MICROMET, INC.
Form 10-K
March 16, 2007

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UNITED STATES SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

Form 10-K

FOR ANNUAL AND TRANSITION REPORTS
PURSUANT TO SECTIONS 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

(Mark One)

þ **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**
For the year ended December 31, 2006

or

o **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**
For the transition period from to

Commission file number: 0-50440

Micromet, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**52-2243564**
(State or other jurisdiction of incorporation or organization)	*(I.R.S. Employer Identification No.)*
2110 Rutherford Road **Carlsbad, CA**	**92008**
(Address of principal executive offices)	*(Zip Code)*

(760) 494-4200
(Registrant s telephone number, including area code)

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

Title of Each Class	**Name of Each Exchange on Which Registered**
Common Stock, par value $0.00004 per share	Nasdaq Global Market

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

Indicate by check mark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. Yes o No þ

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Act. Yes o No þ

Note- checking the box above will not relieve any registrant required to file reports pursuant to Section 13 of 15(d) of the Exchange Act from their obligations under those Sections.

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 þ No o

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 the registrant s knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. þ

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, or a non-accelerated filer. See definition of accelerated filer and large accelerated filer in Rule 12b-2 of the Exchange Act. (Check one):
Large accelerated filer o Accelerated filer þ Non-accelerated filer o

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Exchange Act). Yes o No þ

As of June 30, 2006, the aggregate market value of the registrant s common stock held by non-affiliates of the registrant was approximately $77.5 million, based on the closing price of the registrant s common stock on that date as reported by the NASDAQ Global Market.

The number of outstanding shares of the registrant s common stock, par value $0.00004 per share, as of March 5, 2007 was 31,502,128 shares.

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the registrant s definitive Proxy Statement to be filed with the Securities and Exchange Commission within 120 days after registrant s fiscal year ended December 31, 2006 are incorporated by reference into Part III of this report.

MICROMET, INC.

ANNUAL REPORT ON FORM 10-K
FOR THE YEAR ENDED DECEMBER 31, 2006

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PART I

Item 1. *Business*

INFORMATION REGARDING MICROMET S BUSINESS

Company Overview

We are a biopharmaceutical company focusing on the development of novel, proprietary antibody-based products for cancer, inflammatory and autoimmune diseases. Two of our product candidates are currently in clinical trials, while the remainder of our product pipeline is in preclinical development. MT103 (also known as MEDI-538), which is the first product candidate based on our proprietary BiTE® product development platform, is being evaluated in a phase 1 clinical trial for the treatment of patients with non-Hodgkins lymphoma. The BiTE product development platform is based on a unique, antibody-based class of drugs that leverages the cytotoxic potential of T cells, widely recognized as the most powerful killer cells of the human immune system. Adecatumumab (also known as MT201), a recombinant human monoclonal antibody which targets EpCAM expressing tumors, has completed two phase 2a clinical trials, one in patients with breast cancer and the other in patients with prostate cancer. In addition, a phase 1b trial evaluating the safety and tolerability of adecatumumab in combination with docetaxel is currently ongoing in patients with metastatic breast cancer. We have established collaborations with MedImmune, Inc. for MT103 and Merck Serono for adecatumumab.

Our goal is to develop and commercialize products for the treatment of cancer and inflammatory and autoimmune diseases that have significant unmet medical needs. We believe that our novel technologies, product candidates and product development expertise in these fields will continue to enable us to identify and develop promising new product opportunities for these critical markets.

Corporate History

On May 5, 2006, CancerVax Corporation completed a merger with Micromet AG, a privately-held German company, pursuant to which CancerVax s wholly owned subsidiary, Carlsbad Acquisition Corporation, merged with and into Micromet Holdings, Inc., a newly created parent corporation of Micromet AG. Micromet Holdings became a wholly owned subsidiary of CancerVax and was the surviving corporation in the merger. CancerVax issued to Micromet AG stockholders shares of CancerVax common stock and CancerVax assumed all of the stock options, stock warrants and restricted stock of Micromet Holdings outstanding as of May 5, 2006, such that the former Micromet AG stockholders, option holders, warrant holders and note holders owned, as of the closing, approximately 67.5% of the combined company on a fully-diluted basis and former CancerVax stockholders, option holders and warrant holders owned, as of the closing, approximately 32.5% of the combined company on a fully-diluted basis. In connection with the merger, CancerVax was renamed Micromet, Inc. and our Nasdaq Global Market ticker symbol was changed to MITI.

Micromet AG was founded in 1993 as a spin-off from the Institute for Immunology at Munich University. CancerVax Corporation was incorporated in Delaware in June 1998 and commenced operations in the third quarter of 2000.

Unless specifically noted otherwise, as used throughout this report:

CancerVax Corporation or CancerVax refers to the business, operations and financial results of CancerVax Corporation prior to the closing of the merger between CancerVax Corporation and Micromet AG on May 5,

2006, at which time CancerVax s name was changed to Micromet, Inc. ;

Micromet AG refers to the business, operations and financial results of Micromet AG, a privately-held German company, prior to the closing of the merger and after the merger, as the context requires; and

Micromet, we, our, or us refers to the operations and financial results of Micromet, Inc. and Micromet AG c consolidated basis after the closing of the merger, and Micromet AG prior to the closing of the merger, as the context requires.

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Market Overview

Cancer is among the leading causes of death worldwide. The World Health Organization estimates that more than 10 million people were diagnosed with cancer worldwide in the year 2000 and that this number will increase to 15 million by 2020. In addition, the World Health Organization estimates that 7.6 million people died from the disease in 2005, representing 13% of all deaths worldwide. The American Cancer Society, or ACS, estimates that over 1.4 million people in the U.S. were newly diagnosed with cancer in 2006 and over 560,000 people died from the disease in the U.S. in 2006. Also according to the ACS, in the U.S. one in every four deaths is due to cancer, and as a result it has become the second leading cause of death in all people (exceeded only by heart disease), and the leading cause of death in people over age 85.

The increasing number of people diagnosed with cancer and the approval of new cancer treatments are factors that are expected to continue to fuel the growth of the worldwide market for cancer drugs. The U.S. National Health Information Business Intelligence Reports states that, on a world-wide basis, the revenues for cancer drugs are expected to grow from $35.5 billion in 2003 to $53.1 billion in 2009.

Immunotherapy for the Treatment of Cancer

The body s immune system is a natural defense mechanism tasked with recognizing and combating cancer cells, viruses, bacteria and other disease-causing factors. This defense is carried out by the white blood cells of the immune system through a set of cytolytic, or cell killing, enzymes that either assemble on specific antibodies bound to the cell surface of target cells, or are discharged by certain white blood cells in a highly specific fashion. Specific types of white blood cells, known as T and B cells, are responsible for carrying out cell-mediated immune responses and humoral, or antibody-based, immune responses, respectively.

Cancer cells produce molecules known as tumor-associated antigens, which can also be present in normal cells but are frequently over-produced or modified in cancer cells. T and B cells have receptors on their surfaces that enable them to recognize the tumor-associated antigens. For instance, once a B cell recognizes a tumor-associated antigen, it triggers the production of antibodies that can bind and kill the tumor cells. T cells play more diverse roles, including the identification and destruction of tumor cells by direct cell-to-cell contact.

While cancer cells naturally trigger a T cell-based immune response during the initial appearance of the disease, the immune system response may not be sufficiently robust to eradicate or control the cancer. The human body has developed numerous immune suppression mechanisms to prevent the immune system from destroying the body s normal tissues. Cancer cells have been shown to utilize these same mechanisms to suppress the body s natural immune response against cancer cells. Even with an activated immune system, the number and size of tumors can overwhelm the body s immune response.

Our product candidates aim to efficiently redirect the patient s immune response to tumor cells either through the use of specific recombinant antibodies for the eradication of cancer cells, as is the case with adecatumumab, or through BiTE molecules, which target cancer cells for elimination by the patient s own T cells.

Types of Cancer

Our lead product candidates, MT103 and adecatumumab, target Non-Hodgkins lymphoma and breast cancer, respectively.

Non-Hodgkins Lymphoma

Overview and Current Therapies

The incidence of non-Hodgkins lymphoma, or NHL, is among the fastest growing of all cancers. Indolent NHL tumors grow relatively slowly in most cases and are divided into several subtypes, of which follicular lymphomas are the most common. Approximately 10% of patients with indolent lymphoma are diagnosed at stage I or localized stage II, and are potentially curable with radiotherapy. Patients diagnosed with stage II, III, or IV disease are often asymptomatic and remain under periodic observation. Treatment is generally initiated when patients become symptomatic or when biological evidence of increasingly active disease such as rapidly enlarging lymph nodes

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occurs. First-line treatment for patients with indolent NHL is usually chemotherapy, although recent data indicate that Genentech s, Biogen-Idec s and Roche s rituximab (Rituxan®) added to chemotherapy may provide additional long-term benefit. Rituxan is a monoclonal antibody that targets CD20, an antigen widely expressed on B cells. Patients often cycle between remission and relapse, and may survive for as long as eight to ten years following initial diagnosis. Upon relapse, patients may receive chemotherapy plus Rituxan, Rituxan alone, or chemotherapy alone. Over time, an increasing proportion of patients become refractory to treatments with chemotherapy and/or Rituxan, meaning that they no longer respond to treatment. Refractory patients may then receive radio-labeled monoclonal antibodies targeting CD20 (radioimmunotherapy) and/or experimental regimens including bone marrow transplantation (BMT). A transformation from indolent to aggressive lymphoma is also observed in some patients.

Aggressive NHL tumors are rapidly growing tumors and are divided into various subtypes, with diffuse large B-cell lymphomas (DLBCL) comprising the largest subtype. Current standard first-line treatment for DLBCL is a chemotherapy regimen plus Rituxan, and may result in a cure for approximately 50% of patients treated. The overall survival of patients who do not respond to first-line therapy is generally limited to a few years. Young patients and those with good clinical status may benefit from bone marrow transplantation, but most are treated with combinations of chemotherapy and Rituxan or radioimmunotherapy. Primary treatment for adult patients with acute lymphocytic leukemia/lymphoblastic lymphoma usually involves elaborate chemotherapeutic regimens usually including an induction phase and subsequent treatment(s). BMT may also be considered in specific patients and bcr-abl inhibitors may be considered in patients with bcr-abl mutations (e.g. MRD-positive disease). If patients do not respond to primary treatment or relapse, experimental therapies or re-induction with combination chemotherapy may be attempted, but the overall prognosis for patients in this circumstance is very poor. Altogether, despite recent advances in treatment choices, overall prognosis for survival remains poor in relapsed patients with aggressive NHL.

Our Approach

Our lead product candidate to treat NHL, MT103, also known as MEDI-538, is a recombinant, bispecific single-chain antibody construct, also called a BiTE, that targets CD19, which is exclusively expressed on B cells and B-cell derived B lymphoma cells. Preliminary preclinical and clinical data indicate that MT103 has activity in both indolent and aggressive NHL. Micromet has selected the CD19 target for MT103 for a number of reasons. First, the CD19 antigen is used in the clinic to distinguish lymphoma derived from B cells from those derived from T cells. CD19 serves as a co-receptor of the B cell receptor and is highly specific for the B cells and tumors derived from those B cells. Second, by not binding to CD20, which is the target for a number of antibody-based therapies (Rituxan, Bexxar®, Zevalin®), it may be possible to use MT103 in combination with anti-CD20 therapies, as there will be no competition between the therapeutic agents to bind to the same target antigen. Third, certain human B cell malignancies express CD19 but not CD20, such as those derived from early stages of B cell development. In addition to the treatment of CD20-positive lymphomas, we believe that MT103 will provide an opportunity to treat B cell malignancies that lack CD20, that have a low level of CD20 expression, or that have lost CD20 expression during treatment with Rituxan. We believe that our MT103 product candidate, if approved, may offer patients additional benefit in the treatment of NHL.

Breast Cancer and Other EpCAM-Positive Solid Tumors

Overview and Current Therapies

Breast cancer is the most common cancer in women and the second most common cause of malignancy-related deaths worldwide. Although the incidence of breast cancer is rising in many developed countries, primarily because of the growing number of elderly women, more women are surviving the disease, and those who are not cured are living longer. These achievements result from improved screening methods allowing earlier diagnosis, targeted surgery, post-surgical use of adjuvant treatments, and the use of successive hormonal and cytotoxic treatments for patients with

metastatic disease, as well as the introduction of new targeted anti-cancer therapies. In stage III locally-advanced tumors, neoadjuvant treatment is being increasingly used in order to reduce the size of tumors before standard surgery and radiation therapy.

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Although there is a consensus with regards to the approach to the diagnosis and treatment of patients with breast cancer, medical practice varies most in the treatment of low-risk, early-stage patients. As a consequence of wide-spread mammography screening, more than 80% of all invasive breast tumors are diagnosed in stage I or II. In these stages, the primary treatment is surgery, often combined with radiation. The additional treatment regimen is dependent on several factors, including whether the cancer has infiltrated the patient s lymph nodes. More aggressive therapy, often including chemotherapy, is used to treat patients with a high risk of relapse or who have lymph node metastases. Patients with hormone receptor positive disease usually receive additional anti-hormonal treatment with Tamoxifen alone or Tamoxifen followed by other agents such as aromatase inhibitors.

Research has found that the over-expression of the HER-2 gene contributes to the uncontrolled growth of tumor cells. It is estimated that approximately one in five breast cancer patients is HER-2 positive, and that these patients are likely to have a more aggressive form of cancer. As a result, patients with breast cancer are routinely tested for over-expression of HER-2, and those who test positive are typically treated with Genentech s and Roche s monoclonal antibody, trastuzumab (Herceptin®).

Treatment of patients with metastatic, or stage IV, breast cancer generally intends to prolong progression-free time and improve quality of life. Within this group, prognosis and therapy depend on the presence of hormone receptors for estrogen and progesterone (ER^+/PR^+). Patients with a positive hormone receptor status usually receive hormone therapy (e.g., aromatase inhibitors). Depending on the speed of progression, they then either undergo an additional course of hormone therapy or they are switched to chemotherapy. Patients with more advanced or symptomatic disease or with hormone receptor negative status (ER^-/PR^-) will typically receive chemotherapy. Radiation therapy may be warranted in specific cases with symptomatic metastases. Herceptin® has been marketed since 1998 in the U.S. and since 2000 in the EU for the treatment of patients with HER-2 positive metastatic breast cancer either in combination with paclitaxel after anthracyclin pre-treatment, or as monotherapy in second or third-line metastatic breast cancer patients.

Unmet Medical Needs

Despite recent advances, current breast cancer treatments still do not sufficiently address patients needs. In particular, the following therapies are still needed:

- More effective therapies (defined as prolonged survival and improved quality of life) for patients with stage IV disease, whose cancer has metastasized to another area of the body;
- Less toxic, more convenient secondary therapies to prolong time to disease progression, reduce disease-related symptoms, and improve quality of life; and
- Therapies that increase the overall survival of patients with stage II/III disease, such as neo-adjuvant treatment regimens.

Our Approach

We believe that, if approved, our product candidate adecatumumab, which is a recombinant human monoclonal antibody that targets the epithelial cell adhesion molecule, or EpCAM, may offer a unique approach in treating patients with breast cancer. Over-expression of the EpCAM target itself has been shown to significantly reduce the time and rate of survival of patients with node-positive breast cancer, and has also been shown to promote the proliferation, migration and invasiveness of breast cancer cells. In one study with approximately 1,700 subjects, a high level of EpCAM expression was found in approximately 42% of patients with primary breast cancer. By elimination

of tumor cells that express high levels of EpCAM, we believe that treatment with an anti-EpCAM compound such as adecatumumab may result in an increased time to disease progression, and if added to standard chemotherapy, such as taxanes, may also result in increased response rates and/or time to progression.

Other EpCAM-Positive Solid Tumors

Many solid tumors other than breast cancer, such as colorectal, lung, and ovarian cancer, have also been shown to express high levels of EpCAM. Similar to the data in patients with breast cancer, EpCAM overexpression has been associated with decreased disease-free survival or overall survival of patients in some of these diseases. For

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most solid tumors, the current standard of care consists of surgery, radiotherapy and treatment with certain substances such as chemotherapy, hormonal therapy, targeted therapy, monoclonal antibodies, or anti-angiogenic agents such as Avastin, either as single treatment modality or as a combination of the aforementioned therapy options. Despite recent advances in treating these malignancies over the last two decades, we believe that a tremendous need for further improvement of cancer therapy exists. Depending on the disease type and stage, major medical needs include the quest for improved survival, increased cure rates, prolonged disease-free survival, and improved control of symptoms. The recent approval of various monoclonal antibodies directed against certain tumor antigens (e.g., Herceptin®, Erbitux®) in various indications has confirmed the prospects of targeted therapy. We believe that, if approved, adecatumumab may offer a unique approach in treating patients with EpCAM-expressing solid tumors, or adenocarcinomas.

Our Product Pipeline

Our current product pipeline consists of a variety of different approaches to treating cancer, inflammatory and autoimmune diseases. The following table summarizes the current status of our product candidates in clinical and preclinical development:

Product Candidate	Primary Indication	Collaborator	Status
BiTE® MT103	Non-Hodgkins Lymphoma	MedImmune	Clinical Phase 1
BiTE® MT110	Adenocarcinoma		Pre-clinical
BiTE® EphA2	Selected Cancers	MedImmune	Pre-clinical
BiTE® CEA	Selected Cancers	MedImmune	Pre-clinical
Adecatumumab (MT201)	Metastatic Breast Cancer and other Adenocarcinoma	Merck Serono	Clinical Phase 2a
	Metastatic Breast Cancer in Combination with Docetaxel		Clinical Phase 1b
Antibody D93	Cancer		Open IND
Antibody MT203	Inflammatory Diseases		Pre-clinical
Antibody MORAb28	Melanoma	Morphotek	Pre-clinical
Antibody MT204	Inflammatory Diseases		Pre-clinical

MT103

MT103 is a recombinant, CD19-directed, bispecific single-chain antibody that was generated using our BiTE technology. MT103 consists of four immunoglobulin variable domains assembled into a single polypeptide chain. Two of the variable domains form the binding site for CD19, a cell surface antigen expressed on all B cells and most B tumor cells. The other two variable domains form the binding site for the CD3 present on all T cells. The resulting recombinant molecule is produced by fermentation in eukaryotic cells.

As discussed further under License Agreements and Collaborations below, in June 2003 we announced an agreement to jointly develop MT103 with MedImmune, Inc.

Mechanism of Action

BiTE molecules are designed to direct the body s cytotoxic, or cell-destroying, T cells against tumor cells, and represent a new therapeutic approach to cancer therapy. MT103 has shown cytotoxic efficacy at very low concentrations against CD19-positive lymphoma cells in preclinical tests using cell culture and mouse models and at

low ratios of T, or effector, cells to tumor target cells. Lymphoma-directed cytotoxicity has also been achieved in preclinical tests with unstimulated human T cells and in the absence of additional T cell stimuli.

