ALFACELL CORP
Form 10-K
October 29, 2003

U. S. SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 10-K

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

July 31, 2003	0-11088
For the fiscal year ended	Commission file number

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

Delaware	22-2369085
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification No.)

225 Belleville Avenue, Bloomfield, New Jersey 07003
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: (973) 748-8082

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

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

Common Stock, $.001 par value
(Title of Class)

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

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

Indicate by check mark whether the registrant is an accelerated filer (as defined in Exchange Act Rule 12b-2). Yes |_| No |X|

The aggregate market value of the common stock, par value $.001 per share, held by non-affiliates based upon the reported last sale price of the Common Stock on September 3, 2003 was approximately $22,404,593. As of October 27, 2003 there were 28,183,658 shares of common stock, par value $.001 per share, outstanding.

Documents Incorporated by Reference

Portions of the registrant's definitive Proxy Statement for the Annual Meeting of the Stockholders scheduled to be held on January 14, 2004, to be filed with the Commission not later than 120 days after the close of the registrant's fiscal year, has been incorporated by reference, in whole or in part, into Part III Items 10, 11, 12, 13 and 14 of this Annual Report on Form 10-K.

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The following trademarks appear in this Annual Report: ONCONASE(R) is the registered trademark of Alfacell Corporation, exclusively for the anti-cancer indications; Alimta(R) and Gemzar(R) are registered trademarks of Eli Lilly; Navelbine is a registered trademark of Glaxo Smith Kline.

All information on this Form 10-K is as of October 29, 2003 and we undertake no obligation to update this information.

We maintain a website at www.alfacell.com to provide information to the general public and our stockholders on our products, resources and services along with general information on Alfacell and its management, career opportunities, financial results and press releases. Copies of our most recent Annual Report on Form 10-K, our Quarterly Reports on Form 10-Q or our other reports filed with the Securities and Exchange Commission, or SEC, can be obtained, free of charge as soon as reasonably practicable after such material is electronically filed with, or furnished to the SEC, from our [Investor Relations Department] by calling 973-748-8082, through an e-mail request from our website at www.alfacell.com/info.htm, or through the SEC's website by clicking the direct link from our website at www.alfacell.com/investinfo.htm or directly from the SEC's website at www.sec.gov. Our website and the information contained therein or connected thereto are not intended to be incorporated into this Annual Report on Form 10-K.

Information contained herein contains, in addition to historical information, forward-looking statements that involve risks and uncertainties. All statements, other than statements of historical fact, regarding our financial position, potential, business strategy, plans and objectives for future operations are "forward-looking statements." These statements are commonly identified by the use of forward-looking terms and phrases such as "anticipates," "believes," "estimates," "expects," "intends," "may," "seeks," "should," or "will" or the negative thereof or other variations thereon or comparable terminology, or by discussions of strategy. Actual future results may vary from expectations set forth in these forward-looking statements. The matters set forth in Exhibit 99.1 hereto constitute cautionary statements identifying important factors with respect to these forward-looking statements, including certain risks and uncertainties, that could cause actual results to vary significantly from the future results indicated in these forward-looking statements. Other factors could also cause actual results to differ significantly from the future results indicated in these forward-looking statements.

Part I

Item 1. BUSINESS.

Overview

Alfacell Corporation, a biopharmaceutical company, is a Delaware corporation primarily engaged in the discovery and development of a new therapeutic class of drugs for the treatment of cancer and other pathological conditions. Based on our proprietary Ribonuclease, or RNase technology platform, our drug discovery and development program consists of novel therapeutics developed from amphibian ribonucleases. These primordial enzymes play important roles in nature. They mediate several essential biological activities, namely, regulation of cell proliferation, maturation, differentiation and cell death. Therefore, they are ideal candidates for the development of therapeutics for cancer and other life-threatening diseases, including HIV and autoimmune diseases, that require anti-proliferative and apoptotic, or programmed cell death, properties. We are recognized as a leader in the development of RNase based therapeutics and as such, have both co-sponsored and been a key participant in the International Ribonuclease Meetings held every three years.

ONCONASE(R), our trademark name for ranpirnase and our flagship product, is undergoing the last stage of clinical testing, or Phase III. This international randomized Phase III trial for patients with unresectable malignant mesothelioma, an inoperable form of cancer found in the lining of the lung and

abdomen, is ongoing. We have also conducted other randomized and non-randomized trials with patients with advanced stages of solid tumors in other types of cancers.

ONCONASE(R) is a novel amphibian ribonuclease, unique among the superfamily of pancreatic ribonuclease that has been isolated from the eggs of the leopard frog. We have determined that, thus far, ranpirnase, the generic name of ONCONASE(R) , is the smallest known protein belonging to the superfamily of pancreatic ribonuclease and has been shown, on a molecular level, to re-regulate the unregulated growth and proliferation of cancer cells. ONCONASE(R), unlike most cancer drugs, that attack all cells regardless of their phenotype, malignant vs. normal, and produce a variety of severe toxicities, is not an indiscriminate cytotoxic agent, but rather, its activity is mediated through elegant molecular mechanisms. ONCONASE(R) affects primarily exponentially growing malignant cells.

In December 2002, we received Fast Track Designation from the Food and Drug Administration, or the FDA for the treatment of malignant mesothelioma patients with ONCONASE(R). In February 2001, we received an Orphan Medicinal Product Designation for ONCONASE(R) from the European Agency for the Evaluation of Medicinal

Products, or the EMEA. These designations to ONCONASE(R) may serve to expedite its regulatory review, assuming the clinical trials yield a positive result.

Our proprietary drug discovery program forms the basis for the development of recombinant designer RNases for chemical conjugation, or chemical construct, and gene fusion products with various targeting moieties such as monoclonal antibodies, growth factors, cytokines, etc. This program provides for joint design and generation of new products with outside partners. We may own these new products along with a partner(s), or we may grant an exclusive license to the collaborating partner(s).

We have established a number of scientific collaborations with the National Cancer Institute, or NCI that are designed to develop new therapeutic applications for ONCONASE(R). One collaboration has produced RN321, a conjugate of ranpirnase, with a monoclonal antibody that demonstrated activity in treating non-Hodgkin's lymphoma in preclinical studies. These results were presented by the NCI investigators at the 2002 Ribonuclease Meeting in Bath, England. The NCI has undertaken the manufacturing of RN321 (the conjugate) according to Good Manufacturing Practices, or GMP regulations in preparation for commencing clinical trials for the treatment of patients with non-Hodgkin's lymphoma with RN321.

We have also discovered another series of proteins, collectively named amphinases, that may have therapeutic uses. These proteins are bioactive and have both anti-cancer and anti-viral activity. In addition to ranpirnase, we have isolated several other proteins from eggs of the leopard frog, or Rana pipiens. All of the proteins characterized to date are RNases. Information on four of these proteins was presented at the 2002 Ribonuclease Meeting. These products are currently undergoing preclinical testing. We are currently in negotiations with potential pharmaceutical partners for the development of these new compounds as conjugates and fusion proteins.

We have entered into a research and development collaboration with a major US privately held stent and drug delivery company. ONCONASE(R) is being evaluated in stents and other delivery platforms to treat cardiovascular disease and cancer via direct site delivery. This collaboration may result in licensing

agreement between the companies, however; there is no assurance that such agreement will be reached.

We have entered into a collaborative agreement (antiviral screening, non-SARS) with the National Institute of Allergy and Infectious Diseases, or NIAID in which five potential drug candidates (natural and genetically engineered) are under evaluation against various viruses.

Our research and development collaboration with Wyeth Pharmaceuticals is ongoing to develop a number of designer drugs such as conjugates and fusion proteins for a variety of indications using our proprietary technology. This collaboration may result in a licensing agreement between the companies, however; there is no assurance that such an agreement will be reached.

We have signed confidentiality agreements and have entered into discussions and due diligence with a number of companies for US or non-US marketing rights for ONCONASE(R) and for out-licensing some of our early drug candidates.

We are engaged in the research, development and clinical trials of our products both independently and through research collaborations. We have financed our operations since inception through the sale of our equity securities, private placements, convertible debentures and loans. These funds provide us with the resources to acquire staff, facilities, capital equipment, finance our technology, product development, manufacturing and clinical trials.

RESEARCH AND DEVELOPMENT PROGRAMS

Research and Development

Research and development expenses for the fiscal years ended July 31, 2003, 2002, and 2001 were $1,700,000, $2,033,000, and $1,901,000, respectively. Our research and development programs focus primarily on the development of therapeutics from amphibian ribonucleases. Because ribonucleases have been shown to be involved in the regulation of cell proliferation, maturation, differentiation and programmed cell death, known as apoptosis,

ribonucleases may be ideal candidates for the development of therapeutics for the treatment of cancer and other life-threatening diseases, including viral and autoimmune diseases that require anti-proliferative and pro-apoptotic properties.

Technology Platform and Pipeline

Using ribonucleases as therapeutics is a relatively new approach to drug development. The use of these proteins to re-regulate the unregulated growth and proliferation of cancer cells is unlike most cancer drugs that attack all cells regardless of their phenotype, malignant versus normal. These anticancer drugs are known to produce a variety of severe toxicities. ONCONASE(R) and related drug candidates are not indiscriminate cytotoxic agents, but rather, their activity is mediated through elegant molecular mechanisms. They affect primarily exponentially growing malignant cells.

Cancer is associated with the over or under production of many types of proteins in tumor cells. We believe that the ability to selectively halt the production of certain proteins via ribonuclease activity in tumor cells without damaging normal cells, may make treatment of cancer more effective. To make cancer therapy more effective and less toxic, we are developing ONCONASE(R) and a related family of regulatory proteins, collectively named amphinases. These

novel RNases are being developed as therapeutics as well as effector moieties (payload), or killer molecules for targeted therapies. We believe that selective degradation of intracellular proteins is central to the process of programmed cell death.

We have devoted significant resources towards the development of recombinant designer RNases for chemical conjugation and gene fusion products with various targeting moieties such as monoclonal antibodies, growth factors, cytokines, etc.

Apoptosis

Apoptosis, or programmed cell death, is essential for the proper development of embryos and of many body systems, including the central nervous system, immune regulation and others. Apoptosis is required to accommodate the billions of new cells produced daily by our bodies and to eliminate aged or damaged cells. Abnormal regulation of the apoptosis process can result in disease. For example, cancer, autoimmune disorders and many viral infections are associated with inhibited apoptosis or programmed death of cells occuring too slowly. Conversely, HIV is associated with increased apoptosis or programmed death of cells occuring too rapidly. The process of programmed cell death is genetically regulated. We have been recognized as the first company to discover and develop a novel family of primordial "regulatory" proteins that have been shown to play a fundamental role in this process.

