1903 blood levels were reached that are expected to be clinically effective. Our product candidate for GvHD is in phase 2 development, where the principal challenge is the manufacture of cGMP gene-transfer vector and the development of standardized clinical-scale cell-processing methods to effectively engineer donor T cells for use in our planned multi-center clinical trials.

Our Research Programs

The regulation of cell signaling is a part of normal cellular function, and defects play critical roles in many major diseases. As a result, our technologies have a broad range of potential therapeutic applications, and we can leverage the knowledge and expertise that we have gained in the development of our current lead product candidates for use in the cancer field.

Cell-signaling Inhibitors That Target Bone Diseases: We have developed small-molecule inhibitors of a cell-signaling pathway in bone cells that is critically involved in both bone breakdown and new bone formation. In a special strain of mice that was genetically manipulated to delete a key gene, several groups of researchers found that the deletion prevented bone resorption, increased bone mass, and enhanced bone formation. Extensive *in vivo* and *in vitro* studies by our scientists and collaborators have demonstrated that our small-molecule inhibitors have beneficial effects in animal models of diseases, such as osteoporosis and bone metastases. One such compound with potent anti-resorptive activity, AP23451, was being developed to treat bone metastases. However, it has been replaced by AP23841, which we plan to develop for both bone metastases and primary bone cancers.

Cellular Therapy and Stem Cell Therapy: In our GvHD product candidate, donor T cells are eliminated if they attack the patient's own tissues. Our approach uses a small-molecule drug to activate a "cell-death" switch in the genetically modified T cells, preserving the immune cells that are being produced by the patient's donated bone marrow. We believe the knowledge gained from this program may be applicable to other cellular therapies.

For example, stem cells are master cells that retain the ability to specialize, or differentiate, into many different types of specialized cells. Recent research has emphasized the broad potential of stem cells, both embryonic and adult forms, to treat disease by providing a source of cells that can be used to replace defective cells, tissues or even whole organs. Along with our collaborators, we have demonstrated, both *in vitro* and *in vivo*, that our cell-signaling regulation technologies can potentially overcome the two key limitations to the widespread use of stem-cell based therapies: (1) the inability to transfer therapeutic or corrective genes into stem cells efficiently; and (2) the subsequent difficulty in reliably deriving large numbers of specialized cells of the correct type and purity to patients.

In preclinical studies, our academic collaborators have also demonstrated regulated growth of other potentially useful cell types using ARGENT cell-growth switches customized for the desired cell therapy product. These include liver cells (for the treatment of hepatic disease), muscle cells (for the treatment of heart failure), and pancreatic islet cells (for the treatment of diabetes).

Orally Regulated Protein Therapy: Our ARGENT cell-signaling regulation technology represents a promising general platform for the delivery of secreted therapeutic proteins, because it has the potential to control the level of gene expression using an orally administered drug, and protein levels can be optimized within a therapeutic window. Allowing therapy to be terminated, if necessary, also enhances safety. We believe that results obtained to date from our anemia program with Epo may be used to accelerate the development of other protein therapy candidates. Several biotechnology companies are currently conducting preliminary studies with us to determine the feasibility of jointly developing product candidates incorporating our ARGENT cell-signaling regulation technology.

Our Core Competencies

Our research programs are built around key areas of competency in structure-based drug design and chemo-informatics, functional genomics and proteomics, and protein engineering. The integration of these strengths provides us with unique opportunities in the era of post-genomic drug discovery.

Functional genomics and proteomics are the study of gene and protein function, or more specifically the study of how particular genes regulate cellular function. A further aspect of functional genomics is the study of how the protein products are linked in cell-signaling pathways and how these pathways are regulated. Functional genomics has particular relevance to the process of identifying specific disease-related molecular targets for drug discovery, a process termed target validation.

Protein engineering is the design and modification of proteins based on the knowledge of their atomic level structure, obtained through the use of protein X-ray crystallography or nuclear magnetic resonance spectroscopy. Usually, the design process utilizes the three-dimensional structure of the protein to incorporate non-native amino acids into the protein's structure. This process generates new surface characteristics, thereby altering the small molecule or protein binding properties of the protein.

Structure-based drug design is a computational approach used to design small organic drug molecules that bind specifically to a particular protein in a cell-signaling pathway, for example, the critical molecular target in that pathway known to be linked to a disease. Using the target protein's three-dimensional atomic structure, drugs can be designed and optimized to bind both tightly and selectively to the target, which should lead to more potent drugs with fewer side effects. Structure-based drug design integrates structural biology and computer-assisted molecular modeling methods and has been applied directly to validated molecular targets in our cell-signaling programs to discover and optimize lead compounds. Chemo-informatic techniques and virtual screening further expand the utility of structural methods in drug discovery.

Our Enabling Platform Technologies

NF-κB Cell-signaling Technology

The Science: Dr. David Baltimore, formerly of the Whitehead Institute for Biomedical Research, Dr. Phillip Sharp of the Massachusetts Institute of Technology, and Dr. Thomas Maniatis of Harvard University, together with a team of scientists in their respective laboratories, discovered a family of genes that encode proteins they called NF-κB and I-κB, its inhibitor; the critical role played by NF-κB cell signaling in regulating cellular processes involved in various difficult-to-treat diseases; and methods to determine whether a compound regulates NF-κB cell-signaling activity. NF-κB can be generally thought of as a "biological switch" that can be turned off or on using these methods to treat disorders, such as inflammation, cancer and osteoporosis.

The Patents: ARIAD has a portfolio of four issued patents relating to NF-κB, three in the United States and one in Europe. The most recent U.S. patent, issued in June 2002, contains a range of focused claims to methods useful for treating various disease conditions through modulation of NF-κB activity. Bristol-Myers Squibb Company was the first company to enter into a research and development license for our NF-κB intellectual property in November 2002.

Regulation Technologies to Accelerate Drug Discovery

Overview: Our proprietary portfolio of cell-signaling regulation technologies includes the ARGENT Signaling, ARGENT Transcription, and RPD Secretion Technologies (collectively known as our RegTech technologies). Intracellular processes can be controlled with small molecules providing versatile tools for use in cell biology, functional genomics, proteomics, and drug-discovery research. For example, our ARGENT technologies form the basis of three-hybrid screening approaches that can be used to discover and characterize targets and lead molecules. To maximize their use by the scientific community, we distribute our technologies at no cost to academic investigators in the form of our Regulation Kits. Over 600 investigators worldwide already are using our Regulation Kits in diverse areas of research, and over 150 scientific papers describing their use have been published. For researchers in pharmaceutical and biotechnology companies, we have established an alternative licensing program to provide them with access to our cell-signaling regulation technologies on commercial terms; GPC Biotech AG was the first company to enter into such a license agreement in January 2003.

Target Validation – Cell Signaling: As analysis of the human genome sequence uncovers a wealth of new uncharacterized genes, a key challenge will be validating those genes that are good drug targets. Many of these are likely to be signaling proteins. Our ARGENT Signaling Technology allows single signaling proteins to be activated in isolation, allowing their precise functional role to be assessed *in vitro* and then *in vivo*. ARGENT tools are effective for early analysis of newly identified "orphan" signaling proteins, because no knowledge of natural ligands or binding partners is required. In addition, identification of new pathway components and gene expression changes that occur with activation can be used to identify and further validate new drug targets.

Once a cell-signaling pathway has been validated, the same ARGENT technology can provide useful tools for the next stages of drug development. The inducible gene can be engineered into experimental animals to provide an ARGENT model of the associated disease. ARGENT cell lines in which the validated signaling complex can be inducibly activated also can provide the basis for highly targeted cell-based screening for small-molecule drug candidates.

Target Validation – Gene Transcription: Varying the expression level of a gene is an effective way to study its function. The tight, dose-dependent control of expression afforded by our ARGENT Transcription Technology allows precise correlation of gene expression levels with their physiological consequences. Our technology also can be used to inducibly express inhibitors of supposed targets, such as dominant-negative mutants or gene-specific DNA binding proteins, for validation purposes.

A major application of our ARGENT Transcription Technology, based on its tight regulation of genes, is the creation of inducible knockout mice. Knockout mice in which both copies of a gene of interest have been eliminated are extremely useful for assessing the role of the deleted gene in disease. Unfortunately, many knockout mice are not viable, because expression of the gene is required during embryonic development. In addition, complete knockouts often suffer from changes in the expression of other genes that may compromise interpretation of the resulting physical, biochemical, and physiological makeup of the animal, or its phenotype. We believe that both of these problems can be solved using our ARGENT Transcription Technology by generating inducible knockouts in which genes are eliminated in the adult mouse by administering a small molecule.

Product Validation: The human genome sequence provides a rich source of potential proteins that are themselves drug candidates. In addition, advances in protein and antibody engineering are increasingly yielding large numbers of novel proteins that have therapeutic potential. Validating these molecules as products required extensive efforts in protein manufacturing, purification, scale-up and formulation. Inducible expression in animals can be used to validate therapeutic protein product candidates, in particular, secreted proteins and monoclonal antibodies, without the need to express and purify large amounts of recombinant protein. Since the level of protein delivered can be precisely controlled, this approach offers an effective way to characterize both the therapeutic and safety profiles of protein product candidates.

Our ARGENT Transcription and RPD Secretion Technologies provide complementary alternatives to this approach to product validation. The use of our ARGENT Transcription Technology allows a protein to be delivered over the course of several days, whereas the alternative approach based on our RPD Secretion Technology is particularly useful for generating rapid bursts of protein expression. The use of our ARGENT and RPD technologies to validate protein therapeutic candidates has particular value when a large number of related proteins need to be evaluated, as studies can be done on a high-throughput basis.

Drug Screening: The ability to induce a specific cell signaling, gene activation or protein secretion event in a cell allows the configuration of "targeted" cell-based screens in which the unique cell context of interest for drug design can be chemically induced. These screens very specifically search for drugs affecting cells in which a particular cell signaling or gene activation event has occurred. The tight regulation afforded by our ARGENT and RPD technologies means that highly specific screens can be set up, using the uninduced cell

line as a stringent counter-screen. Because the cellular event of interest can be induced chemically, the induction step can be configured into high-throughput screens.

Our Business Strategy

Our business strategy aims to balance near-term revenues from product partnering and technology licensing with independent product development and commercialization. Our goals are to:

- Enter into a partnership with a major pharmaceutical or biotechnology company to develop and commercialize our lead product candidate, AP23573, to treat cancer;
- Enter into partnerships with medical device companies to develop and commercialize our lead product candidate, AP23573, in drug-delivery stents to decrease reblockage of arteries following angioplasty and stenting;
- Independently develop as many of our product candidates as possible through at least phase 2 before partnering them;
- Establish the commercial infrastructure to market or co-market our anti-cancer product candidates in the United States;
- Enter into commercial partnerships for our other product candidates outside the United States;
- Permit broad use of our NF-κB and RegTech cell-signaling technologies at no cost by investigators at academic and not-for-profit institutions to conduct non-commercial research;
- License our NF-κB technology to pharmaceutical and biotechnology companies conducting research on the discovery of drugs that modulate NF-κB cell signaling and/or marketing such drugs;
- License our RegTech cell-signaling technology to pharmaceutical and biotechnology companies to accelerate their drug discovery; and
- Jointly develop product candidates incorporating our ARGENT cell-signaling regulation technology with companies that have proprietary therapeutic genes, cellular systems, or gene-delivery vectors.

Technology Licenses

We have a non-exclusive worldwide license agreement with Bristol-Myers Squibb Company, or Bristol, that grants Bristol the right to conduct pharmaceutical research and development covered by our NF-κB patents, retroactive to September 8, 1998. In addition to an upfront license fee, Bristol will pay us annual license fees as well as product development and commercialization milestones and royalties based on sales of products discovered using our patented NF-κB drug-discovery methods.

We have a non-exclusive license agreement with GPC Biotech AG, or GPC, that grants GPC the right to use our ARGENT cell-signaling regulation technology in GPC's three-hybrid technology platform. The agreement provides for guaranteed fees of $2.0 million, including $1.0 million upon signing. GPC will pay us a percentage of revenues received by GPC from its partnerships utilizing its three-hybrid drug discovery platform, and make development and commercialization milestone payments and royalty payments to us based on GPC products, if any, resulting from the use of our licensed technology in GPC's internal drug discovery programs.

Research and Development Spending

During each of the three years ended December 31, 2002, 2001 and 2000, respectively, we spent approximately $23.0 million, $16.6 million, and $12.5 million, respectively, on our research and development activities.

Manufacturing

When advantageous, we intend to rely on strategic partners or third-party contractors for manufacturing cGMP material to be used in our product candidates. We believe that our small-molecule drugs can be produced in commercial quantities through conventional synthetic and natural-product fermentation techniques. We expect to access manufacturing for viral vectors from potential partners and third-party manufacturers. Thus far, we have contracted with various commercial and academic entities to develop and optimize our manufacturing methods, but we have not entered into any formal manufacturing agreements adequate to produce our product candidates for large-scale clinical trials or commercial use.

Intellectual Property

Patents and other intellectual property rights are essential to our business. We file patent applications to protect our technology, inventions and improvements to our inventions that are considered important to the development of our business.

As of March 12, 2003, we have 113 patents and pending patent applications in the United States, of which 51 are owned, co-owned or exclusively licensed by us and 62 are owned, co-owned or exclusively licensed by our subsidiary, ARIAD Gene Therapeutics, Inc., or AGTI. In addition, we have filed foreign counterparts, as appropriate. We also have several nonexclusive technology licenses from certain institutions in support of our research programs. We anticipate that we will continue to seek licenses from universities and others where applicable technology complements our research and development efforts.

Many of the patents and patent applications in our portfolio cover our cell-signaling regulation technologies. These patents and pending applications cover regulatory technologies, specialized variants of the technologies, critical nucleic acid components, small-molecule drugs, the identification and use of dimerizer hormone mimetics, and various uses of the technologies in health care and drug discovery. Patents issued to date include 30 patents covering our cell-signaling regulation technologies. These patents issued in the United States beginning in November 1998 and should provide proprietary protection for our protein and cell therapy product candidates until at least 2015. We hope to obtain additional patents in the ensuing years based on pending applications.

Our patent portfolio also covers research tools and methods used in our drug discovery programs, as well as multiple classes of small-molecule drug candidates discovered in those programs. We also have a number of issued patents and pending applications relating to cell-signaling proteins and their use in drug discovery and therapeutics.

We also rely on unpatented trade secrets and proprietary know-how. However, trade secrets are difficult to protect. We enter into confidentiality agreements with our employees, consultants and collaborators. In addition, we believe that certain technologies utilized in our research and development programs are in the public domain. Accordingly, we do not believe that patent or other protection is available for these technologies. If a third party were to obtain patent or other proprietary protection for any of these technologies, we may be required to challenge such protections, obtain a license for such technologies or terminate or modify our programs that rely on such technologies.

ARIAD, ARGENT, RPD, RegTech are our trademarks.

Our Board of Scientific and Medical Advisors

We have assembled a Board of Scientific and Medical Advisors that currently consists of experts in the fields of molecular and cellular biology, biochemistry, immunology, and organic, physical, and computational chemistry, and molecular medicine. Each advisor is engaged under a consulting agreement that requires the advisor to provide consulting services to us in our field of interest and not to disclose any of our confidential information. Our Board of Scientific and Medical Advisors is chaired by Dr. Stuart L. Schreiber, Morris Loeb Professor and Chair, Chemistry and Chemical Biology; Co-Director, Institute of Chemistry and Cell Biology; and Director, Initiative for Chemical Genetics at Harvard University and an Investigator of the Howard Hughes Medical Institute. Dr. Schreiber is one of our scientific founders.

Our Licenses

We and our subsidiary, AGTI, have entered into license agreements with various research institutions and universities pursuant to which we and/or AGTI are the licensee of certain technologies upon which some of our product candidates are based.

We have agreed to pay royalties to our licensors on sales of certain products based on the licensed technologies, as well as, in some instances, milestone payments and patent filing and prosecution costs. The licenses also impose various milestones, commercialization, sublicensing, royalty as well as insurance and other obligations. Failure by us to comply with these requirements could result in the termination of the applicable agreement, which could have a material adverse effect on our business, financial condition, and results of operations.

Competition

The pharmaceutical and biotechnology industries are intensely competitive. We compete directly and indirectly with pharmaceutical companies, biotechnology companies and academic and research organizations, many of whom have greater resources than us. We compete with companies who have products on the market or in development for the same indications as our product candidates. We also complete with organizations that are developing similar technology platforms.

In the area of oncology, companies such as AstraZeneca PLC, Bristol-Myers Squibb Company, Eli Lilly and Company, Genentech, Inc., GlaxoSmithKline plc, Hoffmann LaRoche & Co., Merck KGaA, Novartis AG, Pfizer, Inc., and Wyeth Corp. are developing and marketing drugs to treat cancer. In the area of cell-signaling regulation, companies such as Amgen, Inc., AstraZeneca plc, Biogen, Inc., Eli Lilly and Company, Genentech, Inc., Imclone Systems, Inc., Ligand Pharmaceuticals, Inc., Millennium Pharmaceuticals, Inc., Novartis Pharma AG, OSI Pharmaceuticals, Inc., Tularik, Inc., and Vertex Pharmaceuticals, Inc. are developing and/or marketing drugs to treat human disease by inhibiting cell-signaling pathways. Companies developing gene-delivery technologies related to our programs include Avigen, Inc., Biogen, Inc., Cell Genesys, Inc., Genzyme Corp., MediGene, GmbH, and Targeted Genetics Corp. Certain companies have marketed erythropoietin products including Amgen, Inc., Johnson & Johnson, Transkaryotic Therapies, Aventis and Baxter International Inc., and other companies are developing products that may achieve a similar clinical effect. Several companies are developing products to treat GvHD, including AVAX, Inc., Protein Design Labs, Inc., and Repligen Corp. Other companies have products on the market or in development against which our products may have to compete. We may also experience competition from companies that have acquired or may acquire technology from companies, universities, and other research institutions. As these companies develop their technologies, they may develop proprietary positions that may materially and adversely affect us.

Government Regulation

Our ongoing research and development activities, our clinical trials, the manufacturing and testing procedures and the marketing of our product candidates, if they are approved, all are subject to extensive regulation by numerous governmental authorities in the United States and other countries. Any drug or device developed by us and/or a partner must undergo rigorous preclinical studies and clinical testing and extensive regulatory review administered by the United States Food and Drug Administration , or FDA, under the federal Food, Drug and Cosmetic Act prior to marketing in the United States. Satisfaction of such regulatory requirements, which includes demonstrating that a product is both safe and effective for its intended indications for use, typically takes several years or more depending upon the type, complexity and novelty of the product and requires the expenditure of substantial resources. Preclinical studies must be conducted in conformance with FDA regulations, including its current Good Laboratory Practices, or cGLP, regulations. Before commencing clinical trials in the United States, we must submit extensive information about the results of preclinical studies, toxicity, manufacturing and control procedures and our proposed clinical research protocol to the FDA in an Investigational New Drug application, or IND, or an Investigational Device Exemption, or IDE, as the case may be. If the FDA does not respond with any questions, we can commence clinical trials thirty days after the submission. There can be no assurance that submission of an IND or IDE will result in the commencement of clinical trials. In addition, an independent institutional review board, or IRB, at each institution at which a clinical trial is being performed, must review and approve the clinical protocol before clinical testing may begin, and it will have ongoing overview of the clinical trial at that institution.

In addition, certain clinical studies conducted in the United States involving gene transfer require the review and approval of the National Institutes of Health Recombinant DNA Advisory Committee, or the RAC. There can be no assurance that submission to the RAC will result in clearance to commence clinical trials. We have a limited history of conducting preclinical studies and the clinical trials necessary to obtain regulatory approval. Furthermore, we, the FDA or an IRB may suspend clinical trials at any time if we or they believe that the subjects participating in such trials are being exposed to unacceptable risks or if the FDA finds deficiencies in the conduct of the trials or other problems with our product under development.

Before receiving FDA approval to market a product, we will have to demonstrate that the product is safe and effective in the patients for whom the product is indicated. Data obtained from preclinical studies and clinical trials are susceptible to varying interpretations that could delay, limit or prevent regulatory clearances. In addition, delays or rejections may be encountered 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. Similar or even more extensive delays also may be encountered in foreign countries. There can be no assurance that even after such time and expenditures, regulatory approval will be obtained for any product candidates developed by us, or, even if approval is obtained, that the approved indication and related labeling for such products will not limit the product's condition of use, which could materially impact the marketability and profitability of the product. If regulatory approval of a product is granted, such approval will be limited to those disease states and conditions for which the product has been shown useful, as demonstrated by clinical trials. Furthermore, approval may entail ongoing requirements for post-market studies. Even if such regulatory approval is obtained, a marketed product, its manufacturer and its manufacturing facilities and procedures are subject to continual review and periodic inspections by the FDA. Discovery of previously unknown problems with a product, manufacturer, manufacturing procedures or facility may result in restrictions on such product or manufacturer, including costly recalls, an injunction against continued marketing and manufacturing until the problems have been adequately addressed to the FDA's satisfaction or even withdrawal of the product from the market.

There can be no assurance that any compound developed by us alone or in conjunction with others will prove to be safe and efficacious in clinical trials and will meet all of the applicable regulatory requirements

needed to receive and maintain marketing approval. Additionally, the marketing, labeling and advertising for an approved product is subject to ongoing FDA scrutiny and the failure to adhere to applicable requirements can result in regulatory action that could have a material adverse impact on the profitability of the product.

Outside the United States, our ability to market a product will be contingent upon receiving a marketing authorization from the appropriate regulatory authorities. The requirements governing the conduct of clinical trials, obtaining marketing authorization, and pricing and reimbursement vary widely from country to country. At present, foreign marketing authorizations are applied for at a national level, although within the European Community, certain registration procedures are available to companies wishing to market a product in more than one Member State. If the regulatory authority is satisfied that adequate evidence of safety, quality and efficacy has been presented, a marketing authorization will be granted. This foreign regulatory approval process includes all of the risks associated with FDA clearance set forth above.

Our Employees

As of March 12, 2003, we had 52 employees, 28 of whom hold post-graduate degrees, including 18 with a Ph.D., M.D. or J.D. Most of our employees are engaged directly in research and development. We have entered into confidentiality and noncompetition agreements with all of our employees. None of our employees are covered by a collective bargaining agreement, and we consider relations with our employees to be good.

