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

FORM 10-K

(Mark One)

☒ **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

For the fiscal year ended December 31, 2006

OR

☐ **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**

For the transition period from to

Commission file number: 001-33277

SYNTA PHARMACEUTICALS CORP.
(Exact name of registrant as specified in its charter)

Delaware (State or other jurisdiction of incorporation or organization)	**04-3508648** (I.R.S. Employer Identification No.)
45 Hartwell Avenue **Lexington, Massachusetts** (Address of principal executive offices)	**02421** (Zip Code)

Registrant's telephone number, including area code **(781) 274-8200**

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

Title of each class	Name of each exchange on which registered
Common Stock, $0.0001 Par Value Per Share	The NASDAQ Stock Market, LLC

Securities registered pursuant to Section 12(g) of the Exchange 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 ☐ No ☒

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

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 ☐ No ☒

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

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 ☐ Accelerated filer ☐ Non-accelerated filer ☒

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

The aggregate market value of the registrant's voting and non-voting common stock held by non-affiliates of the registrant (without admitting that any person whose shares are not included in such calculation is an affiliate) computed by reference to the price at which the common stock was last sold on March 15, 2007 was $156,526,648. The registrant has provided this information as of March 15, 2007 because its common stock was not publicly traded as of the last business day of its most recently completed second fiscal quarter.

As of March 15, 2007, the registrant had 33,816,532 shares of common stock outstanding.

PART I

Item 1. BUSINESS

Overview

We are a biopharmaceutical company focused on discovering, developing and commercializing small molecule drugs that address severe medical conditions with large potential markets, including cancer and chronic inflammatory diseases. We have a diverse pipeline of clinical- and preclinical-stage drug candidates with distinct mechanisms of action and novel chemical structures. We discovered and developed each of our drug candidates internally, using our unique chemical compound library and the chemistry, biology, and pharmaceutical development assets and capabilities built over the combined history of Synta and its predecessor companies. At present, we retain all rights to all of our drug candidates and programs, across all geographic markets and therapeutic indications.

Our Lead Drug Candidate, STA-4783

Our most advanced clinical-stage drug candidate, STA-4783, is a novel, injectable, small molecule compound with a unique mechanism of action that has potential for the treatment of a broad range of solid tumor cancers. In September 2006, we announced positive results for STA-4783 in combination with paclitaxel, a leading chemotherapeutic agent, in a double-blind, randomized, controlled, multicenter Phase 2b clinical trial in patients with stage IV metastatic melanoma. We believe this is the first blinded clinical trial of a drug candidate for the treatment of metastatic melanoma in 30 years to meet its primary endpoint with statistical significance. In November 2006, we received Fast Track designation from the U.S. Food and Drug Administration, or FDA, for the development of STA-4783 for the treatment of metastatic melanoma. The FDA grants Fast Track designation for drug candidates intended to treat serious or life threatening conditions and that demonstrate the potential to address unmet medical needs. Designation as a Fast Track product can facilitate the development and expedite the review of a drug candidate by allowing for more frequent and timely meetings with the FDA and submission of a new drug application, or NDA, on a rolling basis. However, Fast Track designation does not alter the standards for approval of a drug candidate, including the need for clinical trials that demonstrate safety and efficacy, nor does it mean that the FDA will expedite approval of a drug candidate. In addition, Fast Track designation does not increase the likelihood of approval of a drug candidate. Based on the results of the Phase 2b trial, we expect to initiate a pivotal Phase 3 clinical trial in metastatic melanoma and initiate clinical trials in one or more additional cancer types in mid-2007.

Our Phase 2b clinical trial enrolled a total of 81 metastatic melanoma patients at 21 centers in the United States. This clinical trial was conducted in a double-blind, randomized, controlled fashion and compared the effects of STA-4783 in combination with paclitaxel, the most widely used taxane, versus paclitaxel alone. The primary endpoint for assessing efficacy was progression-free survival. Progression-free survival measures for each patient the time from when the patient was assigned to a treatment group in the trial until the earlier of tumor progression or death. The FDA has previously indicated this endpoint is acceptable for registration in metastatic melanoma and other cancer types in clinical trials by other companies. Two analyses of trial results were specified in the statistical plan for the trial, one that includes all patients, known as the intent-to-treat analysis, and one that includes only those patients who could be evaluated for efficacy as specified in the protocol, known as the per-protocol analysis. In both of these analyses of the trial results, treatment with STA-4783 plus paclitaxel demonstrated a statistically significant improvement in progression-free survival compared to treatment with paclitaxel alone.

In the intent-to-treat analysis of the trial results, which includes all 81 patients, median progression-free survival increased from 1.84 months for patients treated with paclitaxel alone to 3.68 months for patients treated with STA-4783 plus paclitaxel. The percentage of patients who survived

and were free of tumor progression at six months more than doubled from 15% for patients treated with paclitaxel alone to 35% for patients treated with STA-4783 plus paclitaxel. The statistical significance of the improvement in progression-free survival is described by a p-value, which measures the probability that the difference is due to chance alone. A p-value of less than 0.05 is considered statistically significant and unlikely due to chance. The p-value in this analysis was 0.035.

In the per-protocol analysis of the trial results, which includes the 77 patients who could be evaluated for efficacy as specified in the trial protocol, median progression-free survival increased from 1.84 months for patients treated with paclitaxel alone to 4.40 months for patients treated with STA-4783 plus paclitaxel. The percentage of patients who survived and were free of tumor progression at six months more than doubled from 15% for patients treated with paclitaxel alone to 37% for patients treated with STA-4783 plus paclitaxel. The p-value in this analysis was 0.017.

We have also performed an analysis to determine if factors other than treatment with STA-4783, known as confounding factors, could be responsible for the differences observed between the two treatment groups in this clinical trial. In particular, we analyzed differences in patient characteristics and disease status that can influence disease progression. To date, we have identified no potentially confounding variables which alter the interpretation of the trial results.

We filed the initial Investigational New Drug application, or IND, for STA-4783 in September 2002. Including the patients treated in the Phase 2b metastatic melanoma clinical trial, we have treated a total of approximately 300 patients at over 50 medical centers in the United States and Canada with STA-4783. STA-4783 has been well tolerated, with toxicities of the STA-4783 plus paclitaxel combination generally similar to those of paclitaxel alone, and the incidences of individual severe adverse events generally less than 10%.

Our Other Drug Candidates and Research Programs

STA-4783 was discovered and developed internally from our chemical compound library and using our drug discovery capabilities. In addition to STA-4783, we have discovered and developed three other drug candidates currently in clinical or preclinical development, each of which has a distinct chemical structure, mechanism of action, and presents a differentiated market opportunity. We also have one research-stage program, which is in the lead optimization stage of discovery.

Oncology

STA-9090. STA-9090 is a novel, injectable, small molecule drug candidate we are developing for the treatment of cancer. STA-9090 inhibits heat shock protein 90, or Hsp90, a chaperone protein that regulates the activity of numerous signaling proteins that trigger uncontrolled proliferation in cancer cells, in particular kinase proteins. Examples of kinase proteins include c-Kit, Bcr-Abl, and others that are the targets of approved direct kinase inhibitors such as Gleevec. We believe that inhibiting kinases indirectly, by disrupting the chaperone activities of Hsp90, provides two advantages: first, a means to simultaneously attack multiple cancer-promoting kinases; and, second, an ability to kill tumor cells with mutated kinases that have lost responsiveness to a direct kinase inhibitor. We have shown in preclinical experiments that STA-9090 is significantly more potent against certain types of cancer cells than Gleevec, as well as the two Hsp90 inhibitors furthest along in clinical development, 17-AAG and 17-DMAG. STA-9090 is further differentiated from these Hsp90 inhibitors in that it is a novel chemical structure that is not a derivative or analog of the natural product geldanamycin. We believe this creates a distinct activity profile for STA-9090 and is a competitive advantage. This program is currently in preclinical development, and we expect to file an IND in mid-2007.

STA-9584. STA-9584 is a novel, injectable, small molecule compound that disrupts the blood vessels that supply tumors with oxygen and essential nutrients. In preclinical experiments, STA-9584 has shown strong anti-tumor activity in a broad range of cancer models, including prostate, lung, breast,

melanoma, and lymphoma. In preclinical testing, STA-9584 has been shown to act against established tumor vessels, a mechanism that is differentiated from the mechanism of anti-angiogenesis inhibitors such as Avastin, which prevents the formation of new tumor vessels. This program is currently in preclinical development.

Autoimmune and Inflammatory Diseases

Apilimod (STA-5326). Apilimod is a novel, orally administered, small molecule drug candidate we are developing for the treatment of autoimmune and other chronic inflammatory diseases. Apilimod inhibits the production of the cytokines interleukin-12, or IL-12, and interleukin-23, or IL-23, and thereby down-regulates the inflammation pathways that underlie certain autoimmune and inflammatory diseases. We filed the initial IND for apilimod in March 2003. We are currently conducting a Phase 2a clinical trial of apilimod in patients with rheumatoid arthritis and sponsoring a Phase 2a clinical trial in patients with gastrointestinal manifestations of common variable immunodeficiency, or CVID. We expect to report results from these trials in 2007.

CRAC ion channel inhibitor. We have developed novel, small molecule inhibitors of calcium release activated calcium, or CRAC, ion channels expressed on immune cells. The CRAC ion channel is the primary route for calcium entry into T cells and other immune cells, regulating multiple immune cell processes important for initiating and maintaining an inflammatory immune response. We have demonstrated in preclinical experiments that our CRAC ion channel inhibitors selectively inhibit the production of critical pro-inflammatory cytokines, such as IL-2 and TNFα, by immune cells, and that these compounds are effective in multiple animal models of immune diseases, including models of arthritis. This program is in the lead optimization stage of discovery.

Our Drug Candidate Pipeline

The following table summarizes our most advanced drug candidates currently in clinical or preclinical development:

	Product Candidate	Disease	Stage	Status	Worldwide Commercial Rights
Oncology	**STA-4783** Oxidative stress inducer	Metastatic melanoma	Phase 2b	Completed — met primary endpoint	Synta
			Phase 3	Initiate in mid-2007	
		Additional cancers	Phase 2	Initiate in mid-2007	Synta
	STA-9090 Hsp90 inhibitor	Cancer	Preclinical development	IND expected in mid-2007	Synta
	STA-9584 Vascular disrupting agent	Cancer	Preclinical development	Ongoing	Synta
Inflammatory Diseases	**Apilimod (STA-5326)** Oral IL-12/23 inhibitor	Rheumatoid arthritis	Phase 2a	Results expected in 2007	Synta
		Common variable immunodeficiency	Phase 2a	Results expected in 2007	Synta
	Oral CRAC ion channel inhibitor	Autoimmune diseases, transplant	Lead optimization	Ongoing	Synta

In the above table, lead optimization indicates that compounds have shown activity, selectivity, and efficacy in *in vivo* models, as well as an acceptable preliminary safety profile. These compounds are being optimized for potency, drug-like properties, and safety before entering into preclinical development. Preclinical development activities include manufacturing, formulation, and full toxicology studies in preparation for a Phase 1 clinical trial. Phase 1 indicates initial clinical safety testing and pharmacological profiling in healthy volunteers, with the exception that Phase 1 clinical trials in oncology are typically performed in patients with cancer. Phase 2 involves efficacy testing and continued safety testing in patients with a specific disease, and may include separate Phase 2a and Phase 2b clinical trials. Phase 2a clinical trials typically test the drug candidate in a small number of patients and are designed to provide early information on drug safety and efficacy. Phase 2b clinical trials typically involve larger numbers of patients and comparison with placebo, standard treatments, or other active comparators. Phase 3 indicates a confirmatory study of efficacy and safety in a larger patient population, and typically involves comparison with placebo, standard treatments, or other active comparators.

Oncology Programs

We have one clinical-stage program and two preclinical-stage programs in oncology:

- *STA-4783.* Our most advanced clinical-stage drug candidate, STA-4783, has achieved positive results in a double-blind, randomized, controlled, multicenter Phase 2b clinical trial in patients with stage IV metastatic melanoma. We expect to initiate a pivotal Phase 3 clinical trial in metastatic melanoma and announce plans for initiating clinical trials in one or more additional cancer types in mid-2007.

- *STA-9090.* STA-9090, our novel, small molecule Hsp90 inhibitor, is in preclinical development. We expect to file an IND in mid-2007.

- *STA-9584.* STA-9584, our novel small molecule compound that disrupts the blood vessels that supply tumors with oxygen and essential nutrients, is in preclinical development.

Oncology Background

Cancers are diseases characterized by abnormal and uncontrolled cell growth and division, typically leading to tumor formation. As a tumor grows, it can directly disrupt organ function at its site of origin. In addition, cancer cells can also spread to other organs, such as the brain, bones and liver, by a process called metastasis. The growth of metastatic tumors at these new sites can disrupt the function of these other organs. There are many kinds of cancer, but all are characterized by uncontrolled growth of abnormal cells.

The World Health Organization estimates that more than 11 million people are diagnosed with cancer every year worldwide, and seven million people die from the disease annually. The American Cancer Society estimates that approximately 1.4 million people in the United States will be diagnosed with cancer in 2006, and approximately 565,000 people will die from the disease.

Chemotherapeutics are the second largest therapeutic class of pharmaceuticals in the world, with global sales of $28.5 billion in 2005.

Melanoma

Melanoma is the deadliest type of skin cancer and is the sixth most commonly diagnosed cancer in the United States. The National Cancer Institute has estimated that the prevalence of melanoma in the United States, or the number of patients alive who have been diagnosed with the disease, currently is more than 660,000. The American Cancer Society estimates that in 2006 the incidence, or number of newly diagnosed cases, of melanoma in the United States will be approximately 62,000, with 8,000 deaths from the disease. According to GLOBOCAN, the worldwide incidence of melanoma in 2002 was 160,177, with 40,781 deaths from the disease.

Melanoma is classified into four stages, which are based on well-defined criteria, including characteristics of the primary tumors, involvement of the regional lymph nodes, and the extent and location of metastases. When melanoma is discovered and treated in the early stages, where the cancer is confined to a local area, patients have a relatively high rate of survival. For example, stage I patients have a five-year survival rate of between 90 and 95%. Once melanoma has advanced to stage III, where the cancer has spread to the regional lymph nodes, or stage IV, where the cancer has spread to distant organs, the prognosis for patients is much worse. The five-year survival rate for patients with stage IV melanoma is extremely poor: less than 20%. In 2001, the American Joint Committee on Cancer estimated that approximately 15% of patients with melanoma were initially diagnosed with advanced-stage disease, which consists of stage III and stage IV melanoma. However, recent scientific articles suggest that this percentage may grow significantly with the increased use of improved diagnostic techniques. In a study reported in the February 2003 issue of *The Journal of the American College of*

Surgeons, approximately 38% of 175 patients originally diagnosed with stage I or stage II melanoma should have been categorized with stage III melanoma. The initial target indication for STA-4783 is metastatic melanoma. We are unaware of any reliable industry survey data for the prevalence of metastatic melanoma in the United States or worldwide.

Limitations of Current Treatments for Metastatic Melanoma

For early stage melanoma, surgical removal of the primary melanoma lesion is the standard of care. Surgical removal may also be performed to remove distant skin metastases, lymph nodes or other organs to which the cancer has spread. Sometimes interferon alpha-2b is administered to patients as an adjuvant to surgery to reduce the rate of disease relapse. This is the only drug approved by the FDA for use in such a role.

For metastatic melanoma, treatment options are limited. Single-agent chemotherapy has typically shown progression-free survival of less than two months. Randomized trials comparing combination chemotherapy against single agent chemotherapy have shown significant toxicity with no significant improvement in survival. Dacarbazine, also known as DTIC, has been one of the most studied drugs in this setting, either alone or in combination, and is the only FDA-approved chemotherapy for the treatment of metastatic melanoma. However, when DTIC is used as a single agent, it has been shown to have limited clinical benefits. Various other single-agent chemotherapies such as temozolomide, fotemustine and Genasense have been tested against or in combination with DTIC. Response rates from controlled studies have typically been between 6% to 25% with median time to progression/ progression-free survival of 1.8 to 2.4 months. Immunotherapy with interleukin-2, or IL-2, has been approved by the FDA based on longer duration responses than typically observed with chemotherapy, but these responses occur only in a small subset of patients, and treatment with IL-2 is accompanied by severe toxicities. No agents other than DTIC or IL-2 have been approved by the FDA for the treatment of metastatic melanoma. Therefore, we believe there is an urgent need in metastatic melanoma for additional therapies demonstrating meaningful clinical benefit, favorable safety, and broad patient applicability.

Taxanes

The class of drugs known as taxanes is the market-leading class of chemotherapeutic drugs, with approximately $2.7 billion in worldwide sales in 2005. Approved taxanes include Taxol, a formulation of paclitaxel first approved in 1992 and marketed by Bristol-Myers Squibb, which achieved peak sales of approximately $1.6 billion in 2000 before patent expiry; Taxotere (docetaxel), which is marketed by Sanofi-Aventis and had global sales of approximately $2.0 billion in 2005; Abraxane, a paclitaxel protein conjugate marketed by Abraxis Pharmaceutical Partners; and several generic versions of paclitaxel. Taxanes have shown efficacy across a wide range of cancer types and have been approved by the FDA for the treatment of prostate, ovarian, breast, and non-small cell lung cancers, as well as Kaposi's sarcoma. Additionally, we believe taxanes are prescribed off-label for other cancer types, including metastatic melanoma, head and neck, uterine, stomach, esophageal, and bladder. In metastatic melanoma, the response rate of single agent paclitaxel has been reported as less than 20%. A study published in 2002 in *Cancer Investigation* showed that combining DTIC and paclitaxel for the treatment of metastatic melanoma was not superior to using each agent alone. Other anticancer agents that are sometimes added to taxanes in an attempt to improve efficacy include Paraplatin, a formulation of carboplatin marketed by Bristol-Myers Squibb. While in some cases the addition may increase treatment efficacy, carboplatin has been shown to add substantial toxicity. As a result, we believe there is a significant opportunity for agents that can enhance the anti-tumor effects of taxanes without adding undesirable side effects.

Our Lead Clinical Development Program—STA-4783

STA-4783 is a novel, small molecule drug candidate that induces an oxidative stress response in a wide variety of cancer cell types and has demonstrated, in preclinical models, synergistic anti-tumor activity with the two leading taxanes, paclitaxel and docetaxel. We believe that the anti-cancer activity of STA-4783 is due to its ability to directly increase oxidative stress, as measured by the level of reactive oxygen species, or ROS, inside cancer cells. Because cancer cells have an elevated level of oxidative stress relative to non-cancer cells, we believe that the increase in ROS induced by STA-4783 causes cancer cells to exceed a breaking point that triggers tumor cell death, while causing minimal damage to normal cells.

We have completed six clinical trials with STA-4783 in cancer patients, in which a total of approximately 300 patients have been treated at over 50 medical centers in the United States and Canada. Based on the positive results observed in our recently completed Phase 2b clinical trial in metastatic melanoma, we are now preparing for a pivotal Phase 3 clinical trial in metastatic melanoma, which we expect to initiate in the middle of 2007. We also expect to initiate additional Phase 2 clinical trials in one or more additional cancer indications in mid-2007. STA-4783 has received Fast Track designation from the FDA for the treatment of metastatic melanoma. The FDA grants Fast Track designation for drug candidates intended to treat serious or life threatening conditions and that demonstrate the potential to address unmet medical needs. We also intend to seek orphan drug designations in both the United States and Europe for STA-4783 for the treatment of metastatic melanoma.

Our Phase 2b Clinical Trial in Metastatic Melanoma

Summary

Our Phase 2b clinical trial enrolled a total of 81 metastatic melanoma patients at 21 centers in the United States. This clinical trial was conducted in a double-blind, randomized, controlled fashion and compared the effects of STA-4783 in combination with paclitaxel, the most widely used taxane, versus paclitaxel alone. The primary endpoint for assessing efficacy was progression-free survival. Progression-free survival is considered an acceptable endpoint for registration in metastatic melanoma and other cancer types, as supported by the current FDA draft guidance set forth in *Clinical Trial Endpoints for the Approval of Cancer Drugs and Biologics* issued in April 2005, and by the EMEA guidance set forth in the draft of Appendix 1 *Methodological Considerations for Using Progression-Free Survival (PFS) as Primary Endpoint in Confirmatory Trials for Registration* issued in July 2006 to the *Guideline on the Evaluation of Anticancer Medicinal Products in Man*, which became effective in June 2006.

In September 2006, we presented the results from this clinical trial at the joint meeting of Perspectives in Melanoma X and the Third International Melanoma Research Congress, held in The Netherlands. Patients who received STA-4783 plus paclitaxel showed a statistically significant improvement in progression-free survival compared to those who received paclitaxel alone.

Consistent with safety data for STA-4783 gathered from other clinical trials, STA-4783 was well tolerated in this clinical trial, with toxicities of the STA-4783 plus paclitaxel combination generally similar to those of paclitaxel alone.

Clinical Trial Design

The primary objective of this Phase 2b clinical trial was to assess the efficacy in stage IV metastatic melanoma patients of once-weekly treatment of STA-4783 plus paclitaxel versus paclitaxel alone, based on the endpoint of progression-free survival. Secondary endpoints were objective response rate, duration of tumor responses, and studies of adverse events and laboratory abnormalities. Once-weekly treatments of STA-4783 (213 mg/m^2) plus paclitaxel (80 mg/m^2) or paclitaxel alone (80 mg/m^2) were delivered for three weeks, followed by one week off-treatment. Investigators were permitted to repeat these four-week cycles until disease progression. Tumor assessments were performed at baseline and every other cycle thereafter.

Disease progression and tumor response were defined based on industry standard Response Evaluation Criteria in Solid Tumors, or RECIST, which are the unified response assessment criteria agreed to by the World Health Organization, United States National Cancer Institute, and European Organisation for Research and Treatment of Cancer. RECIST defines disease progression and tumor response based on the sum of the longest diameters of a set of target tumor lesions identified at baseline. A 20% or greater increase in the sum of diameters in target lesions, or unequivocal progression in non-target lesions, or the appearance of a new lesion is defined as disease progression. A reduction in the sum of the diameters of at least 30% as compared to baseline is defined as a partial response. A complete disappearance of target and non-target lesions (and the normalization of any tumor markers) constitutes a complete response. Both partial and complete responses must be confirmed by repeat assessments at least four weeks after the partial or complete response was first documented. Stable disease refers to patients who exhibit neither response nor disease progression. Non-progression refers to patients who exhibit complete response, partial response, or stable disease. Objective response rate is typically defined as the sum of the partial and complete response rates.

In this clinical trial, we enrolled patients who had received up to one prior chemotherapy treatment. An unlimited number of prior immunotherapy treatments were also allowed, provided that a period of four weeks subsequent to the last treatment elapsed prior to trial entry. Patients with Eastern Cooperative Oncology Group, or ECOG, performance status greater than 2 were excluded, as were patients with any brain metastases. The ECOG performance status is a standard patient assessment tool used in determining the care of cancer patients. Patients with an ECOG score of 3 or 4 are significantly disabled by their disease and are often excluded from clinical trials.

Two-thirds of patients were assigned to treatment with STA-4783 plus paclitaxel, with the remaining one-third of patients assigned to treatment with paclitaxel alone. We chose this 2:1 weighting ratio so as to contribute more productively to the safety database for STA-4783 than an even randomization, while still allowing for a statistical comparison of treatment effects. Patients who progressed on paclitaxel alone were given the option to crossover to STA-4783 plus paclitaxel and were then treated until further progression.

Clinical Trial Results

In the intent-to-treat analysis, which includes all 81 patients, patients treated with STA-4783 plus paclitaxel experienced a statistically significant increase in progression-free survival, with a p-value of 0.035. The median progression-free survival in this analysis increased from 1.84 months for patients treated with paclitaxel alone to 3.68 months for patients treated with STA-4783 plus paclitaxel. The percentage of patients who survived and were free of tumor progression at six months more than doubled from 15% for patients treated with paclitaxel alone to 35% for patients treated with STA-4783 plus paclitaxel. The Hazard ratio for progression-free survival in this analysis was 0.5, which indicates that patients treated with STA-4783 plus paclitaxel had a 50% reduction in the risk of disease progression relative to patients treated with paclitaxel alone.

The per-protocol population consists of only those patients who could be evaluated for efficacy as specified in the protocol, in that they received at least one treatment with either paclitaxel or STA-4783 plus paclitaxel and completed at least one tumor assessment following the baseline measurement. Of the 81 patients who were enrolled, 77 qualified for the per-protocol population. In this per-protocol analysis, patients treated with STA-4783 plus paclitaxel also experienced a statistically significant increase in progression-free survival, with a p-value of 0.017. The median progression-free survival in this analysis increased from 1.84 months for patients treated with paclitaxel alone to 4.40 months for patients treated with STA-4783 plus paclitaxel. The percentage of patients who survived and were free of tumor progression at six months more than doubled from 15% for patients treated with paclitaxel alone to 37% for patients treated with STA-4783 plus paclitaxel. The Hazard ratio for progression-free survival in this analysis was 0.42, which indicates that patients treated with STA-4783 plus paclitaxel had a 58% reduction in the risk of disease progression relative to patients treated with paclitaxel alone.

The objective response rate was also assessed, and in the intent-to-treat analysis, found to occur in 15.1% of patients treated with STA-4783 plus paclitaxel, versus 3.6% of patients treated with paclitaxel alone (p-value=0.153). For the per-protocol analysis, the objective response rates were 16% and 3.7% respectively (p-value=0.149). These results showed an encouraging trend but did not reach statistical significance. We were able to obtain complete progression data on only three of the nine patients that were responders in the trial, and as a result, we had insufficient data to perform an analysis on duration of response.

The table below summarizes the median progression-free survival, the progression-free survival at six months, the Hazard ratio, and the objective response rates for the intent-to-treat and the per-protocol populations.

		STA-4783 + Paclitaxel	Paclitaxel alone	P-value[1]	Hazard ratio[2]
		N=53	N=28		
Intent-to-treat analysis (N=81)	Progression-free survival:			0.035	0.50
	• Median (months)	3.68	1.84		
	• At 6 months (% of patients)	35%	15%		
	Objective response rate[3]	15.1%	3.6%	0.153	
		N=50	N=27		
Per-protocol analysis (N=77)	Progression-free survival:			0.017	0.42
	• Median (months)	4.40	1.84		
	• At 6 months (% of patients)	37%	15%		
	Objective response rate[3]	16%	3.7%	0.149	

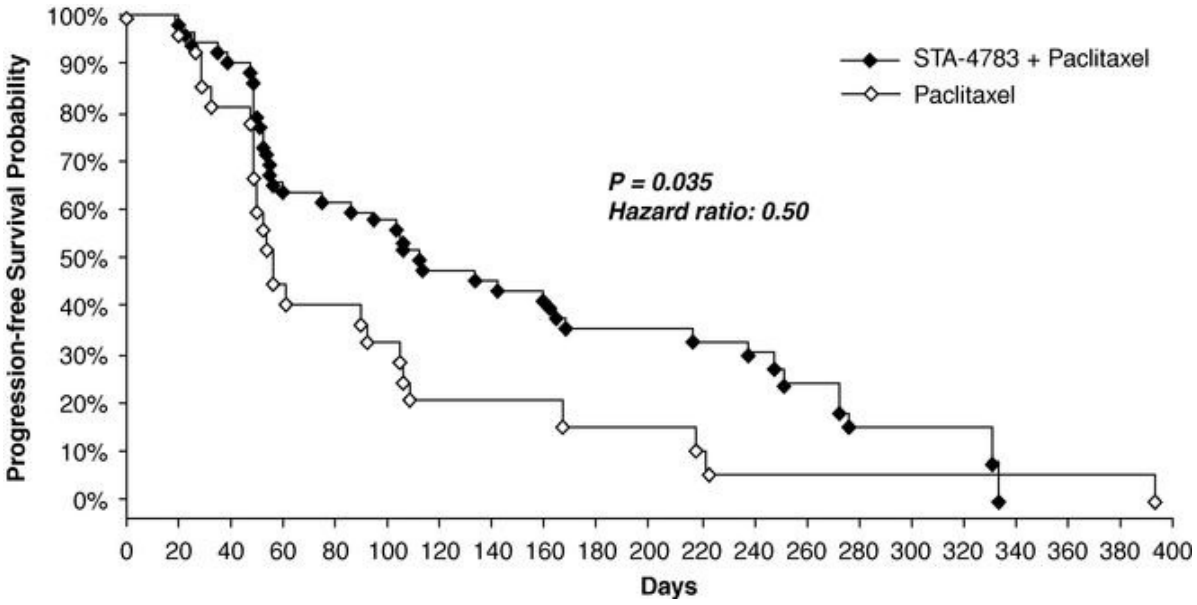
(1) P-value measures the probability that the difference is due to chance alone. A p-value of less than 0.05 is considered statistically significant and unlikely to be due to chance alone.

(2) Hazard ratio is an estimate of comparative risk between the two treatment groups. A hazard ratio of 1 can be interpreted as no decrease in risk, while a hazard ratio of 0.42 can be thought of as a 58% reduction in risk of occurrence for the event as compared to the control group.

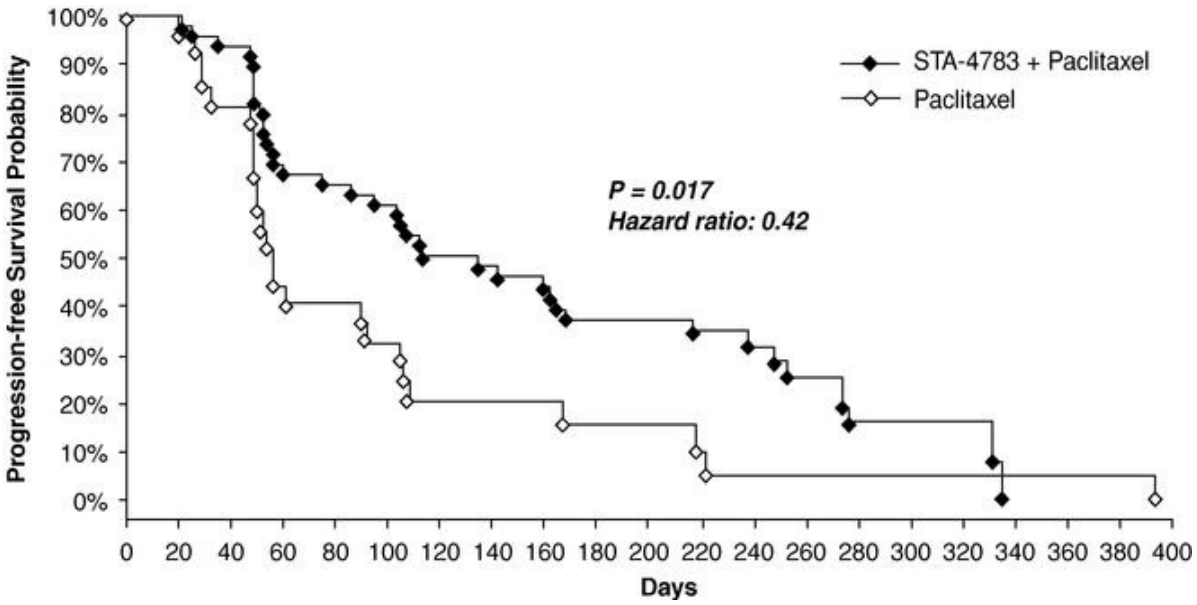
(3) Objective response rate is defined as the sum of complete and partial tumor response rates, as assessed by RECIST.

The figures below show the Kaplan-Meier plots of progression-free survival in this clinical trial for (1) the intent-to-treat population and (2) the per-protocol population.

Intent-to-Treat Population (N=81)



Per-Protocol Population (N=77)



Safety Profile

STA-4783 was well tolerated in this clinical trial. As shown in the table below, the incidence of any specific high severity adverse event, as reported by investigators, was less than 10%. We believe this compares favorably with treatments for metastatic melanoma such as the CVD regimen (cisplatin, vinblastine, and DTIC) or the Dartmouth regimen (DTIC, cisplatin, carmustine, and tamoxifen) that have reported substantially greater incidences of high severity adverse events. The incidence of such events that occurred in 2% or more of the patients treated with STA-4783 plus paclitaxel was as follows:

Grade 3 or Higher Adverse Events (1)(2)

	STA-4783 + Paclitaxel (N=52)	Paclitaxel (N=28)
Neutropenia(3)	4(7.7%)	0 (0%)
Back pain	2(3.8%)	2(7.1%)
Fatigue	2(3.8%)	2(7.1%)
Neuropathy(4)	2(3.8%)	1(3.6%)

(1) As specified in the clinical trial protocol, the patient population for evaluating safety includes only those patients who received at least one treatment with STA-4783 plus paclitaxel or paclitaxel alone. This represents 80 of the total 81 patients enrolled in the trial.