ONCONASE(R) (ranpirnase) Pro-Apoptotic Mechanisms

The molecular mechanisms were identified which determine the apoptotic cell death induced by ranpirnase. Ranpirnase preferentially degrades tRNA, leaving rRNA and mRNA apparently undamaged. The RNA damage induced by ranpirnase appears to represent a "death signal", or triggers a chain of molecular events culminating in the activation of proteolytic enzyme cascades which, in turn, induces disintegration of the cellular components and finally execute tumor cell death. It has been shown that there is a protein synthesis inhibition-independent component, which, together with the changes induced by the protein synthesis inhibition, results in tumor cell death.

Many cancer cells become resistant to most types of cancer treatment, including chemotherapy, radiation and monoclonal antibodies. Overcoming resistance to chemotherapy remains a major challenge for cancer therapy. ONCONASE(R) has shown to overcome multiple drug resistance or prevent resistance to cancer therapy, thereby dramatically increasing the sensitivity of certain cancer cells to chemotherapy and radiation therapy.

It remains unknown whether or not ONCONASE(R) targets and binds preferentially to tumor cells, rather than normal cells of the respective tissues. It is possible that there is no differential targeting and/or binding, but that tumor cells are more susceptible to the cytostatic and cytotoxic effects of ONCONASE(R). The cytostatic effects are manifested by the inhibition of progression in the cell cycle (G1 phase block and by inhibition of expression of cyclin D3). These effects have been associated with induction of parallel differentiation and apoptosis. The cytostatic and

differentiation-inducing effects are reflected in the stabilization of previously progressive tumors observed in our clinical trials.

Preclinical Development and Clinical Studies of ONCONASE(R)

We have been very selective in our product development strategy, which is focused on the use of ONCONASE(R) alone or in combination with drugs which have shown evidence of preclinical and clinical efficacy on tumor types for which median survivals are typically less than a year and for which there are few or no approved treatments.

ONCONASE(R) has been tested in Phase I, Phase II and Phase III clinical trials in more than 40 cancer centers across the United States since 1991 and in Europe since 2000, including major centers such as Columbia-Presbyterian, University of Chicago, M.D. Anderson and Cedars-Sinai Cancer Centers.

ONCONASE(R) has been tested as a single agent in patients with a variety of solid tumors. It has also been tested in combination with tamoxifen in patients with prostate cancer, advanced pancreatic cancer and renal cell carcinoma as well as with doxorubicin in patients with malignant mesothelioma.

In order to affect RNA activity, ONCONASE(R) must enter the cell. After intravenous injection, ONCONASE(R) distributes rapidly to organs, especially the kidney. ONCONASE(R) is excreted predominately by the kidney. Biodistribution studies of ONCONASE(R) in vivo, or studies done in laboratory animals, have demonstrated high tumor tissue uptake rates relative to organ distribution.

We have been in collaboration with the National Institute of Health, or NIH including NCI, as well as a number of well-renowned academic institutions, in the United States, Europe, and Japan and have developed a considerable body of knowledge in RNase technology and novel RNase-based therapeutics. We believe that ONCONASE(R) is recognized as the "gold standard" in RNase research, as reflected by the plethora of peer-reviewed publications. ONCONASE(R) has demonstrated a broad spectrum of anti-tumor activity in vitro, or studies of tumor cell lines in laboratory vessels, and was determined to kill cancer cells and therefore was judged to be "active" in the NCI Cancer Screen.

In vitro and in vivo studies showed both cytostatic (stops cancer cells from further dividing) and cytotoxic (induces cancer cells to disintegrate) antitumor activity when used as a single agent and in combination with other agents.

In Vitro

ONCONASE(R), in combination with other drugs, has been shown to be synergistic which means that the effect of ONCONASE(R) when given in combination with other drugs is greater than if the drugs were given alone. The results of these studies have been published. The combination of ONCONASE(R) and tamoxifen resulted in a significant cell kill in pancreatic, prostate, and ovarian tumor cell lines as compared to each drug alone. Similar results were found with respect to the following:

- ONCONASE(R)+ phenothiazine for non-small cell lung cancer;
- ONCONASE(R)+ lovastatin in pancreatic, ovarian, and two types of non-small cell lung cancer;
- ONCONASE(R)+ cisplatin in ovarian cancer;
- ONCONASE(R)+ all-trans-retinoic acid in glioma (brain) cancer;
- ONCONASE(R)+ vincristine in colorectal cancer and ;
- ONCONASE(R)+ doxorubicin in breast cancer including resistant variants.

In Vivo Anti-Cancer Activity

ONCONASE(R) as a Single Agent

ONCONASE(R) as a single agent has shown in vivo anti-tumor activity in several mouse models of solid tumors:

- In the human squamous A-253 carcinoma and the NIH-OVCAR-3 ovarian adenocarcinoma models, ONCONASE(R) has produced prolonged survival and delayed time to development of ascites (fluid in the abdomen), respectively.

- In mice bearing M109 Madison lung carcinoma cells, time to appearance of ascites and survival were significantly prolonged in ONCONASE(R) treated animals as compared to controls. Several histologically confirmed cures were noted.

- In nude mice bearing human DU-145 prostate carcinoma and pancreatic ASPC-1 carcinoma, ONCONASE(R) inhibited growth of the subcutaneously transplanted tumor.

- In several mouse tumor models, ONCONASE(R) not only demonstrated direct anti-tumor activity but also increased the potential for other drugs to penetrate the tumor tissue as well as increased the tumor sensitivity to radiation therapy.

ONCONASE(R) in Combination With Other Agents

Based on in vivo results, ONCONASE(R) in combination with the following anti-cancer agents has been evaluated by us, in collaboration with the NCI, and the results have been published:

- vincristine

- doxorubicin

- tamoxifen.

ONCONASE(R) prolonged the survival of nude mice bearing vincristine-resistant, HT-29 human colorectal carcinomas transfected with mdr-1 gene, when used in combination with vincristine. These NCI results demonstrated that ONCONASE(R) can restore the sensitivity of resistant tumor cells to chemotherapy.

NCI experiments in nude mice transplanted intravenously with human breast carcinoma cells treated with the combination of ONCONASE(R) and doxorubicin have shown significantly prolonged survival. Tumor growth was significantly inhibited as demonstrated by a decrease in the number pulmonary metastases present at the time of sacrifice.

NCI reported the ability of ONCONASE(R) to overcome multiple drug resistance as well as other forms of drug resistance (referring to a drug that no longer kills cancer cells) both in vitro and in vivo. We believe that these in vivo results demonstrate the therapeutic utility of ONCONASE(R) in chemotherapy-resistant tumors, and the findings suggest that ONCONASE(R) in combination with other agents has broad clinical application in cancer treatments.

Clinical Trials

Onconase(R) Phase III Randomized Clinical Trials

We are currently conducting a two-part Phase III clinical trial of ONCONASE(R) as a treatment for malignant mesothelioma. The first part of the Phase III trial compares ONCONASE(R) alone to doxorubicin. Doxorubicin has been considered by opinion leaders to be the most effective drug for the treatment of malignant mesothelioma. The second part of the trial compares the combination of ONCONASE(R) and doxorubicin versus doxorubicin alone. The trial is a nonrandomized, controlled study. The patient enrollment for the first part of the clinical trial has been completed and the trial is on-going. The second part is currently in the enrollment stage and is being conducted in the United States, Germany and Italy.

Since ONCONASE(R) has Fast Track Designation for the treatment of malignant mesothelioma patients, we continue to have meetings and discussions with the FDA to establish mutually agreed upon parameters for the New Drug Application, or NDA to obtain marketing approval for ONCONASE(R), assuming the Phase III clinical trial yields favorable results.

Phase III Single Agent Results

The single agent Phase III results of the Treatment Target Group, or TTG, which included 104 patients, of which 47 were treated with ONCONASE(R) and 57 were treated with doxorubicin who met the criteria for Cancer Adult Leukemia Group B, or CALGB prognostic groups 1-4, showed a median survival benefit, or MST, of 2 months for ONCONASE(R) treated patients, 11.6 months vs. 9.6 months. This two month median survival difference favoring ONCONASE(R) represents a 20% advantage over the active agent, doxorubicin. Moreover, the clinical activity of ONCONASE(R) is also evident from the overall 1-year and 2-year survival rates of ONCONASE(R) vs. doxorubicin, 46.8% vs. 38.6% and 20.2% vs. 12.3%, respectively. Doxorubicin treatment was associated with a 60% higher risk of death compared to ONCONASE(R) treatment. Tumor assessment by an independent radiologist for 53 patients revealed evidence of objective clinical activity in 17 patients in each treatment arm. Four partial responses and 13 stabilization of previously progressive disease in the ONCONASE(R) treated patients and 7 partial responses and 10 stabilization of previously progressive disease in the doxorubicin treated patients. Despite the small number of patients, the analysis revealed a statistically significant difference, log rank test, p. = 0.037, in survival of the responders favoring ONCONASE(R) treated patients with an MST 23.3 vs. 14.4 months for doxorubicin treated patients as well as the 2 year survival rates of 40% for ONCONASE(R) and 9% for doxorubicin. Preliminary results were presented at the 2000 American Society of Clinical Oncologists, or ASCO, meeting.

These survival advantages were recognized as clinically important in this patient population by opinion leaders and the FDA. Therefore, the FDA has requested confirmation of the survival results in the TTG population in Part II of the ongoing trial.

We have obtained Fast Track Designation from the FDA for the treatment of malignant mesothelioma patients with ONCONASE(R) and doxorubicin. Fast Track is a formal mechanism to interact with the FDA using approaches that are available to all applicants for marketing claims for drugs that are being developed for a serious or life-threatening disease for which there is an unmet medical need. The benefits of Fast Track include scheduled meetings to seek FDA input into development plans, the option of submitting an NDA in sections rather than all components simultaneously, and the option of requesting evaluation of studies using surrogate endpoints. We intend to use this designation to reduce the marketing approval timeline for ONCONASE(R).

In February 2001, we received an Orphan Medicinal Product Designation for

ONCONASE(R) from the EMEA. We continue to fulfill the EMEA requirements regarding the Marketing Authorization Application, or MAA registration requirements for ONCONASE(R) for the treatment of malignant mesothelioma.

In part two of the ongoing Phase III trial, an interim analysis based on the occurrence of 105 deaths is planned. Based upon the results of these analyses, we may be able to file an NDA and an MAA within 6 months after the completion of the analyses. However, we cannot assure you that marketing approval for ONCONASE(R) as a treatment for malignant mesothelioma will be granted by the FDA or EMEA.

