

ANORMED INC.

ANNUAL INFORMATION FORM

FOR THE FISCAL YEAR ENDED MARCH 31, 2002

AUGUST 16, 2002

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GENERAL DEVELOPMENT OF THE BUSINESS

Company Overview

We are focused on the discovery and development of new drugs for the treatment of a variety of diseases including cancer, HIV and inflammation. Our core strengths involve the application of chemistry, biochemistry and biology in order to generate small molecules (chemical compounds) with therapeutic potential. We have four products in our pipeline that are in various stages of clinical development: Fosrenol, ApomateTM, AMD-473 and AMD-3100, and a research program focused on a new class of drugs that target chemokine receptors, specifically the CXCR4 and CCR5 receptors. We also have a number of other early stage projects ongoing and an active in- and out-licensing program.

The most advanced product in our pipeline, Fosrenol (formerly Foznol and prior to that, Lambda), is exclusively licensed to Shire Pharmaceuticals Group plc. Fosrenol has been filed for market approval in Canada, the U.S. and Europe for the treatment of excess phosphate levels associated with end stage kidney failure. Phase III trials for Fosrenol are ongoing in the U.S. Fosrenol has the potential to generate near term revenues.

We have also non-exclusively licensed our chemical linker technology, HYNIC, to North American Scientific, Inc., for use in their medical imaging agent ApomateTM which is in Phase I and II clinical trials in the U.S. and Europe respectively. Additional licensees for HYNIC are also being evaluated.

AMD-473 is a new platinum based anti-cancer agent (chemotherapy) that is active in a variety of tumor types. We are currently overseeing the completion of two clinical studies, a number of preclinical studies and the preparation and manufacture of drug supply for any additional clinical studies. Our plans for AMD-473 are focused on establishing a new development partnership in order to more rapidly maximize the commercial potential of the product.

Our in-house clinical program includes AMD-3100 which is being evaluated for its potential to be a new agent in stem cell transplant for cancer patients. Phase I trials of AMD-3100 are ongoing and Phase II trials are planned. AMD-3100 is an inhibitor of the CXCR4 receptor which is known to be involved in the trafficking of white blood cells and other cells in the immune system. AMD-070, also a CXCR4 inhibitor, has been selected for further evaluation in clinical trials as a new drug for HIV once final toxicology studies have been completed. We have a number of other new chemical compounds that target the CXCR4 receptor that are being evaluated in earlier stage studies as potential agents in asthma and rheumatoid arthritis. In

addition, we are developing new compounds that target the CCR5 receptor which is involved in HIV infection.

Our business strategy involves building upon our drug discovery and clinical development capabilities to support the advancement of our most promising therapeutic candidates into clinical trials and to attract corporate partners.

Our common shares ("Common Shares") are listed for trading on The Toronto Stock Exchange under the symbol "AOM". Our principal office is located at Suite 200, 20353 64th Avenue, Langley, British Columbia, V2Y 1N5, and our registered office is located at 2100 - 1075 West Georgia Street, Vancouver, British Columbia, V6E 3G2.

Development of the Business

We were established in 1996 by a team of seven scientists led by Dr. Michael Abrams and Dr. Geoffrey Henson, both of whom worked together from 1985 as integral members of the biomedical research group of Johnson Matthey plc (together with its affiliates, "JM") of London, U.K. This group was dedicated to the discovery and preclinical development of metal-based pharmaceuticals. In 1995, JM decided to focus its pharmaceutical related business on core activities involving the development and production of bulk drug species (active pharmaceutical ingredients). This decision provided our research team, led by Dr. Abrams, the opportunity to establish and lead an independent entity focused solely on the expansion of the drug discovery and development activities formerly undertaken by the biomedical research group of JM.

In June 1996, we acquired substantially all of the JM biomedical research group's portfolio of proprietary drug candidates then under in-house development and associated intellectual property that had been developed over the previous decade, seven of its key members including the pharmaceutical development management team and all of the related technical expertise (the "Biomedical Research Group").

With JM's permission, Dr. Abrams approached MDS Capital Corporation ("MDS") which manages the largest health care venture capital fund in Canada. Dr. Abrams arranged a meeting between MDS, on behalf of a group of Canadian venture capital investors, and JM, in December 1995, following which an agreement in principle was reached for the sale of the Biomedical Research Group in consideration of U.S.\$10 million. As a result of this we were incorporated in January 1996.

No business was carried out from our incorporation until the closing of the acquisition of the Biomedical Research Group on June 28, 1996. The purchase price was satisfied by the issue of 5,000,000 Common Shares and share purchase warrants (the "Warrants") to purchase 1,250,000 Common Shares in accordance with an asset transfer agreement between ourselves and JM dated June 28, 1996 (the "Asset Transfer Agreement"). JM had no interest in our activities and operated at arm's length until after the completion of the sale of the Biomedical Research Group.

Concurrent with this acquisition, we completed a \$19.8 million equity financing. The cost of the acquisition was allocated to the identifiable assets acquired based on fair values. At the time of this financing, we moved our base of operations, including several fully functional chemistry,

analytical and biology laboratories from West Chester, Pennsylvania, U.S.A. to our present location in Langley, British Columbia, Canada.

2001 Common Share Financing

On February 6, 2001, we completed a public offering of 1,500,000 Common Shares at a price of \$17.00 per share, for a total gross proceeds of \$25,500,000.

2000 Issue of Special Warrants

On February 11, 2000, we closed a private placement of 1,500,000 special warrants at a price of \$14.00 per special warrant for gross proceeds of \$21,000,000. Each special warrant was exercisable for one Common Share for no additional consideration. On March 22, 2000, we filed a long-form prospectus dated March 22, 2000 qualifying for distribution the Common Shares to be issued upon exercise or deemed exercise of the previously issued special warrants, and on March 31, 2000, the previously issued special warrants were deemed to have been exercised and we issued an additional 1,500,000 Common Shares.

1999 Initial Public Offering

On March 5, 1999, we completed an initial public offering of 5,000,000 Common Shares in Canada at \$6.10 per Common Share for total gross proceeds of \$30,500,000. Concurrently with the closing of the initial public offering, our Common Shares were listed on The Toronto Stock Exchange. On April 9, 1999, we issued an additional 160,000 Common Shares for gross proceeds of \$976,000 pursuant to the exercise by the underwriters of an over-allotment option granted under the initial public offering.

Private Placements¹

During the fiscal year-ended March 31, 1998, we completed two private placements issuing 1,790,059 units for gross proceeds of \$8,055,265. Each unit consisted of one Common Share and one price protection warrant ("Price Protection Warrant"). Each Price Protection Warrant entitled the holder to acquire one additional Common Share for no additional consideration if we sold Common Shares for less than \$4.50 per Common Share prior to or at an initial public offering of our Common Shares. Upon the completion of our initial public offering on March 5, 1999, all Price Protection Warrants expired. On February 23, 1998, we repurchased 1,235,112 Common Shares from our existing shareholders and concurrently closed a private placement of 1,235,112 Common Shares, each at a price of \$4.50 per Common Share. Also during fiscal 1998, we issued 718,750 Common Shares on the exercise of 2,875,000 Warrants on December 4, 17 and 24, 1997 at an issue price of U.S.\$2.00 (CDN\$2.86) per share for gross proceeds of \$2,055,546, and issued 113,500 Common Shares to employees on June 27, 1997 at an issue price of U.S.\$2.00 (CDN\$2.74) per share under the terms of employment agreements entered into in the previous year for gross proceeds of \$310,991.

¹ The Canadian equivalent of the various U.S. issue prices referred to in this section are based on the exchange rate used by the Company at the time of the issue.

During the year-ended March 31, 1999, we issued 12,000 Common Shares to an employee on June 1, 1998 at an issue price of \$4.50 per share under the terms of an employment agreement entered into in October, 1997 for gross proceeds of \$54,000, 2,000 Common Shares to an employee on January 26, 1999 at an issue price of U.S.\$2.00 (CDN\$3.09) per share upon the exercise of stock options for gross proceeds of \$6,172 and 187,500 Common Shares on the exercise of 750,000 Warrants on December 21, 1998 at an issue price of U.S.\$2.50 (CDN\$3.88) per share for gross proceeds of \$727,266.

During the year ended March 31, 2000, we issued an aggregate of 959,972 Common Shares on December 24 and 31, 1999, upon the exercise of outstanding Warrants at an issue price of U.S.\$3.00 (approximately CDN\$4.38) per share for gross proceeds of U.S.\$2,879,916 (approximately CDN\$4,205,330). We also issued 7,916 Common Shares for gross proceeds of \$25,000 as a result of the exercise of employee stock options and 6,383 Common Shares for gross proceeds of \$31,000 pursuant to our Employee Share Purchase Plan during fiscal 2000.

During the year ended March 31, 2001, we issued 162,165 Common Shares for gross proceeds of \$586,466 as a result of the exercise of employee stock options and 5,146 Common Shares for gross proceeds of \$59,072 pursuant to our Employee Share Purchase Plan. In December 2000, we issued 847,646 common shares upon the exercise of outstanding warrants at an issue price of U.S.\$3.50 (approximately CDN\$5.33) per share for gross proceeds of U.S.\$2,966,796 (approximately CDN\$4,519,541).

During the year ended March 31, 2002, we issued 22,616 Common Shares for gross proceeds of \$78,000 as a result of the exercise of employee stock options, 87,277 Common Shares for gross proceeds of \$273,000 pursuant to our Employee Share Purchase Plan. As at July 29, 2002 we have issued 7,600 Common Shares pursuant to our Employee Purchase Plan and 20,000 Common Shares to Clearview Venture Partners, L.L.C. at an issue price of \$4.00 as consideration for certain consulting services.

NARRATIVE DESCRIPTION OF THE BUSINESS

Knowledge Platform

The extensive experience of our research team in the development of metal based drugs has provided us with a unique insight into how the physical and chemical properties of compounds relate to their biological effects. In addition, our understanding of medicinal chemistry combined with our biological and pharmaceutical expertise support our activities to develop new product candidates for treating unmet medical needs. We have also built in-house facilities and developed the know how to vary the chemical structure of any class of compound and as a result modify its biological activity and pharmacological profile. The value of both our capabilities and experience is validated by our current and previous partnerships with leading companies in their respective fields, such as AstraZeneca Pharmaceuticals Limited (“AstraZeneca”) of London, U.K., DuPont Pharmaceuticals Company (“DuPont” or “DuPont Pharmaceuticals”) of Wilmington, Delaware and Shire Pharmaceuticals Group plc (“Shire” or “Shire Pharmaceuticals”) of Basingstoke, U.K. Further, our research team, while still at JM, contributed to the design of the first orally administrable platinum anti-cancer drug (JM 216). The rights to this drug were retained by JM and licensed to Bristol Myers Squibb and

subsequently returned to JM and re-licensed to Neotherapeutics Inc.. See “Narrative Description of the Business - Corporate Partnerships”. We have also published numerous scientific articles relating to metal based drugs. See “Directors and Officers”. This collective expertise drives our initiatives in the discovery and development of new pharmaceuticals.

Products in Development

We have a number of product candidates currently in clinical development, however the major focus of our business involves discovering and developing new chemical compounds with therapeutic potential, particularly those targeting specific chemokine receptors. We plan to spend significant resources on this program. In addition to our new programs and product candidates, we have also continued and expanded the discovery and development work related to the product candidates commenced by JM.

Product Candidates	Disease Focus	Stage of Development ⁽¹⁾	Partner
Fosrenol (formerly Foznol and Lambda)	Excess phosphate levels in kidney disease	Filed	Shire Pharmaceuticals
AMD-473	Cancer	Phase II ⁽²⁾	Unpartnered
Apomate™	Medical imaging kit for cancer prognosis	Phase II	North American Scientific
AMD-3100: CXCR4 Chemokine Inhibitor	Stem Cell Transplant	Phase I	Unpartnered
AMD-070: CXCR4 Chemokine Inhibitor	HIV	Late Preclinical	Unpartnered
CXCR4 Chemokine Inhibitors	Asthma, Rheumatoid Arthritis, Cancer	Early Preclinical	Unpartnered
Nitric Oxide Scavengers	Cancer, Inflammation	Preclinical	Unpartnered
Imaging Agents	Inflammation, Cancer	Preclinical	Unpartnered ³
CCR5 Inhibitors	HIV	Research	Unpartnered

Notes:

- (1) For a description of the stages of development in the drug approval process, see “Narrative Description of Business - Product Approval Process”.
- (2) AMD-473 (formerly ZD0473) was exclusively licensed to AstraZeneca between 1998 and 2001. AstraZeneca was responsible for all aspects of product development. As of February 2002 we retain all rights to the product.
- (3) We had licensed this technology for use in infections to Ortho Pharmaceutical Corporation, Inc. (“Ortho”) which has, in turn, sub-licensed it to DuPont Pharmaceuticals. The license with Ortho has been terminated and we are currently negotiating the existing license agreement with Bristol Myers Squibb MI, the acquiree of DuPont Pharmaceuticals. See “Narrative Description of Business - Corporate Partnerships”.

Fosrenol (formerly Foznol and Lambda) – Phosphate binder for end stage kidney dialysis patients

Overview

Fosrenol is a new phosphate binding agent for kidney dialysis patients designed to overcome the limitations of currently available drugs. Kidney failure leads to excess levels of phosphate in the blood, a condition known as hyperphosphatemia. Healthy kidneys maintain a balance between

phosphate and calcium levels in the blood by excreting excess phosphate in the urine. Control of blood phosphate levels is central to the prevention of renal bone disease and hyperparathyroidism in patients with chronic kidney failure. In patients with chronic kidney failure, the kidneys are unable to remove enough phosphate to maintain the necessary balance. Elevated phosphate levels signal the body to excrete parathyroid hormone, which breaks down bone to release calcium into the blood in an effort to re-establish the balance between calcium and phosphate levels. Chronic kidney failure patients with uncontrolled elevated phosphate levels experience bone loss as well as calcification of the circulatory system caused by excessive amounts of phosphate and calcium in the blood. To compensate for the kidney's inability to rid the body of excess phosphate, almost all kidney dialysis patients use a phosphate binder to remove excess phosphate. Phosphate binders bind phosphate in the digestive tract, preventing its absorption into the bloodstream.