RISK FACTORS

THE RISKS AND UNCERTAINTIES DESCRIBED BELOW ARE THOSE THAT WE CURRENTLY BELIEVE MAY MATERIALLY AFFECT OUR COMPANY. ADDITIONAL RISKS AND UNCERTAINTIES THAT WE ARE UNAWARE OF OR THAT WE CURRENTLY DEEM IMMATERIAL ALSO MAY BECOME IMPORTANT FACTORS THAT AFFECT OUR COMPANY.

Risks Relating to Our Business

Insufficient funding may jeopardize our research and development programs and may prevent commercialization of our products and technologies.

We have funded our operations to date through sales of equity securities, debt and operating revenue. Most of our operating revenue to date has been generated through previous collaborative research and development agreements. We do not have any committed funding from any pharmaceutical company to advance any of our product development programs. Although we intend to seek additional funding from product-based collaborations, technology licensing, and public or private financings, additional funding may not be available on terms acceptable to us, or at all. Accordingly, we may not be able to secure the significant funding which is required to maintain and continue each of our research and development programs at their current levels or at levels that may be required in the future. If we cannot secure adequate financing, we may be required to delay, scale back or eliminate one or more of our research and development programs or to enter into license or other arrangements with third parties to commercialize products or technologies that we would otherwise seek to develop ourselves.

We may never succeed in developing marketable drugs or generating product revenues.

We are a development stage company, and our main focus is drug discovery and product development. We do not currently have any product revenues, and we may not succeed in developing or commercializing any products which will generate product revenues. We do not expect to have any products on the market for several years, if at all. We are exploring human diseases at the cellular level and attempting to develop

product candidates that intervene with these processes. As with all science, we face much trial and error, and we may fail at numerous stages along the way. If we are not able to enter into agreements with one or more companies experienced in the development and manufacture of gene-transfer vectors, we will not be able to market our gene and cellular therapy experienced product candidates. If we are not able to enter into agreements with one or more medical device companies experienced in the development, manufacture, and marketing of vascular stents, we will not be able to generate product revenues from the marketing of vascular stents that deliver AP23573. If we are not successful in developing or marketing our product candidates, we will not be profitable.

We have incurred significant losses to date and may never be profitable.

We have incurred significant operating losses in each year since our formation in 1991 and have an accumulated deficit of approximately $136.3 million from our operations through December 31, 2002. Losses have resulted principally from costs incurred in research and development of product candidates and from general and administrative costs associated with our operations. It is likely that we will incur significant operating losses for the foreseeable future. We currently have no product revenues and limited commitments for future licensing revenues, and may never be able to generate such revenues. If our losses continue and we are unable to successfully develop, commercialize, manufacture and market our product candidates and/or to enter into agreements and licenses of our intellectual property, we may never generate sufficient revenues to achieve profitability. Even if we are able to commercialize any of our product candidates or enter into agreements or licenses in the future, we may never generate sufficient revenues to have profitable operations.

We have no experience in manufacturing any of our product candidates, which raises uncertainty as to our ability to develop and commercialize our product candidates.

We have no experience in, and currently lack the resources and capability to, manufacture any of our product candidates on a large scale. Our ability to conduct clinical trials and commercialize our product candidates will depend, in part, on our ability to manufacture our products on a large scale, either directly or through third parties, at a competitive cost and in accordance with cGMP and other regulatory requirements. We depend on third-party manufacturers or collaborative partners for the production of our product candidates for preclinical studies and clinical trials and intend to use third-party manufacturers to produce any products we may eventually commercialize. We have no experience in developing, manufacturing, or marketing drug-delivery vascular stents, and we will be completely dependent on our medical device partners, if any, to conduct these activities. If we are not able to obtain contract manufacturing on commercially reasonable terms and obtain or develop the necessary technologies for manufacturing, we may not be able to conduct or complete clinical trials or commercialize our product candidates, and we do not know whether we will be able to develop such capabilities. If we are not able to develop cell processing methods that comply with regulatory guidelines known as current Good Tissue Practices, or cGTP, we may not be able to commercialize our regulated cellular therapy product.

Significant additional losses or insufficient funding may cause us to default on certain covenants of our loan documents.

At December 31, 2002, we had $6.9 million in term notes payable with two financial institutions of which $1.5 million is payable within twelve months and classified as a current liability. On March 12, 2003, we entered into a new $7.5 million term loan agreement with a bank and paid off the above-mentioned term notes. Under our new term loan agreement, we are required to maintain certain financial and non-financial covenants, including minimum cash and cash equivalent balances of $10.0 million, a default of any of which would allow the bank to demand payment of its loan. We currently maintain sufficient cash balances to fund payment of this loan if demand for payment were made. However, if we are unable to raise adequate financing to fund continuing operations or otherwise to refinance our loan, we may not be able to maintain

compliance with loan covenants, may be required to pay off the loan and may be required to reduce our spending on operations.

We may expend significant capital resources on the enforcement and licensing of our NF-κB patent portfolio and be unable to generate revenues from these efforts, if we are unable to enforce or license our patents to pharmaceutical and biotechnology companies.

We are the exclusive licensee of a family of patents, three in the U.S. and one in Europe, including a pioneering U.S. patent covering methods of treating human disease by regulating NF-κB cell-signaling activity, or the NF-κB '516 Patent, awarded to a team of inventors from the Whitehead Institute for Biomedical Research, Massachusetts Institute of Technology and Harvard University. We have initiated a licensing program to generate revenues from the discovery, development, manufacture and sale of products covered by our NF-κB patent portfolio. These patents may be challenged and subsequently narrowed, invalidated or circumvented, which would materially impact our ability to generate licensing revenues from them.

On June 25, 2002, we, together with these academic institutions, filed a lawsuit in the United States District Court for the District of Massachusetts, or the U.S. District Court, against Eli Lilly and Company, or Lilly alleging infringement upon issuance of certain claims of the NF-κB '516 Patent, or the NF-κB '516 Claims, through sales of Lilly's osteoporosis drug, Evista®, and its septic shock drug, Xigris®. On August 26, 2002, Lilly filed in the U.S. District Court a motion to dismiss or, alternatively, for summary judgment, or Lilly's Combined Motion, challenging the validity of the NF-κB '516 Claims. We filed a response to Lilly's Combined Motion on October 17, 2002, and Lilly filed a reply on November 17, 2002. Oral argument on Lilly's Combined Motion was heard in the U.S. District Court on November 21, 2002. As of March 12, 2003, the U.S. District Court had not yet ruled on Lilly's Combined Motion. If the NF-κB '516 Claims are invalidated, it could have a significant adverse impact on our ability to generate revenues from our NF-κB licensing program. As exclusive licensee of this patent, we are obligated for the costs expended for its enforcement. Accordingly, we anticipate expending significant capital and management resources pursuing this litigation for an indeterminate period, and the outcome is uncertain. Significant expenditures to enforce these patent rights without generating revenues or accessing additional capital could adversely impact our ability to further our research and development programs at the current levels or at levels that may be required in the future.

Because we do not own all of the outstanding stock of our subsidiary, ARIAD Gene Therapeutics, Inc., or AGTI, we may not realize all of the potential future economic benefit from products developed based on technology licensed to or owned by our subsidiary.

Our subsidiary, AGTI, holds licenses from Harvard University, Stanford University and other universities relating to our ARGENT cell-signaling regulation technology, a key component of our programs in regulated protein therapy and cellular therapy, and owns the intellectual property on our mTOR inhibitors derived from our ARGENT programs. The two directors of AGTI are also members of the Board of Directors of the Company. Minority stockholders of AGTI, including Harvard University, Stanford University, several of our scientific advisors, and several current and former members of our management and Board of Directors, own 20% of the issued and outstanding capital stock of AGTI. We own the remaining 80% of the issued and outstanding capital stock of AGTI.

We do not currently have a license agreement with AGTI that provides us with rights to commercialize product candidates based on our ARGENT cell-signaling regulation technology or mTOR inhibitors derived from our ARGENT programs In the event that we commercialize product candidates based on our ARGENT cell-signaling regulation technology or mTOR inhibitors derived from our ARGENT programs, we will have to negotiate the terms of a license or other agreements with AGTI on terms to be determined or

acquire all of the capital stock of AGTI that we do not currently own. The economic benefit to our stockholders from such products, if any, that we commercialize will be diminished by any royalties or other payments paid under a future agreement, if any, with AGTI. The economic benefit to our stockholders from products, if any, would be reduced in an amount based on such future agreements and the percentage owned by the minority stockholders of AGTI.

Alternatively, we may acquire all of the interests of the minority stockholders in AGTI for cash, shares of our common stock or other securities, if any. AGTI has a right of first refusal on the sale to third parties of 73% of the minority stockholders' AGTI shares. AGTI does not have a call option, or a right to require the minority stockholders to sell their shares to us, for any of these shares. If we acquire these minority interests, it may result in dilution to our stockholders. The economic value of the minority stockholders' interests is difficult to quantify in the absence of a public market, and the market price of our publicly traded common stock may not accurately reflect its value. Accordingly, the market could change its perception of the value of these minority interests in our subsidiary at any time in reaction to our increased emphasis on these product candidates, announcements regarding these product candidates or for other reasons, any of which could result in a decline in our stock price. In addition, if we acquire the minority interests at a cost greater than the value attributed to them by the market, this also could result in a decline in our stock price. If we choose to acquire these minority interests through a short-form merger in which we do not solicit the consent of the minority stockholders of AGTI, we could become subject to an appraisal procedure, which would result in additional expense and diversion of management resources.

Because members of our management team and/or Board of Directors beneficially own a material percentage of the capital stock of our subsidiary, AGTI, and we have agreements with AGTI, there may be conflicts of interest present in dealings between ARIAD and AGTI.

Four members of our management team and/or Board of Directors own or have the right to acquire up to approximately 6.1% of the outstanding capital stock of AGTI. Harvey J. Berger, M.D., our Chairman, Chief Executive Officer and President, owns 3.4%, David L. Berstein, Esq., our Senior Vice President and Chief Patent Counsel, owns 0.3%, John D. Iuliucci, Ph.D., our Senior Vice President, Drug Development, owns 0.7% and Jay R. LaMarche, one of our directors and a part-time employee, owns 1.7%. These same individuals beneficially own approximately 7.0% of our outstanding common stock. Additionally, Dr. Berger, and Mr. LaMarche, are the two members comprising the Board of Directors of AGTI. As part of the formation of AGTI, we entered into certain agreements with AGTI to provide for the operations of AGTI. As a result, the market may perceive conflicts of interest to exist in dealings between AGTI and us. AGTI is the exclusive licensee of the ARGENT cell-signaling intellectual property from Harvard University and Stanford University and of related technologies from other universities, and owns the intellectual property on our mTOR inhibitors derived from our ARGENT programs. Because of the apparent conflicts of interest, the market may be more inclined to perceive the terms of any transaction between us and AGTI as being unfair to us.

The loss of key members of our scientific and management staff could delay and may prevent the achievement of our research, development and business objectives.

Our Chairman, Chief Executive Officer and President, Harvey J. Berger, M.D.; our Senior Vice President and Chief Patent Counsel, David L. Berstein, Esq.; our Senior Vice President, Drug Development, John D. Iuliucci, Ph.D.; our Senior Vice President and Chief Business Officer, Fritz Casselman; our Senior Vice President, Science and Technology, Timothy P. Clackson, Ph.D.; and other key officers and members of our scientific staff responsible for areas such as drug development, clinical trials, regulatory affairs, drug discovery, manufacturing and intellectual property protection and licensing are important to our specialized scientific business. We also are dependent upon a few of our scientific advisors to assist in formulating our research and development strategy. The loss of, and failure to promptly replace, any member of our

management team could significantly delay and may prevent the achievement of our research, development and business objectives. While we have entered into employment agreements with all of our officers, these officers may not remain with us.

We may not be able to protect our intellectual property relating to our research programs, technologies and products.

We and our licensors have issued patents and pending patent applications covering research methods useful in drug discovery, new chemical compounds discovered in our drug discovery programs, certain components, configurations and uses of our cell-signaling regulation technologies, products-in-development, and methods and materials for conducting pharmaceutical research. We have an ongoing licensing program to generate revenues from the use of our cell-signaling regulation technologies and our NF-κB intellectual property. Pending patent applications may not issue as patents and may not issue in all countries in which we develop, manufacture or sell our products or in countries where others develop, manufacture and sell products using our technologies. In addition, patents issued to us or our licensors may be challenged and subsequently narrowed, invalidated or circumvented. In that event, such patents may not afford meaningful protection for our technologies or product candidates, which would materially impact our ability to develop and market our product candidates and to generate licensing revenues from our gene regulation patent portfolio. Certain technologies utilized in our research and development programs are already in the public domain. Moreover, a number of our competitors have developed technologies, filed patent applications or obtained patents on technologies and compositions that are related to our business and may cover or conflict with our patent applications. Such conflicts could limit the scope of the patents that we may be able to obtain or may result in the denial of our patent applications. If a third party were to obtain intellectual proprietary protection for any of these technologies, we may be required to challenge such protections, terminate or modify our programs that rely on such technologies or obtain licenses for use of these technologies.

We may be unable to develop or commercialize our product candidates, if we are unable to obtain or maintain certain licenses.

We have entered into license agreements for some of our technologies, either directly or through AGTI. We are currently attempting to obtain additional licenses for technology useful to our programs. Our inability to obtain any one or more of these licenses, on commercially reasonable terms, or at all, or to circumvent the need for any such license, could cause significant delays and cost increases and materially affect our ability to develop and commercialize our product candidates. We also use gene sequences or proteins encoded by those sequences and other biological materials in each of our research programs which are, or may become, patented by others and to which we would be required to obtain licenses in order to develop or market our product candidates. Some of our programs, including, for example, our regulated protein therapy program, may require the use of multiple proprietary technologies, especially gene-transfer vectors and therapeutic genes. Obtaining licenses for these technologies may require us to make cumulative royalty payments or other payments to several third parties, potentially reducing amounts paid to us or making the cost of our products commercially prohibitive. Manufacturing of gene-transfer vectors may also require licensing technologies and intellectual property from third parties.

Some of our licenses obligate us to exercise diligence in pursuing the development of product candidates, to make specified milestone payments and to pay royalties. In some instances, we are responsible for the costs of filing and prosecuting patent applications. These licenses generally expire upon the earlier of a fixed term of years after the date of the license or the expiration of the applicable patents, but each license is also terminable by the other party upon default by us of our obligations. Our inability or failure to meet our diligence requirements or make any payments required under these licenses would result in a reversion to the licensor of the rights granted which, with respect to the licenses pursuant to which we have obtained

exclusive rights, would materially and adversely affect our ability to develop and market products based on our licensed technologies.

We may be unable to access or manufacture vectors or other gene-transfer technologies that we will need to develop and commercialize our regulated protein and cellular therapy product candidates.

We may not be able to access the gene-transfer technologies required to develop, manufacture, and commercialize our regulated protein therapy and cellular therapy product candidates. We are reliant on our ability to enter into license agreements with academic institutions, gene-therapy companies and/or contract manufacturers that can provide us with rights to the necessary technology, production methods, and components of gene-delivery systems. The inability to reach an appropriate agreement with such an entity on reasonable commercial terms could delay or prevent the preclinical evaluation, clinical testing and/or commercialization of our product candidates. Our inability to access gene-transfer technology, including suitable manufacturing methods, would have significant adverse effects on some of our product candidates, including their development timelines. If we do not market our product candidates, we will never become profitable. In addition, the intellectual property landscape covering gene-transfer technologies is uncertain and fragmented. Accordingly, if we select one partner as a source for selected intellectual property rights, we may find that we have not licensed sufficient rights to be able to commercialize our products or we may be forced to acquire additional rights or discontinue marketing our product candidates unexpectedly.

Competing technologies may render some or all of our programs or future products noncompetitive or obsolete.

Many well-known pharmaceutical, healthcare and biotechnology companies, academic and research institutions and government agencies, which have substantially greater capital, research and development capabilities and experience than us, are presently engaged in one or more of the following activities:

- Developing products based on cell signaling, genomics, proteomics, computational chemistry and protein and cellular therapies;
- Conducting research and development programs for the treatment of each of the disease areas in which we are focused; and
- Manufacturing, promoting, marketing and selling pharmaceutical or medical device products for treatment of diseases in all of the disease areas in which we are focused.

Some of these entities already have product candidates in clinical trials or in more advanced preclinical studies than we do. These entities may succeed in commercializing competitive products before us, which would give them a competitive advantage. Competing technologies may render some or all of our programs or future products noncompetitive or obsolete, and we may not be able to make the enhancements to our technology necessary to compete successfully with newly emerging technologies. If we are unable to successfully compete in our chosen markets, we will not become profitable.

If our product candidates are not accepted by physicians and insurers, we will not be successful.

Our success is dependent on the acceptance of our product candidates. Our product candidates may not achieve significant market acceptance among patients, physicians or third-party payors, even if we obtain necessary regulatory and reimbursement approvals. Failure to achieve significant market acceptance of our product candidates will harm our business. We believe that recommendations by physicians and health care payors will be essential for market acceptance of any product candidates. There is continued concern in the marketplace regarding the potential safety and effectiveness of gene therapy products generally. Physicians and health care payors may conclude that any of our product candidates are not safe.

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

We currently have no sales, marketing or distribution capabilities. If we are unable to establish sales, marketing or distribution capabilities either by developing our own sales, marketing and distribution organization or by entering into agreements with others, we may be unable to successfully sell any products that we are able to begin to commercialize. If we are unable to effectively sell our products, our ability to generate revenues will be harmed. We may not be able to hire, in a timely manner, the qualified sales and marketing personnel we need, if at all. In addition, we may not be able to enter into any marketing or distribution agreements on acceptable terms, if at all. If we cannot establish sales, marketing and distribution capabilities as we intend, either by developing our own capabilities or entering into agreements with third parties, sales of future products, if any, may be harmed.

If we develop a product for commercial use, a subsequent product liability-related claim or recall could have an adverse effect on our business.

Our business exposes us to potential product liability risks inherent in the testing, manufacturing and marketing of pharmaceutical products, and we may not be able to avoid significant product liability exposure. A product liability-related claim or recall could be detrimental to our business. In addition, except for insurance covering product use in our clinical trials, we do not currently have any product liability insurance, and we may not be able to obtain or maintain such insurance on acceptable terms, or we may not be able to obtain any insurance to provide adequate coverage against potential liabilities. Our inability to obtain sufficient insurance coverage at an acceptable cost or otherwise to protect against potential product liability claims could prevent or limit the commercialization of any products that we develop.

Risks Relating to Governmental Approvals

We have limited experience in conducting clinical trials, which may cause delays in commencing and completing clinical trials of our product candidates.

Clinical trials must meet FDA and foreign regulatory requirements. We have limited experience in conducting and managing the preclinical studies and clinical trials necessary to obtain regulatory approval for our product candidates in any country. We may encounter problems in clinical trials that may cause us or the FDA or foreign regulatory agencies to delay, suspend or terminate our clinical trials at any phase. These problems could include the possibility that we may not be able to manufacture sufficient quantities of cGMP materials for use in our clinical trials, conduct clinical trials at our preferred sites, enroll a sufficient number of patients for our clinical trials at one or more site or begin or successfully complete clinical trials in a timely fashion, if at all. Furthermore, we, the FDA or foreign regulatory agencies may suspend clinical trials at any time if we or they believe the subjects participating in the trials are being exposed to unacceptable health risks or if we or they find deficiencies in the clinical trial process or conduct of the investigation. If clinical trials of any of our product candidates fail, we will not be able to market the product candidate which is the subject of the failed clinical trials. The FDA and foreign regulatory agencies could also require additional clinical trials, which would result in increased costs and significant development delays. Our failure to adequately demonstrate the safety and effectiveness of a pharmaceutical product candidate under development could delay or prevent regulatory approval of the product candidate and could have a material adverse effect on our business.

We may not be able to obtain government regulatory approval for our product candidates prior to marketing.

To date, we have not submitted a marketing application for any product candidate to the FDA or any foreign regulatory agency, and none of our product candidates have been approved for commercialization in any

country. Prior to commercialization, each product candidate would be subject to an extensive and lengthy governmental regulatory approval process in the United States and in other countries. We may not be able to obtain regulatory approval for any product candidate we develop or even if approval is obtained, the labeling for such products may place restrictions on their use that could materially impact the marketability and profitability of the product subject to such restrictions. We have limited experience in conducting and managing the clinical testing necessary to obtain such regulatory approval. Satisfaction of these regulatory requirements, which includes satisfying the FDA and foreign regulatory authorities that the product is both safe and effective for its intended therapeutic uses, typically takes several years or more depending upon the type, complexity and novelty of the product and requires the expenditure of substantial resources.

Furthermore, the regulatory requirements governing our product candidates are uncertain. In particular, the FDA and other regulatory agencies have suspended certain gene and cellular therapy clinical trials due to concerns about the potential safety of certain gene-transfer vectors, which may lead to additional regulatory requirements for such products. Uncertainty with respect to the regulatory requirements for all of our product candidates may result in excessive costs or extensive delays in the regulatory approval process, adding to the already lengthy review process. If regulatory approval of a product is granted, such approval will be limited to those disease states and conditions for which the product is proven safe and effective, as demonstrated by clinical trials, and our products will be subject to ongoing regulatory reviews. Although we have been granted orphan drug designation by the FDA for AP1903, the small-molecule drug used in our graft-vs-host disease cellular therapy product candidate, this designation may be challenged by others or may prove to be of no practical benefit.

We will not be able to sell our product candidates, if we or our third-party manufacturers fail to comply with FDA manufacturing regulations.

Before we can begin to commercially manufacture our product candidates, we must either secure manufacturing in an approved manufacturing facility or obtain regulatory approval of our own manufacturing facility and processes. In addition, the manufacturing of our product candidates must comply with cGMP and/or cGTP requirements of the FDA and requirements by regulatory agencies in other countries. These requirements govern, among other things, quality control and documentation procedures. We, or any third-party manufacturer of our product candidates, may not be able to comply with these requirements, which would prevent us from selling such products. Material changes to the manufacturing processes of our products after approvals have been granted are also subject to review and approval by the FDA or other regulatory agencies.