BiTE molecules have been shown to induce an immunological synapse between a T cell and a tumor cell in the same manner as observed in physiological T cell attacks. These cytolytic synapses mediate the delivery of cytotoxic proteins called perforin and granzymes from T cells into tumor cells, ultimately inducing a self-destruction process in the tumor cell referred to as apoptosis, or programmed cell death. In the presence of BiTE molecules, T cells have been demonstrated to serially eliminate tumor cells, which explains the activity of BiTE molecules at very low

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ratios of T cells to target cells. Through the tumor killing process, T cells start to proliferate, which leads to an increased number of T cells at the site of attack. It is believed that this effect may have the potential to improve the function of a patient s immune system.

Clinical Trials

Clinical Trial MT103-104 (Relapsed-refractory NHL)

Based on the data from the previous phase 1 clinical trials using short-term infusion regimens (mentioned below), we initiated a phase 1 dose finding clinical trial in April 2004 designed to evaluate the safety and tolerability of the continuous intravenous infusion of MT103 over 4-8 weeks at different dose levels in patients with relapsed or refractory NHL. The phase 1 clinical trial protocol is an open-label, multi-center, dose escalation study, which is being conducted by investigators in Germany. Patients are being enrolled sequentially into cohorts with increasing doses. A maximum tolerated dose has not yet been reached.

Of 12 patients in dose cohort numbers 1 through 3 who have received at least two weeks of treatment and who have passed the first control CT scan at week 4, nine patients have shown stable disease and one patient a minor response. No patient in cohort numbers 1 through 3 has shown a partial or complete tumor response, based on reference radiology assessment according to standardized Cheson criteria for tumor response assessment of NHL. However, partial and complete responses were observed in patients treated with the highest dose so far (dose level 4 = 15 µg/m^2/24 h). As presented at the annual meeting of the American Association for Hematology (ASH) in 2006, four of the eight evaluable patients at this highest dose level in this clinical trial showed a clinically relevant reduction in tumor lesions (1 complete response, 2 partial responses, and 1 minor response, which is defined in the protocol as a 25 to 50 percent decrease in tumor mass). Investigators also observed a reduction of circulating B cells, which appeared to be correlated with increasing doses, with full depletion of B cells observed in all 8 evaluable patients at dose level 4. Furthermore, all patients at dose level 4 with bone marrow infiltration at initial screening showed a reduction or complete disappearance of lymphoma cells from bone marrow after treatment with MT103. Overall, MT103 showed acceptable tolerability in this ongoing clinical trial, and a maximum tolerated dose has yet to be reached. So far, the most frequent adverse side effects related to the administration of MT103 were lymphopenia, leukopenia, fever and elevation of liver enzymes. The most frequent adverse events of grade 3 or higher were lymphopenia in 11 patients (50%) and leukopenia in 9 patients (41%).

Side Effect Profile of MT103 as Observed in Early Clinical Trials

The most frequent clinical adverse events observed so far in previous short-term infusion trials with MT103 were related to the release of cytokines by the patients immune cells. Cytokines are small proteins that allow communication between cells of the immune system and between immune cells and other types of cells. Cytokines are typically produced by activated immune cells, such as T cells, and thus were expected in connection with the treatment of patients with MT103. Cytokine release was transient and decreased after multiple administrations of MT103. Clinically, the most frequent side effects included fever, rigor, fatigue, vomiting, tachycardia, hypertension, headache and back pain. Most of these events were of mild or moderate severity. The most frequent laboratory abnormalities were seen in various hematological parameters, coagulation parameters, and blood chemistry, including liver function tests, and were mostly mild to moderate and transient in nature. About 80% of all clinical adverse events and laboratory abnormalities occurred on the day of the first infusion, with a decreasing incidence during the subsequent infusions.

In our earlier phase 1 clinical trials of MT103 given repeatedly as short-term infusions (Study Numbers MT103 I/01-2001, MT103 I/01-2002, and MT103 I/01-2003), serious adverse side effects included infections, dyspnoea, hypersensitivity and various symptoms of the central nervous system (CNS), including tremor, speech disorder,

somnolence, disorientation, confusion, fatigue, urinary incontinence and vertigo. CNS-related side effects led to termination of the treatment in a total of six patients in these short-term infusion trials. All these side effects fully resolved within a period of a few hours to a few days, with the exception of one patient, who suffered from seizures and a myocardial ischemia, or loss of blood flow to the heart. This patient ultimately died 49 days after receiving the last dose, and the cause of death was determined to be pneumonia. The autopsy and histopathological analysis of this patient suggested a CNS-related lymphoma lesion underlying these events. Based on adverse events

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and the lack of tumor responses in patients treated with the short-term infusion regimen, we terminated those short-term infusion studies and developed a new dosing regimen continuous infusion designed to reduce side effects and to obtain tumor responses in NHL patients.

Based on patients in cohort numbers 1 through 4 in our ongoing trial, the frequency of adverse side effects in the ongoing continuous infusion clinical trial has been lower when compared to the previous short-term infusion regimens, despite the fact that MT103 was present for four to eight weeks in patients in the continuous infusion study while it was only present for a few hours in the patients in the short-term infusion studies. We did not observe the CNS-related side effects in cohort numbers 1 through 3 that were seen in the short-term infusion trials, and no dose-limiting toxicity was observed. As presented at the ASH 2006 annual meeting, one out of 10 patients of cohort number 4 available for safety evaluation has shown transient and fully reversible confusion and disorientation. The safety evaluation of cohort number 4 is still ongoing.

Regulatory Pathway

MT103 is currently under clinical development in Europe. In addition, our collaborator MedImmune has submitted an IND to commence clinical testing of MT103 in the United States in the third quarter of 2006.

We have received orphan drug designation from the European Medicines Agency (EMEA) for the use of MT103 as a treatment for mantle cell lymphoma, or MCL, and chronic lymphatic lymphoma, or CLL. Orphan drug designation from the EMEA is designed to encourage manufacturers to develop drugs intended for rare diseases or conditions affecting fewer than 5 in 10,000 individuals in the European Union. Orphan drug designation also qualifies the applicant for tax credits and marketing exclusivity for seven years following the date of the drug s marketing approval by the EMEA. MedImmune has received orphan drug designation from the EMEA for the use of MT103 in the treatment of indolent B-cell lymphoma, excluding CLL and NHL with CNS involvement.

MT110

Another of our BiTE product candidates, MT110, is a molecule that combines binding specificities for EpCAM and for CD3 on T cells. EpCAM is a cell surface antigen that is over-expressed by many types of solid tumors.

Mechanism of Action and Preclinical Activities

As described above, BiTE molecules are designed to direct the body s cytotoxic T cells against tumor cells. MT110 has shown cytotoxic efficacy against EpCAM-positive tumor cells at very low concentrations and at low ratios of T cells to tumor target cells in preclinical tests using cell culture and mouse models. Of note, MT110 and other EpCAM-specific BiTE molecules were capable of inducing durable elimination of established tumors in mouse models. Likewise, human metastatic tissue from ovarian cancer patients implanted under the skin of mice was eliminated by low doses of intravenously administered MT110. We believe that this suggests that MT110 penetrated the human tumor and re-directed human tumor-infiltrating T cells for the destruction of tumor cells.

As a BiTE molecule, and as described above with respect to MT103, MT110 has been shown to induce an immunological synapse between a T cell and a tumor cell in the same manner as observed in physiological T cell attacks. The mechanism of action of MT110 is similar to that of MT103, by mediating the targeted delivery of perforin and granzymes from T cells into tumor cells.

Regulatory Pathway

We plan to submit an IND for MT110 with the FDA or an investigational medicinal product dossier, or IMPD, with European authorities in 2007.

BiTE molecules targeting EphA2 and CEA

Under our collaboration with MedImmune, as further described below under License Agreements and Collaborations, we are developing BiTE molecules targeting EphA2 and CEA for the treatment of cancer. Both molecules are currently in preclinical development.

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Mechanisms of Action and Preclinical Activities

EphA2 is a cell surface membrane-associated receptor tyrosine kinase that, in normal cells, is thought to function in suppressing cell growth and migration. Studies have indicated that EphA2 may be a molecule candidate for a targeted therapeutic antibody approach for the treatment of cancer. EphA2 is frequently overexpressed in a number of different tumor types, including renal cell carcinoma, breast, prostate, colon, esophageal, cervical, lung, ovarian and bladder cancers and melanoma. The highest levels of EphA2 expression are observed in the most aggressive tumor cells, suggesting that it may play a role in disease progression. High levels of EphA2 have also been correlated with poor survival in patients with non-small cell lung, esophageal, cervical, and ovarian cancers. Additionally, in pre-clinical models, it has been demonstrated that the addition of EphA2 is sufficient to make non-tumorous cells tumorous in both in vitro and in vivo settings. Therefore, we believe that a BiTE approach holds promise for the treatment of many cancer types that overexpress EphA2.

Carcinoembryonic antigen, or CEA, is expressed in a number of tumors of epithelial origin such as colorectal carcinoma, lung adenocarcinoma, mucinous ovarian carcinoma and endometrial adenocarcinoma. Furthermore, it is considered to be upregulated in gastric carcinoma and possibly also in colorectal carcinoma. Therefore, we believe that CEA is an excellent molecule candidate for a targeted therapeutic antibody approach for the treatment of cancer utilizing our BiTE technology. In the progress of cancer, members of the CEA family may play a role as contact-mediating adhesion molecules when tumor cells are moving to new sites. It has been shown that increased adhesion enhances the spread of cancer. Therefore, we believe that a BiTE approach may hold promise for the treatment of many cancer types that overexpress CEA.

Adecatumumab (MT201)

Our product candidate, adecatumumab, which we also refer to as MT201, is a recombinant human monoclonal antibody of the IgG1 subclass that targets the EpCAM molecule. EpCAM is a cell surface protein that is overexpressed on most solid tumor types, including prostate, breast, colon, gastric, ovarian, pancreatic and lung cancers. Overexpression of EpCAM has been shown to promote the proliferation, migration and invasiveness of breast cancer cells. Moreover, highly tumorous human breast and colon cancer stem cells are characterized by expression of EpCAM. In addition, expression of EpCAM has been shown to be associated with decreased survival in a number of cancer indications, including breast, gall bladder, bile duct, ovarian and ampullary pancreatic cancers.

Adecatumumab is administered intravenously. The anticipated treatment regimen consists of intravenous application over a 60-minute period every one to three weeks, either as a monotherapy or in combination with standard chemotherapy. Adecatumumab is expected to bind to EpCAM on tumor tissue and recruit natural killer cells and other immune cells to the tumor. Complement-dependent and antibody-dependent cellular cytotoxicity are believed to be the key modes of action of adecatumumab that trigger tumor cell destruction.

As discussed further under License Agreements and Collaborations below, adecatumumab is the subject of an exclusive worldwide collaboration with Ares Trading S.A., a wholly-owned subsidiary of Merck Serono Biopharmaceuticals, a Swiss corporation. We entered into the collaboration with Merck Serono in December 2004.

Clinical Trials

The following table describes the status of the clinical trials for adecatumumab:

Number of

Phase of Clinical Trial	Indication	Status	Subjects
Phase 2 (adecatumumab monotherapy)	Metastatic breast cancer	Completed	112
Phase 2 (adecatumumab monotherapy)	Prostate cancer	Completed	84
Phase 1 (adecatumumab plus docetaxel)	Metastatic breast cancer	Ongoing	Up to 36
Phase 1 (adecatumumab monotherapy)	Adenocarcinomas	Planned	
Phase 1 (adecatumumab monotherapy)	Hormone refractory prostate cancer	Completed	20

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Phase 2 Clinical Trial Patients with Metastatic Breast Cancer (Adecatumumab monotherapy)

Adecatumumab has been evaluated in a phase 2 clinical trial in patients with metastatic breast cancer. We initiated enrollment in this clinical trial in February 2004 and completed enrollment in October 2005, with a total of 112 patients from 26 sites in five European countries. This clinical trial was a randomized, open-label, multi-center, parallel group study designed to provide preliminary information regarding the efficacy and safety of adecatumumab when administered up to 24 weeks to patients who tested positive for expression of EpCAM.

The patients in this clinical trial were stratified into two groups (high and low) according to their level of EpCAM expression. Of the 109 positive patients, 71 were grouped in the high EpCAM expression group, while 38 were in the low to moderate EpCAM expression group. Patients in each expression group were then randomly divided into two equal dosage groups, either the low dose treatment group (2 mg/kg body weight) or the high dose treatment group (6 mg/kg body weight).

Treatment Groups	**EpCAM Expression**	**MT201 Dosing**
Group I	Moderate EpCAM Expression on Primary Tumor	2 mg/kg
Group II	Moderate EpCAM Expression on Primary Tumor	6 mg/kg
Group III	High EpCAM Expression on Primary Tumor	2 mg/kg
Group IV	High EpCAM Expression on Primary Tumor	6 mg/kg

The protocol called for each patient to receive an intravenous infusion every two weeks, for a total of up to 14 infusions of adecatumumab over 24 weeks of therapy unless disease progression or treatment-limiting toxicity occurred. Patients with at least stable disease after 24 weeks were allowed to continue treatment with adecatumumab in a follow-up study.

The primary endpoint of the study evaluated the clinical benefit rate, which comprises the percentage of patients whose disease was stabilized over 24 weeks of therapy or whose tumors demonstrated a partial response or a complete response, each as defined using standard Response Evaluation Criteria in Solid Tumors, or RECIST. Efficacy evaluations occurred every six weeks after the first administration of adecatumumab until week 24, and then every eight weeks thereafter. These evaluations included a thoracic CT scan or chest X-rays, an abdominal CT scan or MRI, and bone scintigraphy for patients with bone metastasis. Responses were assessed using RECIST and had to be confirmed by an Independent Review Board.

Final data for this trial were presented during the 31st Congress of the European Society of Medical Oncology (ESMO) in Istanbul, Turkey in October 2006 and the annual San Antonio Breast Cancer Meeting in December 2006. While the primary endpoint of the study (25 percent clinical benefit rate at week 24) was not reached, our secondary endpoint analysis showed a significant prolongation of time-to-progression (or TTP) in patients treated with the higher dose of adecatumumab ($p=0.0465$) compared to patients receiving the lower dose. In addition, the importance of target presence was underscored by a trend towards increased TTP in patients expressing high EpCAM levels as opposed to patients with low or moderate EpCAM expression on their primary tumor tissue. Patients receiving the high dose of adecatumumab and expressing high EpCAM (high/high) on their tumor tissue had a significantly longer TTP compared to patients with low EpCAM on their tumor tissue and treated with the low dose of adecatumumab (low/low) ($p=0.0057$). Progression-free survival was 336 days, 128 days and 49 days for 10% (10-percentile), 25% (25-percentile) and 50% (median) of patients in the high/high group compared to 112 days, 59 days and 42 days, respectively, in the low/low group.

The overall safety profile of the two doses of adecatumumab given (2 mg/kg and 6 mg/kg) indicated that both doses were well tolerated. When comparing the high dose with the low dose, an increase in mild-moderate toxicities (grade 1-2 according to CTC-AE) was reported with noted side effects mostly being infusion-related (pyrexia, flush) and gastrointestinal (nausea, diarrhea). No relevant increase in grade 3-4 toxicities was observed when comparing the two dosing regimens. Some increases in the pancreatic enzymes lipase and amylase were observed, but no dose-dependency could be determined nor was any acute clinical pancreatitis reported.

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Phase 2 Clinical Trial Patients with Prostate Cancer (Adecatumumab as a Single Agent)

In May 2005, we completed enrollment for this study of 84 patients at 20 sites in four European countries. The last patient received treatment in October 2005. We reported final data for this trial in October 2006. The trial was a double-blind, randomized, placebo-controlled, multi-center study to investigate the efficacy and safety of two different dose regimens of adecatumumab in patients with increasing serum Prostate Specific Antigen (or PSA) after radical prostatectomy for prostate cancer. The study was designed to evaluate the anti-tumor activity of adecatumumab by delaying further PSA increase in patients with increasing serum PSA after radical prostatectomy for treatment of prostate cancer.

Each patient had a total of 12 scheduled visits, including one screening visit, seven visits during the treatment period, and four follow-up visits. PSA was measured at the screening visit, during treatment at day 1, 29 and 57 and during follow-up at week 13, 15, 20 and 24. Bone scintigraphy, chest X-ray and pelvic CT scanning were performed at the screening and at any time during the study in case of confirmed biological progression, if PSA was ≥ 20 ng/ml, or in case of clinical disease progression.

The primary endpoint of this study was mean change in PSA at week 24 compared to baseline. While the primary end point was not reached, the analysis of the final data of the study indicated that treatment with a dose of 6 mg/kg of adecatumumab had a beneficial trend when compared to placebo (0.46 ng/ml versus 1.24 ng/ml; p=0.0879). Upon additional analysis, this trend was only seen for patients having high EpCAM expression levels (p=0.0884), but not for patients with low EpCAM expression (p=0.7947). The patients included in this trial had a high variability of PSA at study start (variation by a factor of 100 with baseline PSA values ranging from 0.2 to 20 ng/ml in serum). The clinical experts advising us in connection with this trial determined that this high variability of PSA may have confounded the results and recommended that a retrospective sub-group analysis of the primary endpoint should be performed in a more homogeneous patient population. According to these clinical experts, patients predominantly with PSA levels of 1 ng/ml at baseline or below would define a so-called minimal residual disease setting. The retrospective sub-group analysis for this specific patient population with high EpCAM expression (n=23) showed that both high (6 mg/kg) and low (2 mg/kg) adecatumumab doses given every other week for seven weeks led to a statistically significant smaller increase in PSA (0.38 ng/ml; p=0.0356, and 0.21 ng/ml; p=0.0014, respectively) compared to the placebo group (0.76 ng/ml) in patients with a high EpCAM expression. No such differences were observed in patients expressing low levels of EpCAM. No PSA responses according to formalized criteria (>50% decrease in PSA compared to baseline) were observed in either group.

The overall safety profile of the two doses of adecatumumab given (2 mg/kg and 6 mg/kg) indicated that both doses were well tolerated. Similar to the breast cancer trial, when comparing the high dose with the low dose, an increase in mild to moderate toxicities (grade 1-2 according to CTC-AE) was reported with side effects being infusion-related (pyrexia, flush) and gastrointestinal (nausea, diarrhea). No relevant increase in grade 3-4 toxicities was observed when comparing the two dosing regimens. Comparison of the low dose to the placebo group revealed only a slight increase in overall side effects. Some increases in lipase and amylase have been observed, but no acute clinical pancreatitis was reported.

Phase 1 Clinical Trial Patients with Metastatic Breast Cancer (Adecatumumab in Combination with Docetaxel)

Our ongoing phase 1 clinical trial in patients with metastatic breast cancer is an open-label, multi-center study to investigate the safety and tolerability of intravenous infusions of a combination of increasing doses of adecatumumab and a standard dose of docetaxel in patients with EpCAM-positive advanced-stage breast cancer. The first patient was enrolled in this study in April 2005. We are conducting this clinical trial currently in five locations, three in Germany and two in Austria. Final results from this study are expected in 2008.