Currently available phosphate binders include Renagel, a polymer based phosphate binder currently sold by Genzyme Inc., calcium acetate, calcium carbonate and aluminum hydroxide. See "Narrative Description of the Business - Competition". In order to achieve adequate reductions in phosphate absorption, calcium acetate and calcium carbonate, the most commonly used agents, must be taken at doses which can lead to constipation and failure of patients to follow prescribed dosages. In addition, calcium therapy requires frequent monitoring because its use may result in dangerous elevations of blood calcium levels. This condition occurs in 25% to 50% of patients taking calcium-based binders. Aluminum hydroxide is more effective at lower doses than calcium acetate or calcium carbonate, but it is infrequently used because aluminum absorbed from the intestinal tract accumulates in the tissues of patients with chronic kidney failure, causing softening of the bones, anemia and dialysis-related deterioration of intellectual function. Fosrenol is a non-calcium based phosphate binding agent and due to its strong binding affinity for phosphate and low systemic absorption, it controls phosphate levels in the blood and may also protect against bone loss.

Fosrenol, the active ingredient of which is lanthanum carbonate, is taken orally and is designed to bind phosphate in the digestive tract. Fosrenol is not significantly absorbed by the body, lowering the potential for side effects. The activity of Fosrenol is based on the low solubility of lanthanum phosphate, the product formed when Fosrenol is exposed to phosphate in the digestive tract. Therefore, it converts free phosphate into an insoluble substance that is readily excreted from the body.

We granted a worldwide exclusive license to Shire, a leader in developing treatments for renal bone disease, for the development and commercialization of Fosrenol. See "Narrative Description of the Business - Corporate Partnerships". Shire is assuming all responsibility and expenses for the development and commercialization of Fosrenol. Therefore, we have not allocated any funds for expenditures on Fosrenol. We receive a royalty on sales of Fosrenol upon its regulatory marketing approval.

A Phase I dose escalating study was conducted on 14 healthy volunteers. Different dose levels, to a maximum of 4.7 grams of lanthanum per day, were administered. This dose level was well tolerated. It is expected that the effective dose of Fosrenol in kidney failure patients will be substantially lower.

The results from the Phase I study of healthy volunteers show that Fosrenol decreases phosphate levels excreted in the urine relative to placebo. In healthy people, phosphate levels in blood are maintained at normal levels by excretion of excess dietary phosphate into the urine. In patients with kidney failure this ability is reduced or eliminated and phosphate levels increase in the blood. Fosrenol, by binding phosphate in the digestive tract, reduces the amount of dietary phosphate that enters the bloodstream, which in healthy subjects results in decreased levels of phosphate in the urine.

The United States Phase II trial treated 145 kidney dialysis patients for up to six weeks with either placebo or Fosrenol in divided daily doses of 225, 675, 1350 or 2250 mg of lanthanum. The study results are positive with dose-related reductions in phosphate levels at doses of 675, 1350 and 2250 mg and a good tolerability profile. At the end of the six-week study period, serum phosphate was reduced by 0.95 mg/dl in patients receiving 1350 mg and 1.13 mg/dl in patients receiving 2250 mg of lanthanum. These reductions were highly statistically significant when compared to a phosphate increase of about 0.75 mg/dl for patients on placebo. Fosrenol was generally well tolerated, although there were slightly more gastrointestinal complaints in patients who had taken Fosrenol compared with those taking placebo. However, these complaints were generally mild and transient.

A Phase III clinical trial for Fosrenol commenced in Europe. Initiation of the Phase III trial followed positive results from a sub-group of patients who successfully completed the first four weeks of the Phase II study in the U.K. These results confirm those seen earlier in the Phase I study. Of the 28 patients in this sub group, 26 re-established control of their blood phosphate levels. Of these, 21 patients had phosphate concentrations below the target level set for the study. Adverse events were generally mild and transient. A Phase I study, initiated in Japan in August, 1998, directly managed by an in-country caretaker on behalf of Shire, is now complete. Shire also commenced Phase III trials for Fosrenol in the United States in July 1999. These trials are ongoing, however the Phase III trial in Europe is now complete.

Preliminary data from a U.S. Phase III study demonstrate that 59% of patients treated with Fosrenol achieved serum phosphate control versus 23% on placebo. These results were statistically significant and support the potential of Fosrenol to maintain and control the blood phosphate levels in patients with hyperphosphataemia. This study, together with longer term Phase III European clinical data in over 800 patients, formed part of the clinical dataset that was submitted to the regulatory authorities, more specifically a Reference Member State in the European Union (EU) Mutual Recognition Procedure. These regulatory authorities review the data provided and determine whether the product has an appropriate safety, efficacy and quality profile to be granted an approval to market.

Preliminary data from a European Phase III renal bone disease study on a total of 98 patients demonstrated that Fosrenol was well-tolerated and controlled phosphate levels in patients throughout the trial and after one year. In addition, data from 60 patients in this trial who had bone biopsies demonstrated that after one year of treatment with Fosrenol no adverse effects on bone were observed. As of April 2002, Shire reported that they had 77 pre-clinical studies completed; 19 clinical studies completed; 1668 total number of patients treated; 650 patients treated for 6 months; 395 patients treated for 12 months; 87 patients treated for 24 months or longer. Studies are ongoing and as of August 2002 over 1700 had been treated with Fosrenol.

On June 10, 2002, April 30, 2002 and March 14, 2001 Shire filed regulatory submissions seeking marketing approval for Fosrenol in Canada, the U.S. and Europe respectively for the management of phosphate levels in chronic renal failure patients.

Market

It is estimated that there are approximately 280,000 kidney dialysis patients in the U.S., 225,000 in Europe, 190,000 in Japan and 90,000 in the Pacific Rim region. According to the Canadian Institute for Health Information, over 14,000 Canadians underwent dialysis in 2000, with approximately 4,400 new patients beginning dialysis treatment in Canada each year. (*Numbers of patients on dialysis broadly equates to patients with end stage kidney disease. Source: Market Research, Insight International, 2001/2002*).

AMD-473 – A new platinum based anti-cancer agent (chemotherapy)

Overview

AMD-473 is a new generation platinum based anti-cancer drug or chemotherapy. Cancer refers to a broad family or group of diseases caused by the uncontrolled growth and proliferation of cells that have undergone genetic changes or mutations. These mutations are characterized by a breakdown of the normal cellular mechanisms that regulate cell growth and cell death, which give the cancer cells a selective growth and survival advantage over normal cells. Cancer becomes more prevalent with age and with increased exposure to toxic agents such as chemicals or radiation that damage genes.

The current treatment options for most cancers are surgery, radiation therapy and chemotherapy. Often, a combination of these treatment methods is used to overwhelm the ability of cancer cells to develop resistance to the prescribed treatment. Chemotherapy generally involves the delivery of specialized pharmaceutical products that are designed to interfere with cell division and are therefore more toxic to rapidly dividing cancer cells than normal cells. Chemotherapy is the principal mode of treatment where the primary tumor is either inoperable or has spread to other parts of the body.

Platinum based drugs are commonly used as chemotherapeutic agents that bind to a cell's DNA, initiating a complex series of events that ultimately lead to the death of the cell. The platinum based anti-tumor agents, first identified in 1969, are important drugs in the current treatment of testicular, ovarian, lung, head and neck, bladder and cervical cancers.

The platinum drugs currently approved in the U.S. market are cisplatin and carboplatin. One of the major difficulties with these therapies is that the tumors typically develop a resistance to the treatment. In preclinical in vitro studies performed in conjunction with the Institute of Cancer Research ("ICR") of Sutton, U.K., it was demonstrated that AMD-473 circumvents resistance in a number of human ovarian cancer cell lines with decreased sensitivity to cisplatin. In vivo studies of AMD-473 have consistently shown anti-tumor activity in human ovarian cancer xenografts at least equal to and in many cases superior to cisplatin.

Preclinical tests were conducted comparing the activity of AMD-473 versus cisplatin, in which both drugs were given at their maximum tolerated dose and administered on days zero, seven, 14

and 21. While the tumors initially responded to both drugs, after the treatment was terminated the tumors in the cisplatin treated mice recurred. However, the tumors in the AMD-473 treated mice did not recur over the course of the experiment.

Further preclinical tests indicate that AMD-473 has a much improved toxicity profile over cisplatin as well as oral bioavailability. Both of the currently approved platinum drugs, cisplatin and carboplatin, are administered by injection only.

In November 1997, we entered into a collaboration with the U.K. Cancer Research Campaign (“CRC”) and commenced a Phase I clinical trial with AMD-473 at the Royal Marsden Hospital, U.K. A total of 43 patients were recruited and received the drug intravenously. The interim results of this Phase I trial were presented at the 35th Annual American Society of Clinical Oncologists Meeting in Atlanta in May, 1999. This presentation indicated that the dose-limiting toxicity of AMD-473 was bone marrow suppression with no evidence of kidney or nerve toxicity. Initial indications of anti-tumor activity were observed in several patients.

Effective March 31, 1998, we entered into a licensing agreement with Zeneca Pharmaceuticals Limited (now AstraZeneca), whereby we exclusively licensed AMD-473 to AstraZeneca for worldwide development and commercialization. Under the licensing agreement, AstraZeneca assumed all responsibility and expenses for development of AMD-473 including any services we performed on or for the development of this compound. See “Narrative Description of the Business - Corporate Partnerships”.

In the fourth quarter of 1999, AstraZeneca initiated the first of several planned combination Phase I studies of AMD-473 with other chemotherapeutic agents as well as the first of several monotherapy Phase II trials with the drug.

On April 5, 2001, AstraZeneca announced that they planned to initiate the first Phase III clinical trial in ovarian cancer using AMD-473. This decision was based on preliminary clinical results presented at the American Society of Clinical Oncology (“ASCO”) meeting held in May 2001 and at the European Lung Cancer conference in June 2001 indicating that AMD-473 has the potential to offer an effective treatment option for both platinum therapy resistant and sensitive ovarian cancer patients. Data presented also demonstrate that the side effect profile of AMD-473 is manageable with no evidence of clinically significant nerve or kidney toxicity.

Platinum resistant ovarian cancer patients are those who relapse *on or within* 6 months of completion of prior platinum therapy. Sensitive ovarian cancer patients are those who relapsed *after* 6 months of completion of prior platinum therapy. Approximately 25% of ovarian cancer patients will be resistant to their first platinum therapy and 75% will be sensitive. However, of these sensitive patients, close to 80% will relapse again after their initial treatment and may require a second round of treatment.

The preliminary data presented at ASCO 2001 was from a Phase II monotherapy trial of AMD-473 in 29 ovarian cancer patients. Fourteen of these patients were categorized as platinum therapy-sensitive (relapse/disease progression >26 weeks after initial therapy) and 15 as platinum therapy-resistant (relapse/disease progression <26 weeks). Patients received AMD-473 as a one hour infusion every three weeks, initially at a dose of 120mg/m², which was subsequently

increased to 150mg/m² as the original dose was well tolerated. In total, 84 treatment cycles have been administered. Objective responses were observed in 2/14 (14%) 'resistant' patients and 5/12 (42%) 'sensitive' patients. In addition, disease stabilization was observed in 2 'resistant' and 3 'sensitive' patients, with evidence of tumor shrinkage in 3 patients.

Responses to AMD-473 were also observed among patients with small-cell lung cancer who had relapsed after initial chemotherapy. The response rates in both ovarian and lung cancer were considered encouraging compared to response rates achieved with existing platinum chemotherapy.

However, on August 31, 2001, we announced that AstraZeneca would be re-assessing their Phase III clinical trial program for AMD-473 based on updated clinical data in the Phase II ovarian cancer study. This data, which was presented at the European Conference of Clinical Oncology ("ECCO") in October 2001, showed a decreased and disappointing response rate in resistant ovarian patients from the previously reported 2/14 (14%) to 3/42 (7%). Updated data in sensitive ovarian patients remains encouraging with a response rate of 12/31 (39%) compared to the preliminary data of 5/12 (42%). Additional AMD-473 data were also presented at ECCO demonstrating encouraging anti-tumor activity including confirmed responses with mono and combination therapy in a number of other tumors including pancreatic cancer and hormone resistant prostate cancer, among others. The data also confirmed that AMD-473 has a manageable toxicity profile and it has not been associated with clinically significant neuro- or nephrotoxicity.

On November 26, 2001 we announced that AstraZeneca would not continue to develop AMD-473 and would return all commercial development rights to AnorMED. This decision was primarily based on the updated response rate in resistant ovarian cancer patients. Regardless of the promising response rate in second line platinum sensitive ovarian patients, activity in a variety of tumor types and manageable safety profile, AMD-473 did not meet the differentiated profile required by AstraZeneca, particularly in overcoming platinum resistance in ovarian and lung cancer patients who have previously failed a platinum based therapy.

Upon return of AMD-473 to AnorMED, ongoing clinical studies in hormone resistant prostate cancer and bladder cancer continued with recruitment/follow-up in accordance with the existing protocols. These trials were prepaid by AstraZeneca and have completed enrollment.

In order to evaluate further development options for AMD-473, we held a clinical advisory board meeting on January 2002 and also commissioned market analyses. Based on all available clinical data from over 500 patients and a solid safety profile, in conjunction with the clinical advisory board recommendations and the market analyses, we are continuing to evaluate the potential of AMD-473 as a new platinum based chemotherapy that has a broad spectrum of anti-tumor activity, particularly in tumor types where existing platinum have shown limited activity such as hormone resistant prostate cancer (HRPC), among others. In addition, we are continuing with early studies to support the potential of AMD-473 as an oral chemotherapy for use in combination with other oral chemotherapies or in conjunction with radiation therapy. However, our main focus, which we are actively pursuing, for furthering the development of AMD-473 is to establish a new partnership in order to support the initiation of additional clinical studies in a variety of tumor types, including HRPC.