We had initiated a Phase III trial in patients with advanced pancreatic cancer in 1995 after meeting with the FDA, based on the Phase II trial results. The median survival time of 5.5 months for 47 patients with stage 4 disease and liver involvement treated with the combination of ONCONASE(R) weekly and tamoxifen daily was more than double the median survival of such patients reported in previously published trials treated with a variety of other systemic therapies (published median survival times ranged from 2.0 to 2.5 months). Multicenter randomized trials were designed to evaluate ONCONASE(R) and tamoxifen regimen in untreated patients as well as patients who had failed GEMZAR(R), an approved drug for pancreatic cancer. The primary endpoint of both trials was survival. Early survival analyses of both trials did not reveal a significant survival advantage over the controls. Therefore, we made a decision that further evaluation of this end-stage patient population was not warranted at that time and our resources were refocused on the ongoing malignant mesothelioma program.

ONCONASE(R) Phase II Clinical Trials

ONCONASE(R) as a single agent, demonstrated objective clinical activity in 105 patients with uresectable malignant mesothelioma that included many heavily pretreated patients with refractory tumors. Analysis of the TTG population confirmed the importance of the CALGB prognostic groups and their utility for evaluating systemic therapies in this patient population.

41 patients, 39%, reported evidence of clinical activity of which there were four partial responses, two minor responses and 35 stabilization of previously progressive disease. The MST of these patients was 18.5 months and the overall 1-year and 2-year survival rates were 61% and 40.8%, respectively. Two patients, 1 PR and 1 SD had their residual tumors resected after termination from the study and remain alive and tumor free for over 4 years after resection. The results of this trial demonstrated a survival benefit for both newly diagnosed patients and patients who failed prior therapies. The presentation of these data to the FDA resulted in the design of our Phase III malignant mesothelioma trial.

A multicenter Phase II Broad Eligibility trial designed to evaluate ONCONASE(R) as a single agent has been conducted and results of the findings for patients with non-small cell lung cancer, or NSCLC, and advanced breast cancer have been published.

ONCONASE(R) as a single agent, demonstrated objective clinical activity in patients with advanced NSCLC and breast cancer. The median survival time of 30 patients with advanced NSCLC was greater than that in 19 of 20 regimens when supportive care, a placebo or another single agent was given. Furthermore it was greater than 75% of the reported MSTs in combination chemotherapy trials. The MST and 1 year survival rates of 7.7 months and 27% for ONCONASE(R) treated patients compared favorably to 7.2 months and 30% for patients treated with Navelbine(R) (an approved drug for this indication) as a single agent.

Thirty percent of 17 patients with advanced breast cancer demonstrated objective clinical activity, which included, one partial response, two minor responses and significant reduction in bone pain and control of uncontrollable malignant fluid in the lungs (one patient each).

A series of pilot Phase II studies to evaluate ONCONASE(R) as a single agent, and ONCONASE(R) and tamoxifen in previously treated patients with unresectable renal cell cancer were conducted. The results of both the Phase II single agent and ONCONASE(R) and tamoxifen have been published. Although the single agent study did not demonstrate evidence of clinical activity, the regimen of ONCONASE(R) and tamoxifen did demonstrate evidence of clinical activity which indicated further evaluation in untreated patients is warranted.

US Phase II telescopic studies to evaluate the regimen of ONCONASE(R) and Gemzar(R), in patients with NSCLC as well as the regimen of ONCONASE(R) and an approved taxane, in patients with advanced breast cancer are planned for 2004.

Research Collaborations

We are pursuing some of these programs independently, while others are being undertaken in collaboration with the NIH and other United States, European and Japanese institutions.

We have established a number of scientific collaborations with the NIH and NCI. The objective of our collaborations with the NIH and NCI is to develop new therapeutic applications for ONCONASE(R) as well as other drug candidates.

The pleiotropic pattern of biological activity of ONCONASE(R) led to research in other areas of cancer biology. Two important areas associated with significant market opportunities are radiation therapy and control of tumor angiogenesis, or new tumor blood vessel formation. Many types of cancers undergo radiation therapy at early stages of the disease; however, success of such treatment is often limited. We believe any agent capable of enhancing tumor radiosensitivity has great market potential. Moreover, since the growth of essentially all types of cancer is dependent on new blood vessel formation, any agent that has anti-angiogenic activity, we believe, is most desirable.

Evaluation Of ONCONASE(R) As A Radiation Enhancer

Published studies have demonstrated that ONCONASE(R) causes an increase in both tumor blood flow and in median tumor oxygen partial pressure causing tumor cells to become less resistant to radiation therapy regardless of the presence or absence of the functional p53 tumor-suppressor gene.

We believe these findings further expand the profile of ONCONASE(R) in vivo activities and its potential clinical utility and market potential. These findings have led to the collaboration with the Molecular Radiation Oncology Sciences Program of the NCI. The Molecular Radiation Therapeutic Branch in collaboration with the Radiation Biology Branch of the NCI is conducting this research.

The University of Pennsylvania Medical Center, Metabolic Magnetic Resonance Research and Computing Center will further evaluate ONCONASE(R) in combination with radiation and cisplatin in human lung adenocarcinoma in a series of animal models as well as look at the effects of ONCONASE(R) in the inhibition of sub-lethal damage repair (SLDR) and potentially lethal damage repair (PLDR) in human lung carcinoma cells.

ONCONASE(R) As a Resistance-Overcoming and Apoptosis-Enhancing Agent

The Fas (CD95) cell surface receptor (and its Fas ligand [FasL]) has been recognized as an important "death" receptor involved in the induction of the "extrinsic" pathway of apoptosis. The apoptotic pathways have been the preferred target for new drug development in cancer, autoimmune, and other therapeutic areas.

The Thoracic Surgery Branch of the NCI confirmed the synergy between ranpirnase and soluble Fas ligand (sFasL) in inducing significant apoptosis in sFasL-resistant Fas+tumor cells. These results provided rationale for using ONCONASE(R) as a potential treatment of FasL-resistant tumors and possibly other disorders such as the autoimmune lympho-proliferative syndrome (ALPS). Further research in this area is ongoing.

Evaluation Of ONCONASE(R) As An Anti-Viral Agent

A collaborative agreement (antiviral screening, non-SARS) with the NIAID has yielded positive results, which have been confirmed with one of our amphinases. Further evaluation of this potential therapeutic is ongoing.

The ribonucleolytic activity was the basis for testing ONCONASE(R) as a potential anti-viral agent against HIV. The NIH has performed an independent in vitro screen of ONCONASE(R) against the HIV virus type 1. The results showed ONCONASE(R) to inhibit replication of HIV by up to 99.9% after a four-day incubation period at concentrations not toxic to uninfected cells. In vitro findings by the NIH revealed that ONCONASE(R) significantly inhibited production of HIV in several persistently infected human cell lines, preferentially breaking down viral RNA and cellular transfer RNA while not affecting normal cellular ribosomal RNA and messenger RNAs.

Moreover, the NIH, Division of AIDS also screened ONCONASE(R) for anti-HIV activity. ONCONASE(R) demonstrated highly significant anti-HIV activity in the monocyte/macrophage system. Ranpirnase may inhibit viral replication at several points during the life cycle of HIV, including its early phases. Ranpirnase is likely to inhibit replication of all different HIV-1 subtypes. These properties of ranpirnase are particularly relevant in view of the extremely high and exponentially increasing rate of mutations of HIV that occur during infection, and which are primarily responsible for the development of resistance to several currently available antiviral drugs. At present, over 50% of clinical isolates of HIV are resistant to both reverse transcriptase and protease inhibitors drugs, and an additional 25%, while being sensitive to protease inhibitors, are resistant to RT inhibitor(s) drugs. German collaborators continue to investigate the anti-viral properties of ONCONASE(R). The ribonucleolytic activity of ONCONASE(R) suggested that it might be active against a variety of RNA viruses, including HIV and hepatitis C. We believe treatments for both viruses have huge market potentials.

Research And Development Pipeline Of Targeted Therapies

Our proprietary drug discovery program forms the basis for the development of recombinant designer RNases for chemical conjugation and gene fusion products with various targeting moieties such as monoclonal antibodies, growth factors, cytokines, etc. We believe these products can be produced in a cost effective and controlled manufacturing environment.

This program also provides for joint design and generation of new products with

outside partners. We, along with any outside partners, may own these new products jointly, or we may grant an exclusive license to the collaborating partner(s).

Ranpirnase Conjugates and Fusion Proteins

The concept of targeting potent toxins as effector molecules to kill cancer or other specifically targeted cells has been extensively evaluated over the last 2 decades. Several immunotoxins containing bacterial and plant toxins or other biotoxins, have been evaluated in human clinical trials. Efficacy has always been limited due to the high incidence of immunogenicity and other intolerable toxicities, including death. Conjugation of ranpirnase to targeting ligands appears to eliminate this safety problem.

We have established a number of scientific collaborations with the NCI. The objective of our collaboration with the NCI is to develop new therapeutic applications for ONCONASE(R). This collaboration has produced RN321, a conjugate of ranpirnase, with a monoclonal antibody that demonstrated activity in treating non-Hodgkin's lymphoma in preclinical studies. The relative benefit in killing targeted tumor cells versus non-targeted healthy cells, or the therapeutic index, is greater than 200,000-fold with this conjugate. These striking "proof-of-concept" results were presented at the 2002 Ribonuclease Meeting in Bath, England. The NCI has undertaken the manufacturing of RN321 (the conjugate) according to GMP regulations in preparation for commencing clinical trials for the treatment of patients with non-Hodgkin's lymphoma with RN321.

Although ranpirnase is active against a variety of human cancers, its activity is not uniform across different tumor types. However, whether the tumor is more or less sensitive to ranpirnase as a single agent, its anti-tumor activity can be greatly augmented by conjugation to different targeting moieties. One of these moieties is the epidermal growth factor, or EGF, which is a ligand for the EGF receptor often hyperexpressed on malignant cells. The genetically engineered ranpirnase conjugates with EGF (rRNP-EGF) exerted significant anti-tumor activity in human squamous cell head and neck and pancreatic carcinomas, and human D54MG glioblastoma. Other constructs target tumor blood vessel formation, which could be potentially used in a broad spectrum of solid tumors. They are in pre-clinical evaluation by our European collaborators.

