ALFACELL CORP
Form 10-K
November 14, 2002

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, 2002	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. [X]

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 average of the high and low sale prices as reported by the OTC Bulletin Board on November 8, 2002 was $4,139,518. As of November 8, 2002 there were 22,872,958 shares of common stock, par value $.001 per share, outstanding.

The Index to Exhibits appears on page 29.

Documents Incorporated by Reference

None

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The following trademark appear in this Annual Report: ONCONASE(R) is the registered trademark of Alfacell Corporation, exclusively for the anti-cancer indications.

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 is a biopharmaceutical company, 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 Alfacell's 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 properties. Alfacell is recognized as a leader in the development of RNase based therapeutics and as such, has both co-sponsored and been a key participant in the International Ribonuclease Meetings held every three years. ONCONASE(R), our trademark name for ranpirnase, our flagship product, is undergoing the last stage of clinical testing which is called "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 active conponent 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 February 2001, we received an Orphan Medicinal Product Designation for ONCONASE(R) from The European Agency for the Evaluation of Medicinal Products (EMEA). Alfacell has also applied for Fast Track Designation for the indication

of malignant mesothelioma. We will receive a decision from the FDA no later than December 23, 2002. These designations to ONCONASE(R) may in the future serve to expedite its regulatory review, assuming the clinical trials have yielded 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. Both companies may own these new products, or Alfacell may grant an exclusive license to the collaborating partner(s).

We have established a number of scientific collaborations with the National Cancer Institute, 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 NCI investigators at the 6th International Ribonuclease Meeting in Bath, England, June 2002, and the manuscript will be submitted for publication. Preclinical studies are ongoing at NCI in preparation for commencing clinical trials for the treatment of patients with non-Hodgkin's lymphoma with this new conjugate.

We have also discovered another series of proteins that may have therapeutic uses. These proteins appear to be involved in the regulation of both early embryonic and malignant cell growth. In addition to ranpirnase, Alfacell has isolated several other proteins from eggs of the leopard frog (Rana pipiens). All of the proteins characterized to date are RNases. Information on four of these proteins was presented at the 6th International Ribonuclease Meeting in Bath, England, June 2002. 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 Wyeth Pharmaceuticals to co-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 negotiations with a number of companies for US or non-US marketing rights for ONCONASE(R).

Alfacell is engaged in the research, development and clinical trials of its 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.

Alfacell Corporation, a Delaware corporation, was incorporated in 1981. The common stock is traded on the OTC Bulletin Board under the symbol "ACEL".

RESEARCH AND DEVELOPMENT PROGRAMS

Research and Development Programs

Research and development expenses for the fiscal years ended July 31, 2002,

2001, and 2000 were $2,033,000, $1,901,000, and $1,880,000, 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 vs. normal, 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 more effective. To make cancer therapy more effective and less toxic, Alfacell is developing ONCONASE(R) as a therapeutic and as an effector moiety, killer molecule for targeted therapies. We believe that selective degradation of intracellular proteins is central to the process of programmed cell death, known as apoptosis.

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 occurs too slowly. Conversely, HIV is associated with increased apoptosis or programmed death of cells occurs too rapidly. This process of programmed cell death is genetically regulated. Alfacell has 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 (programmed) 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 caspase proteolytic enzyme cascades which, in turn, induce disintegration of the cellular components and finally execute 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 cell death. Ranpirnase-induced apoptosis did not require the functional p53 tumor suppressor gene product, or the Fas ligand/Fas/Fas-associating protein with death domain (FADD) and caspase 8, but appeared to involve an activation of the mitochondrial pro-caspase 9, and also caspase 3 and 7. However, this activation of the mitochondrial pro-caspase was associated with atypically little release of cytochrome c from mitochondria, and a lack of the cytosol-to-mitochondria translocation of the pro-apoptotic bax protein.

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 cancer cells to chemotherapy and radiation therapy.

It remains unknown whether or not ONCONASE(R) targets and binds preferentially 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.

Overview of Preclinical and Clinical Studies of ONCONASE(R)

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 Institutes of Health or NIH, including NCI, as well as a number of well-renowned academic institutions, in the US, 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 be active in the NCI Cancer Screen.

In vitro and in vivo studies showed both cytostatic and cytotoxic 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) + 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 for the following combinations:

- 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 and ONCONASE(R) + Taxol 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 vitro results, ONCONASE(R) in combination with the following anti-cancer agents has been evaluated by Alfacell and in collaboration with 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 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 Programs

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 in clinical trials since 1991 in the US and 2000 in Europe. 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 Europe 2001, 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.

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 patient enrollment for the first part of the clinical trial has been completed. The second part is currently ongoing and is being conducted in the US, Germany and Italy.

We have had a series of meetings with the Food and Drug Administration or FDA to establish mutually agreed upon parameters for the New Drug Application or NDA to obtain marketing approval for ONCONASE(R).

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 an 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. A manuscript has been prepared for publication.

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 applied for Fast Track Designation for the malignant mesothelioma indication. We will receive a decision from the FDA no later than December 23, 2002. 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 EMEA. We are continuing discussions with EMEA regarding the Marketing Authorization Application or MAA registration requirements for ONCONASE(R) for the treatment of malignant mesothelioma.

In the ongoing 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. Marketing approval for ONCONASE(R) as a treatment for malignant mesothelioma may not be granted by the FDA or EMEA.

We had initiated a Phase III program in patients with advanced pancreatic cancer in 1995 after meeting with FDA, based on the Phase II 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) + 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) + tamoxifen regimen in untreated patients as well as patients who had failed GEMZAR(R). 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 indication 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.