Market

Cancer is the third leading cause of death worldwide, accounting for 21% of all deaths (World Health Organization, 1998 Fact Sheet). According to estimates by the World Health Organization, more than 10 million people developed cancer worldwide in 2000 and over six million others, already having the disease, died from it (GLOBOCAN 2000). Worldwide, the cancers with the highest incidences in 2000 were lung, breast, and colon/rectum with total incidence of 1,238,861, 1,050,346, and 944,719 respectively. A significant development in cancer treatment in recent years is the use of platinum drugs in combination with a number of new non-platinum based anti-tumor agents (F-D-C Reports, April, 1998). In the U.S. platinum drugs are approved for the treatment of ovarian, testicular, bladder, and head and neck cancers and are also commonly used in other cancers including lung (Mattson Jack Consulting Group, IDDB). A new platinum drug with enhanced anti-tumor activity has the potential to be used in similar combinations. Given the prevalence of cancer and the continued unmet medical needs in this area, new therapies and treatments are clearly needed.

As of April 2002, world drug purchases of cytostatic agents, which includes platinum anti-cancer agents, were U.S.\$10.7 billion compared to sales of U.S.\$9.7 billion for the previous 12 month period (IMS Health Drug Monitor). In 2000, sales of platinum oncology agents were U.S.\$915 million (Med Ad News, May 2001) compared to sales of platinum oncology agents of U.S.\$819 million in 1999 (Med Ad News, May 2000).

Medical Imaging Agents – Apomate™ & HYNIC Linker Technology

Overview

We have developed a proprietary linker technology (HYNIC linker) for attaching technetium, an imaging radioisotope, to biological molecules. This technology is applicable in medical imaging (nuclear medicine) for a variety of indications including inflammation and cancer, among others.

Nuclear medicine imaging is a diagnostic tool that uses a gamma emitting radioisotope incorporated into a molecule capable of localizing in specific parts of the body associated with a particular disease. This radiopharmaceutical is administered to a patient and the resulting radioactive signal is detected with a gamma radiation detecting camera. Nuclear medicine is widely used in the diagnosis of heart, bone, liver and kidney disease as well as infection and cancer.

Due to its favourable physical properties, ready availability and low cost, the isotope of choice for most nuclear medicine imaging procedures is technetium-99m. In the United States 68% of the nuclear medicine diagnostic imaging procedures are performed using technetium-based agents (J. of Nuc. Med., March 1998).

Although diagnostic imaging programs are not a major focus of our business, our research team, through a detailed understanding of metal binding chemistry, has developed a new methodology to link technetium-99m to biological molecules, including antibodies and peptides, with high specificity for disease targets. Since technetium-99m is a desirable isotope to use for nuclear medicine, the ability to link this isotope to other molecules specific for certain disease targets is important. Our research team discovered a novel aromatic hydrazine molecule, the HYNIC

linker. This same linker can be used to connect technetium-99m to different biological molecules specific to certain diseases such as blood clots, infections or tumors. This linking chemistry allows the labelling of biological molecules in convenient radiopharmaceutical kits, which is important in a hospital setting. The high labelling efficiency obtained with the HYNIC linker, as noted in a scientific review of the field has the potential to provide superior imaging and improved disease detection over other imaging techniques (Bioconjugate Chemistry, 1997).

In November 2001, we announced that we signed a non-exclusive licensing agreement with North American Scientific, Inc. ("NASI") for incorporation of our linker technology into NASI's second generation version of its Apomate™. NASI designs, develops and produces innovative radioisotopic products, including brachytherapy seeds and radiopharmaceuticals, principally for the treatment and diagnosis of disease. Its lead radiopharmaceutical product candidate is Apomate™, a kit for the preparation of Technetium 99m labelled Annexin-V (a naturally occurring human protein produced by recombinant techniques). It is administered intravenously and is intended for the in vivo imaging of apoptosis and necrosis, two forms of cell death.

Apomate™ is being evaluated in Phase I and Phase II clinical trials in the U.S. and Europe as an early indicator of response to anti-cancer treatment. It is also under study for its ability to determine the extent and location of heart injury after a heart attack and the response to treatment of heart attacks. Apomate™ will potentially allow clinicians to make better therapeutic decisions for individual patients by monitoring the patient's condition or response to treatment with greater sensitivity and speed. The use of our linker technology in second generation kits may improve the imaging quality and utility of the Apomate™ kit and may (i) allow access to important new market opportunities in abdominal disease, (ii) reduce end-user preparation time to minutes which provides the flexibility needed to utilize Apomate™ in the acute setting, and (iii) reduce manufacturing costs with significantly improved radiolabeling efficiency.

In the second generation kit very little of the imaging agent in the liver enters the intestine via the biliary system. Thus, the largest fraction of the administered dose of the Apomate™ is cleared from the blood by the kidneys and, in the absence of disease, the remainder of the abdomen shows no localization of radioisotope. This normal absence of activity in the abdomen after administration of the second-generation agent should permit clinicians to readily identify areas of abnormality. Such areas of abnormality might be expected in subjects with gastrointestinal cancer, ovarian cancer and prostate cancer. The first-generation kit did not allow imaging of those disorders because the large amount of imaging agent normally excreted in the bowel would obscure uptake by the cancer.

NASI has successfully performed human testing of a second generation technology for its Apomate™. Phase I/II core studies in various cancers are ongoing and clinical data presented at both the American Association of Cancer Research in April 2002 and at the Society of Nuclear Medicine in June 2002 demonstrate use of Apomate™ can image tumor response to treatment within hours of initial course of therapy. NASI has plans for late stage clinical development and a process in place with regulatory authorities.

In addition to our agreement with NASI, DuPont Pharmaceuticals, a leader in diagnostic radiopharmaceuticals, has used our HYNIC linker technology for the detection and diagnosis of

a variety of disease states and is incurring all costs associated with the development and commercialization. See “Narrative Description of the Business - Corporate Partnerships”.

DuPont Pharmaceuticals screened and selected a novel agent, RP-517, which contains the HYNIC linker, for the detection of inflammation and infection. The ability to image sites of infection is of particular use in managing conditions such as fever of unknown origin and post-operative infections. Identifying the site of infection can facilitate the treatment of such infections by surgical drainage and can monitor the progress of antibiotic therapy. DuPont Pharmaceuticals also used the linker technology for the development of a novel agent for cancer imaging which is at the preclinical stage. This is a potentially broad area that could assist in the early detection of cancer as well as in the treatment planning of patients with known tumors. Clinical development of these programs did not advance due to a change in market business strategy within Dupont. However, we are currently in negotiation to establish a new non-exclusive license agreement with Bristol-Myers Squibb MI, the acquirer of the Dupont Medical Imaging Business, to initiate a new development program for the HYNIC linker in medical imaging (See “Narrative Description of the Business - Corporate Partnerships”). We are also evaluating additional out-licensing opportunities for the HYNIC linker technology.

Market

In 1996, revenues for the total world radiopharmaceutical market were U.S.\$1.1 billion. Of this figure North America accounted for 48%, Europe 19.5%, Asia Pacific 26.4%, and the rest of the world 2.5%. The growth rate for 1996 was 9.5%, and this is expected to increase as new applications to different disease areas, such as oncology, are developed (Journal of Nuclear Medicine, Vol. 39, No.3, March 1998).

Chemokine Inhibitors – HIV, Inflammation and Cancer

Overview

Chemokines and their receptors are important to medicine and have been implicated in a number of diseases including HIV, inflammation and cancer. Chemokines are proteins produced in the body that regulate the development and migration of various cell types, such as white blood cells. Chemokine receptors are specialized proteins that function at the surface of cells, and enable chemokines to trigger an internal cellular process referred to as chemotaxis. Chemokine inhibitors block chemokine receptors, and can prevent this chemotaxis from occurring. For example, chemokines attract tumor cells to other organs forming metastases. Blocking the chemokine receptor can potentially prevent cancerous cells from being drawn into other organs. In rheumatoid arthritis, blocking the chemokine receptor potentially prevents white blood cells from infiltrating the joint as white blood cells are attracted to inflamed tissue areas by chemotaxis. Chemokines are also one of the factors responsible for keeping white blood cells, including stem cells, in the bone marrow. Blocking the chemokine receptor mobilizes white blood cells. These cells then move into the blood. Once in the blood these cells can potentially be collected and may be administered to cancer patients whose white blood cells have been depleted due to radiation and chemotherapy. In a more specific example, the CXCR4 or CCR5 chemokine receptors are used by the different strains of the human immunodeficiency virus

(HIV) to infect the target cells. Blocking the CXCR4 or CCR5 receptors may potentially prevent the relevant HIV strains from entering and therefore infecting the target cells.

We believe chemokine inhibitors hold significant potential as new and potentially breakthrough drugs. We have initiated a number of studies and are currently evaluating the potential of a number of chemokine receptor inhibitors in the areas of oncology, inflammation and virology. Our lead targets in this program are the CXCR4 and CCR5 receptors, and our lead CXCR4 chemokine inhibitor is AMD-3100 which is described below. We have a number of ongoing collaborations within this program with the Rega Institute in Belgium, University of Copenhagen, University of Michigan, Indiana University at Indianapolis and the Institute of Cancer Research in Sutton, U.K. See "Narrative Description of the Business - Research Collaborators".

On May 1, 2001, we expanded our discovery and development efforts in this program through the acquisition of a chemokine receptor inhibitor database from British Biotech Pharmaceuticals Limited, as a means of identifying new drug candidates. See "Narrative Description of the Business - Research Collaborators".

AMD-3100 – Peripheral Blood Stem Cell Transplant

Overview

Stem cell transplant is used to restore the immune system of patients who have been treated for cancers involving the blood and immune system such as leukemias, multiple myeloma and lymphomas. Transplantation involves the collection of certain types of stem cells, specifically CD34⁺ cells. These cells are then re-administered to the patient after the cancer treatment. In stem cell transplant it is very important to be able to collect as many stem cells as possible from the patient prior to their cancer treatment. This can greatly improve the chances that their immune system can be restored.

Stem cells used to be collected from patients using an invasive procedure called bone marrow transplant. This technique is now being replaced by a new procedure called peripheral blood stem cell transplant (PBSC). In this procedure, stem cells are collected from the circulating blood for transplantation. Prior to collection patients are given a growth factor called Neupogen (G-CSF -granulocyte colony stimulating factor) which causes stem cells in the body to multiply. The objective of this procedure is to get as many stem cells as possible into the circulating blood where they can be collected. However, many stem cells remain locked up in the bone marrow and require more than one session of Neupogen and harvesting in order to collect a sufficient number of cells.

AMD-3100 is an inhibitor of the CXCR4 chemokine receptor. The CXCR4 receptor is present on white blood cells and, among other functions, has been shown to play a key regulatory role in the trafficking and homing of human CD34⁺ stem cells in the bone marrow.

On March 19, 2001 we initiated a Phase I clinical trial, using low doses of AMD-3100, in healthy volunteers to study increases in white blood cell counts previously observed in the first AMD-3100 trial. This trial was designed to determine precisely which populations of white blood cells, including possibly stem cells, are mobilized by AMD-3100, and to investigate the

mechanism of action responsible for this activity. This program was initiated due to observations made in a concurrent clinical program of AMD-3100 in HIV. See “AMD-070 – A new HIV Entry Inhibitor”.

AMD-3100 has been shown to mobilize stem cells, causing them to move out of the bone marrow and into the circulating blood. When used in combination, AMD-3100 and Neupogen work together to generate an increase in the number of circulating stem cells. In this capacity, AMD-3100 may enhance the stem cells available for collection as well as improve the transplantation procedure by reducing the number of times cells would have to be collected.

These study results were presented at the International Society of Hematology meeting in July 2002 and confirm that the increase in stem cells induced by AMD-3100, given as a single subcutaneous injection, is dose-dependent and that AMD-3100 enhances the effect of Neupogen. In addition, results from a second study also presented at the conference confirm that stem cells mobilized by AMD-3100 alone in healthy volunteers can successfully re-establish a damaged immune system and function normally in preclinical models. To date, AMD-3100 has been generally well tolerated in all volunteer studies.

We plan to initiate a third Phase I trial in a small number of cancer patients to confirm that AMD-3100 alone increases stem cells in this patient population. Based on the positive data from this study, we plan to initiate a Phase II trial of stem cell transplantation in cancer patients using AMD-3100 in combination with Neupogen in 2002.

Market

In 1999 it is estimated that over 31,900 PBSCT procedures were performed in North America and Europe. The use of PBSCT is increasing in some cancers for a number of reasons including: an overall increased use of autografts for certain indications as well as a shift towards use in older patients, an increase in the number of allogenic transplants, increased donor availability, continued adoption of PBSCT in all recipient groups, and expansion of use in older patients.

This market is segmented into procedures for allogenic transplants and procedures for autologous transplants. However, for both allo-and autotransplants, the proportion of recipients aged > 40 years continues to increase. This may reflect advances in supportive care with a resultant decrease in transplant-related toxicity (TRM) and the increased application of transplantation to diseases affecting older patients (e.g. multiple myeloma [MM]). Patients aged > 50 years now account for more than 10% of allograft recipients and 50% of autograft recipients.

The International Bone Marrow Transplant Registry (IBMTR)/Autologous Blood and Marrow Transplant Registry (ABMTR) estimate(s) that 15,000 allogenic and over 25,000 autologous transplants were carried out in 2000. Based on these numbers and sales estimates from use of Neupogen and other agents in stem cell transplant we estimate a potential market of approximately U.S. \$100M.

Autologous: Donor is the Patient - Over 95% of autotransplants in adults and 80% in children and adolescents use hematopoietic progenitor cells collected from blood. The remainder use bone marrow alone or in combination with cells collected. In 1999 more than 28,900 autografts were reported to European and North American registries. The most common indications for

autotransplants were breast cancer, non-Hodgkin's lymphoma, multiple myeloma and Hodgkin's disease. Strong growth appears to continue in indications such as multiple myeloma where PBSCT transplants are considered the therapy of choice. In the 1970s, the median age of a transplant recipient was 17 years compared to the median age of 44 from 1995 to 1997. This is important as most of the cancers employing PBSCT are characterized by onset later in life.