Novel Amphibian Ribonucleases

In addition to ONCONASE(R), we have isolated several other novel proteins from eggs of the leopard frog. All of the proteins characterized to date are RNases. Information on four new proteins was presented at the 2002 Ribonuclease Meeting. Preclinical testing of the new candidates collectively called amphinases showed them to be similarly active to ranpirnase. Their chemical structure makes them ideal candidates for genetic engineering of designer products.

Collaborations with Pharmaceutical/Drug Delivery Companies

A research and development collaboration with a major US privately held stent and drug delivery company is ongoing. ONCONASE(R) is being evaluated in stents and other delivery platforms to treat cardiovascular disease and cancer via direct site delivery. This collaboration may result in licensing agreement between the companies, however; there is no assurance that such agreement will be reached.

Our research and development collaboration with Wyeth Pharmaceuticals is ongoing to develop a number of designer drugs such as conjugates and fusion proteins for a variety of indications using our proprietary technology. This collaboration may result in a licensing agreement between the companies, however; there is no assurance that such an agreement will be reached.

Raw Materials

The major active ingredient derived from leopard frog eggs is the protein ranpirnase. We have sufficient egg inventory on hand to produce enough ONCONASE(R) to complete the current Phase III clinical trial for malignant mesothelioma and supply ONCONASE(R) for up to two years after commercialization. In addition, we have successfully completed the cloning of the gene of the natural protein ranpirnase; however, the use of this recombinant technology may not be more cost effective than the natural source.

Manufacturing

We have signed an agreement with Scientific Protein Laboratories, a subsidiary of a division of Wyeth Pharmaceuticals, which will perform the intermediary manufacturing process of purifying ranpirnase. Scientific Protein Laboratories sends the intermediate product to a contract filler for the final manufacturing step and vial filling. Other than these arrangements, we do not have specific arrangements for the manufacture of our product. Products manufactured for use in Phase III clinical trials and for commercial sale must be manufactured in compliance with Current Good Manufacturing Practices. Both Scientific Protein Laboratories and the contract filler, to whom the intermediate product is sent, manufacture in accordance with Current Good Manufacturing Practices. For the foreseeable future, we intend to rely on these manufacturers, or substitute manufacturers, if necessary, to manufacture our product. We might not be able to find substitute manufacturers, if necessary. We are dependent upon our contract manufacturers to comply with Current Good Manufacturing Practices and to meet our production requirements. It is possible that our contract manufacturers may not comply with Current Good Manufacturing Practices or deliver sufficient quantities of our products on schedule.

Marketing

We do not plan to market our products at this time. We have entered into a number of Confidential Disclosure Agreements and have been in discussions with several United States and multinational biopharmaceutical companies for the selection of suitable marketing partners for our lead product ONCONASE(R), our proprietary ribonuclease technology pipeline, as well as several patented product candidates.

We intend to enter into development and marketing agreements with third parties. We expect that under such arrangements we would grant exclusive marketing rights to our corporate partners in return for assuming further research and development cost, up-front fees, milestone payments and royalties on sales. Under these agreements, our marketing partner may have the responsibility for a significant portion of product development and regulatory approval. In the event that our marketing partner fails to develop a marketable product or fails to market a product successfully, our business may be adversely affected.

Government Regulation

The manufacturing and marketing of pharmaceutical products in the United States requires the approval of the FDA under the Federal Food, Drug and Cosmetic Act. Similar approvals by comparable regulatory agencies are required in most foreign countries. The FDA has established mandatory procedures and safety standards that apply to the clinical testing, manufacturing and marketing of pharmaceutical products in the United States. Obtaining FDA approval for a new therapeutic may take many years and involve substantial expenditures. State, local and other authorities also regulate pharmaceutical manufacturing

facilities.

As the initial step in the FDA regulatory approval process, preclinical studies are conducted in laboratory dishes and animal models to assess the drug's efficacy and to identify potential safety problems. Moreover manufacturing processes and controls for the product are required. The manufacturing information along with the results of these studies is submitted to the FDA as a part of the IND, which is filed to obtain approval to begin human clinical testing. The human clinical testing program typically involves up to three phases. Data from human trials as well as other regulatory requirements such as chemistry, manufacturing and controls, pharmacology and toxicology sections, are submitted to the FDA in an NDA or Biologics License Application, or BLA. Preparing an NDA or BLA involves considerable data collection, verification and analysis. A similar process in accordance with EMEA regulations is

required to gain marketing approval in Europe. Moreover, a commercial entity must be established and approved by the EMEA in a member state of the EU at least three months prior to filing the MAA.

We have not received United States or other marketing approval for any of our product candidates and may not receive any approvals. We may encounter difficulties or unanticipated costs in our effort to secure necessary governmental approvals, which could delay or preclude us from marketing our products.

With respect to patented products, delays imposed by the governmental approval process may materially reduce the period during which we may have the exclusive right to exploit them.

Patents and Proprietary Technology

We have protected our business by applying for, and obtaining, patents and trademark registrations.. We have also relied on trade secrets and know-how to protect our proprietary technology. We continue to develop our portfolio of patents, trade secrets, and know how. We have obtained, and continue to apply for, patents concerning our RNase-based technology.

In addition, we have filed (and we intend to continue to file) foreign counterparts of certain U.S. patent applications. Generally, we apply for patent protection in the United States, selected European countries, and Japan.

We own the following patents in the United States:

- Patent No. US 6,423,515 B1 issued on July 23, 2002, which covers the methodology for synthesizing gene sequences of ranpirnase and a genetically engineered variant of ranpirnase.
- Patent No. US 6,290,951 B1 issued on September 18, 2001, which covers alteration of the cell cycle in vivo, particularly for inducing apoptosis of tumor cells.
- Patent No. US 6,239,257 B1, issued on May 29, 2001, which covers a family of variants of ONCONASE(R).
- Patent No. US 6,175,003 B1, issued January 16, 2001, which covers the genes of ONCONASE(R)and a variant of ONCONASE(R).
- U.S. Patent No. 5,728,805, issued in 1998, which covers a family of

variants of ONCONASE(R).

- U.S. Patents Nos. 5,529,775 and 5,540,925, issued in 1996, and U.S. Patent No. 5,595,734, issued in 1997, which cover combinations of ONCONASE(R) with certain other pharmaceuticals.

- U.S. Patent No. 5,559,212, issued in 1996, which covers the amino acid sequence of ONCONASE(R).

- U.S. Patent No. 4,888,172, issued in 1989, which covers a pharmaceutical produced from fertilized frog eggs (Rana pipiens) and the methodology for producing it.

We own four European patents, which have been validated in certain European countries. These patents cover ONCONASE(R), a variant of ONCONASE(R), process technology for making ONCONASE(R), and combinations of ONCONASE(R) with certain other chemotherapeutics. We also have patent applications pending in the United States, Europe, and Japan. Additionally, we own one Japanese patent and have an undivided interest in two US patent applications, each relating to a Subject Invention (as that term is defined in Cooperative Research and Development Agreements, or CRADAs, to which we and the NIH are parties.)

The scope of protection afforded by patents for biotechnological inventions can be uncertain, and such uncertainty may apply to our patents as well. The patent applications we have filed, or that we may file in the future, may not result in patents. Our patents may not give us competitive advantages, may be wholly or partially invalidated or held unenforceable, or may be held uninfringed by products that compete with our products. Patents owned by others may adversely affect our ability to do business. Furthermore, others may independently develop products that are similar to our products or that duplicate our products, and may design around the claims of our patents. Although we believe that our patents and patent applications are of substantial value to us, we cannot assure you that such patents and patent applications will be of commercial benefit to us, will adequately protect us from competing products or will not be challenged, declared invalid, or uninfringed upon. We also rely on proprietary know-how and on trade secrets to develop and maintain our competitive position. Others may independently develop or obtain access to such know-how or trade secrets. Although our employees and consultants having access to proprietary information

are required to sign agreements that require them to keep such information confidential, our employees or consultants may breach these agreements or these agreements may be held to be unenforceable.

Competition

Currently, there are no approved systemic treatments for malignant mesothelioma. To our knowledge, no other company is developing a product with the same mechanism of action as ONCONASE(R). There are several companies, universities and research teams which are engaged in research similar, or potentially similar to those performed by us. Eli Lilly is developing a multi-targeted antifolate ALIMTA(R) (pemetrexed) for patients with malignant mesothelioma. Final results have been published in the Journal of Clinical Oncology, July 2003. Some of our competitors have far greater financial resources, larger research staffs and more extensive physical facilities. These competitors may develop products that are more effective than ours and may be more successful than us at producing and marketing their products. We are not aware, however, of any product currently being marketed that has the same mechanism of action as our proposed anti-tumor

agent, ONCONASE(R). Search of scientific literature reveals no published information that would indicate that others are currently employing this method or producing such an anti-tumor agent. Others may develop new treatments that are more effective than ONCONASE(R).

Employees

As of October 24, 2003, we have 13 employees, of whom 10 were engaged in research and development activities and three were engaged in administration and management. We have six employees who hold Ph.D. degrees. All of our employees are covered by confidentiality agreements. We consider relations with our employees to be excellent. None of our employees are covered by a collective bargaining agreement.

Environmental Matters

Our operations are subject to comprehensive regulation with respect to environmental, safety and similar matters by the United States Environmental Protection Agency and similar state and local agencies. Failure to comply with applicable laws, regulations and permits can result in injunctive actions, damages and civil and criminal penalties. If we expand or change our existing operations or propose any new operations, we may need to obtain additional or amend existing permits or authorizations. We spend time, effort and funds in operating our facilities to ensure compliance with environmental and other regulatory requirements.

Such efforts and expenditures are common throughout the biotechnology industry and generally should have no material adverse effect on our financial condition. The principal environmental regulatory requirements and matters known to us requiring or potentially requiring capital expenditures by us do not appear likely, individually or in the aggregate, to have a material adverse effect on our financial condition. We believe that we are in compliance with all current laws and regulations.

Item 2. PROPERTIES

We lease a total of approximately 17,000 square feet in an industrial office building located in Bloomfield, New Jersey. Our lease expired on December 31, 2001 and we have been leasing the property on a month-to-month basis. The monthly rental obligation is $11,333. We believe that the facility is sufficient for our needs in the foreseeable future.

Item 3. LEGAL PROCEEDINGS.

We are presently not involved in any legal proceedings.

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

None.

Part II

Item 5. MARKET FOR COMMON EQUITY AND RELATED STOCKHOLDER MATTERS.

Our common stock is traded on the OTC Bulletin Board, or OTCBB, under the symbol "ACEL". At the close of business April 27, 1999, we were delisted from The Nasdaq SmallCap Market, or Nasdaq, for failing to meet the minimum bid price requirements set forth in the NASD Marketplace Rules. As of October 23, 2003,

there were approximately 1,195 stockholders of record of our common stock.