Forty-one patients, 39% reported evidence of clinical activity: 4 partial responses, 2 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 program.

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

Phase II telescopic studies to evaluate the regimen of ONCONASE(R) + gemcitabine, in patients with NSCLC as well as the regimen of ONCONASE(R) + Taxatere(R), in patients with advanced breast cancer are planned for 2003.

In summary, results of clinical trials performed to date suggest that ONCONASE(R) can be safely administered to patients with cancer and that such treatment may overcome multiple drug resistance and interfere with tumor growth.

Research Collaborations

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

We have established a number of scientific collaborations with NCI. The objective of our collaboration with NCI is to develop new therapeutic applications for ONCONASE(R). This collaboration has produced RN321, a conjugate, or chemical construct, 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, therapeutic index, is greater than 200,000-fold with this conjugate. These striking "proof-of-concept" results were

presented at the 6th International Ribonuclease Meeting in Bath, England, in June 2002 and resulted in approval of an NCI sponsored clinical trial and the associated manuscript will be submitted for publication.

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, 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.

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

The ribonucleolytic activity was the basis for testing ONCONASE(R) as a potential anti-viral agent against the human immunodeficiency virus, or HIV. NIH have 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 inhibitors 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 (AIDS) 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. Alfacell with any outside partners may both own these new products, or Alfacell 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.

RN321 is comprised of ranpirnase conjugated to an anti-CD22 monoclonal antibody for the treatment of non-Hodgkin's Lymphoma. Alfacell, in collaboration with the NCI, is developing this product. The relative benefit in killing targeted tumor cells versus non-targeted healthy cells is greater than 200,000-fold with this conjugate. These striking "proof-of-concept" results were presented at the 6th International Ribonuclease Meeting in Bath, England, in June 2002 and resulted in approval of an NCI sponsored clinical trial and the associated manuscript will be submitted for publication.

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, Alfacell has 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 6th International Ribonuclease Meeting., in Bath , England. Preclinical testing of the new candidates tested shows them to be similarly active to ranpirnase. Their chemical structure makes them ideal candidates for genetic engineering of designer products. We are currently in negotiations with potential pharmaceutical partners for the development of these new compounds.

Collaborations with Pharmaceutical Companies

We have entered into research and development collaboration with Wyeth Pharmaceuticals to co-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. Although we currently acquire our natural source material from a single supplier, we believe that it is abundantly available from other sources. 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, 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 U.S. 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 US. 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

Since our inception, it has been our policy to protect our proprietary technology and know-how by filing for and obtaining patents and trademarks which we consider important to the development of our business. We rely on trade

secrets and know-how and continue to develop our intellectual property rights. We have obtained, and continue to file, patents concerning our RNase based technology, drug candidates including but not limited to genes, conjugates and fusion proteins, novel composition of matter, and methods of manufacture and use.

In addition, foreign counterparts of certain applications have been filed or will be filed at the appropriate time. In the United States patents filed prior to June 8, 1995 will expire 17 years after the date of allowance or, in other cases 20 years from the date of application. Generally, it is our strategy to 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 in July 23, 2002, which covers the methodology for synthesizing gene sequences of ranpirnase and the genetically engineered variant.

- Patent No. US 6,290,951 B1 issued in 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 patents ,each relating to a Subject Invention, as that term is defined in Cooperative Research and Development Agreement, or CRADA 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 declared uninfringed. 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, research teams which are engage 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. Preliminary Phase III results namely response rate and median survival, were presented at ASCO 2002, however many patients were censored at the time of the analysis. Final results are not yet published. ALIMTA-related deaths have been reported. Monitoring folate levels and requiring folic acid + vitamin B12 supplementation reduced the risk of drug-related deaths.

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 November 8, 2002, we have 14 employees, of whom 11 were engaged in research and development activities and three were engaged in administration and management. We have six employees who hold Ph.D. or M.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. Alfacell has retained the services of independent contractors and companies with a proven track record for the significant clinical, regulatory and manufacturing activities required for product development.

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 are currently negotiating a new lease agreement under similar terms with the landlord. 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 November 8, 2002, there were approximately 1,224 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, 2002 and 2001. 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, 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

Year Ended July 31, 2001:		
First Quarter	1.56	0.75
Second Quarter	1.38	0.53
Third Quarter	2.19	0.72
Fourth Quarter	1.59	0.81

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.

Recent Sales of Unregistered Securities

In June 2002, we sold an aggregate of 285,714 shares of common stock to private investors at a price of $0.35 per share resulting in gross proceeds of $100,000. In addition, the private investors were granted five-year warrants to purchase an aggregate of 285,714 shares of common stock at per share exercise price of $1.00. These transactions were consummated as a private sale pursuant to Section 4(2) of the Securities Act of 1933, as amended.

In April, June, July and September 2002, we issued warrants to purchase an aggregate 350,000 shares of common stock in connection with notes payable to unrelated parties in an aggregate amount of $350,000. The notes were due

thirty days from the date of issuance bearing interest at 8% per annum. The warrants to purchase have an exercise price of $0.60 per share. The total non-cash interest expense recorded for these warrants was $40,690, based upon the fair value of such warrants on the date of issuance as estimated by the Black-Scholes options-pricing model. The notes were either extended for eighteen months or the lenders can convert the notes at a conversion price of $0.40 per share plus a five-year warrant for each share of Alfacell common stock issued upon conversion at an exercise price of $1.00 per share. These transactions were consummated as a private sale pursuant to Section 4(2) of the Securities Act of 1933, as amended.