(2) Grade refers to the National Cancer Institute's Common Terminology Criteria, or CTC, for adverse events. The CTC are based on a 5-point severity scale with the following classifications: mild=1, moderate=2, severe=3, life-threatening=4, and fatal=5, and are commonly used in cancer clinical trials.

(3) Neutropenia is an abnormal decrease in white blood cells.

(4) Neuropathy is any disorder affecting any segment of the nervous system.

The adverse events seen across all severity grades in this clinical trial were typical of those expected from paclitaxel alone. The most common adverse events seen in the STA-4783 plus paclitaxel group included fatigue, alopecia, constipation, nausea, hypoaesthesia, arthralgia, insomnia, diarrhea, and anemia.

Tests for Confounding Factors

In order to determine whether any imbalances in the characteristics of the patients between the two treatment arms might have influenced the outcome of this clinical trial, we studied certain factors that might have an impact on progression-free survival. None of the potentially confounding factors we have analyzed to date have been found to have influenced the trial outcome. The factors we studied were:

- *Demographic characteristics.* Demographic characteristics, such as age, sex, ethnicity, race, and ECOG status, were found to have been distributed evenly between the treatment groups.

- *Days between tumor assessment.* To address a potential bias in assessment interval between treatment groups, we examined the number of days between the last tumor assessment prior to progression and progression and found this interval to be closely comparable. For the STA-4783 plus paclitaxel and paclitaxel treatment groups, the means were 55.4 days (standard deviation 14.4) and 52.8 days (standard deviation 12.2), respectively, and the medians were 56.0 and 55.5 days, respectively.

- *Elevated LDH levels.* An elevated level of the enzyme lactate dehydrogenase, or LDH, is considered a negative predictor of outcome and correlates with shorter progression-free survival. Accordingly, a comparable distribution of patients with elevated LDH across treatment arms is important in clinical trials of metastatic melanoma. In our Phase 2b clinical trial, patients with elevated LDH levels prior to treatment were distributed evenly between treatment groups: STA-4783 plus paclitaxel (43%) and paclitaxel alone (44%).

- *Liver metastases.* The presence of liver metastases is considered a negative predictor of outcome and correlates with shorter progression-free survival. Accordingly, a comparable distribution of patients with liver metastases is important in clinical trials of metastatic melanoma. In our trial, patients with liver metastases at baseline were also distributed evenly between treatment groups: STA-4783 plus paclitaxel (32%) and paclitaxel alone (32%).

- *M-class.* We also studied the relationship between the nature of patients' distant metastases and progression-free survival. Within stage IV metastatic melanoma there are three classifications, or M-classes, for the nature of distant metastases: M1a, M1b, and M1c. M1a patients have metastases limited to skin and subcutaneous tissue. M1b patients have metastases to lungs. M1c patients have metastases to liver or other distant organs or have elevated LDH. In general, the higher the M-class, the more advanced the disease and the worse the prognosis. In this clinical trial, patients with different M-classes by investigator assessment were distributed as follows, with some imbalance observed at each M-class: STA-4783 plus paclitaxel and paclitaxel alone, respectively: M1a: 13%, 7%; M1b: 34%, 18%; and, M1c: 53%, 75%.

In order to understand the impact of these prognostic factors, we performed a Cox proportional hazards regression analysis. The Cox analysis is designed to allow for a comparison of treatment arms while adjusting for different patient characteristics. For this clinical trial we tested the following characteristics, in addition to treatment with STA-4783, that are believed to be associated with an effect on the clinical outcome of progression-free survival: LDH level, presence of liver metastases, and M1 sub-class. Elevated LDH and the presence of liver metastases were found, as expected, to significantly worsen progression-free survival with p-values of <0.0001 and 0.0136, respectively. Conversely, M1 stage, as assessed by investigators in this clinical trial, was not found to have an impact on progression-free survival (p-value=0.841). The lack of impact of M-grade on progression-free survival is demonstrated in the following table:

	STA-4783 + Paclitaxel	**Paclitaxel alone**
Low M-grade (M1a or M1b)	3.48 months N=25	1.84 months N=7
High M-grade (M1c)	3.70 months N=28	1.81 months N=21

The treatment effect of STA-4783 on progression-free survival in the full Cox analysis, of all patients in the trial, which adjusts for the three variables above, remained statistically significant, with an adjusted hazard ratio of 0.54 and a p-value of 0.023.

Although not specified in the statistical analysis plan for the trial, in order to further elucidate the dependency of the results on the M1c classification we performed a subset analysis consisting of only those patients reported as M1c. This analysis, the results of which are tabulated below, showed comparable results to the analysis for all patients: a greater than doubling of median progression-free

survival, with a p-value of 0.041 in the intent-to-treat analysis and a p-value of 0.022 in the per-protocol analysis:

	STA-4783 + Paclitaxel 100% M1c	Paclitaxel alone 100% M1c	P-value
Intent-to-treat analysis	3.70 months N=28	1.81 months N=21	0.041
Per-protocol analysis	4.04 months N=26	1.81 months N=20	0.022

The results of the M1c subset analysis are consistent with the results of the Cox analysis, which showed that the M1 stage classification did not have an impact on progression-free survival.

Subset Analysis of Chemotherapy-Naïve Patients

Our Phase 2b clinical trial included patients who had received either no prior chemotherapy treatment or one prior chemotherapy treatment before beginning our trial. In order to understand the impact of the prior treatment, we examined the median progression-free survival and response rates in our trial for both subsets of patients. While this subset analysis is based on a small number of patients and is not statistically significant, the results indicated a trend towards additional enhancement of progression-free survival from treatment with STA-4783 in those patients who had received no prior chemotherapy. The results are illustrated in the table below. Of the 81 total patients, information on prior treatment was available for 80 patients, and was not available for one patient.

Prior chemotherapy treatment		STA-4783 + Paclitaxel	Paclitaxel alone
None (N=32)	Median progression-free survival	8.3 months	2.4 months
	Objective response rate	22% (5/23)	0% (0/9)
One (N=48)	Median progression-free survival	3.1 months	1.8 months
	Objective response rate	10% (3/29)	5.3% (1/19)

The results for treatment with paclitaxel alone in patients who have received no prior chemotherapy are comparable to results previously reported for patients treated with DTIC alone who had received no prior chemotherapy. In a 771 patient, randomized clinical trial comparing DTIC with DTIC plus oblimersen in patients with no prior chemotherapy, which was published in the *Journal of*

Clinical Oncology in October 2006, the median progression-free survival in patients who were treated with DTIC alone was 1.6 months, and the objective response rate was 7.5%.

Crossover Analysis

A total of 19 patients in our Phase 2b clinical trial who were treated with paclitaxel alone elected, subsequent to disease progression, to receive treatment with STA-4783 plus paclitaxel. Of those 19 patients, known as crossover patients, complete data on time to progression subsequent to the crossover are available for 14 patients. The progression-free survival times for these 14 patients ranged from 0.72 to 5.5 months. Three of these 14 patients had progressed rapidly on paclitaxel alone, but experienced a prolonged stabilization of disease after crossing over. These three patients progressed at 0.95, 1.6, and 1.7 months on paclitaxel alone; following treatment with STA-4783 plus paclitaxel their subsequent progression occurred after 2.3 months, 5.5 months, and 4.2 months, respectively.

Results From the Lead-in, Phase 2a Stage of the Trial

Our clinical trial employed a two-stage, lead-in design, with an open-label, single-arm Phase 2a stage prior to the commencement of the blinded, randomized, controlled Phase 2b stage. The objective of the Phase 2a stage was to evaluate the safety of STA-4783 plus paclitaxel and to assess whether it demonstrated sufficient activity to warrant further study. A total of 31 patients were enrolled in this stage, of which 28 were treated at the STA-4783 dose of interest (213 mg/m^2). Of these 28 patients, four achieved an objective response as assessed by RECIST, and an additional 11 achieved stable disease, for a total non-progression rate of 15 out of 28 (54%). This met the pre-specified efficacy criteria, supporting the decision to proceed with enrolling the 81 additional patients for the Phase 2b stage of the trial. The addition of STA-4783 to paclitaxel was well tolerated on the weekly schedule.

Plans for Our Phase 3 Clinical Trial

Based on the results of our Phase 2b clinical trial, we intend to initiate a Phase 3 clinical trial of STA-4783 in combination with paclitaxel in the middle of 2007. In January 2007, we had initial discussions with the FDA regarding our Phase 3 clinical trial and are incorporating their guidance into the trial design. We currently expect the trial to consist of patients who have been diagnosed with stage IV metastatic melanoma and who have received no prior treatment with a cytotoxic chemotherapeutic agent. This patient population has been selected based on data from our Phase 2b clinical trial, as well as our understanding of the mechanism of action of STA-4783.

As in the Phase 2b clinical trial, we anticipate that our Phase 3 clinical trial will evaluate treatment with STA-4783 plus paclitaxel versus paclitaxel alone. Also, as in the Phase 2b trial design, we anticipate that the Phase 3 trial will have progression-free survival as the primary endpoint. Based on our statistical and clinical assumptions, we estimate that enrolling and evaluating between 250 and 300 patients will be sufficient to detect a statistically significant difference between the two treatment arms for the progression-free survival primary endpoint. However, based on our discussions to date with the FDA and with our medical advisors, we also plan to assess overall survival as a secondary endpoint which will require us to recruit a greater total number of patients. We estimate that enrolling approximately 600 patients will be sufficient to detect a statistically significant difference in overall survival between the two treatment arms.

Our current plan is to conduct a single, planned analysis for the primary endpoint of the trial on the smaller number of patients needed for the progression-free survival analysis. Based on the FDA's recommendation, we intend to continue the trial after the progression-free survival analysis until the larger, total number of patients has been treated and assessed for the overall survival secondary endpoint. More specific details of clinical trial design, including estimates of timing for the analysis of the progression-free survival primary endpoint, are currently being developed and will be guided by

additional discussions with the FDA and our medical and regulatory advisors. Although the foregoing description represents our current plans for the Phase 3 trial design, based on our initial discussions with the FDA and feedback from our advisors, these plans may change based on new data or information that we receive, new analyses that we conduct, as well as further discussions we have or input we receive from the FDA or our advisors.

Additional Clinical Trial Results

We completed a Phase 1 clinical trial of STA-4783 in combination with paclitaxel in October 2004. This clinical trial, which enrolled 35 patients, was designed to assess the safety, pharmacokinetics, and efficacy of STA-4783 with paclitaxel in a broad cancer patient population. The combination of STA-4783 plus paclitaxel was well tolerated, with minimal toxicity attributed to STA-4783 at all doses tested. Partial response or stable disease was observed in several cancer types, including melanoma, ovarian, Kaposi's sarcoma, angiosarcoma, parotid gland adenocarcinoma, colorectal, pancreatic and paraganglioma. In some of these patients, their cancers had previously progressed to more advanced stages during treatment with paclitaxel alone.

In addition to measuring safety, efficacy, and pharmaceutical properties in our Phase 1 clinical trial, we also measured biological markers of activity, including levels of circulating heat shock protein 70, or Hsp70, in the blood. We observed time-dependent and dose-dependent increases in levels of Hsp70 circulating in the blood of patients following administration of STA-4783 plus paclitaxel. At the most relevant therapeutic doses, following treatment with STA-4783 plus paclitaxel, every patient was observed to have substantial increases of circulating Hsp70.

Based on the promising signs of activity and safety results we observed in our Phase 1 clinical trial, we initiated Phase 2 clinical trials in malignant melanoma, soft tissue sarcoma, and non-small cell lung cancer. Together these trials have enrolled approximately 300 patients at over 50 medical centers throughout the United States and Canada. These trials were designed to assess response rates, non-progression rates, and progression-free survival, and to further expand the safety database for STA-4783.

We completed a Phase 2 clinical trial of STA-4783 in 84 patients with soft tissue sarcoma in 2005, the results of which were inconclusive. We designed this two-stage Phase 2 clinical trial to assess activity based on response rate and non-progression rate, or NPR. This clinical trial utilized a single-arm design. All patients received weekly treatments of the combination of paclitaxel (80 mg/m ^{2}) and STA-4783 (213 mg/m^2) for three weeks, followed by one week off-treatment. These four-week cycles were repeated until the earlier of disease progression, or a minimum of four months. We enrolled patients with soft tissue sarcoma who had failed at least one prior chemotherapy treatment. In the first stage, 30 eligible patients were evaluated for objective response or disease stabilization after three months and met the predefined criteria for expansion of enrollment. Upon completion of the trial, the Kaplan-Meier estimate of NPR at three months was 35%, with a 95% confidence interval of between 24.3% and 45.8%. A recent publication by Van Glabbeke, et al., proposed a criterion of NPR at three months >=40% to suggest drug activity in this indication. Given that the observed confidence interval includes 40%, this result did not definitively establish evidence of clinical activity or lack thereof. The observed safety profile of STA-4783 plus paclitaxel was acceptable. We are in the process of analyzing these data further to assess future development plans in sarcoma.

We completed a Phase 2 clinical trial of STA-4783 in 103 patients with non-small cell lung cancer in 2005. We designed this two-stage trial to compare the effect of a standard first-line lung cancer combination therapy, paclitaxel and carboplatin, with the effect of this same combination therapy plus STA-4783. Patients included in this study were diagnosed with either stage IIIb or stage IV non-small cell lung cancer and had not received prior chemotherapy. The objective of the first stage, open-label portion was to determine the recommended dose for the second stage. In the second stage, patients

were randomly assigned either to receive STA-4783 plus paclitaxel and carboplatin, or to receive paclitaxel and carboplatin alone. Patients received one treatment of paclitaxel and carboplatin, with or without STA-4783, every three weeks. These three-week cycles were repeated until the earlier of disease progression or completion of six cycles. Efficacy was assessed using RECIST, and the primary endpoint in this clinical trial was time-to-progression. No improvement was observed in time-to-progression between STA-4783 plus paclitaxel plus carboplatin, compared to paclitaxel plus carboplatin. In comparison to patients in our Phase 2b metastatic melanoma trial, patients in this clinical trial received both a less frequent dose of STA-4783 (once every three weeks compared to once a week for three weeks), and a lower total dose of STA-4783 during each monthly cycle (266 mg/m^2 compared to 639 mg/m^2). We are considering the possibility of performing a future study of STA-4783 in non-small cell lung cancer at a more frequent dosing schedule and higher total monthly dose.

Safety Results from all Clinical Trials to Date with STA-4783

In order to assess the safety profile of STA-4783 based on all of the clinical trials completed to date, we collected and integrated the adverse event data for all 352 subjects who participated in the six clinical trials conducted with STA-4783, including the Phase 2b melanoma trial.

Of the 352 subjects in these trials, 298 received the STA-4783 plus paclitaxel combination. Of these 298 subjects, 239 received STA-4783 in combination with paclitaxel, and 59 received STA-4783 in combination with paclitaxel and carboplatin. All participating subjects suffered from solid tumor cancers.

The following table compares the grade 3 or higher adverse events that were reported in greater than 4% of subjects in either the STA-4783 plus paclitaxel treatment groups or the paclitaxel alone treatment group.

Grade 3 or Higher Adverse Events

	STA-4783 + Paclitaxel N=239(1)	Paclitaxel Alone N=28(2)
Neutropenia	15 (6%)	0
Fatigue	8 (3%)	2 (7%)
Neuropathy	4 (2%)	2 (7%)
Back Pain	4 (2%)	2 (7%)
Pain	2 (<1%)	3 (11%)

(1) Of the 239 patients, 224 received the same or higher dose of STA-4783 plus paclitaxel as we used in the Phase 2b melanoma trial. Of these 224 patients, 201 patients were on the same once per week schedule as in the Phase 2b melanoma trial and 23 patients were on a once every three week schedule.

(2) Consists of the 28 patients in the control arm of the Phase 2b melanoma trial.

Consistent with the results observed in our melanoma Phase 2b trial, there was a small increase in observations of neutropenia: 6% of STA-4783 plus paclitaxel subjects versus 0% of the paclitaxel alone subjects. Frequencies of other grade 3 or higher adverse events were similar for the two treatment groups, and in some cases, occurred at slightly lower frequencies in the STA-4783 plus paclitaxel group. In addition, we did not observe any clinically relevant trends in any of the other hematology, serum chemistry, or urinalysis testing on these patients.

Frequencies of adverse events of all grades of severity were comparable between the two groups. Adverse events that were reported as occurring in at least 20% of subjects who received STA-4783 plus paclitaxel were as follows, for the combination and for paclitaxel alone, respectively: asthenic conditions (54% vs. 54%), nausea and vomiting symptoms (44% vs. 54%), alopecia (44% vs. 54%), musculoskeletal and connective tissue signs and symptoms (36% vs. 43%), edema (27% vs. 21%), gastrointestinal atonic and hypomotility disorders (24% vs. 29%), non-infective diarrhea (23% vs. 18%), peripheral neuropathies (23% vs. 21%), anemias (21% vs. 21%), appetite disorders (21% vs. 21%), joint related signs and symptoms (21% vs. 11%), and coughing and associated symptoms (21% vs. 29%). Asthentic conditions generally refers to lack of strength or weakness throughout or in a particular area of the body. Edema is swelling caused by fluid accumulation in bodily tissues. Gastrointestinal atonic and hypermotility disorders generally refer to muscle weakness and decreased movement, respectively, in the gastrointestinal tract. Anemia is the abnormal reduction in red blood cells.

We believe the integrated analysis of adverse event data from all 239 subjects who received the STA-4783 plus paclitaxel combination shows that STA-4783 plus paclitaxel was well tolerated and that the adverse events and laboratory results were similar to those expected for paclitaxel alone.

STA-4783 Mechanism of Action

STA-4783 is a novel, injectable small molecule that we believe rapidly and potently induces the generation of reactive oxygen species, or ROS, in cancer cells, increasing the level of oxidative stress in the cancer cell.

Oxidative stress in a cell is characterized by the increased presence of various ROS molecules, such as superoxide and hydrogen peroxide; stress proteins, such as Hsp70; and other proteins that ordinarily confer a protective response to the cell, such as metallothioneins and antioxidants. Cancer cells are known to have a substantially heightened level of oxidative stress compared to normal cells. These levels are close to an internal breaking point, above which further increases in ROS levels can lead to programmed cell death, or apoptosis.

We believe that STA-4783 causes cancer cell death by inducing ROS at such high levels and for such an extended duration that the cellular protective responses are no longer sufficient, triggering programmed cell death. In addition, we believe that the favorable safety profile that has been observed clinically and preclinically with STA-4783 may be due to the pronounced difference between cancer cells and normal cells in their respective ability to recover from such an increase in oxidative stress. While cancer cells exist close to an oxidative stress breaking point, normal cells have far lower levels of ROS and significantly greater anti-oxidant capacity.

The evidence that the primary mechanism of action of STA-4783 is through induction of ROS includes the following:

- Gene transcript profiles of cancer cells before versus after application of STA-4783 show the characteristic signatures of an immediate, potent oxidative stress response.
- Direct cellular measurements of ROS show strong time-dependent and dose-dependent induction by STA-4783.
- The anti-cancer effects of STA-4783 are eliminated by applying antioxidants which scavenge ROS or an inhibitor which blocks the generation of ROS.

Once ROS levels in cancer cells exceed the breaking point, cell death occurs through two processes:

- *Triggering of apoptosis.* Programmed cell death occurs through the release of cytochrome c from mitochondria, activating the caspase pathway.

- *Increase in signals targeting the cancer cell for elimination.* One example of such a signal is the increased expression of Hsp70 on the cancer cell surface, which activates the natural killer, or NK, cells of the immune system to attack and kill the cancer cell.

In preclinical *in vitro* experiments, we have observed strong anti-cancer activity of STA-4783 across a broad range of cancer cell types, as well as an enhancement of cancer cell killing by paclitaxel, which is known to be sensitive to the level of ROS in cells. In preclinical *in vivo* experiments, we observed minimal activity of STA-4783 as a single agent at therapeutically relevant doses, but a strong enhancement of tumor cell killing by the taxanes paclitaxel and docetaxel in a variety of animal models of cancer, including breast, lung, lymphoma, colorectal and melanoma. Our preclinical safety studies showed that the addition of STA-4783 added little or no toxicity to that seen with paclitaxel alone, and that STA-4783 has a relatively high therapeutic index, or margin between effective dose and toxic dose.

Elevated oxidative stress is one of the most basic features of cancer cells that differentiates them from normal cells. By taking advantage of this fundamental difference, we believe STA-4783 offers the potential for a novel anti-cancer approach that is broadly effective across cancer types in conjunction with ROS-sensitive chemotherapeutic agents such as paclitaxel, without adding substantially to the toxicity of those agents.

Additional Cancer Types for Future Clinical Development

Based on the activity seen in a broad range of tumor models in preclinical experiments, and our understanding of the mechanism of action, which is not specific to melanoma, we believe that STA-4783 has the potential to treat many forms of cancer. We prioritize our clinical development plans based on a number of criteria, including scientific rationale and degree of unmet medical need. Based on these criteria, we believe there are several attractive opportunities for the further clinical development of STA-4783, including:

- *Cancers having elevated levels of ROS.* We believe that cancer types having elevated levels of oxidative stress may be particularly susceptible to the increase in ROS caused by treatment with STA-4783. In addition to melanoma, other cancer types known to have high levels of oxidative stress include pancreatic, prostate, and breast cancer.

- *Cancers in which taxanes are used.* Based on the synergistic activity of STA-4783 and taxanes seen in our melanoma clinical trial and in our preclinical models in other cancer types, we believe other cancers in which taxanes are used may be promising opportunities. Prostate, breast, ovarian, and lung cancers are commonly treated with taxanes. In addition, taxanes have been tested in pancreatic cancer, with significant room for improvement and a high unmet need. In our Phase 2 clinical trial in non small-cell lung cancer, we studied a dosing regimen of once every three weeks; we believe a more frequent dosing schedule, with a higher total monthly drug exposure, such as we used in our melanoma trial, may warrant further exploration.

- *Cancers in which we have observed signs of activity of STA-4783 in our Phase 1 clinical trial.* In our Phase 1 clinical trial, partial response or disease stabilization was observed in several cancer types, including melanoma, ovarian, Kaposi's sarcoma, angiosarcoma, parotid gland adenocarcinoma, colorectal, pancreatic and paraganglioma. In particular, one patient with a history of recurrent ovarian cancer had a documented partial response to treatment with STA-4783 plus paclitaxel after having failed multiple prior chemotherapeutic regimens. This patient received a Special Protocol Exception from the FDA in order to continue on STA-4783 plus paclitaxel beyond the end of the clinical trial and received a total of eight cycles of treatment. We believe that these cancer types may warrant further exploration.

- *Adjuvant treatment of earlier-stage melanoma.* Adjuvant therapy with interferon alfa-2b, an immunotherapy marketed as Intron A by Schering-Plough, is FDA-approved for use following

surgical removal of melanoma to reduce the likelihood of disease recurrence. We believe the mechanism of action and the favorable safety profile of STA-4783 suggest a potential role in adjuvant therapy.

We are evaluating these opportunities and expect to initiate Phase 2 clinical trials in one or more of these indications in mid-2007.

New Formulations

To date, all of our clinical trials have been conducted using the free acid form of STA-4783, which we intend to continue to use in our clinical trials planned for 2007 as well as our commercial product if STA-4783 is approved. This form is dissolved in the paclitaxel solution, diluted in a saline infusion bag and co-administered via the same infusion line. In order to use the free acid form of STA-4783 with other oncology products, including taxanes other than paclitaxel, it must be dissolved in an organic solvent, which may cause additional toxicity due to the presence of the organic solvent. We have also developed a water soluble salt form of STA-4783 that may be more easily used with other taxanes and oncology products that have a different formulation than paclitaxel. We intend to explore the use of this new salt form of STA-4783 and alternative STA-4783 formulations in future clinical trials in order to expand the potential use of STA-4783 in combination with other chemotherapies. In addition, in preclinical studies, we have observed oral bioavailability of STA-4783, and are exploring the possibilities of an oral formulation, such as a tablet or capsule.

Other Oncology Programs

STA-9090 and Our Hsp90 Inhibitor Program

We are using our internal chemistry and drug optimization expertise in the area of heat shock proteins to develop novel synthetic small molecule inhibitors of Hsp90 for the treatment of cancer. STA-9090 is a novel chemical entity that selectivity inhibits the activity of Hsp90. This program is currently in preclinical development, and we expect to file an IND in mid-2007.

Hsp90 is a chaperone protein that regulates the folding, stability, and function of numerous signaling proteins that trigger uncontrolled proliferation in cancer cells. Many of the proteins that require Hsp90 for their folding and activity are kinases that regulate tumor survival, proliferation, and angiogenesis. These include well-recognized cancer targets such as Bcr-Abl, Her2, EGFR, c-Kit, c-Met, Flt3, and BRAF, which are the targets of approved anticancer drugs such as Gleevec, Herceptin, Tarceva, and Erbitux, all of which are direct inhibitors of these kinase proteins. We believe that inhibiting kinases indirectly, by disrupting the chaperone activities of Hsp90, provides two advantages: first, a means to simultaneously attack multiple cancer-promoting kinases; and, second, an ability to kill tumor cells with mutated kinases that have lost responsiveness to direct kinase inhibitors. Furthermore, since cancer cells have far greater levels of active Hsp90 than normal cells, we believe that inhibitors of Hsp90 may selectively halt proliferation of tumor cells and thereby cause cancer cell death.

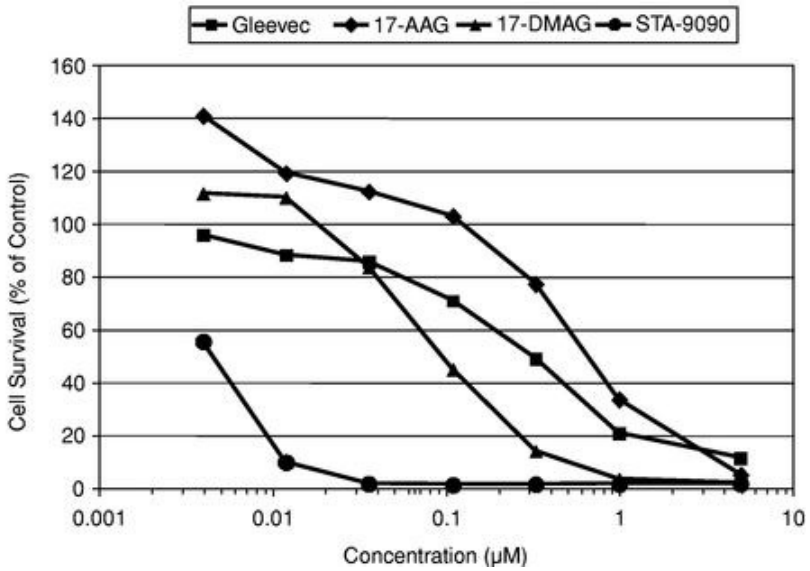
A number of companies have programs targeting inhibition of Hsp90 for the treatment of various forms of cancer. Based on results from experiments we conducted in both cell models and preclinical animal models, we believe that our lead compound, STA-9090, displays substantially higher potency than competing Hsp90 inhibitors in development. In addition to the higher potency of STA-9090 in certain cancer types, these experiments also demonstrated that STA-9090 may be active against cancer cell types for which other Hsp90 inhibitors have not shown activity. We believe these findings suggest a potential competitive advantage for STA-9090 in treating those cancers.

To our knowledge, the Hsp90 inhibitors that are furthest along in clinical development are 17-AAG, or tanespimycin, and 17-DMAG, or alvespimycin. These compounds are being developed by Kosan for several cancer types including multiple myeloma, breast cancer, and melanoma. Both of

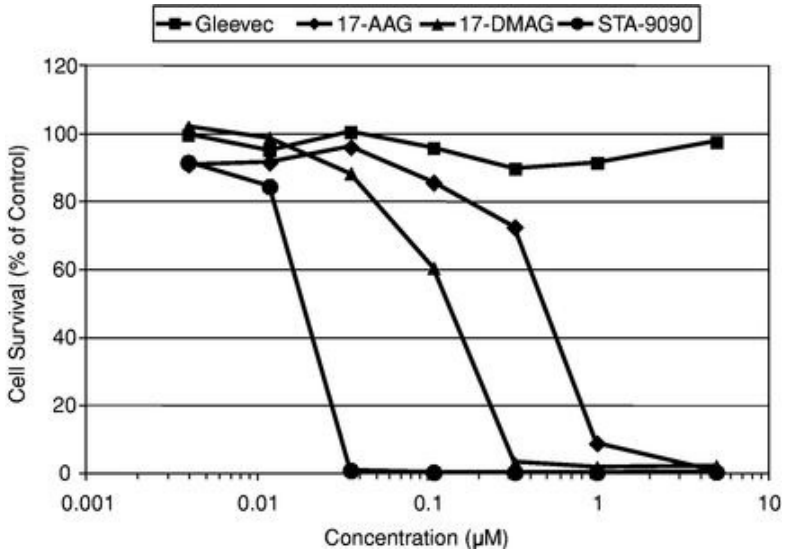
these compounds are derivatives of the natural product, geldanamycin, and have been observed to have certain serious side effects, including liver toxicities. In contrast, STA-9090 is a novel small molecule compound that is not a geldanamycin derivative or analog. In addition, while 17-AAG and 17-DMAG have complex routes of synthesis, STA-9090 has a relatively simple route of synthesis.

In the figures below we illustrate what we believe are the two key potential advantages of our Hsp90 inhibitor, STA-9090: improved potency and the activity against cancers that have developed resistance to kinase inhibitors.

Improved potency. One of the several kinases that we have observed in preclinical testing to be more sensitive to STA-9090 than to other Hsp90 inhibitors is c-Kit. c-Kit plays a critical role in two cancer types: gastro-intestinal stromal tumors, or GIST, and acute myelogenous leukemia, or AML. The c-Kit gene is often mutated in cancers and can drive uncontrolled cancer cell proliferation. Inhibition of Hsp90 leads to the degradation and loss of c-Kit. In preclinical testing we have found that STA-9090 is more effective in causing the loss of c-Kit relative to other Hsp90 inhibitors such as 17-AAG and 17-DMAG. This loss of c-Kit leads to the death of those cancer types that depend upon c-Kit for their growth and survival. The figure below shows the result of an *in vitro* experiment we conducted comparing the activity of STA-9090 against human AML tumor cells with the two leading Hsp90 inhibitors, 17-AAG and 17-DMAG, and with the Bcr-Abl and c-Kit kinase inhibitor Gleevec. This figure shows that STA-9090 was 25-fold to 170-fold more effective in tumor cell killing than these other agents in this experiment, as measured by the IC_{50} (the dose that killed 50% of tumor cells).



Activity against cancers that develop resistance to kinase inhibitors. In patients who are treated for cancers with kinase inhibitors such as Gleevec, an initial period of responding to treatment can be followed by a relapse, in which the disease rapidly worsens and no longer responds to further treatment with that kinase inhibitor. This relapse is believed to be due to the appearance of new mutations in the target kinase. In contrast to direct kinase inhibitors, STA-9090 is an indirect kinase inhibitor that acts by inhibiting Hsp90 rather than the kinases themselves. STA-9090 therefore has the potential to be effective in inhibiting both the original and the mutant kinases. The figure below illustrates this point. In an *in vitro* experiment, a tumor cell line with a Gleevec-resistant mutation in c-Kit is no longer killed by Gleevec. In contrast, STA-9090 demonstrates potent killing of these cells. This figure also shows that STA-9090 is substantially more potent than the competing Hsp90 inhibitors, 17-AAG or 17-DMAG, in this model, as with the previous model.



In addition to the activity shown in cancer cells in the figures above, we have shown that STA-9090 is more potent than 17-AAG in a range of additional cancer cell models as well as in multiple preclinical animal models of human cancer types including lung, prostate carcinoma, breast, gastric, melanoma, lymphoma, multiple myeloma, acute myelogenous leukemia, and chronic myeloid leukemia.

We believe that our preclinical data suggest the potential for using STA-9090 to treat patients whose cancers have relapsed following treatment with small molecule kinase inhibitors such as Gleevec or Tarceva. In addition, we believe that knowledge of which cancer-causing proteins are most susceptible to treatment with STA-9090 will help us to focus our clinical development on cancer types most likely to respond to treatment with our drug candidate.