Phase 1 Clinical Trial Patients with Hormone Refractory Prostate Cancer (Adecatumumab monotherapy)

In 2003, we completed a phase 1/2 open-label, dose escalation clinical trial of adecatumumab in patients with hormone refractory prostate cancer. Patients were treated with two intravenous infusions of adecatumumab at doses up to 262 mg/m^2 body surface. No dose-limiting toxicity was reported at any of the doses investigated, and the

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maximum tolerated dose was not reached. The results of this study were published in 2006 in the European Journal of Oncology.

Additional Phase 1 Clinical Trial (Adecatumumab monotherapy)

We and Merck Serono jointly decided in 2006 to commence a new monotherapy study of adecatumumab in additional indications. The filing of a respective IND/IMPD is anticipated later in 2007.

MT203

MT203 is a human antibody that we believe has the potential to treat a wide variety of acute and chronic inflammatory diseases, including rheumatoid arthritis, asthma, psoriasis and multiple sclerosis. It neutralizes granulocyte/macrophage colony stimulating factor, or GM-CSF, a pro-inflammatory cytokine controlling the innate arm of the immune system. GM-CSF primarily acts in chronic phases of numerous human diseases, including rheumatoid arthritis, asthma, psoriasis and multiple sclerosis. Using an antibody to neutralize GM-CSF has been shown to prevent or even cure symptoms in numerous animal models mimicking the respective human diseases. We generated MT203 using phage display-guided selection licensed from Cambridge Antibody Technology Limited.

Mechanism of Action and Preclinical Activities

Like marketed antibody drugs Humira®, Avastin® and Remicade®, MT203 acts by neutralizing a soluble protein ligand, thereby preventing it from binding to its high-affinity cell surface receptor. We believe that this therapeutic principle is well-validated. MT203 is one of the first human antibodies neutralizing the biological activity of human and non-human primate GM-CSF. The binding characteristics of MT203 to GM-CSF have been characterized in a number of studies, and MT203 has shown biological activity in numerous cell-based assays. We have used a surrogate antibody neutralizing mouse GM-CSF to demonstrate that inhibition of GM-CSF is highly potent in preventing rheumatoid arthritis in a mouse model in which TNF neutralization is largely ineffective and in preventing other inflammatory and autoimmune diseases, such as asthma and multiple sclerosis. This surrogate antibody has comparable binding characteristics to MT203, and therefore we believe that MT203 could have similar positive effects.

MT204

MT204 is a humanized antibody that we believe has the potential to treat a wide variety of acute and chronic inflammatory diseases, including rheumatoid arthritis, asthma, acute transplant rejection, uveitis, psoriasis and multiple sclerosis. We designed MT204 to neutralize interleukin-2, or IL-2, an inflammation-causing cytokine which controls activation of T cells and natural killer cells. Interference with IL-2 signaling is a well-validated anti-inflammatory therapeutic approach as exemplified by small molecule drugs, such as cyclosporine or tacrolimus, and by antibodies blocking the high-affinity IL-2 receptor (Simulect® and Zenapax®). MT204 is the first humanized antibody targeting soluble human and non-human primate IL-2 by a unique mode of action, and has been shown in preclinical models to have inhibitory properties superior to those of IL-2 receptor-blocking antibody Zenapax.

Mechanism of Action and Preclinical Activities

Like marketed antibody drugs Humira®, Avastin® and Remicade®, MT204 acts by neutralizing a soluble protein ligand. MT204 prevents binding of IL-2 to its intermediate-affinity receptor on natural killer cells, and also inactivates the high-affinity receptor with bound IL-2. This is a novel mode of antibody action, which we believe could cause MT204 to have potent anti-inflammatory activity. The binding characteristics of MT204 to IL-2 and IL-2 receptors have been characterized in studies using various assay systems. While the mechanism of action of MT204 is

understood, the antibody is still in an early stage of development.

D93

D93 is a humanized, anti-metastatic and anti-angiogenic monoclonal antibody, for treatment of patients with solid tumors.

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In March 2007, we entered into an agreement with Tracon Pharmaceuticals, Inc. under which we have granted Tracon an exclusive, worldwide license to develop and commercialize D93. Tracon will be responsible for all development and commercial activities and plans to initiate a phase 1 clinical trial in the second half of 2007.

Mechanism of Action and Preclinical Activities

The extracellular matrix is a molecular network that provides mechanical support to cells and tissues but also contains biochemical information important to cellular processes such as cell proliferation, adhesion and migration. D93 binds specifically to hidden, or cryptic, binding sites on extracellular matrix proteins that become exposed as a result of the denaturation of collagen that typically occurs during tumor formation. Binding of D93 to these denatured extracellular matrix proteins has the potential to inhibit angiogenesis, and the growth, proliferation and metastasis of tumor cells.

We believe that this approach to inhibiting angiogenesis and metastasis may have several therapeutic advantages. Because D93 binds preferentially to extracellular matrix proteins that have been denatured during angiogenesis and tumor growth rather than to the native, undenatured forms of collagen, we believe that the D93 antibody may have greater specificity for the tumor site than other therapies. Additionally, denatured proteins in the extracellular matrix may provide a better therapeutic target for long-term treatment than binding sites found directly on tumor cells since the proteins in the extracellular matrix represent a stable structure and are less likely to undergo mutations that are typical for cancer cells. Due to the specific mechanism through which D93 inhibits angiogenesis and metastasis, we believe that it may have the potential to be used in combination with other anti-angiogenic agents or with treatments such as chemotherapy and radiation.

We believe that D93 may be also useful in other pathological conditions associated with angiogenesis, such as choroidal neovascularization, or CNV, an ophthalmologic condition caused by excess growth of blood vessels within the eye, which is the major cause of severe visual loss in patients with age-related macular degeneration. Data presented during the 2004 Annual Meeting of the Association for Research in Vision and Ophthalmology demonstrated that in a murine model of CNV, another of our anti-angiogenic monoclonal antibodies, H8, preferentially recognized areas of new vascular growth but not existing normal vasculature and inhibited angiogenesis in a dose-dependent manner.

In February 2006, we filed an IND to initiate a phase 1 clinical trial with D93 in patients with solid tumors. The FDA approved the IND in April 2006. We have not commenced any clinical testing of D93, but a comprehensive pre-clinical analysis of D93 showing anti-angiogenic as well as anti-metastatic activity in various animal models and targeting of D93 to sites of tumor growth was published in 2006 in the International Journal of Oncology. We have conducted a number of presentations on D93 and related antibodies at annual meetings of the American Association for Cancer Research (AACR) between 2004 and 2006.

Product Candidates Targeting the EGF Receptor Signaling Pathway

In July 2004 our wholly-owned subsidiaries Tarcanta, Inc. and Tarcanta, Limited signed an agreement with CIMAB, S.A., a Cuban company, whereby Tarcanta obtained the exclusive rights to develop and commercialize SAI-EGF, a product candidate that targets the EGF receptor signaling pathway in a specific territory, which includes the United States, Canada, Japan, Australia, New Zealand, Mexico and certain countries in Europe. In addition, these two subsidiaries signed an agreement with CIMAB and YM BioSciences, Inc., a Canadian company, to obtain the exclusive rights to develop and commercialize SAI-TGF, which targets transforming growth factor-alpha, and SAI-EGFR-ECD, which targets the extracellular domain of the EGF receptor, within the same territory. Both of these product candidates are in preclinical development. We are actively seeking to sublicense or sell all three of these product candidates.

MORAb28

In October 2002, we signed an agreement with M-Tech Therapeutics, Inc., obtaining the worldwide exclusive rights and license to develop and commercialize a human IgM monoclonal antibody binding to an antigen that has been identified as a cell-surface antigen present on human melanomas and tumors of neuroectodermal origin. In December 2004, we entered into an agreement with Morphotek, Inc., pursuant to which Morphotek was granted the

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right to evaluatie this antibody with an option to obtain a license from us. In December 2006, Morphotek exercised its option and obtained an exclusive sublicense to the intellectual property rights and property rights associated with this monoclonal antibody. We understand that Morphotek plans to file an IND later this year and commence clinical trials in 2008. As discussed under License Agreements and Collaboration Agreements below, our agreement with Morphotek entitles us to certain milestone payments, royalties and re-acquisition rights.

Our Business Strategy

Our objective is to establish a position as a leader in the research, development and commercialization of highly active, antibody-based drugs for the treatment of patients with cancer. Key aspects of our corporate strategy include the following:

Co-develop Compounds with Established Pharmaceutical and Biopharmaceutical Companies. We are working with Merck Serono and MedImmune to complete ongoing clinical trials and enter into the next stage of clinical development for two of our product candidates.

Maintain Commercialization Opportunities in Collaborations. We have retained an option to co-promote adecatumumab in Europe and the U.S., and have full commercialization rights for MT103 outside of North America. We intend to continue to pursue this partnering strategy in future collaborations.

Advance the Development of Our Preclinical Product Candidates. We have initiated the production of clinical trial-grade material for MT110, and we plan to commence clinical trials upon the availability of such material and the approval of an IND or IMPD.

Pursue Additional Collaborations for Our Product Candidates. We will continue to seek strategic collaborations for some or all of the product candidates in our product portfolio.

Leverage Our Internal Pipeline Generating Capabilities. Our current pipeline of BiTE molecules as well as human IgG1 antibodies have all been generated by personnel employed by us, with the exception of D93 and MORAb28, which were in-previously in-licensed by CancerVax. We will continue to leverage that capability for our early-stage development collaborations, as well as for generating additional product candidates for our own pipeline, especially new product candidates based on the proprietary BiTE development platform.

Intellectual Property

Our success will depend in large part on our ability to:

1. Obtain and maintain patent protection and other intellectual property for cell lines, antibodies, delivery systems, production methods and compositions of matter;

2. Achieve a scope of protection as broad as possible;

3. Defend our patents;

4. Enforce our patents;

5. Extend the patent life for our product candidates;

6. Preserve trade secrets and proprietary know-how; and

7. Operate without infringing the patents and proprietary rights of third parties.

We actively seek appropriate patent protection for our proprietary technologies by filing appropriate patent applications in the United States, Europe and selected other countries. Our policy is to seek patent protection for the inventions that we consider important to the development of our business. Particularly within the BiTE platform, our patent strategy aims to generate protection on different aspects of the BiTE technology with respect to specific antibody constructs or key BiTE-related technology. Our key goals are to expand the patent portfolio, generate patent protection for new product candidates, protect further developments of BiTE-related technologies and harmonize our filing and prosecution strategy with respect to the portfolio.

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As of December 31, 2006, we owned or have licensed approximately 42 U.S. patents, 27 U.S. patent applications, 47 foreign and international patents, and 118 foreign and international patent applications related to our technologies, compounds, and their use for the treatment of human diseases. The number of licensed patents does not include various divisionals, continuations and continuations-in-part of the licensed patents and patent applications, which are also licensed to us. Our issued patents in the United States expire during 2008 and 2018, and our issued patents in Europe and other jurisdictions expire in 2019. We have additional patent applications that, if granted, would extend our patent lives significantly beyond these expirations. We intend to continue using our scientific expertise to pursue and file patent applications on new developments with respect to products, uses, methods and compositions of matter to enhance our intellectual property position in the field of antibody therapeutics for the of treatment of human diseases.

License Agreements and Collaborations

We have entered into several significant license and collaboration agreements for our research and development programs, as further outlined below. These agreements typically provide for the payment by us or to us of license fees, milestone payments, and royalties on net sales of product candidates developed and commercialized under these agreements.

Agreements Relevant for the Generation of Antibodies and for the BiTE Platform in General

License Agreement with Isogenis/Biohybrid

In October 1999, we entered into an agreement with Biohybrid, Inc. (now called Isogenis, Inc.) granting us a worldwide, exclusive license under U.S. Patent No. 5,078,998 entitled *Hybrid Ligand Directed to Activation of Cytotoxic Effector Lymphocytes and Associated Target Antigens* as well as certain related technologies. We are obligated to pay a royalty on net sales in the United States of products derived from the intellectual property that we licensed under this agreement. If we were to sublicense our rights under this agreement, Isogenis would be entitled to a portion of the payments received by us from the sublicensee. The agreement also requires us to pay a minimum annual royalty of $100,000 which started in 2000. Finally, we are obligated to make a milestone payment upon receipt of the first marketing approval in the United States of each product derived from the intellectual property that we licensed under this agreement.

The term of this agreement continues until expiration of the last valid claim in the licensed patents. Either party may terminate the agreement for the other party s uncured material breach. In addition, Isogenis may terminate the agreement in the case of our bankruptcy, insolvency, or cessation of business. We may terminate the agreement if Isogenis converts the exclusive license to a non-exclusive license following certain breaches of the agreement by us, or if the claims of the licensed patent are declared invalid.

License Agreement with Roche

In September 2000, we entered into a license agreement with F. Hoffmann-La Roche and Roche Diagnostics Corporation and obtained an exclusive license to use Roche s proprietary metal chelate affinity purification technology for the BiTE molecules. We paid an initial license fee, have paid and will pay annual license fees and will pay a royalty on the costs of filled bulk product.

Purchase Agreement with Curis

In June 2001, we entered into an agreement with Curis, Inc. to purchase certain single-chain antigen binding molecule patents and license rights from Curis. In exchange for these patent and license rights, we paid to Curis an initial license fee, issued to Curis shares of our common stock, and provided a convertible note in the amount of 4.1 million, or $3.7 million. In addition, we are obligated to pay royalties on net sales of products based on the acquired technology. We are also required to pay to Curis 20% of all supplemental revenues in excess of $8.0 million in the aggregate. Supplemental Revenues includes both (i) proceeds received by us as damages or settlements for infringement of the purchased technology, and (ii) amounts received by us from licensing or sublicensing the purchased technology. In October 2004, we exchanged the convertible note issued to Curis for an

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interest-free note in the amount of 4.5 million, or $5.6 million. The remaining balance on this note will be paid in the second quarter of 2007.

Research and License Agreement with Merck KGaA/Biovation

In August 2001, we entered into a research and license agreement with Biovation Limited, a wholly owned subsidiary of Merck KGaA, Darmstadt, Germany, under which Biovation used their proprietary technology and generated certain variants of the anti-CD3 single-chain antibody used in our BiTE molecules with the aim of reducing the likelihood of potential immune responses upon administration of such molecules to human beings. We received and tested such deimmunized anti-CD3 domains in connection with our BiTE molecules. We paid a license fee and research fees to Biovation and will make milestone payments and pay royalties on net sales of any resultant BiTE products that include such deimmunized anti-CD3 to Biovation s parent company.

License Agreement with Enzon

In April 2002, we entered into a cross-license agreement with Enzon, Inc. (now Enzon Pharmaceuticals, Inc.) relating to each party s portfolio of patents relating to single-chain antibodies and their use in the treatment of disease. This agreement was amended and restated by mutual agreement of the parties in June 2004. Under the cross-license agreement, we receive a non-exclusive, royalty- bearing license under Enzon s single-chain antibody patent portfolio to exploit licensed products other than BiTE products, as well as an exclusive, royalty-free license under such portfolio to exploit BiTE products. We also granted to Enzon a non-exclusive, royalty-bearing license under our single-chain antibody patent portfolio to exploit licensed products. Each party s license is subject to certain narrow exclusions for exclusive rights previously granted to third parties.

Each party is obligated to make milestone payments and pay royalties on net sales to the other party with respect to products that are covered by any patents within the consolidated patent portfolio, irrespective of which party owns the relevant patent(s). As noted above, we do not owe a royalty under this agreement to Enzon on net sales of BiTE products.

The term of the cross-license agreement continues until expiration of the last valid claim in the consolidated patent portfolio. Either party may terminate the agreement upon determination by a court of competent jurisdiction that the other party has committed a material breach of the agreement. Neither party has the right to unilaterally terminate the agreement without cause.

License Agreement with Cambridge Antibody Technology and Enzon

In September 2003, we entered into a cross-license agreement with Cambridge Antibody Technology Limited, or CAT, and Enzon to provide each party access to the other parties proprietary technology. This agreement superseded an existing cross-license arrangement among the parties. Pursuant to the current cross-license agreement, each party licenses to and from the others patents and know-how relating to the field of single-chain antibodies (in the case of licenses granted by Enzon and us) or phage display technology (in the case of licenses granted by CAT). This technology may be used by the parties for the research and development of antibody products in certain defined fields.

Pursuant to the cross-license agreement, we have the right to obtain a non-exclusive, worldwide license to use certain patented technology and know-how controlled by CAT in the field of phage display technology to develop and commercialize antibodies that bind to targets identified by us from time to time and approved by CAT through a predetermined process.

CAT paid an initial license fee to us under this agreement. Additionally, CAT is obligated to pay to us and Enzon: (i) annual license maintenance fees and fees for sublicenses granted by CAT to third parties, and (ii) annual maintenance fees on each sublicense until the termination of such sublicense or the expiration of all licensed patents included in such sublicense, whichever occurs first. We and Enzon are obligated to pay to CAT maintenance and sublicense fees based on the use of the licensed phage display technology by our respective sublicensees.

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Agreements Relevant for MT103

We entered into license and transfer agreements with certain individuals and research institutions to obtain certain intellectual property related to MT103. Under these agreements, we paid certain fees and will make milestone payments and pay royalties based on net sales of MT103.

Collaboration Agreement with MedImmune

In June 2003, we entered into a collaboration and license agreement with MedImmune to jointly develop MT103. Under the terms of the agreement, MedImmune received a license to MT103 and assumed responsibility for clinical development, registration and commercialization of MT103 in North America. We retained all rights to MT103 outside of North America. As part of the agreement, MedImmune is developing the manufacturing process for MT103 and will scale up that process to commercial scale. Also, MedImmune will supply MT103 for clinical trials and commercial sale for North America and for the markets outside of North America which we have retained. MedImmune will make milestone payments and will pay royalties to us on net sales of MT103 in North America. Until submission of the IND in September 2006, MedImmune reimbursed a portion of the clinical development costs incurred in Europe by us prior to the filing of such IND. Going forward, we have elected to share development costs of jointly conducted clinical trials.

During the years ended December 31, 2006, 2005 and 2004, this collaboration generated revenues to us of approximately 19%, 22% and 41%, respectively, of our total revenues.

Transfer Agreements around MT110

We entered into transfer agreements with certain individuals and another company to obtain certain intellectual property related to MT110. Under these agreements, we paid certain fees and will make milestone payments and pay royalties based on net sales of MT110. In addition, the license agreement with CAT discussed below in connection with agreements relating to adecatumumab also covers MT110.

Agreements Relevant for EphA2 BiTE and CEA BiTE

BiTE Research Collaboration Agreement with MedImmune

In June 2003, we entered in a BiTE Research Collaboration Agreement with MedImmune pursuant to which we have generated a BiTE binding to tyrosine kinase EphA2 and a BiTE binding to carcinoembryonic antigen (CEA) based on the BiTE platform. MedImmune is obligated to make milestone payments and pay royalties to us on net sales of the EphA2 BiTE and CEA BiTE. Furthermore, we have exclusive rights to develop and sell the CEA BiTE in Europe, and we also retain the option to obtain the right to co-promote the EphA2 BiTE in Europe. MedImmune is obligated to reimburse our full development costs incurred pursuant to development activities under the agreement up to and including phase 1 clinical trials.