The following table provides additional information on the Company's equity based compensation plans as of July 31, 2002:

Plan Category	Number of securities to be issued upon exercise of outstanding options, warrants and rights (a)	Weighted-average exercise price of outstanding options, warrants and rights (b)	Number remaini future equity (exclud reflect
Equity compensation plans approved by security holders	2,297,784	$ 1.79	

Equity compensation plans not approved by security holders	1,426,556	$ 1.22

Item 6. SELECTED FINANCIAL DATA.

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

	Year Ended July 31,			
	2002	2001	2000	1999
Interest Income	$ 4,838	$ 13,121	$ 51,144	$ 168,372
Net Loss (1)	$(2,591,161)	$(2,294,936)	$(1,722,298)	$(3,156,636)
Net Loss Per Basic and Diluted Share	$ (.12)	$ (.12)	$ (.10)	$ (.18)
Dividends	None	None	None	None

	As of July 31,			
	2002	2001	2000	1999
Total Assets	$ 228,871	$ 201,609	$ 488,099	$ 1,728,648
Long-term Debt	$ 315,929	$ 23,663	$ 30,251	None
Total Equity (Deficiency)	$(1,885,437)	$ (740,378)	$ (131,860)	$ 757,200

(1) Included in the net loss of $2,591,161, $2,294,936 and $1,722,298 for fiscal years ended July 31, 2002, 2001 and 2000, respectively, are tax benefits of $353,732, $451,395 and $755,854, 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. After we obtained the results of our preliminary analysis of the Phase III clinical trial for advanced pancreatic cancer, we closed the pancreatic cancer trials and redirected our resources towards the completion of the clinical program for unresectable malignant mesothelioma.

We have had a series of meetings and communications with the FDA and EMEA to establish mutually agreed upon parameters for the NDA and MAA filings for the malignant mesothelioma indication. In the ongoing Phase III trial in this indication, 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 are also exploring out-licensing and various strategic alternatives for our business, research and development operations with other companies.

In October 2002, we entered into a research collaboration with Wyeth Pharmaceuticals to co-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 us, however, there is no assurance that such agreement will be reached.

We are currently funding the research and development of our products from cash receipts resulting from the private sales of our securities and from certain debt financings. The termination of the Phase III clinical trials for advanced pancreatic cancer had a significant and detrimental impact on the price of our common stock and our ability to raise additional capital for future operations. We may not have, or may not be able to obtain, the financial resources required to pay for all the associated costs of the malignant mesothelioma program to file in the Unites States and/or foreign registration for the marketing approval of ONCONASE(R) for this indication.

Results of Operations

Fiscal Years Ended July 31, 2002, 2001 and 2000

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 2002, 2001 and 2000. 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. Investment income for fiscal 2001 was $13,000 compared to $51,000 for fiscal 2000, a decrease of $38,000. This decrease was due to lower balances of cash and cash equivalents.

Research and Development

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 trials for ONCONASE(R) resulting from the expansion of our Phase III clinical trials 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 application for ONCONASE(R).

Research and development expense for fiscal 2001 was $1,901,000 compared to $1,880,000 for fiscal 2000, an increase of $21,000, or 1%. This increase was primarily due to an increase in costs in support of ongoing clinical trial and increase in costs related to ONCONASE(R) clinical supplies, both primarily due to the expansion of our

Phase III clinical trial for malignant mesothelioma in Europe. These increases were offset by a decrease in expenses related to the NDA filing for ONCONASE(R) with the FDA.

General and Administrative

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, increase in legal costs associated with business development activities and increase in insurance expenses offset by a decrease in non-cash expense relating to stock options issued for consulting services.

General and administrative expense for fiscal 2001 was $706,000 compared to $645,000 for fiscal 2000, an increase of $61,000, or 9%. This increase was primarily due to a 58% increase in costs related to public relations activities, a 30% increase in non-cash expense relating to stock options issued for consulting services, a 12% increase in personnel costs and an 87% increase in costs associated with business development activities.

Interest

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 life of the notes.

Interest expense for fiscal 2001 was $153,000 compared to $5,000 in fiscal 2000, an increase of $148,000. The increase was primarily due to the interest expense on convertible notes and related warrants issued in April 2001 to related and unrelated parties. 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 life 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 2002 (July 1, 2001 to June 30, 2002), we have $1,535,000 total available tax benefits of which $426,000 was allocated to be sold between July 1, 2001 and June 30, 2002. 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. In December 2000, we received $451,000 from the sale of an aggregate of $602,000 tax benefits which was recognized as a tax benefit for our fiscal 2001. We will attempt to sell the remaining balance of our tax benefits in the amount of approximately $1,109,000 between July 1, 2002 and June 30, 2003, 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 2002 was $2,591,000 as compared to $2,295,000 in fiscal 2001 and $1,722,000 in fiscal 2000. The cumulative loss from the date of inception, August 24, 1981, to July 31, 2002 amounted to $61,563,000. 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 financed our operations since inception primarily through equity and debt financing, research product sales and interest income. During the fiscal year 2002, we had a net increase in cash and cash equivalents of $41,000. This increase primarily resulted from net cash provided by financing activities in the amount of $1,572,000, primarily from the private placement of common stock and warrants, proceeds from long-term borrowings, loans

from related parties and proceeds from the exercise of warrants, offset by net cash used in operating activities of $1,531,000. Total cash resources as of July 31, 2002 were $86,000 compared to $45,000 at July 31, 2001.