STA-9584—Our Vascular Disrupting Agent

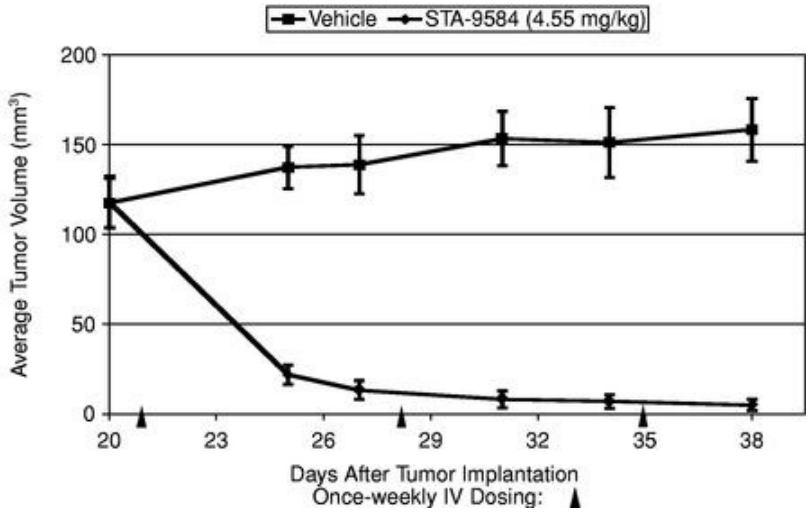
STA-9584 is a novel anticancer agent with a dual mechanism of action: STA-9584 disrupts the vessels feeding tumors, which can choke off the supply of oxygen and nutrients, and, in addition, STA-9584 directly causes tumor cell death by inhibiting microtubules, which are cellular structures that play an important role in cell division and proliferation. STA-9584 has demonstrated strong activity in a range of animal models of human tumors, including prostate, lung, breast, melanoma, and lymphoma. This program is in preclinical development.

Because rapidly growing cancer cells have a high demand for oxygen and nutrients, tumors cause new blood vessels to grow in order to supply those needs. Those new vessels differ from normal blood vessels in that they are fragile and weak, forming disorganized and tortuous networks. We believe that drugs that disrupt tumor vessels, or tumor vasculature, could therefore starve tumor cells of oxygen and nutrients, leading to the rapid death of these cells, including tumor cells resistant to other therapies. Vascular disruption contrasts with anti-angiogenic approaches, such as the proposed mechanism of action of approved cancer drugs such as Avastin, which inhibit the growth of new tumor blood vessels but are not believed to affect established tumor vasculature.

To our knowledge, of the drug candidates in the category of vascular disrupting agents, combretastatin is one of the furthest along in development. We believe the dual mechanism of action of STA-9584 represents an important difference from combretastatin, in that STA-9584 both disrupts tumor vasculature and directly kills tumor cells through inhibiting microtubules. Consistent with this dual mechanism, we have observed in our preclinical models that STA-9584 causes tumor cell death throughout the tumor, both at the tumor core and rim, whereas vascular disrupting agents such as

combretastatin cause tumor cell death primarily at the core of tumors, where the demand for oxygen and nutrients is most pronounced.

We believe the high potency of STA-9584 and acceptable therapeutic index in our preclinical models make this compound a promising candidate for treatment of a wide range of solid-tumor cancers. An example of the potency of STA-9584 is shown in the figure below, in which treatment with STA-9584 leads to complete tumor elimination in a preclinical model of prostate cancer. In this preclinical study, PC-3 human prostate cancer cells were implanted subcutaneously into nude mice. Once tumors reached over 100 mm ^{3} in size, mice were treated with a placebo control or STA-9584 by intravenous injection once per week. Three doses of STA-9584 caused the regression of tumors.



Inflammatory Disease Programs

We have the following two inflammatory disease programs in development:

- *Apilimod (STA-5326).* Apilimod is our novel, orally administered, small molecule drug candidate that inhibits the production of the cytokines interleukin-12, or IL-12, and interleukin-23, or IL-23, which are believed to be important regulators of the biological processes underlying certain autoimmune and inflammatory diseases. We are currently conducting a Phase 2a clinical trial in patients with rheumatoid arthritis and sponsoring a Phase 2a clinical trial in patients with CVID. We expect to report results from both of these trials in 2007.

- *CRAC ion channel inhibitors.* We are developing inhibitors of calcium release-activated calcium, or CRAC, ion channels expressed on immune cells, for the treatment of autoimmune diseases, transplant rejection, asthma, and allergy. We have discovered a family of novel, small molecule, orally administered CRAC ion channel inhibitors that are both selective and highly potent.

Inflammatory Disease Background

Inflammatory diseases are typically caused by aberrant activity of the immune system. The immune system normally protects the body from injury and infection, but in autoimmune diseases it attacks and damages the body's own tissues. Major autoimmune diseases include rheumatoid arthritis, psoriasis, Crohn's disease, and multiple sclerosis. Together, these diseases afflict over seven million people in the United States and over 21 million people worldwide.

Despite the availability of numerous therapeutic options for these diseases, inflammatory diseases remain major causes of impairment of daily activities, reduced quality of life, significant disability, and sometimes death. Current therapeutic treatments for chronic inflammatory diseases have the potential

to cause musculo-skeletal, endocrinologic, neurologic, and metabolic side effects, which can limit their long-term use. The limitations of conventional treatments, together with a growing understanding of the pathogenesis of inflammatory diseases, have stimulated significant interest in the development of targeted immune modulators for the management of chronic inflammatory diseases.

Apilimod (STA-5326) and Our Oral IL-12/23 Inhibitor Program

We believe we have discovered the first oral, small molecule, selective inhibitors of the cytokines IL-12 and IL-23. We have conducted or sponsored eleven Phase 1 and Phase 2 clinical trials with our lead compound, apilimod, also designated STA-5326, or its salt form, apilimod mesylate, also designated STA-5326m. To our knowledge, there are no other oral, selective IL-12 and IL-23 inhibitor drug candidates from other companies currently in clinical development. Although our clinical trials in psoriasis and Crohn's disease did not achieve their primary endpoints, we have ongoing Phase 2a clinical trials of apilimod for rheumatoid arthritis and CVID, and we continue to believe that oral small molecules targeting IL-12 and IL-23 represent a promising therapeutic approach. We may continue to pursue this program with new generations of compounds that improve upon the pharmaceutical properties of apilimod.

The IL-12 cytokine is an important "master switch" that triggers the immune response of the T cell known as T helper type 1, or T_h1. T cells play a critical role in the coordination of the body's immune response, and while T_h1 cells are normally involved in the body's defense against intracellular attack by bacteria and other micro-organisms, an overactive T_h1 response can lead to various autoimmune or inflammatory diseases including Crohn's disease, psoriasis, rheumatoid arthritis, multiple sclerosis, and CVID. The IL-23 cytokine is critical to the generation of the T cells which produce other pro-inflammatory proteins believed to be important to maintaining the immune response. We believe that the Phase 2 clinical trial results observed with anti-IL-12/23 antibody therapies validate the inhibition of IL-12/23 activity as a promising approach for the treatment of inflammatory and autoimmune diseases.

Rheumatoid Arthritis

Rheumatoid arthritis is a chronic autoimmune disease that is primarily characterized by joint synovial inflammation that can lead to long-term joint damage, chronic pain, loss of function and disability. Over two million people suffer from the disease in the United States. We are currently conducting a randomized, placebo-controlled Phase 2a clinical trial of apilimod in rheumatoid arthritis patients with moderate to severe disease. All patients in this clinical trial are to be treated with methotrexate, a commonly used drug to treat rheumatoid arthritis, in addition to receiving either apilimod or placebo. The primary endpoint of this trial is based on an assessment of markers of inflammation in joint tissue after four to eight weeks of treatment. We believe that tissue assessments will provide an objective measure that will allow conclusions regarding potential efficacy to be based on a smaller number of patients. We plan to enroll approximately 20 patients and expect results from this trial to be available in 2007.

Common Variable Immunodeficiency

CVID is a disease characterized by the defective production of antibodies, which exposes patients to increased risk of life-threatening infections and, in some patients, autoimmune conditions and gastrointestinal diseases. In addition, CVID patients are at increased risk of cancer and inflammatory conditions. The incidence of CVID is poorly understood and is estimated to be between 1:25,000 and 1:66,000, with the highest incidence seen in Caucasian and European populations. More than 10% of CVID patients experience gastrointestinal manifestations that are believed to be associated with high levels of IL-12 expression in the digestive tract. In collaboration with the National Institutes of Health, we initiated an exploratory open-label Phase 2a clinical trial of apilimod in up to five CVID patients

with gastrointestinal manifestations. This study is designed to assess changes in clinical symptoms, changes in objective measures of disease activity, including tests for malabsorption, and changes in biopsy samples of the gastrointestinal tract, including measurements of IL-12 production in the gut, before and after treatment with apilimod. We expect results from this trial to be available in 2007.

Psoriasis

Psoriasis is a chronic, inflammatory skin disorder that is characterized by thickened, red areas of skin that are covered with scales. The area of skin affected can range from discrete, localized patches, to extensive areas of the body. The joints, nails, and mucous membranes may also be affected by the disease. Chronic plaque psoriasis is the most common form of psoriasis. This disease involves the formation of plaques, which are circular-to-oval, elevated, and often scaly skin lesions that contain swollen blood vessels and infiltrating immune cells. Affected areas are characterized by itching, swelling, and pain, all of which can impair daily activities and sleep.

We conducted two complementary Phase 2 clinical trials of apilimod for the treatment of moderate to severe chronic plaque psoriasis. In each of these trials patients were treated for 12 consecutive weeks. One psoriasis trial was an open-label Phase 2a clinical trial designed to assess the biological response to apilimod through histological studies of skin biopsies. While the data showed signs of activity, as assessed both histologically and clinically, strong clinical benefit was not demonstrated. Another psoriasis trial was a double-blind, randomized, placebo-controlled, multicenter Phase 2b clinical trial of 212 patients. Despite observing a difference between apilimod and placebo, the primary endpoint of the trial was not achieved, and the magnitude of clinical benefit did not warrant advancement into Phase 3 clinical trials at the doses and with the formulation tested. We are exploring whether inadequate distribution of apilimod to the skin could underlie the insufficient clinical benefit observed in these clinical trials and are developing a topical formulation of apilimod to test this hypothesis.

Crohn's Disease

Crohn's disease is a chronic inflammatory bowel disease characterized by inflammation at points throughout the length of the gastrointestinal, or digestive, tract. Symptoms can be severe and include abdominal pain, frequent diarrhea and intestinal bleeding. In addition, patients with Crohn's disease may experience malnutrition and an increased risk of colorectal cancer.

We initiated three Phase 2 clinical trials in moderate-to-severe Crohn's disease: a 73-patient Phase 2a clinical trial, a planned 282-patient Phase 2b clinical trial and a planned 12-patient biomarker trial. The Phase 2a clinical trial was an open-label, dose-escalating study to assess the safety, pharmacokinetics, and efficacy of apilimod. In this trial, a capsule formulation containing the free base form of apilimod was studied. Promising signs of activity were observed. In the Phase 2b study, we switched formulation to a tablet containing the mesylate form of apilimod. This Phase 2b study was a double-blind, randomized, placebo-controlled, multicenter clinical trial with two treatment arms and one placebo arm. As specified in the protocol, an interim analysis was performed after half the patients expected to be enrolled in the trial had completed treatment. This analysis indicated a low likelihood of achieving the primary endpoint in the trial, and thus, the Phase 2b and biomarker trials were terminated at that point.

We are currently exploring whether the change in formulation and drug form, from the free base capsule form used in the Phase 2a study to the mesylate tablet form used in the Phase 2b study, could underlie the lower response rates observed in the Phase 2b study, or whether the Phase 2a response rates were contaminated with substantial placebo effect bias. We have initiated work on a follow-on generation of IL-12/23 inhibitors, which we believe may have improved pharmaceutical properties.

CRAC Ion Channel Inhibitors

Ion channels have proven to be very attractive targets for small molecule drug development. Examples of successful ion channel modulating drugs include Norvasc, which is marketed by Pfizer for the treatment of hypertension, and Ambien, which is marketed by Sanofi-Aventis for the treatment of insomnia. Ion channel modulators developed to date target channels on excitable cells, which are cells that transmit electrical signals, such as muscle cells and nerve cells, and have been primarily developed for treating cardiac or central nervous system conditions. While ion channels in excitable cells are involved in the electrical signaling of those cells, ion channels are also known to play an important role in the signaling pathways and function of certain non-excitable cell types, such as immune cells.

We are developing small molecule inhibitors of calcium release-activated calcium, or CRAC, ion channels expressed on immune cells. The CRAC ion channel is the primary route for calcium entry into T cells and mast cells. Calcium entry regulates multiple immune cell processes, including T cell proliferation and cytokine secretion, which are important for initiating and sustaining an inflammatory immune response. The relevance of inhibiting this biological pathway has been validated by the clinical and market success of the calcineurin inhibitors, cyclosporin and tacrolimus, in treating autoimmune diseases and transplant rejection. The calcineurin inhibitors, however, act on both immune and non-immune cell types and have substantial toxicities. By more selectively inhibiting the same biological pathway, therapies that inhibit CRAC ion channels offer the potential of modulating the immune system with fewer toxicities. Such therapies may hold promise for treating immune disorders such as rheumatoid arthritis, psoriasis, multiple sclerosis, transplant rejection, allergy, or asthma.

We have discovered a family of novel, small molecule, orally administered CRAC ion channel inhibitors that are both selective and highly potent. We have demonstrated in preclinical experiments that these compounds inhibit the production by immune cells of multiple critical pro-inflammatory cytokines, such as IL-1, IL-2, IL-6, and TNF-α, which are critical to immune disorders such as rheumatoid arthritis and transplant rejection. We have also demonstrated that some of these compounds inhibit mast cell degranulation and the release of histamines, which is believed to be important for the treatment of allergy and asthma. We have shown that our compounds are effective in multiple animal models of immune diseases, including models of arthritis. This program is in the lead optimization stage of discovery. We may seek to enter into a strategic partnership or collaboration with respect to this program.

Our Drug Discovery Capabilities

Our drug discovery approach is based on the close integration and rapid cycle times among our chemistry, biology, and pharmaceutical development groups. Drug candidates are typically identified using novel chemical structures from our chemical compound library in cell-based assays that are designed to preserve the complexity of biological signaling. Early *in vivo* testing and a rapid optimization process allow us to generate a high number of promising leads from our screening hits, improve the profiles of our compounds, and, in some cases, discover novel pathways or mechanisms of action with the potential to define entirely new categories of treatment.

Our approach integrates the following capabilities and resources:

- *Unique chemical compound library.* Our chemical library contains over 100,000 small molecules and numerous plant extracts collected from universities, non-profit institutions, other organizations, and commercial sources. Many of our compounds are proprietary and not available from commercial sources. This library represents a diverse and distinct set of chemical structures that was not generated using combinatorial chemistry and continues to be a valuable source of lead compounds for drug discovery. We are continuing our compound collection efforts. In addition, for each of our discovery programs we build focused libraries dedicated to

particular drug targets. We have the three-dimensional structure of most of our compounds, allowing us to use computer-based, or *in silico*, screening to identify new drug candidates.

- *Broad set of screening assays.* We have high throughput screening capabilities linked to our chemical library that facilitate the rapid identification of new drug candidates. We have developed a wide variety of biochemical and cell-based *in vitro* assays designed to identify promising compounds for treating cancer, immune disorders and other diseases, which form the basis of our initial screening efforts. In addition to assays for identifying new compounds, we have also developed assays we use for early optimization of safety and pharmacokinetic properties.

- *Robust in vivo testing capabilities.* We have substantial *in vivo* testing facilities that we use for evaluating the safety, efficacy, and pharmaceutical properties of our compounds, including absorption, distribution, metabolism, excretion, and toxicology properties. These facilities are equipped for detailed experimental measurements and surgical tasks, such as the rodent microsurgery we use for sophisticated toxicology assessments. We have experience with a wide range of animal models of disease, including multiple models in cancer, inflammatory diseases and metabolic diseases. We believe the ability to complete early testing of compounds *in vivo*, internally and without dependencies on third parties, is a valuable advantage in our ability to rapidly optimize the pharmaceutical properties of our most promising compounds.

- *Multi-functional chemistry capabilities.* We possess a full range of chemistry capabilities, including medicinal chemistry, analytical chemistry, physical chemistry, process development and computational chemistry. Our approach to medicinal chemistry applies the rigorous exploration of permutations of biologically active molecular components to optimize lead compounds. Our in-house process development capability of characterizing and specifying manufacturing processes for our compounds allows us to reduce dependencies on third parties and is an important advantage in our ability to successfully commercialize our drug candidates.

- *Methods for novel target elucidation and validation.* Our scientists use expression profiling, RNA interference, affinity purification, proteomics, electrophysiology, and other methods to identify the therapeutic intervention points of novel, promising compounds.

Manufacturing

Our drug candidates and preclinical compounds are small molecules that can be readily synthesized by processes that we have developed. Utilizing our medicinal chemistry and process development capabilities, we have developed manufacturing processes to produce the active pharmaceutical ingredients, or API, for our drug candidates. We also have the internal capability to synthesize small molecule compounds in quantities of up to several hundred grams for use in our preclinical studies, including proof-of-concept studies in animal models, early pharmacokinetic assays, initial toxicology studies, and formulation development. We currently contract with third parties for the synthesis of all materials used in our clinical trials and rely on third party manufacturers for the supply of our drug candidates in bulk quantities and for the production of suitable dosage forms.

The starting materials and reagents required for synthesizing our drug candidates and preclinical compounds are commercially available from multiple sources. We have established a quality control and quality assurance program, including a set of standard operating procedures, analytical methods, and specifications, designed to ensure that our drug candidates are manufactured in accordance with the FDA's current Good Manufacturing Practices, or cGMP, and other applicable domestic and foreign regulations. We have selected manufacturers that we believe comply with cGMP and other applicable regulatory standards. We do not currently expect to manufacture cGMP material internally for our clinical trials nor undertake the commercial scale manufacture of our drug candidates after approval.

We are discussing with our current suppliers and other third party manufacturers the long-term supply and manufacture of these and other drug candidates we may develop.

STA-4783

We are currently working with two contract manufacturers to produce STA-4783 in its free acid form, which is the active pharmaceutical ingredient, or API, that will be used in the Phase 3 clinical trial of STA-4783 for metastatic melanoma and any Phase 2 clinical trials we may initiate in other cancer indications in 2007. We intend to use one of these manufacturers as the primary supplier of STA-4783 API and the other as a backup manufacturer for the clinical trials initiated in 2007 and potentially, for commercial supply in the future. We have contracts with each of these manufacturers to produce STA-4783 API in quantities we believe will be sufficient for our current clinical trial needs, but to date, these manufacturers have only produced pilot batches of STA-4783, and there can be no assurances that they will be able to produce STA-4783 API in the quantities and to the specifications needed for our clinical trials. If the primary manufacturer we choose to provide STA-4783 API should become unavailable to us for any reason, we believe the backup manufacturer will be able to provide us with sufficient STA-4783 API with little or no delays in our trials. If both of these manufacturers should become unavailable, we believe that there are a number of potential replacements, as our processes are not technically complex nor manufacturer-specific. However, we may incur some added cost and delay in identifying or qualifying such replacements, including delays associated with transferring the process to the new manufacturer and conducting manufacturing runs. We do not currently have a contract with any manufacturer for commercial supply of STA-4783 API.

We intend to use a single manufacturer for the preparation of STA-4783 drug product. This preparation involves highly specialized processing, including the automated filling of vials with STA-4783 API. We believe that our selected manufacturer may be one of a limited number of third party contract manufacturers currently capable of conducting this process on our behalf. To date, this third-party manufacturer has verbally agreed and provided a term sheet to meet our manufacturing requirements for the planned Phase 3 clinical trial of STA-4783 for metastatic melanoma and additional Phase 2 trials of STA-4783 for other cancer indications in 2007. Although we are currently in discussions with this manufacturer regarding a contract for the supply of STA-4783 drug product for clinical trials and potentially, for commercial supply, there can be no assurances that we will be able to enter into a contract with this or another manufacturer on acceptable terms, if at all.

Sales and Marketing

We currently have no marketing, sales or distribution capabilities. In order to commercialize any of our drug candidates, we must develop these capabilities internally or through collaboration with third parties. In selected therapeutic areas where we feel that any approved products can be commercialized by a specialty sales force that calls on a limited and focused group of physicians, we currently plan to participate in the commercialization of these drug candidates. In therapeutic areas that require a large sales force selling to a large and diverse prescribing population, we currently plan to partner our drug candidates for commercialization.

Our plan is to retain commercial rights to our lead oncology drug candidate, STA-4783, in North America either exclusively or through a co-development and/or co-promotion arrangement with a larger company. This arrangement may be part of a global strategic alliance or a more geographically focused partnership. While the primary diagnosing physicians for melanoma are dermatologists and primary care physicians, care of patients with metastatic melanoma is referred to oncologists, surgical oncologists and dermatological oncologists. In the United States, oncology is a highly concentrated specialty, with approximately 650 community cancer programs and oncology private practices and approximately 9,000 oncologists in private practice. We believe this concentration of target physicians can be effectively addressed by a small focused sales force. Companies with comparable products target oncologists with sales forces of approximately 70 to 100 sales representatives. As we obtain additional label indications for STA-4783 in other types of cancer, we may choose to increase our sales force size to promote these new uses. Due to their concentrated and focused nature, specialty target audiences may be reached with more focused and cost-effective marketing campaigns.

We intend to build the commercial infrastructure necessary to bring STA-4783 to market alone or in collaboration with a co-development and/or co-promotion partner. In addition to a specialty sales force, sales management, internal sales support, and an internal marketing group, we will need to establish capabilities to manage key accounts, such as managed care organizations, group purchasing organizations, specialty pharmacies, and government accounts including Veterans Affairs and the Department of Defense. We may also choose to employ medical sales liaisons personnel to support the product.

Competition

The development and commercialization of new drugs is highly competitive. We will face competition with respect to all drug candidates we may develop or commercialize in the future from pharmaceutical and biotechnology companies worldwide. The key competitive factors affecting the success of any approved product will be its efficacy, safety profile, price, method of administration and level of promotional activity. The efficacy and safety profile of our drug candidates relative to competitors will depend upon the results of our clinical trials and experience with the approved product in the commercial marketplace.

STA-4783. If approved, STA-4783 may compete with:

- Drugs that are approved by the FDA for the treatment of metastatic melanoma. Currently, in the United States, there are only two drugs approved for the treatment of metastatic melanoma: dacarbazine/DTIC and the injectable protein interleukin 2, or IL-2. In addition, interferon alfa-2b, also an injectable protein, is the only drug approved for use as an adjuvant to surgery to prevent relapse of melanoma;

- Drugs that are not approved for the treatment of metastatic melanoma, but are used "off-label" to treat the disease, including taxanes, temozolomide, vincristine, carmustine, melphalan, and platinum-chemotherapeutics, such as cisplatin and carboplatin; and

- Compounds in development for metastatic melanoma. Compounds in clinical development may be grouped into five categories: (1) the kinase inhibitors such as Nexavar, being developed by Bayer and Onyx, Sutent, being developed by Pfizer, and ispinesib, being developed by Cytokinetics and GlaxoSmithKline; (2) the anti-CTLA-4 monoclonal antibodies, ipilimumab and ticilimumab; (3) the anti-integrin volociximab; (4) cancer vaccines such as M-Vax and MDX-1379; and (5) derivatives, analogs, or reformulations of known chemotherapies, such as Abraxane, or other chemotherapies.

Apilimod. If approved, apilimod is expected to compete against the currently approved therapies for the treatment of chronic inflammatory diseases, including:

- large-molecule, injectable TNF-antagonists, including: Remicade, marketed by Johnson & Johnson; Enbrel, marketed by Amgen and Wyeth Pharmaceuticals; and Humira, marketed by Abbott Laboratories; and

- broadly immunosuppressive small molecule agents including corticosteroids and azathioprine.

Apilimod may also compete with CNTO-1275 and ABT-874, two injectable antibody-based clinical candidates targeting IL-12 currently in clinical trials that are being developed by Johnson & Johnson and Abbott Laboratories, respectively. We expect that as an oral, small molecule drug, apilimod may prove competitive relative to current and future biologic therapies in manufacturing costs and convenience of administration. We are not aware of any orally administered, selective inhibitors of IL-12 production in clinical trials. Other novel, oral agents in development for inflammatory diseases represent potential competition to apilimod. These include chemokine inhibitors, oral fumarates, and calcineurin inhibitors.

STA-9090. If approved, STA-9090 may compete against the currently approved therapies for the treatment of cancers and other cancer treatments currently under development. In particular, STA-9090 may compete with 17-AAG, being developed by Kosan, and other agents that inhibit Hsp90, including Hsp90 inhibitors from Medimmune/Infinity, BiogenIdec, and Novartis/Vernalis.

STA-9584. If approved, STA-9584 may compete with the currently approved therapies for the treatment of cancers, and other cancer treatments currently under development, including other vascular disrupting agents, such as ABT-751, being developed by Abbott; AS1404, being developed by Antisoma; CA4P, being developed by Oxigene; EXEL-0999, being developed by Exelixis; and ZD6126, being developed by Angiogene.

Many of our potential competitors have substantially greater financial, technical, and personnel resources than us. In addition, many of these competitors have significantly greater commercial infrastructures. Our ability to compete successfully will depend largely on our ability to leverage our experience in drug discovery, development and commercialization to:

- discover and develop medicines that are superior to other products in the market;

- attract high-quality scientific, product development, and commercial personnel;

- obtain patent and/or proprietary protection for our medicines and technologies;

- obtain required regulatory approvals;

- selectively commercialize certain drug candidates in indications treated by specialist physicians; and

- selectively partner with pharmaceutical companies in the development and commercialization of certain drug candidates.

Patents and Proprietary Rights

Our success depends in part on our ability to obtain and maintain proprietary protection for our drug candidates, technology, and know-how, to operate without infringing on the proprietary rights of others, and to prevent others from infringing our proprietary rights. Our policy is to seek to protect our proprietary position by, among other methods, filing U.S. and foreign patent applications related to our proprietary technology, inventions, and improvements that are important to the development of our business. We also rely on trade secrets, know-how, continuing technological innovation, and in-licensing opportunities to develop and maintain our proprietary position.

As of March 15, 2007, our patent portfolio consisted of a total of 520 patents and patent applications worldwide, including specific patent filings with claims to the composition-of-matter and

methods of use of our two clinical stage compounds. We own or have exclusively licensed a total of 23 issued U.S. patents and 106 U.S. patent applications, as well as 391 foreign counterparts to these patents and patent applications. With respect to STA-4783, we have two issued U.S. patents that claim the chemical structure of STA-4783 that expire no earlier than 2022. Both of these issued U.S. patents also claim related chemical structures, pharmaceutical compositions, and methods for treating a subject with cancer. In addition, we have filed several U.S. patent applications that have the potential to extend the patent life of STA-4783, including U.S. patent applications claiming aspects of the treatment regimen for metastatic melanoma which, if issued, would expire no earlier than 2026. We have also filed a U.S. patent application claiming the salt form of STA-4783 which, if issued, would expire no earlier than 2025.

With respect to apilimod, we have three issued U.S. patents that claim the chemical structure of apilimod and two issued U.S. patents that claim pharmaceutical compositions that include apilimod and methods for treating specific disorders using apilimod. These patents expire no earlier than 2021.

We have pending U.S. applications covering compositions-of-matter, methods of treatment and other aspects of our preclinical- and discovery-stage programs, including STA-9090, STA-9584 and our CRAC ion channel program. The patent term of our U.S. patents may potentially be extended under applicable law or regulations, such as the Patent Term Restoration Act. Counterpart filings to these patents and patent applications have been made in a number of other jurisdictions, including Europe and Japan.

We have also in-licensed various technologies to complement our ongoing clinical and research programs. These licenses generally extend for the term of the related patent and contain customary royalty, termination, and other provisions. We have license agreements with Beth Israel Deaconess Medical Center and The Queen's Medical Center, Inc. that provide us with the exclusive commercial right to certain patent filings made by Beth Israel and Queen's Medical in the field of ion channels. We do not believe that these license agreements are currently material to our business. We have exclusive license rights to a patent filing made by Dana-Farber Cancer Institute covering combinations of ingredients that could potentially cover our STA-4783/taxane combination therapy, should such patent claims issue. We would owe nominal royalty payments to Dana-Farber if any of the claims which ultimately issue under a patent or that are pending in an application from this patent filing cover our commercial product. We also have a non-exclusive license to a U.S. patent assigned to Columbia University that could potentially cover a possible aspect of the STA-4783 mechanism. This license is not royalty bearing unless we include specific mechanism language on the label of any approved product, in which case a nominal royalty would be owed.

Government Regulation and Product Approval

Government authorities in the United States, at the federal, state and local level, and other countries extensively regulate, among other things, the research, development, testing, manufacture, labeling, packaging, promotion, storage, advertising, distribution, marketing and export and import of products such as those we are developing. Our drugs must be approved by the FDA through the new drug application, or NDA process before they may be legally marketed in the United States.

United States Government Regulation

NDA Approval Processes

In the United States, the FDA regulates drugs under the Federal Food, Drug and Cosmetic Act, FDCA, and implementing regulations. Failure to comply with the applicable United States

requirements at any time during the product development process, approval process or after approval, may subject an applicant to administrative or judicial sanctions. These sanctions could include:

- the FDA's refusal to approve pending applications;
- license suspension or revocation;
- withdrawal of an approval;
- a clinical hold;
- warning letters;
- product recalls;
- product seizures;
- total or partial suspension of production or distribution; or
- injunctions, fines, civil penalties or criminal prosecution.

Any agency or judicial enforcement action could have a material adverse effect on us. The process of obtaining regulatory approvals and the subsequent substantial compliance with appropriate federal, state, local, and foreign statutes and regulations require the expenditure of substantial time and financial resources.

The process required by the FDA before a drug may be marketed in the United States generally involves the following:

- completion of preclinical laboratory tests according to Good Laboratory Practices;
- submission of an IND, which must become effective before human clinical trials may begin;
- performance of adequate and well-controlled human clinical trials according to Good Clinical Practices to establish the safety and efficacy of the proposed drug for its intended use;
- submission to the FDA of a NDA;
- satisfactory completion of an FDA inspection of the manufacturing facility or facilities at which the product is produced to assess compliance with current good manufacturing practice, or cGMP, to assure that the facilities, methods and controls are adequate to preserve the drug's identity, strength, quality and purity; and
- FDA review and approval of the NDA.

Once a pharmaceutical candidate is identified for development, it enters the preclinical testing stage. Preclinical tests include laboratory evaluations of product chemistry, toxicity and formulation, as well as animal studies. An IND sponsor must submit the results of the preclinical tests, together with manufacturing information and analytical data, to the FDA as part of the IND. Some preclinical or nonclinical testing may continue even after the IND is submitted. In addition to including the results of the preclinical studies, the IND will also include a protocol detailing, among other things, the objectives of the clinical trial, the parameters to be used in monitoring safety and the effectiveness criteria to be evaluated if the first phase lends itself to an efficacy determination. The IND automatically becomes effective 30 days after receipt by the FDA, unless the FDA, within the 30-day time period, specifically places the sponsor on clinical hold. In such a case, the IND sponsor and the FDA must resolve any outstanding concerns before clinical trials can begin.

All clinical trials must be conducted under the supervision of one or more qualified investigators in accordance with good clinical practice regulations. These regulations include the requirement that all research subjects provide informed consent. Further, an institutional review board, or IRB, at each

institution participating in the clinical trial must review and approve the plan for any clinical trial before it commences at that institution. Each new clinical protocol must be submitted to the FDA as part of the IND. Progress reports detailing the results of the clinical trials must be submitted at least annually to the FDA and more frequently if serious adverse events occur.

Human clinical trials are typically conducted in three sequential phases that may overlap or be combined:

- *Phase 1.* The drug is initially introduced into healthy human subjects or patients with the disease and tested for safety, dosage tolerance, pharmacokinetics, pharmacodynamics, absorption, metabolism, distribution and excretion. In the case of some products for severe or life-threatening diseases, especially when the product may be too inherently toxic to ethically administer to healthy volunteers, the initial human testing is often conducted in patients.

- *Phase 2.* Involves studies in a limited patient population to identify possible adverse effects and safety risks, to preliminarily evaluate the efficacy of the product for specific targeted diseases and to determine dosage tolerance and optimal dosage.

- *Phase 3.* Clinical trials are undertaken to further evaluate dosage, clinical efficacy and safety in an expanded patient population at geographically dispersed clinical study sites. These studies are intended to establish the overall risk-benefit ratio of the product and provide, if appropriate, an adequate basis for product labeling.

Phase 1, Phase 2, and Phase 3 testing may not be completed successfully within any specified period, if at all. The FDA or an IRB or the sponsor may suspend a clinical trial at any time on various grounds, including a finding that the research subjects or patients are being exposed to an unacceptable health risk.