Licensing of Single-Chain Antibody Patents

Exclusive IP Marketing Agreement with Enzon

In April 2002, we entered into an Exclusive Single-Chain Antibody IP Marketing Agreement with Enzon, which was amended and restated by the parties in June 2004. Under the 2004 agreement, we serve as the exclusive marketing partner for both parties consolidated portfolio of patents relating to single-chain antibody technology licensed under

the 2004 cross-license agreement discussed above. Licensing revenues are shared equally with Enzon, as are associated marketing and legal costs.

The term of the IP marketing agreement continues until expiration of the last valid claim in the consolidated patent portfolio. Either party may terminate the agreement upon determination by a court of competent jurisdiction that the other party has committed a material breach of the agreement. In addition, the marketing agreement terminates automatically upon termination of the cross-license agreement between us and Enzon. Neither party has the right to unilaterally terminate the agreement without cause prior to September 30, 2007. After such date, either party may terminate it at will.

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License Agreements with Various Parties pursuant to the Exclusive IP Marketing Agreement

Since April 2002 we have entered into several license agreements with third parties under the Enzon IP Marketing Agreement, and we have received license fees and milestone payments under several of these agreements, which aggregated approximately $2.0 million and $2.5 million in revenues for the years ended December 31, 2006 and 2005, respectively. In 2003, we entered into a research license agreement with ESBATech AG. ESBATech also has the option to extend the scope of the license to use single-chain antibodies in the development of therapeutics. In 2004, we entered into research license agreements with BioInvent AB, Merck & Co., Inc., and EvoGenix Pty Ltd. Also in 2004, Arizeke Pharmaceuticals Inc. entered into a license agreement with us for development and commercialization of single-chain antibodies targeting its proprietary pIgR antigen. In October 2006, we terminated the license agreement with Arizeke on the basis of the failure of Arizeke to meet its contractual payment obligations. In 2005, we entered into research license agreements with Abbott Laboratories, Alligator Bioscience AB, and Haptogen Ltd. Further, we entered into a product license agreement with Viventia Barbados, Inc. for the development and commercialization of a single-chain antibody for the treatment of cancer. In 2006, we entered into a license agreement with Antigenics, Inc. for the use of single-chain antibody technology in the research, development and manufacturing of non-single-chain antibody products.

Agreements Relevant for Adecatumumab (MT201)

Transfer Agreement with Inventors

In October 1998, we entered into an asset transfer agreement with a group of inventors at the University of Munich pursuant to which we acquired certain rights to adecatumumab. We have paid an initial fee and milestone payments under this agreement, and will make additional milestone payments and pay royalties based on net sales of adecatumumab.

License Agreement with Dyax

In October 2000, we entered into a non-exclusive license agreement with Dyax Corporation for the use of certain patented technology (including certain phage display processes) for screening and research of antibody products binding to EpCAM, including adecatumumab. We have paid an initial license fee and made a milestone payment under this agreement, and we will make additional milestone payments upon the achievement of specified events. No royalties are due under this agreement. The term of this agreement continues until expiration of the last valid claim in the licensed patents. Either party may terminate the agreement for the other party s uncured material breach. In addition, we may terminate the agreement at will.

License Agreement with Cambridge Antibody Technology

In September 2003, we entered into an agreement with CAT granting us a non-exclusive, worldwide license to use certain patented technology and know-how controlled by CAT in the field of phage display technology to develop and commercialize antibodies binding to EpCAM, the target of adecatumumab. We paid an initial license fee, and will make additional milestone payments and pay royalties based on net sales of adecatumumab.

Manufacturing and Supply Agreement with Boehringer Ingelheim

In December 2003, we entered into a process development agreement with Boehringer Ingelheim Pharma GmbH & Co. KG. Under the agreement, Boehringer Ingelheim is developing a commercial scale process for adecatumumab. Boehringer Ingelheim will supply us with material for clinical trials.

If we do not enter into a commercial supply agreement with Boehringer Ingelheim, or if we intend to establish a second source of supply, we have the right to manufacture adecatumumab under a license to Boehringer Ingelheim s high expression technology and the process developed for adecatumumab. Such license would carry an obligation for us to make milestone payments and pay royalties based on net sales of adecatumumab.

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Collaboration Agreement with Merck Serono

In December 2004, we entered into a collaboration agreement with Ares Trading S.A., a wholly-owned subsidiary of Serono International S.A., a leading Swiss biotechnology firm that was recently acquired by Merck KGaA and that is now called Merck Serono Biopharmaceuticals S.A. Pursuant to the agreement, we granted Merck Serono a worldwide license under our relevant patents and know-how to develop, manufacture, commercialize and use adecatumumab for the prevention and treatment of any human disease. Merck Serono paid an initial license fee of $10 million and has made three milestone payments in the total amount of $12 million to date. The most recent milestone paid was a $10 million payment made in November 2006 after the delivery by us of the study reports on two phase 2a clinical trials conducted with adecatumumab. Overall, the agreement provides for Serono to pay up to $138 million in milestone payments (in which the $12 million above is included) if adecatumumab is successfully developed and registered worldwide in at least three indications. The revenues from this collaboration agreement represented approximately 66% and 52% of our total revenues for the years ended December 31, 2006 and 2005, respectively.

Under the terms of the agreement, Serono bears all costs of product development and manufacturing subject to our participation right as described below. The original agreement provided that upon the completion of both phase 2 clinical studies in September 2006, Serono would assume the leading role in the management of any further clinical trials with adecatumumab, and at that time, we would have to decide whether or not to exercise our co-development option and participate in the costs and expenses of developing and selling adecatumumab in the United States and/or Europe. On November 24, 2006, we and Merck Serono amended the agreement to extend our leading role in the management of the clinical trials with adecatumumab until completion of the phase 1b clinical trial currently being conducted to evaluate the combination of adecatumumab and docetaxel in patients with metastatic breast cancer and the completion of an additional phase 1 clinical trial designed to demonstrate the safety of adecatumumab as a monotherapy in patients with other kinds of solid tumors. Merck Serono will continue to bear all of the development expenses associated with the collaboration. Further, under the amended agreement we can exercise our co-development option and participate in the costs and expenses of developing and selling adecatumumab in the United States and/or Europe after the end of both phase 1 clinical trials. If we exercise our option, we will then share up to 50% of the development costs, as well as certain other expenses, depending on the territory for which we exercise our co-development option. The parties will co-promote and share the profits from sales of adecatumumab in the territories for which the parties shared the development costs. In the other territories, Merck Serono will pay a royalty on net sales of adecatumumab.

Merck Serono may terminate the agreement following receipt by Merck Serono of the final study report for either of the ongoing and planned phase 1 trials, and for convenience upon specified prior notice. Either party may terminate for material breach or bankruptcy of the other. In the event of a termination of the agreement, all product rights will revert to us.

Agreements Relevant for D93

License Agreement with the University of Southern California

In September 1999, we entered into an exclusive license agreement with the University of Southern California (USC) with respect to patents for anti-angiogenic monoclonal antibody products binding to denatured collagen, which cover D93. We paid an initial license fee and will pay royalties on net sales of D93 including an annual minimum royalty. In February 2007, we amended the license agreement with USC to clarify the scope of the license and to exclude certain patents that claim antibody molecules that do not bind to denatured collagen.

Collaboration Agreement with Applied Molecular Evolution/Eli Lilly

In November 1999, we entered into a collaboration agreement with Applied Molecular Evolution, Inc. (AME , which was subsequently acquired by Eli Lilly and Company) to have AME humanize two of our murine monoclonal antibodies, which resulted in the development of the antibody now designated as D93. In February 2006, we filed an IND for D93, as required under our agreement with AME. If we intend to seek a licensee for D93, AME has a right of negotiation to obtain from us an exclusive license under our intellectual property rights related to

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the making, using and selling of D93, and under certain circumstances, AME has a right of first refusal with respect to such license agreement. We have an obligation to make milestone payments and pay royalties on net sales of D93.

License Agreement with Tracon Pharmaceuticals

In March 2007, we entered into an agreement with Tracon Pharmaceuticals, Inc. (Tracon), under which we granted Tracon an exclusive, worldwide license to develop and commercialize D93. Under the agreement, Tracon also has an option to expand the license to an additional antibody, and upon the exercise of the option, the financial and other terms applicable to D93 would become applicable to such other antibody. Under the terms of the agreement, Tracon will be responsible for the development and commercialization of D93 on a worldwide basis, as well as the costs and expenses associated with such activities. We will transfer to Tracon certain materials, including the stock of D93 clinical trial materials, stored at our contract manufacturer. Tracon is obligated to pay us an upfront license fee, make development and sales milestone payments, and pay a royalty on worldwide net sales of D93. In addition, Tracon will make certain payments for the delivery of the materials and has an obligation to pay us a portion of sublicensing revenues, which portion decreases based on the timepoint in the development of D93 when Tracon enters into the sublicense agreement. If D93 is successfully developed and commercialized in three indications in three major markets, we would be entitled to receive total payments, exclusive of royalties on net sales, of more than $100 million. Tracon may terminate the agreement at any time upon a specified prior notice period, and either party may terminate the agreement for material breach by the other party. In the event of termination, all product rights would revert back to us under the agreement.

Agreements Relevant for MT203

License Agreement with Enzon

In November 2005, we terminated a multi-year strategic collaboration with Enzon on mutually agreeable terms. We had entered into that agreement in April 2002 to identify and develop the next generation of antibody-based therapeutics. This collaboration had generated revenues during the years ended December 31, 2005 and 2004 that represented approximately 16% and 20%, respectively, of our total revenues in those years. In connection with the termination, we entered into a license agreement with Enzon for an antibody program targeting the human cytokine granulocyte/macrophage colony-stimulating factor (GM-CSF) that we had focused on under the collaboration. The agreement grants us the rights to certain patents of Enzon and patents and know-how created under the collaboration. We are obligated to pay royalties to Enzon upon the sale of products against GM-CSF using such patents or know-how.

License agreement with Cambridge Antibody Technology

In November 2003, we entered into an agreement with CAT granting us a non-exclusive, worldwide license to use certain patented technology and know-how controlled by CAT in the field of phage display technology to develop and commercialize antibodies binding to GM-CSF. We paid an initial license fee and are obligated to make additional milestone payments and pay royalties based on net sales of MT203.

Agreements Relevant for MT204

License Agreement with Enzon

In June 2004, we entered into a license agreement with Enzon for an antibody program targeting interleukin-2 (Il-2), which had been developed by the parties pursuant to the collaboration that has since been terminated. The agreement grants to us the rights to certain patents of Enzon and patents and know-how created under the collaboration. We are

obligated to pay royalties to Enzon upon the sale of products targeting Il-2 using such patents or know-how.

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Agreements Relevant for the Antibody MORAb28

License Agreement with M-Tech

In October 2002, we entered into an exclusive license agreement with M-Tech regarding patents, know-how and antibodies binding to different tumor antigens. The license agreement was amended in November 2004 and June 2006 and is limited to patents, know-how and antibodies, including antibody MORAb28 (formerly known as L72), which bind to a specific antigen. Under the M-Tech agreement we, or our sublicensee, have the obligation to perform development and achieve certain development milestones within certain timeframes. If we or our sublicensee fail to achieve these milestones, M-Tech has the right to terminate the agreement and we have to pay a termination fee. We paid M-Tech a license fee upon execution of the agreement, reimbursed M-Tech for certain development costs, and we have paid and are obligated to pay annual license maintenance fees, milestone payments, royalties on the net sales of resultant products, and a share of certain sublicensing revenues.

Sublicense Agreement with Morphotek

In December 2004, we entered into an exclusive sublicense agreement with Morphotek under which we granted Morphotek the right to evaluate certain antibodies, including antibody MORAb28, and an option to obtain an exclusive worldwide sublicense under our license from M-Tech. In December 2006, Morphotek exercised the option. Under the sublicense agreement, Morphotek has the obligation to perform development and achieve certain development milestones within certain timeframes. If Morphotek fails to achieve the milestones, we have the right to terminate the agreement, in which case Morphotek would be required to pay a termination fee. Morphotek paid us a license fee upon the execution of the option and is obligated to pay annual license maintenance fees, milestone payments, and royalties on the net sales of resultant products. Following commencement of phase 1 clinical trials and phase 2 clinical trials, we have the right to terminate and re-acquire Morphotek s rights for North America at pre-defined terms. If Morphotek intends to sublicense the rights for countries outside of North America to third parties, we have a right of first refusal to license back these rights.

Agreements Relevant for the EGF, TGF-alpha and HER-1 Vaccine Programs

License, Development, Manufacturing and Supply Agreement with CIMAB and YM BioSciences

In July 2004, we entered into a license agreement with CIMAB, S.A., a Cuban corporation, and YM BioSciences, Inc., a Canadian corporation, whereby we obtained the exclusive rights to develop and commercialize in a specific territory, which includes the U.S., Canada, Japan, Australia, New Zealand, Mexico and certain countries in Europe, three specific active immunotherapeutic product candidates that target the epidermal growth factor receptor (EGFR) signaling pathway for the treatment of cancer. Following the merger between Micromet AG and CancerVax Corporation, we decided to seek a suitable sublicense to continue with the development of these vaccine programs. Pursuant to letter agreements executed in October and November 2006, we agreed to terminate the agreements if no suitable partner was identified by the end of 2006, and to postpone the payment of a $1 million milestone payment that became due in the first quarter of 2006 until the earlier of i) the closing of a transaction in which a new partner obtains the rights to develop and commercialize the EGF vaccine and ii) June 12, 2007. In December 2006, we agreed to postpone the termination of the agreements until the end of February 2007, provided that the agreements would not terminate as of that date if certain conditions were met. We have satisfied the conditions, and the agreements currently remain in force and effect.

Other Licensing and Research and Development Agreements

Prior to the merger with Micromet AG, CancerVax had executed licensing and research and development agreements with various universities, research organizations and other third parties under which it received licenses to certain intellectual property, scientific know-how and technology. In consideration for the licenses received, we are required to pay license and research support fees, milestone payments upon the achievement of certain success-based objectives and/or royalties on future sales of commercialized products, if any. We may also be required to pay minimum annual royalties and the costs associated with the prosecution and maintenance of the patents covering the licensed technology.

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Manufacturing and Supply

Beside manufacturing and supply agreements for our clinical stage programs described above, non-GMP and GMP production agreements have been established with various manufacturers for our pre-clinical compounds.

Government Regulation and Product Approval

General

Governmental authorities in the United States and other countries extensively regulate the preclinical and clinical testing, manufacturing, labeling, storage, record-keeping, advertising, promotion, export, marketing and distribution of biologic products. In the United States, the FDA under the Federal Food, Drug, and Cosmetic Act, the Public Health Service Act and other federal statutes and regulations subjects pharmaceutical and biologic products to rigorous review. Parties that fail to comply with applicable requirements may be fined, may have their marketing applications rejected, and may be criminally prosecuted. The FDA also has the authority to revoke previously granted marketing authorizations upon failure to comply with regulatory standards or in the event of serious adverse events following initial marketing.

FDA Approval Process

The process required by the FDA before a new drug or biologic may be marketed in the United States generally involves the following: completion of preclinical laboratory and animal testing; submission of an IND, which must become effective before human clinical trials may begin; performance of adequate and well-controlled human clinical trials to establish the safety and efficacy of the proposed drug or biologic for its intended use; and submission and approval of a New Drug Application, or NDA, for a drug, or a Biologics License Application, or BLA, for a biologic. The sponsor typically conducts human clinical trials in three sequential phases, but the phases may overlap. In phase 1 clinical trials, the product is tested in a small number of patients or healthy volunteers, primarily for safety at one or more doses. In phase 2, in addition to safety, the sponsor evaluates the efficacy of the product in targeted indications, and identifies possible adverse effects and safety risks, in a patient population that is usually larger than phase 1 clinical trials. Phase 3 clinical trials typically involve additional testing for safety and clinical efficacy in an expanded patient population at geographically-dispersed clinical trial sites. Clinical trials must be conducted in accordance with the FDA s Good Clinical Practices requirements. Prior to commencement of each clinical trial, the sponsor must submit to the FDA a clinical plan, or protocol, accompanied by the approval of the ethics committee responsible for overseeing the clinical trial sites. The FDA may order the temporary or permanent discontinuation of a clinical trial at any time if it believes that the clinical trial is not being conducted in accordance with FDA requirements or presents an unacceptable risk to the clinical trial patients. The ethics committee at each clinical site may also require the clinical trial at that site to be halted, either temporarily or permanently, for the same reasons.

The sponsor must submit to the FDA the results of the preclinical and clinical trials, together with, among other things, detailed information on the manufacture and composition of the product, in the form of an NDA, or, in the case of a biologic, a BLA. In a process that may take from several months to several years, the FDA reviews these applications and, when and if it decides that adequate data are available to show that the new compound is both safe and effective and that other applicable requirements have been met, approves the drug or biologic for sale. The amount of time taken for this approval process is a function of a number of variables, including whether the product has received a fast track designation, the quality of the submission and studies presented, the potential contribution that the compound will make in improving the treatment of the disease in question, and the workload at the FDA. It is possible that our product candidates will not successfully proceed through this approval process or that the FDA will not approve them in any specific period of time, or at all.

The FDA may, during its review of a NDA or BLA, ask for additional test data. If the FDA does ultimately approve the product, it may require additional testing, including potentially expensive phase 4 studies, to monitor the safety and effectiveness of the product. In addition, the FDA may in some circumstances impose restrictions on the use of the product, which may be difficult and expensive to administer and may require prior approval of promotional materials.

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We will also be subject to a variety of regulations governing clinical trials and sales of our products outside the United States. Whether or not FDA approval has been obtained, approval of a product by the comparable regulatory authorities of foreign countries and regions must be obtained prior to the commencement of selling the product in those countries. The approval process varies from one regulatory authority to another and the time may be longer or shorter than that required for FDA approval. In the European Union, Canada, and Australia, regulatory requirements and approval processes are similar, in principle, to those in the United States.

Ongoing Regulatory Requirements

Before approving an NDA or BLA, the FDA will inspect the facilities at which the product is manufactured and will not approve the product unless the manufacturing facilities are in compliance with FDA s Good Manufacturing Practices, or GMP, regulations which govern the manufacture, storage and distribution of a product. Manufacturers of biologics also must comply with FDA s general biological product standards. Following approval, the FDA periodically inspects drug and biologic manufacturing facilities to ensure continued compliance with the GMP regulations. Manufacturers must continue to expend time, money and effort in the areas of production, quality control, record keeping and reporting to ensure full compliance with those requirements. Failure to comply with GMP regulations subjects the manufacturer to possible legal or regulatory action, such as suspension of manufacturing or recall or seizure of product. Adverse experiences with the product must be reported to the FDA and could result in the imposition of marketing restrictions through labeling changes or market removal. Product approvals may be withdrawn if compliance with regulatory requirements is not maintained or if problems concerning safety or efficacy of the product occur following approval.

The labeling, advertising, promotion, marketing and distribution of a drug or biologic product also must be in compliance with FDA and Federal Trade Commission, or FTC, requirements which include, among others, standards and regulations for off-label promotion, industry sponsored scientific and educational activities, promotional activities involving the internet, and direct-to-consumer advertising. The FDA and FTC have very broad enforcement authority, and failure to abide by these regulations can result in penalties, including the issuance of a warning letter directing the company to correct deviations from regulatory standards and enforcement actions that can include seizures, injunctions and criminal prosecution.