Our current liabilities as of July 31, 2002 were $1,798,000 compared to $918,000 at July 31, 2001, an increase of $880,000. The increase was primarily due to an increase in expenses related to the expansion of our Phase III clinical trial for malignant mesothelioma in Europe and increasing business development, investor and public relations activities. As of July 31, 2002 our current liabilities exceeded our current assets and we had a working capital deficit of $1,667,000.

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 2002 (July 1, 2001 to June 30, 2002), we have $1,535,000 total available tax benefits of which $426,000 was allocated to be sold between July 1, 2001 and June 30, 2002. 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. In December 2000 and 1999, we received $451,000 and $756,000 from the sale of an aggregate of $602,000 and $1,008,000 tax benefits which was recognized as a tax benefit for our fiscal years 2001 and 2000, respectively. We will attempt to sell the remaining balance of our tax benefits in the amount of approximately $1,109,000 between July 1, 2002 and June 30, 2003, 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), 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. To date, 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. From August through November 6, 2002, we received gross proceeds of approximately $277,000 from long-term and short-term borrowings from unrelated parties and from the private placement of common stock and warrants. 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 January 2003.

Management is continuing its fund raising efforts and anticipates securing required financing in the first calendar quarter of 2003. The report 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.

Below is a table that presents our contractual obligations and commercial commitments as of July 31, 2002:

	Payments Due by Fiscal Year			
	Total	2003	2004	20
Research and development commitments	$ - 0 -	$ - 0 -	$ - 0 -	$ -
Operating leases	58,200	19,800	19.200	19,
Total contractual cash obligations	$ 58,200	$ 19,800	$ 19.200	$ 19,

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.

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 KPMG LLP with respect to our financial statements from inception to July 31, 2002 is based on the report 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.

Part III

Item 10. DIRECTORS AND EXECUTIVE OFFICERS OF THE REGISTRANT.

Name	Age	Director Since	Position with the Company
Kuslima Shogen	57	1981	Chairman of the Board, Chief Exec Acting Chief Financial Officer
Stanislaw M. Mikulski, M.D.	58	1986	Executive Vice President, Medical Director
Stephen K. Carter, M.D.(1)	64	1997	Director and Chairman of the Scie Board

Donald R. Conklin (1)(2)	66	1997	Director
Martin F. Stadler (1)(2)	60	1997	Director

(1) Member of Compensation Committee
(2) Member of Audit Committee

Business Experience of Directors and Executive Officers

Kuslima Shogen has served as our Chief Executive Officer since September 1986, as Chairman of the Board since August 1996, as a Director since our inception and as Acting Chief Financial Officer since June 23, 1999. She also served as our Chief Financial Officer from September 1986 through July 1994 and as our President from September 1986 through July 1996. Ms. Shogen formed the company in 1981 to pursue research that she had initiated while a biology student in the University Honors Program at Fairleigh Dickenson University. Prior to our founding, from 1976 to 1981 she was founder and president of a biomedical research consortium specializing in Good Laboratory Practices and animal toxicology. During that time, she also served as a consultant for the Lever Brothers Research Group. Ms. Shogen has received numerous awards for achievements in biology, including the Sigma Xi first prize from the Scientific Research Society of North America in 1974 and first prize for the most outstanding research paper in biology at the Eastern College Science Conferences competitions in 1972, 1973, and 1974. She earned a B.S. degree in 1974 and an M.S. degree in 1976 in biology from Fairleigh Dickenson University, or FDU, and also completed graduate studies in 1978 in embryology. She is a Phi Beta Kappa graduate. In April 1998, Ms. Shogen received the Pinnacle Award from FDU, the highest honor the University bestows on its graduates.

Stanislaw M. Mikulski, M.D., F.A.C.P. has served as our Executive Vice President and Medical Director since 1987 and as a Director since 1986. Prior to his affiliation with us, Dr. Mikulski was Special Assistant to the Chief of the Investigational Drug Branch of the National Cancer Institute, and the Coordinator for Immunotherapy Trials in Cancer for the Division of Cancer Treatment. Prior to joining us, he maintained a private practice in medical oncology for over eight years. He is a diplomate of the American Board of Internal Medicine and Medical Oncology as well as a Fellow of the American College of Physicians and a member of the American Society of Clinical Oncology, The American Association for Cancer Research and the American Association for the Advancement of Science. Dr. Mikulski is currently a clinical assistant Professor of Medicine at the University of Medicine and Dentistry of New Jersey. He received his M.D. in 1967 from the Medical School of Warsaw, Poland and subsequently performed post-doctoral studies in human tumor immunology at the University of California in Los Angeles.

Stephen K. Carter, M.D. joined the Board of Directors in May 1997 and serves as Chairman of our Scientific Advisory Board. In addition to his positions with us, Dr. Carter also serves as a senior clinical consultant to Sugen, Inc. From 1995 through 1997, he served as Senior Vice President of Research and Development for Boehringer-Ingelheim Pharmaceuticals. Before this, Dr. Carter spent over 13 years with Bristol-Myers Squibb, an international leader in the development of innovative anti-cancer and anti-viral therapies. He held a variety of senior executive research and development positions while at Bristol-Myers, including serving for five years as Senior Vice President of worldwide clinical research and development of its Pharmaceutical Research Institute. From 1976 to 1982, he

established and directed the Northern California Cancer Program. Prior to this, he held a number of positions during a nine-year tenure at the National Cancer Institute, including the position of Deputy Director at the National Institutes of Health. He has also been a member of the faculties of the medical schools of Stanford University, the University of California at San Francisco and New York University. Dr. Carter has published extensively on the development of anti-cancer drugs, was the co-founding editor of journals devoted to cancer therapeutics or immunology, and has served on the editorial boards of a number of additional journals dedicated to cancer treatment. He is a member of the American Society of Clinical Oncology, the American Association for Cancer Research, and the Society of Surgical Oncology, as well as several other medical societies. Dr. Carter earned his B.A. from Columbia University and his M.D. from New York Medical College. He currently serves on the Board of Directors of Allos Therapeutics.