Even if we bring products to market, we may be unable to effectively price our products or obtain adequate reimbursement for sales of our products, which would prevent our products from becoming profitable.

If we succeed in bringing our product candidates to the market, they may not be considered cost-effective, and coverage and adequate payments may not be available or may not be sufficient to allow us to sell our products on a competitive basis. In both the United States and elsewhere, sales of medical products and treatments are dependent, in part, on the availability of reimbursement from third-party payors, such as health maintenance organizations and other private insurance plans and governmental programs such as Medicare. Third-party payors are increasingly challenging the prices charged for pharmaceutical products and services. Our business is affected by the efforts of government and third-party payors to contain or reduce the cost of health care through various means. In the United States, there have been and will continue to be a number of federal and state proposals to implement government controls on pricing. In addition, the emphasis on managed care in the United States has increased and will continue to increase the pressure on the pricing of pharmaceutical products. We cannot predict whether any legislative or regulatory proposals will be adopted or the effect these proposals or managed care efforts may have on our business.

Risks Relating to Our Common Stock

Results of our operations and general market conditions for biotechnology stocks could result in the sudden change in the value of our stock.

As a biopharmaceutical company, we have experienced significant volatility in our common stock. Fluctuations in our operating results and general market conditions for biotechnology stocks could have a significant impact on the volatility of our common stock price. During 2002, our stock price ranged from a high bid price of $6.25 to a low bid price of $1.58. Factors contributing to such volatility include: results and timing of preclinical studies and clinical trials; evidence of the safety or effectiveness of pharmaceutical products; announcements of new collaborations; failure to enter into collaborations; our funding requirements; announcements of technological innovations or new therapeutic products; developments relating to intellectual property rights, including licensing and litigation, including our litigation with Eli Lilly and Company; governmental regulation; policies regarding recombinant DNA and gene therapy; healthcare or cost-containment legislation; general market trends for the biotechnology industry and related high-technology industries; the impact of changing interest rates and policies of the Federal Reserve; and public policy pronouncements.

ITEM 2: PROPERTIES

We have leased approximately 100,000 square feet (approximately 37,000 square feet currently under sublease to third parties) of laboratory and office space at 26 Landsdowne Street, located at University Park at Massachusetts Institute of Technology in Cambridge, Massachusetts. The lease originally had a ten-year term, which ended in July of 2002, with two consecutive five-year renewal options. We have extended the lease for the first five-year option period through July of 2007. We believe that our currently leased facility will, in large part, be adequate for our research and development activities at least through the year 2007.

ITEM 3: LEGAL PROCEEDINGS

We were named as a defendant in a purported class action lawsuit commenced in June 1995 in the U.S. District Court for the Southern District of New York. The action named as defendants ARIAD Pharmaceuticals, Inc.; the underwriter of our initial public offering and a market maker in our stock, D. Blech & Co.; the managing director and sole shareholder of D. Blech & Co. and one of our former directors, David Blech; certain other of our directors, and the qualified independent underwriter for the initial public offering, Shoenberg Hieber, Inc., or SHI.

Counsel for the plaintiff class, counsel for the named director defendants of the Company (excluding David Blech), or the Company Defendants, and the Company, and counsel for SHI have executed a stipulation of settlement in the action, or the Settlement. The Settlement was approved pursuant to entry by the Court of a Final Order and Judgment on December 4, 2002. The Final Order and Judgment orders that a payment of $620,000 (of which our contribution was not material and no liability was recorded on the 2002 balance sheet) be distributed to the plaintiffs from a legal escrow account, that this action and a related action entitled In re: Blech Securities Litigation, 94 Civ. 7696 (RWS) are dismissed with prejudice, and that non-settling parties are barred from pursuing contribution-type claims against the Company Defendants.

On May 19, 1999, we filed suit in the Massachusetts Superior Court against Michael Z. Gilman, Ph.D., or Dr. Gilman, our former Chief Scientific Officer, seeking equitable relief for breach of his employment agreements in accepting a position as the research director of molecular biology at Biogen, Inc., or Biogen. The Superior Court issued a temporary injunction on May 19, 1999 restraining Dr. Gilman from using any of our confidential information in his new employment. On June 21, 1999, Dr. Gilman filed counterclaims against us seeking an order awarding damages for breach of contract and barring us from enforcing any provisions of our employment agreements with Dr. Gilman. On May 26, 1999, Biogen filed a motion to intervene as a

defendant in the action which the Superior Court granted on August 2, 1999. Discovery in the case has been completed, and Summary Judgment Motions have been filed, heard and ruled upon.

Counsel for us, counsel for Biogen and counsel for Dr. Gilman have executed a stipulated partial judgment, or the Stipulated Judgment, which was approved pursuant to entry by the Court on January 13, 2003. The Stipulated Judgment dismisses with prejudice our claim for breach of contract against Dr. Gilman and dismisses Biogen as a party to the action. Dr. Gilman's counterclaims will now proceed to trial which we anticipate will be set in 2003. The ultimate outcome of the litigation with Dr. Gilman is not determinable at this time, and as a result, we cannot estimate whether any damages will be awarded or what the range of such an award might be and have not recorded any liability on the December 31, 2002 balance sheet.

On June 25, 2002, we, together with Massachusetts Institute of Technology, The Whitehead Institute for Biomedical Research and Harvard University, filed a lawsuit in the United States District Court for the District of Massachusetts, or the U.S. District Court, against Eli Lilly and Company, or Lilly, alleging infringement upon issuance of certain claims of our U.S. patent covering methods of treating human disease by regulating NF-κB cell-signaling activity, or the NF-κB '516 Claims, through sales of Lilly's osteoporosis drug, Evista®, and Lilly's septic shock drug, Xigris®, and seeking monetary damages from Lilly. On August 26, 2002, Lilly filed a motion to dismiss or, alternatively, for summary judgment, or Lilly's Combined Motion, challenging the validity of the NF-κB '516 Claims. We filed a response to Lilly's Combined Motion on October 17, 2002 and Lilly filed a reply on November 17, 2002. Oral argument on Lilly's Combined Motion was heard in the U.S. District Court on November 21, 2002. As of March 12, 2003, the U.S. District Court had not yet ruled on Lilly's Combined Motion. While the ruling on Lilly's Combined Motion is not currently determinable, if Lilly were to be successful and its Combined Motion is granted, we will consider filing an appeal with the Court of Appeals for the Federal Circuit. If Lilly's Combined Motion is denied, a trial scheduling conference pursuant to Rule 16(b) of the Federal Rules of Civil Procedure will be scheduled by the U.S. District Court, and the case will proceed to the discovery phase leading to trial. The ultimate outcome of the litigation cannot be determined at this time, and, as a result, an estimate of a damage award or range of awards, if any, cannot be made.

ITEM 4: SUBMISSION OF MATTERS TO A VOTE OF SECURITY HOLDERS

No matters were submitted to a vote of security holders during the quarter ended December 31, 2002.

PART II

ITEM 5: MARKET FOR REGISTRANT'S COMMON EQUITY AND RELATED STOCKHOLDER MATTERS

Market Information

Our common stock is traded on the Nasdaq National Market under the symbol "ARIA". The following table sets forth the high and low sales prices of our common stock as quoted on the Nasdaq National Market for the periods indicated.

	High	Low
2002:		
First Quarter	$5.65	$3.32
Second Quarter	6.25	3.55
Third Quarter	4.47	2.75
Fourth Quarter	4.13	1.58
2001:		
First Quarter	$8.38	$2.78
Second Quarter	6.84	3.80
Third Quarter	5.40	1.66
Fourth Quarter	6.40	2.41

On March 12, 2003, the last sale price of our common stock was $1.35.

Stockholders

The approximate number of holders of record of our common stock as of March 4, 2003 was 478, and the approximate total number of beneficial holders of our common stock as of March 4, 2003 was 25,000.

Dividends

We have not declared or paid dividends on our common stock in the past and do not intend to declare or pay such dividends in the foreseeable future. Our long-term debt agreement prohibits the payment of cash dividends. (See "Management's Discussion and Analysis of Financial Condition and Results of Operations – Liquidity and Capital Resources" and Note 4 of "Notes to Consolidated Financial Statements.")

ITEM 6: SELECTED FINANCIAL DATA

The selected financial data set forth below as of December 31, 2002, 2001, 2000, 1999 and 1998 and for each of the years then ended have been derived from the audited consolidated financial statements of the Company, of which the financial statements as of December 31, 2002 and 2001 and for the years ended December 31, 2002, 2001 and 2000 are included elsewhere in this Annual Report on Form 10-K, and are qualified by reference to such financial statements. The information set forth below should be read in conjunction with "Management's Discussion and Analysis of Financial Condition and Results of Operations" and the audited consolidated financial statements, and the notes thereto, and other financial information included herein.

	Years Ended December 31,				
In thousands, except share and per share data	2002	2001	2000	1999	1998
Consolidated Statements of Operations Data:					
Research revenue (principally related parties prior to 2000)	$ 67	$ 4	$ 128	$ 12,468	$ 12,143
Operating expenses:					
Research and development	23,018	16,587	12,467	28,844	35,515
General and administrative	5,718	4,469	3,318	3,938	2,634
Total operating expenses	28,736	21,056	15,785	32,782	38,149
Loss from operations	(28,669)	(21,052)	(15,657)	(20,314)	(26,006)
Other income (expense):					
Interest income	615	1,578	2,050	445	999
Interest expense	(323)	(285)	(225)	(522)	(481)
Other income – tax refund	534				
Gain on sale of Genomics Center				46,440	
Equity in net loss of Genomics Center				(1,493)	(660)
Total other income (expense), net	826	1,293	1,825	44,870	(142)
Income (loss) before cumulative effect of change in accounting principle	(27,843)	(19,759)	(13,832)	24,556	(26,148)
Cumulative effect of change in accounting principle				(364)	
Net income (loss)	(27,843)	(19,759)	(13,832)	24,192	(26,148)
Repurchase and accretion costs attributable to redeemable convertible preferred stock				(6,435)	(36)
Net income (loss) attributable to common stockholders	$ (27,843)	$ (19,759)	$ (13,832)	$ 17,757	$ (26,184)
Earnings (loss) per share:					
Per common share (basic):					
Income (loss) attributable to common stockholders before cumulative effect of change in accounting principle	$ (.86)	$ (.68)	$ (.53)	$ .82	$ (1.25)
Cumulative effect of change in accounting principle				(.02)	
Net income (loss) – basic	$ (.86)	$ (.68)	$ (.53)	$ .80	$ (1.25)
Weighted average number of shares of common stock outstanding – basic	32,475,083	29,256,767	25,875,663	22,004,646	20,966,586
Per common share (diluted):					
Income (loss) before cumulative effect of change in accounting principle	$ (.86)	$ (.68)	$ (.53)	$ .71	$ (1.25)
Cumulative effect of change in accounting principle				(.01)	
Net income (loss) – diluted	$ (.86)	$ (.68)	$ (.53)	$ .70	$ (1.25)
Weighted average number of shares of common stock					

outstanding – diluted	32,475,083	29,256,767	25,875,663	34,448,015	20,966,586

In thousands	As of December 31, 2002	2001	2000	1999	1998
Consolidated Balance Sheet Data:					
Cash, cash equivalents and marketable securities	$ 26,850	$ 47,186	$ 39,781	$ 28,320	$ 14,176
Working capital	21,126	43,249	37,165	22,875	5,852
Total assets	35,104	55,361	48,813	44,236	30,786
Long-term debt	5,437	6,847	3,700	1,900	3,295
Redeemable convertible preferred stock				8,070	5,036
Accumulated deficit	(136,317)	(108,474)	(88,715)	(74,883)	(92,640)
Stockholders' equity	21,852	43,093	40,851	27,068	11,733

ITEM 7: MANAGEMENT'S DISCUSSION AND ANALYSIS OF FINANCIAL CONDITION AND RESULTS OF OPERATIONS

You should read the following discussion and analysis in conjunction with "Selected Financial Data" and our consolidated financial statements and the related notes included elsewhere in this report.

Overview

We are engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo,* that may be used to treat diseases in innovative ways. We are developing a comprehensive approach to the treatment of cancer and are primarily focused on a series of product candidates for targeted oncology indications. We have an exclusive license to pioneering technology and patents related to the discovery, development, and use of drugs that regulate NF-κB cell-signaling activity, which has been implicated in many major diseases.

General

Since our inception in 1991, we have devoted substantially all of our resources to our research and development programs. We receive no revenue from the sale of pharmaceutical products, and most of our revenue to date has been received in connection with our past relationship with Aventis Pharmaceuticals, Inc. ("Aventis"). Except for the gain on the sale of our fifty percent interest in the Hoechst-ARIAD Genomics Center LLC (the "Genomics Center") to Aventis in December 1999, which resulted in net income for fiscal 1999, we have not been profitable since inception. We expect to incur substantial operating losses for the foreseeable future, primarily due to costs associated with our pharmaceutical product development programs, clinical trials, and product manufacturing. We expect that losses will fluctuate from quarter to quarter and that these fluctuations may be substantial. As of December 31, 2002, we had an accumulated deficit of $136.3 million.

Our business strategy aims to balance near-term revenues from product partnering and technology licensing with independent product development and commercialization. With respect to the development and commercialization of our lead product candidates, our goals are to: (1) enter into a partnership with a pharmaceutical or biotechnology company to develop and commercialize our lead product candidate, AP23573, to treat cancer; (2) enter into partnerships with medical device companies to develop and commercialize our lead product candidate, AP23573, in drug-delivery stents to decrease reblockage of arteries following angioplasty and stenting; (3) independently develop as many of our product candidates as possible through at least phase 2 before partnering them; (4) establish the commercial infrastructure to market or co-market our anti-cancer product candidates in the United States; and (5) enter into partnerships for our other product candidates outside the United States. With respect to our core technologies and intellectual property, our goals are to license our NF-κB technology to pharmaceutical and biotechnology companies conducting research on the discovery of

drugs that modulate NF-κB cell signaling and/or marketing such drugs and to license our RegTech cell-signaling technologies to pharmaceutical and biotechnology companies to accelerate their drug discovery. In addition, we may jointly develop product candidates incorporating our ARGENT cell-signaling regulation technology, especially with companies that have proprietary therapeutic genes, cellular systems or gene delivery vectors. However, there can be no assurance that we will be successful in achieving our strategies and generating future revenue streams.

Critical Accounting Policies

Our financial position and results of operations are affected by subjective and complex judgments, particularly in the areas of stock-based compensation to consultants, deferred compensation benefits for executives and key employees, and the carrying value of intangible assets. In determining expense related to stock-based compensation and deferred compensation, recorded balances are adjusted at each reporting period to reflect fair value utilizing the Black Scholes financial model that takes into account, among other things, the price and volatility of our common stock or other underlying securities, an interest-free discount rate, and an estimate of the life of the option contract. Fluctuations in those factors result in uneven expense charges or credits to our statements of operations. If, for example, the price and volatility of our common stock were 10% greater as of December 31, 2002, we would have recognized an increase of $10,000 in stock-based compensation to consultants in 2002. Similarly, if the market price of the underlying securities in our executive deferred compensation plan was 10% higher at December 31, 2002, we would have recognized an additional $214,000 in compensation expense in 2002.

At December 31, 2002, we reported $5.5 million of intangible assets consisting of costs related primarily to purchased patents, patent applications and licenses. These costs are being amortized over the estimated useful lives of the underlying patents or licenses. Changes in these lives or a decision to discontinue using the technologies could result in material changes to our balance sheet and statements of operations. For example, during 2002, we expensed $591,000 of unamortized costs related to certain intangible assets which we are not actively developing any longer. We recently announced that we are focusing our resources primarily on developing our three lead anti-cancer, small-molecule product candidates and have extended our development timelines on several other programs. We have concluded that the carrying value of our intangible assets is not currently impaired because they are utilized in our anti-cancer programs and/or continue to be viable technologies for collaborations or licensing efforts which we continue to pursue. If we were to abandon the underlying technologies or terminate our efforts to pursue collaborations or license agreements, we may be required to write off a portion of the carrying value of our intangible assets.

Results of Operations

Years Ended December 31, 2002 and 2001

Revenue

We recognized research revenue of $67,000 for the year ended December 31, 2002 compared to $4,000 for the year ended December 31, 2001. The increase in research revenue was due to license agreements into which we have entered related to our NF-κB and ARGENT cell-signaling technologies.

Operating expenses

Research and development expenses increased 39% to $23.0 million in 2002 from $16.6 million in 2001. This $6.4 million increase in 2002 expenses as compared to those incurred in 2001 was primarily due to the costs of advancing our product candidates through preclinical and clinical phases of development. As of December 31, 2002, we had seven development candidates of which one was in Phase 2 development. Preclinical costs include product development costs (including pharmacology and toxicology studies) and manufacturing costs to produce and scale-up material for various studies and clinical trials. Our increase in development activity

resulted in a higher level of spending on product development of $4.7 million, product manufacturing and external activities in support of clinical development of $940,000 and increased personnel expenses of $780,000. In addition, we wrote off $591,000 of capitalized patent and license costs reflecting our evaluation of several technologies in our portfolio which we are not actively developing at this time. Such increases in costs were partially offset by decreased overhead expenses of $207,000 and decreased consulting costs of $513,000.

General and administrative expenses increased 28% to $5.7 million in 2002 from $4.5 million in 2001. This $1.2 million increase in 2002 expenses as compared to those incurred in 2001 was primarily due to increased professional expenses of $676,000 resulting principally from our litigation with Eli Lilly and Company regarding infringement of certain claims of one of our patents, personnel expenses of $321,000 resulting from additions to our legal and business development personnel and other general expenses of $252,000.

In March 2003, we announced that we are focusing our research and development efforts primarily on our anti-cancer small-molecule product candidates and reducing or deferring our research and development efforts in certain other programs. As a result of this decision, we have reduced our workforce by 20% and expect to reduce our overall expenses by approximately 33% from those amounts incurred in 2002. Actual expenses will be determined in part by our ability to realize revenue through partnerships, licensing, joint ventures or similar arrangements, progress in the development and testing of our product candidates, progress and status of our litigation with Eli Lilly and Company, and our ability to raise additional funding through the sale of equity securities.

Interest income/expense

Interest income decreased 61% to $615,000 in 2002 from $1.6 million in 2001 primarily as a result of declining interest rates during the year and a lower level of funds invested. Interest expense increased 13% to $323,000 in 2002 from $285,000 in 2001. This increase was primarily due to a higher level of long-term debt outstanding in 2002 offset, in part, by lower interest rates for 2002.

Other income – tax refund

Other income for the year ended December 31, 2002 consisted of a one-time tax refund of $534,000, received in June 2002, due to changes in the tax laws. As a result of these changes, we were able to carry back a portion of the 2001 loss to offset the taxes resulting from the sale of our 50% interest in the Genomics Center to Aventis. In December 1999, we recognized a gain on the sale of $46.4 million, net of $534,000 in Alternative Minimum Tax, and reported the gain in other income.

Operating results

We reported a loss from operations of $28.7 million in 2002 compared to a loss from operations of $21.1 million in 2001, an increase in loss of $7.6 million or 36%. We expect that our loss from operations will decrease in 2003 due to focusing our research and development efforts primarily on our three anti-cancer small-molecule product candidates. Losses may fluctuate depending on the extent to which, if at all, we enter into collaborations or partnerships for one or more of our product candidates or licenses for our technologies. The extent of operating losses will also depend on our ability to raise funding from other sources, such as the capital markets, which will influence the amount we will spend on research and development and the development timelines for our product candidates.

We reported a net loss of $27.8 million in 2002 or $.86 per share (basic and diluted). We reported a net loss of $19.8 million in 2001 or $.68 per share (basic and diluted).

Years Ended December 31, 2001 and 2000

Revenue

We recognized research revenue of $4,000 for the year ended December 31, 2001 compared to $128,000 for the year ended December 31, 2000. The decrease in research revenue was due to the termination of our services agreements with the Genomics Center as a result of the sale of our 50% ownership interest in the Genomics Center to Aventis on December 31, 1999. Research revenue for the year ended December 31, 2000 was comprised principally of transitional research revenue for services provided to Aventis following the December 31, 1999 sale of our interest in the Genomics Center.

Operating expenses

Research and development expenses increased 33% to $16.6 million in 2001 from $12.5 million in 2000. This $4.1 million increase in 2001 expenses as compared to those incurred in 2000 was primarily due to advancing our lead product candidates further through development resulting in a higher level of spending on product development of $867,000, product manufacturing and external activities in support of clinical trials of $1.7 million, costs associated with the launch of our initiatives to promote the commercialization and licensing of our cell-signaling regulation technologies by both corporate and academic researchers of $325,000, increased personnel expenses of $567,000 and overhead expenses of $471,000.

General and administrative expenses increased 35% to $4.5 million in 2001 from $3.3 million in 2000. This $1.2 million increase in 2001 expenses as compared to those incurred in 2000 was primarily due to increased professional expenses of $419,000, resulting from increased use of certain consultants, and personnel expenses of $825,000.

Interest income/expense

Interest income decreased 23% to $1.6 million in 2001 from $2.1 million in 2000 primarily as a result of declining interest rates during the year. Interest expense increased 27% to $285,000 in 2001 from $225,000 in 2000. This increase was primarily due to a higher level of long-term debt outstanding in 2001.

Operating results

We reported a loss from operations of $21.1 million in 2001 compared to a loss from operations of $15.7 million in 2000, an increase in loss of $5.4 million or 34%. We reported a net loss of $19.8 million in 2001 or $.68 per share (basic and diluted). We reported a net loss of $13.8 million in 2000 or $.53 per share (basic and diluted).

Selected Quarterly Financial Data

Summarized quarterly financial data are as follows:

In thousands, except per share amounts	2002 Quarters			
	First	Second	Third	Fourth
Total research revenue	$ —	$ 13	$ 12	$ 42
Net loss	(6,167)	(7,043)	(7,819)	(6,814)
Net loss per share (basic and diluted)	(.19)	(.22)	(.24)	(.21)
	2001 Quarters			
	First	Second	Third	Fourth
Total research revenue	$ 1	$ 1	$ 1	$ 1
Net loss	(4,157)	(4,716)	(4,745)	(6,141)
Net loss per share (basic and diluted)	(.15)	(.17)	(.16)	(.20)

Liquidity and Capital Resources

We have financed our operations and investments primarily through private placements and public offerings of our equity securities and research revenue and other transactions resulting from our collaboration with Aventis from 1995 to 1999, including the sale of our 50% interest in the Genomics Center in December 1999. In addition, we have financed our operations through the issuance of long-term debt, operating and capital lease transactions, certain licensing transactions, interest income, and government- sponsored research grants.