During the development of a new drug, sponsors are given an opportunity to meet with the FDA at certain points. These points are prior to submission of an IND, at the end of Phase 2, and before an NDA is submitted. These meetings can provide an opportunity for the sponsor to share information about the data gathered to date, for the FDA to provide advice, and for the sponsor and FDA to reach agreement on the next phase of development. Sponsors typically use the end of Phase 2 meeting to discuss their Phase 2 clinical results and present their plans for the pivotal Phase 3 clinical trial that they believe will support approval of the new drug. If a Phase 2 clinical trial is the subject of discussion at an end of Phase 2 meeting with the FDA, a sponsor may be able to request a special protocol assessment, or SPA, the purpose of which is to reach agreement with the FDA on the design and size of the Phase 3 clinical trial. If such an agreement is reached, it will be documented and made part of the administrative record. This agreement may not be changed by the sponsor or the FDA after the trial begins, except (1) with the written agreement of the sponsor and the FDA or (2) if the FDA determines that a substantial scientific issue essential to determining the safety or effectiveness of the drug was identified after the testing began.

Concurrent with clinical trials, companies usually complete additional animal studies and must also develop additional information about the chemistry and physical characteristics of the drug and finalize a process for manufacturing the product in accordance with cGMP requirements. The manufacturing process must be capable of consistently producing quality batches of the drug candidate and the manufacturer must develop methods for testing the quality, purity and potency of the final drugs. Additionally, appropriate packaging must be selected and tested and stability studies must be conducted to demonstrate that the drug candidate does not undergo unacceptable deterioration over its shelf-life.

The results of product development, preclinical studies and clinical studies, along with descriptions of the manufacturing process, analytical tests conducted on the chemistry of the drug, results of chemical studies and other relevant information are submitted to the FDA as part of an NDA requesting approval to market the product. The submission of an NDA is subject to the payment of

user fees, but a waiver of such fees may be obtained under specified circumstances. The FDA reviews all NDAs submitted before it accepts them for filing. It may request additional information rather than accept a NDA for filing. In this event, the NDA must be resubmitted with the additional information. The resubmitted application also is subject to review before the FDA accepts it for filing.

Once the submission is accepted for filing, the FDA begins an in-depth review. The FDA may refuse to approve an NDA if the applicable regulatory criteria are not satisfied or may require additional clinical or other data. Even if such data is submitted, the FDA may ultimately decide that the NDA does not satisfy the criteria for approval. The FDA reviews an NDA to determine, among other things, whether a product is safe and effective for its intended use and whether its manufacturing is cGMP-compliant to assure and preserve the product's identity, strength, quality and purity. Before approving an NDA, the FDA will inspect the facility or facilities where the product is manufactured and tested.

Satisfaction of FDA requirements or similar requirements of state, local and foreign regulatory authorities typically takes at least several years and the actual time required may vary substantially, based upon, among other things, the type, complexity and novelty of the product or disease. Government regulation may delay or prevent marketing of potential products for a considerable period of time and impose costly procedures upon our activities. Success in early stage clinical trials does not assure success in later stage clinical trials. Data obtained from clinical activities are not always conclusive and may be susceptible to varying interpretations, which could delay, limit or prevent regulatory approval. The FDA may not grant approval on a timely basis, or at all. Even if a product receives regulatory approval, the approval may be significantly limited to specific diseases and dosages or the indications for use may otherwise be limited, which could restrict the commercial application of the product. Further, even after regulatory approval is obtained, later discovery of previously unknown problems with a product may result in restrictions on the product or even complete withdrawal of the product from the market. Delays in obtaining, or failures to obtain, regulatory approvals for any drug candidate could substantially harm our business and cause our stock price to drop significantly. In addition, we cannot predict what adverse governmental regulations may arise from future U.S. or foreign governmental action.

Expedited Review and Approval

The FDA has various programs, including Fast Track, priority review, and accelerated approval, that are intended to expedite or simplify the process for reviewing drugs, and/or provide for approval on the basis of surrogate endpoints. Even if a drug qualifies for one or more of these programs, we cannot be sure that the FDA will not later decide that the drug no longer meets the conditions for qualification or that the time period for FDA review or approval will be shortened. Generally, drugs that may be eligible for these programs are those for serious or life-threatening conditions, those with the potential to address unmet medical needs, and those that offer meaningful benefits over existing treatments. Fast Track designation applies to the combination of the product and the specific indication for which it is being studied. Although Fast Track and priority review do not affect the standards for approval, FDA will attempt to facilitate early and frequent meetings with a sponsor of a Fast Track designated drug and expedite review of the application for a drug designated for priority review. Drugs that receive an accelerated approval may be approved on the basis of adequate and well-controlled clinical trials establishing that the drug product has an effect on a surrogate endpoint that is reasonably likely to predict clinical benefit or on the basis of an effect on a clinical endpoint other than survival or irreversible morbidity. As a condition of approval, the FDA may require that a sponsor of a drug receiving accelerated approval perform post-marketing clinical trials. We have applied for and received Fast Track designation from the FDA for STA-4783 for the treatment of metastatic melanoma. However, there can be no assurance that STA-4783 will be reviewed or approved more expeditiously than would otherwise have been the case.

Orphan Drug Designation

Under the Orphan Drug Act, the FDA may grant orphan drug designation to drugs intended to treat a rare disease or condition, which is generally a disease or condition that affects fewer than 200,000 individuals in the United States, or more than 200,000 individuals in the United States and for which there is no reasonable expectation that the cost of developing and making available in the United States a drug for this type of disease or condition will be recovered from sales in the United States for that drug. Orphan drug designation must be requested before submitting an NDA. After the FDA grants orphan drug designation, the identity of the therapeutic agent and its potential orphan use are disclosed publicly by the FDA. Orphan drug designation does not convey any advantage in or shorten the duration of the regulatory review and approval process.

If a product that has orphan drug designation subsequently receives the first FDA approval for the disease for which it has such designation, the product is entitled to orphan product exclusivity, which means that the FDA may not approve any other applications to market the same drug for the same indication, except in very limited circumstances, for seven years. Orphan drug exclusivity, however, also could block the approval of one of our products for seven years if a competitor obtains approval of the same drug as defined by the FDA or if our drug candidate is determined to be contained within the competitor's product for the same indication or disease.

We intend to file for orphan drug designation for STA-4783 for the treatment of stage IV metastatic melanoma and potentially for other indications for STA-4783 and for other drug candidates that meet the criteria for orphan designation. We may not be awarded orphan exclusivity for STA-4783 or any of our other drug candidates or indications. In addition, obtaining FDA approval to market a product with orphan drug exclusivity may not provide us with a material commercial advantage.

Pediatric Exclusivity

The FDA Modernization Act of 1997 included a pediatric exclusivity provision that was extended by the Best Pharmaceuticals for Children Act of 2002. Pediatric exclusivity is designed to provide an incentive to manufacturers for conducting research about the safety of their products in children. Pediatric exclusivity, if granted, provides an additional six months of market exclusivity in the United States for new or currently marketed drugs. Under Section 505A of the FDCA, six months of market exclusivity may be granted in exchange for the voluntary completion of pediatric studies in accordance with an FDA-issued "Written Request." The FDA may not issue a Written Request for studies on unapproved or approved indications or where it determines that information relating to the use of a drug in a pediatric population, or part of the pediatric population, may not produce health benefits in that population.

We have not requested or received a Written Request for such pediatric studies, although we may ask the FDA to issue a Written Request for such studies in the future. To receive the six-month pediatric market exclusivity, we would have to receive a Written Request from the FDA, conduct the requested studies in accordance with a written agreement with the FDA or, if there is no written agreement, in accordance with commonly accepted scientific principles, and submit reports of the studies. The FDA would then have to accept the reports. The FDA may not issue a Written Request for such studies or accept the reports of the studies. The current pediatric exclusivity provision is scheduled to end on October 1, 2007, and it may not be reauthorized.

Post-approval Requirements

Once an approval is granted, the FDA may withdraw the approval if compliance with regulatory standards is not maintained or if problems occur after the product reaches the market. After approval, some types of changes to the approved product, such as adding new indications, manufacturing changes and additional labeling claims, are subject to further FDA review and approval. In addition, the FDA may require testing and surveillance programs to monitor the effect of approved products that have been commercialized, and the FDA has the power to prevent or limit further marketing of a product based on the results of these post-marketing programs.

Any drug products manufactured or distributed by us pursuant to FDA approvals are subject to continuing regulation by the FDA, including, among other things:

- record-keeping requirements;
- reporting of adverse experiences with the drug;
- providing the FDA with updated safety and efficacy information;
- drug sampling and distribution requirements;
- notifying the FDA and gaining its approval of specified manufacturing or labeling changes;
- complying with certain electronic records and signature requirements; and
- complying with FDA promotion and advertising requirements.

Drug manufacturers and their subcontractors are required to register their establishments with the FDA and some state agencies, and are subject to periodic unannounced inspections by the FDA and some state agencies for compliance with cGMP and other laws.

We rely, and expect to continue to rely, on third parties for the production of clinical and commercial quantities of our products. Future FDA and state inspections may identify compliance issues at the facilities of our contract manufacturers that may disrupt production or distribution, or require substantial resources to correct.

From time to time, legislation is drafted, introduced and passed in Congress that could significantly change the statutory provisions governing the approval, manufacturing and marketing of products regulated by the FDA. In addition, FDA regulations and guidance are often revised or reinterpreted by the agency in ways that may significantly affect our business and our products. It is impossible to predict whether legislative changes will be enacted, or FDA regulations, guidance or interpretations changed or what the impact of such changes, if any, may be.

Foreign Regulation

In addition to regulations in the United States, we will be subject to a variety of foreign regulations governing clinical trials and commercial sales and distribution of our products. Whether or not we obtain FDA approval for a product, we must obtain approval by the comparable regulatory authorities of foreign countries before we can commence clinical trials or marketing of the product in those countries. The approval process varies from country to country and the time may be longer or shorter than that required for FDA approval. The requirements governing the conduct of clinical trials, product licensing, pricing and reimbursement vary greatly from country to country.

Under European Union regulatory systems, we may submit marketing authorization applications either under a centralized or decentralized procedure. The centralized procedure, which is compulsory for medicines produced by biotechnology or those medicines intended to treat AIDS, cancer, neurodegenerative disorders, or diabetes and optional for those medicines which are highly innovative, provides for the grant of a single marketing authorization that is valid for all European Union member

states. The decentralized procedure provides for mutual recognition of national approval decisions. Under this procedure, the holder of a national marketing authorization may submit an application to the remaining member states. Within 90 days of receiving the applications and assessments report each member state must decide whether to recognize approval. If a member state does not recognize the marketing authorization, the disputed points are eventually referred to the European Commission, whose decision is binding on all member states.

As in the United States, we may apply for designation of a product as an orphan drug for the treatment of a specific indication in the European Union before the application for marketing authorization is made. Orphan drugs in Europe enjoy economic and marketing benefits, including up to 10-years of market exclusivity for the approved indication unless another applicant can show that its product is safer, more effective or otherwise clinically superior to the orphan-designated product.

Reimbursement

Sales of pharmaceutical products depend in significant part on the availability of third-party reimbursement. We anticipate third-party payors will provide reimbursement for our products. It is time consuming and expensive for us to seek reimbursement from third-party payors. Reimbursement may not be available or sufficient to allow us to sell our products on a competitive and profitable basis.

The passage of the Medicare Prescription Drug and Modernization Act of 2003, or the MMA, imposes new requirements for the distribution and pricing of prescription drugs for Medicare beneficiaries, which may affect the marketing of our products. The MMA also introduced new reimbursement methodologies, which have been phased in since January 1, 2005. For example, new reimbursement methodologies under the MMA with respect to drugs administered by physicians, such as STA-4783 if approved, were phased in during 2005 and 2006. Under these reimbursement methods, physicians and hospitals are reimbursed at a rate equal to 106% of the average sales price, or ASP, of the particular physician-administered drug. The Centers for Medicare & Medicaid Services, or CMS, monitors the calculation of a product's ASP and publishes a product's ASP quarterly in advance of the quarter in which it is applicable. Physicians administering drugs in an office setting have a choice between obtaining and billing for these kinds of drugs under the ASP plus 6% methodology or to obtain drugs from vendors selected by the CMS under the competitive acquisition program, or CAP. Physicians who select to obtain drugs under CAP do not purchase or obtain reimbursement directly for such drugs. It is not clear what effect the MMA will have on the prices paid for currently approved drugs and the pricing options for new drugs approved after January 1, 2006. Moreover, while the MMA applies only to drug benefits for Medicare beneficiaries, private payors often follow Medicare coverage policy and payment limitations in setting their own payment rates. Any reduction in payment that results from the MMA may result in a similar reduction in payments from non-governmental payors.

In addition, in some foreign countries, the proposed pricing for a drug must be approved before it may be lawfully marketed. The requirements governing drug pricing vary widely from country to country. For example, the European Union provides options for its member states to restrict the range of medicinal products for which their national health insurance systems provide reimbursement and to control the prices of medicinal products for human use. A member state may approve a specific price for the medicinal product or it may instead adopt a system of direct or indirect controls on the profitability of the company placing the medicinal product on the market.

We expect that there will continue to be a number of federal and state proposals to implement governmental pricing controls. While we cannot predict whether such legislative or regulatory proposals will be adopted, the adoption of such proposals could have a material adverse effect on our business, financial condition and profitability.

Employees

We believe that our success will depend greatly on our ability to identify, attract, and retain capable employees. As of March 15, 2007, we had 146 full time employees, including a total of 59 employees who hold M.D. or Ph.D. degrees. One hundred and fourteen of our employees are primarily engaged in research and development activities, and 32 are primarily engaged in general and administrative activities. Our employees are not represented by any collective bargaining unit, and we believe our relations with our employees are good.

Company History and Available Information

We commenced operations in July 2001. In September 2002, we acquired Principia Associates, Inc., which had previously acquired Shionogi BioResearch Corp., a U.S.-based drug discovery subsidiary of the Japanese pharmaceutical company, Shionogi & Co., Ltd. In this acquisition, we acquired a unique chemical compound library, an integrated set of drug discovery capabilities, and a pipeline of preclinical and research programs. Since 2002, we have been advancing these programs into later stages of development; discovering and developing additional drug candidates; and expanding our management and scientific teams and capabilities to support more advanced stages of drug development and commercialization.

Our principal executive offices are located at 45 Hartwell Avenue, Lexington, Massachusetts 02421, and our telephone number is (781) 274-8200. Our website address is www.syntapharma.com. The information contained on our website is not incorporated by reference into, and does not form any part of, this Annual Report on Form 10-K. We have included our website address as a factual reference and do not intend it to be an active link to our website. Our trademarks include Synta Pharmaceuticals and our logo. Other service marks, trademarks and trade names appearing in this Annual Report on Form 10-K are the property of their respective owners. Our Annual Reports on Form 10-K, Quarterly Reports on Form 10-Q, Current Reports on Form 8-K, and all amendments to those reports, are available free of charge through the Investors section of our website as soon as reasonably practicable after such materials have been electronically filed with, or furnished to, the Securities and Exchange Commission.

Item 1A. RISK FACTORS

If any of the following risks occurs, our business, business prospects, financial condition, results of operations, or cash flows could be materially harmed.

Risks Related to Our Financial Position and Need for Additional Capital

We have incurred significant losses since our inception, and we expect to incur losses for the foreseeable future and may never reach profitability.

Since inception we have incurred significant operating losses and, as of December 31, 2006, we had an accumulated deficit of $236.6 million, which includes research and development expense of $180.4 million. We expect to continue to incur significant operating expenses and capital expenditures and anticipate that our expenses and losses will increase substantially in the foreseeable future as we:

- initiate a pivotal Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma and announce plans for initiating clinical trials in one or more additional cancer types in mid-2007;
- begin to establish sales and marketing functions and commercial manufacturing arrangements for STA-4783;

- complete the current Phase 2a clinical trials of apilimod for the treatment of rheumatoid arthritis and CVID, and possibly initiate Phase 2 clinical trials of apilimod in additional inflammatory disease indications;
- initiate additional Phase 3 clinical trials of STA-4783 and one or more Phase 3 clinical trials of apilimod, if supported by Phase 2 results;
- complete preclinical development of STA-9090 and initiate clinical trials, if supported by positive preclinical data;
- complete preclinical development of STA-9584 and initiate clinical trials, if supported by positive preclinical data;
- advance our preclinical CRAC ion channel inhibitor program into clinical trials, if supported by positive preclinical data;
- discover, develop, and seek regulatory approval for backups of our current drug candidates and other new drug candidates;
- identify additional compounds or drug candidates and acquire rights from third parties to those compounds or drug candidates through licenses, acquisitions or other means;
- commercialize any approved drug candidates;
- hire additional clinical, scientific, and management personnel; and
- add operational, financial, and management information systems and personnel.

We must generate significant revenue to achieve and maintain profitability. Even if we succeed in developing and commercializing one or more of our drug candidates, we may not be able to generate sufficient revenue and we may never be able to achieve or maintain profitability.

Our operating history may make it difficult to evaluate the success of our business to date and to assess our future viability.

We commenced operations in July 2001 and are a development-stage company. Our operations to date have been limited to organizing and staffing our company, acquiring, developing, and securing our technology, and undertaking preclinical studies and clinical trials of our drug candidates. We have not yet demonstrated an ability to obtain regulatory approval, formulate and manufacture a commercial-scale product, or conduct sales and marketing activities necessary for successful product commercialization. Consequently, any predictions about our future success or viability may not be as accurate as they could be if we had a longer operating history or had previously discovered, developed, and/or commercialized an approved product.

If we fail to obtain the capital necessary to fund our operations, we will be unable to successfully develop and commercialize our lead drug candidates.

Although we have raised substantial capital to date, we will require substantial future capital in order to complete clinical development and commercialize our lead drug candidates, STA-4783, apilimod, STA-9090, and STA-9584, and to conduct the research and development and clinical and regulatory activities necessary to bring other drug candidates to market. In particular, based upon our current estimates, we expect the cost to complete our pivotal Phase 3 clinical trial of STA-4783 in metastatic melanoma will be in the range of $40 to $60 million. We may not have sufficient capital to complete this trial and fund the continued clinical development of our other lead drug candidates and other programs. Our future capital requirements will depend on many factors that are currently unknown to us, including:

- the timing of initiation, progress and results of our planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma;

- the costs of establishing sales and marketing functions and of establishing commercial manufacturing arrangements for STA-4783;

- the progress and results of any additional Phase 2 clinical trials of STA-4783 for other cancer indications that we may initiate;

- the progress and results of the current Phase 2a clinical trials of apilimod for the treatment of rheumatoid arthritis and CVID and any future Phase 2 clinical trials we may initiate for other inflammatory disease indications;

- the need for, and the progress and results of, any additional Phase 3 clinical trials of STA-4783 and any Phase 3 clinical trial of apilimod we may initiate in the future based on the results of Phase 2 clinical trials;

- the results of our preclinical studies and testing of STA-9090, STA-9584 and our CRAC ion channel inhibitor program, and our decision to initiate clinical trials, if supported by the preclinical results;

- the costs, timing, and outcome of regulatory review of STA-4783, apilimod and our preclinical drug candidates;

- the scope, progress, results, and cost of preclinical development, clinical trials, and regulatory review of any new drug candidates we may discover or acquire;

- the costs of preparing, filing, and prosecuting patent applications and maintaining, enforcing, and defending intellectual property-related claims;

- our ability to establish strategic collaborations and licensing or other arrangements on terms favorable to us;

- the costs to satisfy our obligations under potential future collaborations; and

- the timing, receipt, and amount of sales or royalties, if any, from STA-4783, apilimod, STA-9090, STA-9584, and our other potential products.

There can be no assurance that additional funds will be available when we need them on terms that are acceptable to us, or at all. If adequate funds are not available on a timely basis, we may be required to:

- terminate or delay clinical trials or other development for one or more of our drug candidates;

- delay our establishment of sales and marketing capabilities, our contracting for commercial manufacturing capacity, or other activities that may be necessary to commercialize our drug candidates; or

- curtail significant drug development programs that are designed to identify new drug candidates.

We believe that our existing cash and investment securities will be sufficient to support our current operating plan through at least mid-2008. However, our operating plan may change as a result of many factors currently unknown to us, and we may need additional funds sooner than planned. In addition, we may seek additional capital due to favorable market conditions or strategic considerations even if we believe we have sufficient funds for our current or future operating plans.

Raising additional capital may cause dilution to existing stockholders, restrict our operations or require us to relinquish rights.

We may seek the additional capital necessary to fund our operations through public or private equity offerings, debt financings, and collaborative and licensing arrangements. To the extent that we raise additional capital through the sale of equity or convertible debt securities, existing stockholders ownership interests will be diluted and the terms may include liquidation or other preferences that

adversely affect their rights as a stockholder. Debt financing, if available, may involve agreements that include covenants limiting or restricting our ability to take specific actions such as incurring additional debt, making capital expenditures, or declaring dividends. If we raise additional funds through collaboration and licensing arrangements with third parties, we may have to relinquish valuable rights to our technologies or drug candidates, or grant licenses on terms that are not favorable to us.

Risks Related to the Development and Regulatory Approval of Our Drug Candidates

Our success is largely dependent on the success of our lead drug candidate, STA-4783, as well as our other drug candidates, and we cannot be certain that we will be able to obtain regulatory approval for or successfully commercialize any of these drug candidates.

We have invested a significant portion of our time and financial resources in the development of our lead drug candidate, STA-4783 for the treatment of cancer. We have also invested a significant amount of time and financial resources in the development of our other drug candidates, apilimod, STA-9090 and STA-9584. We anticipate that our success will depend largely on the receipt of regulatory approval and successful commercialization of these drug candidates. The future success of these drug candidates will depend on several factors, including the following:

- our ability to provide acceptable evidence of their safety and efficacy;
- receipt of marketing approval from the FDA and any similar foreign regulatory authorities;
- successful formulation of an efficacious and commercially viable form of apilimod;
- obtaining and maintaining commercial manufacturing arrangements with third-party manufacturers or establishing commercial-scale manufacturing capabilities;
- establishing an internal sales force or collaborating with pharmaceutical companies or contract sales organizations to market and sell any approved drug; and
- acceptance of any approved drug in the medical community and by patients and third-party payors.

Many of these factors are beyond our control. Accordingly, there can be no assurance that we will ever be able to generate revenues through the sale of STA-4783, apilimod, STA-9090, or STA-9584.

If we do not obtain required regulatory approval, we will be unable to market and sell our drug candidates.

STA-4783, apilimod, STA-9090, STA-9584, and any other drug candidates we may discover or acquire and seek to commercialize are subject to extensive governmental regulations relating to development, clinical trials, manufacturing, and commercialization. Rigorous preclinical testing and clinical trials and an extensive regulatory approval process are required to be successfully completed in the United States and in many foreign jurisdictions before a new drug can be sold. Satisfaction of these and other regulatory requirements is costly, time consuming, uncertain, and subject to unanticipated delays. The time required to obtain approval by the FDA is unpredictable but typically exceeds five years following the commencement of clinical trials, depending upon the complexity of the drug candidate. We initiated clinical development of STA-4783 and apilimod in 2002 and 2003, respectively, and thus far, these drug candidates have been studied in only a relatively small number of patients. We have recently completed a Phase 2b clinical trial of STA-4783 for the treatment of metastatic melanoma and intend to initiate a pivotal Phase 3 clinical trial for this indication in the middle of 2007. Apilimod is currently in Phase 2a clinical trials for the treatment of rheumatoid arthritis and CVID. STA-9090 and STA-9584 are still in preclinical development.

We have limited experience in conducting and managing the clinical trials necessary to obtain regulatory approvals, including approval by the FDA. In connection with the clinical trials of STA-4783, apilimod, STA-9090, and STA-9584 and any other drug candidate we may seek to develop in the future, we face risks that:

- the drug candidate may not prove to be efficacious;
- the dosing of the drug candidate in a particular clinical trial may not be at an optimal level (for example, we are currently evaluating whether the Phase 2 clinical trial results for STA-4783 in sarcoma and non-small cell lung cancer and Phase 2 clinical trial results for apilimod in psoriasis and Crohn's disease were the result of suboptimal dosing amounts and/or dosing schedules);
- patients may die or suffer other adverse effects for reasons that may or may not be related to the drug candidate being tested;
- the results may not confirm the positive results of earlier clinical trials; and
- the results may not meet the level of statistical significance required by the FDA or other regulatory agencies for marketing approval.

Of the large number of drugs in development, only a small percentage result in the submission of a new drug application, or NDA, to the FDA and even fewer are approved for commercialization. Furthermore, even if we do receive regulatory approval to market a commercial product, any such approval may be subject to limitations on the indicated uses for which we may market the product.

We will need to demonstrate the safety and efficacy of STA-4783 in one or more Phase 3 clinical trials in order to obtain FDA approval of STA-4783 for use in the treatment of metastatic melanoma, and there can be no assurance that STA-4783 will achieve positive results in further clinical testing.

Positive results in early clinical trials of a drug candidate may not be replicated in later clinical trials. A number of companies in the pharmaceutical and biotechnology industries have suffered significant setbacks in late-stage clinical trials even after achieving promising results in earlier-stage development. Although our Phase 2b clinical trial of STA-4783 for the treatment of metastatic melanoma achieved the primary endpoint of increasing progression-free survival, there can be no assurance that the planned Phase 3 clinical trial for the treatment of metastatic melanoma we intend to initiate in 2007 will achieve positive results. A number of factors could contribute to a lack of positive results in our planned Phase 3 clinical trial. For example, in our Phase 2b clinical trial, the majority of patients had been treated with prior chemotherapy, whereas we expect our Phase 3 clinical trial to enroll only patients who have received no prior treatment with chemotherapy. We have based the trial design on positive trends seen in a subset analysis of our Phase 2b clinical trial results, as well as our understanding of the mechanism of action of STA-4783. However, because of the small number of patients in each subset, the results of this analysis were not statistically significant, and there can be no assurance that we will achieve positive results in a larger group of patients who have received no prior chemotherapy in the Phase 3 clinical trial. In addition, the clinical investigators involved in the Phase 2b clinical trial used their judgment to determine when a patient's melanoma had progressed, using the criteria defined in the trial protocol and, among other factors, either CT or magnetic resonance imaging scans of a patient's tumors. In our Phase 2b clinical trial, each clinical trial site determined when patients enrolled at the site experienced a progression of their melanoma. In some past clinical trials by other companies involving similar subjective judgments, it has been reported that the variation among clinical trial sites in determining progression contributed to positive results. In our Phase 3 clinical trial, we plan to use a single centralized radiological reading center to review all patient scans, which could cause the results of our Phase 3 clinical trial to differ from those observed in our Phase 2b clinical trial.

Furthermore, although we did not identify any confounding factors in the Phase 2b clinical trial of STA-4783 for the treatment of metastatic melanoma, we did not evaluate every factor that may have potentially influenced the trial results and can give no assurance that there were no such confounding factors. In our planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma, we may stratify, or evenly allocate to each trial arm, patients having certain strong prognostic factors, such as elevated lactate dehydrogenase, or LDH, levels and liver metastases. However, we may not be able to stratify all such prognostic factors evenly or we may not require the stratification of one or more prognostic factors if the clinical trial timelines would be adversely impacted. Although we found that patients with elevated LDH and liver metastases were evenly distributed between the STA-4783 plus paclitaxel arm and the paclitaxel control arm in our Phase 2b clinical trial, we noted an imbalance in the M-class distribution of patients. M-class is a measure of disease progression that is generally viewed as a prognostic factor. In our Phase 2b clinical trial, 53% of the patients in the STA-4783 plus paclitaxel group were classified by the clinical investigator as M1c, the most advanced stage of metastatic melanoma, compared to 75% in the paclitaxel alone group. We performed a statistical analysis which showed that, firstly, investigator-reported M-class was not a prognostic factor in this study, and secondly, the imbalance in M-class distribution between the two arms did not contribute to the positive outcome of this clinical trial. However, we cannot provide complete assurance that the imbalance in M1c classification did not have an impact on the Phase 2b trial results or that if evenly balanced in a future trial, that the clinical trial outcome would not be altered.

If we do not receive positive results in a Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma, we may not be able to obtain regulatory approval or commercialize STA-4783 for this indication and our development of STA-4783 for other indications may be delayed or cancelled.

If the FDA requires an efficacy endpoint other than progression-free survival, or requires more than one pivotal Phase 3 clinical trial, for registration, we may be required to conduct more, larger or longer Phase 3 clinical trials than currently planned.

The primary endpoint of our recently-completed Phase 2b clinical trial of STA-4783 for treating metastatic melanoma was progression-free survival, and we currently intend to use progression-free survival as the primary endpoint of our planned pivotal Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma. Progression-free survival, which measures for each patient the time from assignment to a treatment group until the earlier of tumor progression or death, is an endpoint that the FDA has previously indicated is acceptable for registration in melanoma and other cancer types in clinical trials by other companies. However, no therapy for the treatment of melanoma has been approved based on a progression-free survival endpoint. In our initial meeting with the FDA in January 2007 on the design of our Phase 3 clinical trial for STA-4783, the FDA raised no objection to our use of progression-free survival as the primary endpoint in this trial and overall survival as the secondary endpoint, although the FDA noted that the magnitude of an increase in progression-free survival would need to be clinically meaningful in order to support approval of STA-4783 based on the progression-free survival endpoint. We can give no assurances, however, that the FDA or any other regulatory body will not require a different primary endpoint, such as overall survival, or additional efficacy endpoints for registration. If the FDA requires a different or any additional efficacy endpoints, we may be required to conduct larger or longer Phase 3 clinical trials than currently planned to achieve a statistically significant result to enable approval of STA-4783 for the treatment of metastatic melanoma.

In addition, in order to detect a statistically significant result in our Phase 3 trial for the primary endpoint of progression-free survival, we believe that we will need to enroll and evaluate between 250 and 300 patients. However, based on our initial discussions with the FDA and our medical advisors, we intend to use overall survival as a secondary endpoint, and estimate that we will need to enroll between 600 and 900 patients to detect a statistically significant benefit in this endpoint. Although we expect to

conduct a single, planned analysis for the progression-free survival primary endpoint on the smaller number of patients needed to detect the statistically significant result, the timing of the analysis will be guided by additional discussions we have with the FDA and our medical and regulatory advisors. Although we do not currently expect a delay in the availability of the progression-free survival results, there can be no assurance that further discussions with the FDA will not result in a delay in the release of this data. In addition, even if the Phase 3 trial shows statistically and clinically meaningful benefits in the progression-free survival primary endpoint, the FDA may decide to wait for data on the overall survival secondary endpoint prior to considering STA-4783 for approval. If the FDA were to approve STA-4783 based on the data from the progression-free survival endpoint and the results of the overall survival secondary endpoint are not positive, the FDA may limit the use of STA-4783 or even withdraw it from the market.

Furthermore, prior to approving a new drug, the FDA typically requires that the efficacy of the drug be demonstrated in two double-blind, controlled studies. In light of the unmet medical need in metastatic melanoma and the results of our Phase 2b clinical trial, we believe we will be required to conduct only a single Phase 3 clinical trial of STA-4783. However, the FDA has indicated that the trial must provide compelling evidence of clinically meaningful benefit in order to warrant consideration for marketing approval, and the FDA has noted that a trial that is merely statistically positive may not provide sufficient evidence to support an NDA filing or approval of a drug candidate. If the FDA determines that our Phase 3 results do not have a clinically meaningful benefit, or if the FDA requires us to conduct additional Phase 3 clinical trials of STA-4783 prior to seeking approval, we will incur significant additional development costs and commercialization of STA-4783 may be prevented or delayed.

We have not determined if we will submit a request for a special protocol assessment for our planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma.

We are currently evaluating whether to submit a request for a Special Protocol Assessment, or SPA, with the FDA regarding our planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma. The SPA process provides for official FDA evaluation of a Phase 3 clinical trial and provides a product sponsor with a binding agreement, unless circumstances change, confirming that the design and size of the Phase 3 trial will be appropriate to form the primary basis of an effectiveness claim for an NDA if the study is performed according to the SPA. However, an SPA must be approved by the FDA before the trial can be initiated, and there is no guarantee that an SPA would be granted on a timely basis. Accordingly, if we submit a request for an SPA, the initiation of this trial may be delayed. If we believe that the submission of a request for an SPA will significantly delay the initiation of this trial, we may determine not to submit a request for an SPA. However, without the FDA's concurrence on an SPA, we cannot be certain that the design, conduct and data analysis approach for this clinical trial will be sufficient to allow us to submit or receive approval of an NDA for STA-4783.