Donald R. Conklin joined the Board of Directors in May 1997. Prior to his retirement in May 1997, Mr. Conklin was a senior executive with Schering-Plough, a major worldwide pharmaceutical firm. During his more than 35 years with Schering-Plough, he held a variety of key management positions within the firm. From 1986 to 1994, he served as President of Schering-Plough Pharmaceuticals and Executive Vice-President of Schering-Plough Corporation. In this position, he was responsible for worldwide pharmaceutical operations, including the launch of INTRON A(R) (interferon alfa-2b). Prior to this, Mr. Conklin had served as President of Schering USA and had held a variety of executive marketing positions in the United States, Europe, and Latin America. Immediately preceding his retirement, he was Chairman of Schering-Plough Health Care Products and an Executive Vice President of Schering-Plough Corporation. Mr. Conklin received his B.A. with highest honors from Williams College and his M.B.A. degree from the Rutgers University School of Business. He currently serves on the Board of Directors of Vertex Pharmaceuticals, Inc. and Ventiv Health, Inc.

Martin F. Stadler joined the Board of Directors in November 1997. At the end of 1996, Mr. Stadler retired from Hoffmann La-Roche, Inc. after 32 years of pharmaceutical, chemical and diagnostic experience. Mr. Stadler served as senior vice president and chief financial officer, and was a member of the Hoffmann La-Roche, Inc. Board of Directors from 1985 through 1996. His responsibilities included finance, information technology, human resources, quality control and technical services. Prior to 1985, Mr. Stadler served as vice-president of strategic planning and business development. Mr. Stadler received his B.S. degree from Rutgers University and his M.B.A. from Fairleigh Dickenson University. In April 1999, he received the Pinnacle Award from FDU, the highest honor the University bestows on its graduates. Mr. Stadler is a member of the Finance Council of the American Management Association.

In March 1998 the SEC approved the settlement previously disclosed in our November 1997 Proxy Statement of allegations by the SEC of violations of Sections 13 and 16 of the Securities Exchange Act of 1934, as amended (the Exchange Act) by Kuslima Shogen, Chairman and Chief Executive Officer and Stanislaw Mikulski, Executive Vice President. Ms. Shogen and Dr. Mikulski agreed to the entry of a cease and desist order and the payment of monetary penalties totaling $40,000 (payable by us under our indemnity agreements with these individuals) without admitting or denying any of the SEC's allegations concerning certain allegedly late filings required to be made by them pursuant to Sections 13 and 16 of the Exchange Act with respect to changes in beneficial ownership of our securities. With the exception of one late filing by Ms. Shogen in 1996, each of the allegedly unreported transactions occurred during the years 1983 to 1994. The alleged reporting violations relate solely to the filings of required forms. There was no allegation by the SEC of any fraudulent or willful

misconduct. No action was brought against us.

Section 16(a) Beneficial Ownership Reporting Compliance

Ownership of and transactions in our stock by our executive officers and directors and owners of 10% or more of our outstanding common stock are required to be reported to the Securities and Exchange Commission pursuant to Section 16(a) of the Securities Exchange Act of 1934, as amended. During the fiscal year ended July 31, 2002, all reports required to be filed pursuant to Section 16(a) of the Exchange Act were filed in a timely manner.

Item 11. EXECUTIVE COMPENSATION.

Directors' Compensation

Directors receive no cash compensation in consideration for their serving on the Board of Directors.

In November 1993 and January 1994, the Board of Directors and the stockholders, respectively, approved our 1993 Stock Option Plan, or the 1993 Plan, which, among other things, provides for automatic grants of options under a formula to non-employee directors or independent directors on an annual basis.

The formula provides that (i) on each December 31st each independent director receives automatically an option to purchase 15,000 shares of our common stock, or the regular grant; and (ii) on the date of each independent director's

initial election to the Board of Directors, the newly elected independent director automatically receives an option to purchase the independent director's pro rata share of the regular grant which equals the product of 1,250 multiplied by the number of whole months remaining in the calendar year, or the pro rata grant. Each option granted pursuant to a regular grant and a pro rata grant vests and becomes exercisable on December 30th following the date of grant. An option will not become exercisable as to any shares unless the independent director has served continuously on the Board during the year preceding the date on which such options are scheduled to vest and become exercisable, or from the date the independent director joined the Board until the date on which the options are scheduled to vest and become exercisable. However, if an independent director does not fulfill such continuous service requirement due to the independent director's death or disability all options held by the independent director nonetheless vest and become exercisable as described herein. An option granted pursuant to the formula remains exercisable for a period of five years after the date the option first becomes exercisable. The per share exercise price of an option granted under the formula is equal to the average of the high and low trade prices of our common stock for the twenty (20) trading days preceding the date of grant.

During the fiscal year ended July 31, 2002, the following independent directors listed below were granted options under our 1997 Stock Option Plan, or the 1997 Plan, pursuant to the same formula under the 1993 Plan as set forth above. The exercise prices of the options are equal to the formula set forth above.