At December 31, 2002, we had cash and cash equivalents totaling $26.9 million and working capital of $21.1 million compared to cash, cash equivalents and marketable securities totaling $47.2 million and working capital of $43.2 million at December 31, 2001.

The primary uses of cash during the year ended December 31, 2002 were $24.0 million to finance our operations and working capital requirements, $1.5 million to repay long-term debt, $1.4 million to invest in intellectual property and $269,000 to purchase equipment. We expect that our working capital requirements for 2003 will be reduced from 2002 as a result of focusing our research and development efforts on our three lead anti-cancer small-molecule product candidates.

The primary sources of funds during the year ended December 31, 2002, were $5.6 million from the sale of common stock, $999,000 from the issuance of common stock related to the exercise of stock options and purchases under the employee stock purchase plan, $442,000 from the sales and maturities of marketable securities and $77,000 from additional borrowing to finance purchases of equipment.

On June 22, 2001, we filed a shelf registration statement with the United States Securities and Exchange Commission ("SEC") for the issuance of up to 4.5 million registered shares of our common stock, which was declared effective by the SEC on August 1, 2001 (the "2001 Shelf Registration Statement"). At December 31, 2001, we had 2,572,288 registered shares remaining available for sale at our discretion under this shelf registration, following the sale of 1,927,712 shares of common stock on October 31, 2001. On November 13, 2002, we sold an additional 2,200,000 shares of our common stock registered under such shelf registration statement to existing and new institutional investors at a price of $2.75 per share and received gross proceeds of $6.1 million before commissions and expenses of $415,000. This net amount of $5.6 million was included in the 2002 primary sources of funds mentioned in the preceding paragraph. On January 9, 2002, we filed an additional shelf registration statement with the SEC for the issuance of up to 3.0 million registered shares of our common stock,

which was declared effective on February 13, 2002. The 3.0 million registered shares, as well as the 372,288 shares remaining on the 2001 Shelf Registration Statement, remain available for sale at our discretion, subject to certain limitations under federal securities laws and the rules of the Nasdaq Stock Market.

We have substantial fixed contractual obligations under various research and licensing agreements, consulting and employment agreements, lease agreements and long-term debt instruments. These contractual obligations were comprised of the following as of December 31, 2002:

In thousands	Payments Due By Period				
Contractual Obligations	**Total**	**In 2003**	**2004 through 2006**	**2007 through 2008**	**After 2008**
Long-term debt	$ 6,915	$1,478	$ 5,437	$ —	$ —
Operating leases	2,938	904	1,714	320	
Other long-term obligations *	7,949	3,240	4,105	244	360
Total fixed contractual obligations	$17,802	$5,622	$11,256	$564	$360

*Other long-term obligations are comprised primarily of employment agreements and license agreements. The license agreements generally provide for payment by us of annual license fees, milestone payments and royalties upon successful commercialization of products. All license agreements are cancelable by us. The above table reflects remaining license fees for the lives of the agreements but excludes milestone and royalty payments, as such amounts are not probable or estimable at this time.

In March 2003, we entered into a term loan agreement with a bank for $7.5 million, the proceeds of which were used to repay existing long-term debt, to pay off our obligations under certain operating leases for equipment and for general working capital purposes. The term loan is payable in monthly installments of $125,000 beginning in April 2003 plus interest with a final payment due in March 2006 of $3.0 million. Had this term loan been effective on December 31, 2002, our total fixed contractual obligations in the table above would have been $5.2 million in 2003, $11. 8 million in the period 2004 through 2006, $564,000 in the period 2007 through 2008 and $360,0000 thereafter.

We will require substantial additional funding for our research and development programs for operating expenses, for the pursuit of regulatory approvals and for establishing manufacturing, marketing and sales capabilities. Adequate funds for these purposes, whether obtained through financial markets or other arrangements with collaborative partners, or from other sources, may not be available when needed or on terms acceptable to us.

Based on our current operating plans and assuming no further funding or potential revenues that may be generated from product partnering or licensing initiatives we are currently pursuing, we believe that our current available funds will be adequate to satisfy our capital and operating requirements into the second quarter of 2004. However, there can be no assurance that changes in our research and development plans or other future events affecting our revenues or operating expenses will not result in the earlier depletion of our funds.

Recently Issued Accounting Pronouncements

In June 1998, the Financial Accounting Standards Board (“FASB) issued SFAS No. 133, *Accounting for Derivative Instruments and Hedging Activities*. This standard, requires that all companies record derivatives on the balance sheet as assets or liabilities, measured at fair value. Gains or losses resulting from changes in the values of those derivatives are accounted for depending on the use of the derivative and whether it qualifies for hedge accounting. In April 2002, the EITF issued EITF 02-8, *Accounting for Options Granted to Employees in Unrestricted, Publicly Traded Shares of an Unrelated Entity*. This consensus requires that companies account for such benefits as derivatives under SFAS No. 133. We adopted the provisions of these pronouncements in connection with our executive compensation plan in 2002. See Note 5 of notes to consolidated financial statements.

In December 2002, the EITF issued EITF 00-21, *"Accounting for Revenue Arrangements with Multiple Deliverables."* This consensus provides guidance in determining when a revenue arrangement with multiple deliverables should be divided into separate units of accounting, and, if separation is appropriate, how the arrangement consideration should be allocated to the identified accounting units. The provisions of EITF 00-21 are effective for revenue arrangements entered into in fiscal periods beginning after June 15, 2003. We will evaluate multiple element arrangements in accordance with this EITF conclusion upon its effective date for new arrangements into which it enters.

In December 2002, the FASB issued SFAS No. 148, *"Accounting for Stock-Based Compensation —Transition and Disclosure, an amendment of FASB Statement No. 123."* This Statement amends SFAS No. 123, *"Accounting for Stock-Based Compensation,"* to provide alternative methods of transition for a voluntary change to the fair value based method of accounting for stock-based employee compensation. In addition, this Statement amends the disclosure requirements of SFAS No. 123 to require prominent disclosures in both annual and interim financial statements about the method of accounting for stock-based employee compensation and the effect of the method used on reported results. We have determined that we will continue to account for stock-based compensation to employees under the provisions of APB No. 25 and will make all required disclosures in our annual and interim financial statements.

In June 2002, the FASB issued SFAS No. 146, "*Accounting for Costs Associated with Exit or Disposal Activities*." This statement replaces EITF 94-3 "*Liability Recognition for Certain Employee Termination Benefits and Other Costs to Exit an Activity (including Certain Costs Incurred in a Restructuring)*" and requires that a liability for costs associated with a disposal or exit activity be recognized only when an actual liability is incurred and not when management has committed to a plan as is the case under EITF 94-3. The application of this statement is required for exit or disposal activities that are initiated after December 31, 2002.

ITEM 7A: QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK

We invest our available funds in accordance with our investment policy to preserve principal, maintain proper liquidity to meet operating needs and maximize yields. Our investment policy specifies credit quality standards for our investments and limits the amount of credit exposure to any single issue, issuer or type of investment.

We invest cash balances in excess of operating requirements first in short-term, highly liquid securities, with maturities of 90 days or less, and money market accounts. Depending on our level of available funds and our expected cash requirements, we may invest a portion of our funds in marketable securities, consisting generally of corporate debt and U.S. government securities with maturities of one year or less, but generally less than six months. These securities are classified as available-for-sale. Available-for-sale securities are recorded on the balance sheet at fair market value with unrealized gains or losses reported as a separate component of stockholders' equity (accumulated other comprehensive loss). Gains and losses on marketable security transactions are reported on the specific-identification method. Interest income is recognized when earned. A decline in the market value of any available-for-sale security below cost that is deemed other than temporary results in a charge to earnings and establishes a new cost basis for the security.

Our investments are sensitive to interest rate risk. We believe, however, that the effect, if any, of reasonable possible near-term changes in interest rates on our financial position, results of operations and cash flows generally would not be material due to the short-term nature of these investments. In particular, at December 31, 2002, because our available funds are invested solely in cash equivalents and short-term securities with maturities less than 90 days, our risk of loss due to changes in interest rates is not material.

We have an executive compensation plan which provides participants, in lieu of a cash bonus, an option to purchase certain designated mutual funds at a discount equal to the amount of the bonus. These deferred compensation arrangements are accounted for as derivatives under SFAS No. 133. The fair value of the derivatives are reflected as a liability on our balance sheet. As of December 31, 2002, in the event of a hypothetical 10% increase (decrease) in the fair market value of the underlying mutual funds, we would incur approximately $214,000 of additional (less) compensation expense.

At December 31, 2002, we have a bank term note which bears interest at prime plus 1%. This note is sensitive to interest rate risk. In the event of a hypothetical 10% increase in the prime rate (42.5 basis points), we would incur approximately $30,000 of additional interest expense per year. Our refinancing in March 2003 would not affect this sensitivity analysis materially.

Certain Factors That May Affect Future Results of Operations

The SEC encourages companies to disclose forward-looking information so that investors can better understand a company's future prospects and make informed investment decisions. This Annual Report contains such "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These statements may be made directly in this Annual Report, and they may also be made a part of this Annual Report by reference to other documents filed with the SEC, which is known as "incorporation by reference."

Such statements in connection with any discussion of future operating or financial performance may be identified by use of words such as "may," "anticipate," "estimate," "expect," "project," "intend," "plan," "believe" and other words and terms of similar meaning. Such statements are based on management's expectations and are subject to certain factors, risks and uncertainties that may cause actual results, outcome of events, timing and performance to differ materially from those expressed or implied by such forward-looking statements. These risks include, but are not limited to, risks and uncertainties regarding our ability to conduct preclinical and clinical studies of our product candidates and the results of such studies, regulatory oversight, intellectual property claims, the timing, scope, cost and outcome of legal proceedings, future capital needs, key employees, dependence on our collaborators and manufacturers, markets, economic conditions, products, services, prices,

reimbursement rates, competition and other factors. Please also see the discussion of risks and uncertainties under “Risk Factors” in Item 1 of this Annual Report.

In light of these assumptions, risks and uncertainties, the results and events discussed in the forward-looking statements contained in this Annual Report or in any document incorporated by reference might not occur. Stockholders are cautioned not to place undue reliance on the forward-looking statements, which speak only of the date of this Annual Report or the date of the document incorporated by reference in this Annual Report. We are not under any obligation, and we expressly disclaim any obligation, to update or alter any forward-looking statements, whether as a result of new information, future events or otherwise. All subsequent forward-looking statements attributable to us or to any person acting on our behalf are expressly qualified in their entirety by the cautionary statements contained or referred to in this section.

ITEM 8: FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA

Independent Auditors' Report

The Board of Directors and Stockholders of
ARIAD Pharmaceuticals, Inc.:

We have audited the accompanying consolidated balance sheets of ARIAD Pharmaceuticals, Inc. and its subsidiaries (the "Company") as of December 31, 2002 and 2001, and the related consolidated statements of operations, stockholders' equity, and cash flows for each of the three years in the period ended December 31, 2002. These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these consolidated financial statements based on our audits.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. 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, such consolidated financial statements present fairly, in all material respects, the financial position of ARIAD Pharmaceuticals, Inc. and its subsidiaries as of December 31, 2002 and 2001, and the results of their operations and their cash flows for each of the three years in the period ended December 31, 2002, in conformity with accounting principles generally accepted in the United States of America.

/s/ DELOITTE & TOUCHE LLP

Boston, Massachusetts
March 13, 2003

ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES
CONSOLIDATED BALANCE SHEETS

In thousands, except share and per share data	December 31, 2002	December 31, 2001
ASSETS		
Current assets:		
Cash and cash equivalents	$ 26,850	$ 46,742
Marketable securities (Note 2)		444
Inventory and other current assets	847	1,010
Total current assets	27,697	48,196
Property and equipment:		
Leasehold improvements	12,642	12,624
Equipment and furniture	5,668	5,417
Total	18,310	18,041
Less accumulated depreciation and amortization	(17,269)	(16,190)
Property and equipment, net	1,041	1,851
Intangible and other assets, net (Note 3)	6,366	5,314
Total assets	$ 35,104	$ 55,361
LIABILITIES AND STOCKHOLDERS' EQUITY		
Current liabilities:		
Accounts payable	$ 2,145	$ 1,505
Current portion of long-term debt	1,478	1,443
Accrued compensation and benefits	399	348
Accrued product developments expenses	1,006	1,073
Other accrued expenses	1,310	578
Deferred revenue	233	
Total current liabilities	6,571	4,947
Long-term debt (Notes 4 and 12)	5,437	6,847
Deferred executive compensation (Note 5)	1,244	474
Commitments, contingent liabilities and minority interest (Notes 1, 6, 11)		
Stockholders' equity:		
Preferred stock, authorized, 10,000,000 shares, none issued and outstanding		
Common stock, $.001 par value, authorized, 60,000,000 shares, issued and outstanding, 34,828,689 shares in 2002 and 32,146,774 shares in 2001	35	32
Additional paid-in capital	158,147	151,638
Deferred compensation	(13)	(106)
Accumulated other comprehensive income		3
Accumulated deficit	(136,317)	(108,474)
Total stockholders' equity	21,852	43,093
Total liabilities and stockholders' equity	$ 35,104	$ 55,361

See notes to consolidated financial statements

ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES
CONSOLIDATED STATEMENTS OF OPERATIONS

In thousands, except share and per share data	Years Ended December 31, 2002	2001	2000
Research revenue	$ 67	$ 4	$ 128
Operating expenses:			
Research and development *	23,018	16,587	12,467
General and administrative *	5,718	4,469	3,318
Total operating expenses	28,736	21,056	15,785
Loss from operations	(28,669)	(21,052)	(15,657)
Other income (expense):			
Interest income	615	1,578	2,050
Interest expense	(323)	(285)	(225)
Other income – tax refund	534		
Total other income, net	826	1,293	1,825
Net loss	$ (27,843)	$ (19,759)	$ (13,832)
Net loss per share (basic and diluted)	$ (.86)	$ (.68)	$ (.53)
Weighted average number of shares of common stock outstanding – basic and diluted	32,475,083	29,256,767	25,875,663
*Includes non-cash stock-based compensation expense			
- Research and development expense	$ (29)	$ 146	$ 142
- General and administrative expense		$ 57	

See notes to consolidated financial statements.

ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES
CONSOLIDATED STATEMENTS OF STOCKHOLDERS' EQUITY
For the Years Ended December 31, 2000, 2001 and 2002

In thousands, except share data	Common Stock Shares	Common Stock Amount	Additional Paid-in Capital	Deferred Compensation
Balance, January 1, 2000	22,031,888	$ 22	$101,928	$ 0
Issuance of common stock, series C repurchase	1,078,038	1	1,144	
Issuance of common stock, net of issuance costs	857,024	1	9,742	
Exercise of warrants	1,389,498	1	11,636	
Issuance of shares pursuant to ARIAD and AGTI stock option plans and ARIAD stock purchase plan	1,935,690	2	4,952	
Stock-based compensation to consultants			359	(359)
Amortization of stock-based compensation				142
Comprehensive loss:				
Net loss				
Other comprehensive loss-				
Unrealized loss on marketable securities				
Comprehensive loss				
Balance, December 31, 2000	27,292,138	27	129,761	(217)
Issuance of common stock, net of issuance costs	4,551,541	5	21,296	
Issuance of shares pursuant to ARIAD stock option and purchase plans	303,095		489	
Stock-based compensation to consultants			92	(92)
Amortization of stock-based compensation				203
Comprehensive loss:				
Net loss				
Other comprehensive income -				
Unrealized gains on marketable securities				
Comprehensive loss				
Balance, December 31, 2001	32,146,774	32	151,638	(106)
Issuance of common stock, net of issuance costs	2,200,000	2	5,633	
Issuance of shares pursuant to ARIAD stock option and purchase plans	481,915	1	998	
Stock-based compensation to consultants			(122)	122
Amortization of stock-based compensation				(29)
Comprehensive loss:				
Net loss				
Other comprehensive loss -				
Unrealized loss on marketable securities				
Comprehensive loss				
Balance, December 31, 2002	34,828,689	$ 35	$158,147	$ (13)

[Additional columns below]

[Continued from above table, first column(s) repeated]

In thousands, except share data	Accumulated Other Comprehensive Income (Loss)	Accumulated Deficit	Stockholders' Equity
Balance, January 1, 2000	$ 0	$ (74,883)	$ 27,067
Issuance of common stock, series C repurchase			1,145
Issuance of common stock, net of issuance costs			9,743
Exercise of warrants			11,637
Issuance of shares pursuant to ARIAD and AGTI stock option plans and ARIAD stock purchase plan			4,954
Stock-based compensation to consultants			

Amortization of stock-based compensation			142
Comprehensive loss:			
Net loss		(13,832)	(13,832)
Other comprehensive loss-			
Unrealized loss on marketable securities	(5)		(5)
Comprehensive loss			(13,837)
Balance, December 31, 2000	(5)	(88,715)	40,851
Issuance of common stock, net of issuance costs			21,301
Issuance of shares pursuant to ARIAD stock option and purchase plans			489
Stock-based compensation to consultants			
Amortization of stock-based compensation			203
Comprehensive loss:			
Net loss		(19,759)	(19,759)
Other comprehensive income -			
Unrealized gains on marketable securities	8		8
Comprehensive loss			(19,751)
Balance, December 31, 2001	3	(108,474)	43,093
Issuance of common stock, net of issuance costs			5,635
Issuance of shares pursuant to ARIAD stock option and purchase plans			999
Stock-based compensation to consultants			
Amortization of stock-based compensation			(29)
Comprehensive loss:		(27,843)	(27,843)
Net loss			
Other comprehensive loss -			
Unrealized loss on marketable securities	(3)		(3)
Comprehensive loss			(27,846)
Balance, December 31, 2002	$ —	$(136,317)	$ 21,852

See notes to consolidated financial statements

ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES
CONSOLIDATED STATEMENTS OF CASH FLOWS

In thousands	Years Ended December 31, 2002	2001	2000
Cash flows from operating activities:			
Net loss	$(27,843)	$(19,759)	$(13,832)
Adjustments to reconcile net loss to net cash used in operating activities:			
Depreciation and amortization	2,253	1,889	1,675
Stock-based compensation to consultants	(29)	203	142
Executive compensation expense	205	181	163
Increase (decrease) from:			
Inventory and other current assets	163	337	262
Other assets	(135)	(53)	(408)
Accounts payable	640	70	(842)
Accrued compensation and benefits	51	282	58
Accrued product development expenses	(67)	438	103
Other accrued expenses	732	(50)	(2,419)
Deferred revenue	233		
Deferred executive compensation	(161)	(6)	
Net cash used in operating activities	(23,958)	(16,468)	(15,098)
Cash flows from investing activities:			
Acquisitions of marketable securities		(7,585)	(42,965)
Proceeds from sales and maturities of marketable securities	442	34,356	15,805
Investment in property and equipment	(269)	(614)	(447)
Investment in intangible assets	(1,366)	(1,002)	(1,205)
Net cash provided by (used in) investing activities	(1,193)	25,155	(28,812)
Cash flows from financing activities:			
Proceeds from long-term debt borrowings	77	4,590	3,000
Repayment of long-term debt borrowings	(1,452)	(1,200)	(1,200)
Proceeds from issuance of common stock, net of issuance costs	5,635	21,633	9,743
Proceeds from issuance of common stock pursuant to stock option and purchase plans	999	489	4,953
Proceeds from exercise of warrants, net			11,637
Net cash provided by financing activities	5,259	25,512	28,133
Net increase (decrease) in cash and cash equivalents	(19,892)	34,199	(15,777)
Cash and cash equivalents, beginning of year	46,742	12,543	28,320
Cash and cash equivalents, end of year	$ 26,850	$ 46,742	$ 12,543
Interest paid	$ 288	$ 250	$ 215

See notes to consolidated financial statements.

1. Nature of Business and Summary of Significant Accounting Policies

Nature of Business

The Company is engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo,* that may be used to treat diseases in innovative ways. The Company is developing a comprehensive approach to the treatment of cancer and is primarily focused on a series of product candidates for targeted oncology indications. The Company also has an exclusive license to pioneering technology and patents related to the discovery, development and use of drugs that regulate NF-κB cell-signaling activity, which has been implicated in many major diseases.

Principles of Consolidation

The consolidated financial statements include the accounts of ARIAD Pharmaceuticals, Inc., its wholly owned subsidiary, ARIAD Corporation, and its 80% owned subsidiary ARIAD Gene Therapeutics, Inc. (“AGTI”) (Note 7). The Company’s development of product candidates based on the ARGENT cell-signaling regulation technology and mTOR inhibitors derived from the ARGENT programs are conducted on behalf of AGTI. Intercompany accounts and transactions have been eliminated in consolidation. There is no liability recorded for minority interest for AGTI in the consolidated balance sheets, because AGTI currently has a deficiency in its stockholders’ equity and, accordingly, the Company currently records 100% of the losses incurred by AGTI. The Company accounts for any gain from the exercise of AGTI options as an adjustment to additional paid-in capital. Because AGTI is a research and development company, the Company believes that the gain realization from the exercise of AGTI options cannot be assured and therefore should be accounted for as a capital transaction in the Company’s consolidated financial statements.

Basis of Presentation

As shown in the consolidated financial statements, the Company incurred net losses of $27.8 million and $19.8 million and used $24.0 million and $16.5 million of cash in operations for the years ended December 31, 2002 and 2001, respectively. At December 31, 2002, the Company had cash and cash equivalents of $26.9 million, long-term debt of $6.9 million and stockholders’ equity of $21.9 million. Also at December 31, 2002, the Company was in violation of one of its bank term loan covenants but had received a waiver of any and all covenant violations from the lender. The Company pledged $7.0 million of its cash and cash equivalents to the bank as security for the loan (see Note 4).