The following table sets forth the range of high and low sale prices of our common stock for the two fiscal years ended July 31, 2003 and 2002. The prices were obtained from OTCBB and are believed to be representative of inter-dealer quotations, without retail mark-up, mark-down or commission, and may not necessarily represent actual transactions.

	High	Low
Year Ended July 31, 2003:		
First Quarter	$ 0.36	$ 0.18
Second Quarter	1.01	0.19
Third Quarter	0.85	0.39
Fourth Quarter	1.45	0.64
Year Ended July 31, 2002:		
First Quarter	0.96	0.33
Second Quarter	1.01	0.35
Third Quarter	0.77	0.42
Fourth Quarter	0.47	0.27

We have not paid dividends on our common stock since inception and we do not plan to pay dividends in the foreseeable future. Any earnings we may realize will be retained to finance our growth.

The following table provides additional information on the Company's equity based compensation plans as of July 31, 2003:

Plan Category	Number of securities to be issued upon exercise of outstanding options, warrants and rights	Weighted-average exercise price of outstanding options, warrants and rights
	(a)	(b)
Equity compensation plans approved by security holders	2,393,666	$ 1.26
Equity compensation plans not approved by security holders	779,556(1)	$ 1.02

(1) The following 779,556 securities to be issued upon the exercise of outstanding options and warrants that were not approved by the shareholders, relate to options and warrants we issued to third parties in connection with services rendered.

On October 1, 1998, we issued options to purchase 200,000 shares of common stock at an exercise price of $1.00 per share to Sage Partners as payment for services to be rendered. 150,000 of such options were cancelled in November 1999 upon the cancellation of the contract with Sage Partners. The remaining options vested as to 2,500 shares per month from October 31, 1998 through September 30, 1999 and as to 20,000 shares

on October 1, 1999. The options expire five years from the respective vesting date. As of July 31, 2003, options to purchase 50,000 shares remained outstanding. On September 10, 2003, the option to purchase these remaining 50,000 shares was exercised.

In August 2001, we converted $50,000 of our accounts payable owed to DZS Computer Solutions, Inc., into 55,556 shares of common stock. In addition, we issued to DZS Computer Solutions, Inc. 55,556 five-year warrants to purchase 55,556 shares of common stock at an exercise price of $1.50 per share.

In February 2002, we issued 1,500,000 five-year warrants to purchase an aggregate of 1,500,000 shares of common stock in connection with the engagement of a consultant. We received $1,500 for the issuance of the warrants. Of such warrants 500,000 are exercisable immediately, 250,000 at an exercise price of $0.50 and 250,000 at an exercise price of $1.00. The remaining 1,000,000 warrants will become exercisable if the consultant is successful in helping us raise capital. For each $1 million in capital financing raised with the assistance of the consultant, 200,000 warrants will become exercisable up to 1,000,000 warrants in the aggregate. Of these 1,000,000 warrants, 400,000 are exercisable at $1.00 per share and 600,000 are exercisable at $1.50 per share. During the fiscal year 2003, the vesting of the 600,000 warrants was amended to vest immediately and the exercise price was amended from $1.50 to $.50 per share. As of July 31, 2003, warrants to purchase 826,000 shares of common stock had been exercised and warrants to purchase 674,000 shares of common stock remained outstanding.

Recent Sales of Unregistered Securities

In May 2003, we issued a $100,000 8% note payable to an unrelated party, which will become due in November 2004. The unrelated party can convert the note into shares of our common stock at a conversion rate of $0.50 per share. In addition, upon conversion of the note, we will issue warrants to purchase an equal number of shares of common stock at an exercise price of $1.00 per share, expiring five years from the date of issuance. This transaction was exempt from registration under Section 4(2) of the Securities Act of 1933, as amended.

From May 2003 to July 2003, we settled $84,223 of our accounts payable by issuing 149,171 shares of restricted common stock. These transactions were exempt from registration under Section 4(2) of the Securities Act of 1933, as amended.

From May 2003 to July 2003, we issued to private investors an aggregate 730,000 shares of restricted common stock and five-year warrants to purchase an aggregate 730,000 shares of common stock at exercise prices ranging from $1.25 to $1.50 per share. We received an aggregate of $411,000 from such private placements. These transactions were exempt from registration under Section 4(2) of the Securities Act of 1933, as amended.

From May 2003 to July 2003, we issued an aggregate of 724,000 shares of common stock upon the exercise of warrants and options by unrelated parties, which resulted in aggregate gross proceeds of $358,000 to us. These transactions were exempt from registration under Section 4(2) of the Securities Act of 1933, as amended.

The net proceeds from the above mentioned transactions will be used for general corporate purposes.

Item 6. SELECTED FINANCIAL DATA.

Set forth below is the selected financial data for our company for the five fiscal years ended July 31, 2003.

	Year Ended July 31,				
	2003	2002	2001	2000	19
Interest Income	$ 9,877	$ 4,838	$ 13,121	$ 51,144	$ 16
Other Income	30,000	--	--	--	
Net Loss (1)	(2,411,532)	(2,591,162)	(2,294,936)	(1,722,298)	(3,15
Net Loss Per Basic and Diluted Share	(.10)	(.12)	(.12)	(.10)	
Dividends	None	None	None	None	
Total Assets	495,322	228,871	201,609	488,099	1,72
Long-term Debt	242,516	315,929	23,663	30,251	
Total Equity (Deficiency)	(2,491,681)	$(1,885,437)	(740,378)	(131,860)	75

(1) Included in the net loss of $2,411,532, $2,591,162 and $2,294,936 for fiscal years ended July 31, 2003, 2002 and 2001, respectively, are tax benefits of $231,357, $353,732 and $451,395, respectively, related to the sale of certain state tax operating loss carryforwards.

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

Overview

Since our inception, we have devoted the majority of our resources to the research and development of ONCONASE(R) and related drug candidates. We have focused our resources towards the completion of the clinical program for unresectable malignant mesothelioma.

Since ONCONASE(R) has Fast Track Designation for the treatment of malignant mesothelioma patients, we continue to have meetings and discussions with the FDA to establish mutually agreed upon parameters for the New Drug Application, or NDA to obtain marketing approval for ONCONASE(R), assuming the Phase III clinical trial yields favorable results.

We received an Orphan Medicinal Product Designation for ONCONASE(R) from the European Agency for the Evaluation of Medicinal Products, or the EMEA. We continue to fulfill the EMEA requirements regarding the Marketing Authorization Application, or MAA registration requirements for ONCONASE(R) for the treatment of malignant mesothelioma.

In the ongoing Phase III trial, an interim analysis based on the occurrence of 105 deaths is planned. Based upon the results of these analyses, we may be able to file an NDA and an MAA within six months after the completion of the analyses. Marketing approval for ONCONASE(R) as a treatment for malignant mesothelioma may not be granted by the FDA or EMEA.

We fund the research and development of our products from cash receipts resulting from the private sales of our securities, sale of our tax benefits and from certain debt financings. Presently, our cash balance is sufficient to fund our operations in the near term, however, we intend to raise additional capital through the sale of our securities and strategic alliance(s). However, there are no assurances that such funds will be obtained.

Results of Operations

Fiscal Years Ended July 31, 2003, 2002 and 2001

Revenues

We are a development stage company as defined in the Financial Accounting Standards Board's Statement of Financial Accounting Standards No. 7. We are devoting substantially all our present efforts to establishing a new business and developing new drug products. Our planned principal operations of marketing and/or licensing of new drugs have not commenced and, accordingly, we have not derived any significant revenue from these operations. We focus most of our productive and financial resources on the development of ONCONASE(R). We did not have any sales in fiscal 2003, 2002 and 2001. Investment income for fiscal 2003 was $10,000 compared to $5,000 for fiscal 2002, an increase of $5,000. The increase was due to higher balances of cash and cash equivalents. Investment income for fiscal 2002 was $5,000 compared to $13,000 for fiscal 2001, a decrease of $8,000. This decrease was due to lower balances of cash and cash equivalents.

Research and Development

Research and development expense for fiscal 2003 was $1,700,000 compared to $2,033,000 for fiscal 2002, a decrease of $333,000, or 16.4%. This decrease was primarily due to decreases in personnel costs, regulatory consulting costs and a reduction of non-cash expenses relating to stock options issued for consulting services. These decreases were partially offset by an increase in costs relating to patent and trademark applications for ONCONASE(R).

Research and development expense for fiscal 2002 was $2,033,000 compared to $1,901,000 for fiscal 2001, an increase of $132,000, or 7%. This increase was primarily due to an increase in costs in support of ongoing clinical

trial for ONCONASE(R) resulting from the expansion of our Phase III clinical trial for malignant mesothelioma in Europe. This increase was partially offset by a decrease in expenses related to outside consultants, reduction of non-cash expenses relating to stock options issued for consulting services and a decrease in costs relating to patent and trademark applications for ONCONASE(R).

General and Administrative

General and administrative expense for fiscal 2003 was $624,000 compared to $798,000 for fiscal 2002, a decrease of $174,000, or 21.8%. This decrease was primarily due to decreases in costs related to public relations activities, insurance expenses, personnel costs and reduction in non-cash expense relating to stock options issued for consulting services.

General and administrative expense for fiscal 2002 was $798,000 compared to $706,000 for fiscal 2001, an increase of $92,000, or 13%. This increase was primarily due to an increase in costs related to public relations activities, an increase in legal costs associated with business development activities and an

increase in insurance expenses offset by a decrease in non-cash expense relating to stock options issued for consulting services.

Interest

Interest expense for fiscal 2003 was $358,000 compared to $119,000 in fiscal 2002, an increase of $239,000. The increase was primarily due to the interest expense on the beneficial conversion feature of the notes payable issued to unrelated parties, the related warrants and the increase in total borrowing levels. The interest expense was based on the value of the warrants using the Black-Scholes options-pricing model, amortized on a straight-line basis over the term of the notes.

Interest expense for fiscal 2002 was $119,000 compared to $153,000 in fiscal 2001, a decrease of $34,000. The decrease was primarily due to the interest expense on convertible notes and related warrants issued during the fiscal year ended 2001. The interest expense was based on the value of the warrants using the Black-Scholes options-pricing model, amortized on a straight-line basis over the term of the notes.