Name	Number of Options	Exercise Price	Expiration

Stephen K. Carter	15,000	$ 0.66	12/30/07
Donald R. Conklin	15,000	$ 0.66	12/30/07
Martin F. Stadler	15,000	$ 0.66	12/30/07

Compensation Committee Interlocks and Insider Participation

During the fiscal year ended July 31, 2002, the members of our Board of Directors who served on the Compensation Committee were Stephen K. Carter, Donald R. Conklin and Martin F. Stadler, all of whom are non-employee directors and have never been an officer of Alfacell.

In April 2001, our board of directors approved the issuance of 50,000 stock options under the 1997 Plan to Martin Stadler, which vested on the date of grant. The exercise price of the stock options was $0.90 per share which was based on the average of the high and low trade prices of our common stock for the ten trading days preceding the date of grant.

In April 2001, we issued convertible notes to Kuslima Shogen, our Chief Executive Officer and a director, two of our directors, Donald Conklin and Martin Stadler, and unrelated parties in the aggregate amount of $366,993. Messrs. Conklin and Stadler are members of our Compensation Committee. The notes were due within ninety days unless the lenders elect to exercise an option to convert their note into common stock at the conversion price of $0.90 per share. The related parties named above have elected to convert their notes into an aggregate 330,000 shares of common stock. In addition, upon conversion, they received three-year warrants to purchase an aggregate 330,000 shares of common stock at an exercise price of $2.50 per share that will expire on July 7, 2004. In October 2001, the Board of Directors approved a change in the exercise price of the 330,000 warrants issued to related parties from $2.50 per share to $1.50 per share and changed the expiration date to July 7, 2006, to conform with the private placements to unrelated parties. The notes issued to unrelated parties with an aggregate balance of $69,993 were renewed for one hundred twenty (120) days for the same conversion price of $0.90 per share. In addition, upon conversion, they will receive five-year warrants to purchase an aggregate 77,770 shares of common stock at an exercise price of $1.50 per share. In October 2001, the remaining noteholders elected to convert an aggregate $64,993 notes payable into an aggregate 72,214 shares of common stock. In addition, they received five-year warrants to purchase an aggregate 72,214 shares of common stock at an exercise price of $1.50 per share.

Summary Compensation Table

The following table provides a summary of cash and non-cash compensation for each of the last three fiscal years ended July 31, 2002, 2001 and 2000 earned by our Chief Executive Officer and Executive Vice President or our executive officers during the last fiscal year.

	Annual Compensation			Long Term Compensation

Name and Principal Position	Year	Salary(5) ($)	Bonus ($)	Other Annual Compensation ($)(1)	Securities Underlying Options/SARs(#)
Kuslima Shogen	2002	$150,000	- 0 -	- 0 -	115,000
Chief Executive	2001	150,000	- 0 -	- 0 -	115,000
Officer, Chairman of the Board of Directors and Acting Chief Financial Officer	2000	150,000	- 0 -	- 0 -	215,000(3)
Stanislaw M. Mikulski					
Executive Vice	2002	$130,000	- 0 -	- 0 -	55,000
President and	2001	130,000	- 0 -	- 0 -	50,000
Medical Director	2000	130,000	- 0 -	- 0 -	130,000(4)

(1) Excludes perquisites and other personal benefits which in the aggregate do not exceed 10% of our executive officers' total annual salary and bonus.

(2) Consists of our contributions to a 401(k) plan.

(3) Of these options, 100,000 expired in December 2001.

(4) Of these options, 75,000 expired in December 2001.

(5) Fiscal year 2002 includes $13,000 and $33,900 of unpaid salary for K. Shogen and S. Mikulski, respectively.

Option Grants in Last Fiscal Year

The following table contains information concerning the grant of stock options to our executive officers during the fiscal year ended July 31, 2002:

	Individual Grants				Potential Re Assumed Annu Price Apprec T
Name	Number of Securities Underlying Options Granted (#)	% of Total Options Granted to Employees in Fiscal Year	Exercise or Base Price ($/Share)(1)	Expiration Date	0%($) 5
Kuslima Shogen	115,000(3)	33.92%	$.49	(4)	-- $
Stanislaw M. Mikulski	50,000(3)	14.75%	$.49	(4)	-- $

(1) The exercise price of these options was based on the average of the high and low trade prices of our common stock for the twenty trading days preceding the date of grant.

(2) The amounts set forth in the three columns represent hypothetical gains that might be achieved by the optionees if the respective options are exercised at the end of their terms. These gains are based on assumed rates of stock price appreciation of 0%, 5% and 10%. The 0% appreciation column is included because the exercise prices of the options equal the market price of the underlying common stock on the date the options were granted, and thus the options will have no value unless our stock price increases above the exercise prices.

(3) These options vested and became exercisable as to 20% of the shares on October 4, 2001 and 20% of the shares each year thereafter.

(4) These options will expire five years after the vesting date.

Option Exercises and Fiscal Year-End Values

The following table sets forth the information with respect to our executive officers concerning the exercise of options during the fiscal year ended July 31, 2002 and unexercised options held as of July 31, 2002.

			Number of Securities Underlying Unexercised Options at Fiscal Year-End (#)		Value o In-The- at Fisca
Name	Shares Acquired on Exercise (#)	Value Realized ($) (1)	Exercisable	Unexercisable	Exercisable
Kuslima Shogen	None	None	512,685	307,000	$-0-
Stanislaw M. Mikulski	None	None	221,281	170,000	$-0-

(1) Based upon the fair market value of the purchased shares on the option exercise date less the exercise price paid for the shares.