Manufacturers are also subject to various laws and regulations governing laboratory practices, the experimental use of animals, and the use and disposal of hazardous or potentially hazardous substances in connection with their research. In each of these areas, as above, the FDA has broad regulatory and enforcement powers, including the ability to levy fines and civil penalties, suspend or delay issuance of product approvals, seize or recall products, and deny or withdraw approvals.

Competition

We face competition from a number of companies that are marketing products or evaluating various product candidates, technologies and approaches for the treatment of cancer and inflammatory diseases. Specifically, we face competition from a number of companies working in the fields of antibody-derived therapies for the treatment of solid tumors and B cell lymphomas. We expect that competition among products approved for sale will be based on various factors, including product efficacy, safety, reliability, convenience, availability, pricing and patent position. Some of these products use therapeutic approaches that may compete directly with our product candidates, and the companies developing these competing technologies may have significantly more resources than we do, and may succeed in obtaining approvals from the FDA and foreign regulatory authorities for their products sooner than we do for ours.

Non-Hodgkins Lymphoma

There are numerous cytotoxic/cytostatic agents (chemotherapy) licensed or in development to treat non-Hodgkins lymphoma as single agents or as combination regimens. For most lymphoma indications, consensus recommendations and guidelines, such as those of the National Comprehensive Cancer Network, are available, recommending the preferred treatment regimens according to disease subtype and stage of disease.

In addition to standard chemotherapy regimens, a growing number of targeted therapies have been developed to treat aggressive and indolent NHL. Among those, Rituximab (Genentech/Idec/Zenyaku Kogyo s Rituxan®;

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Roche s MabThera®), a chimeric human-mouse monoclonal antibody active against the CD20 antigen, is the most advanced product in the treatment algorithm of those diseases. The FDA has approved its use for certain stages of aggressive as well as indolent NHL.

In addition, radiolabeled antibodies to CD20 have been developed, including:

Biogen Idec s Ibritumomab tiuxetan (Zevalin®), a murine labeled with yttrium-90 (murine CD20 antibody);

GlaxoSmithKline s tositumomab (Bexxar®), labeled with iodine-131(murine CD20 antibody);

Zevalin® is marketed for the radioimmunotherapy of relapsed/refractory low-grade (indolent) CD20-positive NHL; and

Bexxar® is marketed for the treatment of low-grade (indolent) NHL.

Moreover, fully human antibodies against CD20 are under development by companies such as Genmab and Genentech for various lymphomas, including chronic lymphocytic leukemia. In addition, other monoclonal antibodies against various antigens, such as the anti-CD22 monoclonal antibody epratuzumab (Immunomedics LymphoCide), are under clinical investigation in its naked (unlabeled) and radiolabeled (90 Y) forms.

Additional targeted therapies include small molecules binding to specific targets, such as protein kinases. Among those, imatinib (Gleevec®) probably is the most prominent example, recently gaining label extensions for bcr-abl positive lymphoblastic lymphomas; similarily, other bcr-abl inhibitors (dasatinib, BMS-354825) are being developed for various indications.

Breast Cancer

The treatment of breast cancer employs a multimodal approach**,** using hormone therapy, chemotherapy, biological agents, radiotherapy, and surgery. Treatment selection is tied primarily to disease stage, estrogen and progesterone receptor status, performance status, and, increasingly, HER-2 expression. Hormone therapy and**/**or chemotherapy are given in the following circumstances:

Neoadjuvant therapy (prior to surgery) to reduce tumor size and facilitate surgery;

Adjuvant therapy (postsurgery) to prevent recurrence (both local and distant); and

Palliative treatment of metastatic disease, which is not considered to be curable, where it might be used to prolong survival.

A large number of cytotoxic/cytostatic drugs, whether given alone, in combination, or in sequence, have demonstrated clinical benefit in breast cancer patients and have been adopted into clinical practice. Generally, neoadjuvant and adjuvant chemotherapy uses combinations of drugs each with a different mechanism of action and complementary toxicity profile to maximize efficacy while minimizing toxicity. Palliative treatment of metastatic disease usually but not exclusively employing single-agent chemotherapy intends to ameliorate symptomatic disease and/or to delay the progression of disease. A variety of newly developed chemotherapeutic agents are currently under clinical investigation, including Epothilones (e.g. ixabepilone, KOS-862), Pemetrexed (Alimta), and Temsirolimus, among others.

Hormone-receptor positive disease is usually been treated with Tamoxifen and/or second generation anti-hormonal agents such as aromatase inhibitors, either exclusively or after chemotherapy, depending on stage of primary disease.

Trastuzumab (Roche/Genentech/Chugai s Herceptin®) has been developed as a targeted therapy for HER-2 positive disease and is licensed for treatment of metastatic breast cancer either as monotherapy or in combination with taxanes. Recently, the label was extended to adjuvant treatment of HER-2 positive early breast cancer after various large studies confirmed significant benefit on disease-free survival.

GlaxoSmithKline s lapatinib (Tycerb) is an orally administered EGFR tyrosine kinase inhibitor also blocking ErbB-2/HER-2 tyrosine kinase. Recently, a BLA has been filed by Roche for the combination of Tycerb with capecitabine (Xeloda). Genentech/Roche/Chugai s next-generation HER-2 directed monoclonal antibody,

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pertuzumab (Omnitarg), inhibits HER-2 dimerization and is currently in clinical trials for a range of solid cancers, including breast cancer.

Angiogenesis, the formation of new blood vessels, plays a major role in many normal physiological processes and in several pathological conditions, including solid tumor growth and metastasis. Numerous companies are developing compounds that inhibit angiogenesis. Bevacizumab (Genentech s/Roche/Chugai s Avastin®), a humanized monoclonal antibody designed to inhibit angiogenesis, is approved for marketing in the United States and the European Union for colorectal cancer, non-small-cell lung, and breast cancer. Various other approaches to inhibit neo-vascularisation are under investigation. Examples of agents within this class in early-phase development for breast cancer include:

AstraZeneca s ZD-6474;

EntreMed s 2-methoxyestradiol (2-ME2); and

Bayer/Onyx s sorafenib (BAY-43-9006).

Employees

As of December 31, 2006, we had 79 full-time employees. As of that date 62 full-time employees were engaged in research and development and 17 were engaged in general and administrative activities. We believe that we have good relations with our employees. None of our employees is covered by a collective bargaining agreement.

Available Investor Information

We file electronically with the Securities and Exchange Commission our annual reports on Form 10-K, quarterly interim reports on Form 10-Q, current reports on Form 8-K, and amendments to those reports filed or furnished pursuant to Section 13(a) or 15(d) of the Securities Exchange Act of 1934. We make available on or through our website, free of charge, copies of these reports as soon as reasonably practicable after we electronically file or furnish it to the SEC. Our website is located at http://www.micromet-inc.com. You can also request copies of such documents by contacting our Investor Relations Department at (760) 494-4235 or sending an email to investors@micromet-inc.com.

Item 1A. ***Risk Factors***

The following information sets forth factors that could cause our actual results to differ materially from those contained in forward-looking statements we have made in this report and the information incorporated herein by reference and those we may make from time to time. Certain factors individually or in combination with others may have a material adverse effect on our business, financial condition and results of operations and you should carefully consider them.

Risks Relating to Our Financial Results, Financial Reporting and Need for Financing

We have a history of losses, we expect to incur substantial losses and negative operating cash flows for the foreseeable future and we may never achieve profitability.

We have incurred losses from the inception of Micromet through December 31, 2006, and we expect to incur substantial losses for the foreseeable future. We have no current sources of material ongoing revenue, other than the reimbursement of development expenses and potential future milestone payments from our current collaborators, Merck Serono and MedImmune. We have not commercialized any products to date, either alone or with a third party

collaborator. If we are not able to commercialize any products, whether alone or with a collaborator, we may not achieve profitability. Even if our collaboration agreements provide funding for a portion of our research and development expenses for some of our programs, we expect to spend significant capital to fund our internal research and development programs for the foreseeable future. As a result, we will need to generate significant revenues in order to achieve profitability. We cannot be certain whether or when this will occur because of the significant uncertainties that affect our business. Our failure to become and remain profitable may depress the market price of

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our common stock and could impair our ability to raise capital, expand our business, diversify our product offerings or continue our operations.

We will require additional financing, which may be difficult to obtain and may dilute your ownership interest in us. If we fail to obtain the capital necessary to fund our operations, we will be unable to develop or commercialize our product candidates and our ability to operate as a going concern may be adversely affected.

We will require substantial funds to continue our research and development programs and our future capital requirements may vary from what we expect. There are factors, many of which are outside our control, that may affect our future capital requirements and accelerate our need for additional financing. Among the factors that may affect our future capital requirements and accelerate our need for additional financing are:

- continued progress in our research and development programs, as well as the scope of these programs;
- our ability to establish and maintain collaborative arrangements for the discovery, research or development of product candidates;
- the timing, receipt and amount of research funding and milestone, license, royalty and other payments, if any, from collaborators;
- the timing, receipt and amount of sales revenues and associated royalties to us, if any, from our product candidates in the market;
- our ability to sell shares of our common stock under our committed equity financing facility, or CEFF, with Kingsbridge Capital Limited, or Kingsbridge;
- the costs of preparing, filing, prosecuting, maintaining, defending and enforcing patent claims and other patent-related costs, including litigation costs and technology license fees;
- costs associated with litigation; and
- competing technological and market developments.

We filed a shelf registration statement, declared effective by the Securities and Exchange Commission on December 9, 2004, under which we may raise up to $80 million through the sale of our common stock. This shelf registration statement became inactive in March 2006, and we may decide to activate it by filing a post-effective amendment in the future, although our ability to do so will depend on our eligibility to use a shelf registration statement at such time, under applicable SEC rules. We expect to seek additional funding through public or private financings or from new collaborators with whom we enter into research or development collaborations with respect to programs that are not currently licensed. However, the market for stock of companies in the biotechnology sector in general, and the market for our common stock in particular, is highly volatile. Due to market conditions and the status of our product development pipeline, additional funding may not be available to us on acceptable terms, or at all. Having insufficient funds may require us to delay, scale back or eliminate some or all of our research or development programs or to relinquish greater or all rights to product candidates at an earlier stage of development or on less favorable terms than we would otherwise choose. Failure to obtain adequate financing also may adversely affect our ability to operate as a going concern.

If we raise additional funds through the issuance of equity securities, our stockholders may experience substantial dilution, or the equity securities may have rights, preferences or privileges senior to those of existing stockholders. If

we raise additional funds through debt financings, these financings may involve significant cash payment obligations and covenants that restrict our ability to operate our business and make distributions to our stockholders. We also could elect to seek funds through arrangements with collaborators or others that may require us to relinquish rights to certain technologies, product candidates or products.

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Our committed equity financing facility with Kingsbridge may not be available to us if we elect to make a draw down, may require us to make additional blackout or other payments to Kingsbridge and may result in dilution to our stockholders.

In August 2006, we entered into a CEFF with Kingsbridge. The CEFF entitles us to sell and obligates Kingsbridge to purchase, from time to time over a period of three years, shares of our common stock for cash consideration up to an aggregate of $25 million, subject to certain conditions and restrictions. Kingsbridge will not be obligated to purchase shares under the CEFF unless certain conditions are met, which include:

- a minimum price for our common stock that is not less than 85% of the closing price of the day immediately preceding the applicable eight-day pricing period, but in no event less than $2.00 per share;
- the accuracy of representations and warranties made to Kingsbridge;
- our compliance with all applicable laws which, if we failed to so comply, would have a Material Adverse Effect (as that term is defined in the purchase agreement with Kingsbridge); and
- the effectiveness of a registration statement registering for resale the shares of common stock to be issued in connection with the CEFF.

Kingsbridge is permitted to terminate the CEFF by providing written notice to us upon the occurrence of certain events. If we are unable to access funds through the CEFF, or if Kingsbridge terminates the CEFF, we may be unable to access capital from other sources on favorable terms, or at all.

We are entitled, in certain circumstances, to deliver a blackout notice to Kingsbridge to suspend the use of the resale registration statement and prohibit Kingsbridge from selling shares under the resale registration statement for a certain period of time. If we deliver a blackout notice during the fifteen trading days following our delivery of shares to Kingsbridge in connection with any draw down, then we may be required to make a payment to Kingsbridge, or issue to Kingsbridge additional shares in lieu of this payment, calculated on the basis of the number of shares purchased by Kingsbridge in the most recent draw down and held by Kingsbridge immediately prior to the blackout period and the decline in the market price, if any, of our common stock during the blackout period. If the trading price of our common stock declines during a blackout period, this blackout payment could be significant.

In addition, if we fail to maintain the effectiveness of the resale registration statement or related prospectus in circumstances not permitted by our agreement with Kingsbridge, we may be required to make a payment to Kingsbridge, calculated on the basis of the number of shares held by Kingsbridge during the period that the registration statement or prospectus is not effective, multiplied by the decline in market price, if any, of our common stock during the ineffective period. If the trading price of our common stock declines during a period in which the resale registration statement or related prospectus is not effective, this payment could be significant.

Should we sell shares to Kingsbridge under the CEFF or issue shares in lieu of a blackout payment, it will have a dilutive effect on the holdings of our current stockholders and may result in downward pressure on the price of our common stock. If we draw down under the CEFF, we will issue shares to Kingsbridge at a discount of 6% to 14% from the volume weighted average price of our common stock. If we draw down amounts under the CEFF when our share price is decreasing, we will need to issue more shares to raise the same amount than if our stock price was higher. Issuances in the face of a declining share price will have an even greater dilutive effect than if our share price were stable or increasing and may further decrease our share price. Moreover, the number of shares that we will be able to issue to Kingsbridge in a particular draw down may be materially reduced if our stock price declines

significantly during the applicable eight-day pricing period.

Our quarterly operating results and stock price may fluctuate significantly.

We expect our results of operations to be subject to quarterly fluctuations. The level of our revenues, if any, and results of operations at any given time, will be based primarily on the following factors:

the status of development of our product candidates;

the time at which we enter into research and license agreements with strategic collaborators that provide for payments to us, and the timing and accounting treatment of payments to us, if any, under those agreements;

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whether or not we achieve specified research, development or commercialization milestones under any agreement that we enter into with collaborators and the timely payment by commercial collaborators of any amounts payable to us;

the addition or termination of research programs or funding support;

the timing of milestone payments under license agreements, repayments of outstanding amounts under loan agreements, and other payments that we may be required to make to others; and

variations in the level of research and development expenses related to our product candidates or potential product candidates during any given period.

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

If the estimates we make and the assumptions on which we rely in preparing our financial statements prove inaccurate, our actual results may vary significantly.

Our financial statements have been prepared in accordance with generally accepted accounting principles in the United States. The preparation of these financial statements requires us to make estimates and judgments that affect the reported amounts of our assets, liabilities, revenues and expenses, the amounts of charges taken by us and related disclosure. We base our estimates on historical experience and on various other assumptions that we believe to be reasonable under the circumstances. We cannot assure you that our estimates, or the assumptions underlying them, will be correct. Accordingly, our actual financial results may vary significantly from the estimates contained in our financial statements.

Changes in, or interpretations of, accounting rules and regulations, such as expensing of stock options, could result in unfavorable accounting charges or require us to change our compensation policies.

Accounting methods and policies for biopharmaceutical companies, including policies governing revenue recognition, expenses, accounting for stock options and in-process research and development costs are subject to further review, interpretation and guidance from relevant accounting authorities, including the SEC. Changes to, or interpretations of, accounting methods or policies in the future may require us to reclassify, restate or otherwise change or revise our financial statements, including those contained in this filing.

Our operating and financial flexibility, including our ability to borrow money, is limited by certain debt arrangements.

Our loan agreements contain certain customary events of default, which generally include, among others, non-payment of principal and interest, violation of covenants, cross defaults, the occurrence of a material adverse change in our ability to satisfy our obligations under our loan agreements or with respect to one of our lenders security interest in our assets and in the event we are involved in certain insolvency proceedings. Upon the occurrence of an event of default, our lenders may be entitled to, among other things, accelerate all of our obligations and sell our assets to satisfy our obligations under our loan agreements. In addition, in an event of default, our outstanding obligations may be subject to increased rates of interest.

In addition, we may incur additional indebtedness from time to time to finance acquisitions, investments or strategic alliances or capital expenditures or for other purposes. Our level of indebtedness could have negative consequences for us, including the following:

- our ability to obtain additional financing, if necessary, for working capital, capital expenditures, acquisitions or other purposes may be impaired or such financing may not be available on favorable terms;

- payments on our indebtedness will reduce the funds that would otherwise be available for our operations and future business opportunities;

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we may be more highly leveraged than our competitors, which may place us at a competitive disadvantage; and

our debt level may reduce our flexibility in responding to changing business and economic conditions.

We have determined and further received an opinion from our independent registered public accounting firm in connection with our year-end audit for 2006 that our system of internal control over financial reporting does not meet the requirements of Section 404 of the Sarbanes-Oxley Act of 2002. As a result, investors could lose confidence in the reliability of our internal control over financial reporting, which could have a material adverse effect on our stock price.

As a publicly traded company, we are required to comply with the Sarbanes-Oxley Act of 2002 and the related rules and regulations of the SEC, including Section 404 of the Sarbanes-Oxley Act of 2002. As a result of the merger between CancerVax Corporation and Micromet AG, we are in the process of upgrading the existing, and implementing additional, procedures and controls to incorporate the operations of Micromet AG, which had been a private German company prior to the merger. The process of updating the procedures and controls is requiring significant time and expense. The integration of the two companies finance and accounting systems, procedures and controls, and the implementation of procedures and controls at Micromet AG are more time-consuming and expensive than we previously anticipated.

Our internal control system is designed to provide reasonable assurance to our management and board of directors regarding the preparation and fair presentation of published financial statements. In connection with the audit of our consolidated financial statements for the year ended December 31, 2006, our independent registered public accounting firm provided us with an unqualified opinion on our consolidated financial statements, but it identified material weaknesses in our internal control over financial reporting based on criteria established in Internal Control Integrated Framework, issued by the Committee of Sponsoring Organizations (COSO) of the Treadway Commission. These material weaknesses relate to certain of our estimation and accrual processes, procedures relating to analysis and recording of revenue transactions with unusual terms, and an insufficient level of management review due to lack of resources. These weaknesses resulted, in part, from our inability to sufficiently upgrade our existing procedures and controls and to implement new procedures and controls to integrate the operations of Micromet AG prior to December 31, 2006. Because of these material weaknesses in our internal control over financial reporting, there is heightened risk that a material misstatement of our annual or quarterly financial statements will not be prevented or detected.

We are in the process of expanding our internal resources and implementing additional procedures in order to remediate these material weaknesses in our internal control over financial reporting; however, we cannot guarantee that these efforts will be successful. If we do not adequately remedy these material weaknesses, and if we fail to maintain proper and effective internal control over financial reporting in future periods, our ability to provide timely and reliable financial results could suffer, and investors could lose confidence in our reported financial information, which may have a material adverse effect on our stock price.