Income Taxes

New Jersey has enacted legislation permitting certain corporations located in New Jersey to sell state tax loss carryforwards and state research and development credits, or tax benefits. For the state fiscal year 2003 (July 1, 2002 to June 30, 2003), we have $1,373,000 total available tax benefits of which $273,000 was allocated to be sold between July 1, 2002 and June 30, 2003. In December 2002, we received $231,000 from the sale of an aggregate of $273,000 tax benefits which was recognized as a tax benefit for fiscal 2003. In December 2001, we received $354,000 from the sale of an aggregate of $426,000 tax benefits which was recognized as a tax benefit for our fiscal 2002. We will attempt to sell the remaining balance of our tax benefits in the amount of approximately $1,100,000 between July 1, 2003 and June 30, 2004, subject to all existing laws of the State of New Jersey. However, we may not be able to find a buyer for our tax benefits or that such funds may not be available in a timely manner.

Net Loss

We have incurred net losses during each year since our inception. The net loss for fiscal 2003 was $2,411,000 as compared to $2,591,000 in fiscal 2002 and 2,295,000 in fiscal 2001. The cumulative loss from the date of inception, August 24, 1981, to July 31, 2003 amounted to $63,974,000. Such losses are attributable to the fact that we are still in the development stage and accordingly have not derived sufficient revenues from operations to offset the development stage expenses.

Liquidity and Capital Resources

We have reported net losses of approximately $2,411,000, $2,591,000, and $2,295,000 for the fiscal years ended July 31, 2003, 2002 and 2001, respectively. The loss from date of inception, August 24, 1981, to July 31, 2003 amounts to $63,974,000. Also, we have a working capital deficit and limited liquid resources.

We have financed our operations since inception primarily through equity and debt financing, research product sales and interest income. During the fiscal year 2003, we had a net increase in cash and cash equivalents of $244,000. This

increase primarily resulted from net cash provided by financing activities in the amount of $1,798,000, primarily due to proceeds from short and long-term borrowings, from the private placement of common stock and warrants and proceeds from the exercise of warrants, offset by net cash used in operating activities of $1,554,000. Total cash resources as of July 31, 2003 were $330,000 compared to $86,000 at July 31, 2002.

Our current liabilities as of July 31, 2003 were $2,744,000 compared to $1,798,000 at July 31, 2002, an increase of $946,000. The increase was primarily due to the short-term maturity of notes payable, accrued payroll and payroll taxes offset by the reduction of a loan payable to a related party. As of July 31, 2003, we had a total of $644,023 in unpaid payroll and $240,784 in unpaid payroll taxes. As of September 2003, all unpaid payroll taxes have been fully satisfied. In addition, $115,000 in unpaid payroll was paid and since July 31, 2003, we have been current in our payroll and payroll taxes. As of July 31, 2003 our current liabilities exceeded our current assets and we had a working capital deficit of $2,404,000.

The following transactions occurred after July 31, 2003:

In August 2003, the Company issued an aggregate of 120,000 shares of common stock to private investors resulting in aggregate gross proceeds of $60,000 to the Company. In addition, the private investors were granted five-year warrants to purchase 120,000 shares of common stock at an exercise of price of $1.25 per share.

In September 2003, the Company issued 1,704,546 shares of common stock to an institutional investor resulting in gross proceeds of $1,500,000 to the Company. In addition, the private investors were granted five-year warrants to purchase 852,273 shares of common stock at an exercise of price of $1.50 per share.

From August 2003 through October 14, 2003, the Company issued to unrelated parties, an aggregate of 1,165,773 shares of common stock upon the exercise of warrants and stock options at per share exercise prices ranging from $0.43 to $1.00. The Company realized aggregate gross proceeds of approximately $861,225.

In September 2003, the terms of our notes payable were amended such that (i) they are convertible into shares of Series A Preferred Stock rather than common stock, and (ii) the warrants to be issued upon the due date of the notes are warrants to purchase shares of Series A Preferred Stock rather than common stock. In the event the stockholders approve an increase in the number of shares of common stock authorized, the terms of the notes will revert to the original terms to the extent the notes have not been converted.

In September 2003, our Board of Directors designated 200,000 of the 1,000,000 shares of preferred stock as Series A Preferred Stock. 105,666 shares of our Series A Preferred Stock has been reserved for issuance upon the conversion of certain of our outstanding notes.

New Jersey has enacted legislation permitting certain corporations located in New Jersey to sell state tax loss carryforwards and state research and development credits, or tax benefits. For the state fiscal year 2003 (July 1, 2002 to June 30, 2003), we have $1,373,000 total available tax benefits of which $273,000 was allocated to be sold between July 1, 2002 and June 30, 2003. In December 2002, we received $231,000 from the sale of an aggregate of $273,000 tax benefits which was recognized as a tax benefit for our fiscal 2003. In December 2001 and 2000, we received $354,000 and $451,000 from the sale of an aggregate of $426,000 and $602,000 tax benefits which was recognized as tax benefits for our fiscal years 2002 and 2001, respectively. We will attempt to sell the remaining

balance of our tax benefits in the amount of approximately $1,100,000 between July 1, 2003 and June 30, 2004, subject to all existing laws of the State of New Jersey. However, we may not be able to find a buyer for our tax benefits or that such funds may not be available in a timely manner.

Our continued operations will depend on our ability to raise additional funds through various potential sources such as equity and debt financing, collaborative agreements, strategic alliances, sale of tax benefits, revenues from the commercial sale of ONCONASE(R), our primary anti-cancer product being developed, licensing of our proprietary RNase technology and our ability to realize the full potential of our technology and our drug candidates via out-licensing agreements with other companies. Such additional funds may not become available as we need them or be available on acceptable terms. Through July 31, 2003, a significant portion of our financing has been through private placements of common stock and warrants, the issuance of common stock for stock options and warrants exercised and for services rendered, debt financing and financing provided by our Chief Executive Officer. Additionally, we have raised capital through the sale of our tax benefits. Until our operations generate significant revenues, we will continue to fund operations from cash on hand and through the sources of capital previously described. During the fiscal year ended July 31, 2003, the Company received gross proceeds of approximately $2,241,000 from long-term and short-term borrowings from unrelated parties, from the private placement of common stock and warrants, proceeds from the exercise of warrants and options and from the sale of our tax benefits. After taking into account these net proceeds and the anticipated proceeds from the sale of the balance of our tax benefits, we believe that our cash and cash equivalents will be sufficient to meet our anticipated cash needs through October 2004. We continue our fund raising efforts and anticipates securing additional financing in the first calendar quarter of 2004. The reports of our independent auditors on our financial statements includes an explanatory paragraph which states that our recurring losses, working capital deficit and limited liquid resources raise substantial doubt about our ability to continue as a going concern. Our financial statements do not include any adjustments that might result from the outcome of this uncertainty.

We will continue to incur costs in conjunction with our U.S. and foreign registrations for marketing approval of ONCONASE(R). We are currently in discussions with several potential strategic alliance partners including major international biopharmaceutical companies to further the development and marketing of ONCONASE(R) and other related products in our pipeline, as well as our proprietary technology. However, we cannot be certain that any such alliances will materialize.

Our common stock was delisted from The Nasdaq SmallCap Market effective at the close of business April 27, 1999 for failing to meet the minimum bid price requirements set forth in the NASD Marketplace Rules. Since April 28, 1999, our common stock has traded on the OTC Bulletin Board under the symbol "ACEL". Delisting of our common stock from Nasdaq could have a material adverse effect on our ability to raise additional capital, our stockholders' liquidity and the price of our common stock.

The market price of our common stock is volatile, and the price of the stock could be dramatically affected one way or another depending on numerous factors. The market price of our common stock could also be materially affected by the marketing approval or lack of approval of ONCONASE(R).

Critical Accounting Policies

In December 2001, the SEC requested that all registrants discuss their most

"critical accounting policies" in management's discussion and analysis of financial condition and results of operations. The SEC indicated that a "critical accounting policy" is one which is both important to the portrayal of the company's financial condition and results and requires management's most difficult, subjective or complex judgments, often as a result of the need to make estimates about the effect of matters that are inherently uncertain. We believe based on our current business that there are no critical accounting policies. Our accounting policies are described in Note 1 to the financial statements.

Contractual Obligations

Our major outstanding contractual obligations relate to our equipment operating lease and convertible notes.

Below is a table that presents our contractual obligations and commercial commitments as of July 31, 2003:

	Payments Due by Fiscal Year			
	Total	2004	2005	2006 and Thereafter
Research and development commitments	$ -0-	$ -0-	$ -0-	$ -0-
Operating lease	30,600	17,500	13,100	-0-
Total contractual cash obligations	$ 30,600	$ 17,500	$ 13,100	$ -0-

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

Not Applicable.

Item 8. FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA.

The response to this Item is submitted as a separate section of this report commencing on Page F-1.

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

As described in the current report on Form 8-K filed by the Company on December 12, 2002 which is incorporated by reference into this Item 9, on December 6, 2002 KPMG LLP resigned as our independent accountants and was replaced by J.H. Cohn LLP as our independent accountants for fiscal 2003. The engagement of J.H. Cohn was approved by our Audit Committee. The reports of KPMG on the financial statements for the past two fiscal years contained no adverse opinion or disclaimer of opinion and were not qualified or modified as to uncertainty, audit scope or accounting principle except that the report on our financial statements for the fiscal years ended July 31, 2002 and 2001 contained a separate paragraph stating that "the Company has suffered recurring losses from operations, has a working capital deficit and has limited liquid resources which raise substantial doubt about its ability to continue as a going concern.

Management's plans in regard to these matters are also described in Note 2. The financial statements do not include any adjustments that might result from the outcome of this uncertainty." During our two most recent fiscal years through December 6, 2002, there were no disagreements between us and KPMG on any matter of accounting principles or practices, financial statement disclosures or auditing scope or procedures, which disagreements if not resolved to the satisfaction of KPMG would have caused them to make reference thereto in their report on the financial statements for such years.

On December 1, 1993, certain shareholders of Armus Harrison & Co., or AHC, terminated their association with AHC, or the AHC termination, and AHC ceased performing accounting and auditing services, except for limited accounting services to be performed on our behalf. In June 1996, AHC dissolved and ceased all operations. The report of J.H. Cohn LLP with respect to our financial statements from inception to July 31, 2003 is based on the report of KPMG LLP from August 1, 1992 to July 31, 2002 and of AHC for the period from inception to July 31, 1992, although AHC has not consented to the use of such report herein and will not be available to perform any subsequent review procedures with respect to such report. Accordingly, investors will be barred from asserting claims against AHC under Section 11 of the Securities Act on the basis of the use of such report in any registration statement into which such report is incorporated by reference. In addition, in the event any persons seek to assert a claim against AHC for false or misleading financial statements and disclosures in documents previously filed by us, such claim will be adversely affected and possibly barred. Furthermore, as a result of the lack of a consent from AHC to the use of its audit report herein, or to its incorporation by reference into a registration statement, our officers and directors will be unable to rely on the authority of AHC as experts in auditing and accounting in the event any claim is brought against such persons under Section 11 of the Securities Act based on alleged false and misleading Financial Statements and disclosures attributable to AHC. The discussion regarding certain effects of the AHC termination is not meant and should not be construed in any way as legal advice to any party and any potential purchaser should consult with his, her or its own counsel with respect to the effect of the AHC termination on a potential investment in our common stock or otherwise.