Allogenic: Donor to Patient - Most allogeneic transplants use bone marrow grafts. However, in 1998-2000 there was a steady increase in use of peripheral blood stem cells, especially in older recipients.

In 1999, approximately 4,500 PBSCT allografts were performed in Europe and North America. The majority of these transplants (74% in 1998) were done to treat Leukemia, although the use for other indications such as lymphomas has continued to grow. The percentage of allografts using PBSCT also continues to grow. In 1999 45% of allografts reported to the European registry used PBSCT as the stem cell source as compared to less than 15% in 1995. European registry data suggest that allogeneic transplants have grown at an average annual rate of 10% and that PBPC allografts grew in excess of that. Allografts in the U.S. have also grown significantly in the last 5 years combined with a continued shift towards the use of PBSCT as the cell source.

AMD-070 – A new HIV Entry Inhibitor

Overview

In order to enter and infect cells, HIV must bind to either the CXCR4 (X4) or CCR5 (R5) chemokine receptor. Different strains of HIV prefer one receptor or the other. Blocking the CXCR4 and CCR5 receptors can prevent the relevant HIV strains from entering and infecting the target cells. Drugs that block or inhibit the CXCR4 and CCR5 receptors are referred to as HIV entry inhibitors. An infected individual may harbor different levels of both X4 and R5-using strains. A new product combining both a CXCR4 and a CCR5 inhibitor would offer a potent new way to target HIV.

AMD-3100 was our first anti-HIV drug candidate. It is a CXCR4 inhibitor. Our research team, in collaboration with the Rega Institute of Leuven, Belgium, discovered that AMD-3100 had the potential to be an entirely new class of anti-HIV agent.

AMD-3100 was originally licensed by JM to Novartis AG (formerly Sandoz AG), who returned the rights when they changed the focus of their research programs. Subsequently, we discovered that AMD-3100 inhibits the binding action of HIV with the CXCR4 chemokine receptor. In preclinical studies, AMD-3100 inhibits HIV in vitro and has shown significant in vivo therapeutic activity in a mouse model of HIV infection, both as a single agent and in combination with approved anti-HIV agents. Our collaboration on AMD-3100 with the Rega Institute is ongoing. See "Narrative Description of the Business - Research Collaborators".

In August 1998, we initiated a Phase Ib/IIa clinical trial of AMD-3100 in healthy volunteers at the Johns Hopkins University School of Medicine in Baltimore, Maryland. The goal of this trial was to obtain initial safety and pharmacokinetic data in humans. AMD-3100 was administered intravenously and sub-cutaneously in this trial. Thirteen volunteers were enrolled in this study and the drug was well tolerated and demonstrated linear pharmacokinetics both intravenously

and sub-cutaneously. No oral bioavailability was demonstrated. A full report of this trial was published in the June, 2000 edition of the Journal "Antimicrobial Agents and Chemotherapy". Enrolment in a Phase Ib/IIa study to test the effect of the intravenously administered drug on HIV infected individuals was commenced at Johns Hopkins University in May, 1999 and shortly thereafter at Case Western Reserve University in Cleveland and at the University of Washington in Seattle. Since that time, three other clinical sites were added, University of Texas Medical Branch in Galveston, Cornell University in New York and the AIDS Research Alliance in Los Angeles. In this Phase Ib/IIa HIV study for AMD-3100, the drug was administered as a 10 day continuous intravenous infusion. Patients were pre-selected for the strain of HIV believed to infect cells via the CXCR4 chemokine receptor, using a test called "MT2", that is not very sensitive but was the only assay available at that time

On May 25, 2001, after enrolling 40 patients, we halted the study. This decision was based on three factors: drug level had reached the maximal level feasible for subcutaneous administration planned for subsequent phase II trials; insufficient antiviral activity as defined by the assays available at the time; and two patients, both of whom may have had pre-existing cardiac conditions, experienced abnormal cardiac activity at high doses. It is not believed that this cardiac activity was related to blocking the CXCR4 receptor. Although the abnormal activity was not serious, it would have required impractical patient monitoring in subsequent phase II trials. More importantly, we had developed a new series of CXCR4 inhibitors that have the potential to be administered as a pill.

Later in 2001, Virologics Inc. developed a more sensitive test that detected the presence and amount of each HIV strain. For example, the test can determine the percentage of HIV using CXCR4 and the percentage of HIV using CCR5 in each individual patient. A significant number of patients will be infected with both strains of HIV. Using this new assay, AnorMED identified that 19/40 patients in the Phase Ib/IIa study actually had the HIV virus strain that uses the CXCR4 chemokine receptor and therefore may have benefited from AMD-3100.

Subsequent analysis of data collected from these 19 patients confirmed efficacy of AMD-3100 in blocking the CXCR4 receptor and--importantly--proof of principle in anti-HIV activity. Of the 19 truly eligible patients, AMD-3100 eliminated the CXCR4 receptor-using virus in 9 patients. This activity was dose-dependent.

These breakthrough results were presented at the 9th Retroviral and Opportunistic Infections Conference in February 2002, at the Collaborative HIV Research Seminar in April 2002 and at the International AIDS Conference in July 2002.

In July 2002, we announced that we selected a new CXCR4 HIV entry inhibitor, AMD-070 for further evaluation in clinical trials and also have backup candidates currently in preclinical studies. AMD-070, and the backups, have the potential to be administered to HIV patients as a pill. We are preparing to file an Investigational New Drug (IND) application with the U.S. Food and Drug Administration (FDA) and begin Phase I clinical studies in 2003 for AMD-070 as a new anti-HIV agent. We are exploring partnering opportunities which would allow the evaluation of AMD-070 in combination with another HIV entry inhibitor or existing agent.

Market

Acquired immunodeficiency syndrome (“AIDS”) is caused by HIV infection. AIDS is characterized by a gradual decline in the body’s immune system, leading to opportunistic infections and certain cancers, dementia and, in many cases, death. Drugs for the treatment of AIDS include agents that treat the secondary conditions caused by the weakened immune system of the patient as well as drugs that directly inhibit HIV. HIV infects by fusing with target cells and injecting its genetic material into the T-cells. The CXCR4 chemokine receptor is found on T-cells. Loss of T-cells is a major factor in the decline of the immune system in HIV infected patients. Once HIV infects the target cell during fusion, the virus uses the target cell in order to replicate. Drugs that prevent HIV from fusing with T-cells are potential anti-HIV agents.

In 2001, the number of people living worldwide with HIV/AIDS was 40 million with 5 million people newly infected with the virus. In North America and Western Europe, approximately 1.5 million people were living with HIV/AIDS at the end of 2001. HIV/AIDS includes all people with HIV infection, whether or not they have developed symptoms of AIDS. (AIDS Epidemic Update December 2001 - UNAIDS). It is generally believed that in the absence of therapeutic intervention the vast majority of people infected with HIV will ultimately develop AIDS.

In 1999, worldwide sales of AIDS and AIDS related drugs were U.S.\$5.2 billion. (Med Ad News, May 2000). In 1998, sales for this category were U.S. \$4.4 billion. (Med Ad News, May 1999). The key factors driving the growth of drug treatment of HIV include the recent adoption of combination therapies, whereby two, three, or even four antiretroviral drugs are taken together. As there is still no cure for HIV or AIDS, an infected individual must continue antiretroviral therapy for life (Datamonitor - Market Dynamics 1999:HIV)

Chemokine Inhibitors – CXCR4 in Asthma, Rheumatoid Arthritis and Cancer and CCR5 in HIV

Overview

The CXCR4 receptor is present on white blood cells and, among other functions, has been shown to play a key regulatory role in the trafficking and homing of a variety of cells involved in the immune system, including white blood cells. Inflammatory diseases such as rheumatoid arthritis are caused by the attack of the immune system, particularly white blood cells, in the joints. In a disease state this attack is directed, at least to some extent, by increased levels of chemokine molecules that attract white blood cells to joint tissue, a process called chemotaxis, where they cause tissue destruction. Chemokine receptor inhibitors block this attraction and are therefore potential therapeutics in this area. We believe chemokine inhibitors hold significant potential as new and potentially breakthrough drugs.

Our drug development program is focused on the CXCR4 receptor. Promising results from a number of preclinical studies with new compounds targeting the CXCR4 receptor have shown encouraging activity in rheumatoid arthritis and asthma models. Additional preclinical studies are ongoing to evaluate these compounds in cancer and other diseases. We will also explore potential collaboration and partnering opportunities in this area.

In addition, work continues on the identification of preclinical leads for CCR5 as a new HIV entry inhibitor (see “AMD-070 – A new HIV Entry Inhibitor”) as well as other chemokine receptors. We plan to select a preclinical candidate in 2003 and will also expand drug discovery and development in this program through a number of ongoing collaborations.

Nitric Oxide Scavengers

Overview

We have discontinued our research and development efforts on this product candidate. Instead we are seeking corporate partnership and out-licensing opportunities prior to continuing further development and commercialization of this compound. Nitric oxide scavengers have therapeutic potential in a variety of indications.

Nitric oxide (“NO”) is a molecule produced by the body that plays an essential role in brain function, immune system function and the regulation of blood pressure. Whereas minimum levels of NO are essential for normal function, excess levels of NO have toxic effects and are potentially involved in such diseases as inflammatory bowel disease, septic shock, arthritis, stroke, psoriasis and some degenerative eye diseases.

An important property of the metallic element ruthenium is its ability to bind with NO to form stable compounds. Our recognition of the potential applications of this property, in combination with our ability to design ruthenium compounds of low toxicity, led us to develop a series of ruthenium based NO scavengers. Our scavengers work by tightly binding NO, thus neutralizing its toxicity. A wide variety of ruthenium compounds were screened by our scientists for their ability to bind and scavenge NO. These experiments resulted in the selection of several compounds for further evaluation in animal models.

Our compounds have several potential advantages over alternative methods for lowering NO levels. Since our compounds bind directly with NO, they have the potential to be faster acting than those that interfere with the synthesis of NO. Compared to competing scavengers that are based on blood components, our compounds are less complex, readily synthesized molecules that are potentially easier and less expensive to manufacture. We are currently seeking corporate partners prior to continuing further development and commercialization of these compounds.

Business Strategy

We plan to continue and expand our drug discovery and development activities through the strategy outlined below.

Expand drug discovery capability. In order to enhance our ability to identify and develop a steady stream of new product candidates, we are enlarging the scope of our biological research activities. We are focussing on the identification of clinical candidates in our chemokine inhibitor program. This will require the addition of molecular biology, enzymology, rapid throughput screening and structural biology capabilities to enable us to produce these candidates and evaluate their therapeutic potential. In conjunction with the in-house activities described above, we will continue to evaluate promising opportunities to in-license from third parties, such as research centres, the rights to further develop work using small molecule compounds.

Continue strong preclinical collaborations with international research centres to identify and test new lead compounds. Our scientists interact extensively with our Scientific Advisory Board (see “Directors and Officers - Scientific Advisory Board”) and a broad network of research associates and collaborators to identify areas in medicine with large potential markets and compounds with therapeutic potential. Innovative ideas are initially tested by the in-house synthesis of prototype compounds followed by in-house screening using biochemical or cell culture experiments. If results at this stage are promising, external collaborators who are recognized in their field are identified to perform more advanced tests.

Progress lead compounds through early stage clinical trials. We intend to add value to our product candidates by taking them through Phase II clinical trials, thereby establishing efficacy in human patients, before negotiating the financial terms of corporate partnerships. We may enter into partnerships at earlier stages of development, preclinical or Phase I, if such collaborations would substantially accelerate product development and the likelihood of clinical and market success. Our in-house clinical and regulatory capabilities in the design and implementation of clinical trials are supplemented through the use of external collaborators and contractors and the use of recognized clinical and regulatory consultants.

Extend process development capability. We plan to extend our ability to design processes for the synthesis of our product candidates. Our research team has considerable experience in this area, having worked on the process development and regulatory chemistry aspects of several drugs including carboplatin, JM-216 and AMD-3100. We expect that commercial production will be outsourced since there are many inexpensive, efficient sources for synthesis of cGMP bulk active ingredients and finished drug product.

Establish corporate partnerships for late stage trials and marketing. We will continue to seek corporate partnerships with established pharmaceutical companies in order to carry on Phase III clinical trials, NDA filings and ultimately marketing of our products. This will mitigate the financial risk inherent in carrying out large multi-centre Phase III clinical trials, and the significant investment of money and resources to build a sales force required to address these markets.

Corporate Partnerships

We develop our product candidates both independently and in collaboration with established pharmaceutical companies or other suitable partners. Corporate partners may provide financial resources, research and development and manufacturing capabilities, and sales and marketing infrastructure, to aid in the commercialization of our potential products. Our corporate partnership agreements, some of which were initially entered into by JM and assigned to us pursuant to the Asset Transfer Agreement, are described above. See “General Development of the Business – Development of the Business”.

AstraZeneca. Effective March 31, 1998, we reached an agreement with AstraZeneca whereby we exclusively licensed to AstraZeneca, our novel anti-cancer agent AMD-473 for further development and commercialization. Under the terms of the agreement, AstraZeneca made an initial payment of \$8.6 million in consideration for patent rights and recognition of our research and early development efforts on AMD-473.

On April 5, 2001 we earned a second milestone payment of approximately U.S.\$4 million. Total upfront and milestones payments under the license agreement were approximately U.S.\$33 million of which U.S.\$10 million was earned.

On November 26th, 2001 AstraZeneca announced that they would not continue with the development of ZD-0473 (now AMD-473). This license agreement was legally terminated on February 28, 2002 and all the data and development rights were returned to AnorMED.

DuPont Pharmaceuticals. The rights to non-cancer applications to our HYNIC linker technology were originally licensed to Ortho Pharmaceuticals Corporation (“Ortho”) and subsequently sub-licensed to DuPont on June 7, 1994. Our license agreement with Ortho has been terminated.