Risks Relating to Our Common Stock

Substantial sales of shares may adversely impact the market price of our common stock and our ability to issue and sell shares in the future.

Substantially all of the outstanding shares of our common stock are eligible for resale in the public market and certain holders of our shares have the right to require us to file a registration statement for purposes of registering their shares for resale. A significant portion of these shares is held by a small number of stockholders. We have also registered

shares of our common stock that we may issue under our equity incentive compensation plans and our employee stock purchase plan. These shares generally can be freely sold in the public market upon issuance. If our stockholders sell substantial amounts of our common stock, the market price of our common stock may decline, which might make it more difficult for us to sell equity or equity-related securities in the future at a time and price that we deem appropriate. We are unable to predict the effect that sales of our common stock may have on the prevailing market price of our common stock.

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Our stock price may be volatile, and you may lose all or a substantial part of your investment.

The market price for our common stock is volatile and may fluctuate significantly in response to a number of factors, a number of which we cannot control. Among the factors that could cause material fluctuations in the market price for our common stock are:

our ability to upgrade and implement our disclosure controls and our internal control over financial reporting;

our ability to successfully raise capital to fund our continued operations;

our ability to successfully develop our product candidates within acceptable timeframes;

changes in the regulatory status of our product candidates;

changes in significant contracts, new technologies, acquisitions, commercial relationships, joint ventures or capital commitments;

the execution of new contracts or termination of existing contracts related to our clinical or preclinical product candidates or our BiTE technology platform;

announcements of the invalidity of, or litigation relating to, our key intellectual property;

announcements of the achievement of milestones in our agreements with collaborators or the receipt of payments under those agreements;

announcements of the results of clinical trials by us or by companies with product candidates in the same therapeutic category as our product candidates;

events affecting our collaborators;

fluctuations in stock market prices and trading volumes of similar companies;

announcements of new products or technologies, clinical trial results, commercial relationships or other events by us, our collaborators or our competitors;

our ability to successfully complete sublicensing arrangements with respect to our product candidates that target the EGFR signaling pathway, denatured collagen, GM-CSF and interleukin-2;

variations in our quarterly operating results;

changes in securities analysts estimates of our financial performance or product development timelines;

changes in accounting principles;

sales of large blocks of our common stock, including sales by our executive officers, directors and significant stockholders;

additions or departures of key personnel; and

discussions of Micromet or our stock price by the financial and scientific press and online investor communities such as chat rooms.

If our officers and directors choose to act together, they can significantly influence our management and operations in a manner that may be in their best interests and not in the best interests of other stockholders.

Our officers and directors, together with their affiliates, collectively own an aggregate of approximately 33% of our outstanding common stock, and, as a result, may significantly influence all matters requiring approval by our stockholders, including the election of directors and the approval of mergers or other business combination transactions. The interests of this group of stockholders may not always coincide with our interests or the interests of other stockholders, and this group may act in a manner that advances their best interests and not necessarily those of other stockholders.

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Our stockholder rights plan, anti-takeover provisions in our organizational documents and Delaware law may discourage or prevent a change in control, even if an acquisition would be beneficial to our stockholders, which could affect our stock price adversely and prevent attempts by our stockholders to replace or remove our current management.

Our stockholder rights plan and provisions contained in our amended and restated certificate of incorporation and amended and restated bylaws may delay or prevent a change in control, discourage bids at a premium over the market price of our common stock and adversely affect the market price of our common stock and the voting and other rights of the holders of our common stock. The provisions in our amended and restated certificate of incorporation and amended and restated bylaws include:

dividing our board of directors into three classes serving staggered three-year terms;

prohibiting our stockholders from calling a special meeting of stockholders;

permitting the issuance of additional shares of our common stock or preferred stock without stockholder approval;

prohibiting our stockholders from making certain changes to our amended and restated certificate of incorporation or amended and restated bylaws except with 662/3% stockholder approval; and

requiring advance notice for raising matters of business or making nominations at stockholders meetings.

We are also subject to provisions of the Delaware corporation law that, in general, prohibit any business combination with a beneficial owner of 15% or more of our common stock for five years unless the holder s acquisition of our stock was approved in advance by our board of directors.

We may become involved in securities class action litigation that could divert management s attention and harm our business and our insurance coverage may not be sufficient to cover all costs and damages.

The stock market has from time to time experienced significant price and volume fluctuations that have affected the market prices for the common stock of pharmaceutical and biotechnology companies. These broad market fluctuations may cause the market price of our common stock to decline. In the past, following periods of volatility in the market price of a particular company s securities, securities class action litigation has often been brought against that company. We may become involved in this type of litigation in the future. Litigation often is expensive and diverts management s attention and resources, which could adversely affect our business.

Risks Relating to Our Collaborations and Clinical Programs

We are dependent on collaborators for the development and commercialization of many of our product candidates. If we lose any of these collaborators, or if they fail or incur delays in the development or commercialization of our current and future product candidates, our operating results would suffer.

The success of our strategy for development and commercialization of our product candidates depends upon our ability to form and maintain productive strategic collaborations. We currently have strategic collaborations with Merck Serono and MedImmune. We expect to enter into additional collaborations in the future. Our existing and any future collaborations may not be scientifically or commercially successful. The risks that we face in connection with these collaborations include the following:

Each of our collaborators has significant discretion in determining the efforts and resources that it will apply to the collaboration. The timing and amount of any future royalty and milestone revenue that we may receive under such collaborative arrangements will depend on, among other things, such collaborator s efforts and allocation of resources.

All of our strategic collaboration agreements are for fixed terms and are subject to termination under various circumstances, including, in some cases, on short notice without cause. If either Merck Serono or MedImmune were to terminate its agreement with us, we may attempt to identify and enter into an agreement with a new collaborator with respect to the product candidate covered by the terminated agreement. If we are not able to do so, we may not have the funds or capability to undertake the

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development, manufacturing and commercialization of that product candidate, which could result in a discontinuation or delay of the development of that product candidate.

Our collaborators may develop and commercialize, either alone or with others, products and services that are similar to or competitive with the product candidates and services that are the subject of their collaborations with us.

Our collaborators may discontinue the development of our product candidates in specific indications, for example as a result of their assessment of the results obtained in clinical trials, or fail to initiate the development in indications that have a significant commercial potential.

Pharmaceutical and biotechnology companies from time to time re-evaluate their research and development priorities, including in connection with mergers and consolidations, which have been common in recent years in these industries. The ability of our partnered product candidates to reach their potential could be limited if, as a result of such changes, our collaborators decrease or fail to increase spending related to such product candidates, or decide to discontinue the development of our product candidates and terminate their collaboration agreement with us.

We may not be successful in establishing additional strategic collaborations, which could adversely affect our ability to develop and commercialize product candidates.

As an integral part of our ongoing research and development efforts, we periodically review opportunities to establish new collaborations for development and commercialization of new BiTE molecules or existing product candidates in our development pipeline. We face significant competition in seeking appropriate collaborators and the negotiation process is time-consuming and complex. We may not be successful in our efforts to establish additional collaborations or other alternative arrangements. Even if we are successful in our efforts to establish a collaboration, the terms of the agreement may not be favorable to us. Finally, such collaborations or other arrangements may not result in successful products and associated revenue from milestone payments, royalties or profit share payments.

If the combination of adecatumumab (MT201) with cytotoxics, such as docetaxel, is not tolerable or safe, if higher serum levels of adecatumuab cannot be administered safely, or if sufficient anti-tumor activity cannot be shown, we and our collaborator Merck Serono may decide to abandon all or part of the development program, and we could experience a material adverse impact on our results of operations.

We previously have reported that the phase 2 clinical trials of adecatumumab did not reach their respective primary endpoint in patients with metastatic breast cancer (clinical benefit rate at week 24) and in patients with prostate cancer (mean change in prostate specific antigen, compared to placebo control). We have also reported that we are continuing the development of adecatumumab in a clinical trial in combination with docetaxel with escalating doses of adecatumumab to investigate the tolerability and the safety of this combination. We have also reported that we, in collaboration with Merck Serono, are planning to start a new phase 1 monotherapy study for the treatment of patients with solid tumors estimated to begin in 2007. If the combination of adecatumumab with docetaxel proves not to be tolerable or safe or if no higher serum levels of adecatumuab compared to previous clinical trials can be administered safely or if sufficient anti-tumor activity cannot be shown, we and our collaborator Merck Serono may decide to abandon all or part of the development program of adecatumumab and as a result we may experience a material adverse impact on our results of operations.

We previously terminated three phase 1 trials involving short-term infusion regimens of MT103 due to adverse side effects and a lack of perceived tumor response, and there can be no assurance that our current continuous infusion phase 1 clinical trial of MT103 will produce a different outcome.

In April 2004, we initiated a phase 1, dose finding clinical trial designed to evaluate the safety and tolerability of a continuous intravenous infusion of MT103 over 4-8 weeks at different dose levels in patients with relapsed non-Hodgkin s lymphoma. We previously terminated three other phase 1 clinical trials for MT103, which involved a short-term infusion, as opposed to a continuous infusion dosing regimen of MT103, due to adverse side effects and the lack of observed tumor responses. We have redesigned the dosing regimen for our ongoing phase 1 clinical trial

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and, based upon the preliminary clinical data, we currently are seeing a considerably more favorable safety profile in response to the new continuous infusion dosing regimen. We have also seen objective tumor responses at the highest dose level tested (15 µg/m2/d) with the continuous infusion regimens. While this preliminary data suggest that MT103 has anti-tumor activity, there can be no assurance that we will not encounter unacceptable adverse events during the continued dose escalation of our ongoing, continuous-infusion phase 1 clinical trial or that the preliminary suggestion of anti-tumor activity will be confirmed during the ongoing or any future study.

Changes in the laws or regulations of the United States or Cuba related to the conduct of our business with CIMAB may adversely affect our ability to sublicense or otherwise transfer our rights to SAI-EGF and our two other product candidates that we have licensed from that company.

The United States government has maintained an embargo against Cuba for more than 40 years. The embargo is administered by the Office of Foreign Assets Control, or OFAC, of the U.S. Department of Treasury. Without a license from OFAC, U.S. individuals and companies may not engage in any transaction in which Cuba or Cubans have an interest. In order to enter into and carry out our licensing agreements with CIMAB S.A., a Cuban company, we have obtained from OFAC a license authorizing us to carry out all transactions set forth in the license agreements that we have entered into with CIMAB for the development, testing, licensing and commercialization of our product candidate SAI-EGF, and with CIMAB and its affiliate YM BioSciences, Inc., a Canadian company, for our two other product candidates that target the EGF receptor signaling pathway. In the absence of such a license from OFAC, the execution of and our performance under these agreements could have exposed us to legal and criminal liability. At any time, there may occur for reasons beyond our control a change in United States or Cuban law, or in the regulatory environment in the U.S. or Cuba, or a shift in the political attitudes of either the U.S. or Cuban governments, that could result in the suspension or revocation of our OFAC license or in our inability to carry out part or all of the licensing agreements with CIMAB. There can be no assurance that the U.S. or Cuban governments will not modify existing law or establish new laws or regulations that may adversely affect our ability to develop, test, license and commercialize these product candidates. Our OFAC license may be revoked or amended at anytime in the future, or the U.S. or Cuban governments may restrict our ability to carry out all or part of our respective duties under the licensing agreements between us, CIMAB and YM BioSciences. Similarly, any such actions may restrict CIMAB s ability to carry out all or part of its licensing agreements with us. In addition, we cannot be sure that the FDA, EMEA or other regulatory authorities will accept data from the clinical trials of these product candidates that were conducted in Cuba as the basis for our applications to conduct additional clinical trials, or as part of our application to seek marketing authorizations for such product candidates.

In 1996, a significant change to the United States embargo against Cuba resulted from congressional passage of the Cuban Liberty and Democratic Solidarity Act, also known as the Helms-Burton Bill. That law authorizes private lawsuits for damages against anyone who traffics in property confiscated, without compensation, by the government of Cuba from persons who at the time were, or have since become, nationals of the United States. We do not own any property in Cuba and do not believe that any of CIMAB s properties or any of the scientific centers that are or have been involved in the development of the technology that we have licensed from CIMAB were confiscated by the government of Cuba from persons who at the time were, or who have since become, nationals of the U.S. However, there can be no assurance that our understanding in this regard is correct. We do not intend to traffic in confiscated property, and we have included provisions in our licensing agreements to preclude the use of such property in association with the performance of CIMAB s obligations under those agreements, although we cannot ensure that CIMAB or other third parties will comply with these provisions.

As part of our interactions with CIMAB, we are subject to the U.S. Commerce Department s export administration regulations that govern the transfer of technology to foreign nationals. Specifically, we or our sublicensees, if any, will require a license from the Commerce Department s Bureau of Industry and Security, or BIS, in order to export or otherwise transfer to CIMAB any information that constitutes technology under the definitions of the Export

Administration Regulations, or EAR, administered by BIS. The export licensing process may take months to be completed, and the technology transfer in question may not take place unless and until a license is granted by the Commerce Department. Due to the unique status of the Republic of Cuba, technology that might otherwise be transferable to a foreign national without a Commerce Department license requires a license for export or transfer to a Cuban national. If we or our sublicensees fail to comply with the export administration

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regulations, we may be subject to both civil and criminal penalties. There can be no guarantee that any license application will be approved by BIS or that a license, once issued, will not be revoked, modified, suspended or otherwise restricted for reasons beyond our control due to a change in U.S.-Cuba policy or for other reasons.

Risks Relating to Our Operations, Business Strategy, and the Life Sciences Industry

We face substantial competition, which may result in our competitors discovering, developing or commercializing products before or more successfully than we do.

Our product candidates face competition with existing and new products being developed by biotechnology and pharmaceutical companies, as well as universities and other research institutions. For example, research in the fields of antibody-based therapeutics for the treatment of cancer, and autoimmune and inflammatory diseases, is highly competitive. A number of entities are seeking to identify and patent antibodies, potentially active proteins and other potentially active compounds without specific knowledge of their therapeutic functions. Our competitors may discover, characterize and develop important inducing molecules or genes in advance of us.

Many of our competitors have substantially greater capital resources, research and development staffs and facilities than we have. Efforts by other biotechnology and pharmaceutical companies could render our programs or product candidates uneconomical or result in therapies that are superior to those that we are developing alone or with a collaborator. We and our collaborators face competition from companies that may be more experienced in product development and commercialization, obtaining regulatory approvals and product manufacturing. As a result, they may develop competing products more rapidly and at a lower cost, or may discover, develop and commercialize products, which render our product candidates non-competitive or obsolete. We expect competition to intensify in antibody research as technical advances in the field are made and become more widely known.

We may not be successful in our efforts to expand our portfolio of product candidates.

A key element of our strategy is to discover, develop and commercialize a portfolio of new drugs. We are seeking to do so through our internal research programs and in-licensing activities. A significant portion of the research that we are conducting involves new and unproven technologies. Research programs to identify new disease targets and product candidates require substantial technical, financial and human resources regardless of whether or not any suitable candidates are ultimately identified. Our research programs may initially show promise in identifying potential product candidates, yet fail to yield product candidates suitable for clinical development. If we are unable to discover suitable potential product candidates, develop additional delivery technologies through internal research programs or in-license suitable product candidates or delivery technologies on acceptable business terms, our business prospects will suffer.

The product candidates in our pipeline are in early stages of development and our efforts to develop and commercialize these product candidates are subject to a high risk of delay and failure. If we fail to successfully develop our product candidates, our ability to generate revenues will be substantially impaired.

The process of successfully developing product candidates for the treatment of human diseases is very time-consuming, expensive and unpredictable and there is a high rate of failure for product candidates in preclinical development and in clinical trials. The preclinical studies and clinical trials may produce negative, inconsistent or inconclusive results, and the results from early clinical trials may not be statistically significant or predictive of results that will be obtained from expanded, advanced clinical trials. Further, we or our collaborators may decide, or regulators may require us, to conduct preclinical studies or clinical trials in addition to those planned by us or our collaborators, which may be expensive or could delay the time to market for our product candidates. In addition, we do not know whether the clinical trials will result in marketable products.

All of our product candidates are in early stages of development, so we will require substantial additional financial resources, as well as research, product development and clinical development capabilities, to pursue the development of these product candidates, and we may never develop an approvable or commercially viable product.

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We do not know whether our planned preclinical development or clinical trials for our product candidates will begin on time or be completed on schedule, if at all. The timing and completion of clinical trials of our product candidates depend on, among other factors, the number of patients that will be required to enroll in the clinical trials, the inclusion and exclusion criteria used for selecting patients for a particular clinical trial, and the rate at which those patients are enrolled. Any increase in the required number of patients, tightening of selection criteria, or decrease in recruitment rates or difficulties retaining study participants may result in increased costs, delays in the development of the product candidate, or both.

Since our product candidates may have different efficacy profiles in certain clinical indications, sub-indications or patient profiles, an election by us or our collaborators to focus on a particular indication, sub-indication or patient profile may result in a failure to capitalize on other potentially profitable applications of our product candidates.

Our product candidates may not be effective in treating any of our targeted diseases or may prove to have undesirable or unintended side effects, toxicities or other characteristics that may prevent or limit their commercial use. Institutional review boards or regulators, including the FDA and the EMEA, may hold, suspend or terminate our clinical research or the clinical trials of our product candidates for various reasons, including non-compliance with regulatory requirements or if, in their opinion, the participating subjects are being exposed to unacceptable health risks, or if additional information may be required for the regulatory authority to assess the proposed development activities. Further, regulators may not approve study protocols at all or in a timeframe anticipated by us if they believe that the study design or the mechanism of action of our product candidates poses an unacceptable health risk to study participants.

We have limited financial and managerial resources. These limitations require us to focus on a select group of product candidates in specific therapeutic areas and to forego the exploration of other product opportunities. While our technologies may permit us to work in multiple areas, resource commitments may require trade-offs resulting in delays in the development of certain programs or research areas, which may place us at a competitive disadvantage. Our decisions as to resource allocation may not lead to the development of viable commercial products and may divert resources away from other market opportunities, which would otherwise have ultimately proved to be more profitable.

We rely heavily on third parties for the conduct of preclinical and clinical studies of our product candidates, and we may not be able to control the proper performance of the studies or trials.

In order to obtain regulatory approval for the commercial sale of our product candidates, we and our collaborators are required to complete extensive preclinical studies as well as clinical trials in humans to demonstrate to the FDA, EMEA and other regulatory authorities that our product candidates are safe and effective. We have limited experience and internal resources for conducting certain preclinical studies and clinical trials and rely primarily on collaborators and contract research organizations for the performance and management of certain preclinical studies and clinical trials of our product candidates. If our collaborators or contractors fail to properly perform their contractual or regulatory obligations with respect to conducting or overseeing the performance of our preclinical studies or clinical trials, the completion of these studies or trials may be delayed, or the results may not be useable and the studies or trials may have to be repeated. Any of these events could delay or create additional costs in the development of our product candidates and could adversely affect our and our collaborators ability to market a product after marketing approvals have been obtained.

Even if we complete the lengthy, complex and expensive development process, there is no assurance that we or our collaborators will obtain the regulatory approvals necessary for the launch and commercialization of our product candidates.