In March 2003, the Company announced that it is focusing its resources primarily on developing its lead anti-cancer small-molecule product candidates. As a result of this decision, the Company expects to reduce its operating expenses in 2003 by approximately 33% from those amounts incurred in 2002. In addition, the Company has entered into a new term loan agreement with a bank for $7.5 million (see Notes 4 and 12), the proceeds of which were used to repay existing long-term debt, to pay off obligations under certain operating leases and for general working capital purposes. This refinancing also lowered the Company’s cost of financing its equipment. The Company expects to remain in compliance with all covenants of the new term loan through at least December 31, 2003.

The Company will continue to pursue additional funding through the capital markets, collaborations for one or more of its product candidates and additional licenses for its technologies. Based on its current operating plans and the effect of the above actions and assuming no further funding, management believes that the Company’s

current available funds will be adequate to satisfy its capital and operating requirements into the second quarter of 2004.

Fair Value of Financial Instruments

The carrying amounts of cash, cash equivalents, accounts payable and accrued liabilities approximate fair value because of their short-term nature. Marketable securities are recorded in the consolidated financial statements at aggregate fair value (Note 2). The carrying amount of the Company's bank term note of $6.3 million at December 31, 2002 approximates fair value due to its variable interest rate (Note 4). The Company's term note with General Electric Capital Corporation, with an outstanding principal balance of $615,000 at December 31, 2002, bears a fixed interest rate of 9.48% (Note 4). Based on current interest rates for comparable debt, the estimated fair value of this term note is $654,000 at December 31, 2002. The Company's obligation under its executive compensation plan (Note 5) is based on the current fair market value of the underlying securities and is therefore stated at its estimated current fair value.

Accounting Estimates

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

Cash Equivalents

Cash equivalents include short-term, highly liquid investments, which consist principally of United States government securities and high-grade domestic corporate securities, purchased with remaining maturities of 90 days or less, and money market accounts.

Marketable Securities

The Company has classified its marketable securities as "available-for-sale" and, accordingly, carries such securities at aggregate fair value. The difference between fair value and original cost is reflected as a component of accumulated other comprehensive income (loss). Fair value has been determined based on quoted market prices, in a dealer market, at the closing bid for each individual security held.

Inventory

Inventory consists of bulk pharmaceutical material to be used for multiple development programs. Inventories are carried at cost using the first-in, first-out method and are charged to research and development expense when consumed. The carrying value of inventory amounted to $430,000 and $682,000 at December 31, 2002 and 2001, respectively.

Property and Equipment

Property and equipment are recorded at cost. Depreciation is recorded using the straight-line method over the estimated useful lives of the assets (3 to 10 years). Assets acquired under capital lease obligations are stated at the lower of the present value of the minimum lease payments or the fair market value at the inception of the lease. Assets recorded under capital leases and leasehold improvements are amortized over the shorter of their useful lives or lease term using the straight-line method (4 to 10 years).

Intangible and Other Assets

Intangible and other assets consist primarily of capitalized patent and license costs, deposits and the unvested portion of the fair value of outstanding grants under the Company's executive compensation plan (Note 5). The cost of purchased patents and patent applications, costs incurred in filing patents and certain license fees are capitalized. Capitalized costs related to issued patents are amortized over a period not to exceed seventeen years or the remaining life of the patent, whichever is shorter, using the straight-line method. Capitalized license fees are amortized over the periods to which they relate. Amortization expense for intangible assets amounted to $548,000, $502,000, and $431,000 for 2002, 2001 and 2000, respectively. In addition, capitalized costs are expensed when it becomes determinable that such patent applications or technology will not be pursued. The Company expensed $591,000, $0, and $58,000 for the years ended December 31, 2002, 2001 and 2000, respectively, in accordance with this policy.

Impairment of Long-Lived Assets

The Company reviews its long-lived assets for impairment when events or changes in circumstances indicate that the carrying amount of a long-lived asset may not be recoverable. Recoverability of assets to be held and used is measured by a comparison of the carrying amount of an asset to future net cash flows expected to be generated by the asset. If such assets are considered to be impaired, the impairment to be recognized is measured by the amount by which the carrying amount of the assets exceeds the fair value of the assets.

Revenue Recognition

The Company recognizes revenue in accordance with the Securities and Exchange Commission ("SEC") Staff Accounting Bulletin 101 *Revenue Recognition in Financial Statements.* Revenue is principally comprised of license fees received under agreements that provide the licensee with access to and/or review and evaluation of certain technology owned or controlled by the Company. Upfront and annual license fees are recorded as deferred revenue upon receipt and recognized as revenue on a systematic basis over the period of time they are earned in accordance with the terms of the agreements. Such agreements may also include milestone and royalty payments. Such payments will be recognized as revenue when earned in accordance with the terms of the related agreements.

Income Taxes

The Company accounts for income taxes in accordance with Statement of Financial Accounting Standard ("SFAS") SFAS No. 109, "Accounting for Income Taxes," which requires an asset and liability approach to financial accounting and reporting for income taxes. Deferred income tax assets and liabilities are computed annually for differences between financial statement basis and the income tax basis of assets and liabilities that will result in taxable or deductible amounts in the future. Such deferred income tax liability computations are based on enacted tax laws and rates applicable to the years in which the differences are expected to affect taxable income. A valuation allowance is established when it is necessary to reduce deferred income tax assets to the expected realized amounts.

Segment Reporting

The Company organizes itself into one segment reporting to the chief executive officer. No significant revenues from product sales or services occurred in the years ended December 31, 2002, 2001 or 2000.

Stock-Based Compensation

SFAS No. 123, *Accounting for Stock-Based Compensation*, addresses the financial accounting and reporting standards for stock or other equity-based compensation arrangements. The Company accounts for stock or other equity-

based compensation for non-employees under the fair value-based method as required by SFAS No. 123 and Emerging Issues Task Force ("EITF") 96-18, *Accounting for Equity Instruments That Are Issued to Other Than Employees for Acquiring, or in Conjunction with Selling, Goods or Services"* and other related interpretations. Under this method, the equity-based instrument is valued at either the fair value of the consideration received or the equity instrument issued on the date of grant. The resulting compensation cost is recognized and charged to operations over the service period, which is usually the vesting period. The unearned portion of these awards is classified as a component of stockholders' equity and is listed as "deferred compensation" on the consolidated balance sheet.

The Company uses the intrinsic value method to measure compensation expense associated with grants of stock options to employees. On a pro forma basis, had the Company used the fair value method to measure compensation for all stock options, the net loss and net loss per share would have been reported as follows:

	Years ended December 31,		
In thousands (except per share data)	**2002**	**2001**	**2000**
Net loss, as reported	$(27,843)	$(19,759)	$(13,832)
Effect of stock options if valued at fair value	(4,239)	(3,617)	(2,835)
Pro forma net loss	$(32,082)	$(23,376)	$(16,667)
Net loss per share, as reported	$ (.86)	$ (.68)	$ (.53)
Effect of stock options if valued at fair market	(.13)	(.12)	(.11)
Pro forma net loss per share	$ (.99)	$ (.80)	$ (.64)

The above disclosure, required by SFAS No. 123, includes only the effect of grants made subsequent to January 1, 1996. For purposes of calculating the above disclosure, the fair value of options on their grant date was measured using the Black-Scholes option pricing model. Key assumptions used to apply this pricing model included a risk-free interest rate of 3.0% for 2002, 4.2% for 2001, and 5.9% for 2000, expected lives of the option grants ranging from one to six years and expected rates of volatility for the underlying stock of 106% for 2002, 108% for 2001, and 109% for 2000. Using this model, the weighted average fair value per option for all options granted to employees in 2002, 2001 and 2000 was $4.01 $4.71, and $9.35, respectively.

Earnings Per Share

Basic earnings per common share are computed using the weighted average number of common shares outstanding during each year. Diluted earnings per common share reflect the effect of the Company's outstanding options, warrants and convertible securities, except where such items would be anti-dilutive. In years in which a net loss is reported, basic and diluted per share amounts are the same. In 2002, 2001 and 2000, options amounting to 5,392,311, 4,639,782, and 3,480,360 shares of common stock, respectively, were not included in the computation of dilutive earnings per share, because the effect would be anti-dilutive. There were no warrants or convertible securities outstanding at December 31, 2002, 2001 or 2000.

Recently Issued Accounting Pronouncements

In June 1998, the Financial Accounting Standards Board ("FASB") issued SFAS No. 133, *Accounting for Derivative Instruments and Hedging Activities*. This standard requires that all companies record derivatives on the balance sheet as assets or liabilities, measured at fair value. Gains or losses resulting from changes in the values of those derivatives are accounted for depending on the use of the derivative and whether it qualifies for hedge accounting. In April 2002, the EITF issued EITF 02-8, *Accounting for Options Granted to Employees in*

Unrestricted, Publicly Traded Shares of an Unrelated Entity. This consensus requires that companies account for such benefits as derivatives under SFAS No. 133. The Company adopted the provisions of these pronouncements in 2002 in connection with its executive compensation plan (Note 5).

In December 2002, the EITF issued EITF 00-21, *"Accounting for Revenue Arrangements with Multiple Deliverables."* This consensus provides guidance in determining when a revenue arrangement with multiple deliverables should be divided into separate units of accounting, and, if separation is appropriate, how the arrangement consideration should be allocated to the identified accounting units. The provisions of EITF 00-21 are effective for revenue arrangements entered into in fiscal periods beginning after June 15, 2003. Management will evaluate multiple element arrangements in accordance with this EITF conclusion upon its effective date for new arrangements into which it enters.

In December 2002, the FASB issued SFAS No. 148, *"Accounting for Stock-Based Compensation —Transition and Disclosure, an amendment of FASB Statement No. 123."* This Statement amends SFAS No. 123, *"Accounting for Stock-Based Compensation,"* to provide alternative methods of transition for a voluntary change to the fair value based method of accounting for stock-based employee compensation. In addition, this Statement amends the disclosure requirements of SFAS No. 123 to require prominent disclosures in both annual and interim financial statements about the method of accounting for stock-based employee compensation and the effect of the method used on reported results. The Company has determined that it will continue to account for stock-based compensation to employees under the provisions of APB No. 25 and will make all required disclosures in its annual and interim financial statements.

In June 2002, the FASB issued SFAS No. 146, "*Accounting for Costs Associated with Exit or Disposal Activities."* This statement replaces EITF 94-3 "*Liability Recognition for Certain Employee Termination Benefits and Other Costs to Exit an Activity (including Certain Costs Incurred in a Restructuring)*" and requires that a liability for costs associated with a disposal or exit activity be recognized only when an actual liability is incurred and not when management has committed to a plan as is the case under EITF 94-3. The application of this statement is required for exit or disposal activities that are initiated after December 31, 2002.

Reclassifications

Certain reclassifications have been made to prior year financial statements to conform to the 2002 presentation.

2. Marketable Securities

The Company has classified its marketable securities as available-for-sale and, accordingly, carries such securities at aggregate fair value. At December 31, 2001, all of the Company's marketable securities consisted of corporate debt securities. At December 31, 2002, the Company held no marketable securities.

At December 31, 2001, the aggregate fair value and amortized cost of the Company's marketable securities were $444,000 and $441,000, respectively. Gross unrealized gains and losses were $3,000 and $0, respectively, at December 31, 2001.

Gains and losses on investment security transactions are reported on the specific-identification method. Realized gains and losses on sales of marketable securities were not material during the years ended December 31, 2002, 2001 and 2000. Changes in market values resulted in an increase (decrease) in net unrealized gains (losses) of ($3,000) $8,000, and ($5,000) for the years ended December 31, 2002, 2001 and 2000, respectively.

3. Intangible and Other Assets, Net

Intangible and other assets, net, was comprised of the following at December 31:

	2002	2001
	In thousands	
Capitalized patent and license costs	$ 8,130	$ 7,356
Less accumulated amortization	(2,680)	(2,130)
	5,450	5,226
Unvested executive deferred compensation (Note 5)	726	
Other	190	88
	$ 6,366	$ 5,314

4. Long-Term Debt

Long-term debt was comprised of the following at December 31:

	2002	2001
	In thousands	
Bank term note at prime plus 1% (5.25%, at December 31, 2002) payable in monthly installments of $100,000 plus interest, through January 1, 2005	$ 6,300	$ 7,500
General Electric Capital Corporation term notes at average interest rate of 9.48% payable in monthly installments including interest of $27,015, through December 2004 and $1,911 from January 2005 through June 2006	615	790
Less current portion	(1,478)	(1,443)
Long-term debt	$ 5,437	$ 6,847

The bank term note is collateralized by all assets of the Company with the exception of the assets to collateralize the General Electric Capital Corporation ("G.E.") term notes. The Company may, at its option, pledge marketable securities under the bank term note, and, in such event, the interest rate would be adjusted to the equivalent of 90-day LIBOR plus 1.25%.

The bank term note agreement contained certain covenants that would require consent from the bank to change the Company's Chief Executive Officer or increase indebtedness, as well as covenants that limited capital spending and stock redemption and restricted dividend distributions. The agreement also required the Company to pledge its marketable securities or maintain minimum levels of tangible net worth of $15.0 million, working capital of $7.0 million and liquid assets of $15.0 million plus the outstanding principal balance of the G.E. term notes, all as defined. At December 31, 2002, the Company was in violation of one of its bank term loan covenants but had received a waiver of any and all covenant violations from the lender. The Company pledged $7.0 million of its cash and cash equivalents to the bank as security for the loan.

The G.E. term notes were collateralized by certain assets of the Company. The G.E. term notes contained a covenant that required the Company to maintain a minimum unrestricted cash balance of $10.0 million. As noted below, the G.E. term note was paid off in March 2003.

In March 2003, the Company entered into a term loan agreement with another bank for $7.5 million (see Note 12), the proceeds of which were used to repay the existing bank term note and the G.E. term notes as well as to pay off remaining obligations under certain operating leases, such repayments totaling $6.9 million in the

aggregate. The Company expects to remain in compliance with all covenants of the new term loan through at least December 31, 2003. Therefore, the bank term note continues to be classified as a long-term liability at December 31, 2002.

Prior to the March 2003 refinancing, the annual aggregate future principal payments of the above debt agreements were $1.5 million for 2003, $1.5 million for 2004 and $3.9 million in 2005. Interest expense during 2002, 2001, and 2000 were $289,000 $255,000 and $204,000 respectively.

5. Executive Compensation Plan

Since 1998, the Company has maintained an executive compensation plan which provides participants, in lieu of a cash bonus, an option to purchase certain designated mutual funds at a discount (75% for each year since the plan's inception) equal to the amount of the bonus. The options vest equally over four years. For awards granted prior to 2002, the benefit obligation had been recorded as compensation expense and a liability as the obligation vested based on the fair market value of the underlying designated mutual funds.

In April 2002, the EITF issued EITF 02-8, *Accounting for Options Granted to Employees in Unrestricted, Publicly Traded Shares of an Unrelated Entity*. This consensus requires that the Company account for such benefits as derivatives under FAS No. 133, *Accounting for Derivative Instruments and Hedging Activities.* Under these pronouncements, the fair value of the derivative should be recorded at its inception as an asset and liability, with the asset amortized to expense over the vesting period. Subsequent changes in the fair value of the derivative should be included in the determination of net income or loss.

In July 2002, the Company approved the 2002 grants to certain executives and key employees and modified all prior year grants to conform certain terms with current year grants. As a result, the Company recorded (1) an asset of $877,000 which represents the unvested portion of the fair value of all outstanding grants, (2) an increase to the deferred executive compensation liability of $910,000 to $1,393,000, and (3) a resulting charge to income of $33,000. Total expense related to the executive compensation plan amounted to $205,000, $181,000 and $163,000 in 2002, 2001 and 2000, respectively. Changes in the fair value of the deferred executive compensation liability during 2002 were not material.

6. Leases, Licensed Technology and Other Commitments

Facility Lease

The Company conducts its operations in a 100,000 square foot office and laboratory facility under a non-cancelable operating lease. The original ten-year term of the lease expired in July 2002 and the Company has extended the lease for the first of two five-year extension periods. The Company has sublet approximately 37,000 square feet of space to two tenants. Rent expense, net of sublease income of $1.1 million, $950,000, and $1.2 million for the years ended December 31, 2002, 2001, and 2000, respectively, amounted to $601,000, $654,000, and $477,000, respectively. Future minimum annual rental payments, net of sublease income, for the next five years are approximately $654,000 for 2003 and $686,000 for 2004, 2005 and 2006, respectively, and $400,000 in 2007.

Equipment Leases

The Company has utilized a lease credit facility from an equipment leasing company to acquire equipment, which is resold to a lessor at cost, with no resulting gain or loss recognized. The lease agreement, which is classified as an operating lease for financial reporting purposes, has a term of five years with various lease renewal or purchase options at the end of the initial term. The Company did not enter into any new equipment lease agreements in 2002, 2001 or 2000. Equipment rental expense for the years ended December 31, 2002, 2001 and 2000 amounted to $788,000, $897,000, and $933,000, respectively. Minimum future rental payments under the initial terms of the leases are approximately $196,000 for 2003, and $33,000 for 2004.

At the end of the five-year lease terms, the Company has the option to purchase the equipment at a negotiated purchase price. In 2002, the Company paid a total of $105,000 to exercise options to purchase such equipment and recorded the expenditure as property and equipment. The assets are being amortized over their estimated remaining useful lives.

In March 2003, the Company utilized a portion of the proceeds of $7.5 million-term loan to pay off the remaining obligations under this lease agreement and purchase the associated assets.

Licensed Technology

The Company and AGTI have entered into agreements with several universities under the terms of which the Company and/or AGTI have received exclusive licenses or options to technology and intellectual property. The agreements, which are generally cancelable by the Company and AGTI, provide for the payment of license fees and/or minimum payments, which are generally creditable against future royalties. Fees paid by the Company on behalf of the Company and/or AGTI amounted to $317,000, $127,000 and $127,000 for 2002, 2001 and 2000, respectively, and are expected to amount to approximately $177,000 annually for 2003 through 2007. In addition, the agreements provide for payments upon the achievement of certain milestones in product development. The agreements also require the Company to fund certain costs associated with the filing and prosecution of patent applications.

Other Commitments

The Company has entered into various employment agreements with its senior officers. The agreements provide for aggregate annual base salaries of $2.8 million and remaining terms of employment of up to three years.

7. Stockholders' Equity

Preferred Stock

The Company has authorized 10 million shares of preferred stock which the Board of Directors is empowered to designate and issue in different series. As of December 31, 2002, the Board of Directors had designated 500,000 shares as series A preferred stock, and 9.5 million shares remained undesignated.

Series C Redeemable Convertible Preferred Stock ("Series C Preferred Stock")

On January 14, 2000, the Company completed the repurchase of the remaining 3,000 shares of its Series C Preferred Stock (initially issued in November 1998) for an aggregate consideration of $6.9 million plus 1,078,038 shares of common stock.

Common Stock – Shelf Registration

On June 22, 2001, the Company filed a shelf registration statement with the SEC for the issuance of up to 4.5 million registered shares of its common stock, which was declared effective by the SEC on August 1, 2001. The registered shares are available for sale at the Company's discretion. On October 31, 2001, the Company sold 1,927,712 registered shares of its common stock to institutional investors at a price of $4.15 per share and received gross proceeds of $8.0 million before commissions and expenses of $464,000. On November 13, 2002, the Company sold 2.2 million registered shares of its common stock to new and existing institutional investors at a price of $2.75 per share and received gross proceeds of $6.1 million before commissions and expenses of $415,000 At December 31, 2002 the Company had 372,288 registered shares remaining available for sale under this shelf registration.

On January 9, 2002, the Company filed a second shelf registration statement with the SEC for the issuance of up to an additional 3.0 million registered shares of its common stock, which was declared effective by the SEC on February 13, 2002. All of these shares remain available for sale under this shelf registration.

Redemption of Warrants

The Company received funds aggregating approximately $11.6 million from the exercise of approximately 1.4 million of its publicly traded warrants during the first and second quarters of 2000. Each warrant was exercisable for one share of common stock at an exercise price of $8.40 per share. The warrants had been called for redemption effective April 27, 2000. At December 31, 2002 and 2001, there were no warrants outstanding.

Common Stock – Other Sales

In 2001, in addition to the sale of shares under the shelf registration mentioned above, the Company sold an aggregate of 2.6 million shares of common stock in a direct placement and received net proceeds of $13.8 million. All of these shares were publicly registered.

Stockholder Rights Plan

The Board of Directors of the Company adopted a Rights Agreement, dated as of June 8, 2000 (the "2000 Rights Agreement"), between the Company and State Street Bank and Trust Company, as Rights Agent, and approved the declaration of a dividend distribution of one Preferred Share Purchase Right (a "Right") on each outstanding share of its Common Stock. In general, the Rights become exercisable if a person or group hereafter acquires 15% or more of the Common Stock of the Company or announces a tender offer for 15% or more of the Common Stock. The Board of Directors will, in general, be entitled to redeem the Rights at one cent per Right at any time before any such person hereafter acquires 15% or more of the outstanding Common Stock. The plan is designed to protect the Company's stockholders in the event that an attempt is made to acquire the Company without an offer of fair value.

If a person hereafter acquires 15% or more of the outstanding Common Stock of the Company (the "Acquiring Person"), each Right will entitle its holder to purchase, for an initial exercise price of $65, a number of shares of Common Stock having a market value at that time of twice the Right's exercise price. Rights held by the Acquiring Person will become void. If the Company is acquired in a merger or other business combination transaction after a person acquires 15% or more of the Company's Common Stock, each Right will entitle its holder to purchase, at the Right's then-current exercise price, a number of the acquiring company's common shares having a market value at that time of twice the Right's exercise price.

The dividend distribution of Rights was payable on July 19, 2000 to shareholders of record on June 19, 2000. The Rights will expire in ten years. The Rights distribution is not taxable to the Company's stockholders.

The Board of Directors also adopted two amendments to the Rights Agreement dated December 15, 1994, (the "1994 Rights Agreement"), between the Company and State Street Bank and Trust Company, as Rights Agent. As a result of these amendments, the adoption of the 2000 Rights Agreement and the setting of a record date to distribute new Rights, the 1994 Rights Agreement is no longer in effect.