On December 15, 1996, we entered into a license agreement with DuPont, whereby they were granted an exclusive worldwide license to our HYNIC linker technology for use in cancer applications. We have given notice of termination of this license and are currently negotiating, in conjunction with the license for non-cancer applications, an overall license agreement for use of the linker technology with Bristol-Myers Squibb MI, the acquirer of Dupont Medical Imaging business.

Shire Pharmaceuticals. On February 2, 1996, Shire exercised its option under an option agreement dated January 10, 1995 to acquire the worldwide exclusive license to our patent on rare earth anti-hyperphosphatemia agents. As consideration for the license, Shire will pay a royalty to us on worldwide product sales derived from this license. Under the terms of the license agreement Shire has assumed all development and commercialization expenses for Fosrenol. The license may be terminated by either party if there is a breach of material obligations or, in certain circumstances, the failure to meet specified milestones. Shire has the right to terminate development under this license agreement at any time prior to product approval. Upon termination of the license agreement all the underlying technology will revert to us.

North American Scientific, Inc. On July 20, 2001 we signed a non-exclusive licensing agreement with NASI for incorporation of our HYNIC linker technology into NASI’s second generation version of its ApomateTM, an imaging agent now in clinical trials. Under the terms of this agreement, we received a non-refundable licensing fee of U.S.\$75,000 and an upfront payment of U.S.\$62,500 for ongoing technical support. We will also receive milestone payments and future single digit royalties based on sales of products that incorporate our technology.

Research Collaborators

An important aspect of our product development strategy is the establishment of collaborations with research centres with resources and expertise vital to our programs. These collaborations provide us with access to *in vivo* disease models and *in vitro* testing capacity that would be difficult and expensive to establish in-house. In each collaboration, we either have an exclusive license to all patented or patentable technology developed under the research agreement or a right to an exclusive license on commercially reasonable terms. See “Narrative Description of

Business - Prior Royalty Agreements". We currently collaborate with the following research centres:

Rega Institute, Leuven, Belgium. On June 22, 1987, JM entered into a research agreement with the Rega Institute, a virology laboratory at Katholieke Universiteit. The agreement was assigned to us pursuant to the Asset Transfer Agreement and was amended by ourselves and the Rega Institute on April 1, 1999. Professor De Clercq of the Rega Institute is a world renowned researcher in antiviral chemotherapy and is a member of our Scientific Advisory Board. The Rega Institute will be responsible for primary anti-HIV screening of our compounds and is also capable of evaluating new compounds for oral bioavailability, a crucial component of the research plan. Under this research agreement, we will pay the Rega Institute a research fee and a percentage of any royalty we receive on any product developed through the Rega Institute's laboratory.

Cancer UK, Sutton, UK (Previously Institute of Cancer Research, Sutton, U.K.) On November 15, 1989, JM entered into a collaboration agreement with the ICR, a major cancer research centre, for the evaluation of new platinum anti-cancer agents. The agreement was assigned to us and subsequently amended on February 25 and March 24, 1998. Under the agreement, ICR is entitled to a percentage share of our revenues from products developed through ICR's laboratory.

A letter stating our intent to terminate our collaboration with ICR, under another agreement with the Institute dated December 31, 1999, for the evaluation of anti-cancer compounds was sent on April 15, 2002.

Johns Hopkins University, School of Medicine, Baltimore, Maryland, U.S.A. On August 1, 1998, we entered into a clinical trial agreement with the Johns Hopkins University to conduct clinical trials on AMD-3100. Under the agreement, we will pay a fee for each patient in the trial conducted by the University. We renewed this clinical trial agreement, under the same terms as the original agreement, on May 1, 1999.

University of Washington, Seattle, Washington; Case Western Reserve University, Cleveland, Ohio, U.S.A. On June 1, 1999 we entered into clinical trial agreements with the University of Washington and the Case Western Reserve University to conduct clinical trials on AMD-3100. Under the agreements, we will pay a fee for each patient in the trial at each University. The University of Washington research agreements were renewed on December 13, 2001 and April 25, 2002, under the same terms as the original agreement, for the evaluation of AMD-3100 in stem cell transplant.

Indiana University, Indiana, U.S.A. On September 29, 2000, we entered into an agreement with the Indiana University to study the role of stem cell mobilization using AMD-3100. Under this agreement, we will pay Indiana University costs associated with this study. This agreement was renewed under the same terms on December 12, 2001.

The Panum Institute, University of Copenhagen, Copenhagen, Denmark. On January 25, 2000, we entered into a research and consulting agreement with the University of Copenhagen to evaluate our chemokine inhibitors against a library of chemokine receptors for the purpose of

mapping molecular interactions. Under this agreement, we will pay the University of Copenhagen costs associated with this study.

University of Michigan Medical School, Michigan, U.S.A. On January 28, 2001, we entered into a research agreement with the University of Michigan to conduct studies relating to inhibiting allergen-induced lung inflammation and airway hyperactivity using our chemokine inhibitors. Under this agreement, we will pay the University of Michigan research cost associated with this agreement. On February 16, 2001, we entered into an additional research agreement with the University of Michigan to perform studies relating to the evaluation of our chemokine inhibitors to study the compound's role and its relationship with chemokine receptors during allergic airway fibrosis. Under this agreement, we will pay the University of Michigan research costs associated with this agreement.

British Biotech Pharmaceuticals Limited, Oxford, UK On April 24, 2001 we entered into a database purchase agreement with British Biotech. We purchased and acquired the full title and ownership rights to British Biotech database concerning their chemokine antagonists screening data and antagonist compounds against select chemokine receptors molecules. Under the terms of the acquisition, we paid approximately \$450,000 for exclusive rights to information generated from the screening of over 60,000 compounds against a variety of chemokine receptors.

Queen Mary and Westfield College, St. Bartholomew's and the Royal London School of Medicine and Dentistry, London, U.K. On May 16, 2001 we entered into a research agreement with the College to conduct research on chemokine inhibitors, i.e., AMD-3100, and its role as a potential anti-tumor compound using both invitro tumor cell lines and invivo tumor models. Under the agreement, we will pay a fee the College the research cost associated with such research. This study was discontinued in April 2002.

Prior Royalty Agreements

Under the terms of the Asset Transfer Agreement, we assumed the obligations of JM under several prior royalty agreements arising out of the transfer of intellectual property and, in some cases, funds for research and development, from related corporate and research collaborators.

In addition to its current collaborations with research institutes (see "Narrative Description of Business - Research Collaborators"), we are subject to additional royalty arrangements. The State University of New York is entitled to a percentage share of our royalties on certain of our HYNIC linker technology. Novartis AG is entitled to a percentage of any running royalties received by us under any licenses that we grant for products relating to AMD-3100, up to an aggregate maximum amount.

Intellectual Property

We regard our patent and other proprietary technology rights as one of the key strengths upon which we continue to build a successful pharmaceutical development company and therefore we intend to diligently file worldwide patent applications to protect our proprietary discoveries.

We hold the rights to 52 patents or patent applications in the United States relating to our technology, as well as foreign counterparts for most of these patents and patent applications. To

date, 31 patents have been issued in the United States. As with the patent positions of other pharmaceutical and biotechnology firms, we do not know whether any patent applications will result in the issuance of patents or, for patents that are issued, whether they will provide significant proprietary protection or will be circumvented or invalidated.

In addition to the patent strategy outlined above, we also rely upon trade secrets, know-how and continuing technological innovations to develop and maintain our competitive position. It is our policy to require our employees, consultants, members of the Scientific Advisory Board and parties to collaborative agreements to execute confidentiality agreements upon the commencement of employment, consulting relationships or a collaboration with us. These agreements provide that all confidential information developed or made known during the course of these relationships are to be kept confidential. In the case of our senior scientific staff, agreements are in place providing that all inventions resulting from work performed for us utilizing our property or relating to our business and conceived or completed by the individual during employment are our exclusive property to the extent permitted by law.

Competition

Based on our review of the industry and within a given market segment, competition is based on drug efficacy, safety, ease of use, patient compliance, price, marketing and distribution. Our ability to succeed in business will depend on our and our partner's or our licensee's ability to compete effectively in all these areas. There can be no assurance that our competitors will not succeed in developing products which are more effective than any that are being developed by ourselves, or which would render our technologies and products obsolete and non-competitive. See "Risk Factors".

The following is a discussion of all principal competitors which we have been able to identify as being active in related product areas. The discussion is based on our current understanding and should not be construed as identifying all our potential competitors.

Phosphate Scavengers. Our principal competitors in the area of new anti-hyperphosphatemia agents include Genzyme Inc. and Nephro-Tech Inc.

Platinum Anti-cancer Drugs. Our principal competitors in the area of platinum anti-cancer drugs include Access Pharmaceuticals, Inc., Neotherapeutics, Novuspharma SpA, Bristol-Myers Squibb, Sanofi SA, Alza Corporation, Shionogi & Co. and Asta Medica SA.

Chemokine Inhibitors. Our principal competitors in the area of chemokine inhibitors, specifically CXCR4 and CCR5 chemokine inhibitors, include Merck, Schering-Plough and GlaxoSmithKline.

Anti-HIV Drugs. There are numerous companies with anti-HIV drugs currently under development or approved for marketing. Companies with products which would directly compete with AnorMED in the area of anti-HIV drugs which inhibit viral fusion include Trimeris Inc., Millennium Pharmaceuticals Inc., Progenics Pharmaceuticals Inc., Schering-Plough Corporation and Takeda Chemical Industries Ltd.

Imaging Agents. Our principal competitors marketing or developing products using radiopharmaceuticals for the indications of infection and cancer include Nycomed Amersham PLC, Mallinckrodt Inc., Schering AG, Cytogen Corp., Palatin Technologies Inc. and Immunomedics Inc.

Product Approval Process

The production and manufacture of our new drug product candidates, and our research and development activities, are subject to regulation for safety and efficacy by applicable government authorities. In Canada, these activities are regulated by the Food and Drug Act (Canada) and the rules and regulations thereto, which are enforced by the Inspectorate of the Health Products and Food Branch (the “HPFB”). In the United States, drugs and biological products are subject to regulation by the Food and Drug Administration (“FDA”). Drug licensing laws require licensing of manufacturing facilities, carefully controlled research and testing of products, government review and approval of results prior to marketing products, and adherence to Canadian and U.S. cGMP during production.

The principal activities which must be completed before obtaining approval for marketing of any new drug product in Canada and the United States are as follows:

- (a) *Preclinical Studies.* Preclinical studies are conducted in animals to test pharmacology, efficacy and toxicology and to do formulation work based on in vivo results.
- (b) *Phase I Clinical Trials.* Phase I clinical trials consist of testing a product in a small number of humans, usually healthy subjects, to determine its toxicity, dose tolerance and pharmacokinetic properties.
- (c) *Phase II Clinical Trials.* Phase II clinical trials usually involve a larger patient population than is required for Phase I trials and are conducted to evaluate the effectiveness of a drug in patients having the disease or medical condition for which the drug is indicated. These trials also serve to identify possible common short-term side effects and risks in a larger group of patients.
- (d) *Phase III Clinical Trials.* Phase III clinical trials involve conducting tests in an expanded patient population at geographically dispersed test sites (multi-centre trials) to establish clinical safety and effectiveness. These trials also generate information from which the overall benefit-risk relationship relating to the drug can be determined and provide a basis for drug labelling.

Two key factors influencing the rate of progression of clinical trials are the rate at which patients can be recruited to participate in the research program and whether effective treatments are currently available for the disease the drug is intended to treat. Patient recruitment is largely dependent upon the incidence and severity of the disease, the treatments available and the potential side effects of the drug to be tested. An IND application must be filed and accepted by the HPFB or FDA, as applicable, before each phase of human clinical trials may begin. The IND application must contain specified information including the results of the preclinical or clinical tests completed at the time of the IND application. In addition, since the method of manufacture may affect the efficacy and safety of a drug, information on manufacturing methods and

standards and the stability of the drug substance and dosage form must be presented so that the HPFB can ensure that the product which may eventually be sold to the public has the same composition as that determined to be effective and safe in the clinical trials. Production methods and quality control procedures must be in place to ensure a relatively pure compound, essentially free of contamination and uniform with respect to all quality aspects.

Upon completion of all clinical trials the results are submitted to the HPFB as part of a new drug submission (“NDS”) or to the FDA as part of a product license application or New Drug Application (“NDA”) to obtain approval to commence marketing the drug. We anticipate that HPFB and FDA marketing approval for the majority of products will take between 12 and 36 months from the date an NDS in Canada or an NDA in the United States is submitted. In addition, an establishment license application must be filed and approved by the HPFB or FDA, as applicable, for the production of a product and test sites must demonstrate that cGMP standards have been maintained during preclinical and clinical evaluation. The HPFB and FDA may require post-market surveillance programs to monitor a product’s side-effects. Results of post-marketing programs may limit or expand the further marketing of products. A serious safety or effectiveness problem involving an approved drug may result in HPFB or FDA action requiring withdrawal of the product from the market and possible recall action.

Our or our corporate partners’ success in ultimately obtaining marketing approval for products currently under development will depend on their ability to comply with worldwide regulations governing the manufacturing, quality control, preclinical evaluation and clinical testing of INDs. Depending upon the circumstances surrounding the clinical evaluation of a product candidate, we may ourselves undertake clinical trials, contract clinical trial activities to contract research organizations or rely upon corporate partners for such development. We believe that this approach will allow us to make cost effective development decisions in a timely fashion.

Process Development And Manufacturing

We currently have no plans to establish manufacturing facilities for the commercial production of our product development candidates. Our strategy is to develop, manufacture and commercialize our therapeutic products through arrangements with major pharmaceutical and biotechnology companies and we may rely on such companies, licensees or other contract manufacturing entities for commercial scale manufacturing and marketing of our products. There can be no assurance, however, that we will be able to reach satisfactory arrangements with such parties, that such arrangements will be successful or that our partners or contractors will be able to develop adequate manufacturing capabilities for commercial scale quantities.