To the extent that we or our collaborators are able to successfully complete the clinical development of a product candidate, we or our collaborators will be required to obtain approval by the FDA, EMEA or other regulatory authorities prior to marketing and selling such product candidate in the United States, the European Union or other countries.

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The process of preparing and filing applications for regulatory approvals with the FDA, EMEA and other regulatory authorities, and of obtaining the required regulatory approvals from these regulatory authorities is lengthy and expensive, and may require two years or more. This process is further complicated because some of our product candidates use non-traditional or novel materials in non-traditional or novel ways, and the regulatory officials have little precedent to follow. Moreover, an unrelated biotech company recently observed multiple severe adverse reactions in a phase 1 trial of an antibody that stimulates T cells. This development could cause the FDA and EMEA or other regulatory authorities to require additional preclinical data or certain precautions in the designs of clinical protocols that could cause a delay in the development of our BiTE product candidates or make the development process more expensive.

Any marketing approval by the FDA, EMEA or other regulatory authorities may be subject to limitations on the indicated uses for which we or our collaborators may market the product candidate. These limitations could restrict the size of the market for the product and affect reimbursement levels by third-party payers.

As a result of these factors, we or our collaborators may not successfully begin or complete clinical trials and launch and commercialize any product candidates in the time periods estimated, if at all. Moreover, if we or our collaborators incur costs and delays in development programs or fail to successfully develop and commercialize products based upon our technologies, we may not become profitable and our stock price could decline.

We and our collaborators are subject to governmental regulations other than those imposed by the FDA and EMEA, and we or our collaborators may not be able to comply with these regulations. Any non-compliance could subject us or our collaborators to penalties and otherwise result in the limitation of our or our collaborators operations.

In addition to regulations imposed by the FDA, EMEA and other health regulatory authorities, we and our collaborators are subject to regulation under the Occupational Safety and Health Act, the Environmental Protection Act, the Toxic Substances Control Act, the Research Conservation and Recovery Act, as well as regulations administered by the Nuclear Regulatory Commission, national restrictions on technology transfer, import, export and customs regulations and certain other local, state or federal regulations, or their counterparts in Europe and other countries. From time to time, other governmental agencies and legislative or international governmental bodies have indicated an interest in implementing further regulation of biotechnology applications. We are not able to predict whether any such regulations will be adopted or whether, if adopted, such regulations will apply to our or our collaborators business, or whether we or our collaborators would be able to comply, without incurring unreasonable expense, or at all, with any applicable regulations.

Our growth could be limited if we are unable to attract and retain key personnel and consultants.

We have limited experience in filing and prosecuting regulatory applications to obtain marketing approval from the FDA, EMEA or other regulatory authorities. Our success depends on the ability to attract, train and retain qualified scientific and technical personnel, including consultants, to further our research and development efforts. The loss of services of one or more of our key employees or consultants could have a negative impact on our business and operating results. Locating candidates with the appropriate qualifications can be difficult, and we may not be able to attract and retain sufficient numbers of highly skilled employees.

Any growth and expansion into areas and activities that may require additional personnel or expertise, such as in regulatory affairs and compliance, would require us to either hire new key personnel or obtain such services from a third party. The pool of personnel with the skills that we require is limited, and we may not be able to hire or contract such additional personnel.

If our third-party manufacturers do not follow current good manufacturing practices or do not maintain their facilities in accordance with these practices, our product development and commercialization efforts may be harmed.

Product candidates used in clinical trials or sold after marketing approval has been obtained must be manufactured in accordance with current good manufacturing practices regulations. There are a limited number of manufacturers that operate under these regulations, including the FDA s and EMEA s good manufacturing

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practices regulations, and that are capable of manufacturing our product candidates. Third-party manufacturers may encounter difficulties in achieving quality control and quality assurance and may experience shortages of qualified personnel. Also, manufacturing facilities are subject to ongoing periodic, unannounced inspection by the FDA, the EMEA, and other regulatory agencies or authorities, to ensure strict compliance with current good manufacturing practices and other governmental regulations and standards. A failure of third-party manufacturers to follow current good manufacturing practices or other regulatory requirements and to document their adherence to such practices may lead to significant delays in the availability of product candidates for use in a clinical trial or for commercial sale, the termination of, or hold on a clinical trial, or may delay or prevent filing or approval of marketing applications for our product candidates. In addition, as a result of such a failure, we could be subject to sanctions, including fines, injunctions and civil penalties, refusal or delays by regulatory authorities to grant marketing approval of our product candidates, suspension or withdrawal of marketing approvals, seizures or recalls of product candidates, operating restrictions and criminal prosecutions, any of which could significantly and adversely affect our business. If we were required to change manufacturers, it may require additional clinical trials and the revalidation of the manufacturing process and procedures in accordance with applicable current good manufacturing practices and may require FDA or EMEA approval. This revalidation may be costly and time-consuming. If we are unable to arrange for third-party manufacturing of our product candidates, or to do so on commercially reasonable terms, we may not be able to complete development or marketing of our product candidates.

Even if regulatory authorities approve our product candidates, we may fail to comply with ongoing regulatory requirements or experience unanticipated problems with our product candidates, and these product candidates could be subject to restrictions or withdrawal from the market following approval.

Any product candidates for which we obtain marketing approval, along with the manufacturing processes, post-approval clinical trials and promotional activities for such product candidates, will be subject to continual review and periodic inspections by the FDA, EMEA and other regulatory authorities. Even if regulatory approval of a product candidate is granted, the approval may be subject to limitations on the indicated uses for which the product may be marketed or contain requirements for costly post-marketing testing and surveillance to monitor the safety or efficacy of the product. Post-approval discovery of previously unknown problems with any approved products, including unanticipated adverse events or adverse events of unanticipated severity or frequency, difficulties with a manufacturer or manufacturing processes, or failure to comply with regulatory requirements, may result in restrictions on such approved products or manufacturing processes, withdrawal of the approved products from the market, voluntary or mandatory recall, fines, suspension of regulatory approvals, product seizures, injunctions or the imposition of civil or criminal penalties.

The procedures and requirements for granting marketing approvals vary among countries, which may cause us to incur additional costs or delays or may prevent us from obtaining marketing approvals in different countries and regulatory jurisdictions.

We intend to market our product candidates in many countries and regulatory jurisdictions. In order to market our product candidates in the United States, the European Union and many other jurisdictions, we must obtain separate regulatory approvals in each of these countries and territories. The procedures and requirements for obtaining marketing approval vary among countries and regulatory jurisdictions, and can involve additional clinical trials or other tests. Also, the time required to obtain approval may differ from that required to obtain FDA and EMEA approval. The various regulatory approval processes may include all of the risks associated with obtaining FDA and EMEA approval. We may not obtain all of the desirable or necessary regulatory approvals on a timely basis, if at all. Approval by a regulatory authority in a particular country or regulatory jurisdiction, such as the FDA in the United States and the EMEA in the European Union, generally does not ensure approval by a regulatory authority in another country. We may not be able to file for regulatory approvals and may not receive necessary approvals to commercialize our product candidates in any or all of the countries or regulatory jurisdictions in which we desire to

market our product candidates.

If we fail to obtain an adequate level of reimbursement for any approved products by third-party payers, there may be no commercially viable markets for these products or the markets may be much smaller

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than expected. The continuing efforts of the government, insurance companies, managed care organizations and other payers of health care costs to contain or reduce costs of healthcare may adversely affect our ability to generate revenues and achieve profitability, the future revenues and profitability of our potential customers, suppliers and collaborators, and the availability of capital.

Our ability to commercialize our product candidates successfully will depend in part on the extent to which governmental authorities, private health insurers and other organizations establish appropriate reimbursement levels for the price charged for our product candidates and related treatments. The efficacy, safety and cost-effectiveness of our product candidates as well as the efficacy, safety and cost-effectiveness of any competing products will determine in part the availability and level of reimbursement. These third-party payors continually attempt to contain or reduce the costs of healthcare by challenging the prices charged for healthcare products and services. Given recent federal and state government initiatives directed at lowering the total cost of healthcare in the United States, the U.S. Congress and state legislatures will likely continue to focus on healthcare reform, the cost of prescription pharmaceuticals and on the reform of the Medicare and Medicaid systems. In certain countries, particularly the countries of the European Union, the pricing of prescription pharmaceuticals is subject to governmental control. In these countries, pricing negotiations with governmental authorities can take six to twelve months or longer after the receipt of regulatory marketing approval for a product. To obtain reimbursement or pricing approval in some countries, we may be required to conduct clinical trials that compare the cost-effectiveness of our product candidates to other available therapies. If reimbursement for our product candidates were unavailable or limited in scope or amount or if reimbursement levels or prices are set at unsatisfactory levels, our projected and actual revenues and our prospects for profitability would be negatively affected.

Another development that may affect the pricing of drugs in the United States is regulatory action regarding drug reimportation into the United States. The Medicare Prescription Drug, Improvement and Modernization Act of 2003, which became law in December 2003, requires the Secretary of the U.S. Department of Health and Human Services to promulgate regulations allowing drug reimportation from Canada into the United States under certain circumstances. These provisions will become effective only if the Secretary certifies that such imports will pose no additional risk to the public s health and safety and result in significant cost savings to consumers. Proponents of drug reimportation may also attempt to pass legislation that would remove the requirement for the Secretary s certification or allow reimportation under circumstances beyond those anticipated under current law. If legislation is enacted, or regulations issued, allowing the reimportation of drugs, it could decrease the reimbursement we would receive for any product candidates that we may commercialize, or require us to lower the price of our product candidates then on the market that face competition from lower-priced supplies of that product from other countries. These factors would negatively affect our projected and actual revenues and our prospects for profitability.

If physicians and patients do not accept the product candidates that we may develop, our ability to generate product revenue in the future will be adversely affected.

Our product candidates, if successfully developed and approved by the regulatory authorities, may not gain market acceptance among physicians, healthcare payers, patients and the medical community. Market acceptance of and demand for any product candidate that we may develop will depend on many factors, including:

- our ability to provide acceptable evidence of safety and efficacy;
- convenience and ease of administration;
- prevalence and severity of adverse side effects;
- availability of alternative treatments;

cost effectiveness;

effectiveness of our marketing and pricing strategy for any product candidates that we may develop;

publicity concerning our product candidates or competitive products; and

our ability to obtain third-party coverage or reimbursement.

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If any product candidates for which we may receive marketing approval fail to gain market acceptance, our ability to generate product revenue in the future will be adversely affected.

We face the risk of product liability claims and may not be able to obtain insurance.

Our business exposes us to the risk of product liability claims that is inherent in the testing, manufacturing, and marketing of drugs and related devices. Although we have product liability and clinical trial liability insurance that we believe is appropriate, this insurance is subject to deductibles and coverage limitations. We may not be able to obtain or maintain adequate protection against potential liabilities. If any of our product candidates are approved for marketing, we may seek additional insurance coverage. If we are unable to obtain insurance at acceptable cost or on acceptable terms with adequate coverage or otherwise protect ourselves against potential product liability claims, we will be exposed to significant liabilities, which may harm our business. These liabilities could prevent or interfere with our product commercialization efforts. Defending a suit, regardless of merit, could be costly, could divert management attention and might result in adverse publicity or reduced acceptance of our product candidates in the market.

Our operations involve hazardous materials and we must comply with environmental laws and regulations, which can be expensive.

Our research and development activities involve the controlled use of hazardous materials, including chemicals and radioactive and biological materials. Our operations also produce hazardous waste products. We are subject in the United States to a variety of federal, state and local regulations, and in Europe to European, national, state and local regulations, relating to the use, handling, storage and disposal of these materials. We generally contract with third parties for the disposal of such substances and store certain low-level radioactive waste at our facility until the materials are no longer considered radioactive. We cannot eliminate the risk of accidental contamination or injury from these materials. We may be required to incur substantial costs to comply with current or future environmental and safety regulations. If an accident or contamination occurred, we would likely incur significant costs associated with civil penalties or criminal fines and in complying with environmental laws and regulations. We do not have any insurance for liabilities arising from hazardous materials. Compliance with environmental laws and regulations is expensive, and current or future environmental regulation may impair our research, development or production efforts.

Risks Relating to Our Intellectual Property and Litigation

We may not be able to obtain or maintain adequate patents and other intellectual property rights to protect our business and product candidates against competitors.

Our value will be significantly enhanced if we are able to obtain adequate patents and other intellectual property rights to protect our business and product candidates against competitors. For that reason, we allocate significant financial and personnel resources to the filing, prosecution, maintenance and defense of patent applications, patents and trademarks claiming or covering our product candidates and key technology relating to these product candidates.

To date, we have sought to protect our proprietary positions related to our important proprietary technology, inventions and improvements by filing of patent applications in the U.S., Europe and other jurisdictions. Because the patent position of pharmaceutical and biopharmaceutical companies involves complex legal and factual questions, the issuance, scope and enforceability of patents cannot be predicted with certainty, and we cannot be certain that patents will be issued on pending or future patent applications that cover our product candidates and technologies. Claims could be restricted in prosecution that might lead to a scope of protection which is of minor value for a particular product candidate. Patents, if issued, may be challenged and sought to be invalidated by third parties in litigation. In addition, U.S. patents and patent applications may also be subject to interference proceedings, and U.S. patents may

be subject to reexamination proceedings in the U.S. Patent and Trademark Office. European patents may be subject to opposition proceedings in the European Patent Office. Patents might be invalidated in national jurisdictions. Similar proceedings may be available in countries outside of Europe or the U.S. These proceedings could result in either a loss of the patent or a denial of the patent application or loss or

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reduction in the scope of one or more of the claims of the patent or patent application. Thus, any patents that we own or license from others may not provide any protection against competitors. Furthermore, an adverse decision in an interference proceeding could result in a third party receiving the patent rights sought by us, which in turn could affect our ability to market a potential product or product candidate to which that patent filing was directed. Our pending patent applications, those that we may file in the future, or those that we may license from third parties may not result in patents being issued. If issued, they may not provide us with proprietary protection or competitive advantages against competitors with similar technology. Furthermore, others may independently develop similar technologies or duplicate any technology that we have developed, which fall outside the scope of our patents. Products or technology could also be copied by competitors after expiration of the patent life.

We rely on third-party payment services for the payment of foreign patent annuities and other fees. Non-payment or delay in payment of such fees, whether intentional or unintentional, may result in loss of patents or patent rights important to our business.

We may incur substantial costs enforcing our patents against third parties. If we are unable to protect our intellectual property rights, our competitors may develop and market products with similar features that may reduce demand for our potential products.

We own or control a substantial portfolio of issued patents. From time to time, we may become aware of third parties that undertake activities that infringe on our patents. We may decide to grant those third parties a license under our patents, or to enforce the patents against those third parties by pursuing an infringement claim in litigation. If we initiate patent infringement litigation, it could consume significant financial and management resources, regardless of the merit of the claims or the outcome of the litigation. The outcome of patent litigation is subject to uncertainties that cannot be adequately quantified in advance, including the demeanor and credibility of witnesses and the identity of the adverse party, especially in biotechnology-related patent cases that may turn on the testimony of experts as to technical facts upon which experts may reasonably disagree. Some of our competitors may be able to sustain the costs of such litigation or proceedings more effectively than we can because of their substantially greater financial resources. Uncertainties resulting from the initiation and continuation of patent litigation or other proceedings could harm our ability to compete in the marketplace.

Our ability to enforce our patents may be restricted under applicable law. Many countries, including certain countries in Europe, have compulsory licensing laws under which a patent owner may be compelled to grant licenses to third parties. For example, compulsory licenses may be required in cases where the patent owner has failed to work the invention in that country, or the third-party has patented improvements. In addition, many countries limit the enforceability of patents against government agencies or government contractors. In these countries, the patent owner may have limited remedies, which could materially diminish the value of the patent. Moreover, the legal systems of certain countries, particularly certain developing countries, do not favor the aggressive enforcement of patent and other intellectual property rights, which makes it difficult to stop infringement. In addition, our ability to enforce our patent rights depends on our ability to detect infringement. It is difficult to detect infringers who do not advertise the compounds that are used in their products or the methods they use in the research and development of their products. If we are unable to enforce our patents against infringers, it could have a material adverse effect on our competitive position, results of operations and financial condition.

If we are not able to protect and control our unpatented trade secrets, know-how and other technological innovation, we may suffer competitive harm.

We rely on proprietary trade secrets and unpatented know-how to protect our research, development and manufacturing activities and maintain our competitive position, particularly when we do not believe that patent protection is appropriate or available. However, trade secrets are difficult to protect. We attempt to protect our trade

secrets and unpatented know-how by requiring our employees, consultants and advisors to execute confidentiality and non-use agreements. We cannot guarantee that these agreements will provide meaningful protection, that these agreements will not be breached, that we will have an adequate remedy for any such breach, or that our trade secrets or proprietary know-how will not otherwise become known or independently developed by a third party. Our trade secrets, and those of our present or future collaborators that we utilize by agreement, may become known or may be independently discovered by others, which could adversely affect the competitive position of our product

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candidates. If any trade secret, know-how or other technology not protected by a patent or intellectual property right were disclosed to, or independently developed by a competitor, our business, financial condition and results of operations could be materially adversely affected.

If third parties claim that our product candidates or technologies infringe their intellectual property rights, we may become involved in expensive patent litigation, which could result in liability for damages or require us to stop our development and commercialization of our product candidates after they have been approved and launched in the market, or we could be forced to obtain a license and pay royalties under unfavorable terms.

Our commercial success will depend in part on not infringing the patents or violating the proprietary rights of third parties. Competitors or third parties may obtain patents that may claim the composition, manufacture or use of our product candidates, or the technology required to perform research and development activities relating to our product candidates.

From time to time we receive correspondence inviting us to license patents from third parties. While we believe that our pre-commercialization activities fall within the scope of an available exemption against patent infringement provided in the United States by 35 U.S.C. § 271(e) and by similar research exemptions in Europe, claims may be brought against us in the future based on patents held by others. Also, we are aware of patents and other intellectual property rights of third parties relating to our areas of practice, and we know that others have filed patent applications in various countries that relate to several areas in which we are developing product candidates. Some of these patent applications have already resulted in patents and some are still pending. The pending patent applications may also result in patents being issued. In addition, the publication of patent applications occurs with a certain delay after the date of filing, so we may not be aware of all relevant patent applications of third parties at a given point in time. Further, publication of discoveries in the scientific or patent literature often lags behind actual discoveries, so we may not be able to determine whether inventions claimed in patent applications of third parties have been made before or after the date on which inventions claimed in our patent applications and patents have been made. All issued patents are entitled to a presumption of validity in many countries, including the United States and many European countries. Issued patents held by others may therefore limit our freedom to operate unless and until these patents expire or are declared invalid or unenforceable in a court of applicable jurisdiction.