If the current formulation and method of administering STA-4783 is not commercially feasible, we may not be able to commercialize STA-4783 without reformulation and conducting additional clinical trials.

To date, all of our clinical trials have been conducted using the free acid form of STA-4783, which we intend to continue to use in our clinical trials planned for 2007, as well as in our commercial product. Because this free acid form of STA-4783 is not water soluble, prior to administration, it must be dissolved in an organic solvent. In the recently completed Phase 2b clinical trial in metastatic melanoma, this was achieved by combining the STA-4783 with a volume of organic solvent included in the paclitaxel solution and agitating the resulting mixture with a sonication machine for up to 45 minutes. Once the STA-4783 was fully dissolved, the resulting solution was added to the remaining paclitaxel solution, and the combined STA-4783/paclitaxel solution was administered to the patient. We have improved the process for preparing the active pharmaceutical ingredient, or API, and drug

product of STA-4783, such that STA-4783 can now be dissolved in the paclitaxel solution without sonication. We believe these improved procedures replicate the results of the prior methods and are suitable for preparing drug product for clinical trials and commercialization. We anticipate that these improved procedures will be used in the planned Phase 3 clinical trial for STA-4783 in metastatic melanoma and any Phase 2 clinical trials that we may initiate in additional cancer indications in 2007. Although we believe that the changes in the procedures for preparing and dissolving STA-4783 prior to administration will not affect the efficacy or pharmaceutical properties of the treatment, there can be no assurance that the results of future trials will not be affected by these changes in process. In addition, in order to use the free acid form of STA-4783 with other oncology products, including taxanes other than paclitaxel, it must be dissolved in an organic solvent, which may cause additional toxicity due to the presence of the organic solvent.

We have developed a water soluble salt form of STA-4783 that does not need to be dissolved in an organic solvent and therefore may be used more easily with other oncology products. We intend to explore the use of this new salt form of STA-4783 in future clinical trials in order to expand its potential use in combination with other chemotherapies, but it is also our intention to use the free acid form of STA-4783 in our clinical trials planned for 2007 as well as in our commercial product. If the free acid form does not prove to be commercially feasible and we are required to commercialize the salt form of STA-4783, it will require additional formulation development efforts and clinical studies which would delay the commercialization of this drug candidate.

While we believe STA-4783 may have applicability to a broad range of solid tumor cancers, including tumor types other than melanoma, our clinical trials of STA-4783 in non-small cell lung cancer and soft tissue sarcoma have shown negative or inconclusive results.

Based on our understanding of the mechanism of action and the preclinical activity we have seen with STA-4783, which included showing activity in a broad range of cancer types, we intend to conduct clinical trials of STA-4783 in a number of other cancer indications in addition to melanoma. In addition to our Phase 2b clinical trial in metastatic melanoma, we have also conducted Phase 2 clinical trials of STA-4783 in sarcoma and non-small cell lung cancer. The results of the soft tissue sarcoma clinical trial did not definitively establish evidence of clinical activity. In the non-small cell lung cancer clinical trial, no improvement was observed in time-to-progression between combination treatment with STA-4783 and a standard first-line combination therapy. Although we are currently analyzing these data further to assess future development of STA-4783 in sarcoma and non-small cell lung cancer, including assessing the possibility for a potential future clinical trial in non-small cell lung cancer at a more frequent dosing schedule and higher dose than previously tested, there can be no assurances that we will continue the development of STA-4783 in these indications or that STA-4783 will prove effective in and be approved for treating these or other forms of cancer.

Because our drug candidates are in an early stage of development, there is a high risk of failure, and we may never succeed in developing marketable products or generating product revenue.

We have no drug candidates that have received regulatory approval for commercial sale. We do not expect to have any commercial products on the market until at least 2009, if at all. We are exploring human diseases at the cellular level and attempting to develop drug candidates that intervene with cellular processes. Drug development is an uncertain process that involves trial and error, and we may fail at numerous stages along the way. Success in preclinical studies of a drug candidate may not be predictive of similar results in humans during clinical trials, and successful results from early or small clinical trials of a drug candidate may not be replicated in later and larger clinical trials. For example, although preclinical data and Phase 2a clinical trial results suggested that apilimod had activity in psoriasis and Crohn's disease, our Phase 2b clinical trials of apilimod in those indications did not demonstrate clinical benefit. Accordingly, the results from preclinical studies and the completed

and ongoing clinical trials for our drug candidates may not be predictive of the results we may obtain in later stage clinical trials.

If clinical trials for our drug candidates, including STA-4783 and apilimod, are prolonged or delayed, we may be unable to commercialize our drug candidates on a timely basis, which would require us to incur additional costs and delay our receipt of any revenue from potential product sales.

We cannot predict whether we will encounter problems with any of our completed, ongoing or planned clinical trials that will cause us or any regulatory authority to delay or suspend those clinical trials or delay the analysis of data derived from them. A number of events, including any of the following, could delay the completion of our ongoing and planned clinical trials and negatively impact our ability to obtain regulatory approval for, and to market and sell, a particular drug candidate, including our clinical drug candidates STA-4783 and apilimod:

- conditions imposed on us by the FDA or any foreign regulatory authority regarding the scope or design of our clinical trials, particularly with respect to the planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma;
- delays in obtaining, or our inability to obtain, required approvals from institutional review boards or other reviewing entities at clinical sites selected for participation in our clinical trials;
- insufficient supply or deficient quality of our drug candidates or other materials necessary to conduct our clinical trials;
- delays in obtaining regulatory agency agreement for the conduct of our clinical trials, including setting the primary endpoints or establishing the appropriate comparator treatment for our planned Phase 3 clinical trial of STA-4783 in metastatic melanoma;
- lower than anticipated enrollment and retention rate of subjects in clinical trials;
- negative or inconclusive results from clinical trials, or results that are inconsistent with earlier results, that necessitate additional clinical studies (for example, due to patient-to-patient pharmacokinetic variability);
- serious and unexpected drug-related side effects experienced by patients in clinical trials; or
- failure of our third-party contractors to comply with regulatory requirements or otherwise meet their contractual obligations to us in a timely manner.

Commercialization of our drug candidates may be delayed by the imposition of additional conditions on our clinical trials by the FDA or the requirement of additional supportive studies by the FDA. In addition, clinical trials require sufficient patient enrollment, which is a function of many factors, including the size of the patient population, the nature of the trial protocol, the proximity of patients to clinical sites, the availability of effective treatments for the relevant disease, the conduct of other clinical trials that compete for the same patients as our clinical trials, and the eligibility criteria for our clinical trials. For example, competing trials for melanoma treatments or the emergence of new approved therapies may make it more difficult to enroll patients in our Phase 3 clinical trial of STA-4783 for metastatic melanoma on the schedule currently planned. We are aware of other ongoing clinical trials of drug candidates for the treatment of metastatic melanoma, including Nexavar, Sutent, ipilimumab, and ticilimumab. Enrollment efforts and future results with respect to these trials could also adversely impact patient enrollment in our Phase 3 clinical trial. We have had satisfactory patient enrollment in our clinical trials to date. However, in our planned Phase 3 clinical trial, we expect to enroll approximately 600 patients with stage IV metastatic melanoma, which is significantly more patients than we enrolled in our Phase 2b clinical trial for STA-4783. Future delays in patient enrollment can result in increased costs and longer development times. Our failure to enroll patients in our clinical trials could delay the completion of the clinical trial beyond our current expectations. In

addition, the FDA could require us to conduct clinical trials with a larger number of subjects than we have projected for any of our drug candidates. We may not be able to enroll a sufficient number of patients in a timely or cost-effective manner. Furthermore, enrolled patients may drop out of our clinical trials, which could impair the validity or statistical significance of the clinical trials.

We do not know whether our clinical trials will begin as planned, will need to be restructured, or will be completed on schedule, if at all. Delays in our clinical trials will result in increased development costs for our drug candidates. In addition, if our clinical trials are delayed, our competitors may be able to bring products to market before we do and the commercial viability of our drug candidates, including our drug candidates STA-4783 and apilimod, could be limited.

Failure to comply with foreign regulatory requirements governing human clinical trials and marketing approval for drugs could prevent us from selling our drug candidates in foreign markets, which may adversely affect our operating results and financial condition.

The requirements governing the conduct of clinical trials, product licensing, pricing, and reimbursement for marketing our drug candidates outside the United States vary greatly from country to country and may require additional testing. We have no experience in obtaining foreign regulatory approvals. We expect that our future clinical development of STA-4783 and apilimod will involve a number of clinical trials in foreign jurisdictions, particularly in Europe. The time required to obtain approvals outside the United States may differ from that required to obtain FDA approval. We may not obtain foreign regulatory approvals on a timely basis, if at all. Approval by the FDA does not ensure approval by regulatory authorities in other countries, and approval by one foreign regulatory authority does not ensure approval by regulatory authorities in other countries or by the FDA. Failure to comply with these regulatory requirements or obtain required approvals could impair our ability to develop foreign markets for our drug candidates and may have a material adverse effect on our results of operations and financial condition.

Our drug candidates will remain subject to ongoing regulatory review even if they receive marketing approval, and if we fail to comply with continuing regulations, we could lose these approvals and the sale of any approved commercial products could be suspended.

Even if we receive regulatory approval to market a particular drug candidate, the manufacturing, labeling, packaging, adverse event reporting, storage, advertising, promotion, and record keeping related to the product will remain subject to extensive regulatory requirements. If we fail to comply with the regulatory requirements of the FDA and other applicable U.S. and foreign regulatory authorities or previously unknown problems with any approved commercial products, manufacturers, or manufacturing processes are discovered, we could be subject to administrative or judicially imposed sanctions, including:

- restrictions on the products, manufacturers, or manufacturing processes;
- untitled or warning letters;
- civil or criminal penalties;
- fines;
- injunctions;
- product seizures or detentions;
- import bans;
- voluntary or mandatory product recalls and related publicity requirements;
- suspension or withdrawal of regulatory approvals;

- total or partial suspension of production; and

- refusal to approve pending applications for marketing approval of new products or supplements to approved applications.

If side effects increase or are identified during the time our drug candidates are in development or after they are approved and on the market, we may be required to perform lengthy additional clinical trials, change the labeling of any such products, or withdraw any such products from the market, any of which would hinder or preclude our ability to generate revenues.

In our recently completed Phase 2b clinical trial of STA-4783 for metastatic melanoma there were four patients with possible or probable drug-related serious adverse events related to treatment with STA-4783. The first event involved a patient who developed lichenoid dermatitis, a severe rash-like condition, which was considered by the investigator to be possibly related to treatment. The second event involved a patient who experienced atrial fibrillation with rapid ventricular response. This event was also considered by the investigator to be possibly related to treatment. The third event involved an infection which, despite a normal absolute neutrophil count, or ANC, was considered by the investigator to be possibly related to treatment. The fourth event involved severe dehydration that was considered by the investigator to be probably related to treatment. If the incidence of these events increases or if other effects are identified after any of our drug candidates are approved and on the market:

- regulatory authorities may withdraw their approvals;

- we may be required to reformulate any such products, conduct additional clinical trials, make changes in labeling of any such products, or implement changes to or obtain new approvals of our or our contractors' manufacturing facilities;

- we may experience a significant drop in the sales of the affected products;

- our reputation in the marketplace may suffer; and

- we may become the target of lawsuits, including class action suits.

Any of these events could harm or prevent sales of the affected products or could substantially increase the costs and expenses of commercializing and marketing any such products.

We have also observed significant toxicities in preclinical animal studies of our preclinical drug candidate, STA-9090. As a result of these observed toxicities, we may need to begin our Phase 1 clinical trial at a sub-optimal starting dose, which may delay the completion of our Phase 1 clinical trial and the initiation of any future STA-9090 clinical trials. If significant toxicities occur at a clinical dose of STA-9090 which is not sufficiently efficacious, we may not be able to demonstrate an adequate therapeutic index to obtain regulatory approval for STA-9090.

While we chose to test our drug candidates in specific clinical indications based on our understanding of their mechanisms of action, our understanding may be incorrect or incomplete and, therefore, our drugs may not be effective against the diseases tested in our clinical trials.

Our rationale for selecting the particular therapeutic indications for each of our drug candidates is based on our understanding of the mechanism of action of these drug candidates. However, our understanding of the drug candidate's mechanism of action may be incomplete or incorrect, or the mechanism may not be clinically relevant to diseases treated. In such cases, our drug candidates may prove to be ineffective in the clinical trials for treating those diseases.

We deal with hazardous materials and must comply with environmental laws and regulations, which can be expensive and restrict how we do business.

Our activities involve the controlled storage, use, and disposal of hazardous materials, including cytotoxic agents, genotoxic agents, infectious agents, corrosive, explosive and flammable chemicals, and various radioactive compounds. We are subject to federal, state, and local laws and regulations governing the use, manufacture, storage, handling, and disposal of these hazardous materials. Although we believe that our safety procedures for the handling and disposing of these materials comply with the standards prescribed by these laws and regulations, we cannot eliminate the risk of accidental contamination or injury from these materials.

In the event of an accident, state or federal authorities may curtail our use of these materials, and we could be liable for any civil damages that result, which may exceed our financial resources and may seriously harm our business. We currently maintain insurance covering hazardous waste clean up costs in an amount of up to $250,000 per site. Because we believe that our laboratory and materials handling policies and practices sufficiently mitigate the likelihood of materials liability or third-party claims, we currently carry no insurance covering such claims. While we believe that the amount of insurance we carry is sufficient for typical risks regarding our handling of these materials, it may not be sufficient to cover pollution conditions or other extraordinary or unanticipated events. Additionally, an accident could damage, or force us to shut down, our operations. In addition, if we develop a manufacturing capacity, we may incur substantial costs to comply with environmental regulations and would be subject to the risk of accidental contamination or injury from the use of hazardous materials in our manufacturing process.

Risks Related to Our Dependence on Third Parties

We rely on third parties to conduct our clinical trials, and those third parties may not perform satisfactorily, including failing to meet established deadlines for the completion of such clinical trials.

We do not have the ability to independently conduct clinical trials for our drug candidates, and we rely on third parties such as contract research organizations, medical institutions, and clinical investigators to perform this function. Our reliance on these third parties for clinical development activities reduces our control over these activities. Furthermore, these third parties may also have relationships with other entities, some of which may be our competitors. To date, our contract research organizations and other similar entities with which we are working have performed well; however, if these third parties do not successfully carry out their contractual duties or meet expected deadlines, we may be delayed in obtaining regulatory approvals for our drug candidates and may be delayed in our efforts to successfully commercialize our drug candidates for targeted diseases.

We have no manufacturing capacity and depend on third-party manufacturers to produce our clinical trial drug supplies.

We do not currently operate manufacturing facilities for clinical or commercial production of STA-4783 or apilimod, or any of our preclinical drug candidates. We have limited experience in drug manufacturing, and we lack the resources and the capabilities to manufacture any of our drug candidates on a clinical or commercial scale. As a result, we currently rely on third-party manufacturers to supply, store, and distribute drug supplies for our clinical trials and anticipate future reliance on a limited number of third-party manufacturers until we increase the number of manufacturers with whom we contract. Any performance failure on the part of our existing or future manufacturers could delay clinical development or regulatory approval of our drug candidates or commercialization of any approved products, producing additional losses and depriving us of potential product revenue. For example, we have engaged two contract manufacturers to produce the API of STA-4783 for use in our Phase 3 clinical trial for metastatic melanoma. To date, these manufacturers have only produced pilot

batches of STA-4783 API, and there can be no assurances that they will be able to produce STA-4783 API in the quantities and to the specifications needed for our clinical trials.

Our drug candidates require precise, high quality manufacturing. Failure by our contract manufacturers to achieve and maintain high manufacturing standards could result in patient injury or death, product recalls or withdrawals, delays or failures in testing or delivery, cost overruns, or other problems that could seriously hurt our business. Contract manufacturers may encounter difficulties involving production yields, quality control, and quality assurance. These manufacturers are subject to ongoing periodic unannounced inspection by the FDA and corresponding state and foreign agencies to ensure strict compliance with current Good Manufacturing Practice, or cGMP, and other applicable government regulations and corresponding foreign standards; however, we do not have control over third-party manufacturers' compliance with these regulations and standards.

If for some reason our contract manufacturers cannot perform as agreed, we may be unable to replace such third-party manufacturers in a timely manner and the production of our drug candidates would be interrupted, resulting in delays in clinical trials and additional costs. Switching manufacturers may be difficult because the number of potential manufacturers is limited and the FDA must approve any replacement manufacturer prior to manufacturing our drug candidates. Such approval would require new testing and compliance inspections. In addition, a new manufacturer would have to be educated in, or develop substantially equivalent processes for, production of our drug candidates after receipt of FDA approval. It may be difficult or impossible for us to find a replacement manufacturer on acceptable terms quickly, or at all.

In late 2004, we observed granules in some of the capsules of apilimod manufactured by the third-party contractor used in our Phase 2 Crohn's disease and psoriasis clinical trials. We conducted analytical testing and animal studies of the capsules containing the granules and determined that the granules consisted of the API of apilimod rather than impurities. Based on these studies, we believe that the capsules containing the granules were comparable to the capsules without the granules, including with respect to pharmacokinetics and expected absorption in patients. We do not believe that this had any adverse effect on our clinical trials, but there can be no assurance that it did not. We submitted a summary of our findings from the preclinical studies on this issue to the FDA, and the FDA requested the data from these studies that support these findings. We provided these data to the FDA in early February 2005. We have received no further inquiry from the FDA and do not know whether the FDA will require additional information or require that corrective action be taken. Since the identification of these granules, we have performed a comprehensive investigation and believe we identified the cause of the granule formation. We have made improvements to the manufacturing process, and thereafter, no granules have been observed in subsequent batches. Although in our current Phase 2a clinical trials of apilimod in rheumatoid arthritis and CVID we are using a mesylate tablet form of STA-5326, if we decide to use the capsule formulation of apilimod in the future, we do not expect any delay in the clinical development of apilimod due to this issue, but there can be no assurance that no such delay will occur.

We intend to use a single manufacturer for the supply of STA-4783 powder-filled vials for our planned Phase 3 clinical trial and potentially, for commercial supply, and the failure of this manufacturer to supply sufficient quantities of STA-4783 powder-filled vials could have a material adverse effect on our business.

We intend to use a single manufacturer for the supply of STA-4783 powder-filled vials for our planned Phase 3 clinical trial and potentially, for commercial supply, if approved. This process involves highly specialized processing, including the automated filling of vials with STA-4783 under sterile conditions. We believe that this manufacturer may be one of a limited number of third-party contract manufacturers currently capable of conducting this process on our behalf. To date, this third-party manufacturer has verbally agreed and provided a term sheet to meet our manufacturing requirements for the planned Phase 3 clinical trial of STA-4783 for metastatic melanoma and additional Phase 2

clinical trials of STA-4783 for other cancer indications. Although we are currently in discussions with this manufacturer regarding a definitive contract for the supply of STA-4783 for clinical trials and potentially, for commercial supply, there can be no assurances that we will be able to enter into a contract with it on acceptable terms, if at all. Any performance failure on the part of this manufacturer or the failure to enter into a contract with this manufacturer could delay clinical development, regulatory approval or commercialization of STA-4783, which could have a material adverse effect on our business. Moreover, although we believe we have identified a suitable backup manufacturer for STA-4783 powder-filled vials, we do not have an agreement with this manufacturer and there can be no assurance that we will be able to enter into such an agreement on favorable terms, if at all.

We anticipate continued reliance on third-party manufacturers if we are successful in obtaining marketing approval from the FDA and other regulatory agencies for any of our drug candidates.

To date, our drug candidates have been manufactured in small quantities for preclinical testing and clinical trials by third-party manufacturers. If the FDA or other regulatory agencies approve any of our drug candidates for commercial sale, we expect that we would continue to rely, at least initially, on third-party manufacturers to produce commercial quantities of our approved drug candidates. These manufacturers may not be able to successfully increase the manufacturing capacity for any of our approved drug candidates in a timely or economic manner, or at all. Significant scale-up of manufacturing may require additional validation studies, which the FDA must review and approve. If they are unable to successfully increase the manufacturing capacity for a drug candidate, particularly STA-4783, or we are unable to establish our own manufacturing capabilities, the commercial launch of any approved products may be delayed or there may be a shortage in supply.

If we do not establish collaborations, we may have to alter our development plans.

Our drug development programs and potential commercialization of our drug candidates will require substantial additional cash to fund expenses. Our strategy includes potentially selectively collaborating with leading pharmaceutical and biotechnology companies to assist us in furthering development and potential commercialization of some of our drug candidates. Although we are not currently a party to any such collaboration, we may enter into one or more of such collaborations in the future, especially for target indications in which the potential collaborator has particular therapeutic expertise or that involve a large, primary care market that must be served by large sales and marketing organizations or for markets outside of North America. We face significant competition in seeking appropriate collaborators and these collaborations are complex and time-consuming to negotiate and document. We may not be able to negotiate collaborations on acceptable terms, or at all. If that were to occur, we may have to curtail the development of a particular drug candidate, reduce or delay its development program or one or more of our other development programs, delay its potential commercialization or reduce the scope of our sales or marketing activities, or increase our expenditures and undertake development or commercialization activities at our own expense. If we elect to increase our expenditures to fund development or commercialization activities on our own, we may need to obtain additional capital, which may not be available to us on acceptable terms, or at all. If we do not have sufficient funds, we will not be able to bring our drug candidates to market and generate product revenue.

If we are unable to establish sales and marketing capabilities or enter into agreements with third parties to market and sell our drug candidates, we may be unable to generate product revenue.

We do not currently have an organization for the sales, marketing, and distribution of pharmaceutical products. In order to market any products that may be approved by the FDA, we must build our sales, marketing, managerial, and other non-technical capabilities or make arrangements with third parties to perform these services. If we are unable to establish adequate sales, marketing, and

distribution capabilities, whether independently or with third parties, we may not be able to generate product revenue and may not become profitable.

Risks Related to Our Intellectual Property

If our patent position does not adequately protect our drug candidates or any future products, others could compete against us more directly, which would harm our business

As of March 15, 2007, our patent portfolio consisted of a total of 520 patents and patent applications worldwide with claims covering the composition-of-matter and methods of use for both of our clinical stage compounds. We own or license a total of 23 issued U.S. patents and 106 U.S. patent applications, as well as 391 foreign patents and patent applications. We have issued U.S. composition-of-matter patents claiming the chemical structures of STA-4783 and apilimod.

Our commercial success will depend in part on our ability to obtain additional patents and protect our existing patent position as well as our ability to maintain adequate protection of other intellectual property for our technologies, drug candidates, and any future products in the United States and other countries. If we do not adequately protect our intellectual property, competitors may be able to use our technologies and erode or negate any competitive advantage we may have, which could harm our business and ability to achieve profitability. The laws of some foreign countries do not protect our proprietary rights to the same extent as the laws of the United States, and we may encounter significant problems in protecting our proprietary rights in these countries.

The patent positions of biotechnology and pharmaceutical companies, including our patent position, involve complex legal and factual questions, and, therefore, validity and enforceability cannot be predicted with certainty. Patents may be challenged, deemed unenforceable, invalidated, or circumvented. We will be able to protect our proprietary rights from unauthorized use by third parties only to the extent that our proprietary technologies, drug candidates, and any future products are covered by valid and enforceable patents or are effectively maintained as trade secrets.

In addition, although we do not believe that any of the patents or patent applications that we currently license are material to our business, we may in the future license intellectual property that is material to us. In such cases, we may be dependent upon the licensors to obtain, maintain and enforce patent protection for the licensed intellectual property. These licensors may not successfully prosecute patent applications or may fail to maintain issued patents. The licensors may also determine not to pursue litigation against other companies that infringe the patents, or may pursue such litigation less aggressively than we would. If any of the foregoing occurs, and the terms of any such future license do not allow us to assume control of patent prosecution, maintenance and enforcement, any competitive advantage we may have due to the license may be diminished or eliminated.

The degree of future protection for our proprietary rights is uncertain, and we cannot ensure that:

- we or our licensors were the first to make the inventions covered by each of our pending patent applications;
- we or our licensors were the first to file patent applications for these inventions;
- others will not independently develop similar or alternative technologies or duplicate any of our technologies;
- any of our or our licensors' pending patent applications will result in issued patents;
- any of our or our licensors' patents will be valid or enforceable;
- any patents issued to us or our licensors and collaborators will provide a basis for commercially viable products, will provide us with any competitive advantages or will not be challenged by third parties;

- we will develop additional proprietary technologies or drug candidates that are patentable; or
- the patents of others will not have an adverse effect on our business.

We typically file for patent protection first on the composition-of-matter of our drug candidates and also claim their activities and methods for their production and use to the extent known at that time. As we learn more about the mechanisms of action and new methods of manufacture and use of these drug candidates, we generally file additional patent applications for these new inventions. Although our patents may prevent others from making, using, or selling similar products, they do not ensure that we will not infringe the patent rights of third parties. For example, because we sometimes identify the mechanism of action or molecular target of a given drug candidate after identifying its composition-of-matter and therapeutic use, we may not be aware until the mechanism or target is further elucidated that a third party has an issued or pending patent claiming biological activities or targets that may cover our drug candidate. If such a patent exists or is granted in the future, we cannot provide assurances that a license will be available on commercially reasonable terms, or at all.

We may be unable to adequately prevent disclosure of trade secrets and other proprietary information.

We rely on trade secrets to protect our proprietary technologies, especially where we do not believe patent protection is appropriate or obtainable. However, trade secrets are difficult to protect. We rely in part on confidentiality agreements with our employees, consultants, outside scientific collaborators, sponsored researchers, and other advisors to protect our trade secrets and other proprietary information. These agreements may not effectively prevent disclosure of confidential information and may not provide an adequate remedy in the event of unauthorized disclosure of confidential information. In addition, others may independently discover our trade secrets and proprietary information. Costly and time-consuming litigation could be necessary to enforce and determine the scope of our proprietary rights, and failure to obtain or maintain trade secret protection could adversely affect our competitive business position.

Litigation or other proceedings or third-party claims of intellectual property infringement would require us to spend time and money and could prevent us from developing or commercializing our drug candidates.

Our commercial success will depend in part on not infringing upon the patents and proprietary rights of other parties and enforcing our own patents and proprietary rights against others. Certain of our research and development programs are in highly competitive fields in which numerous third parties have issued patents and patent applications with claims closely related to the subject matter of our programs. We are not currently aware of any litigation or other proceedings or claims by third parties that our drug candidates, technologies or methods infringe their intellectual property.

However, while it is our practice to conduct freedom to operate searches and analyses, we cannot guarantee that we have identified every patent or patent application that may be relevant to the research, development or commercialization of our drug candidates. Moreover, there can be no assurance that third parties will not assert against us patents that we believe are not infringed by us or are invalid. For example, we are aware of a U.S. patent and a related European patent that claim generic chemical structures, pharmaceutical formulations and methods of treatment relating to compounds similar to STA-9090 and a U.S. patent that claims methods of treating certain cancers using Hsp90 inhibitors. The claims of these patents may be relevant to the commercialization of our drug candidate, STA-9090. However, based on our analysis of these patents, we do not believe that the manufacture, use, importation or sale of STA-9090 would infringe any valid claim of these patents. However, we cannot guarantee that these patents would not be asserted against us and, if asserted, that a court would find these patents to be invalid or not infringed.

In the event of a successful infringement action against us with respect to any third party patent rights, we may be required to:

- pay substantial damages;
- stop developing, commercializing, and selling the infringing drug candidates or approved products;
- stop utilizing the infringing technologies and methods in our drug candidates or approved products;
- develop non-infringing products, technologies, and methods; and
- obtain one or more licenses from other parties, which could result in our paying substantial royalties or our granting of cross licenses to our technologies.

We may not be able to obtain licenses from other parties at a reasonable cost, or at all. If we are not able to obtain necessary licenses at a reasonable cost, or at all, we could encounter substantial delays in product introductions while we attempt to develop alternative technologies, methods, and products, which we may not be able to accomplish. Although third parties may challenge our rights to, or the scope or validity of our patents, to date, we have not received any communications from third parties challenging our patents or patent applications covering our drug candidates.

We may be subject to claims that we have wrongfully hired an employee from a competitor or that we or our employees have wrongfully used or disclosed alleged confidential information or trade secrets of their former employers.

As is commonplace in our industry, we employ individuals who were previously employed at other biotechnology or pharmaceutical companies, including our competitors or potential competitors. Although no claims against us are currently pending, we have previously been subject to a claim by an alleged competitor that a prospective employee we sought to hire was bound by an ongoing non-competition obligation which prevented us from hiring this employee. We may be subject in the future to claims that our employees or prospective employees are subject to a continuing obligation to their former employers (such as non-competition or non-solicitation obligations) or claims that our 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.

Risks Related to the Commercialization of Our Drug Candidates

If physicians and patients do not accept our future products or if the markets for indications for which any drug candidate is approved is smaller than expected, we may be unable to generate significant revenue, if any.

Even if STA-4783, apilimod, STA-9090, or any other drug candidates we may develop or acquire in the future obtain regulatory approval, they may not gain market acceptance among physicians, healthcare payors, patients, and the medical community. Physicians may elect not to recommend these drugs for a variety of reasons including:

- timing of market introduction of competitive products, including other melanoma treatments, currently in development (such as Nexavar, Sutent, ispinesib, ipilimumab, ticilimumab, volociximab, M-Vax and MDX-1379, as well as forms of chemotherapy);
- demonstration of clinical safety and efficacy compared to other products;
- cost-effectiveness;

- availability of reimbursement from managed care plans and other third-party payors;
- convenience and ease of administration;
- prevalence and severity of adverse side effects;
- other potential advantages of alternative treatment methods; and
- ineffective marketing and distribution support of our products.

If our approved drugs fail to achieve market acceptance, we may not be able to generate significant revenue and our business would suffer.

In addition, we intend to initiate a Phase 3 clinical trial for our most advanced clinical-stage candidate, STA-4783, in patients with stage IV metastatic melanoma in the middle of 2007. We currently estimate that there are relatively few people with metastatic melanoma in the United States. Even if we are successful in obtaining regulatory approval to market STA-4783 for this indication, the market for this indication may not be sufficient to generate significant revenue and our business would suffer.

If the government and third-party payors fail to provide adequate coverage and reimbursement rates for our future products, if any, our revenue and prospects for profitability will be harmed.

In both domestic and foreign markets, our sales of any future products will depend in part upon the availability of reimbursement from third-party payors. Such third-party payors include government health programs such as Medicare, managed care providers, private health insurers, and other organizations. These third-party payors are increasingly attempting to contain healthcare costs by demanding price discounts or rebates and limiting both coverage and the amounts that they will pay for new drugs, and, as a result, they may not cover or provide adequate payment for our drugs. We might need to conduct post-marketing studies in order to demonstrate the cost-effectiveness of any future products to such payors' satisfaction. Such studies might require us to commit a significant amount of management time and financial and other resources. Our future products might not ultimately be considered cost-effective. Adequate third-party reimbursement might not be available to enable us to maintain price levels sufficient to realize an appropriate return on investment in product development.

U.S. and foreign governments continue to propose and pass legislation designed to reduce the cost of healthcare. For example, in some foreign markets, the government controls the pricing and profitability of prescription pharmaceuticals. In the United States, we expect that there will continue to be federal and state proposals to implement similar governmental controls. In addition, recent changes in the Medicare program and increasing emphasis on managed care in the United States will continue to put pressure on pharmaceutical product pricing. Cost control initiatives could decrease the price that we would receive for any products in the future, which would limit our revenue and profitability. Accordingly, legislation and regulations affecting the pricing of pharmaceuticals might change before our drug candidates are approved for marketing. Adoption of such legislation could further limit reimbursement for pharmaceuticals.

For example, the Medicare Prescription Drug Improvement and Modernization Act of 2003, or MMA, changes the way Medicare will cover and pay for pharmaceutical products. The legislation expanded Medicare coverage for drug purchases by the elderly and disabled and introduced new reimbursement methodologies, based on average sales prices for drugs that are administered in an in-patient setting or by physicians, such as STA-4783, if approved. In addition, this legislation provides authority for limiting the number of drugs that will be covered in any therapeutic class. Although we do not know what the full impact of the new reimbursement methodologies will have on the prices of new drugs, we expect that there will be added pressure to contain and reduce costs. These cost reduction initiatives and other provisions of this legislation could decrease the coverage and price that we receive

for any approved products and could seriously harm our business. While the MMA applies only to drug benefits for Medicare beneficiaries, private payors often follow Medicare coverage policy and payment limitations in setting their own reimbursement rates, and any reduction in reimbursement that results from the MMA may result in a similar reduction in payments from private payors.

If a successful product liability claim or series of claims is brought against us for uninsured liabilities or in excess of insured liabilities, we could be forced to pay substantial damage awards.