Human Resources

As of March 31, 2002, we employed or retained 103 persons, 72 of whom hold advanced degrees in science or business, including 40 who hold Ph.D. or M.Sc. degrees. In addition, we maintain affiliations with research centres including the Cancer UK, in London, U.K., the Rega Institute of Leuven, Belgium, the Johns Hopkins University School of Medicine of Baltimore, Maryland, and the University of Washington in Seattle.

Facilities

Our head office and main laboratory is located in Langley, British Columbia, Canada. The 32,000 square foot facility currently houses a modern laboratory and administrative offices. We moved into these facilities in February of 1997 under a 10 year lease with the option of three additional five year renewal terms. Annual lease commitments are approximately \$590,000 over the term of the lease. We have postponed plans for an expansion of an addition of approximately 11,500 square feet in our facility.

RISK FACTORS

Investment in the Common Shares involves a high degree of risk and should be regarded as speculative due to the nature of our business and because our product candidates are still in research and development. We have incurred losses and expect to incur further losses.

In addition to the other information contained in this Annual Information Form, the following factors should be considered carefully by investors when evaluating an investment in our securities.

Early Stage Development

We were founded in 1996 and are at an early stage of development. We have not completed the development of any products and, accordingly, have not begun to market or generate revenues from the commercialization of our products. Our products will require significant additional clinical testing and investment prior to commercialization. A commitment of substantial resources by ourselves and our partners to conduct time-consuming research and clinical trials will be required if we are to complete the development of our portfolio of product candidates. There can be no assurance that any of our products will meet applicable regulatory standards, obtain required regulatory approvals, be capable of being produced in commercial quantities at reasonable costs or be successfully marketed. Most of our products are not expected to be commercially available for several years.

Lack of Product Revenues; History of Operating Losses

To date, we have not recorded any revenues from the sale of products. From our date of incorporation to March 31, 2002, we have accumulated net losses of approximately \$47.3 million. We realized a net profit of \$1,696,000 during the year ended March 31, 1999 due to the initial payment from AstraZeneca; however, typically such payments only occur when significant development milestones are achieved. There can be no assurance that any such milestones will be achieved in the future. See "Narrative Description of the Business - Corporate Partnerships". Consequently, we expect losses to increase in the near term as we fund our clinical trials and eventually seek regulatory approval for the sale of our products. We expect to continue to incur substantial operating losses unless and until such time as product sales and royalty payments generate sufficient revenues to fund our continuing operations.

Additional Financing Requirements and Access to Capital

Since inception, we have raised approximately \$113 million, net of offering costs, from the sale of equity securities in private placements and public offerings. We will require substantial additional funds for further research and development, planned clinical trials and regulatory approvals. Further funding for these purposes may be achieved through public or private equity or debt financings, collaborative arrangements with pharmaceutical companies and/or from other sources. Further equity financings may substantially dilute the interest of our shareholders. There can be no assurance that additional funding will be available at all or on acceptable terms to permit successful commercialization of our products. Other than leases for certain capital equipment, we have no established bank financing arrangements, and there can be no assurance that we will be able to establish such arrangements on satisfactory terms.

Patents and Proprietary Rights

Our success will depend, in part, on our ability to obtain patents, maintain trade secret protection and operate without infringing on the proprietary rights of third parties or having third parties circumvent our rights. We have filed and are actively pursuing applications for U.S., Canadian and foreign patents. The patent positions of biotechnology and pharmaceutical companies can be highly uncertain and involve complex legal and factual questions. Thus, there can be no assurance that any of our patent applications will result in the issuance of patents, that we will develop additional proprietary products that are patentable, that any patents issued to us or those that already have been issued will provide us with any competitive advantages or will not be challenged by any third parties, that the patents of others will not impede our ability to do business or that third parties will not be able to circumvent our patents. Furthermore, there can be no assurance that others will not independently develop similar products, duplicate any of our products not under patent protection, or, if patents are issued to us, design around the patented products we developed or will develop.

We may be required to obtain licenses from third parties to avoid infringing patents or other proprietary rights. No assurance can be given that any licenses required under any such patents or proprietary rights would be made available, if at all, on terms we find acceptable. If we do not obtain such licenses, we could encounter delays in the introduction of products, or could find that the development, manufacture or sale of products requiring such licenses could be prohibited.

A number of pharmaceutical and biotechnology companies and research and academic institutions have developed technologies, filed patent applications or received patents on various technologies that may be related to or affect our business. Some of these technologies, applications or patents may conflict with our technologies or patent applications. Such conflict could limit the scope of the patents, if any, that we may be able to obtain or result in the denial of our patent applications. In addition, if patents that cover our activities are issued to other companies, there can be no assurance that we would be able to obtain licenses to these patents at a reasonable cost or be able to develop or obtain alternative technology. If we do not obtain such licenses, we could encounter delays in the introduction of products, or could find that the development, manufacture or sale of products requiring such licenses could be prohibited. In addition, we could incur substantial costs in defending ourselves in suits brought against us on patents it might infringe or in filing suits against others to have such patents declared invalid.

Since patent applications in the United States are maintained in secrecy until the patents issue or foreign counterparts, if any, publish and, since publication of discoveries in the scientific or patent literature often lag behind actual discoveries, we cannot be certain that we or any licensor were the first creator of inventions covered by pending patent applications or that we or such licensor was the first to file patent applications for such inventions. Moreover, we might have to participate in interference proceedings declared by the U.S. Patent and Trademark Office to determine priority of invention, which could result in substantial cost to us, even if the eventual outcome were favourable to us. There can be no assurance that our patents, if issued, would be held valid or enforceable by a court or that a competitor's technology or product would be found to infringe such patents.

Much of our know-how and technology may not be patentable. To protect our rights, we require employees, consultants, advisors and collaborators to enter into confidentiality agreements. There can be no assurance, however, that these agreements will provide meaningful protection for our trade secrets, know-how or other proprietary information in the event of any unauthorized use or disclosure. Further, our business may be adversely affected by competitors who independently develop competing technologies, especially if we obtain no, or only narrow, patent protection. See "Narrative Description of the Business - Intellectual Property".

Government Regulations

The manufacture and sale of human therapeutic and diagnostic products in the U.S. and Canada are governed by a variety of statutes and regulations in both countries. These laws require approval of manufacturing facilities, controlled research and testing of products and government review and approval of a submission containing manufacturing, preclinical and clinical data in order to obtain marketing approval based on establishing the safety and efficacy of the product for each use sought, including adherence to cGMP during production and storage, and control of marketing activities, including advertising and labelling.

The products we are currently developing will require significant development, preclinical and clinical testing and investment of substantial funds prior to their commercialization. The process of obtaining required approvals can be costly and time-consuming, and there can be no assurance that future products will be successfully developed and will prove to be safe and effective in clinical trials or receive applicable regulatory approvals. Markets other than the United States and Canada have similar restrictions. Potential investors and shareholders should be aware of the risks, problems, delays, expenses and difficulties which we may encounter in view of the extensive regulatory environment which controls our business.

Rapid Technological Change and Substantial Competition

We are engaged in a rapidly changing field. Other products and therapies that will compete directly with the products that we are seeking to develop and market currently exist or are being developed. Competition from fully integrated pharmaceutical companies and more established biotechnology companies is intense and is expected to increase. Most of these companies have significantly greater financial resources and expertise in discovery and development, manufacturing, preclinical and clinical testing, obtaining regulatory approvals and marketing than ourselves. Smaller companies may also prove to be significant competitors, particularly

through collaborative arrangements with large pharmaceutical and established biotechnology companies. Many of these competitors have significant products that have been approved or are in development and operate large, well funded discovery and development programs. Academic institutions, governmental agencies and other public and private research organizations also conduct research, seek patent protection and establish collaborative arrangements for therapeutic products and clinical development and marketing. These companies and institutions compete with us in recruiting and retaining highly qualified scientific and management personnel. In addition to the above factors, we will face competition based on product efficacy and safety, the timing and scope of regulatory approvals, availability of supply, marketing and sales capability, reimbursement coverage, price and patent position. There is no assurance that our competitors will not develop more effective or more affordable products, or achieve earlier patent protection or product commercialization, than our own.

Other companies may succeed in developing products earlier than ourselves, obtaining HPFB and FDA approvals for such products more rapidly than we will, or in developing products that are more effective than products we propose to develop. While we will seek to expand our technological capabilities in order to remain competitive, there can be no assurance that research and development by others will not render our technology or products obsolete or non-competitive or result in treatments or cures superior to any therapy we develop, or that any therapy we develop will be preferred to any existing or newly developed technologies. See “Narrative Description of the Business - Competition”.

Unproven Market

We believe that there will be many different applications for our products. We also believe that the anticipated market for our products will continue to expand. These assumptions may prove to be incorrect for a variety of reasons, including competition from other products and the degree of our products’ commercial viability.

Lack of Manufacturing and Marketing Experience

We have not yet introduced any products and have no manufacturing or marketing experience. To be successful, our products must be manufactured in commercial quantities in compliance with regulatory requirements and at acceptable costs. In order to manufacture our products in commercial quantities, if we elect to do so, we will need to develop our own manufacturing facilities or contract with third parties to manufacture our products. No assurance can be given that we will be able to make the transition to commercial production.

In addition, production of our products may require raw materials for which the sources and amount of supply are limited. An inability to obtain adequate supplies of such raw materials could significantly delay the development, regulatory approval and marketing of our products.

Reliance on Key Personnel

We are dependent on certain members of our management and scientific staff, the loss of services of one or more of whom could materially adversely affect us. Currently, five members of our management and scientific staff are permitted to work in Canada pursuant to employment authorizations issued by Immigration Canada. Employment authorizations are required to be

renewed periodically. There is no assurance that the employment authorizations will be renewed.

Our ability to manage growth effectively will require us to continue to implement and improve our management systems and to recruit and train new employees. Although we have done so in the past and expect to do so in the future, there can be no assurance that we will be able to successfully attract and retain skilled and experienced personnel.

Status of Healthcare Reimbursement

Our ability to commercialize products successfully may depend in part on the extent to which reimbursement for the cost of such products and related treatments will be available from government health administration authorities, private health coverage insurers and other organizations. Third-party payers in the U.S. are increasingly challenging the price of medical products and services, and new legislation has been proposed to attempt to provide broader and better health care and to manage and contain its cost. Significant uncertainty exists as to the reimbursement status of newly approved healthcare products, and there can be no assurance that adequate third-party coverage will be available to establish price levels sufficient for us to realize an appropriate return.

Foreign Exchange Fluctuation

We maintain our accounts in Canadian dollars. A portion of our revenue and expenditures are in foreign currencies, most notably in U.S. dollars, and therefore we are subject to foreign currency fluctuations which may, from time to time, impact our financial position and results. We enter into hedging arrangements typically through the use of forward or futures contracts to minimize the impact of decreases in the value of the U.S. dollar.

Potential Product Liability

Our business exposes us to potential product liability risks which are inherent in the testing, manufacturing, marketing and sale of therapeutic products. Human therapeutic products involve an inherent risk of product liability claims and associated adverse publicity. While we will continue to take precautions we deem appropriate, there can be no assurance that we will be able to avoid significant product liability exposure. We have product liability insurance coverage to a maximum of \$5 million per incident and an aggregate of \$10 million. Such insurance is expensive, difficult to obtain and may not continue to be available on acceptable terms, if at all. An inability to obtain sufficient insurance coverage on reasonable terms or to otherwise protect against potential product liability claims could prevent or inhibit the commercialization of our current or potential products. A product liability claim brought against us or a product withdrawal could have a material adverse effect upon us and our financial condition.

Dependence on Collaborative Partners

Our strategy is to enter into various arrangements with corporate and academic collaborators, licensors, licensees and others for the research, development, clinical testing, manufacturing, marketing and commercialization of our products. To date, we have also entered into research collaborations for the potential development and commercialization of our product candidates

with pharmaceutical firms, pursuant to which we could receive additional funding, including milestone payments from those parties. We intend to enter into additional corporate partnering agreements to develop and commercialize products based upon our drug delivery technology. There can be no assurance, however, that we will be able to establish such additional collaborations on favourable terms, if at all, or that our current or future collaborative arrangements will be successful.

Should any collaborative partner fail to develop or commercialize successfully any product to which we have rights, or any of the partner's products to which we have rights, our business may be adversely affected. In addition, while we believe that our collaborative partners will have sufficient economic motivation to continue their funding, there can be no assurance that any of these collaborations will be continued or result in successfully commercialized products. Failure of a collaborative partner to continue funding any particular program could delay or halt the development or commercialization of any products arising out of such program. In addition, there can be no assurance that the collaborative partners will not pursue alternative technologies or develop alternative products either on their own or in collaboration with others, including our competitors, as a means for developing treatments for the diseases targeted by our programs.

Hazardous Materials; Environmental Matters

Our discovery and development processes involve the controlled use of hazardous and radioactive materials. We are subject to federal, provincial and local laws and regulations governing the use, manufacture, storage, handling and disposal of such materials and certain waste products. Although we believe that our safety procedures for handling and disposing of such materials comply with the standards prescribed by such laws and regulations, the risk of accidental contamination or injury from these materials cannot be completely eliminated. In the event of such an accident, we could be held liable for any damages that result and any such liability could exceed our resources. We are not specifically insured with respect to this liability. Although we believe that we are in compliance in all material respects with applicable environmental laws and regulations and currently do not expect to make material capital expenditures for environmental control facilities in the near-term, there can be no assurance that we will not be required to incur significant costs to comply with environmental laws and regulations in the future, or that our operations, business or assets will not be materially adversely affected by current or future environmental laws or regulations.