(2) The fair market value of the common stock at the fiscal year end was based on the average of the high and low trade prices ($0.34) for the common stock obtained from the OTC Bulletin Board on the last trading day of the fiscal year July 31, 2002.

Item 12. SECURITY OWNERSHIP OF CERTAIN BENEFICIAL OWNERS AND MANAGEMENT.

The following table sets forth certain information concerning stock ownership of each person who is the beneficial owner of five percent or more of our outstanding common stock, each of the current directors, each of our executive officers and all directors and executive officers as a group as of September 30,

2002. Except as otherwise noted, each person has sole voting and investment power with respect to the shares shown as beneficially owned.

Directors, Officers or 5% Stockholders (1)	Number of Shares (2)	Perce Stock
Kuslima Shogen	2,073,305 (4)	
Stanislaw M. Mikulski	624,531 (5)	
Stephen K. Carter	183,750 (6)	

Donald R. Conklin	454,250 (7)	
Martin F. Stadler	466,250 (8)	
All executive officers and directors as a group (five persons)	3,802,086 (9)	

* Less than one percent.

(1) The address of all officers and directors listed above is in the care of the company.

(2) All shares listed are common stock. Except as discussed below, none of these shares are subject to rights to acquire beneficial ownership, as specified in Rule 13d-3(d)(1) under the Exchange Act, and the beneficial owner has sole voting and investment power, subject to community property laws where applicable.

(3) The percentage of stock outstanding for each stockholder is calculated by dividing (i) the number of shares of Common Stock deemed to be beneficially held by such stockholder as of September 30, 2002 by (ii) the sum of (A) the number of shares of common stock outstanding as of September 30, 2002 plus (B) the number of shares issuable upon exercise of options or warrants held by such stockholder which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002.

(4) Includes 604,685 shares underlying options which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002 and 110,000 shares underlying warrants which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002.

(5) Includes 263,281 shares underlying options which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002.

(6) Includes 183,750 shares underlying options which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after

September 30, 2002.

(7) Includes 113,750 shares underlying options which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002 and 110,000 shares underlying warrants which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002.

(8) Includes 131,250 shares underlying options which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002 and 110,000 shares underlying warrants which were exercisable as of September 30, 2002 or which will become exercisable within 60 days after September 30, 2002.

(9) Includes all shares owned beneficially by the directors and the executive officers named in the table.

Item 13. CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS.

On July 23, 1991, the Board of Directors authorized us to pay Kuslima Shogen an amount equal to 15% of any gross royalties which may be paid to us from any license(s) with respect to our principal product, ONCONASE(R), or any other products derived from amphibian source extract, produced either as a natural, synthesized, and/or genetically engineered drug for which we own or are a co-owner of the patents, or acquire such rights in the future, for a period not to exceed the life of the patents. If we manufacture and market the drugs ourselves, we will pay an amount equal to 5% of gross sales from any products sold during the life of the patents. On April 16, 2001, this agreement was

amended and clarified to provide that Ms. Shogen would receive the 15% royalty payment relating to license(s) or the 5% of the net sales from any products sold during the life of the patents but not both, unless we and a licensee both market the licensed product.

In December 1999, our compensation committee approved the issuance of an aggregate total of 75,000 stock options to our outside board of directors, which vested on the date of grant. The exercise price of the stock options was $0.47 per share which was based on the average of the high and low trade prices of our common stock for the twenty trading days preceding the date of grant. An aggregate 50,000 of these options were exercised.

In April 2001, our board of directors approved the issuance of 50,000 stock options under the 1997 Plan to Martin Stadler, which vested on the date of grant. The exercise price of the stock options was $0.90 per share which was based on the average of the high and low trade prices of our common stock for the ten trading days preceding the date of grant.

In April 2001, we issued convertible notes to Kuslima Shogen, our Chief Executive Officer and a director, two of our directors, Donald Conklin and Martin Stadler, and unrelated parties in the aggregate amount of $366,993. Messrs. Conklin and Stadler are members of our Compensation Committee. The notes are due within ninety days unless the lenders elect to exercise an option to convert their note into common stock at the conversion price of $0.90 per share. The related parties named above have elected to convert their notes into an aggregate 330,000 shares of common stock. In addition, upon conversion, they received three-year warrants to purchase an aggregate 330,000 shares of common stock at an exercise price of $2.50 per share that will expire on July 7, 2004. In October 2001, the Board of Directors approved a change in the exercise price

of the 330,000 warrants issued to related parties from $2.50 per share to $1.50 per share and changed the expiration date to July 7, 2006, to conform with the private placements to unrelated parties. The notes issued to unrelated parties with an aggregate balance of $69,993 were renewed for one hundred twenty (120) days for the same conversion price of $0.90 per share. In addition, upon conversion, they will receive five-year warrants to purchase an aggregate 77,770 shares of common stock at an exercise price of $1.50 per share. In October 2001, the remaining noteholders elected to convert an aggregate $64,993 notes payable into an aggregate 72,214 shares of common stock. In addition, they received five-year warrants to purchase an aggregate 72,214 shares of common stock at an exercise price of $1.50 per share.

During the fiscal year ended July 31, 2002, Kuslima Shogen, the Company's CEO has made loans to the Company repayable upon demand bearing interest at 8% per annum. As of July 31, 2002, the Company owes Ms. Shogen $139,800.

Item 14. 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 November 11, 2002, 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.

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

(a)(1) and (2) The response to these portions of Item 14 is submitted as a separate section of this report commencing on page F-1.