The use of any of our drug candidates in clinical trials, and the sale of any approved products, might expose us to product liability claims. We currently maintain product liability insurance coverage in an amount of up to $10.0 million, which we believe is adequate for our clinical trials currently in progress. We monitor the amount of coverage we maintain as the size and design of our clinical trials evolve and intend to adjust the amount of coverage we maintain accordingly. However, there can be no assurance that such insurance coverage will fully protect us against some or all of the claims to which we might become subject. We might not be able to maintain adequate insurance coverage at a reasonable cost or in sufficient amounts or scope to protect us against potential losses. In the event a claim is brought against us, we might be required to pay legal and other expenses to defend the claim, as well as uncovered damages awards resulting from a claim brought successfully against us. Furthermore, whether or not we are ultimately successful in defending any such claims, we might be required to direct financial and managerial resources to such defense and adverse publicity could result, all of which could harm our business.

If we inadvertently violate the guidelines pertaining to promotion and advertising of our clinical candidates or approved products, we may be subject to disciplinary action by the FDA's Division of Drug Marketing, Advertising, and Communications or other regulatory bodies.

The FDA's Division of Drug Marketing, Advertising, and Communications, or DDMAC, is responsible for reviewing prescription drug advertising and promotional labeling to ensure that the information contained in these materials is not false or misleading. There are specific disclosure requirements and the applicable regulations mandate that advertisements cannot be false or misleading or omit material facts about the product. Prescription drug promotional materials must present a fair balance between the drug's effectiveness and the risks associated with its use. Most warning letters from DDMAC cite inadequate disclosure of risk information.

DDMAC prioritizes its actions based on the degree of risk to the public health, and often focuses on newly introduced drugs and those associated with significant health risks. There are two types of letters that DDMAC typically sends to companies which violate its drug advertising and promotional guidelines: notice of violation letters, or untitled letters, and warning letters. In the case of an untitled letter, DDMAC typically alerts the drug company of the violation and issues a directive to refrain from future violations, but does not typically demand other corrective action. A warning letter is typically issued in cases that are more serious or where the company is a repeat offender. Although we have not received any such letters from DDMAC, we may inadvertently violate DDMAC's guidelines in the future and be subject to a DDMAC untitled letter or warning letter, which may have a negative impact on our business.

Risks Related to Our Industry

We may not be able to keep up with the rapid technological change in the biotechnology and pharmaceutical industries, which could make any future approved products obsolete and reduce our revenue.

Biotechnology and related pharmaceutical technologies have undergone and continue to be subject to rapid and significant change. Our future will depend in large part on our ability to maintain a competitive position with respect to these technologies. Our competitors may render our technologies

obsolete by advances in existing technological approaches or the development of new or different approaches, potentially eliminating the advantages in our drug discovery process that we believe we derive from our research approach and proprietary technologies. In addition, any future products that we develop, including our clinical drug candidates, STA-4783 and apilimod, and our preclinical drug candidates, STA-9090 and STA-9584, may become obsolete before we recover expenses incurred in developing those products, which may require that we raise additional funds to continue our operations.

Our market is subject to intense competition. If we are unable to compete effectively, our drug candidates may be rendered noncompetitive or obsolete.

We are engaged in segments of the pharmaceutical industry that are highly competitive and rapidly changing. Many large pharmaceutical and biotechnology companies, academic institutions, governmental agencies, and other public and private research organizations are pursuing the development of novel drugs that target cancer and chronic inflammatory diseases. We face, and expect to continue to face, intense and increasing competition as new products enter the market and advanced technologies become available. In addition to currently approved drugs, there are a significant number of drugs that are currently under development and may become available in the future for the treatment of cancer and chronic inflammatory diseases. We would expect STA-4783 and apilimod to compete with marketed drugs and drug candidates currently under development, including the following:

- *STA-4783.* If approved, we would expect STA-4783 to compete with currently approved drugs for the treatment of metastatic melanoma, including dacarbazine/DTIC marketed by Bayer, and generic versions thereof, the injectable protein interleukin 2, or IL-2, marketed by Chiron, and the injectable protein interferon alfa-2b, marketed by Schering-Plough. STA-4783 may also compete with drug candidates currently in clinical development by other companies, including: (1) kinase inhibitors such as Nexavar, being developed by Bayer and Onyx, Sutent, being developed by Pfizer, and ispinesib, being developed by Cytokinetics and GlaxoSmithKline; (2) the anti-CTLA-4 monoclonal antibodies, ipilimumab and ticilimumab; (3) the anti-integrin volociximab; (4) cancer vaccines such as M-Vax and MDX-1379; and (5) derivatives, analogs, or reformulations of known chemotherapies, such as Abraxane, or other cytotoxic chemotherapies. In addition, STA-4783 may compete against drugs not currently approved for the treatment of metastatic melanoma, but which are commonly used "off-label" to treat this disease, such as taxanes, temozolomide, vincristine, carmustine, melphalan, and platinum-chemotherapeutics, such as cisplatin and carboplatin.

- *Apilimod.* If approved, we would expect apilimod to compete with other treatments of chronic inflammatory diseases, including (1) large-molecule, injectable TNF α antagonists, such as Remicade, marketed by Johnson & Johnson, Enbrel, marketed by Amgen and Wyeth Pharmaceuticals, and Humira, marketed by Abbott Laboratories, (2) broadly immunosuppressive small molecule agents, including corticosteroids, methotrexate, and azathioprine, and (3) CNTO-1275 and ABT-874, two injectable antibody-based clinical candidates targeting IL-12 currently in clinical trials that are being developed by Johnson & Johnson and Abbott Laboratories, respectively.

- *STA-9090.* If approved, we would expect STA-9090 to compete with the currently approved therapies for the treatment of cancers, and other cancer treatments currently under development, including 17-AAG, being developed by Kosan, and other agents that inhibit Hsp90, including Hsp90 inhibitors being developed by Medimmune/Infinity, BiogenIdec, and Novartis/Vernalis.

- *STA-9584.* If approved, we would expect STA-9584 to compete with the currently approved therapies for the treatment of cancers, and other cancer treatments currently under development, including other vascular disrupting agents, such as ABT-751, being developed by Abbott; AS1404, being developed by Antisoma, CA4P, being developed by Oxigene, EXEL-0999, being developed by Exelixis, and ZD6126, being developed by Angiogene.

Many of our competitors have:

- significantly greater financial, technical and human resources than we have and may be better equipped to discover, develop, manufacture and commercialize drug candidates;

- more extensive experience in preclinical testing and clinical trials, obtaining regulatory approvals and manufacturing and marketing pharmaceutical products;

- drug candidates that have been approved or are in late-stage clinical development; and/or

- collaborative arrangements in our target markets with leading companies and research institutions.

Competitive products may render our products obsolete or noncompetitive before we can recover the expenses of developing and commercializing our drug candidates. Furthermore, the development of new treatment methods and/or the widespread adoption or increased utilization of any vaccine for the diseases we are targeting could render our drug candidates noncompetitive, obsolete or uneconomical. If we successfully develop and obtain approval for our drug candidates, we will face competition based on the safety and effectiveness of our drug candidates, the timing of their entry into the market in relation to competitive products in development, the availability and cost of supply, marketing and sales capabilities, reimbursement coverage, price, patent position and other factors. If we successfully develop drug candidates but those drug candidates do not achieve and maintain market acceptance, our business will not be successful.

Risks Related to Employee Matters and Managing Growth

Our future success depends on our ability to retain our chief executive officer and other key executives and to attract, retain, and motivate qualified personnel.

We are highly dependent on Safi R. Bahcall, Ph.D., our President and Chief Executive Officer, and the other principal members of our executive and scientific teams listed under Item 10 "Directors and Executive Officers of the Registrant" below. All of the agreements with these principal members of our executive and scientific teams provide that employment is at-will and may be terminated by the employee at any time and without notice. Although we do not have any reason to believe that we may lose the services of any of these persons in the foreseeable future, the loss of the services of any of these persons might impede the achievement of our research, development, and commercialization objectives. Recruiting and retaining qualified scientific personnel and possibly sales and marketing personnel will also be critical to our success. We may not be able to attract and retain these personnel on acceptable terms given the competition among numerous pharmaceutical and biotechnology companies for similar personnel. We also experience competition for the hiring of scientific personnel from universities and research institutions. We do not maintain "key person" insurance on any of our employees. In addition, we rely on consultants and advisors, including scientific and clinical advisors, to assist us in formulating our research and development and commercialization strategy. Our consultants and advisors may be employed by employers other than us and may have commitments under consulting or advisory contracts with other entities that may limit their availability to us.

We expect to expand our development, clinical research, and marketing capabilities, and as a result, we may encounter difficulties in managing our growth, which could disrupt our operations.

We expect to experience significant growth in the number of our employees and the scope of our operations. To manage our anticipated future growth, we must continue to implement and improve our managerial, operational, and financial systems, expand our facilities, and continue to recruit and train additional qualified personnel. Due to our limited resources, we may not be able to effectively manage the expansion of our operations or recruit and train additional qualified personnel. The physical expansion of our operations may lead to significant costs and may divert our management and business development resources. Any inability to manage growth could delay the execution of our business plans or disrupt our operations.

If we make strategic acquisitions, we will incur a variety of costs and might never realize the anticipated benefits.

All of our acquisitions to date have been of related parties. Accordingly, we have very limited experience in independently identifying acquisition candidates and integrating the operations of truly independent acquisition candidates with our company. Currently we are not a party to any acquisition agreements, nor do we have any understanding or commitment with respect to any such acquisition. If appropriate opportunities become available, however, we might attempt to acquire approved products, additional drug candidates, or businesses that we believe are a strategic fit with our business. If we pursue any transaction of that sort, the process of negotiating the acquisition and integrating an acquired product, drug candidate, or business might result in operating difficulties and expenditures and might require significant management attention that would otherwise be available for ongoing development of our business, whether or not any such transaction is ever consummated. Moreover, we might never realize the anticipated benefits of any acquisition. Future acquisitions could result in potentially dilutive issuances of equity securities, the incurrence of debt, contingent liabilities, or impairment expenses related to goodwill, and impairment or amortization expenses related to other intangible assets, which could harm our financial condition.

Risks Related to Our Common Stock

Our stock price is likely to be volatile and the market price of our common stock may drop.

Prior to our February 2007 initial public offering, there was not a public market for our common stock. There is a limited history on which to gauge the volatility of our stock price. In addition, the stock market has recently experienced significant volatility, particularly with respect to pharmaceutical, biotechnology, and other life sciences company stocks. The volatility of pharmaceutical, biotechnology, and other life sciences company stocks often does not relate to the operating performance of the companies represented by the stock. Some of the factors that may cause the market price of our common stock to fluctuate include:

- plans for, progress in, and results from our planned Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma or any other future clinical trials of STA-4783 we may initiate;
- results of our current Phase 2a or any future clinical trials of apilimod we may initiate;
- results of clinical trials conducted by others on drugs that would compete with our drug candidates;
- failure or delays in advancing STA-9090, STA-9584 or our CRAC ion channel inhibitor program, or other drug candidates we may discover or acquire in the future, into clinical trials;
- failure or discontinuation of any of our research programs;
- issues in manufacturing our drug candidates or approved products;

- regulatory developments or enforcement in the United States and foreign countries;
- developments or disputes concerning patents or other proprietary rights;
- introduction of technological innovations or new commercial products by us or our competitors;
- changes in estimates or recommendations by securities analysts, if any cover our common stock;
- public concern over our drug candidates or any approved products;
- litigation;
- future sales of our common stock;
- general market conditions;
- changes in the structure of healthcare payment systems;
- failure of any of our drug candidates, if approved, to achieve commercial success;
- economic and other external factors or other disasters or crises;
- period-to-period fluctuations in our financial results; and
- overall fluctuations in U.S. equity markets.

These and other external factors may cause the market price and demand for our common stock to fluctuate substantially, which may limit or prevent investors from readily selling their shares of common stock and may otherwise negatively affect the liquidity of our common stock. In addition, in the past, when the market price of a stock has been volatile, holders of that stock have instituted securities class action litigation against the company that issued the stock. If any of our stockholders brought a lawsuit against us, we could incur substantial costs defending the lawsuit. Such a lawsuit could also divert the time and attention of our management.

Insiders have substantial control over us which could delay or prevent a change in corporate control or result in the entrenchment of management and/or the board of directors.

Our directors, executive officers and principal stockholders, together with their affiliates and related persons, beneficially own, in the aggregate, approximately 49.3% of our outstanding common stock. These stockholders, if acting together, may have the ability to determine the outcome of matters submitted to our stockholders for approval, including the election and removal of directors and any merger, consolidation, or sale of all or substantially all of our assets. In addition, these persons, acting together, may have the ability to control the management and affairs of our company. Accordingly, this concentration of ownership may harm the market price of our common stock by:

- delaying, deferring, or preventing a change in control;
- entrenching our management and/or the board of directors;
- impeding a merger, consolidation, takeover, or other business combination involving us; or
- discouraging a potential acquirer from making a tender offer or otherwise attempting to obtain control of us.

Future sales of a large number of shares of our common stock by our stockholders after the lock-up agreements from our initial public offering expire may cause our stock price to fall.

As of March 15, 2007, we had 33,816,532 outstanding shares of common stock. Approximately 29,474,996 of such shares are subject to lock-up agreements that the majority of our stockholders entered into with the underwriters for our initial public offering. These lock-up agreements restrict the

ability of such stockholders to transfer their shares of stock until August 5, 2007, unless such date is extended pursuant to the terms of the lock-up agreements, at which time their shares will be eligible for sale in the public market, subject to volume limitations for any such shares held by our affiliates. The market price of our common stock may drop significantly when these restrictions on transfer lapse and our stockholders are able to sell shares of our common stock into the market.

Provisions of our charter, bylaws, and Delaware law may make an acquisition of us or a change in our management more difficult.

Certain provisions of our restated certificate of incorporation and restated bylaws could discourage, delay, or prevent a merger, acquisition, or other change in control that stockholders may consider favorable, including transactions in which stockholders might otherwise receive a premium for their shares. These provisions also could limit the price that investors might be willing to pay in the future for shares of our common stock, thereby depressing the market price of our common stock. Stockholders who wish to participate in these transactions may not have the opportunity to do so. Furthermore, these provisions could prevent or frustrate attempts by our stockholders to replace or remove our management. These provisions:

- allow the authorized number of directors to be changed only by resolution of our board of directors;
- establish a classified board of directors, providing that not all members of the board of directors be elected at one time;
- authorize our board of directors to issue without stockholder approval blank check preferred stock that, if issued, could operate as a "poison pill" to dilute the stock ownership of a potential hostile acquirer to prevent an acquisition that is not approved by our board of directors;
- require that stockholder actions must be effected at a duly called stockholder meeting and prohibit stockholder action by written consent;
- establish advance notice requirements for stockholder nominations to our board of directors or for stockholder proposals that can be acted on at stockholder meetings;
- limit who may call stockholder meetings; and
- require the approval of the holders of 80% of the outstanding shares of our capital stock entitled to vote in order to amend certain provisions of our restated certificate of incorporation and restated bylaws.

In addition, because we are incorporated in Delaware, we are governed by the provisions of Section 203 of the Delaware General Corporation Law, which may, unless certain criteria are met, prohibit large stockholders, in particular those owning 15% or more of our outstanding voting stock, from merging or combining with us for a prescribed period of time.

We do not anticipate paying cash dividends, and accordingly, stockholders must rely on stock appreciation for any return on their investment.

We currently intend to retain our future earnings, if any, to fund the development and growth of our business. As a result, capital appreciation, if any, of our common stock will be the sole source of gain on an investment in our common stock for the foreseeable future.

Item 1B. UNRESOLVED STAFF COMMENTS

Not applicable.

Item 2. PROPERTIES

Our operations are based primarily in Lexington, Massachusetts, which is located approximately 10 miles west of Boston, Massachusetts. We currently lease a total of 68,730 square feet of office and laboratory space in Lexington and 15,000 square feet of office and laboratory space in the neighboring town of Bedford, Massachusetts. We lease the following properties:

Location	Approximate Square Feet	Use	Lease Expiration Date
45 Hartwell Avenue Lexington, Massachusetts	24,420	Office and Laboratory	Nov. 2011
91 Hartwell Avenue Lexington, Massachusetts	21,830	Office	Feb. 2008
125 Hartwell Avenue Lexington, Massachusetts	22,480	Office and Laboratory	Jan. 2008
45-47 Wiggins Avenue Bedford, Massachusetts	15,000	Office and Laboratory	Oct. 2011

We believe our facilities are adequate for our current needs.

Item 3. LEGAL PROCEEDINGS

We are currently not a party to any material legal proceedings.

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

No matters were submitted to a vote of security holders during the fourth quarter of the year ended December 31, 2006.

PART II

Item 5. MARKET FOR REGISTRANT'S COMMON EQUITY, RELATED STOCKHOLDER MATTERS AND ISSUER PURCHASES OF EQUITY SECURITIES

Market Information

Our common stock began trading on the Nasdaq Global Market on February 6, 2007 under the symbol "SNTA." Prior to that time, there was no established public trading market for our common stock. The high and low sales prices per share of our common stock on the Nasdaq Global Market for the period from February 6, 2007 through March 15, 2007 were $10.10 and $8.19, respectively.

Stockholders

As of March 15, 2007, there were approximately 197 stockholders of record of the 33,816,532 outstanding shares of our common stock.

Dividends

We have never paid or declared any cash dividends on our common stock. We currently intend to retain all available funds and any future earnings to fund the development and expansion of our business, and we do not anticipate paying any cash dividends in the foreseeable future. Any future determination to pay dividends will be at the discretion of our board of directors and will depend on our financial condition, results of operations, capital requirements, and other factors that our board of directors deems relevant. In addition, the terms of any future debt or credit facility may preclude us from paying dividends.

Unregistered Sales of Securities

During the year ended December 31, 2006, we made the following issuances of securities which were not registered under the Securities Act of 1933, as amended.

- on April 14, 2006, we issued 4,875 shares of our common stock at a purchase price per share of $14.00 for an aggregate purchase price of $68,260.50 to our President and Chief Executive Officer as partial payment for his annual bonus;
- on June 2, 2006, we issued and sold 8,000,000 shares of our Series A convertible preferred stock at a purchase price per share of $5.00 to 42 accredited investors for an aggregate purchase price of $40,000,000;
- on November 17, 2006, we issued 12,142 shares of restricted common stock to our non-employee directors as compensation for services as a director at a purchase price of $0.0004 per share for an aggregate purchase price of $4.86; and
- During 2006, we granted options to employees, consultants and directors to purchase 750,987 shares of our common stock. In addition, 125 shares of common stock were issued during 2006 upon the exercise of stock options.

All of these issuances were made in reliance on Section 4(2) of the Securities Act of 1933, as amended, or Regulation D promulgated thereunder as sales not involving a public offering, except with respect to the issuance of stock options and shares of common stock issued upon exercise of stock options, which were issued pursuant to written compensatory plans or arrangements with our employees consultants and directors in reliance on the exemption from registration under the Securities Act of 1933, as amended, provided by Rule 701 promulgated under the Securities Act of 1933, as amended. No underwriters were involved in the foregoing sales of securities. The recipients of securities in each of the above-referenced transactions represented their intentions to acquire the securities for

investment purposes only and not with a view to, or for sale in connection with, any distribution thereof and appropriate legends were affixed to the instruments representing such securities issued in such transactions. All recipients either received adequate information about us or had, through their relationship with us, adequate access to such information.

Issuer Purchases of Equity Securities

None.

Use of Proceeds from Registered Securities

We registered shares of our common stock in connection with our initial public offering under the Securities Act of 1933, as amended. Our Registration Statement on Form S-1 (Reg. No. 333-138894) in connection with our initial public offering was declared effective by the Securities and Exchange Commission on February 6, 2007. The offering commenced on February 6, 2007 and terminated before all securities were sold. 5,750,000 shares of our common stock were registered, of which 5,000,000 were sold. The underwriters of the offering were Bear, Stearns & Co. Inc., Lehman Bothers Inc., Lazard Capital Markets LLC and Montgomery & Co., LLC. The 5,000,000 shares of our common stock sold in the offering were sold at the initial public offering price per share of $10.00. The aggregate proceeds of the offering was $50,000,000. The net offering proceeds to us after deducting total expenses were $44,700,000. We incurred total expenses in connection with the offering of $5,300,000, which consisted of direct payments of:

- $1,550,000 in legal, accounting and printing fees;
- $3,500,000 in underwriters' discounts, fees and commissions; and
- $149,305 in registration, listing and filing fees;
- $100,695 in miscellaneous expenses.

No payments for such expenses were made directly or indirectly to (i) any of our directors, officers or their associates, (ii) any person(s) owning 10% or more of any class of our equity, securities or (iii) any of our affiliates.

We estimate that we will use the net proceeds of the initial public offering as follows:

- approximately $22 to $24 million of these net proceeds to fund the continued clinical development of STA-4783, including the initiation of a pivotal Phase 3 clinical trial in metastatic melanoma in 2007 and the initiation of Phase 2 clinical trials in up to two other indications in 2007;
- approximately $2 to $3 million of these net proceeds to fund the continued clinical development of apilimod, including the completion of our current Phase 2a clinical trials in rheumatoid arthritis and CVID and potentially the initiation of Phase 2b clinical trials in these indications, depending on the results of the Phase 2a trials;
- approximately $10 to $12 million of these net proceeds to fund the continued research, preclinical and future clinical development of STA-9090, STA-9584 and our CRAC ion channel program; and
- approximately $8 million to fund general corporate purposes, such as general and administrative expenses, capital expenditures, working capital needs, prosecution and maintenance of our intellectual property, and the potential acquisition of, or investment in, technologies, products, or companies that complement our business.

We cannot predict with certainty all of the particular uses for the proceeds from the initial public offering, or the amounts that we will actually spend on the uses set forth above. The amounts and timing of our actual expenditures will depend upon numerous factors, including the progress of our research, development, and commercialization efforts, the progress of our clinical trials, whether or not we enter into strategic collaborations or partnerships, and our operating costs and expenditures. Accordingly, our management will have significant flexibility in applying the net proceeds of the initial public offering.

The net offering proceeds have been invested into short-term investment-grade securities and money market accounts.

There has been no material change in the planned use of proceeds from our initial public offering as described in our final prospectus dated February 6, 2007 filed with the Securities and Exchange Commission pursuant to Rule 424(b)(4).

Item 6. SELECTED FINANCIAL DATA

The following table sets forth our selected consolidated financial data and has been derived from our audited consolidated financial statements. Consolidated balance sheets as December 31, 2006 and 2005, as well as consolidated statements of operations for the years ended December 31, 2006, 2005, and 2004 and the period from inception (March 10, 2000) through December 31, 2006, and the report thereon are included elsewhere in this Annual Report on Form 10-K. The information below should be read in conjunction with our audited consolidated financial statements (and notes thereon) and "Management's Discussion and Analysis of Financial Condition and Results of Operations," included below in Item 7.

	Years ended December 31,					Period from inception (March 10, 2000) through December 31, 2006
	2006	2005	2004	2003	2002	
Consolidated Statement of Operations Data:						
Revenues	$ —	$ —	$ 173	$ 1,304	$ —	$ 1,477
Operating expenses						
Research and development	50,503	59,901	38,136	24,337	7,292	180,446
In-process research and development(1)	—	—	1,583	—	18,088	19,671
General and administrative	8,648	11,279	7,383	5,261	1,569	34,342
Other compensation expense	—	—	—	—	9,315	9,315
Total operating expenses	59,151	71,180	47,102	29,598	36,264	243,774
Loss from operations	(59,151)	(71,180)	(46,929)	(28,294)	(36,264)	(242,297)
Investment income, net	1,881	2,317	995	416	110	5,739
Net loss	(57,270)	(68,863)	(45,934)	(27,878)	(36,154)	(236,558)
Convertible preferred stock dividends	1,859	—	—	—	—	1,859
Net loss attributable to common stockholders	$ (59,129)	$ (68,863)	$ (45,934)	$ (27,878)	$ (36,154)	$ (238,417)
Basic and diluted net loss attributable to common stockholders per share	$ (2.66)	$ (3.09)	$ (2.46)	$ (1.86)	$ (4.37)	
Weighted average shares used in computing basic and diluted net loss per common share	22,265	22,253	18,704	15,024	8,279	

(1) In September 2002 and December 2002 we acquired Principia Associates, Inc. and Diagon Genetics, Inc., respectively. See note 3 to our audited consolidated financial statements.

	As of December 31,					Pro forma as of December 31, 2006(1)
	2006	2005	2004	2003	2002	
Consolidated Balance Sheet Data:						
Cash, cash equivalents and marketable securities	$ 46,824	$ 62,057	$ 124,968	$ 76,226	$ 28,952	$ 91,524
Working capital	36,081	48,476	113,147	73,564	27,574	81,744
Total assets	54,789	71,210	132,019	80,387	33,173	98,526
Capital lease obligations, net of current portion	3,170	4,259	1,188	—	—	3,170
Convertible preferred stock	41,820	—	—	—	—	—
Common stock	2	2	2	2	1	3
Additional paid-in capital	234,807	239,029	238,930	144,154	68,434	321,326
Deficit accumulated during the development stage	(236,558)	(179,288)	(110,425)	(64,491)	(36,613)	(236,558)
Total stockholders' equity (deficit)	(1,747)	52,477	117,956	76,891	31,151	84,773

(1) The pro forma balance sheet data as of December 31, 2006 gives effect to and reflects the $50.0 million in gross proceeds from the sale of 5,000,000 shares of common stock at $10.00 per share in our initial public offering on February 9, 2007, net of $5.3 million in expenses for underwriters' discounts, fees and commissions, legal, accounting, printing and listing and filing fees, and miscellaneous expenses, and the conversion of all outstanding shares of our Series A convertible preferred stock and accumulated dividends into 6,278,765 shares of common stock upon the closing of our initial public offering.

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

This Management's Discussion and Analysis of Financial Condition and Results of Operations should be read together with the consolidated financial statements, related notes and other financial information included elsewhere in this Annual Report on Form-10K.

Overview

We are a biopharmaceutical company focused on discovering, developing and commercializing small molecule drugs that address severe medical conditions with large potential markets, including cancer and chronic inflammatory diseases. We have two drug candidates in clinical trials, two drug candidates in preclinical studies, and one program undergoing lead optimization. In September 2006, we announced positive results for our most advanced drug candidate, STA-4783, in a Phase 2b clinical trial in patients with metastatic melanoma. Based on these positive results, we intend to initiate a pivotal Phase 3 clinical trial in metastatic melanoma and announce plans for initiating clinical trials in one or more additional cancer types in mid-2007. For our second clinical-stage drug candidate, apilimod, we are currently conducting a Phase 2a clinical trial in patients with rheumatoid arthritis and sponsoring a Phase 2a clinical trial in patients with CVID. Our two next most advanced drug candidates, STA-9090 and STA-9584, are currently in preclinical development, and our CRAC ion channel inhibitor program is currently in the lead optimization stage of discovery. All of our drug candidates were discovered and developed internally, using our unique chemical compound library, and the chemistry, biology, and pharmaceutical development assets and capabilities built over the combined history of Synta and its predecessor companies. We have retained all rights to all of our drug candidates and programs across all geographic markets and therapeutic indications.

We were incorporated in March 2000 and commenced operations in July 2001. Since that time, we have been principally engaged in the discovery and development of novel drug candidates. In September 2002, we acquired all of the outstanding stock of Principia Associates, Inc., an operating biopharmaceutical company and a related party, in exchange for our common stock, common stock warrants and forgiveness of notes receivable with an aggregate value of $16.9 million. In July 2002, Principia had acquired all of the outstanding stock of SBR Pharmaceuticals Corp. (formerly Shionogi BioResearch Corp.), an operating biopharmaceutical company, in exchange for cash of $12.5 million. In December 2002, we acquired all of the outstanding stock of Diagon Genetics, Inc., a related party, whose activities consisted of owning the rights to the development of certain intellectual property, in exchange for cash of $5.0 million and $8.5 million of our common stock. In January 2004, we acquired the assets, consisting principally of rights to intellectual property, and assumed certain liabilities of Cancer Genomics, Inc., Kava Pharmaceuticals, Inc. and SinglePixel Biomedical, Inc., collectively referred to herein as CKS, all related parties, in a single transaction in exchange for our common stock with a value of $2.2 million.

Since our inception, we have had no revenues from product sales and have funded our operations primarily through the private placement of our common stock and Series A convertible preferred stock. Through December 31, 2006, we raised net cash proceeds of $236.6 million through the private placement of common stock and Series A convertible preferred stock and the exercise of common stock options and warrants. In June 2006, we raised gross cash proceeds of $40.0 million through the private placement of our Series A convertible preferred stock.

In February 2007, we raised $50.0 million in gross proceeds from the sale of 5,000,000 shares of our common stock in an initial public offering at $10.00 per share. The net offering proceeds to us after deducting approximately $5.3 million in expenses for underwriters' discounts, fees and commissions, legal, accounting, printing and listing and filing fees, and miscellaneous expenses was approximately $44.7 million. Based on our current operating plans, we expect the proceeds of this

offering, together with our existing resources, to be sufficient to fund our planned operations, including our continued research and drug development, through at least mid-2008. All outstanding shares of the Series A convertible preferred stock and $1.9 million in accumulated dividends were converted into 6,278,765 shares of common stock upon the closing of our initial public offering. In accordance with EITF No. 98-5, *Accounting for Convertible Securities with Beneficial Conversion Features or Contingently Adjustable Conversion Ratios*, and EITF No. 00-27, *Application of Issue No. 98-5 to Certain Convertible Instruments,* we will record a non-cash beneficial conversion charge of approximately $58.6 million in the first quarter of 2007 in connection with the contingent adjustable conversion feature of the Series A convertible preferred stock.

We have devoted substantially all of our capital resources to the research and development of our drug candidates and to the acquisitions of Principia and Diagon. We have never been profitable and, as of December 31, 2006, we had an accumulated deficit of $236.6 million. We had net losses attributable to common stockholders of $59.1 million, $68.9 million and $45.9 million for the years ended December 31, 2006, 2005 and 2004, respectively. We expect to incur significant and increasing operating losses for the foreseeable future as we advance our drug candidates from discovery through preclinical and clinical trials and seek regulatory approval and eventual commercialization. In addition to these increasing research and development expenses, we expect general and administrative costs to increase as we add personnel and begin operating as a public company. We will need to generate significant revenues to achieve profitability and may never do so.

Financial Operations Overview

Revenue

We have not yet generated any product revenue and do not expect to generate any product revenue for the foreseeable future. We have recognized, in the aggregate, $1.5 million of revenue since our inception. This revenue was derived entirely from government research grants. We will seek to generate revenue from product sales, and possibly from collaborative or strategic relationships, which could include research and development, profit sharing, and milestone payments, as well as royalties. In the future, we expect that any revenue we generate will fluctuate from quarter-to-quarter as a result of the timing and amount of payments received under any future collaborative or strategic relationships, and the amount and timing of payments we receive upon the sale of our drug candidates, to the extent any is successfully commercialized.

Research and Development

Research and development expense consists of costs incurred in connection with developing and advancing our drug discovery technology and identifying and developing our drug candidates. From inception through December 31, 2006, we incurred research and development expense in the aggregate of $180.4 million. We charge all research and development expenses to operations as incurred.

Our research and development expense consists of:

- internal costs associated with research, preclinical and clinical activities;
- payments to third-party contract research organizations, investigative sites and consultants in connection with our preclinical and clinical development programs;
- costs associated with drug formulation and supply of drugs for clinical trials;
- personnel related expenses, including salaries, stock-based compensation, benefits and travel; and
- overhead expenses, including rent and maintenance of our facilities, and laboratory and other supplies.

We began tracking our internal and external research and development costs and our personnel and related costs on an individual drug candidate basis in 2003. For the periods indicated below, research and development expenses for our clinical-stage drug candidates, STA-4783 and apilimod, and our other early-stage and discontinued programs were as follows (in millions):

	Years ended December 31,		
	2006	2005	2004
STA-4783	$ 9.6	$ 14.0	$ 10.8
Apilimod	16.8	27.5	15.0
Early-stage and discontinued programs	24.1	18.4	12.3
Total	$ 50.5	$ 59.9	$ 38.1

We do not know if we will be successful in developing our drug candidates. While expenses associated with the completion of our current clinical programs are expected to be substantial and increase, we believe that accurately projecting total program-specific expenses through commercialization is not possible at this time. The timing and amount of these expenses will depend upon the costs associated with potential future clinical trials of our drug candidates, and the related expansion of our research and development organization, regulatory requirements, advancement of our preclinical programs and product manufacturing costs, many of which cannot be determined with accuracy at this time based on our stage of development. This is due to the numerous risks and uncertainties associated with the duration and cost of clinical trials, which vary significantly over the life of a project as a result of unanticipated events arising during clinical development, including with respect to:

- the number of clinical sites included in the trial;
- the length of time required to enroll suitable subjects;
- the number of subjects that ultimately participate in the trials; and
- the efficacy and safety results of our clinical trials and the number of additional required clinical trials.