Item 9A. CONTROLS AND PROCEDURES.

(a) Evaluation of disclosure controls and procedures.

Under the supervision and with the participation of our management, including our Chief Executive Officer and acting Chief Financial Officer, we evaluated the effectiveness of the design and operation of our disclosure controls and procedures as of July 31, 2003, the evaluation date. Based upon the evaluation, the Chief Executive Officer and acting Chief Financial Officer concluded that, as of the evaluation date, our disclosure controls and procedures are effective in timely alerting them to the material information relating to us required to be included in our periodic SEC filings.

(b) Changes in internal controls.

There were no significant changes made in our internal controls during the period covered by this report or, to our knowledge, in other factors that could significantly affect these controls subsequent to the date of their evaluation.

Part III

The information required by Item 10 - Directors and Executive Officers of the

Registrant; Item 11 - Executive Compensation; Item 12 - Security Ownership of Certain Beneficial Owners and Management; Item 13 - Certain Relationships and Related Transactions and Item 14 - Principal Accounting Fees and Services is incorporated into Part III of this Annual Report on Form 10-K by reference to the Proxy Statement for our Annual Meeting of Stockholders scheduled to be held on January 14, 2004.

Part IV

Item 15. EXHIBITS, FINANCIAL STATEMENT SCHEDULES, AND REPORTS ON FORM 8-K.

(a)(1) and (2) The response to these portions of Item 15 is submitted as a separate section of this report commencing on page F-1.

(a)(3) Exhibits (numbered in accordance with Item 601 of Regulation S-K).

Exhibit No.	Item Title	Exh Inco
3.1	Certificate of Incorporation	
3.2	By-Laws	
3.3	Amendment to Certificate of Incorporation	
3.4	Amendment to Certificate of Incorporation	
3.5	Certificate of Designation for Series A Preferred Stock	
4.1	Form of Convertible Debenture	
10.1	1993 Stock Option Plan and Form of Option Agreement	
10.2	Debt Conversion Agreement dated March 30, 1994 with Kuslima Shogen	
10.3	Accrued Salary Conversion Agreement dated March 30, 1994 with Kuslima Shogen	
10.4	Accrued Salary Conversion Agreement dated March 30, 1994 with Stanislaw Mikulski	
10.5	Option Agreement dated March 30, 1994 with Kuslima Shogen	
10.6	Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Kuslima Shogen	
10.7	Form of Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Kuslima Shogen	
10.8	Form of Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Stanislaw Mikulski	

Exhibit No.	Item Title	Exh Inco
10.9	1997 Stock Option Plan	
10.10	Form of Subscription Agreement and Warrant Agreement used in Private Placement completed on February 20, 1998	
10.11	Form of Warrant Agreement issued to the Placement Agent in connection with the Private Placement completed on February 20, 1998	
10.12	Form of Subscription Agreement and Warrant Agreement used in Private	

	Placements completed in February 2000
10.13	Form of Subscription Agreement and Warrant Agreement used in the August and September 2000 Private Placements
10.14	Form of Subscription Agreement and Warrant Agreement used in the April 2001 Private Placements
10.15	Form of Convertible Note entered into in April 2001
10.16	Form of Subscription Agreement and Warrant Agreement used in the July 2001 Private Placements
10.17	Form of Subscription Agreement and Warrant Agreement used in the August and October 2001 private placement
10.18	Form of Subscription Agreement and Warrant Agreement used in the September 2001, November 2001 and January 2002 private placements
10.19	Warrant issued in the February 2002 private placement
10.20	Form of Subscription Agreement and Warrant Agreement used in the March 2002, April 2002 and May 2002 private placements
10.21	Form of Subscription Agreement and Warrant Agreement used in the June 2002 and October 2002 private placements
10.22	Form of Note Payable and Warrant Certificate entered into April, June, July, September, November and December 2002
10.23	Form of Note Payable and Warrant Certificate entered into November 2001, January, March and May 2003
10.24	Form of Subscription Agreement and Warrant Agreement used in the February 2003 and April through August 2003 private placements
10.25	Securities Purchase Agreement and Warrant Agreement used in September 2003 private placement
10.26	Registration Rights Agreement used in September 2003 private placement
10.27	Form of Amended Notes Payable which amends the November 2001, April 2002, June 2002, July 2002, September 2002, November 2002 December 2002, January 2003, March 2003 and May 2003 notes payable
21.1	Subsidiaries of Registrant
23.1	Consent of J.H. Cohn LLP
23.2	Consent of KPMG LLP
31.1	Certification of Chief Executive Officer and Chief Financial Officer pursuant to Rule 13a-14(a) (Section 302 Certification), as adapted pursuant to Section 302 of Sarbanes-Oxley Act of 2002
32.1	Certification Chief Executive Officer and Chief Financial Officer pursuant to 18 U.S.C. Section 1350, as adopted pursuant to Section 906 of the Sarbanes-Oxley Act of 2002
99.1	Factors to Consider in Connection with Forward-Looking Statements

* Previously filed as exhibit to the Company's Registration Statement on Form S-18 (File No. 2-79975-NY) and incorporated herein by reference thereto.

** Previously filed as exhibits to the Company's Annual Report on Form 10-K for the year ended July 31, 1993 and incorporated herein by reference thereto.

*** Previously filed as exhibits to the Company's Quarterly Report on Form 10-QSB for the quarter ended April 30, 1994 and incorporated herein by reference thereto.

**** Previously filed as exhibits to the Company's Registration Statement Form SB-2 (File No. 33-76950) and incorporated herein by reference thereto.

\+ Previously filed as exhibits to the Company's Quarterly Report on

Form 10-Q for the quarter ended January 31, 1998 and incorporated herein by reference thereto.

++ Previously filed as exhibits to the Company's Annual Report on Form 10-K for the year ended July 31, 2000 and incorporated herein by reference thereto.

+++ Previously filed as exhibits to the Company's Quarterly Report on Form 10-Q for the quarter ended October 31, 2000 and incorporated herein by reference thereto.

^ Previously filed as exhibits to the Company's Registration Statement on Form S-1 (File No. 333-38136) and incorporated herein by reference thereto.

^^ Previously filed as exhibits to the Company's Registration Statement on Form S-1 (File No. 333-89166) and incorporated herein by reference thereto.

Previously filed as exhibits to the Company's Annual Report on Form 10-KSB for the year ended July 31, 1995 and incorporated herein by reference thereto.

Previously filed as exhibits to the Company's Quarterly Report on Form 10-QSB for the quarter ended April 30, 1997 and incorporated herein by reference thereto.

Filed herewith.

(b) Reports on Form 8-K.

None.

Signature

Pursuant to the requirements of Section 13 or 15(d) of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

ALFACELL CORPORATION

Dated: October 29, 2003

By: /s/ KUSLIMA SHOGEN
Kuslima Shogen, Chief Executive Officer, Acting Chief Financial Officer and Chairman of the Board

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

Dated: October 29, 2003

/s/ KUSLIMA SHOGEN
Kuslima Shogen, Chief Executive Officer, Acting Chief Financial Officer (Principal Executive Officer, Principal Accounting Officer) and Chairman of the Board

Dated: October 29, 2003 /s/ STEPHEN K. CARTER
Stephen K. Carter, M.D., Director

Dated: October 29, 2003 /s/ DONALD R. CONKLIN
Donald R. Conklin, Director

Dated: October 29, 2003 /s/ MARTIN F. STADLER
Martin F. Stadler, Director

Dated: October 29, 2003 /s/ PAUL M. WEISS
Paul M. Weiss, Ph.D., MBA, Director

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REPORT OF INDEPENDENT PUBLIC ACCOUNTANTS

To the Board of Directors
Alfacell Corporation

We have audited the accompanying balance sheet of ALFACELL CORPORATION (A Development Stage Company) as of July 31, 2003, and the related statements of

operations, stockholders' deficiency and cash flows for the year then ended and for the period from August 24, 1981 (date of inception) to July 31, 2003. These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these financial statements based on our audits. The financial statements of Alfacell Corporation for the period from August 24, 1981 to July 31, 2002 were audited by other auditors whose reports dated November 4, 2002 and December 9, 1992, except for Note 18 which is as of July 19, 2003 and Note 3 which is as of October 28, 1993, expressed unqualified opinions on those statements with explanatory paragraphs relating to the Company's ability to continue as a going concern.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, based on our audits and, for the effect on the period from August 24, 1981 to July 31, 2003 of the amounts for the period from August 24, 1981 to July 31, 2002, on the reports of other auditors, the financial statements referred to above present fairly, in all material respects, the financial position of Alfacell Corporation as of July 31, 2003, and its results of operations and cash flows for the year then ended and for the period from August 24, 1981 to July 31, 2003, in conformity with accounting principles generally accepted in the United States of America.

The financial statements referred to above have been prepared assuming that the Company will continue as a going concern. As discussed in Note 2 to the financial statements, the Company has suffered substantial losses from inception and is a development stage company. Such matters raise substantial doubt about the ability of the Company to continue as a going concern. Management's plans in regard to these matters are also described in Note 2. The financial statements referred to above do not include any adjustments that might result from the outcome of this uncertainty.

J.H. Cohn LLP

Roseland, New Jersey
September 26, 2003, except for Note 18,
which is as of October 14, 2003

Independent Auditors' Report

The Stockholders and Board of Directors
Alfacell Corporation:

We have audited the accompanying balance sheet of Alfacell Corporation (a development stage company) as of July 31, 2002, and the related statements of operations, stockholders' equity (deficiency), and cash flows for each of the years in the two-year period ended July 31, 2002 and the period from August 24, 1981 (date of inception) to July 31, 2002 (not presented herein). These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these financial statements based on our audits. The financial statements of Alfacell Corporation for the period from

August 24, 1981 to July 31, 1992 were audited by other auditors whose report dated December 9, 1992, except as to note 18 which is July 19, 1993 and note 3 which is October 28, 1993, expressed an unqualified opinion on those statements with an explanatory paragraph regarding the Company's ability to continue as a going concern.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, based on our audits and, for the effect on the period from August 24, 1981 to July 31, 2002 of the amounts for the period from August 24, 1981 to July 31, 1992, on the report of other auditors, the financial statements referred to above present fairly, in all material respects, the financial position of Alfacell Corporation as of July 31, 2002, and the results of its operations and its cash flows for each of the years in the two-year period ended July 31, 2002 and the period from August 24, 1981 to July 31, 2002 (not presented herein) in conformity with accounting principles generally accepted in the United States of America.