Minority Interest in Subsidiary

The 20% minority interest in AGTI includes shares owned by Stanford University and Harvard University (which together own 2%) issued in 1995 in connection with a license agreement and shares issued to option holders (18%), including certain current and former members of the Company's management, Board of

Directors, and certain consultants. Additional stock options are outstanding and, if all such options were exercised, the minority interest would increase to 21% (Note 8).

8. Stock Option and Stock Purchase Plans

ARIAD Stock Option and Stock Plans

The Company's 1991, 1994 and 2001 Stock Option and Stock Plans (the "Plans") provide for the granting of nonqualified and incentive stock options to officers, directors, employees and consultants of the Company. Options become exercisable as specified in the related option agreement, typically over a four-year period, and expire ten years from the date of grant. As of December 31, 2002, the 1991 Plans have terminated according to their terms, although existing stock options granted under these Plans remain outstanding. Also as of December 31, 2002, there are no remaining options available to be issued under the 1994 Plan and 794,044 options available to be granted under the 2001 Plan.

Transactions under the Plans for the years ended December 31, 2000, 2001 and 2002 are as follows:

		Number of Shares	Weighted Average Exercise Price Per Share
Options outstanding,	January 1, 2000	5,069,369	$ 2.48
Granted		553,300	10.99
Forfeited		(226,668)	4.04
Exercised		(1,915,641)	2.40
Options outstanding,	December 31, 2000	3,480,360	3.77
Granted		1,554,220	5.61
Forfeited		(120,688)	5.75
Exercised		(274,110)	1.51
Options outstanding,	December 31, 2001	4,639,782	4.47
Granted		1,341,300	4.01
Forfeited		(185,830)	5.37
Exercised		(402,941)	1.89
Options outstanding,	December 31, 2002	5,392,311	$ 4.51
Options exercisable,	December 31, 2000	2,268,950	$ 2.64
	December 31, 2001	2,650,830	$ 3.31
	December 31, 2002	3,277,042	$ 3.95

The following table sets forth information regarding options outstanding at December 31, 2002:

Range of Exercise Prices	Number of Shares	Weighted Average Exercise Price	Weighted Average Remaining Life (years)	Number of Option Shares Currently Exercisable	Weighted Average Exercise Price for Currently Exercisable
$.75 - 1.25	525,714	$ .77	6.8	519,289	$ .77
1.34 - 2.31	876,259	1.70	5.4	711,300	1.71
2.68 - 4.88	2,213,366	4.17	7.7	1,228,552	4.09
4.89 - 8.00	1,439,722	6.04	7.9	641,776	6.16
12.56 - 14.63	337,250	3.41	7.5	176,125	13.37
	5,392,311	$4.51		3,277,042	$ 3.95

ARIAD Gene Therapeutics, Inc. Stock Option Plan

The Company's subsidiary, AGTI, adopted a stock option plan in 1993 substantially similar to the Plans and reserved 1,785,714 shares of AGTI's common stock for issuance pursuant to such plan. At December 31, 2002, options with respect to 87,428 shares of AGTI's common stock (all granted in 1994) were outstanding at an exercise price of $.42 per share, and all options were exercisable. During 2002 and 2001, no options were exercised or forfeited. During 2000, 758,282 options were exercised for aggregate proceeds of $318,000, and 25,000 option shares were forfeited. As of December 31, 2002, AGTI had 5,195,779 shares of its common stock outstanding.

Employee Stock Purchase Plan

In 1997, the Company adopted the 1997 Employee Stock Purchase Plan and reserved 500,000 shares of common stock for issuance under this plan. Under this plan, substantially all of its employees may, through payroll withholdings, purchase shares of the Company's stock at a price of 85% of the lesser of the fair market value at the beginning or end of each three-month withholding period. In 2002, 2001 and 2000, 78,974, 28,985 and 20,049 shares of common stock were issued under the plan, respectively.

9. Other Income – Tax Refund

Other income consists of a tax refund of $534,000 received in 2002. In March 2002, the "Job Creation and Worker Assistance Act of 2002 (the "Act") was signed into law. The Act allows taxpayers to carry back net operating losses incurred in 2001 and 2002 to the five prior tax years. Prior tax law limited the carry back to two years. In addition, the Act also suspended certain limitations on the utilization of Alternative Minimum Tax net operating losses. As a result of the Act, the Company was able to carry back a portion of its net loss for the year ended December 31, 2001 to recover taxes previously paid attributable to the sale of the Company's 50% interest in the Hoechst-ARIAD Genomics Center, LLC (the "Genomics Center") to Aventis Pharmaceuticals, Inc. on December 31, 1999. As a result of the sale, the Company had recorded a net gain of $46.4 million, net of $534,000 in Alternative Minimum Tax, in 1999 in other income.

10. Income Taxes

At December 31, 2002, the Company had available, for federal tax reporting purposes, net operating loss carryforwards of approximately $138.8 million, which expire commencing in 2009 and, for state tax reporting purposes, net operating loss carryforwards of approximately $100.4 million, which expire commencing in 2003. The Company also had federal research and development credit carryovers of approximately $6.0 million, which

expire commencing in 2006. Both the net operating loss carryforwards and credits are subject to certain limitations under federal tax law.

The components of deferred income taxes were as follows at December 31:

In thousands	2002	2001
Deferred tax liabilities:		
Intangible and other assets	$ 2,181	$ 2,091
Deferred tax assets:		
Net operating loss carryforwards	53,222	42,590
Federal and State tax credit carryovers	12,714	10,808
Depreciation	3,790	3,612
Other	465	408
Total deferred tax assets	70,191	57,418
Deferred tax assets, net	68,010	55,327
Valuation allowance	(68,010)	(55,327)
Total deferred taxes	$ 0	$ 0

Since the Company has not yet achieved sustained profitable operations, management believes the tax benefits as of December 31, 2002 and 2001 do not satisfy the realization criteria set forth in SFAS No. 109 and has recorded a valuation allowance for the entire net deferred tax asset. The increase in the valuation allowance of $12.7 million in 2002 and $8.9 million in 2001 resulted primarily from net operating loss carryforwards and tax credit carryovers that resulted from operations in those years.

11. Litigation

The Company was named as a defendant in a purported class action lawsuit commenced in June 1995 in the U.S. District Court for the Southern District of New York. The action named as defendants ARIAD Pharmaceuticals, Inc.; the underwriter of our initial public offering and a market maker in our stock, D. Blech & Co.; the managing director and sole shareholder of D. Blech & Co. and one of our former directors, David Blech; certain other of the Company's directors, and the qualified independent underwriter for the initial public offering, Shoenberg Hieber, Inc., or SHI.

Counsel for the plaintiff class, counsel for the named director defendants of the Company (excluding David Blech), or the Company Defendants, and the Company, and counsel for SHI have executed a stipulation of settlement in the action, or the Settlement. The Settlement was approved pursuant to entry by the Court of a Final Order and Judgment on December 4, 2002. The Final Order and Judgment orders that a payment of $620,000 (of which the Company's contribution was not material and no liability was recorded on the 2002 balance sheet) be distributed to the plaintiffs from a legal escrow account, that this action and a related action entitled In re: Blech Securities Litigation, 94 Civ. 7696 (RWS) are dismissed with prejudice, and that non-settling parties are barred from pursuing contribution-type claims against the Company Defendants.

On May 19, 1999, the Company filed suit in the Massachusetts Superior Court against Michael Z. Gilman, Ph.D., or Dr. Gilman, its former Chief Scientific Officer, seeking equitable relief for breach of his employment agreements in accepting a position as the research director of molecular biology at Biogen, Inc., or Biogen. The Superior Court issued a temporary injunction on May 19, 1999 restraining Dr. Gilman from using any of the Company's confidential information in his new employment. On June 21, 1999, Dr. Gilman filed

counterclaims against the Company seeking an order awarding damages for breach of contract and barring the Company from enforcing any provisions of the Company's employment agreements with Dr. Gilman. On May 26, 1999, Biogen filed a motion to intervene as a defendant in the action which the Superior Court granted on August 2, 1999. Discovery in the case has been completed, and Summary Judgment Motions have been filed, heard and ruled upon.

Counsel for the Company, counsel for Biogen and counsel for Dr. Gilman have executed a stipulated partial judgment, or the Stipulated Judgment, which was approved pursuant to entry by the Court on January 13, 2003. The Stipulated Judgment dismisses with prejudice the Company's claim for breach of contract against Dr. Gilman and dismisses Biogen as a party to the action. Dr. Gilman's counterclaims will now proceed to trial which the Company anticipates will be set in 2003. The ultimate outcome of the litigation with Dr. Gilman is not determinable at this time, and as a result, the Company cannot estimate whether any damages will be awarded or what the range of such an award might be and has not recorded any liability on the December 31, 2002 balance sheet.

On June 25, 2002, the Company, together with Massachusetts Institute of Technology, The Whitehead Institute for Biomedical Research and Harvard University, filed a lawsuit in the United States District Court for the District of Massachusetts, or the U.S. District Court, against Eli Lilly and Company, or Lilly, alleging infringement upon issuance of certain claims of our U.S. patent covering methods of treating human disease by regulating NF-κB cell-signaling activity, or the NF-κB '516 Claims, through sales of Lilly's osteoporosis drug, Evista®, and Lilly's septic shock drug, Xigris®, and seeking monetary damages from Lilly. On August 26, 2002, Lilly filed a motion to dismiss or, alternatively, for summary judgment, or Lilly's combined Motion, challenging the validity of the NF-κB '516 Claims. The Company filed a response to Lilly's Combined Motion on October 17, 2002 and Lilly filed a reply on November 17, 2002. Oral argument on Lilly's Combined Motion was heard in the U.S. District Court on November 21, 2002. As of March 12, 2003, the U.S. District Court had not yet ruled on Lilly's Combined Motion. While the ruling on Lilly's Combined Motion is not currently determinable, if Lilly were to be successful and its Combined Motion is granted, the Company will consider filing an appeal with the Court of Appeals for the Federal Circuit. If Lilly's Combined Motion is denied, a trial scheduling conference pursuant to Rule 16(b) of the Federal Rules of Civil Procedure will be scheduled by the U.S. District Court, and the case will proceed to the discovery phase leading to trial. The ultimate outcome of the litigation cannot be determined at this time, and, as a result, an estimate of a damage award or range of awards, if any, cannot be made.

12. Subsequent Events

On January 31, 2003, the Company entered into a non-exclusive license agreement with GPC Biotech AG ("GPC") that gives GPC the right to use the Company's ARGENT cell-signaling regulation technology in GPC's three-hybrid technology platform. The agreement provides for guaranteed fees of $2.0 million, including $1.0 million upon signing. GPC will pay the Company a percentage of revenues received by GPC from its partnerships utilizing its three-hybrid drug discovery platform, and make development and commercialization milestone payments and royalty payments to the Company based on GPC products, if any, resulting from the use of the Company's licensed technology in GPC's internal drug discovery programs.

On March 12, 2003, the Company entered into a $7.5 million term loan agreement with a bank. The loan is secured by a lien on all assets of the Company excluding intellectual property, which the Company has agreed not to pledge to any other party. The proceeds of the loan were used to pay off the existing $6.3 million bank term note and the $615,000 G.E. term notes as well as buy out remaining obligations under certain operating leases for equipment. The term loan carries interest at the bank's prime rate, or at LIBOR plus 2% and is repayable in monthly installments beginning in April 2003 of $125,000 plus interest over 36 months with a balloon payment of $3.0 million at the end of the term. The term loan agreement requires the Company to maintain a minimum of $10.0 million in unrestricted cash, cash equivalents and investments.

The agreement also contains certain covenants that restrict additional indebtedness, additional liens, sales of assets, and dividends, distributions or repurchases of common stock.

ITEM 9: CHANGES IN AND DISAGREEMENTS WITH ACCOUNTANTS ON ACCOUNTING AND FINANCIAL DISCLOSURE

Not Applicable.

PART III

ITEM 10: DIRECTORS AND EXECUTIVE OFFICERS OF THE REGISTRANT

Our directors, officers and key employees are as follows:

Name	Age	Position
Harvey J. Berger, M.D.	52	Chairman of the Board of Directors, Chief Executive Officer and President
Sandford D. Smith	55	Vice Chairman of the Board of Directors
Laurie A. Allen, Esq.	42	Senior Vice President, Chief Legal Officer, and Secretary
David L. Berstein, Esq.	50	Senior Vice President and Chief Patent Counsel
Fritz Casselman	53	Senior Vice President and Chief Business Officer
Timothy P. Clackson, Ph.D.	37	Senior Vice President, Science and Technology
Edward M. Fitzgerald	48	Senior Vice President, Chief Financial Officer and Treasurer
John D. Iuliucci, Ph.D.	60	Senior Vice President, Drug Development
Thomas A. Pearson	61	Senior Vice President, Corporate Strategy and Communications
Camille L. Bedrosian, M.D.	50	Vice President and Chief Medical Officer
David C. Dalgarno, Ph.D.	45	Vice President, Physical and Chemical Sciences
Maryann G. Krane	43	Vice President, Regulatory Affairs
Tomi K. Sawyer, Ph.D.	48	Vice President, Drug Discovery
Joseph Bratica	39	Director of Finance and Controller
Vaughn D. Bryson	64	Director
Jay R. LaMarche	56	Director
Frederick S. Schiff	55	Director
Burton E. Sobel, M.D.	65	Director
Raymond S. Troubh	76	Director
Elizabeth H.S. Wyatt	55	Director

Harvey J. Berger, M.D. is our principal founder and has served as our Chairman of the Board, Chief Executive Officer and President since April 1991. From 1986 to 1991, Dr. Berger held a series of senior management positions at Centocor, Inc., a biotechnology company, including Executive Vice President and President, Research and Development Division. He also has held senior academic and administrative appointments at Emory University, Yale University and the University of Pennsylvania and was an Established Investigator of the American Heart Association. Dr. Berger is a director of PTC Therapeutics, Inc., a closely held biotechnology company. Dr. Berger received his A.B. degree in Biology from Colgate University and his M.D. degree from Yale University School of Medicine and did further medical and research training at the Massachusetts General Hospital and Yale-New Haven Hospital.

Sandford D. Smith**,** one of our Directors since October 1991 and our Vice Chairman since January 1999, is Corporate Vice President and President, Genzyme Europe and International, Genzyme Corporation. From October 1997 to December 2000, he was President, Therapeutics International, Genzyme Corporation, and from May 1996 to September 1996, Vice President and General Manager, Specialty Therapeutics and International Group, Genzyme Corporation, a biotechnology company. Mr. Smith was President and Chief Executive Officer and a Director of Repligen Corporation, a biotechnology company, from 1986 to March

1996. Mr. Smith previously held a number of positions with Bristol-Myers Squibb and Company from 1977 to 1986, including, most recently, Vice President of Corporate Development and Planning for the United States Pharmaceutical and Nutritional Group. Mr. Smith is a Director of CSPI, a software company. Mr. Smith earned his B.A. degree from the University of Denver.

Laurie A. Allen, Esq. has served as our Senior Vice President and Chief Legal Officer since March 2002 and has served continuously as our Secretary since January 1999. Previously, from January 1999 to December 1999, she served as our Senior Vice President, Corporate Development and Legal Affairs and General Counsel. From January 2000 to March 2002, Ms. Allen was Senior Vice President, Business Development and Legal Affairs at Alexandria Real Estate Equities, Inc., a real estate investment trust. Previously, she was a partner with the law firm of Brobeck, Phleger and Harrison, LLP from January 1996 to December 1998. She also was an associate with Brobeck, Phleger & Harrison, LLP from February 1991 to December 1995. Ms. Allen received her A.B. degree in History from the University of California, Los Angeles, her L.L.M. degree in taxation from New York University and her J.D. degree from Emory University School of Law.

***David L. Berstein, Esq*.** has served as our Senior Vice President and Chief Patent Counsel since June 2000. Previously, he served as our Vice President and Chief Patent Counsel from September 1993 to June 2000. Prior to joining us, from 1990 through 1993, Mr. Berstein was Patent Counsel at BASF Bioresearch Corporation, a biotechnology company, where he was responsible for intellectual property matters, including patents and licensing. From 1985 to 1990, Mr. Berstein was a patent attorney at Genetics Institute, Inc., a biotechnology company, where he was involved in various aspects of the patent process from patent procurement through litigation. Mr. Berstein joined Genetics Institute from the law firm of Cooper & Dunham. Mr. Berstein received his B.S. degree from the University of Michigan and his J.D. degree from Fordham University School of Law.

Fritz Casselman has served as our Senior Vice President and Chief Business Officer since February 2001. From February 2000 to January 2001, Mr. Casselman was Senior Vice President, Strategy and Corporate Development at Avant Immunotherapeutics Inc., a biotechnology company. From 1997 to 2000, Mr. Casselman was Director of Worldwide Business Development at SmithKline Beecham, plc, a pharmaceutical company; from 1988 to 1996, Vice President and consultant to Cambridge Biotech Corporation, a biotechnology company; and from 1982 to 1988 an associate and then a partner at the law firm of Bromberg, Sunstein and Casselman. Mr. Casselman received his B.A. degree from the University of Wisconsin (Madison) and his J.D. degree from Boston University School of Law.

Timothy P. Clackson, Ph.D. has served as our Senior Vice President, Science and Technology since June 2002. Previously he served as our Vice President, Gene Therapy and Genomics from June 2000 to June 2002, as our Director, Gene Therapy from August 1999 to June 2000 and as our Department Head, Gene Therapy Biology from March 1999 to August 1999. Prior to joining us in December 1994, Dr. Clackson was a postdoctoral fellow at Genentech, Inc., a biotechnology company from 1991 to 1994, where he studied the molecular basis for human growth hormone function. Dr. Clackson received his B.A. degree in Biochemistry from the University of Oxford. Dr. Clackson received his Ph.D. in Biology from the University of Cambridge, for research conducted at the MRC Laboratory of Molecular Biology into antibody engineering and the development of phage display technology.

Edward M. Fitzgerald has served as our Senior Vice President, Chief Financial Officer and Treasurer since May 2002. From 1998 to April 2002, he served as Senior Vice President, Chief Financial Officer and Secretary at AltaRex Corp., a biotechnology company. From 1992 to 1997, Mr. Fitzgerald held various management positions at BankBoston Corp., a financial services and commercial banking company. From 1989 to 1992, he was a partner at Arthur Andersen & Co. in the audit and business advisory practice. Previously, from 1978 to 1988, he also was at Arthur Andersen & Co. Mr. Fitzgerald received his B.S. degree in accounting and M.B.A. degree in finance from Babson College.

John D. Iuliucci, Ph.D. has served as our Senior Vice President, Drug Development since January 1999. Previously, he also served as our Vice President, Drug Development from October 1996 to December 1998 and our Vice President, Preclinical Development from June 1992 to September 1996. Prior to joining us, Dr. Iuliucci was Director of Preclinical Pharmacology and Toxicology at Centocor, Inc., a biotechnology company, from 1984 to 1992. From 1975 to 1984, Dr. Iuliucci headed the Drug Safety Evaluation Department at Adria Laboratories, a pharmaceutical company. He was a Senior Toxicologist at the Warner-Lambert Pharmaceutical Research Institute from 1972 to 1975. Dr. Iuliucci received his B.S. degree in Pharmacy and M.S. and Ph.D. degrees in Pharmacology from Temple University.

Thomas A. Pearson has served as our Senior Vice President, Corporate Strategy and Communications since June 2002. Previously, he served as our Senior Advisor, Corporate Communications and Planning from January 2001 to June 2002, and as our corporate communications consultant from 1992 to January 2001. Mr. Pearson was an independent business consultant since 1983, specializing in biotechnology and high-technology companies. Previously, Mr. Pearson held various management positions in the television stations division of CBS, an entertainment and broadcasting company. Mr. Pearson received his B.A. degree in liberal arts from Wheaton College.

Camille L. Bedrosian, M.D. has served as our Vice President and Chief Medical Officer since September 2002. From 1997 to 2002, Dr. Bedrosian served in the Clinical Research and Development Department of Wyeth/Genetics Institute, Inc., most recently as Senior Director, Oncology/Hematology. From 1986 to 1997, she was a Fellow, an Associate, and then Assistant Professor of Medicine in the Division of Hematology and Oncology at Duke University Medical Center and the Duke Comprehensive Cancer Center. Dr. Bedrosian received her B.A. degree from Harvard University/Radcliffe College in chemistry, her M.S. in biophysics from M.I.T., and her M.D. from Harvard Medical School.

David C. Dalgarno, Ph.D. has served as our Vice President, Physical and Chemical Sciences since November 1999. Previously, he served as our Director, Physical and Chemical Sciences from September 1998 to November 1999 and as our Director, Spectroscopy from October 1996 to August 1998. Prior to joining us in March 1992, Dr. Dalgarno was a scientist at Schering-Plough Corp. focusing on protein structure determination by nuclear magnetic resonance. Dr. Dalgarno received his B.A. and Ph.D. degrees in Chemistry from the University of Oxford. He received his postdoctoral training in Molecular Biophysics and Biochemistry at Yale University.

Maryann G. Krane has served as our Vice President, Regulatory Affairs since May 2001. From September 2000 to May 2001, she served as Senior Director, Regulatory Affairs and Quality Assurance at Avant Immunotherapeutics, Inc., a biotechnology company. From 1986 to 1992 and from 1993 to 2000, Ms. Krane held various positions in regulatory affairs and research at Genetics Institute, Inc., currently a unit of American Home Products Corporation, a diversified healthcare company. Most recently, she was Head, Regulatory Affairs, Global Development of Hemophilia and Oncology Products at Genetics Institute. From August 1992 to April 1993, she was Manager, Regulatory Affairs at Genzyme Corporation, a biotechnology company. Ms. Krane received her B.S. degree in Microbiology from the University of Massachusetts at Amherst, MA.

Tomi K. Sawyer, Ph.D. has served as our Vice President, Drug Discovery since January 1999. Previously, he served as our Director, Drug Discovery – Signal Transduction from October 1997 to December 1998. From July 1993 to September 1997, he was Head and Associate Research Fellow, Structure-Based Design and Chemistry at Parke-Davis Pharmaceutical Research a Division of Warner-Lambert Company, a pharmaceutical company, and Section Director, Peptide and Peptidomimetic Chemistry at Parke-Davis from July 1991 to July 1993. Dr. Sawyer received his B.S. degree in Chemistry from Moorhead State University and his Ph.D. degree in Organic Chemistry from the University of Arizona.