We and our collaborators may not have rights under some patents that may cover the composition of matter, manufacture or use of product candidates that we seek to develop and commercialize, drug targets to which our product candidates bind, or technologies that we use in our research and development activities. As a result, our ability to develop and commercialize our product candidates may depend on our ability to obtain licenses or other rights under these patents. The third parties who own or control such patents may be unwilling to grant those licenses or other rights to us or our collaborators under terms that are commercially viable or at all. Third parties who own or control these patents could bring claims based on patent infringement against us or our collaborators and seek monetary damages and to enjoin further clinical testing, manufacturing and marketing of the affected product candidates or products. There has been, and we believe that there will continue to be, significant litigation in the pharmaceutical industry regarding patent and other intellectual property rights. If a third party sues us for patent infringement, it could consume significant financial and management resources, regardless of the merit of the claims or the outcome of the litigation.

If a third party brings a patent infringement suit against us and we do not settle the patent infringement suit and are not successful in defending against the patent infringement claims, we could be required to pay substantial damages or we or our collaborators could be forced to stop or delay research, development, manufacturing or sales of the product or product candidate that is claimed by the third party s patent. We or our collaborators may choose to seek, or be required to seek, a license from the third party and would most likely be required to pay license fees or royalties or both. However, there can be no assurance that any such license will be available on acceptable terms or at all. Even if

we or our collaborators were able to obtain a license, the rights may be nonexclusive, which would give our competitors access to the same intellectual property. Ultimately, we could be prevented from commercializing a product candidate, or forced to cease some aspect of our business operations as a result of patent infringement claims, which could harm our business.

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Our success depends on our ability to maintain and enforce our licensing arrangements with various third party licensors.

We are party to intellectual property licenses and agreements that are important to our business, and we expect to enter into similar licenses and agreements in the future. These licenses and agreements impose various research, development, commercialization, sublicensing, milestone and royalty payment, indemnification, insurance and other obligations on us. If we or our collaborators fail to perform under these agreements or otherwise breach obligations thereunder, our licensors may terminate these agreements and we could lose licenses to intellectual property rights that are important to our business. Any such termination could materially harm our ability to develop and commercialize the product candidate that is the subject of the agreement, which could have a material adverse impact on our results of operations.

If licensees or assignees of our intellectual property rights breach any of the agreements under which we have licensed or assigned our intellectual property to them, we could be deprived of important intellectual property rights and future revenue.

We are a party to intellectual property out-licenses, collaborations and agreements that are important to our business, and we expect to enter into similar agreements with third parties in the future. Under these agreements, we license or transfer intellectual property to third parties and impose various research, development, commercialization, sublicensing, royalty, indemnification, insurance, and other obligations on them. If a third party fails to comply with these requirements, we generally retain the right to terminate the agreement and to bring a legal action in court or in arbitration. In the event of breach, we may need to enforce our rights under these agreements by resorting to arbitration or litigation. During the period of arbitration or litigation, we may be unable to effectively use, assign or license the relevant intellectual property rights and may be deprived of current or future revenues that are associated with such intellectual property, which could have a material adverse effect on our results of operations and financial condition.

We may be subject to damages resulting from claims that we or our employees have wrongfully used or disclosed alleged trade secrets of their former employers.

Many of our employees were previously employed at other biotechnology or pharmaceutical companies, including our competitors or potential competitors. Although no claims against us are currently pending, we may be subject to claims that these employees or we have inadvertently or otherwise used or disclosed trade secrets or other proprietary information of their former employers. Litigation may be necessary to defend against these claims. Even if we are successful in defending against these claims, litigation could result in substantial costs and be a distraction to management. If we fail in defending such claims, in addition to paying money claims, we may lose valuable intellectual property rights or personnel. A loss of key personnel or their work product could hamper or prevent our ability to commercialize certain product candidates.

Risks Relating to Manufacturing and Sales of Products

We depend on our collaborators and third-party manufacturers to produce most, if not all, of our product candidates and if these third parties do not successfully manufacture these product candidates our business will be harmed.

We have no manufacturing experience or manufacturing capabilities for the production of our product candidates for clinical trials or commercial sale. In order to continue to develop product candidates, apply for regulatory approvals, and commercialize our product candidates following approval, we or our collaborators must be able to manufacture or

contract with third parties to manufacture our product candidates in clinical and commercial quantities, in compliance with regulatory requirements, at acceptable costs and in a timely manner. The manufacture of our product candidates may be complex, difficult to accomplish and difficult to scale-up when large-scale production is required. Manufacture may be subject to delays, inefficiencies and poor or low yields of quality products. The cost of manufacturing our product candidates may make them prohibitively expensive. If supplies of any of our product candidates or related materials become unavailable on a timely basis or at all or are contaminated

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or otherwise lost, clinical trials by us and our collaborators could be seriously delayed. This is due to the fact that such materials are time-consuming to manufacture and cannot be readily obtained from third-party sources.

To the extent that we or our collaborators seek to enter into manufacturing arrangements with third parties, we and such collaborators will depend upon these third parties to perform their obligations in a timely and effective manner and in accordance with government regulations. Contract manufacturers may breach their manufacturing agreements because of factors beyond our control or may terminate or fail to renew a manufacturing agreement based on their own business priorities at a time that is costly or inconvenient for us. If third-party manufacturers fail to perform their obligations, our competitive position and ability to generate revenue may be adversely affected in a number of ways, including:

- we and our collaborators may not be able to initiate or continue clinical trials of product candidates that are under development;
- we and our collaborators may be delayed in submitting applications for regulatory approvals for our product candidates; and
- we and our collaborators may not be able to meet commercial demands for any approved products.

We have no sales, marketing or distribution experience and will depend significantly on third parties who may not successfully sell our product candidates following approval.

We have no sales, marketing or product distribution experience. If we receive required regulatory approvals to market any of our product candidates, we plan to rely primarily on sales, marketing and distribution arrangements with third parties, including our collaborators. For example, as part of our agreements with Merck Serono and MedImmune, we have granted these companies the right to market and distribute products resulting from such collaborations, if any are ever successfully developed. We may have to enter into additional marketing arrangements in the future and we may not be able to enter into these additional arrangements on terms that are favorable to us, if at all. In addition, we may have limited or no control over the sales, marketing and distribution activities of these third parties, and sales through these third parties could be less profitable to us than direct sales. These third parties could sell competing products and may devote insufficient sales efforts to our product candidates following approval. As a result, our future revenues from sales of our product candidates, if any, will be materially dependent upon the success of the efforts of these third parties.

We may seek to co-promote products with our collaborators, or to independently market products that are not already subject to marketing agreements with other parties. If we determine to perform sales, marketing and distribution functions ourselves, we could face a number of additional risks, including:

- we may not be able to attract and build a significant and skilled marketing staff or sales force;
- the cost of establishing a marketing staff or sales force may not be justifiable in light of the revenues generated by any particular product; and
- our direct sales and marketing efforts may not be successful.

Item 1B. ***Unresolved Staff Comments***

Not applicable.

Item 2. ***Properties***

Our current corporate headquarters are located in Carlsbad, California and are held under a 10-year lease that commenced in 2002. The property held under this lease also included a research and development facility previously operated by CancerVax. In April 2006, we entered into a sublease agreement pursuant to which 46,527 rentable square feet of the 61,618 total rental square feet covered by the original lease was subleased. The sublease has a term through June 2012.

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Shortly after the filing of this report, we expect to move our corporate headquarters to Bethesda, Maryland and to enter into a lease for such offices. We are currently seeking to sublease the remainder of our premises leased in Carlsbad, California.

We also maintain a research and development facility of approximately 81,161 square feet located in Munich, Germany, which is leased under a 10-year operating lease that commenced in July 2002. We have options to renew this lease for additional periods of five years. We believe that this facility will suffice for our anticipated future research and development requirements for the foreseeable future. We also entered into an agreement with the lessor to receive a subsidy in the amount of approximately 365,000, or $345,000, which subsidy would be required to be repaid in full or in part in the event that we terminate the lease for our Munich facility prior to December 2010. In 2005, we also entered into an agreement with the landlord of the Munich facility to defer a portion of our monthly rental payments. Upon consummation of the merger with CancerVax in May 2006, we repaid the full amount of approximately $623,000 in deferred rental payments to the lessor.

CancerVax also maintained a biologics manufacturing facility in Marina Del Rey, California under an operating lease for approximately 51,000 square feet with a term through August 2011. In connection with the merger with Micromet AG, in April 2006 CancerVax entered into an assignment of its obligations under this lease to another company.

In addition, CancerVax leased approximately 43,000 square feet of warehouse, laboratory and office space in the Los Angeles, California area under a seven-year lease with a term through August 2011. This facility was closed in 2006, and in June 2006 we entered into a lease termination agreement with the landlord. In connection with this termination, we paid a termination fee and related costs in the aggregate amount of approximately $560,000.

Item 3. ***Legal Proceedings***

Cell Therapeutics Inc./Novuspharma S.p.A.

On January 2, 2004, our collaborator, Novuspharma S.p.A., was acquired by Cell Therapeutics Inc. (CTI). Subsequently, CTI management announced that it would not make any payments to us for outstanding invoices and contractual obligations. At that date, 4.9 million, or $6.1 million, of invoices submitted for payment to Novuspharma were not paid, of which 2.2 million, or $2.7 million, was invoiced in 2003 and 2.7 million, or $3.4 million, was invoiced in 2004. As collectability was not reasonably assured, we did not record revenues and receivables related to these unpaid invoices.

On February 10, 2004, the collaboration agreement with CTI was terminated on the basis of the failure of CTI to meet its contractual payment obligations. On the same date, we commenced legal proceedings against CTI for breach of contract. On February 23, 2004, CTI filed a counterclaim against us. Based on its assessment of the contract, management believed that we would prevail against the countersuit, and therefore no financial provisions were made in our financial statements. In December 2005, the parties submitted the dispute to non-binding mediation. This mediation led to a settlement agreement with CTI on May 3, 2006, pursuant to which CTI made a payment of $1.9 million to Micromet AG.

Curis, Inc.

On October 2, 2006, a court-proposed settlement agreement with Curis, Inc. became effective that resolved a lawsuit initiated by Curis against Micromet AG in a German court regarding the repayment of an outstanding promissory note in the remaining principal amount of 2.0 million, or $2.6 million. Curis had requested immediate repayment of this amount at the time of the merger between CancerVax and Micromet AG in May 2006. We had disagreed with Curis s

interpretation of the repayment terms of the promissory note. In accordance with the settlement, we paid Curis 1.0 million, or $1.3 million, in October 2006, and will pay 1.0 million on or before May 31, 2007. The second payment will be reduced to 0.8 million if the payment is made on or before April 30, 2007. The payments will be made by us without any interest charges. Both parties bear their own costs incurred in connection with the litigation.

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Patent Opposition in Europe

Micromet AG s patent EP1071752B1 was opposed under Articles 99 and 100 of the European Patent Convention (EPC), by Affimed Therapeutics AG in March 2004. The opponent alleged that the patent does not fulfill the requirements of the EPC. On January 19, 2006, the Opposition Division of the European Patent Office (EPO) revoked the opposition in oral proceedings according to Article 116 of the EPC and maintained the patent as granted. The opponent filed a notice of appeal on May 30, 2006. On August 7, 2006, Micromet AG and Affimed entered into a settlement agreement pursuant to which Micromet AG reimbursed Affimed for a portion of its legal costs in the amount of 75,000, or $96,000, and Affimed agreed to withdraw the opposition. We were notified of the closure of appeal proceedings by the EPO on November 11, 2006.

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

None.

PART II

Item 5. ***Market for Registrant s Common Equity, Related Stockholder Matters and Issuer Purchases of Equity Securities***

Market Information

Our common stock is quoted on the NASDAQ Global Market under the symbol MITI . Prior to May 5, 2006, our common stock was quoted under the symbol CNVX . The following table sets forth, for the periods indicated, the high and low sales prices per share of our common stock as reported on the Nasdaq Global Market (previously the Nasdaq National Market). The data below reflects the 1:3 reverse stock split of our common stock effected on May 5, 2006.

	High	Low
Year Ended December 31, 2005		
First Quarter	$ 33.00	$ 18.06
Second Quarter	$ 20.13	$ 8.10
Third Quarter	$ 12.72	$ 8.28
Fourth Quarter	$ 10.38	$ 3.93
Year Ended December 31, 2006		
First Quarter	$ 10.65	$ 3.96
Second Quarter	$ 10.26	$ 4.07
Third Quarter	$ 4.47	$ 2.25
Fourth Quarter	$ 5.30	$ 1.82

As of February 28, 2007, there were approximately 153 holders of record of our common stock.

Dividend Policy

We have never declared or paid cash dividends on our capital stock. We currently intend to retain all available funds and any future earnings for use in the operation and expansion of our business and do not anticipate paying any cash dividends in the foreseeable future.

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Comparative Stock Performance Graph

The following comparative stock performance graph illustrates a comparison of the total cumulative stockholder return (assuming reinvestment of dividends, if any) from investing $100 in our common stock (traded under the symbol MITI) since October 30, 2003, the date our stock commenced public trading, and plotted at the close of the last trading day of the fiscal year ended December 31, 2003 and the end of each fiscal quarter during 2004, 2005 and 2006, to three indices: the Nasdaq Composite Index of U.S. Companies, the Nasdaq Pharmaceuticals Index and a self-constructed peer group of 23 public biotechnology companies of similar size, market capitalization and stage of development compared to us, except that for the Nasdaq Composite Index and the Nasdaq Pharmaceuticals Index, the stock performance graph below reflects an investment date of September 30, 2003. The returns of each component issuer of our self-constructed peer group has been weighted according to the respective issuer s stock market capitalization at the beginning of each period for which a return is indicated. The comparisons in the graph are required by the Securities and Exchange Commission and are not intended to forecast or be indicative of possible future performance of our common stock.

Our self-constructed peer group consists of the following 23 companies:

ACADIA Pharmaceuticals, Inc.
Alnylam Pharmaceuticals, Inc.
Anadys Pharmaceuticals, Inc.
Anesiva, Inc.
Antigenics, Inc.
Arqule, Inc.
Avant Immunotherapeutics, Inc.
CombinatoRx, Inc.
Curagen Corporation
Cyclacel Pharmaceuticals, Inc.
Cytokinetics, Incorporated
Dyax Corp.
Dynavax Technologies Corporation
Immunogen Inc
Immunomedics, Inc.
Maxygen, Inc.
Metabasis Therapeutics, Inc.
Neurogen Corporation
Panacos Pharmaceuticals, Inc.
Renovis, Inc.
Rigel Pharmaceuticals, Inc.
Seattle Genetics, Inc.
Sunesis Pharmaceuticals, Inc.

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The information included under the heading Comparative Stock Performance Graph in this Item 5 of our annual report on Form 10-K shall not be deemed to be soliciting material or subject to Regulation 14A or 14C, shall not be deemed filed for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date hereof and irrespective of any general incorporation language in any such filing.

COMPARISON OF 39 MONTH CUMULATIVE TOTAL RETURN
Among Micromet, Inc., The NASDAQ Composite Index,
The NASDAQ Pharmaceutical Index and a Peer Group

12/03	3/04	6/04	9/04	12/04	3/05	6/05	9/05	12/05	3/06
$ 79.00	$ 88.42	$ 63.42	$ 67.50	$ 90.42	$ 54.92	$ 23.75	$ 28.67	$ 11.50	$ 23.
$ 112.00	$ 113.33	$ 115.46	$ 110.63	$ 127.81	$ 120.03	$ 122.11	$ 128.97	$ 128.56	$ 138.
$ 100.07	$ 103.74	$ 102.83	$ 101.53	$ 109.26	$ 94.58	$ 100.32	$ 121.31	$ 122.90	$ 127.
$ 105.88	$ 107.05	$ 97.39	$ 86.42	$ 98.78	$ 65.13	$ 71.38	$ 84.13	$ 73.76	$ 89.

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Item 6. ***Selected Consolidated Financial Data.***

The following selected consolidated financial data for the three years ended December 31, 2006 are derived from our audited consolidated financial statements included in this report. The selected financial data as of December 31, 2004 and as of and for the years ended December 31, 2003 and 2002 are derived from unaudited financial statements not included in this report. In May 2006, CancerVax Corporation merged with Micromet AG. For accounting purposes, the business combination was considered a reverse merger under which Micromet AG was considered the acquirer of CancerVax. Accordingly, all financial information prior to the merger date reflects the historical financial results of Micromet AG alone. For 2006, the results of operations of the combined company reflect those of Micromet AG for the full year and, from May 5, 2006 on, the combined financial results of Micromet AG and CancerVax.

The Consolidated Statement of Operations Data and Consolidated Balance Sheet Data presented below is only a summary and should be read in conjunction with Item 7. Management s Discussion and Analysis of Financial Condition and Results of Operations, and our consolidated financial statements and related notes appearing elsewhere in this Form 10-K and incorporated by reference herein. Historical results are not necessarily indicative of the results to be expected in the future. See the notes to our financial statements for an explanation of the method used to determine the number of shares used in computing basic and diluted net loss per share.

	Years Ended December 31,				
	2006	2005	2004	2003	2002
	(In thousands, except per share amounts)				
Consolidated Statement of Operations Data:					
Revenues					
Collaboration agreements	$ 25,449	$ 23,130	$ 14,530	$ 14,844	$
License fees	1,959	2,482	2,103	57	
Other	175	111	108	31	3,539
Total revenues	27,583	25,723	16,741	14,932	3,539
Operating expenses					
Research and development[1]	28,252	28,579	33,084	29,630	21,215
In-process research and development	20,890				
General and administrative[1]	12,012	6,861	5,589	4,433	2,427
Total operating expenses	61,154	35,440	38,673	34,063	23,642
Loss from operations	(33,571)	(9,717)	(21,932)	(19,131)	(20,103)
Other income (expenses)					
Interest expense	(1,725)	(5,176)	(2,944)	(2,346)	(1,141)
Interest income	743	335	264	660	974
Other income (expense)	561	288	(456)	(640)	(124)
Total other income (expense)	(421)	(4,553)	(3,136)	(2,326)	(291)
Net loss	(33,992)	(14,270)	(25,068)	(21,457)	(20,394)
		(4,780)			

Beneficial conversion charge on issuance of preferred shares					
Net loss attributable to common stockholders	$ (33,992)	$ (19,050)	$ (25,068)	$ (21,457)	$ (20,394)
Basic and diluted net loss per common share	$ (1.29)	$ (3.70)	$ (16.64)	$ (14.25)	$ (13.54)
Weighted average shares used to compute basic and diluted net loss per share	26,366	5,147	1,506	1,506	1,506

(1) Includes the following amounts related to stock-based compensation expense (in thousands):

Research and development	$ 2,574	$	$	$	$
General and administrative	$ 3,032	$	$ 2	$ 20	$

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	As of December 31,				
	2006	2005	2004	2003	2002
Consolidated Balance Sheet Data (in thousands):					
Cash and cash equivalents	$ 24,301	$ 11,414	$ 12,749	$ 18,356	$ 21,581
Working capital	11,578	(10,407)	(1,459)	13,339	17,353
Total assets	51,172	28,877	50,002	45,759	46,790
Long-term debt, net of current portion	7,408	5,531	9,878	9,880	7,777
Convertible notes, net of current portion		11,844	40,236	29,936	14,061
Accumulated deficit	(144,807)	(110,815)	(91,768)	(66,699)	(45,242)
Total stockholders equity (deficit)	24,518	(14,533)	(33,231)	(5,250)	15,105

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