Our expenditures are subject to additional uncertainties, including the terms and timing of regulatory approvals and the expense of filing, prosecuting, defending or enforcing any patent claims or other intellectual property rights. In addition, we may obtain unexpected or unfavorable results from our clinical trials. We may elect to discontinue, delay or modify clinical trials of some drug candidates or focus on others. A change in the outcome of any of the foregoing variables in the development of a drug candidate could mean a significant change in the costs and timing associated with the development of that drug candidate. For example, if the FDA or other regulatory authority were to require us to conduct clinical trials beyond those that we currently anticipate, or if we experience significant delays in any of our clinical trials, we would be required to expend significant additional financial resources and time on the completion of clinical development. Additionally, future commercial and regulatory factors beyond our control will evolve and therefore impact our clinical development programs and plans over time.

Despite this uncertainty, however, our development strategy for our lead clinical-stage drug candidate, STA-4783, is currently based on a number of assumptions that allow us to make broad estimates of certain clinical trial expenses. We expect to initiate a pivotal Phase 3 clinical trial of STA-4783 in metastatic melanoma in the middle of 2007, and we expect the cost to complete this trial, including the cost of clinical supplies of STA-4783, together with the costs of related nonclinical toxicology and other testing to support the trial, will be in the range of $40 to $60 million. To date, we have not entered into any collaboration with a strategic corporate partner for the development of this

drug candidate, and unless we do so in the future, we expect to internally finance all clinical development of this candidate. We do not expect to receive regulatory approval of any of our drug candidates until 2009 at the earliest, if at all.

Beyond our two lead drug candidates, we anticipate that we will select drug candidates and research projects for further development on an ongoing basis in response to their preclinical and clinical success, as well as commercial potential.

In-Process Research and Development

Our acquisitions of Principia and Diagon in 2002 and the CKS assets in 2004 resulted in in-process research and development charges to our consolidated statements of operations in the respective periods of the acquisitions. The total amount of in-process research and development charges related to these acquisitions was approximately $19.7 million. We used the income approach to estimate the fair value of in-process research and development for the Principia and Diagon acquisitions and the cost approach for the CKS acquisition. Generally, in cases where we acquired assets and assumed liabilities, and where the purchase price exceeded the fair value of net assets acquired, the excess purchase price has been allocated to acquired intangible assets, principally in-process research and development. If the in-process research and development acquired is incomplete and has no alternative future use, we record the value of the in-process research and development as an expense in our consolidated statement of operations in the period of the acquisition.

Under the income approach, each project was analyzed to determine the utilization of core technology; the complexity, cost and time to complete development; any alternative future use or current technological feasibility; and the stage of completion. Future cash flows were estimated, taking into account the expected life cycles of the product and the underlying technology, relevant market sizes and industry trends. The estimated net cash flows from these products were based on management's estimates of related revenues, cost of goods sold, research and development costs, selling, general and administrative costs, and income taxes. Material cash flows from each of the projects valued under the income approach were assumed to commence in the year following project completion. Discount rates and probability factors were determined based on the nature of the technology, the stage of completion of the projects, the complexity of the development effort and the risks associated with reaching technological feasibility of the projects.

We recorded an in-process research and development charge of $13.9 million as a result of the Principia acquisition, principally comprised of an $8.7 million charge related to STA-4783 and a $3.7 million charge related to apilimod. The discount rates applied in the valuations ranged from 30% to 40%.

Projects acquired in the Diagon acquisition related to ion channel technology and anti-allergy antibody projects and resulted in in-process research and development valuation of approximately $3.0 million and $1.2 million, respectively. The discount rate applied in the valuations was 30%.

The CKS in-process research and development charge of $1.6 million pertained to the technology related to the treatment of anxiety and general pain. The value of the CKS in-process research and development charge was based on the cost approach. During 2004, after an initial investment to advance the technology, we ceased further funding of the project.

We believe each of the acquired technologies for which in-process research and development was recorded was unique and patents were filed for each of the acquired projects. Completion of these projects will be a complex and costly undertaking, involving continuing research, animal studies and human clinical trials.

General and Administrative

General and administrative expense consists primarily of salaries and related expenses for personnel in executive, finance, business development, human resources and administrative functions. Other costs include stock-based compensation costs, legal costs of pursuing patent protection of our intellectual property, fees for general legal, accounting and other professional services, and overhead-related costs not otherwise included in research and development. We anticipate increases in general and administrative expense relating to public-company requirements and initiatives. These increases will likely include legal fees, accounting fees and directors' and officers' liability insurance premiums, as well as fees for investor relations services.

Convertible Preferred Stock Dividends

Convertible preferred stock dividends consists of cumulative but undeclared dividends payable on our Series A convertible preferred stock. The Series A convertible preferred stock accrued dividends at 8% per year. For the year ended December 31, 2006, dividends recorded with respect to the Series A convertible preferred stock totaled $1.9 million.

Critical Accounting Policies and Estimates

Our management's discussion and analysis of our financial condition and results of operations are based on our financial statements which have been prepared in accordance with U.S. generally accepted accounting principles, or GAAP. The preparation of these financial statements requires us to make estimates and judgments that affect the reported amounts of assets and liabilities and the disclosure of contingent assets and liabilities at the date of the financial statements and the reported amounts of revenues and expenses during the reported periods. We are required to make estimates and judgments with respect to accrued expenses, acquisitions and stock-based compensation. We base our estimates on historical experience, known trends and events, and various other factors that are believed to be reasonable under the circumstances, the results of which form the basis for making judgments about the carrying values of assets and liabilities that are not readily apparent from other sources and the reported amounts of revenues and expenses. Actual results may differ from these estimates under different assumptions or conditions.

We believe the following accounting policies and estimates are most critical to aid you in understanding and evaluating our reported financial results.

Accrued Expenses

As part of the process of preparing financial statements, we are required to estimate accrued expenses. This process involves identifying services which have been performed on our behalf, and estimating the level of service performed and the associated cost incurred for such service as of each balance sheet date in our financial statements. Given our current business, the primary area of uncertainty concerning accruals which could have a material effect on our business is with respect to service fees paid to contract manufacturers in conjunction with the production of clinical drug supplies and to contract research organizations in connection with our preclinical studies and clinical trials. In connection with all of the foregoing service fees, our estimates are most affected by our understanding of the status and timing of services provided. The majority of our service providers, including contract research organizations, invoice us in arrears for services performed. In the event that we do not identify some costs which have begun to be incurred, or we under or over estimate the level of services performed or the costs of such services in a given period, our reported expenses for such period would be too low or too high. We currently reflect the over or under accrual of expenses directly in our operations in the period the amount was determined.

Our arrangements with contract research organizations in connection with clinical trials often provide for payment prior to commencing the project or based upon predetermined milestones throughout the period during which services are expected to be performed. We recognize expense relating to these arrangements based on the various services provided over the estimated time to completion. The date on which services commence, the level of services performed on or before a given date, and the cost of such services are often determined based on subjective judgments. We make these judgments based upon the facts and circumstances known to us based on the terms of the contract or our ongoing monitoring of service performance. In the years ended December 31, 2006, 2005 and 2004, we had arrangements with multiple contract research organizations whereby these organizations commit to performing services for us over multiple reporting periods. We currently recognize and plan to continue to recognize the expenses associated with these arrangements based on our expectation of the timing of the performance of components under these arrangements by these organizations. Generally, these components consist of the costs of setting up the trial, monitoring the trial, closing the trial and preparing the resulting data.

With respect to financial reporting periods presented in this Annual Report on Form-10K, and based on our receipt of invoices from our third-party providers, the timing of our actual costs incurred have not differed materially from our estimated timing of such costs. In light of the foregoing, we do not believe our estimates of future expenses and our practice of making judgments concerning the accrual of expenses are reasonably likely to change in the future. There were no changes in our estimates and accruals for contract service fees that had a material effect on our net losses in the years ended December 31, 2006, 2005 and 2004, respectively.

Acquisitions

We apply purchase accounting in our acquisitions. Under purchase accounting, we allocate the purchase price to assets acquired and liabilities assumed based upon our analysis and estimates of fair values. Our analysis generally includes three approaches to estimate the value of acquired assets. The cost approach measures the value of an asset by quantifying the aggregate expenditures that would be required to replace the subject asset, given its future service capability. The market approach employs a comparative analysis of actual transactions in which similar assets have been transferred or in which businesses have been sold whose value is comprised largely of assets similar to the subject assets. The income approach is an estimation of the present value of the future monetary benefits expected to flow to the owner of the asset during its remaining useful life. We generally use the income approach to estimate the fair value of in-process research and development. We perform a discounted cash flow analysis, utilizing anticipated revenues, expenses and net cash flow forecasts related to the technology. Given the high risk associated with the development of new drugs, we adjust the probability of revenue and expense forecasts to reflect the risk of failing to advance through the clinical development and regulatory approval process based on the stage of development in the regulatory process. Such a valuation requires significant estimates and assumptions. We believe the fair value assigned to the in-process research and development reflected in our consolidated financial statements is based on reasonable assumptions. However, these assumptions may be incomplete or inaccurate, and unanticipated events and circumstances may occur. If the in-process research and development is incomplete and has no alternative future value, we record the full value of the in-process research and development as an expense in the period of the acquisition.

Stock-Based Compensation

Prior to January 1, 2006, we applied the intrinsic-value-based method of accounting prescribed by Accounting Principles Board, or APB, Opinion No. 25, *Accounting for Stock Issued to Employees,* or APB Opinion No. 25, and related interpretations, including Financial Accounting Standards Board, or FASB, Interpretation No. 44, *Accounting for Certain Transactions Involving Stock Compensation, an*

Interpretation of APB Opinion No. 25, in accounting for our employee stock options. Under this method, compensation expense is generally recorded on the date of grant only if the estimated fair value of the underlying stock exceeds the exercise price. Given the absence of an active market for our common stock, the board of directors historically has determined the estimated fair value of our common stock on the dates of grant. Historically, the determination was principally based on sales of common stock to outside investors, as well as progress against regulatory, clinical and product development milestones, and the likelihood of achieving a liquidity event such as an initial public offering or sale of the company. As a result, we recorded deferred compensation charges for the excess of the estimated fair value of our common stock over the exercise price of options granted at the date of grant. Compensation expense was recognized over the vesting period of the related options on a straight-line basis.

We account for stock options issued to non-employees in accordance with the provisions of Statement of Financial Accounting Standards, or SFAS, No. 123, *Accounting for Stock-Based Compensation,* or SFAS 123, and Emerging Issues Task Force, or EITF, No. 96-18, *Accounting for Equity Instruments that are Issued to Other than Employees, or in Conjunction with Selling Goods or Services,* which requires valuing and remeasuring such stock options to the current fair value until the performance date has been reached.

Effective January 1, 2006, we adopted SFAS No. 123R, *Share-Based Payment,* or SFAS 123R, for stock-based awards to employees, using the modified prospective method of transition for awards granted after January 17, 2005 (valued using the fair value method), and using the prospective method for awards granted prior to January 17, 2005 (valued using the minimum value method). Therefore, compensation cost recognized in the year ended December 31, 2006 includes: (1) compensation costs related to the vesting of stock options granted after January 17, 2005 but prior to January 1, 2006, based on the grant date fair value method estimated in accordance with the provisions of SFAS 123 adjusted for estimated forfeitures, (2) compensation costs related to the continued vesting of nonvested restricted stock awards granted prior to January 1, 2006, and (3) compensation costs for all share-based payments granted or modified subsequent to January 1, 2006, based on the provisions of SFAS 123R.

We continue to use the Black-Scholes option pricing model as the most appropriate valuation method for our option grants. The Black-Scholes model requires inputs for risk-free interest rate, dividend yield, volatility and expected lives of the options. Since we did not have a history of stock trading activity, expected volatility was based on historical data from several public companies similar in size and value to us. Now that our common stock is publicly traded, in future periods, we will use a weighted average approach using historical volatility and other similar public entity volatility information until historical volatility of our common stock is relevant to measure expected volatility for future option grants. We estimate the forfeiture rate based on historical data. Our options generally vest 25% after one year of service and quarterly over three years thereafter. Based on an analysis of historical forfeitures, we applied a forfeiture rate of 10% to all options that vest upon completion of the first year of service following the date of grant. The analysis will be re-evaluated at least annually and the forfeiture rate will be adjusted as necessary. The risk-free interest rate for periods within the contractual life of the option is based on the U.S. Treasury yield curve in effect at the time of the grant. The expected lives for options granted represents the period of time that options granted are expected to be outstanding. Since January 1, 2006, we have used the simplified method for determining the expected lives of options.

For awards with graded vesting, we allocate compensation costs under SFAS 123R on a straight-line basis over the requisite service period. Accordingly, we amortized the fair value of each option over each option's service period, which is generally the vesting period.

Our net loss for the year ended December 31, 2006 includes $4.8 million of compensation costs and no income tax benefit related to our stock-based compensation arrangements for employee and non-employee awards. As of December 31, 2006, the total amount of unrecognized stock-based compensation expense is $13.3 million and will be recognized over a weighted average period of 3.6 years.

Accounting for equity instruments granted or sold by us requires fair value estimates of the equity instrument granted or sold. If our estimates of the fair value of these equity instruments are too high or too low, our expenses may be over- or understated. We contemporaneously estimated the fair value of the equity instruments based upon consideration of factors which we deemed to be relevant at the time of each respective grant or issuance. These included, depending on the period, the purchase price of our common stock that was sold to investors in December 2003 and throughout 2004 and the impact of our first proposed initial public offering of common stock in 2005. These factors indicated that the deemed fair values of the common stock underlying the options granted to employees and board members during 2003 and 2004 was equivalent to the exercise price of the respective options, except for one grant of an option for 75,000 shares of common stock to a board member in May 2004 at an exercise price that was below the fair value of the common stock. The difference, or the intrinsic value, is being amortized as compensation expense over the vesting period of the stock options. In addition, these factors indicated that the issuance of 365,000 shares of restricted stock, the grant of stock options to purchase 42,250 shares of common stock in December 2004, and the issuance of 96,589 shares of restricted stock in the year ended December 31, 2005, were at sales and exercise prices below the fair value of the common stock and, accordingly, the difference is being amortized as compensation expense over the respective vesting periods.

In late 2005, following negative results in our Phase 2b clinical trial of apilimod in psoriasis and our Phase 2 clinical trial of STA-4783 in non-small cell lung cancer, and inconclusive results in our Phase 2 clinical trial of STA-4783 in soft tissue sarcoma, and without a recent sale of a significant number of shares of common stock that established a fair value, the board of directors evaluated the fair value of our common stock. The principal factors considered by the board of directors in its valuation were the negative impacts of the aforementioned trial results on the values of each of the respective programs and the corresponding effect on the overall enterprise value of the company. The board of directors also considered market factors, including: (1) downward trends in the biotechnology indices during 2005, (2) that many new biotechnology issues during 2005 priced below expectations or were withdrawn due to market conditions, and (3) that many post-money valuations for new biotechnology issues in 2005 were below those of 2004. The fact that we had cash reserves sufficient to fund approximately only one year of operations and would need additional financing, potentially under unfavorable terms, was also considered by the board. The board also considered positive factors including: (1) the initiation of a Phase 2b clinical trial of apilimod in Crohn's disease and a Phase 2a clinical trial of apilimod in rheumatoid arthritis, (2) the addition of a key scientific and medical expert to the board of directors, (3) advancement of our Hsp90 program to the preclinical stage and (4) significant advances in our CRAC ion channel program, each of which the board believed had a positive effect on the valuations of the respective programs and the overall enterprise value of the company. Based upon their review of both positive and negative factors, the board of directors made a determination that the fair value of our common stock as of mid-December 2005 was $14.00 per share, yielding an enterprise value, including cash, of approximately $342 million.

On February 15, 2006, we granted options to purchase 602,587 shares of our common stock at an exercise price of $14.00 per share, in connection with the annual compensation reviews for all employees. On March 1, 2006, the board of directors amended the exercise price of all outstanding options with exercise prices equal to or greater than $16.00 per share held by active employees, directors and consultants. Options to purchase an aggregate of 933,075 shares of common stock were repriced to $14.00 per share, resulting in incremental stock-based compensation of $720,000, of which $269,000 related to vested options and was expensed immediately and $451,000 related to unvested options and will be recognized as expense over the remaining vesting periods. In connection with these stock grants and the repricing, the board determined that the factors affecting the value of the common stock, taken as a whole, had not changed since December 2005, and accordingly, the board determined that $14.00 per share continued to be the fair value of our common stock.

In November 2006, in anticipation of our initial public offering and due to the substantial number of shares involved in the option grants in February 2006, and the repricing of options in March 2006, we performed a retrospective quantitative analysis of the fair value of our common stock for financial reporting purposes as of February 15, 2006, in order to reevaluate the appropriateness of the board of directors' fair value determination of $14.00 per share. Valuation methodologies employed in the analysis included an income approach under which we estimated our capacity to generate financial benefits for our shareholders, converting those projected benefits into a measure of present value, and a market approach under which we measured our value through an analysis of initial public offerings by 16 companies that were considered comparable to us. No allocation of the enterprise valuation to classes of stock was necessary as we only had common stock outstanding as of February 15, 2006. The valuation information considered by us in the income approach included the present value of our projected operating results on a going concern enterprise basis as determined by a probability-weighted, discounted cash flow analysis of our two most advanced drug candidates at the time, STA-4783 and apilimod, and our most advanced preclinical candidate, STA-9090. We assessed the risks associated with achieving our projections in selecting the probability factors and discount rate, which we believe to be reasonable and appropriate based upon our assumptions regarding market growth, estimated costs, and the likelihood and timing of FDA approval. The probability factors for achieving clinical trial success applied to the analysis for STA-4783, which at the time had not successfully completed a Phase 2b clinical trial, were 28% for melanoma and 15% for other potential cancer indications. The probability factor applied to the analysis for apilimod was 21%. The probability factors applied to the analysis for STA-9090, which was in the preclinical stage, were 5% for all potential indications taken as a whole. These probability rates were based upon information available from industry sources and our judgment of the relative likelihood of success in each program in light of the facts and circumstances available to us as of the valuation date. Our projections also considered ongoing research and development and selling and marketing expenses related to development of new compounds other than those for which we had developed specific projections.

The discount rate used in each probability-adjusted case was 19%, which was based on our development of the weighted average cost of capital utilizing market participant assumptions, and which correlated to the venture capital rates of return in the AICPA practice aid on *Valuation of Privately-Held-Company Equity Securities Issued as Compensation*. We weighted the results of the income approach 75% and the market approach 25%. The analysis also considered a discount for lack of marketability of 10%. This analysis resulted in a weighted average enterprise valuation range of approximately $328 million to $364 million, including cash. Based upon this valuation analysis and the resultant enterprise valuation range, we concluded that the board of directors' determination of the fair value per share of common stock of $14.00 per share, or an enterprise value of $342 million, as of the end of 2005 and as of February 2006 was appropriate.

In June 2006, we sold 8,000,000 shares of our Series A convertible preferred stock to investors for $5.00 per share. The Series A convertible preferred stock accrued dividends at 8% per year. The Series A convertible preferred stock had an initial conversion ratio of 1:1 (which was adjusted to 1:4 after giving effect to the one-for-four reverse stock split effected on February 2, 2007) and a conversion ratio upon an initial public offering of our common stock equal to the Series A purchase price of $5.00 per share plus an accumulated dividend of 8% per year (which was earned in arrears on the last day of each fiscal quarter with respect to the prior quarterly period but without prorating for any partially completed fiscal quarter) divided by the lesser of $20.00 or 66.6667% of the initial public offering price. There were no changes in our business, risks or market conditions during the period from February 15, 2006, the effective date of the retrospective valuation, until the date of our sale of the Series A convertible preferred stock, and, accordingly, there was no change attributed to value of the common stock. We believe the difference in the value of the common stock and the Series A convertible preferred stock purchase price reasonably reflected the rights and preferences of the Series A convertible preferred stock, including the 8% dividend and the value of the adjustable conversion

feature of the Series A convertible preferred stock upon the closing of an initial public offering of our common stock.

In June 2006, subsequent to the sale of the Series A convertible preferred stock, we received results from the interim analysis of our Phase 2b clinical trial of apilimod in patients with Crohn's disease, which indicated that it was unlikely the trial would meet its primary endpoint, and thus, the trial was terminated. In August, 2006, we received results from the Phase 2b clinical trial of STA-4783 in patients with metastatic melanoma, which indicated that the trial had achieved its primary endpoint and was well tolerated. We believe these two clinical events were offsetting events in the valuation of the company at the time and there were no other changes to the business, risks or market conditions in the period through December 31, 2006.

The following table summarizes the share-based awards issued to employees during the year ended December 31, 2006:

Month	Shares	Per Share Exercise Price	Per Share Fair Value	Per Share Intrinsic Value
January 2006	1,150	$ 14.00	$ 14.00	$ —
February 2006	677,587	14.00	14.00	—
March 2006	4,500	14.00	14.00	—
May 2006	17,400	14.00	14.00	—
June 2006	6,075	14.00	14.00	—
July 2006	9,625	14.00	14.00	—
August 2006	10,750	14.00	14.00	—
September 2006	6,625	14.00	14.00	—
October 2006	17,275	14.00	14.00	—
Total	750,987			

From October 17, 2006 through December 31, 2006, we did not grant any options, but we allocated 131,337 shares of common stock for options which were granted to new employees on February 6, 2007, the effective date of the registration statement in connection with our initial public offering, at the initial public offering price of $10.00 per share.

Consolidated Results of Operations

Year Ended December 31, 2006 Compared with Year Ended December 31, 2005

Revenue. There were no revenues for the years ended December 31, 2006 and 2005.

Research and development. Research and development expense decreased to $50.5 million in the year ended December 31, 2006 from $59.9 million in the year ended December 31, 2005. This decrease in research and development expense principally resulted from a decrease of $11.2 million for external costs of clinical trials, animal studies and other preclinical testing, clinical product manufacturing and consulting, principally due to the completion of several clinical trials in 2005 and in the first half of 2006, and $0.5 million of expense recorded in 2005 in connection with an agreement and release with our scientific founder. This was offset in part by an increase of $1.3 million for personnel costs and related research supplies and operational overhead due in part to a full year of costs associated with the expansion of one of our research and development facilities completed in 2005 and an increase in stock-based compensation expense of $1.0 million principally related to the net effect of the increased expense in connection with implementation of SFAS 123R less the impact of the conclusion of vesting of certain non-employee options in 2005.

General and administrative. General and administrative expense decreased to $8.6 million in the year ended December 31, 2006 from $11.3 million in the year ended December 31, 2005. The decrease in general and administrative expense was principally due to $2.4 million incurred in connection with the filing of a Registration Statement on Form S-1 with the SEC in 2005 relating to an initial public offering of our common stock. We determined that we would not complete the planned offering and withdrew the filing in June 2005. The related costs were expensed in the year ended December 31, 2005 as we did not reactivate and complete the offering within 90 days of the withdrawal of the filing. This decrease was also due to a decrease of $0.6 million for personnel costs and related overhead due principally to decreased headcount and a decrease of $0.3 million in external professional fees, principally for general legal and other consulting services, offset by an increase in stock-based compensation of $0.6 million principally related to the net effect of the increased expense in connection with implementation of SFAS 123R less the impact of the conclusion of vesting of certain non-employee options in 2005.

Investment income, net. Net investment income decreased to $1.9 million in the year ended December 31, 2006 from $2.3 million in the year ended December 31, 2005. The decrease in investment income was principally due to a decrease in average cash balances as a result of the use of existing cash resources during 2005 and 2006, prior to the net cash proceeds of $40.0 million raised from the sale of our Series A convertible preferred stock in June 2006.

Convertible preferred stock dividends. Series A convertible preferred stock dividends were $1.9 million for the year ended December 31, 2006 due to the issuance of the Series A convertible preferred stock in June 2006. The Series A convertible preferred stock dividends accrued at the rate of 8% per year.

Net loss Net loss for the year ended December 31, 2006 decreased to $57.3 million from $68.9 million for the year ended December 31, 2005. Basic and diluted net loss per share attributable to common stockholders decreased to $2.66 for the year ended December 31, 2006 from $3.09 for the year ended December 31, 2005. The decrease was principally due to the completion of several clinical trials in 2005 and in the first half of 2006.

Year Ended December 31, 2005 Compared with Year Ended December 31, 2004

Revenue. There were no revenues in the year ended December 31, 2005, compared to research grant revenues of $0.2 million in the year ended December 31, 2004. This was due to the fact that research services were concluded during 2004.

Research and development. Research and development expense increased to $59.9 million for the year ended December 31, 2005 from $38.1 million for the year ended December 31, 2004. This increase principally resulted from increases of $10.9 million for external costs of clinical trials, animal studies and other pre-clinical testing, clinical product manufacturing and consulting due principally to several clinical trials commencing in 2004 and continuing in 2005, $9.2 million for personnel costs and related research supplies and operational overhead due principally to an increase in research and development headcount, and $1.2 million in stock-based compensation expense principally resulting from the issuance of restricted stock in 2004. In addition, we recorded $0.5 million in expense in 2005 in connection with an agreement and release with our scientific founder.

In-process research and development. In-process research and development expense of $1.6 million for the year ended December 31, 2004 represents the expensing of the value of incomplete research and development acquired in connection with the purchase of the CKS assets in January 2004.

General and administrative. General and administrative expense increased to $11.3 million for the year ended December 31, 2005 from $7.4 million for the year ended December 31, 2004. The increase

was principally the result of $2.4 million incurred in connection with the filing of a Registration Statement on Form S-1 with the SEC in 2005 relating to an initial public offering of our common stock. We determined that we would not complete the planned offering and withdrew the filing in June 2005. The related costs were expensed in 2005 as we did not reactivate and complete the offering within 90 days of the withdrawal of the filing. The increase was also the result of $1.1 million for personnel costs and related overhead due to increased hiring and a net increase of $0.4 million in stock-based compensation principally resulting from the issuance of restricted stock in 2004 and 2005.

Investment income, net. Net investment income increased to $2.3 million for the year ended December 31, 2005 from $1.0 million for the year ended December 31, 2004. The increase in net investment income in each year was principally due to increases in the average cash balances invested resulting from sales of our common stock.

Net loss Net loss for the year ended December 31, 2005 increased to $68.9 million from $45.9 million for the year ended December 31, 2004. Basic and diluted net loss per share attributable to common stockholders increased to $3.09 for the year ended December 31, 2005 from $2.46 for the year ended December 31, 2004. The increase was principally due to the volume of several clinical trials that commenced in 2004 and early 2005, and the related manufacturing, packaging and distribution costs of drug supply.

Liquidity and Capital Resources

Sources of Liquidity

We have incurred significant operating losses since our inception. We have funded our operations principally with $195.4 million in net proceeds from private placements of our common stock and $40.0 million in gross proceeds from a private placement of our Series A convertible preferred stock, which together with the exercise of common stock warrants and options, provided aggregate net cash proceeds of approximately $236.6 million through December 31, 2006. We have also generated funds from government grant revenues, equipment lease financings and investment income.

As of December 31, 2006, we had cash, cash equivalents and marketable securities of $46.8 million, consisting of cash and highly liquid, short-term investments, which is a decrease of $15.2 million from $62.1 million as of December 31, 2005. The decrease principally reflects the $40.0 million in gross proceeds from the private placement of our Series A convertible preferred stock in June 2006, offset by the net loss of $57.3 million. Our funds are currently invested in investment grade and U.S. government securities with an average duration of less than one year.

In February 2007, we raised $50.0 million in gross proceeds from the sale of 5,000,000 shares of our common stock in an initial public offering at $10.00 per share. The net offering proceeds to us after deducting approximately $5.3 million in expenses for underwriters' discounts, fees and commissions, legal, accounting, printing and listing and filing fees, and miscellaneous expenses were approximately $44.7 million. Based on our current operating plans, we expect the proceeds of this offering, together with our existing resources, to be sufficient to fund our planned operations, including our continued research and drug development, through at least mid-2008.

In November 2004, we entered into an agreement for a revolving property and equipment lease line of credit, which was amended in 2005 and extended in 2006. Under the agreement, we may periodically directly lease, or sell and lease back up to $6.0 million of equipment and leasehold improvements through March 2007. Amounts borrowed under the facility are repayable over 36 or 48 months. As of December 31, 2006, approximately $1.2 million was available under the lease line for future property and equipment expenditures.

Cash Flows

The following table provides information regarding our cash flows and our capital expenditures for the years ended December 31, 2006, 2005 and 2004 (in thousands).

	Years ended December 31,		
	2006	2005	2004
Cash provided by (used in):			
Operating activities	$ (52,985)	$ (61,882)	$ (33,795)
Investing activities	23,574	39,176	(43,811)
Financing activities	39,289	3,779	84,280
Capital expenditures (included in investing activities above)	(1,580)	(4,883)	(1,594)

Our operating activities used cash of $53.0 million, $61.9 million and $33.8 million in the years ended December 31, 2006, 2005 and 2004, respectively. The use of cash in all periods principally resulted from our losses from operations and changes in our working capital accounts. The increase in cash used in operations in each of the periods through December 31, 2006 was due to our continued focus on research and development activities and the related expansion of our organizational infrastructure to support our broadened development activities.

Our investing activities provided cash of $23.6 million and $39.2 million, and used cash of $43.8 million in the years ended December 31, 2006, 2005 and 2004, respectively. Our investing activities in 2006 included sales and maturities of marketable securities in our investment portfolio in the amount of $143.4 million, offset by the purchases of marketable securities in the amount of $118.2 million and purchases of property and equipment in the amount of $1.6 million. Our investing activities in 2005 included sales and maturities of marketable securities in our investment portfolio in the amount of $228.4 million, offset by the purchases of marketable securities in the amount of $184.4 million and purchases of property and equipment in the amount of $4.9 million, including a research and development expansion of one of our facilities. Our investing activities in 2004 included purchases of marketable securities in the amount of $124.7 million and purchases of property and equipment in the amount of $1.6 million, offset by the sales and maturities of marketable securities in our investment portfolio in the amount of $82.5 million.

Our financing activities provided $39.3 million, $3.8 million and $84.3 million in the years ended December 31, 2006, 2005 and 2004, respectively. Our financing activities since inception through December 31, 2006 consisted principally of the sale of our common stock and our Series A convertible preferred stock to private investors and the exercise of stock options and warrants providing net proceeds of $236.6 million, and the sale and lease-back of equipment providing proceeds of $7.5 million, less the repayment of $3.4 million of our capital equipment leases. In June 2006, we raised gross proceeds of $40.0 million from the sale of 8,000,000 shares of our Series A convertible preferred stock. In November 2004, we raised net proceeds of $79.9 million from the sale of 3,999,997 shares of our common stock. We raised $1.4 million, $4.7 million and $1.3 million in proceeds from the sale and lease-back of property and equipment in the years ended December 31, 2006, 2005 and 2004, respectively. We repaid $2.1 million, $1.1 million and $0.2 million in capital equipment leases in the years ended December 31, 2006, 2005 and 2004, respectively.

Contractual Obligations and Commitments

The following tables summarize our contractual obligations at December 31, 2006 and the effects such obligations are expected to have on our liquidity and cash flows in future periods (in thousands).

Contractual Obligations	Total	2007	2008 through 2009	2010 through 2011	More than 5 years
Capital lease obligations(1)	$ 6,256	$ 2,774	$ 3,304	$ 178	$ —
Operating lease obligations	5,734	2,079	1,939	1,716	—
Research and development contracts	1,477	1,445	28	4	—
Consulting	367	167	200	—	—
Total	$ 13,834	$ 6,465	$ 5,471	$ 1,898	—

(1) Including scheduled interest payments.

Research and development contracts principally include contracts for human clinical studies, animal studies and clinical manufacturing. The future research and development contract obligations in the table of Contractual Obligations above assume that each of the studies and related manufacturing contracts is completed as planned. In the event a study or manufacturing contract is terminated prior to planned completion by mutual agreement between the contractor and us, the amount paid under such contracts may be less than the amounts presented.

Under various license agreements, substantially all of which are related to our early-stage discovery programs, we may be obligated to pay up to an aggregate of $3.9 million if specified development and commercialization milestones are met, as follows (in thousands). These amounts are not included in the table of Contractual Obligations above.