(a)(3) and (4) Exhibits (numbered in accordance with Item 601 of Regulation S-K).

Exhibit No.	Item Title
3.1	Certificate of Incorporation
3.2	By-Laws
3.3	Amendment to Certificate of Incorporation
3.4	Amendment to Certificate of Incorporation

4.1	Form of Convertible Debenture
10.1	Form of Stock and Warrant Purchase Agreements used in private placements completed in April 1996 and June 1996
10.2	Lease Agreement - 225 Belleville Avenue, Bloomfield, New Jersey
10.3	Form of Stock Purchase Agreement and Certificate used in connection with various private placements
	Form of Stock and Warrant Purchase Agreement and Warrant Agreement used in Private 10.4 Placement completed on March 21, 1994
10.5	1993 Stock Option Plan and Form of Option Agreement
10.6	Debt Conversion Agreement dated March 30, 1994 with Kuslima Shogen
10.7	Accrued Salary Conversion Agreement dated March 30, 1994 with Kuslima Shogen
10.8	Accrued Salary Conversion Agreement dated March 30, 1994 with Stanislaw Mikulski
10.9	Option Agreement dated March 30, 1994 with Kuslima Shogen
10.10	Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Kuslima Shogen
10.11	Form of Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Kuslima Shogen
10.12	Form of Amendment No. 1 dated June 20, 1994 to Option Agreement dated March 30, 1994 with Stanislaw Mikulski
10.13	Form of Stock and Warrant Purchase Agreement and Warrant Agreement used in Private Placement completed on September 13, 1994
10.14	Form of Subscription Agreements and Warrant Agreement used in Private Placements closed in October 1994 and September 1995
10.15	1997 Stock Option Plan
10.16	Separation Agreement with Michael C. Lowe dated October 9, 1997
10.17	Form of Subscription Agreement and Warrant Agreement used in Private Placement completed on February 20, 1998
10.18	Form of Warrant Agreement issued to the Placement Agent in connection with the Private Placement completed on February 20, 1998
10.19	Placement Agent Agreement dated December 15, 1997
10.20	Separation Agreement with Gail Fraser dated August 31, 1999
10.21	Form of Subscription Agreement and Warrant Agreement used in Private Placements completed in February 2000
10.22	Form of Subscription Agreement and Warrant Agreement used in the August and September 2000 Private Placements

Exhibit No.	Item Title
10.23	Form of Subscription Agreement and Warrant Agreement used in the April 2001 Private Placements
10.24	Form of Convertible Note entered into in April 2001
10.25	Form of Subscription Agreement and Warrant Agreement used in the July 2001 Private Placements
10.26	Form of Subscription Agreement and Warrant Agreement used in the August and October 2001 private placement
10.27	Form of Subscription Agreement and Warrant Agreement used in the September 2001, November 2001 and January 2002 private placements
10.28	Warrant issued in the February 2002 private placement
10.29	Form of Subscription Agreement and Warrant Agreement used in the March 2002, April 2002 and May 2002 private placements.
21.1	Subsidiaries of Registrant
23.1	Consent of KPMG LLP
99.1	Factors to Consider in Connection with Forward-Looking Statements
99.2	Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to section 906 of the Sarbanes-Oxley Act of 2002
99.3	Certification pursuant to 18 U.S.C. Section 1350, as adopted pursuant to section 906 of the Sarbanes-Oxley Act of 2002

* 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 January 31, 1994 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 Registration Statement on

Form SB-2 (File No. 33-83072) 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, 1997 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 Registration statement on Form SB-2 (File No. 333-11575) 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.

Previously filed as exhibits to the Company's Annual Report on Form 10-K for the year ended July 31, 1999 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: November 13, 2002 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: November 13, 2002 /s/ KUSLIMA SHOGEN
Kuslima Shogen, Chief Executive Officer, Acting Chief Financial Officer (Principal Executive Officer, Principal Accounting Officer) and Chairman of the Boa

Dated: November 13, 2002 /s/ STANISLAW M. MIKULSKI
Stanislaw M. Mikulski, M.D., Executive Vice President and Director

Dated: November 13, 2002 /s/ STEPHEN K. CARTER
Stephen K. Carter, M.D., Director

Dated: November 13, 2002 /s/ DONALD R. CONKLIN
Donald R. Conklin, Director

Dated: November 13, 2002 /s/ MARTIN F. STADLER
Martin F. Stadler, Director

CERTIFICATIONS

I, Kuslima Shogen, certify that:

1. I have reviewed this annual report on Form 10-K of Alfacell Corporation;

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

report;

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

Date: November 13, 2002

/s/ Kuslima Shogen

Name: Kuslima Shogen
Title: Chief Executive Officer and Chairman of the Board

CERTIFICATIONS

I, Kuslima Shogen, certify that:

1. I have reviewed this annual report on Form 10-K of Alfacell Corporation;

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

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

Date: November 13, 2002

/s/ Kuslima Shogen

Name: Kuslima Shogen
Title: Acting Chief Financial Officer

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Independent Auditors' Report

The Stockholders and Board of Directors
Alfacell Corporation:

We have audited the accompanying balance sheets of Alfacell Corporation (a development stage company) as of July 31, 2002 and 2001, and the related statements of operations, stockholders' equity (deficiency), and cash flows for each of the years in the three-year period ended July 31, 2002 and the period from August 24, 1981 (date of inception) to July 31, 2002. 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 2001, and the results of its operations and its cash flows for each of the years in the three-year period ended July 31, 2002 and the period from August 24, 1981 to July 31, 2002 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