Joseph Bratica has served as our Director of Finance and Controller since January 1999. Previously, he served as our Assistant Controller from January 1997 to December 1998 and as our Accounting Manager from August 1994 to December 1996. Prior to joining us, he was Accounting Manager at Creative BioMolecules, Inc., a biotechnology company, from 1992 to 1994. Mr. Bratica received his B.A. degree in Accounting from Suffolk University.

Vaughn D. Bryson**,** one of our Directors since February 1995, is President of Life Science Advisors, Inc., a healthcare consulting company. Mr. Bryson was a thirty-two year employee of Eli Lilly & Co., a pharmaceutical company, from 1961 to 1993 and served as President and Chief Executive Officer of Eli Lilly from 1991 to 1993. He served as Executive Vice President of Eli Lilly from 1986 until 1991. He also served as a member of Eli Lilly's Board of Directors from 1984 until his retirement in 1993. Mr. Bryson was Vice Chairman of Vector Securities International Inc., an investment-banking firm, from April 1994 to December 1996. He also is a Director of Chiron Corporation, a biotechnology company, AtheroGenics, Inc., a biotechnology company, Amylin Pharmaceuticals, Inc., a biotechnology company, and Quintiles Transnational Corporation, a pharmaceutical services company. He received his B.S. degree in Pharmacy from the University of North Carolina and completed the Sloan Program at the Stanford University Graduate School of Business.

Jay R. LaMarche**,** one of our Directors since January 1992, has served as a financial advisor since November 2000. Previously, he served as our Chief Financial Officer and Treasurer from January 1992 to November 2000 and as our Executive Vice President from March 1997 to November 2000. Mr. LaMarche was our Senior Vice President, Finance from January 1992 to February 1997. Prior to joining us, he was Chief Financial Officer and a Director of ChemDesign Corporation, a fine chemicals manufacturer. Previously, Mr. LaMarche was a partner with Deloitte Haskins & Sells, a public accounting firm. Mr. LaMarche received his B.B.A. degree in Public Accountancy from the University of Notre Dame and served as an officer in the United States Navy.

Frederick S. Schiff, one of our directors since June 2002, is Executive Vice President and Chief Financial Officer of Vital Signs, Inc. Previously, Mr. Schiff held various senior management positions over a period of twenty years at Bristol-Myers Squibb Company (BMS), most recently, from 2001 to 2002, as Senior Vice President and Chief Financial Officer. He also served as Senior Vice President, Financial Operations and Chief Financial Officer, Medicines Business in 2000, Vice President and Controller from 1990 to 1997, and in various financial positions from 1982 to 1989, all at BMS. Previously, he was a principal at Arthur Young & Company and director of auditing at the New York office. He is also a director of Visiting Nurse Services of New York and the Eugene Lang Entrepreneurship Fund of the Graduate Business School of Columbia University. Mr. Schiff received an M.B.A. degree from Columbia University and a B.A. from New York University.

Burton E. Sobel, M.D., one of our directors since June 2002, is E.L. Amidon Professor, Physician-in-Chief, and Professor of Biochemistry at the University of Vermont and a trustee of the Fletcher Allen Health Care Center, Burlington. Previously, he held senior academic and administrative positions at Washington University School of Medicine, from 1973 to 1994, and at the University of California, San Diego, from 1968 to 1973. Dr. Sobel is a director of Scios, Inc., a biopharmaceutical company, and Corvas International, Inc., a biopharmaceutical company. Dr. Sobel completed postgraduate training at the Peter Bent Brigham Hospital, Boston and the National Institutes of Health, Bethesda and received an M.D. from Harvard University and an A.B. from Cornell University.

Raymond S. Troubh**,** one of our Directors since October 1991, has been a financial consultant for more than five years. Prior to this, he was a general partner of Lazard Freres & Co., an investment banking firm, and a governor of the American Stock Exchange. Mr. Troubh is Chairman of the Board of Directors of Enron Corp., an energy distribution company and is a Director of Diamond Offshore Drilling, Inc., a contract drilling company, General American Investors Company, Inc., an investment trust company, Gentiva Health Services, Inc., a healthcare provider, Petrie Stores Corporation, a liquidating trust, Hercules Incorporated, a

specialty chemicals company, Triarc Companies, Inc., a holding company, and WHX Corporation, a steel products company. He received his A.B. degree from Bowdoin College and his L.L.B. degree from Yale Law School.

Elizabeth H.S. Wyatt, one of our directors since June 2002, held various senior management positions over a period of twenty years at Merck & Co., Inc., most recently, from 1992 to 2000, as Vice President, Corporate Licensing. She also served in leadership positions in corporate licensing from 1980 to 1992 at Merck. Previously, she held academic and administrative positions at Harvard Business School, Doyle Dane Bernbach, and Boston College. Ms. Wyatt is a director of Medimmune, Inc., a biopharmaceutical company and Neose Technologies, Inc., a biopharmaceutical company. She received an M.B.A. from Harvard Business School, an M.S. in education from Boston University, and a B.A. from Sweet Briar College, Virginia.

Section 16(a) Beneficial Ownership Reporting Compliance

Section 16(a) of the Securities Exchange Act of 1934 requires our directors and officers, and persons who own more than 10% of the Common Stock, to file with the Securities and Exchange Commission (the "SEC") initial reports of beneficial ownership and reports of changes in beneficial ownership of the Common Stock and our other equity securities. Officers, directors and greater than 10% beneficial owners are required by SEC regulation to furnish us with copies of all Section 16(a) forms they file.

To our knowledge, based solely on review of the copies of such reports furnished to us and written representations that no other reports were required, during the fiscal year ended December 31, 2002, all Section 16(a) filing requirements applicable to its officers, directors and greater than 10% beneficial owners were complied with.

An Annual Statement of Beneficial Ownership on Form 5 is not required to be filed if there are no previously unreported transactions or holdings to report. Nevertheless, we are required to disclose the names of directors, officers and 10% shareholders who did not file a Form 5 unless we have obtained a written statement that no filing is required. At the date of this report, we had not obtained a written statement from Ironwood Capital Management, LLC.

ITEM 11: EXECUTIVE COMPENSATION

The following table sets forth aggregate amounts of compensation paid or accrued by us for the years ended December 31, 2002, 2001 and 2000 for services rendered in all capacities, by our Chief Executive Officer and our four next most highly compensated executive officers as of December 31, 2002 (the "Named Executive Officers").

Summary Compensation Table

		Annual Compensation		Long-Term Compensation	
Name and Principal Position	Year	Base Salary	Bonus (1)	Number of Shares Underlying Options	All Other Compensation (2)
Harvey J. Berger, M.D.	2002	$433,000	$ -0-	150,000	$ 5,297
Chairman, Chief Executive	2001	395,000	-0-	115,000	3,318
Officer and President	2000	363,000	-0-	100,000	3,400
David L. Berstein, Esq.	2002	242,000	70,000	50,000	6,621
Senior Vice President and	2001	221,000	60,000	60,000	3,951
Chief Patent Counsel	2000	200,000	47,000	35,000	3,082
Fritz Casselman	2002	227,000	60,000	45,000	6,858
Senior Vice President and	2001	180,923	-0-	200,000	3,896
Chief Business Officer	2000	-0-	-0-	-0-	-0-
Timothy P. Clackson, Ph.D.	2002	213,000	65,000	75,000	8,146
Senior Vice President,	2001	165,577	65,000	60,000	3,979
Science and Technology	2000	129,770	31,000	25,000	2,595
John D. Iuliucci, Ph.D.	2002	242,000	70,000	70,000	7,210
Senior Vice President,	2001	221,000	60,000	55,000	5,016
Drug Development	2000	207,500	37,000	-0-	16,485

(1) The amounts listed are for bonuses awarded and deferred under our 1997 Executive Compensation Plan, a non-qualified, unfunded, deferred compensation plan. The amounts awarded vest over a four-year period commencing on the first anniversary of the date of the award.

(2) The amounts listed for each year consist of our matching contributions of up to $6,000 per year under our 401(k) Plan and, in the case of Mr. Berstein, Mr. Casselman, Dr. Clackson and Dr. Iuliucci, include the aggregate difference between the fair market value and the purchase cost of common stock purchased during fiscal year 2002 under our 1997 Employee Stock Purchase Plan. Dr. Berger is not eligible to participate in our Employee Stock Purchase Plan.

Stock Options

The following table sets forth information regarding each stock option granted during the fiscal year ended December 31, 2002 to each of the Named Executive Officers.

Stock Option Grants in Last Fiscal Year

	Individual Grants				Potential Realizable Value at Assumed Annual Rates of Stock Price Appreciation for Option Term (2)	
Name	Number of Shares Underlying Options Granted (1)	Percent of Total Options Granted to Employees in Fiscal Year	Exercise Price (per share)	Expiration Date	5%	10%
Harvey J. Berger, M.D.	20,000(3)	1.5%	$4.44	03/07/12	$ 55,846	$141,524
	130,000(4)	9.7%	4.05	06/12/12	331,113	839,105
David L. Berstein, Esq.	50,000(4)	3.7%	4.19	06/18/12	131,753	333,889
Fritz Casselman	45,000(4)	3.4%	4.19	06/18/12	118,578	300,500
Timothy Clackson, Ph.D.	25,000(4)	1.9%	4.05	06/13/12	63,676	161,366
	50,000(4)	3.7%	4.19	06/18/12	131,753	333,889
John D. Iuliucci, Ph.D.	20,000(3)	1.5%	4.44	03/07/12	55,846	141,524
	50,000(4)	3.7%	4.19	06/18/12	131,753	333,889

(1) Options to purchase shares of our common stock under the 2001 Stock Plan.

(2) These amounts, based on assumed annual appreciation rates of 5% and 10% as prescribed by the rules of the SEC, are for illustration purposes only and are not intended to forecast possible future appreciation, if any, of our stock price. Actual gains, if any, on stock option exercises will depend on the future performance of our common stock, the option holder's continued employment with us through the option exercise period and the date on which the option is exercised.

(3) Options fully vested upon issuance.

(4) Options vest annually over four years commencing on the first anniversary of the award.

Aggregated Option Exercises in Last Fiscal Year and Fiscal Year-End Option Values

The following table provides information regarding the exercise of options by each of the Named Executive Officers during the fiscal year ended December 31, 2002. In addition, this table includes the number of shares covered by both exercisable and unexercisable stock options as of December 31, 2002 and the values of "in-the-money" options, which values represent the positive spread between the exercise price of any such option and either the actual or estimated fair market value of the underlying security as of December 31, 2002, as applicable.

Aggregated Option Exercises in Last Fiscal Year and Fiscal Year-End Option Values

Name	Shares Acquired on Exercise (#)	Value Realized	No. of Shares Underlying Unexercised Options at Fiscal Year-End Exercisable/ Unexercisable	Value of Unexercised In-the-Money Options at Fiscal Year-End
Harvey J. Berger, M.D.	235,714(1)	$353,571	470,250/283,750(2)	$324,363/16,538(4)
	0	0	1,402/0(3)	0/0(5)
David L. Berstein, Esq.	0	0	158,214/117,500(2)	109,908/4,900(4)
	0	0	112/0(3)	0/0(5)
Fritz Casselman	0	0	50,000/195,000(2)	0/0(4)
Timothy P. Clackson, Ph.D.	0	0	43,760/138,900(2)	6,935/6,935(4)
John D. Iuliucci, Ph.D.	58,928	129,858	202,750/96,200(2)	142,960/4,900(4)
	0	0	280/0(3)	0/0(5)

(1) Options exercised are shares of our common stock held by The Berger Family Trust.

(2) Options to purchase shares of our common stock.

(3) Options to purchase common stock of our subsidiary, AGTI.

(4) Based upon a fair market value of $2.32 per share of common stock, which was the closing price of a share of our common stock on the NASDAQ National Market on December 31, 2002, less the per share exercise price.

(5) Based upon an estimated value of the common stock of AGTI, for which there was no public market on December 31, 2002, less the per share exercise price.

Director Compensation

Effective as of 2001, members of the Board of Directors, other than Dr. Berger, receive an annual award of options to purchase 15,000 shares of our common stock, which are exercisable on the date of grant. On December 3, 2002, each such director was awarded options to purchase 15,000 shares of our common stock at $2.66 per share, pursuant to our stock options plans. Such options were fully exercisable on the grant date. Mr. Smith receives $4,000 per month for his services as Vice Chairman of the Board of Directors. No other non-employee director receives any cash compensation for service on the Board of Directors or its committees. Mr. LaMarche receives $42,000 per year for his services as a financial advisor to the Company. Directors are reimbursed for their expenses for each meeting they attend.

Employment Agreements with Named Executive Officers

Dr. Berger, our Chairman of the Board of Directors, Chief Executive Officer and President, has an employment agreement with us which commenced in January 1992 and terminates in December 2004. Dr. Berger's employment agreement is automatically renewable for successive three-year terms unless terminated by either party. The agreement provides that he shall be employed as our Chief Executive

Officer and President, shall be nominated for election to our Board of Directors, serve as Chairman of the Board and receive an annual base salary during 2002 of $433,000, increasing each year by at least 10% of the preceding year's base salary. Dr. Berger is eligible each year to receive a discretionary bonus, determined by the Board of Directors, of up to 50% of his annual base salary. If we fail to renew the employment agreement, we are obligated to pay Dr. Berger, in addition to his compensation for the remainder of the term, a lump sum payment equal to two times Dr. Berger's annual salary for the final year of the term and to provide for the immediate exercisability of all stock options and other equity rights.

Dr. Berger's employment agreement provides that, if the agreement is terminated by either party upon the occurrence of certain events, including (i) our sale or merger (or stockholder approval of a merger agreement) or an acquisition of a substantial equity interest in us by a person or group of persons, (ii) if Dr. Berger is not elected to membership on our Board of Directors, named as Chairman or designated as Chief Executive Officer or ceases to be our highest ranking executive officer or ceases to control personnel decisions with respect to our employees, (iii) if we are in material breach of the terms of his employment agreement, (iv) if we are bankrupt or insolvent or (v) if we terminate Dr. Berger's employment agreement without cause, (1) we will pay Dr. Berger the greater of (x) any remaining salary payable during the term of the agreement plus the maximum possible bonus for each year remaining in the term (taking into account, in both cases, obligated 10% increases in salary) and (y) an amount equal to twice his current annual salary and maximum bonus for the current year of employment (the "Severance Payment") and (2) all of his stock options, stock awards and similar equity rights will immediately vest and become exercisable. We are not obligated to make the Severance Payment if we discharge Dr. Berger for cause. If the vesting of certain benefits and the payment of certain amounts by us to Dr. Berger are treated as payments in the nature of compensation that are contingent on a "change in control" (within the meaning of Section 280G of the Internal Revenue Code of 1986, as amended (the "Code")), the deductibility of such payments could, depending upon the aggregate amount of such payments, be disallowed pursuant to Section 280G of the Code and an excise tax could be imposed on Dr. Berger pursuant to Section 4999 of the Code for which he would, pursuant to the employment agreement, be indemnified by us on a net after-tax basis. The employment agreement contains a non-competition provision that is effective during the term of the agreement and, if Dr. Berger is terminated for cause, for a period of one year following the date of termination.

We also entered into employment agreements with Mr. Berstein, Mr. Casselman, Dr. Clackson and Dr. Iuliucci. The agreements provide for employment through December 31, 2005 for Mr. Berstein and Dr. Iuliucci and Dr. Clackson, and through February 28, 2004 for Mr. Casselman, at annual base salaries during 2002 of $242,000, $242,000, $213,000 and $227,000, respectively, increasing each year by an amount to be determined by the Board of Directors. In addition, each executive is eligible each year to receive a performance bonus, to be determined by the Board of Directors, of up to 30% of his annual base salary, which may be paid in the form of deferred compensation under the 1997 Executive Compensation Plan, awards of our stock options, or cash. The agreements are renewable for successive one-year terms with the mutual consent of the parties.

Our agreements with Mr. Berstein, Mr. Casselman, Dr. Clackson and Dr. Iuliucci also provide that (i) upon a change of control, such officers will be entitled to receive, upon termination by the officer within 90 days after the change in control, any remaining salary payable during the term or six months' salary, whichever is less, and all stock options held by such officers will immediately vest and become exercisable; and (ii) upon termination by us, without cause, such officer will be entitled to receive his current salary for the remaining period of the applicable term and all outstanding options that would have vested during such term shall vest immediately.

Compensation Committee Interlocks and Insider Participation

During the fiscal year ended December 31, 2002, the Compensation Committee was comprised of Dr. Sobel and Messrs. Smith and Troubh. None of our executive officers serve on the Board of Directors or Compensation Committee of any entity that has one or more executive officers serving as a member of our Board of Directors or Compensation Committee. There is no family relationship between or among the members of our Board of Directors or executive officers.

ITEM 12: SECURITY OWNERSHIP OF CERTAIN BENEFICIAL OWNERS AND MANAGEMENT AND RELATED STOCKHOLDERS MATTERS

The following table sets forth, as of March 12, 2003, certain information with respect to (i) each person (including any "group" as defined in Section 13(d)(3) of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), known to us to own beneficially more than 5% of our Common Stock, (ii) each of our directors, (iii) each executive officer named in the Summary Compensation Table under "Executive Compensation" and (iv) all directors and executive officers as a group. In accordance with the rules promulgated by the SEC, such ownership includes shares currently owned, as well as shares that the named person has the right to acquire within 60 days of March 12, 2003, including, but not limited to, shares that the named person has the right to acquire through the exercise of any option. Except as otherwise indicated, we believe that the stockholders listed in the table have sole voting and investment powers with respect to the Common Stock shown as beneficially owned by them based on information provided to us by these stockholders. Percentage ownership is based on 34,846,640 shares of our common stock outstanding as of March 12, 2003.

Stock Ownership by Management, Directors and 5% Beneficial Owners

Name and Address **	Number and Nature of Shares Beneficially Owned ***		Percent of Class
Ironwood Capital Management, LLC 21 Custom House Street, Boston, MA 02110	3,876,595	(1)	11.1%
Harvey J. Berger, M.D.	1,738,997	(2)	4.9%
David L. Berstein, Esq	169,793	(3)	*
Fritz Casselman	106,203	(4)	*
Timothy P. Clackson, Ph.D.	61,700	(5)	*
John D. Iuliucci, Ph.D.	233,450	(6)	*
Vaughn D. Bryson	155,500	(7)	*
Jay R. LaMarche	477,856	(8)	1.4%
Frederick S. Schiff	15,000	(9)	*
Sandford D. Smith	191,705	(10)	*
Burton E. Sobel, M.D.	20,000	(11)	*
Raymond S. Troubh	201,749	(12)	*
Elizabeth H.S. Wyatt	15,000	(13)	*
All directors and executive officers as a group (15 persons)	3,605,053	(14)	9.9%

* Indicates less than one percent of the outstanding shares of common stock.

** Addresses are given for beneficial owners of more than 5% of the outstanding common stock only.

*** Attached to each share of common stock is a preferred share purchase right to acquire a number of shares of common stock having a market value at that time of twice the rights' exercise price, which rights are not presently exercisable.

(1) Such shares are held of record by Ironwood Capital Management, LLC. This information is based solely on review of Schedule 13G, which was filed with the Commission on March 14, 2003.

(2) Includes 487,750 shares issuable upon exercise of stock options. Includes 771,428 shares of Common Stock held of record by The Berger Family Trust and 8,928 shares of Common Stock held of record by the Wolk Family Trust. Wendy S. Berger and Harvey J. Berger, as co-trustees of such trusts, have the right to vote and dispose of the shares held by such trusts; however, in certain circumstances, Wendy S. Berger as co-trustee will have sole voting power with respect to the shares held by each such trust. Includes 40,892 shares held by Wendy S. Berger, Dr. Berger's spouse, and 13,928 shares held by Dr. Berger's children.

(3) Includes 163,214 shares issuable upon exercise of stock options.

(4) Includes 100,000 shares issuable upon exercise of stock options.

(5) Includes 48,910 shares issuable upon exercise of stock options.

(6) Includes 207,750 shares issuable upon exercise of stock options.

(7) Includes 113,000 shares issuable upon exercise of stock options.

(8) Includes 147,750 shares issuable upon exercise of stock options and 6,696 shares held by Carol B. LaMarche, Mr. LaMarche's spouse.

(9) Consists of 15,000 shares issuable upon exercise of stock options.

(10) Includes 163,357 shares issuable upon exercise of stock options.

(11) Consists of 20,000 shares issuable upon exercise of stock options.

(12) Includes 145,500 shares issuable upon exercise of stock options.

(13) Consists of 15,000 shares issuable upon exercise of stock options.

(14) Includes 1,844,731 shares issuable upon the exercise of stock options held by all directors and executive officers as a group.

Securities Authorized for Issuance Under Equity Compensation Plans

The following table sets forth information regarding compensation plans involving our common stock in effect as of December 31, 2002

Equity Compensation Plan Information

Plan Category	Number of Securities to be Issued Upon Exercise of Outstanding Options	Weighted Average Exercise Price of Outstanding Options	Number of Securities Remaining Available for Future Issuance Under Equity Compensation Plans (excluding securities reflected in first column)
Equity Compensation Plans Approved by Security Holders (a)	5,392,311	$4.51	794,044
Equity Compensation Plans not Approved by Security Holders	N/A	N/A	N/A
Total	5,392,311	$4.51	794,044

(a) Consists of options to purchase 2,841,355 shares of common stock granted under our 1991 Stock Option Plans for Employees, Consultants and Directors, options to purchase 415,000 shares of common stock granted under our 1994 Stock Option Plan for Non-Employee Directors, and options to purchase 2,135,956 shares of common stock granted under our 2001 Stock Plan.