SELECTED FINANCIAL INFORMATION

Consolidated financial results for each of our fiscal years ended March 31 since our incorporation summarized below:

Year ended March 31	2002	2001	2000	1999	1998	1997
Revenue	\$8,634,000	\$ 4,402,000	\$ 2,731,000	\$10,546,610	\$ 1,087,080	\$ 1,109,190
Net earnings (loss)	(13,368,000)	(11,299,000)	(10,506,000)	1,696,088	(6,391,499)	(7,438,973)
Total assets	82,220,000	95,602,000	76,534,000	62,839,144	30,858,037	26,543,772
Long-term debt	173,000	335,000	497,000	654,362	162,274	113,492
Per share earnings						
- Basic	(0.52)	(0.48)	(0.51)	0.11	(0.47)	(0.75)
- Fully diluted	(0.52)	(0.48)	(0.51)	0.10	(0.47)	(0.75)
Dividends	0	0	0	0	0	0

DIVIDENDS AND DIVIDEND POLICY

We did not pay any dividends in any of our fiscal years ended March 31, since our incorporation. We intend to retain all available funds, if any, for use in our business and do not anticipate paying any dividends in the foreseeable future.

MANAGEMENT'S DISCUSSION AND ANALYSIS AND FINANCIAL STATEMENTS

Our Management's Discussion and Analysis ("MD&A") and our comparative audited annual financial statements (the "Financial Statements") for the year ended March 31, 2002 (both included in our 2002 Annual Report, commencing at page 18 for MD&A under the heading "management's discussion and analysis" and at page 25 for the Financial Statements), are incorporated herein by this reference. Our MD&A and Financial Statements are available on SEDAR at www.sedar.com under our company name, AnorMED Inc.

MARKET FOR SECURITIES

Our common shares are listed and posted for trading on The Toronto Stock Exchange under the symbol "AOM".

INCORPORATION

We are incorporated under the Canada Business Corporations Act pursuant to articles of incorporation dated January 5, 1996. On March 17, 1999, we amended our articles to create a class of preferred shares (the "Preferred Shares" see below), create special rights and restrictions for our Common Shares and our Preferred Shares and increase the minimum and maximum number of our directors from 1 to 3 and 10 to 18 respectively.

Our authorized share capital consists of an unlimited number of Common Shares without par value and an unlimited number of Preferred Shares issuable in one or more series, without par value.

Common Shares. The holders of Common Shares are entitled to receive notice of any meeting of our shareholders and to attend and vote, except those meetings at which only the holders of shares of another class or of a particular series are entitled to vote. Each Common Share entitles its holder to one vote. Subject to the rights of the holders of Preferred Shares, the holders of Common Shares are entitled to receive on a pro-rata basis such dividends as our Board of Directors may declare out of funds legally available for this purpose. In the event of the dissolution, liquidation, winding-up or other distribution of our assets, such holders are entitled to receive on a pro-rata basis all of our assets remaining after payment of all of our liabilities, subject to the rights of holders of Preferred Shares. The Common Shares carry no pre-emptive or conversion rights. As at March 31, 2002, 25,636,965 Common Shares were issued and outstanding.

Preferred Shares. The Preferred Shares are issuable from time to time in one or more series, each series comprising the number of shares, designation, rights, privileges, restrictions and conditions determined by our Board of Directors. The Preferred Shares will be entitled to priority over the Common Shares with respect to the payment of dividends and distributions in the event of our dissolution, liquidation or winding-up. The holders of Preferred Shares are entitled to receive notice of any meeting of our shareholders and to attend and vote, except as otherwise provided in the rights and restrictions attached to the shares by the Board of Directors. As at March 31, 2002 there were no Preferred shares issued and outstanding we have no present intention to issue any Preferred Shares.

On September 7, 2000, our shareholders approved the adoption of a Shareholder Protection Rights Plan (the "Rights Plan") effective July 28, 2000 and expiring on July 28, 2010. The Rights Plan is designed to protect our shareholders from unfair, abusive or coercive take-over strategies, including the acquisition of control by a bidder in a transaction that may not treat all shareholders equally or fairly nor afford all shareholders an equal opportunity to share in the premium paid upon an acquisition of control.

The rights issued to shareholders under the Rights Plan entitle their holders (other than the acquiror) under certain conditions to acquire Common Shares at a 50% discount from the then prevailing market price if a person or related group acquires 20% or more of the outstanding Common Shares.

The dilutive effects of the rights are not triggered by a permitted bid as defined under the Rights Plan. A permitted bid must meet certain requirements, including the requirements that a take-over bid circular be prepared in compliance with applicable securities laws, the take-over bid be made to all shareholders and the take-over bid remain open for 60 days. If a bidder does not wish to make a permitted bid, the Board of Directors may still elect to redeem the rights or waive the application of the Rights Plan and allow the offer to proceed without dilution to the bidder.

DIRECTORS AND OFFICERS

The names, municipalities of residence and principal occupations within the last five years of each of our directors and officers are as follows:

Name, Municipality of Residence and Present Position with the Company	Date Became a Director/Officer⁽¹⁾	Principal Occupation Last Five Years⁽²⁾
David Scott ⁽³⁾⁽⁴⁾⁽⁶⁾⁽⁷⁾ Vancouver, British Columbia Chairman of the Board and Director	January 6, 1996	Retired December, 1999. Former President, MDS Ventures Pacific Inc., a subsidiary of MDS Capital Corporation (venture capital company)
Michael J. Abrams, Ph.D. Custer, Washington President, Chief Executive Officer and Director	January 6, 1996	Officer of the Company
Geoffrey W. Henson, Ph.D. Ferndale, Washington Executive Vice President and Chief Operating Officer	January 6, 1996	Officer of the Company
W.J. (Bill) Adams, C.A. Langley, British Columbia Vice President Finance, Chief Financial Officer, Secretary and Treasurer	June 27, 1997	Officer of the Company
Michael J. Cleare Ph.D. ⁽³⁾⁽⁷⁾ Kennett Square, Pennsylvania Director	June 28, 1996	Executive Director, Columbia University Innovation Enterprise. Former Managing Director and President, Electronics Materials Division and Pharmaceutical Materials Group, Johnson Matthey PLC (advanced materials company)
Julia Levy, Ph.D. ⁽³⁾⁽⁴⁾ Vancouver, British Columbia Director	September 30, 1996	Executive Chairman Scientific Advisory Board and Director, QLT Inc. (biopharmaceuticals company)
Colin Mallet ⁽⁵⁾⁽⁶⁾ Vancouver, British Columbia Director	September 30, 1996	Corporate Director
Michael E. Phillips ⁽⁴⁾⁽⁵⁾⁽⁶⁾ Vancouver, British Columbia Director	October 31, 1997	Senior Vice President Investment, Growthworks Capital Ltd., Manager of Working Opportunity Fund (EVCC) Ltd. (venture capital fund)
Willem Wassenaar, M.D. ⁽⁵⁾ Toronto, Ontario Director	September 30, 1996	President, Pharmacy.ca (web based retail pharmacy)
Edward J. Wawrzynczak, Ph.D. ⁽⁷⁾ Copenhagen, Denmark Director	March 18, 1998	Senior Portfolio Manager, Biotechnology & Healthcare, BankInvest Asset Management A/S (asset management company)

Name, Municipality of Residence and Present Position with the Company	Date Became a Director/Officer⁽¹⁾	Principal Occupation Last Five Years⁽²⁾
Gary J. Bridger, Ph.D. Bellingham, Washington Vice President, Research and Chief Scientific Officer	July 1, 2000	Officer of the Company
Christen M. Giandomenico, Ph.D. Blaine, Washington Vice President, Development	July 1, 2000	Officer of the Company
Gary Calandra, M.D., Ph.D. Cresco, Pennsylvania Vice President, Clinical Development	September 5, 2000	Officer of the Company
Paul Brennan, M.Sc. Surrey, British Columbia Vice President, Business Development	May 1, 2002	Officer of the Company
Renato Skerlj, Ph.D. Vancouver, British Columbia Vice President, Chemistry	March 13, 2002	Officer of the Company

Notes:

- (1) Each director is elected to hold office, subject to our By-laws, until the next annual meeting of shareholders or until the successor is elected or appointed.
- (2) Our directors and officers have held their present principal occupations noted opposite their respective names through the last five years except as described below.
- (3) Member of the Compensation Committee.
- (4) Member of the Finance Committee.
- (5) Member of the Audit Committee.
- (6) Member of the Corporate Governance Committee.
- (7) Member of the Nominating Committee.

Senior Management and Directors

The following are brief biographies of our senior management team and directors.

David Scott, M.B.A., Chairman of the Board and Director. Mr. Scott has spent over 30 years in investment analysis and management. He founded, built and sold a mutual fund management company, Scotiafund Financial Services Inc. He established the largest independent investment counselling firm in Atlantic Canada and later became president of North American Life's early stage venture capital affiliate, Toronto Shared Ventures Inc. In 1988, Mr. Scott joined Discovery Enterprises Inc. as President. From 1994 until his retirement in December, 1999, Mr. Scott had been the President of MDS Ventures Pacific. He was a founding director of the Academy of Chief Executives of Technology Companies. He is also a director of a number of technology and investment companies. Mr. Scott is a graduate of Bishop's University and holds an M.B.A. from the University of Western Ontario.

Michael J. Abrams, Ph.D., President, Chief Executive Officer and Director. Dr. Abrams has been active in the discovery and development of novel metal based drugs throughout his career. Dr. Abrams is an inventor on the patents that led to the development of the DuPont technetium-99m heart imaging agent, Cardiolite and is a co-inventor on JM 216, an orally administered platinum anti-tumor agent. In 1991, Dr. Abrams was promoted to Manager, Biomedical Research, worldwide for Johnson Matthey PLC (together with its affiliates, "JM"). He is a named inventor on 15 patents and has authored over 50 scientific articles. He received his Ph.D. in Chemistry from the Massachusetts Institute of Technology. Dr. Abrams is also a director of a technology company and a technology investment fund.

Geoffrey W. Henson, Ph.D., Executive Vice President and Chief Operating Officer. Dr. Henson has over 18 years of experience in the fields of cancer chemotherapy and immunology. Prior to joining AnorMED, Dr. Henson was Manager of Preclinical and Clinical Development for JM since 1991. He has more than 15 years experience in pharmaceutical discovery and development. Dr. Henson is a named inventor on three patents and has authored 22 scientific articles. He has a Ph.D. in Biochemistry from New Mexico State University and has held research positions at Roswell Park Memorial Institute and the Basel Institute of Immunology.

W.J. (Bill) Adams, C.A., Vice President Finance, Chief Financial Officer, Secretary and Treasurer. Mr. Adams was Chief Financial Officer and Corporate Secretary of Epic Data International Inc. from March 1993 until he joined AnorMED in June, 1997. Prior to March 1993, he held the position of Controller of Epic Data. Before joining Epic Data in January 1992, Mr. Adams was an audit manager with KPMG, and had audit responsibility for a number of high-tech clients. He is a Chartered Accountant and holds a Bachelor of Commerce Degree from the University of British Columbia.

Michael J. Cleare Ph.D., Director. Effective in August 2000 Dr. Cleare became Executive Director for the Columbia University Innovation Enterprise having retired from JM. After joining JM in 1966, Dr. Cleare participated in the original research into the anti-cancer properties of platinum compounds and is named co-inventor of carboplatin, a major anti-cancer drug. Dr. Cleare has held a number of positions within JM culminating in his appointment to the Executive Board of JM in 1995. In 1995 through 1997 he was Managing Director and President, Catalytic Systems Division Worldwide and Biomedical Products. Since 1997 until his retirement, he was the Managing Director and President of JM's Electronic Materials Division Worldwide and Pharmaceutical Materials Division. Dr. Cleare holds a Ph.D. in Chemistry from London University (U.K.).

Julia Levy, Ph.D., Director. Effective February 18, 2002, Dr. Levy became the Executive Chairman Scientific Advisory Board of QLT Inc. ("QLT"). A co-founder of QLT and Director, Dr. Levy served as President and Chief Executive Officer of QLT from February 1996 to February 2002. QLT is a world leader in photodynamic therapy, a new medical field which employs drugs activated by light. From November, 1995 to February 1996, Dr. Levy was Acting President and Chief Executive Officer of QLT. From 1986 to November, 1995, Dr. Levy was a Senior Vice-President or Vice-President of QLT. In addition, Dr. Levy was Acting President and Chief Executive Officer of QLT from May, 1991 to February, 1992. Dr. Levy has had extensive experience working with major pharmaceutical partners to commercialize photodynamic therapy in North America, Europe and Japan. Dr. Levy is a graduate of the

University of British Columbia and the University of London, and is a Professor of Microbiology at the University of British Columbia. She is a director of a number of technology companies and the Working Opportunity Fund (EVCC) Ltd.

Colin Mallet, Director. Mr. Mallet was Chairman of Synapse Technologies Inc., a biotechnology company in Vancouver, British Columbia, until March 2002. Since 1995, Mr. Mallet has been a Director of a number of public and private biotechnology and pharmaceutical companies; public companies where he is currently a Board member include Axcan Pharmaceuticals Inc. (since Nov 1995), and Micrologix Biotech Inc. (since Dec 1995). Prior to that, Mr. Mallet was President and CEO of Sandoz Canada Inc. for 7 years. Mr Mallet holds a B.A. from Cambridge University and has over 30 years of management experience in the pharmaceutical industry in Asia, Europe and North America.

Michael E. Phillips, Director. Mr. Phillips is a Senior Vice President, Investment of Growthworks Capital Ltd. which on January 1, 1999 became the manager of Working Opportunity Fund (EVCC) Ltd. ("WOF"), a British Columbia venture capital firm. Previous to January 1, 1999 he was a Senior Vice President, Investment of WOF (prior to January 1998, Vice President, Investment). He has over 20 years of venture capital experience and currently shares responsibility for managing venture capital activities of WOF. Prior to joining WOF in April 1993, Mr. Phillips was an investment manager with Vencap Equities Alberta Ltd., a publicly traded Alberta venture fund. He is currently a director of several technology companies.