The accompanying financial statements have been prepared assuming that the Company will continue as a going concern. As discussed in Note 2 to the financial statements, the Company has suffered recurring losses from operations, has a working capital deficit and has limited liquid resources which raise substantial doubt about its ability to continue as a going concern. Management's plans in regard to these matters are also described in Note 2. The financial statements do not include any adjustments that might result from the outcome of this uncertainty.

/s/ KPMG LLP

Short Hills, New Jersey
November 4, 2002

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On December 1, 1993, certain shareholders of Armus Harrison & Co. ("AHC") terminated their association with AHC (the "AHC termination"), and AHC ceased performing accounting and auditing services, except for limited accounting services to be performed on behalf of the Company. In June 1996, AHC dissolved and ceased all operations. The report of AHC with respect to the financial statements of the Company from inception to July 31, 1992 is included herein, although AHC has not consented to the use of such report herein and will not be available to perform any subsequent review procedures with respect to such report. Accordingly, investors will be barred from asserting claims against AHC under Section 11 of the Securities Act of 1933, as amended (the "Securities Act") on the basis of the use of such report in any registration statement of the Company into which such report is incorporated by reference. In addition, in the event any persons seek to assert a claim against AHC for false or misleading financial statements and disclosures in documents previously filed by the Company, such claim will be adversely affected and possibly barred. Furthermore, as a result of the lack of a consent from AHC to the use of its audit report herein, or, to its incorporation by reference into a registration statement, the

officers and directors of the Company will be unable to rely on the authority of AHC as experts in auditing and accounting in the event any claim is brought against such persons under Section 11 of the Securities Act based on alleged false and misleading financial statements and disclosures attributable to AHC. The discussion regarding certain effects of the AHC termination is not meant and should not be construed in any way as legal advice to any party and any potential purchaser should consult with his, her or its own counsel with respect to the effect of the AHC termination on a potential investment in the Common Stock of the Company or otherwise.

REPORT OF INDEPENDENT AUDITORS

Board of Directors
Alfacell Corporation
Bloomfield, New Jersey

We have audited the balance sheets of Alfacell Corporation (a Development Stage Company) as of July 31, 1992 and 1991, as restated, and the related statements of operations, stockholders' deficiency, and cash flows for the three years ended July 31, 1992, as restated, and for the period from inception August 24, 1981 to July 31, 1992, as restated. In connection with our audit of the 1992 and 1991 financial statements, we have also audited the 1992, 1991 and 1990 financial statement schedules as listed in the accompanying index. These financial statements and financial statement schedules are the responsibility of the Company's management. Our responsibility is to express an opinion on these financial statements based on our audit.

We conducted our audit in accordance with generally accepted auditing standards. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audit provides a reasonable basis for our opinion.

In our opinion the financial statements referred to above present fairly in all material respects, the financial position of Alfacell Corporation as of July 31, 1992 and 1991, as restated, and for the three years ended July 31, 1992, as restated, and for the period from inception August 24, 1981 to July 31, 1992, as restated, and the results of operations and cash flows for the years then ended in conformity with generally accepted accounting principles.

The accompanying financial statements have been prepared on a going concern basis which contemplates the realization of assets and the satisfaction of liability in the normal course of business. As shown in the statement of operations, the Company has incurred substantial losses in each year since its inception. In addition, the Company is a development stage company and its principal operation for production of income has not commenced. The Company's working capital has been reduced considerably by operating losses, and has a deficit net worth. These factors, among others, as discussed in Note 2 to the Notes of Financial Statements, indicates the uncertainties about the Company's ability to continue as a going concern. The financial statements do not include any adjustments relating to the recoverability and classification of recorded asset amounts and the amount of classification of liabilities that might be necessary should the Company be unable to continue its existence.

/s/ Armus, Harrison & Co.

Armus, Harrison & Co.

Mountainside, New Jersey
December 9, 1992
Except as to Note 18 which
is July 19, 1993 and Note 3
which is October 28, 1993

ALFACELL CORPORATION
(A Development Stage Company)

Balance Sheets

July 31, 2003 and 2002

	2003	2002
ASSETS		
Current assets:		
Cash and cash equivalents	$ 330,137	$ 85
Other assets	10,103	45
Total current assets	340,240	131
Property and equipment, net of accumulated depreciation and amortization of $1,136,183 in 2003 and $1,120,371 in 2002	12,795	28
Loan receivable, related party	142,287	68
Total assets	$ 495,322	$ 228
LIABILITIES AND STOCKHOLDERS' DEFICIENCY		
Current liabilities:		
Current portion of long-term debt, net of debt discount of $187,121 at July 31, 2003	$ 637,080	$ 8
Loan payable, related party	--	139
Accounts payable	699,429	796
Accrued expenses	1,407,978	854
Total current liabilities	2,744,487	1,798
Long-term debt, less current portion, net of debt discount of $163,687 at July 31, 2003	242,516	315
Total liabilities	2,987,003	2,114
Commitments and contingencies		

Stockholders' deficiency:		
Preferred stock, $.001 par value. Authorized and unissued, 1,000,000 shares at July 31, 2003 and 2002	--	
Common stock $.001 par value. Authorized 40,000,000 shares; issued and outstanding 25,026,129 shares and 22,760,921 shares at July 31, 2003 and 2002, respectively	25,026	22
Capital in excess of par value	61,457,502	59,654
Deficit accumulated during development stage	(63,974,209)	(61,562
Total stockholders' deficiency	(2,491,681)	(1,885
Total liabilities and stockholders' deficiency	$ 495,322	$ 228

See accompanying notes to financial statements.

ALFACELL CORPORATION
(A Development Stage Company)

Statements of Operations

Years ended July 31, 2003, 2002
and 2001, and the Period from August 24, 1981
(Date of Inception) to July 31, 2003

	August 24, 1981 (date of inception) to July 31, 2003	2003	2002
Revenues:			
Sales	$ 553,489	$ --	$ --
Investment income	1,387,000	9,877	4,838
Other income	90,103	30,000	--
	2,030,592	39,877	4,838
Cost and expenses:			
Cost of sales	336,495	--	--
Research and development	41,601,935	1,699,962	2,032,938
General and administrative	22,287,852	624,406	798,053
Interest:			
Related parties	1,147,547	--	4,687
Others	2,423,310	358,398	114,054
	67,797,139	2,682,766	2,949,732
Loss before state tax benefit	(65,766,547)	(2,642,889)	(2,944,894)
State tax benefit	1,792,338	231,357	353,732

Net loss	$(63,974,209)	$ (2,411,532)	$ (2,591,162)
Loss per basic and diluted common share		$ (0.10)	$ (0.12)
Weighted average number of shares outstanding		23,166,000	21,045,000

See accompanying notes to financial statements.

ALFACELL CORPORATION
(A Development Stage Company)

Statement of Stockholders' Equity (Deficiency)

Period from August 24, 1981
(Date of Inception) to July 31, 2003

	Common Stock		
	Number of Shares	Amount	Capital In Excess of par Value
Issuance of shares to officers and stockholders for equipment, research and development, and expense reimbursement	712,500	$ 713	$ 212,987
Issuance of shares for organizational legal service	50,000	50	4,950
Sale of shares for cash, net	82,143	82	108,418
Adjustment for 3 for 2 stock split declared September 8, 1982	422,321	422	(422)
Net loss	--	--	--
Balance at July 31, 1982	1,266,964	1,267	325,933
Issuance of shares for equipment	15,000	15	13,985
Sale of shares to private investors	44,196	44	41,206
Sale of shares in public offering, net	660,000	660	1,307,786
Issuance of shares under stock grant program	20,000	20	109,980
Exercise of warrants, net	1,165	1	3,494
Net loss	--	--	--
Balance at July 31, 1983	2,007,325	2,007	1,802,384
Exercise of warrants, net	287,566	287	933,696
Issuance of shares under stock grant program	19,750	20	101,199
Issuance of shares under stock bonus plan for directors and consultants	130,250	131	385,786
Net loss	--	--	--
Balance at July 31, 1984	2,444,891	2,445	3,223,065

Issuance of shares under stock grant program	48,332	48	478,057
Issuance of shares under stock bonus plan for directors and consultants	99,163	99	879,379
Shares canceled	(42,500)	(42)	(105,783)
Exercise of warrants, net	334,957	335	1,971,012
Net loss	--	--	--
Balance at July 31, 1985	2,884,843	2,885	6,445,730
Issuance of shares under stock grant program	11,250	12	107,020
Issuance of shares under stock bonus plan for directors and consultants	15,394	15	215,385
Exercise of warrants, net	21,565	21	80,977
Net loss	--	--	--
Balance at July 31, 1986 (carried forward)	2,933,052	2,933	6,849,112

	Deficit Accumulated During Development Stage	Subscription Receivable	Deferred compensati restricte stock
Issuance of shares to officers and stockholders for equipment, research and development, and expense reimbursement	$ --	$ --	$ -
Issuance of shares for organizational legal service	--	--	-
Sale of shares for cash, net	--	--	-
Adjustment for 3 for 2 stock split declared September 8, 1982	--	--	-
Net loss	(121,486)	--	-
Balance at July 31, 1982	(121,486)	--	-
Issuance of shares for equipment	--	--	-
Sale of shares to private investors	--	--	-
Sale of shares in public offering, net	--	--	-
Issuance of shares under stock grant program	--	--	-
Exercise of warrants, net	--	--	-
Net loss	(558,694)	--	-
Balance at July 31, 1983	(680,180)	--	-
Exercise of warrants, net	--	--	-
Issuance of shares under stock grant program	--	--	-
Issuance of shares under stock bonus plan for directors and consultants	--	--	-
Net loss	(1,421,083)	--	-
Balance at July 31, 1984	(2,101,263)	--	-
Issuance of shares under stock grant program	--	--	-
Issuance of shares under stock bonus plan for directors and consultants	--	--	-
Shares canceled	--	--	-

Exercise of warrants, net	--	--	-
Net loss	(2,958,846)	--	-
Balance at July 31, 1985	(5,060,109)	--	-
Issuance of shares under stock grant program	--	--	-
Issuance of shares under stock bonus plan for directors and consultants	--	--	-
Exercise of warrants, net	--	--	-