ITEM 13: CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS

Our subsidiary, AGTI, holds licenses from Harvard University, Stanford University and other universities relating to our ARGENT cell-signaling regulation technology, a key component of our programs in regulated protein therapy and cellular therapy and owns the intellectual property on our mTOR inhibitors derived from our ARGENT programs. The two directors of AGTI are also directors of the Company. Minority stockholders of AGTI, including Harvard University, Stanford University, several of our scientific advisors, and several current and former members of our management and Board of Directors, own 20% of the issued and outstanding capital stock of AGTI. We own the remaining 80% of the issued and outstanding capital stock of AGTI. Four members of our management team and/or Board of Directors own or have the right to acquire up to approximately 6.1% of the outstanding capital stock of AGTI. Harvey J. Berger, M.D. owns 3.4%; David L. Berstein, Esq. owns 0.3%; John D. Iuliucci, Ph.D. owns 0.7%; and Jay R. LaMarche owns 1.7%. AGTI has a right of first refusal on the sale to third parties of 73% of the minority stockholders' AGTI shares. AGTI does not have a call option, or a right to require the minority stockholders to sell their shares to us, for any of these shares. As part of the formation of AGTI, we entered into agreements with AGTI to provide for the operations of AGTI.

As part of an employment agreement entered into as of March 4, 2002, we extended a $75,000 relocation loan to Laurie A. Allen, our Senior Vice President and Chief Legal Officer and Secretary, pursuant to a promissory note, and secured by a second mortgage on her residence in Massachusetts. The loan will be forgiven on the third anniversary of the issue date, based on Ms. Allen's continuous service with us. In the event that Ms. Allen terminates her employment prior to such third anniversary, the principal is due and payable within ninety days thereafter, and any unpaid balance as of the due date shall bear interest at a rate of 7% per annum.

ITEM 14: CONTROLS AND PROCEDURES

(a) *Evaluation of Disclosure Controls and Procedures.* Our executive officer and principal financial officer, after evaluating the effectiveness of our disclosure controls and procedures (as defined in Exchange Act Rules 13a-14(c) and 15d-14(c)) as of a date within 90 days of the filing date of this Annual Report on Form 10-K, have concluded that, based on such evaluation, our disclosure controls and procedures were adequate and effective to ensure that material information relating to the Company, including its consolidated subsidiaries, was made known to them by others within those entities, particularly during the period in which this Annual Report on Form 10-K was being prepared.

(b) *Changes in Internal Controls.* There were no significant changes in our internal controls or in other factors that could significantly affect these controls subsequent to the date of their evaluation, nor were there any significant deficiencies or material weaknesses in the our internal controls. Accordingly, no corrective actions were required or undertaken.

PART IV

ITEM 15: EXHIBITS, FINANCIAL STATEMENT SCHEDULES AND REPORTS ON FORM 8-K

(a)(1) The following Consolidated Financial Statements, Notes thereto and Independent Auditors' Report have been presented in Item 8:

Independent Auditors' Report

Consolidated Balance Sheets

Consolidated Statements of Operations

Consolidated Statements of Stockholders' Equity

Consolidated Statements of Cash Flows

Notes to Consolidated Financial Statements

(a)(2) Financial Statement Schedules:

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

(a)(3) The Exhibits listed in the Exhibit Index are filed herewith in the manner set forth therein.

(b) Reports on Form 8-K

We filed a Current Report on Form 8-K on November 1, 2002 announcing that our novel anemia product candidate produced therapeutically effective amounts of the hormone erythropoietin (EPO) more than four years after one-time injection on monkeys of our Company's patented gene complex.

We filed a Current Report on Form 8-K on November 6, 2002 announcing a non-exclusive worldwide license agreement with Bristol-Myers Squibb Company granting Bristol-Myers Squibb the right to conduct pharmaceutical research and development covered by our NF-κB drug discovery patents.

We filed a Current Report on Form 8-K on November 12, 2002 announcing the commencement of the offering of 2.2 million shares of our common stock, par value $0.001 per share, to new and existing investors at a purchase price of $2.75 per share pursuant to a Form S-3 Shelf Registration Statement (Registration No. 333-63708) and the related Prospectus and Prospectus Supplement. Rodman & Renshaw, Inc. served as placement agent for the offering.

We filed a Current Report on Form 8-K on November 12, 2002 announcing that we had entered into definitive agreements with selected existing and new institutional investors for the purchase of 2.2 million shares of our common stock, referred to in the previous Current Report on Form 8-K, at $2.75 per share for gross proceeds of $6.1 million.

SIGNATURES

Pursuant to the requirements of Section 13 or 15(d) of the Securities Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized, in the City of Cambridge and Commonwealth of Massachusetts on the 14th of March, 2003.

ARIAD PHARMACEUTICALS, INC

By: /s/ Harvey J. Berger, M.D.

Name: Harvey J. Berger, M.D.
Title: Chairman, Chief Executive Officer and President

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

Signature	Title	Date
/s/ Harvey J. Berger, M.D. **Harvey J. Berger, M.D.**	**Chairman of the Board of Directors, Chief Executive Officer and President (Principal Executive Officer)**	**March 14, 2003**
/s/ Sandford D. Smith **Sandford D. Smith**	**Vice Chairman of the Board of Directors**	**March 14, 2003**
/s/ Edward M. Fitzgerald **Edward M. Fitzgerald**	**Senior Vice President, Chief Financial Officer (Principal Financial Officer and Principal Accounting Officer)**	**March 14, 2003**
/s/ Vaughn D. Bryson **Vaughn D. Bryson**	**Director**	**March 14, 2003**
/s/ Jay R. LaMarche **Jay R. LaMarche**	**Director**	**March 14, 2003**
/s/ Frederick S. Schiff **Frederick S. Schiff**	**Director**	**March 14, 2003**
/s/ Burton E. Sobel, M.D. **Burton E. Sobel, M.D.**	**Director**	**March 14, 2003**
/s/ Raymond S. Troubh **Raymond S. Troubh**	**Director**	**March 14, 2003**
/s/ Elizabeth H.S. Wyatt **Elizabeth H.S. Wyatt**	**Director**	**March 14, 2003**

CERTIFICATIONS

I, Harvey J. Berger, M.D., certify that:

1. I have reviewed this annual report on Form 10-K of ARIAD Pharmaceuticals, Inc.;

2. Based on my knowledge, this annual report does not contain any untrue statement of material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this annual report;

3. Based on my knowledge, the financial statements, and other financial information included in this annual report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this annual report;

4. The registrant's other certifying officers and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-14 and 15d-14) for the registrant and have:

   a) designed such disclosure controls and procedures to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this annual report is being prepared;

   b) evaluated the effectiveness of the registrant's disclosure controls and procedures as of a date within 90 days prior to the filing date of this annual report (the "Evaluation Date"); and

   c) presented in this annual report our conclusions about the effectiveness of the disclosure controls and procedures based on our evaluation as of the Evaluation Date;

5. The registrant's other certifying officers and I have disclosed, based on our most recent evaluation, to the registrant's auditors and the audit committee of registrant's board of directors (or persons performing the equivalent functions):

   a) all significant deficiencies in the design or operation of internal controls which could adversely affect the registrant's ability to record, process, summarize and report financial data and have identified for the registrant's auditors any material weaknesses in internal controls; and

   b) any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal controls; and

6. The registrant's other certifying officers and I have indicated in this annual report whether there were significant changes in internal controls or in other factors that could significantly affect internal controls subsequent to the date of our most recent evaluation, including any corrective actions with regard to significant deficiencies and material weaknesses.

March 14, 2003

/s/ Harvey J. Berger, M.D.

Harvey J. Berger, M.D.
Chairman of the Board of Directors, Chief Executive Officer and President

CERTIFICATIONS

I, Edward M. Fitzgerald, certify that:

1. I have reviewed this annual report on Form 10-K of ARIAD Pharmaceuticals, Inc.;

2. Based on my knowledge, this annual report does not contain any untrue statement of material fact or omit to state a material fact necessary to make the statements made, in light of the circumstances under which such statements were made, not misleading with respect to the period covered by this annual report;

3. Based on my knowledge, the financial statements, and other financial information included in this annual report, fairly present in all material respects the financial condition, results of operations and cash flows of the registrant as of, and for, the periods presented in this annual report;

4. The registrant's other certifying officers and I are responsible for establishing and maintaining disclosure controls and procedures (as defined in Exchange Act Rules 13a-14 and 15d-14) for the registrant and have:

   a) designed such disclosure controls and procedures to ensure that material information relating to the registrant, including its consolidated subsidiaries, is made known to us by others within those entities, particularly during the period in which this annual report is being prepared;

   b) evaluated the effectiveness of the registrant's disclosure controls and procedures as of a date within 90 days prior to the filing date of this annual report (the "Evaluation Date"); and

   c) presented in this annual report our conclusions about the effectiveness of the disclosure controls and procedures based on our evaluation as of the Evaluation Date;

5. The registrant's other certifying officers and I have disclosed, based on our most recent evaluation, to the registrant's auditors and the audit committee of registrant's board of directors (or persons performing the equivalent functions):

   a) all significant deficiencies in the design or operation of internal controls which could adversely affect the registrant's ability to record, process, summarize and report financial data and have identified for the registrant's auditors any material weaknesses in internal controls; and

   b) any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal controls; and

6. The registrant's other certifying officers and I have indicated in this annual report whether there were significant changes in internal controls or in other factors that could significantly affect internal controls subsequent to the date of our most recent evaluation, including any corrective actions with regard to significant deficiencies and material weaknesses.

March 14, 2003

/s/ Edward M. Fitzgerald

Edward M. Fitzgerald
Senior Vice President,
Chief Financial Officer

EXHIBIT INDEX

Exhibit No.	Title
3.1	Certificate of Incorporation of the Company, as amended. (1)
3.2	Restated By-laws of the Company, as amended. (6)
3.3	Amendment of Certificate of Incorporation of the Company, dated April 8, 1994. (2)
3.4	Amendment of Certificate of Incorporation of the Company, dated October 4, 1994. (5)
3.5	Certificate of Designations in respect of Series A Preferred Stock of the Company dated June 19, 2000. (4)
4.1	Principal Stockholders' Agreement, dated as of January 5, 1992, among ARIAD Pharmaceuticals, Inc., David Blech, David Blech as trustee of The Blech Family Trust, Mark S. Germain, Harvey J. Berger, Harvey J. Berger and Wendy S. Berger as Trustees of the Berger Family Trust, Avalon Ventures and Avalon Ventures IV. (1)
4.2	Rights Agreement, dated as of June 8, 2000, between the Company and State Street Bank and Trust Company, which includes the Form of Certificate of Designations in respect of the Series A Preferred Stock, as Exhibit A, the Form of Right Certificate as Exhibit B and the Summary of Rights to Purchase Series A Preferred Stock as Exhibit C. Pursuant to the Rights Agreement, Right Certificates will not be mailed until after the Separation Date (as defined therein). (4)
4.3	Stock Purchase Agreement, dated as of April 24, 1995, between ARIAD Pharmaceuticals, Inc. and Biotech Target S.A. (7)
10.1	Lease Agreement, dated January 8, 1992, between ARIAD Pharmaceuticals, Inc. and Forest City Cambridge, Inc. (1)
10.2+	Executive Employment Agreement, dated as of January 1, 1992, between ARIAD Pharmaceuticals, Inc. and Harvey J. Berger, M.D. (1)
10.3+	Executive Employment Agreement, dated as of January 1, 1992, between ARIAD Pharmaceuticals, Inc. and Jay R. LaMarche. (1)
10.4	Loan and Security Agreement, dated September 23, 1992, by and between ARIAD Pharmaceuticals, Inc., ARIAD Corporation and BayBank Boston, N.A. and related instruments and documents. (1)
10.5+	ARIAD Pharmaceuticals, Inc. 1991 Stock Option Plan for Employees, as amended. (5)
10.6+	ARIAD Pharmaceuticals, Inc. 1991 Stock Option Plan for Directors. (1)
10.7+	ARIAD Retirement Savings Plan. (1)
10.8**	Amended and Restated Agreement, dated as of December 12, 1997, between The Board of Trustees of The Leland Stanford Junior University and ARIAD Gene Therapeutics, Inc. (9)
10.9+	Amendment, dated April 19, 1994, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Harvey J. Berger, M.D.(3)
10.10+	Amendment, dated March 2, 1994, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Jay R. LaMarche. (3)
10.11+	Amendment No. 2, dated June 30, 1994, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Harvey J. Berger, M.D. (5)
10.12+	ARIAD Pharmaceuticals, Inc. 1994 Stock Option Plan for Non-Employee Directors. (5)
10.13**	License Agreement, dated as of September 12, 1996, between Mochida Pharmaceuticals Co., Ltd. and ARIAD Pharmaceuticals, Inc. (8)

Exhibit No.	Title
1014+	Amendment, dated January 1, 1997, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Harvey J. Berger, M.D. (23)
10.15+	Amendment, dated January, 1, 1997, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Jay R. LaMarche (23)
10.16+	ARIAD Pharmaceuticals, Inc. 1997 Employee Stock Purchase Plan. (23)
10.17+	Amendment to the 1991 Stock Option Plan for Employees and Consultants. (23)
10.18+	Amendment to the 1994 Stock Option Plan for Non-Employee Directors. (23)
10.19	Fourth Amendment to Loan and Security Agreement, dated June 27, 1997, with BankBoston, N.A. as successor in interest to BayBank, N.A. (23)
10.20**	License Agreement, dated July 17, 1997, between ARIAD Pharmaceuticals, Inc. and Mitotix, Inc. (24)
10.21**	Technology Purchase and Sale Agreement and related agreements, dated July 17, 1997, between ARIAD Pharmaceuticals, Inc. and Mitotix, Inc. (24)
10.22+	ARIAD Pharmaceuticals, Inc. 1997 Executive Compensation Plan. (9)
10.23+	Amendment, dated November 10, 2000, to Executive Employment Agreement between ARIAD Pharmaceuticals, Inc. and Jay R. LaMarche. (14)
10.24+	Executive Employment Agreement, dated January 24, 2001, between ARIAD Pharmaceuticals, Inc. and Fritz Casselman. (15)
10.25	Common Stock Purchase Agreement, dated as of June 27, 2000, by and between ARIAD Pharmaceuticals, Inc. and Acqua Wellington North American Equities Fund, Ltd. (10)
10.26	Common Stock Purchase Agreement, dated as of June 27, 2000, by and between ARIAD Pharmaceuticals, Inc. and the Purchaser named therein. (10)
10.27+	Executive Employment Agreement, dated May 1, 1992, Fourth Amendment to Employment Agreement dated June 8, 2000, Third Amendment to Employment Agreement dated January 1, 1999, and Amendments to Employment Agreements dated January 1, 1997 and March 2, 1994 between ARIAD Pharmaceuticals, Inc. and John Iuliucci, Ph.D. (11)
10.28+	Executive Employment Agreement, dated August 1, 1993, Third Amendment to Employment Agreement dated June 8, 2000, and Amendments to Employment Agreements dated January 1, 1997 and March 2, 1994 between ARIAD Pharmaceuticals, Inc. and David L. Berstein, J.D.(11)
10.29**	Restructuring Agreement, dated December 31, 1999, by and between Hoechst Marion Roussel (France) and ARIAD Pharmaceuticals, Inc. (**)(12)
10.30**	Restructuring Agreement, dated December 31, 1999, by and among Aventis Pharmaceuticals, Inc., the Hoechst-ARIAD Genomics Center, LLC and ARIAD Pharmaceuticals, Inc. (**)(12)
10.31	Settlement and Repurchase Agreement by and among ARIAD Pharmaceuticals, Inc., Promethean Investment Group LLC and HFTP Investments, LLC, dated as of January 14, 2000. (13)
10.32+	Amendment, dated as of January 1, 2001, to Executive Employment Agreement with John Iuliucci, Ph.D. (15)
10.33+	Amendment, dated as of January 1, 2001, to Executive Employment Agreement with David Berstein, Esq. (15)
10.34+	ARIAD Pharmaceuticals, Inc. 2001 Stock Plan. (16)
10.35	December 2001 Amendment to Loan and Security Agreement, dated December 26, 2001, by and among ARIAD Pharmaceuticals, Inc., ARIAD Corporation, ARIAD Gene Therapeutics, Inc. and Fleet National Bank, as successor in interest to BankBoston, N.A.,which includes as Exhibit A, the Second Amended and Restated Secured Term Note dated as of December 26, 2001(17)
10.36	Master Security Agreement, dated as of December 27, 2001, by and between ARIAD Pharmaceuticals, Inc. and General Electric Capital Corporation. (17)
10.37	Financial Covenants Addendum No. 001 to Master Lease Agreement between ARIAD Pharmaceuticals, Inc. and General Electric Capital Corporation. (17)
10.38+	Promissory Note to General Electric Capital Corporation dated as of December 28, 2001. (17)

Exhibit No.	Title
10.39	Revised and Restated Research and Development Agreement, dated as of March 15, 2002, by and between ARIAD Pharmaceuticals, Inc. and ARIAD Corporation. (17)
10.40	Revised and Restated Research and Development Agreement, dated as of March 15, 2002, by and between ARIAD Gene Therapeutics, Inc. and ARIAD Corporation. (17)
10.41+	Executive Employment Agreement, dated as of March 4, 2002, between ARIAD Pharmaceuticals, Inc. and Laurie A. Allen, Esq. (17)
10.42	Stock Transfer Agreement between ARIAD Gene Therapeutics, Inc. and the individuals listed on Exhibit A thereto. (17)
10.43	Notice of Extension of Lease, dated October 2, 2001, from ARIAD Corporation to Forest City Commercial Group. (17)
10.44+	Executive Employment Agreement, dated May 6, 2002, between ARIAD Pharmaceuticals, Inc. and Edward M. Fitzgerald (18)
10.45+	ARIAD Pharmaceuticals, Inc. 2001 Stock Plan, as amended (19)
10.46+	Promissory Note issued pursuant to Executive Employment Agreement, dated as of March 4, 1992, by and between ARIAD Pharmaceuticals, Inc. and Laurie A. Allen, Esq., dated as of July 24, 2002 (20)
10.47	Letter Agreement, dated November 7, 2002, between ARIAD Pharmaceuticals, Inc. and Rodman & Renshaw, Inc. (21)
10.48	Amendment to Loan and Security Agreement, dated September 30, 2002, by and among ARIAD Pharmaceuticals, Inc., ARIAD Corporation, ARIAD Gene Therapeutics, Inc. and Fleet National Bank. (22)
10.49+*	Executive Employment Agreement, dated June 8, 2000, between ARIAD Pharmaceuticals, Inc. and Timothy Clackson, Ph.D.
10.50+*	Amendment to Employment Agreement, dated July 1, 2001, between ARIAD Pharmaceuticals, Inc. and Timothy Clackson, Ph.D.
10.51+*	Amendment to Employment Agreement, dated July 12, 2002, between ARIAD Pharmaceuticals, Inc. and Timothy Clackson, Ph.D.
10.52+*	Executive Employment Agreement, dated July 1, 2002, between ARIAD Pharmaceuticals, Inc. and Thomas A. Pearson.
10.53*	Form of Stock Purchase Agreement, dated November 8, 2002, between ARIAD Pharmaceuticals, Inc. and each of the parties thereto.
10.54*	Letter Agreement, dated December 27, 2002, by and among ARIAD Pharmaceuticals, Inc., ARIAD Corporation, ARIAD Gene Therapeutics, Inc. and Fleet National Bank.
10.55*	Agreement of Sublease, dated December 31, 1999, between ARIAD Corporation and Aventis Pharmaceuticals Inc.
10.56*	First Amendment to Sublease, dated July 26, 2002, between ARIAD Corporation and Aventis Pharmaceuticals Inc.
21.1	Subsidiaries of the Company. (3)
23.1*	Consent of Deloitte & Touche LLP.
99.1*	Certification Pursuant to Section 906 of the Sarbanes-Oxley Act of 2002.

Notes to Exhibits:

(+)	Management Contract or Compensatory Plan or Arrangement
(*)	Filed Herewith.
(**)	Confidential treatment has been granted by the Securities and Exchange Commission as to certain portions.
(1)	Incorporated by reference to Registration Statement on Form 10 of the Company filed with the Securities and Exchange Commission on June 25, 1993.
(2)	Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 1993 filed with the Securities and Exchange Commission on April 15, 1994.
(3)	Incorporated by reference to Registration Statement on Form S-1 of the Company (No. 33-76414) filed with the Securities and Exchange Commission on March 11, 1994.

(4) Incorporated by reference to Form 8-A of the Company filed with the Securities and Exchange Commission on June 19, 2000.
(5) Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 1994 filed with the Securities and Exchange Commission on March 31, 1995.
(6) Incorporated by reference to Amendment No. 1 to the Registration Statement on Form S-3 of the Company (No. 333-38664) filed with the Securities and Exchange Commission on June 23, 2000.
(7) Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 1995 filed with the Securities and Exchange Commission on March 8, 1996.
(8) Incorporated by reference to Forms 10-Q of the Company filed with the Securities and Exchange Commission on May 3, 1997.
(9) Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 1997 filed with the Securities and Exchange Commission on March 10, 1998.
(10) Incorporated by reference to Form 8-K of the Company filed with the Securities and Exchange Commission on July 7, 2000.
(11) Incorporated by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on August 10, 2000.
(12) Incorporated by reference to Form 8-K of the Company, dated December 31, 1999 and filed with the Securities and Exchange Commission on January 18, 2000.
(13) Incorporated by reference to Form 8-K of the Company, dated January 14, 2000 and filed with the Securities and Exchange Commission on January 18, 2000.
(14) Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 2000 filed with the Securities and Exchange Commission on March 29, 2001.
(15) Incorporated herein by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on May 14, 2001.
(16) Incorporated herein by reference to Form S-8 of the Company (No. 333-63706) filed with the Securities and Exchange Commission on June 22, 2001.
(17) Incorporated by reference to Form 10-K of the Company for the fiscal year ended December 31, 2001 filed with the Securities and Exchange Commission on March 22, 2002.
(18) Incorporated herein by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on May 9, 2002.
(19) Incorporated by reference to Registration Statement on Form S-8 of the Company (No. 333-90480) filed with the Securities and Exchange Commission on June 14, 2002.
(20) Incorporated herein by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on July 31, 2002.
(21) Incorporated herein by reference to Form 8-K of the Company filed with the Securities and Exchange Commission on November 12, 2002.
(22) Incorporated herein by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on November 14, 2002.
(23) Incorporated by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on August 12, 1997.
(24) Incorporated by reference to Form 10-Q of the Company filed with the Securities and Exchange Commission on November 12, 1997.