Willem Wassenaar, M.D., M.B.A., Director. Dr. Wassenaar is President and founder of Pharmacy.ca, a web-based retail pharmacy, focused on personalized medicine. From May 1999 until July 2001 Dr. Wassenaar was the Chief Operating Officer of Transplantation Technologies Inc., a biotechnology Company. Dr. Wassenaar is a director on the boards of GlycoDesign Inc. Spectral Diagnostics Inc. and Sunnybrook Working Ventures Medical Break Through Fund. Dr. Wassenaar has over 20 years of experience in the pharmaceutical industry and has held the position of President for several companies including Sterling Drug Ltd. and Ferring Inc. In his professional career, Dr. Wassenaar's responsibilities in the pharmaceutical and biotechnology fields have been in areas of product development, marketing and sales and general management. Dr. Wassenaar holds an M.Sc. from the University of Toronto, an M.B.A. from York University and a M.D. from the University of Western Ontario.

Edward J. Wawrzynczak, Ph.D., Director. Dr. Wawrzynczak is the Senior Portfolio Manager, Biotechnology & Healthcare for BankInvest Asset Management A/S, Denmark. Formerly the Principal of Tardis Consulting, U.K., from February 2001 to September 2001. From July 1992 to January 2001 he has been an investment executive with Rothschild Asset Management Limited, an asset management company, most recently as the head of the Bioscience Unit and a director of several biotechnology companies. Dr. Wawrzynczak was Head of the Drug Targeting Laboratory at the Institute of Cancer Research in the U.K. until 1992. Dr. Wawrzynczak received a Ph.D. in Biochemistry from Cambridge University (U.K.).

Gary J. Bridger, Ph.D., Vice President, Research and Chief Scientific Officer. Dr. Bridger joined JM's biomedical research group in 1990 where he played a leading role in all aspects of chemistry research. Dr. Bridger is a named inventor on twelve patents and is the author of more

than 25 scientific articles. He received a Ph.D. in Chemistry from the University of Manchester Institute of Science and Technology (U.K.) and did post-doctoral research at Boston College.

Christen M. Giandomenico, Ph.D., Vice President, Development. Dr. Giandomenico joined JM in 1985 where he was a co-inventor of JM 216, the first orally active platinum anti-cancer drug. Dr. Giandomenico is a named inventor on six patents and is the author of 19 scientific articles. He received a Ph.D. in Chemistry from Columbia University and has held post-doctoral positions at the University of Chicago and the Stanford Research Institute.

Gary Calandra, M.D., Ph.D., Vice President, Clinical Development. Dr. Calandra has over 20 years of clinical trial experience in the areas of infectious and inflammatory disease. He was formerly the Vice-President Clinical Development at Pasteur Merieux Connaught in Swiftwater, Pennsylvania and prior to that he was Senior Director, Clinical Research at the Merck Research Laboratories in West Point, Pennsylvania. While at Merck, one of Dr. Calandra's areas of responsibility was the development of Crixivan for HIV infection.

Paul A. Brennan, M.Sc., Vice President, Business Development. Mr. Brennan brings to AnorMED over 13 year's global experience in the biotechnology and pharmaceutical industries. As Director, Global Licensing at AstraZeneca his responsibilities included product licensing, technology evaluation and acquisitions. Prior to his involvement with Global Licensing, Mr. Brennan held the position of Director of International Regulatory Affairs at Astra Draco AB. Mr. Brennan is responsible for leading our partnering initiatives and ongoing licensing activities, as well as strategic planning for new product development. Mr. Brennan received a Masters of Science degree from Queen's University, Ontario.

Renato T. Skerlj, Ph.D, Vice President, Chemistry. Dr. Skerlj held the position of Senior Research Chemist at Merck & Co., Inc. from October 1995 until he joined AnorMED in May 1998 as Director of Chemistry. Prior to October 1995 Dr. Skerlj was a Senior Scientist with Johnson Matthey's biomedical research group. Dr. Skerlj is a named inventor on 12 patents and is the author of 25 scientific articles. He received a Ph.D. in Chemistry from the University of British Columbia and did post-doctoral research at the University of Oxford (U.K.) and Ohio State University.

Scientific Advisory Board

We have formed a Scientific Advisory Board composed of scientists having professional experience and valuable expertise in various therapeutic or research fields that relate to our research and development programs. At our request, these scientific advisors review and provide us with advice regarding individual research and development projects. Advisors have all executed confidentiality agreements. The Scientific Advisory Board meets at least annually and makes its recommendations directly to management. Members of the Scientific Advisory Board are paid a fee of U.S.\$2,500 for each meeting attended plus travel expenses and each member has been awarded options under our incentive stock option plan. The following are brief biographies of the members of this advisory board.

Dr. A. Hilary Calvert, M.Sc., M.D., FRCP, Professor of Clinical Oncology and Director of the Cancer Research Unit, The University of Newcastle-upon-Tyne, U.K. Professor Calvert obtained

his medical qualifications (M.B.) from Cambridge University (U.K.) and from University College Hospital Medical School, London, in 1972. His continued studies in the field of cancer chemotherapy culminated in an MD Thesis in 1981 on the clinical applications of anti-folates. Professor Calvert is a world leader in the clinical introduction of new anti-cancer drugs. He played a key role in the introduction of carboplatin into clinical practice. He is on the editorial board of five cancer/pharmacology journals and has published 140 full papers in referred journals. Professor Calvert is the Chairman of the Cancer Research Campaign Phase I/II Committee (Clinical), is a Committee Member of the European Organization for Research and Treatment of Cancer (EORTC) Pharmacokinetics and Molecular Mechanisms Group and an active member of the Early Clinical Studies Group. In addition, he is a member of the European Drug Development Network, devoted to coordinating early clinical trials being conducted by the various European organizations and in 2001 was elected as a Fellow of the Academy of Medical Sciences, UK (F MedSci).

Dr. Erik De Clercq, M.D., Ph.D., Rega Institute, Katholieke Universiteit, Leuven, Belgium. Dr. De Clercq is an internationally recognized expert in antiviral chemotherapy. He received his MD in 1966 and his Ph.D. in 1972, both from the Katholieke Universiteit Leuven in Belgium. After post-doctoral training at Stanford University, first as a Lilly International Fellow and subsequently as a Damon Runyon Cancer Research Fellow, Dr. De Clercq returned to Leuven University Medical School where he became Professor in 1975. He served as Chairman of the Department of Microbiology from 1986 until 1991. In 1986, he also became Chairman of the Directory Board of the Rega Institute. In 1994, Dr. De Clercq was elected a Fellow of the American Association for the Advancement of Science and in 1995 was awarded the Professor P. De Somer Chair for Microbiology of the Katholieke Universiteit Leuven.

Dr. Thue W. Schwartz, M.D., University of Copenhagen, Copenhagen, Denmark. Professor Schwartz obtained an MD from the University of Copenhagen, trained in protein chemistry at the University of Aarhus, was a post-Doctoral Fellow at the department of biochemistry at The University of Chicago and holds a D.M.Sc. From the University of Copenhagen. He is currently a Professor of Molecular Pharmacology at the University of Copenhagen where his research is primarily focused on studying the structural and molecular biology of seven transmembrane G-coupled receptors. He was previously research professor at the Danish Medical Research Council and prior to that he was a research director at Novo Nordisk A/S where he lead departments of computational chemistry, molecular genetics, and molecular pharmacology. Professor Schwartz has published approximately 190 scientific papers, has received many prizes and honors including the Jahre Prize (Oslo 1990), the Viktor Mutt award (Santa Barbara, 1995), and recently the Lundbeck Foundation prize and the Novo Nordisk Prize. He is also member of the Royal Danish Academy of Sciences. Professor Schwartz is the Chief Scientific Officer of 7TM Pharma A/S and is on the board of directors of NeuroSearch A/S and Profound Pharma A/S, of which he was a co-founder.

Dr. Robert Schooley, Director of the Infectious Disease Division, Professor of Medicine at the University of Colorado Health Sciences Center. Dr. Schooley is a new member of our Scientific Advisory Board. He has replaced Dr. Robert Kerbel. Dr. Schooley has served as Chair of the Executive Committee and of the Immunology Committee of the National Institutes of Health (NIH) AIDS Clinical Trials Group, and as a member and Chair of the AIDS and Related

Retroviruses I Study Section of the NIH. He is also a staff physician for the University of Colorado Hospital and the Denver Veterans Affairs Medical Center.

Our directors and officers, as a group, beneficially own or exercise control or direction over an aggregate of 448,600 Common Shares (2,213,600 on a fully diluted basis) representing approximately 1.7% (7.8% on a fully diluted basis) of the Common Shares issued and outstanding as of the date hereof.

ADDITIONAL INFORMATION

We will provide to any person or company, on written request to our Corporate Secretary copies of the following documents:

- (a) when our securities are in the course of a distribution pursuant to a preliminary short form prospectus or a short form prospectus:
 - (i) one copy of this Annual Information Form, together with one copy of any document, or the pertinent pages of any document, incorporated by reference in this Annual Information Form;
 - (ii) one copy of our comparative audited financial statements for our most recently completed financial year for which financial statements have been filed, together with the auditors' report thereon, and one copy of any of our interim financial statements that have been filed subsequent to the financial statements for our most recently completed financial year;
 - (iii) one copy of our management proxy circular in respect of our most recent annual meeting of shareholders that involved the election of directors; and
 - (iv) one copy of any other documents that are incorporated by reference into our preliminary short form prospectus or short form prospectus and are not provided under (i) to (iii) above; or
- (b) at any other time, one copy of any of our documents referred to in (a)(i), (ii) and (iii) above, provided that we may require payment of a reasonable charge for such copy if the request is made by a person who is not our security holder

Additional information, including directors' and officers' remuneration and indebtedness, principal holders of our securities, options to purchase securities and interests of insiders in material transactions, where applicable, is contained in the Management Proxy Circular for our most recent annual general meeting of shareholders that involves the election of directors. Additional financial information is provided in our comparative audited annual financial statements for the year ending March 31, 2002. A copy of these documents may be obtained upon request from our Corporate Secretary or can be obtained from SEDAR at www.sedar.com under our company name, AnorMED Inc.

GLOSSARY OF TERMS

<u>Term</u>	<u>Definition</u>
Analog	A structural derivative of a compound.
cGMP (current Good Manufacturing Practices)	Codes of manufacturing practices published by the FDA and the HPFB intended to ensure that drug products are consistently manufactured to a quality appropriate for their intended use.
Chemotherapy	The treatment of disease with drugs, most often used for describing the treatment of cancer. These drugs interfere with the growth and division of cancer cells.
Chemokine	A type of biological signalling molecule.
Clinical trials	Organized studies with human patients designed to provide statistically relevant clinical data for determining the efficacy or safety of new therapeutic agents and diagnostic and medical devices.
Combination therapy	Patients are given two or more drugs in combination in order to increase efficacy by attacking the disease using two or more different routes.
Coordination chemistry	The science of how metal atoms interact with molecules and ions.
DNA (Deoxyribonucleic acid)	The chemical compound present in all cells of the body that is the carrier of genetic information.
Efficacy	The ability to produce a desired effect.
FDA (Food and Drug Administration)	The United States federal government agency that regulates the production, safety and efficacy of biological and pharmaceutical products in the United States.
Formulation	Preparation of the end-use dosage form of a drug by combining a pharmacologic product with non-medicinal agents. These agents can include solvents, preservatives, stabilizers, colorants, fillers and disintegrating agents to create the final dosage form (e.g. capsule, tablet or injection).
Genes	Small sections of a chromosomal DNA that carry the specific genetic information required for the production of a protein molecule by a cell.
HPFB (Health Products and Food Branch)	An agency of the Department of Health Canada that regulates the production, safety and efficacy of biological and pharmaceutical products in Canada.
In Vitro	Taking place outside the living body; sometimes used to include the growth of cells from multicellular organisms under cell culture conditions.
In Vivo	Taking place in a living organism.
IND	Investigational new drug.
IND application	A request for acceptance from the FDA or the HPFB to conduct a clinical evaluation of an IND. An IND application must include information on animal tests, a description of the proposed clinical trial and a list of investigators and their qualifications.
i-NOS	An inducible enzyme which produces Nitric Oxide (NO) as a response to stress.
Intravenous	Within a vein or veins.
Isotopes	Variant forms of a chemical element (at the atomic level) that differ from each other in atomic mass.
Kidney dialysis	A procedure for removing nitrogenous wastes (urea) from the body when the kidneys are not functioning effectively.
Linker	A chemical entity which holds two separate molecules together.

<u>Term</u>	<u>Definition</u>
MRI (Magnetic Resonance imaging)	A computerized diagnostic imaging system that uses radio frequency radiation as a source of energy for visualizing internal organs.
NDA (New Drug Application)	An application to the FDA for marketing approval for a new therapeutic agent made upon successful completion of clinical trials. The review time for a NDA is typically between 12 and 36 months and encompasses a review of all information related to animal tests, clinical trials, pharmacokinetics, drug manufacturing, processing and packaging.
Nitric Oxide (NO)	A molecule containing one atom of nitrogen and one atom of oxygen.
NOS (Nitric Oxide Synthase)	An enzyme which produces NO.
Pharmacokinetics	The study of how a drug is absorbed by, distributed through and excreted by the body.
Radioisotopes	Isotopes which emit radiation.
Receptor	A structure within a cell or on the surface of a cell that selectively binds a specific substance resulting in a specific physiologic effect.
Scavenger	A substance added to remove unwanted substances.
Sub-cutaneous	Under the skin.
Technetium	A radioactive element widely used in nuclear medicine imaging studies.
Therapeutics	Products for treating medical disorders or diseases, including synthetic small molecule drugs (pharmaceuticals), biologically derived agents (biopharmaceuticals), plant or animal extracts, human tissue products, genetic factors, and immune components.
TNF	Tumor necrosis factor, a protein involved in inflammation.
Toxicity	A condition that results from exposure to a poison or to poisonous amounts of a substance that does not cause side effects in smaller amounts but can exert harmful side effects in large doses.
Xenografts	Grafts of tissue that have been transplanted between animals of different species.

All references in this document to “we”, “us”, “our” or similar terms means AnorMED Inc.