Milestone	Amount
Phase 1 clinical trials	$ 150
Phase 2 clinical trials	250
Phase 3 clinical trials	350
Completion of Phase 3 clinical trials	75
FDA new drug approval	1,875
European market approval	500
Other	650
Total	$ 3,850

In November 2004, we entered into an agreement for a revolving property and equipment lease line of credit, which was amended in 2005 and extended in 2006. Under the agreement, we may periodically directly lease, or sell and lease back up to $6.0 million of equipment and leasehold improvements through March 2007, with repayment periods of 36 or 48 months and a $1.00 purchase option at the end of each lease period. In the years ended December 31, 2006, 2005 and 2004 we sold and leased back under this agreement an aggregate of approximately $7.5 million of our previously purchased equipment and leasehold improvements, of which approximately $2.8 million and $4.7 million were capitalized and will be paid over 36 and 48 month periods, respectively.

Funding Requirements

We expect to incur substantial expenses and generate significant operating losses as we continue to advance our drug candidates into preclinical studies and clinical trials and as we:

- initiate a pivotal Phase 3 clinical trial of STA-4783 for the treatment of metastatic melanoma and announce plans for initiating clinical trials in one or more additional cancer types in mid-2007;
- begin to establish sales and marketing functions and commercial manufacturing arrangements for STA-4783;
- complete the current Phase 2a clinical trials of apilimod for the treatment of rheumatoid arthritis and CVID, and possibly initiate Phase 2 clinical trials of apilimod in additional inflammatory disease indications;
- initiate additional Phase 3 clinical trials of STA-4783 and one or more Phase 3 clinical trials of apilimod, if supported by Phase 2 results;
- complete preclinical development of STA-9090 and initiate clinical trials, if supported by positive preclinical data;
- complete preclinical development of STA-9584 and initiate clinical trials, if supported by positive preclinical data;
- advance our preclinical CRAC ion channel inhibitor program into clinical trials, if supported by positive preclinical data;
- discover, develop, and seek regulatory approval for backups of our current drug candidates and other new drug candidates;
- identify additional compounds or drug candidates and acquire rights from third parties to those compounds or drug candidates through licenses, acquisition or other means;
- commercialize any approved drug candidates;
- hire additional clinical, scientific, and management personnel; and
- add operational, financial, and management information systems and personnel.

Our funding requirements will depend on a number of factors, including:

- the progress of our research and development programs, including the completion of preclinical and clinical trials for our current drug candidates and the results from these studies and trials;
- the number of drug candidates we advance into later-stage clinical trials and the scope of our research and development programs;
- our ability to discover additional drug candidates using our drug discovery technology and advance them into clinical development;
- the costs involved in preparing, filing, prosecuting, maintaining and enforcing patent claims for our drug discovery technology and drug candidates and avoiding infringing the intellectual property of others;
- the time and costs involved in obtaining regulatory approvals for our drug candidates;
- our ability to establish and maintain collaborative arrangements;
- the potential in-licensing of other products or technologies or the acquisition of complementary businesses;

- the cost of manufacturing, marketing and sales activities, if any; and
- the timing, receipt and amount of revenue, if any, from our drug candidates.

We do not anticipate that we will generate product revenue for the next several years. We expect our continuing operating losses to result in increases in cash used in operations over the next several years. Our future capital requirements will depend on a number of factors including the progress and results of our clinical trials, the costs, timing and outcome of regulatory review of our drug candidates, our revenue, if any, from successful development and commercialization of our products, the costs of commercialization activities, the scope, progress, results and costs of preclinical development, laboratory testing and clinical trials for other drug candidates, the emergence of competing therapies and other market developments, the costs of preparing, filing and prosecuting patent applications and maintaining, enforcing and defending intellectual property rights, the extent to which we acquire or invest in other drugs and technologies, and our ability to establish collaborations and obtain milestone, royalty or other payments from any collaborators.

We may require significant additional funds earlier than we currently expect to conduct additional clinical trials and seek regulatory approval of our drug candidates. Because of the numerous risks and uncertainties associated with the development and commercialization of our drug candidates, we are unable to estimate the amounts of increased capital outlays and operating expenditures associated with our current and anticipated clinical trials.

Additional funding may not be available to us on acceptable terms or at all. In addition, the terms of any financing may adversely affect the holdings or the rights of our stockholders. For example, if we raise additional funds by issuing equity securities or by selling debt securities, if convertible, further dilution to our existing stockholders may result. To the extent our capital resources are insufficient to meet our future capital requirements, we will need to finance our future cash needs through public or private equity offerings, collaboration agreements, debt financings or licensing arrangements.

If adequate funds are not available, we may be required to terminate, significantly modify or delay our research and development programs, reduce our planned commercialization efforts, or obtain funds through collaborators that may require us to relinquish rights to our technologies or drug candidates that we might otherwise seek to develop or commercialize independently. We may elect to raise additional funds even before we need them if the conditions for raising capital are favorable.

Off-Balance Sheet Arrangements

We do not have any off-balance sheet arrangements or relationships with unconsolidated entities of financial partnerships, such as entities often referred to as structured finance or special purpose entities.

Tax Loss Carryforwards

In 2005 and in 2007, we performed analyses to determine if there were changes in ownership, as defined by Section 382 of the Internal Revenue Code, that would limit our ability to utilize certain net operating loss and tax credit carryforwards. We determined that we experienced a change in ownership, as defined by Section 382, in connection with the acquisition of Principia on September 20, 2002, but did not experience a change in ownership upon the effectiveness of our initial public offering. As a result, the utilization of our federal tax net operating loss carryforwards generated prior to the ownership change is limited. As of December 31, 2006 we have net operating loss carryforwards for U.S. federal tax purposes of approximately $201.4 million, after taking into consideration net operating losses expected to expire unused as a result of this limitation, and the remainder will expire in varying amounts through 2026 unless utilized. In addition, as of December 31, 2006, we have state net operating loss carryforwards of approximately $186.1 million, which will expire through 2010 unless

utilized. The utilization of these net operating loss carryforwards may be further limited as we experience future ownership changes as defined in Section 382 of the Internal Revenue Code, including changes resulting from the issuance of common stock in this offering.

Recently Issued Accounting Pronouncements

In May 2005, the FASB issued SFAS No. 154, *Accounting Changes and Error Correction*, or SFAS No. 154. SFAS No. 154 is a replacement of APB Opinion No. 20, *Accounting Changes*, or APB Opinion No. 20, and SFAS No. 3, *Reporting Accounting Changes in Interim Financial Statements.* This statement applies to all voluntary changes in accounting principle, and changes the accounting for, and reporting of, a change in accounting principle. SFAS No. 154 requires retrospective application to prior periods' financial statements of a voluntary change in accounting principle unless it is impracticable to do so. APB Opinion No. 20 previously required that most voluntary changes in accounting principle be recognized by including in net income of the period of the change the cumulative effect of changing to the new accounting principle. SFAS No. 154 carries forward many provisions of APB Opinion No. 20 without change, including the provisions related to the reporting of a change in accounting, a change in the reporting entity, and the correction of an error. SFAS No. 154 does not change the transition provisions of any existing account pronouncements, including those that are in a transition phase as of the effective date of the statement. We adopted the provisions of SFAS No. 154 on January 1, 2006, and the adoption of the new standard did not have a material impact on our consolidated financial position or consolidated statement of operations.

In June 2005, the FASB issued FSP 150-5. The FSP clarifies that freestanding warrants and similar instruments on shares that are redeemable should be accounted for as liabilities under SFAS No. 150, regardless of the timing of the redemption feature or price, even though the underlying shares may be classified as permanent or temporary equity. The FSP was effective for the first reporting period beginning after June 30, 2005. We adopted FSP 150-5 in 2006 and the impact was not material to our consolidated financial position or consolidated statement of operations.

In July 2006, the FASB issued FASB Interpretation No. 48, *Accounting for Uncertainty in Income Taxes—an interpretation of FAS 109*, (Interpretation No. 48). This interpretation clarifies the accounting for uncertainty in income taxes recognized in a company's financial statements in accordance with FASB Statement No. 109, *Accounting for Income Taxes.* This interpretation prescribes a recognition threshold and measurement attribute for the financial statement recognition and measurement of a tax position taken in a tax return. It also provides guidance on derecognition, classification, interest and penalties, accounting in interim periods, disclosure and transition. Interpretation No. 48 is effective for fiscal years beginning after December 15, 2006. Earlier application is encouraged if a company has not yet issued financial statements, including interim financial statements, in the period that Interpretation No. 48 is adopted. We are currently evaluating the impact the adoption of this interpretation will have on our consolidated results of operations and financial position.

In September 2006, the FASB issued SFAS No. 157, *Fair Value Measurements* (SFAS No. 157), which provides guidance for using fair value to measure assets and liabilities. The pronouncement clarifies (1) the extent to which companies measure assets and liabilities at fair value; (2) the information used to measure fair value; and (3) the effect that fair value measurements have on earnings. SFAS No. 157 will apply whenever another standard requires (or permits) assets or liabilities to be measured at fair value. SFAS No. 157 will be applicable to us as of January 1, 2008. We do not believe the adoption of SFAS No. 157 will have a material impact on our overall financial position or results of operations.

In September 2006, the U.S. Securities and Exchange Commission issued Staff Accounting Bulletin No. 108, *Considering the Effects of Prior Year Misstatements when Quantifying Misstatements in Current Year Financial Statements* (SAB No. 108). SAB No. 108 provides guidance on the consideration of the

effects of prior year misstatements in quantifying current year misstatements in determining whether the current year's financial statements are materially misstated. SAB No. 108 is effective for fiscal years ending after November 15, 2006. We have adopted SAB No. 108 as of December 31, 2006 and its adoption did not have a material impact on our consolidated financial statements.

In February 2007, the FASB issued SFAS No. 159, *The Fair Value Option for Financial Assets and Financial Liabilities,* including an amendment of SFAS No. 115, which permits entities to choose to measure many financial instruments and certain other items at fair value that are not currently required to be measured at fair value. SFAS No. 159 is effective for the Company beginning in fiscal 2009. The Company is currently evaluating SFAS No. 159 and the impact that it may have on its results of operations or financial position.

Certain Factors That May Affect Future Results of Operations

The Securities and Exchange Commission encourages companies to disclose forward-looking information so that investors can better understand a company's future prospects and make informed investment decisions. This Annual Report on Form 10-K contains such "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995.

Words such as "may," "anticipate," "estimate," "expects," "projects," "intends," "plans," "believes" and words and terms of similar substance used in connection with any discussion of future operating or financial performance, identify forward-looking statements. All forward-looking statements are management's present expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those described in the forward-looking statements. These risks include, but are not limited to those set forth under the heading "Risk Factors" contained in Item 1A of this Annual Report on Form 10-K.

In light of these assumptions, risks and uncertainties, the results and events discussed in the forward-looking statements contained in this Annual Report on Form 10-K or in any document incorporated by reference might not occur. Stockholders are cautioned not to place undue reliance on the forward-looking statements, which speak only as of the date of this Annual Report on Form 10-K. We are not under any obligation, and we expressly disclaim any obligation, to update or alter any forward-looking statements, whether as a result of new information, future events or otherwise. All subsequent forward-looking statements attributable to Synta or to any person acting on its behalf are expressly qualified in their entirety by the cautionary statements contained or referred to in this section.

Item 7A. QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK

We are exposed to market risk related to changes in interest rates. As of December 31, 2006, we had cash, cash equivalents and marketable securities of $46.8 million consisting of cash and highly liquid, short-term investments. Our cash is deposited in and invested through highly rated financial institutions in North America. Our marketable securities are subject to interest rate risk and will fall in value if market interest rates increase. If market interest rates were to increase immediately and uniformly by 10% from levels at December 31, 2006, we estimate that the fair value of our investments will decline by an immaterial amount, and therefore, our exposure to interest rate changes is not significant.

Item 8. FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA

The information required by this Item 8 is included at the end of this Annual Report on Form 10-K beginning on page F-1.

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

Not applicable.

Item 9A. CONTROLS AND PROCEDURES

(a) *Evaluation of Disclosure Controls and Procedures.* Our principal executive officer and principal financial officer, after evaluating the effectiveness of our disclosure controls and procedures (as defined in Rules 13a-15(e) and 15d-15(e) under the Securities Exchange Act of 1934, as amended) as of the end of the period covered by this Annual Report on Form 10-K, have concluded that, based on such evaluation, our disclosure controls and procedures were adequate and effective. In designing and evaluating our disclosure controls and procedures, our management recognized that any controls and procedures, no matter how well designed and operated, can provide only reasonable assurance of achieving the desired control objectives, and our management necessarily was required to apply its judgment in evaluating the cost-benefit relationship of possible controls and procedures.

(b) *Changes in Internal Controls.* There were no changes in our internal control over financial reporting, identified in connection with the evaluation of such internal control that occurred during the fourth quarter of our last fiscal year, that have materially affected, or are reasonably likely to materially affect, our internal control over financial reporting.

Item 9B. OTHER INFORMATION

Not applicable.

PART III

Item 10. DIRECTORS AND EXECUTIVE OFFICERS OF THE REGISTRANT

Executive Officers and Directors

The following table sets forth certain information concerning our executive officers and directors as of March 15, 2007:

Name	Age	Position
Executive Officer		
Safi R. Bahcall, Ph.D.	38	President and Chief Executive Officer and Director
James G. Barsoum, Ph.D.	50	Senior Vice President, Research
Jeremy G. Chadwick, Ph.D.	44	Senior Vice President, Program Management and Clinical Operations
Keith S. Ehrlich, C.P.A.	56	Vice President, Finance and Administration, Chief Financial Officer
Eric W. Jacobson, M.D.	50	Senior Vice President, Clinical Research and Regulatory Affairs, Chief Medical Officer
Keizo Koya, Ph.D.	49	Senior Vice President, Drug Development
Wendy E. Rieder, Esq.	39	Vice President, Intellectual Property and Legal Affairs, General Counsel
Martin D. Williams	42	Senior Vice President, Commercial and Business Development, Chief Business Officer
Non-Employee Directors		
Keith R. Gollust(1)(2)(3)	61	Chairman of the Board of Directors
Lan Bo Chen, Ph.D.	63	Director
Judah Folkman, M.D.	74	Director
Bruce Kovner(2)(3)	62	Director
William S. Reardon, C.P.A.(1)	60	Director
Robert N. Wilson(1)(2)(3)	66	Director

(1) Member of our audit committee. Mr. Reardon is the chairman of the committee.

(2) Member of our compensation committee. Mr. Wilson is the chairman of the committee.

(3) Member of our nominating and governance committee. Mr. Gollust is the chairman of the committee.

Safi R. Bahcall, Ph.D. co-founded Synta with Dr. Lan Bo Chen and has been our Chief Executive Officer and a member of our board of directors since July 2001. Dr. Bahcall has served as our President since December 2003. From 1998 to 2001, Dr. Bahcall was a consultant at McKinsey & Company, a management consulting firm, serving investment banks and pharmaceutical companies on key issues of strategy, technology, and operations. Dr. Bahcall also co-founded a drug discovery company focused on novel ion channel research in November 2001, which was acquired by Synta in December 2002. He received his B.A. *summa cum laude* from Harvard University, was awarded his Ph.D. from Stanford University in theoretical physics, and was a Miller postdoctoral research fellow at the University of California, Berkeley.

James G. Barsoum, Ph.D. has served as our Senior Vice President, Research since October 2006. He served as our Vice President, Biology from February 2003 to September 2006. From February 1987 to February 2003, Dr. Barsoum held various leadership roles at Biogen, Inc., now Biogen Idec Inc., a publicly traded biopharmaceutical company, most recently as the Director of Molecular and Cellular Biology. From January 1984 to January 1987, Dr. Barsoum held research fellowships at Stanford University and the Whitehead Institute for Biomedical Research. Dr. Barsoum received a Ph.D. in Biology from the Massachusetts Institute of Technology.

Jeremy G. Chadwick, Ph.D. has served as our Senior Vice President, Program Management and Clinical Operations since October 2006. He served as our Vice President, Program Management and Clinical Operations from May 2004 to September 2006. From January 2002 to May 2004, Dr. Chadwick served as Vice President, Development Operations at Vertex Pharmaceuticals, Inc., a publicly traded biopharmaceutical company. From December 1995 to September 1998, Dr. Chadwick held various positions at Parexel International, a publicly traded pharmaceutical services company, most recently as Vice President, U.S. Biostatistics and Data Management. From September 1985 to October 1995, Dr. Chadwick held various positions at Glaxo Group Research, most recently as Senior Manager, Medical Data Sciences Division. From September 1998 to October 2001, Dr. Chadwick was the Chief Operating Officer at Foliage Software Systems, a privately held software development company. Dr. Chadwick obtained both his Masters and Ph.D. in statistics from the University of London, U.K.

Keith S. Ehrlich, C.P.A. has served as our Chief Financial Officer since October 2006 and as our Vice President, Finance and Administration since March 2004. From November 2003 to February 2004, Mr. Ehrlich served as a financial consultant to us. From September 1999 to April 2003, Mr. Ehrlich was Vice President, Finance and Administration and Chief Financial Officer and Treasurer at Argentys Corporation, a private software development company. From January 1998 to July 1999, Mr. Ehrlich served as Senior Vice President, Finance and Administration, Chief Financial Officer and Treasurer of Dyax Corp., a publicly traded biopharmaceutical company. From October 1993 to January 1998, he served as Vice President, Finance and Administration and Chief Financial Officer and Treasurer of Oravax, Inc., a publicly traded biopharmaceutical company since acquired by Peptide Therapeutics Group. From May 1991 to October 1993, he served as Treasurer and Director of Finance of Vertex Pharmaceuticals, Inc., a publicly traded biopharmaceutical company. From January 1980 to April 1991, Mr. Ehrlich was an auditor with Coopers & Lybrand LLP. Mr. Ehrlich received his B.A. in Biology from Drew University and his M.B.A. in Finance and Accounting from Rutgers University.

Eric W. Jacobson, M.D. has served as our Senior Vice President, Clinical Research and Regulatory Affairs since October 2006 and as our Chief Medical Officer since January 2006. He served as our Vice President, Medical Research from April 2005 to December 2005. From January 2002 until April 2005, Dr. Jacobson held positions of increasing responsibility at Millennium Pharmaceuticals, Inc., a publicly traded biopharmaceutical company, most recently serving as Senior Director, Clinical Research and previously as Director, Clinical Research. From June 2000 until January 2002, Dr. Jacobson was the U.S. Medical Director, New Clinical Therapies for Serono Laboratories, Inc., a publicly traded biotechnology company. Dr. Jacobson was employed as an Academic Rheumatologist at the University of Massachusetts Medical Center from April 1991 until June 2000. From 1998 through 2000, he was also a consultant for the Center for Clinical and Lifestyle Research assisting with study design, data interpretation, report generation and journal publication. From July 1993 through June 1995, Dr. Jacobson was Adjunct Faculty at Northeastern University in their Physician Assistant Program, and previous to this Dr. Jacobson was a Rheumatologist at the North Carolina Arthritis and Allergy Care Center from July 1989 until April 1991. Dr. Jacobson received his B.S. at the University of Illinois at Champaign/Urbana and his M.D. at Rush Medical College of Rush University. Dr. Jacobson has had numerous academic appointments and has published over 25 abstracts, papers and book chapters.

Keizo Koya, Ph.D. has served as our Senior Vice President, Drug Development since September 2002. From September 1997 to August 2002, Dr. Koya worked for Shionogi BioResearch Corp. as Vice President, Research and Development. From April 1995 to August 1997, Dr. Koya was the Director, Drug Discovery and Development at Fuji ImmunoPharmaceuticals Corp., now EMD Lexigen Research Center Corp., a biopharmaceutical company. From October 1990 to March 1995 he was employed by Fuji Photo Film Co., Ltd., a global imaging and information company, where he was most recently the Head of Pharmaceutical R&D, U.S. Representative Office. He earned his Ph.D. in organic chemistry at Kyushu University.

Wendy E. Rieder, Esq. has served as our General Counsel since October 2006 and as our Vice President, Intellectual Property and Legal Affairs since December 2002. In August 1998, Ms. Rieder co-founded Microbiotix, Inc., a privately held biotechnology company developing small- molecule anti-infectives, and served as its Chief Operating Officer and Vice President, Business Development and Intellectual Property from January 2000 to December 2002. From August 1997 to December 1999 Ms. Rieder served as the Vice President, Business Development and Intellectual Property at LipoGenics, Inc., a subsidiary of a publicly traded biopharmaceutical company. Ms. Rieder was a patent attorney at Boehringer Ingelheim Pharmaceuticals, a U.S. affiliate of Boehringer Ingelheim GmbH, a global pharmaceutical company, from August 1995 to July 1997, and a patent agent at Fish & Neave LLP from January 1991 to July 1995. Ms. Rieder received an M.S. in organic chemistry from Columbia University and a J.D. from Fordham Law School.

Martin D. Williams has served as our Senior Vice President, Commercial and Business Development since October 2006 and as our Vice President, Commercial and Business Development, Chief Business Officer since February 2006. From December 2004 until December 2005, Mr. Williams was Head of Corporate Development for Altus Pharmaceuticals Inc., a publicly traded biopharmaceutical company. From July 2001 to June 2004, Mr. Williams was Senior Vice President, Corporate Development and Marketing at Oscient Pharmaceuticals Corporation, a publicly traded biopharmaceutical company. From November 1999 to March 2001, Mr. Williams was President and Chief Executive Officer of U.S. Marketer, Inc., an information technology and software company. From 1987 to 1993, he held various sales and sales management positions with Glaxo Laboratories (now GlaxoSmithKline, Inc.). From 1993 to 1995, Mr. Williams was international marketing director of anti-infectives for Lederle Laboratories/Wyeth. From 1995 to 1996, Mr. Williams was Group Director, Metabolic Products Business Development & Strategic Planning at Hoffman-La Roche and from 1997 to 1999, he was Vice President, Business Development, Sales & Marketing at Pentose Pharmaceuticals. Mr. Williams holds an M.B.A. from Harvard Business School, an M.S. from the University of Manchester, England, and a B.A. in biology from the University of Humberside, Hull, England.

Keith R. Gollust has been a member of our board of directors since July 2002 and has been our Chairman since September 2002. Mr. Gollust is a private investor and founded Gollust, Tierney, and Oliver, a private investment firm, in 1978. Mr. Gollust also was a Managing Director of Caxton Associates, L.L.C., a hedge fund firm, from July 2003 through December 2004. Mr. Gollust received a B.A. from Princeton University and an MSIA from Carnegie Mellon University.

Lan Bo Chen, Ph.D. co-founded Synta with Dr. Safi Bahcall and has been a member of our board of directors since July 2001, and a member of our scientific advisory board and its Chairman since July 2001. Dr. Chen is a Professor of Pathology, Emeritus, at Harvard Medical School. He has been at the Dana-Farber Cancer Institute and Harvard Medical School since July 1977. Dr. Chen is the founder of several biotechnology companies, including Fuji ImmunoPharmaceuticals Corp. and Shionogi BioResearch Corp. Dr. Chen received his B.S. in chemistry from National Taiwan University and his Ph.D. in cell biology from the Massachusetts Institute of Technology.

Judah Folkman, M.D. has been a member of our board of directors since September 2005 and has been a member of our scientific advisory board since September 2003. He began his career in 1965 as

an Instructor in Surgery for Harvard's Surgical Service at Boston City Hospital, and he became the Julia Dyckman Andrus Professor of Pediatric Surgery in 1968. For 14 years, he served as Surgeon-in-Chief at Children's Hospital Boston. Since 1971, when Dr. Folkman founded the field of angiogenesis research, he has made seminal discoveries on the mechanisms of angiogenesis that have opened a field of investigation now pursued worldwide. His laboratory reported the first purified angiogenesis molecule, the first angiogenesis inhibitor and proposed the concept of angiogenic disease. All of these discoveries have been translated into numerous clinical trials. Dr. Folkman is currently the Director of the Vascular Biology Program in the Department of Surgery at Children's Hospital. He holds honorary degrees from 17 universities and is the author of more than 400 original peer-reviewed papers and 109 book chapters and monographs. Dr. Folkman received his B.A. (*cum laude*) from Ohio State University in 1953 and his M.D. (*magna cum laude*) from Harvard Medical School in 1957. He is a member of the National Academy of Sciences, the American Academy of Arts and Sciences, and the American Philosophical Society.

Bruce Kovner has been a member of our board of directors since July 2002. In 1983, Mr. Kovner founded Caxton Corporation, a diversified trading company and manager of client funds active in currency, interest rate, commodity and equity markets, and has acted as its Chairman since its inception. He is also Chairman of Caxton Associates, L.L.C., which succeeded to a significant portion of Caxton Corporation's trading and investment activities in 1996. Prior to the formation of Caxton, Mr. Kovner served as a Vice President of Commodities Corporation, a private commodities trading company since acquired by Goldman Sachs. Mr. Kovner is also Chairman of the Board of the American Enterprise Institute, Chairman of the Board of the Juilliard School, and Vice Chairman of Lincoln Center for the Performing Arts. In addition, he is the Founder and Chairman of the School Choice Scholarships Foundation, which provides scholarships to low-income students in New York City to attend primary schools of their choice. Mr. Kovner received his B.A. from Harvard College in 1966. He continued his studies at the John F. Kennedy School of Government until 1970.

William S. Reardon, C.P.A. has been a member of our board of directors since August 2004. Until his retirement in 2002 from PricewaterhouseCoopers LLP, an international accounting firm, where he was employed from June 1973 to July 2002, Mr. Reardon was a business assurance (audit) partner at the firm's Boston office and leader of its life sciences industry practice for New England and the eastern United States. From 1998 to 2000, Mr. Reardon served on the board of the emerging companies section of the Biotechnology Industry Organization. He also served on the board of the Massachusetts Biotechnology Council from 2000 until his retirement in 2002. Mr. Reardon is currently a member of the board of directors and the chairman of the audit committees of Idera Pharmaceuticals, Inc., and Oscient Pharmaceuticals Corporation, both of which are publicly traded pharmaceutical companies. He is also an advisor to the audit committee at Momenta Pharmaceuticals, Inc., a publicly traded pharmaceutical company. Mr. Reardon received both his undergraduate degree in East Asian history and his M.B.A. from Harvard University.

Robert N. Wilson has been a member of our board of directors since June 2003. Mr. Wilson served as Vice Chairman of the board of directors of Johnson & Johnson, a global manufacturer of healthcare products, from 1986 until 2003. Mr. Wilson joined Johnson & Johnson in 1964. He was appointed to Johnson & Johnson's executive committee in 1983 and was elected to its board of directors in 1986. Mr. Wilson is also a director of The Charles Schwab Corporation, a publicly traded retail brokerage firm, U.S. Trust Corporation, United States Trust Company of New York and Amerada Hess Corporation, an integrated oil and gas company and is the Chairman of Caxton Health Holdings LLC, a healthcare investment firm that is an affiliate of Caxton Associates, L.L.C. Mr. Wilson received his B.A. in business administration from Georgetown College in Kentucky, and completed the Executive Program at Columbia University Graduate School of Business.

Scientific Advisory Board

We have established a scientific advisory board comprised of leading experts in their fields. Members of our scientific advisory board consult with us regularly on matters relating to:

- our research and development programs;
- the design and implementation of our clinical programs;
- market opportunities from a clinical perspective;
- new technologies relevant to our research and development programs; and
- scientific and technical issues relevant to our business.

The current members of our scientific advisory board are:

Name	Professional Affiliations/Honors
Lan Bo Chen, Ph.D., Chairman	See biography above.
Sir James W. Black, O.M., F.R.S.	Emeritus Professor of Analytical Pharmacology at King's College London; previously conducted research with Imperial Chemical Industries plc, SmithKline French and Wellcome Laboratories; was awarded the Nobel Prize in Medicine in 1989 for his work in pharmotherapeutic potential of receptor blocking drugs; knighted by the Queen of England in 1981; received the Order of Merit from the Queen in 2000.
Judah Folkman, M.D.	See biography above.
Marc B. Garnick, M.D.	Clinical Professor of Medicine at Harvard Medical School and physician at the Beth Israel Deaconess Medical Center; devoted his career to the development of novel cancer therapeutics, both from the academic and biotechnology perspective; previously served as the academic principal investigator for the development of leuprolide acetate (Lupron), a hormonal therapy for prostate cancer, and as the head of clinical and biometric departments at Genetics Institute (now Wyeth) and Praecis Pharmaceuticals, where he served as chief medical and regulatory officer; directed development of Plenaxis for prostate cancer, and gained its approval in United States and Europe; involved in the development of cisplatin, Interleukin 11, recombinant factor VIII and IX and other novel therapies; serves on the Board of Trustees of Bowdoin College and University of Pennsylvania School of Medicine and is editor in chief of *Perspectives on Prostate Diseases,* a quarterly journal of Harvard Health Publications.
Nir Hacohen, Ph.D.	Assistant Professor at Massachusetts General Hospital and Harvard Medical School; founder of the RNAi consortium, a group of Harvard and Massachusetts Institute of Technology researchers who are working to create and apply genome-wide gene silencing libraries to accelerate gene discovery in humans; honors include the Sandler Memorial first prize Ph.D. thesis award, Helen Hay Whitney Fellowship with David Baltimore and Whitehead Institute Fellowship.

Jean-Pierre Kinet, M.D.	Professor of Pathology at Harvard Medical School; Director of the Division of Allergy and Immunology at the Beth Israel Deaconess Medical Center; previously the head of the Molecular Allergy and Immunology section of the National Institute of Allergy and Infectious Diseases at the National Institutes of Health; scientific founder of Astarix Institute, Inc., an early- stage drug discovery company later sold to Heska Corporation.
Christopher J. Logothetis, M.D.	Professor and Chairman of the Department of Genitourinary Medical Oncology at the University of Texas M.D. Anderson Cancer Center; Principal Investigator of the M.D. Anderson SPORE in Prostate Cancer; Director of the Genitourinary Cancer Center and the Prostate Cancer Research Program, which are multidisciplinary collaborations of physicians and scientists dedicated to genitourinary cancer treatment, research, prevention, and education; leader in the Therapy Consortium, an active group of researchers involved in the development of innovative therapy for prostate cancer.
Mace L. Rothenberg, M.D.	Ingram Professor of Cancer Research at the Vanderbilt-Ingram Cancer Center and Professor of Medicine at Vanderbilt University Medical Center; Medical Oncologist with appointments at the Vanderbilt University Medical Center and the Department of Veterans Affairs Medical Center; Director of the Phase 1 Drug Development Program at Vanderbilt-Ingram Cancer Center; serves on a number of committees including the Vanderbilt-Ingram Cancer Center Gastrointestinal Cancer SPORE Executive Committee and Lung Cancer SPORE Steering Committee, the Clinical Cancer Research Committee for the American Association for Cancer Research, and the Medical Oncology Committee for the American College of Surgeons.
Daniel D. Von Hoff, M.D.	Professor of Medicine, Pathology, Molecular and Cellular Biology, at the University of Arizona; Director of the Arizona Health Sciences Center's Cancer Therapeutics Program; Executive Vice President of the Translational Genomics Research Institute, or TGen; Director of TGen's Translational Drug Development Division; Head, Pancreatic Cancer Research Program; Chief Medical Officer for U.S. Oncology, the nation's largest health-care services network devoted exclusively to cancer treatment and research; past President of the American Association for Cancer Research; past board member of the American Society of Clinical Oncology; founder and editor emeritus of *Investigational New Drugs — The Journal of New Anticancer Agents*; editor-in-chief of *Molecular Cancer Therapeutics*; appointed to President Bush's National Cancer Advisory Board in June 2004.

Michael E. Weinblatt, M.D.	Co-Director of Clinical Rheumatology at the Brigham and Women's Hospital and Professor of Medicine at Harvard Medical School; published over 127 papers, reviews and invited chapters in the field of rheumatology primarily rheumatoid arthritis therapeutics; co-editor of the textbook, *Treatment of Rheumatic Diseases,* the textbook, *Rheumatology* 3rd edition; author of the Arthritis Action Program; co-received in 1997 the Arthritis Foundation Virginia P. Engalitcheff Award for Impact on Quality of Life for work on methotrexate; served as an Associate Editor of Arthritis and Rheumatism; currently sits on the editorial board of *Journal of Rheumatology*; was a member of the Rheumatology Subspecialty Board of the American Board of Internal Medicine; in 2001, served as the President of the American College of Rheumatology.
Bruce R. Zetter, Ph.D.	Charles Nowiszewski professor in the departments of cell biology and surgery at Harvard Medical School; Chief Scientific Officer at Boston Children's Hospital; has won numerous national and international awards for his work in the field of cancer research including a Faculty Research Award from the American Cancer Society and the MERIT award from the National Cancer Institute; served as an expert witness on cancer to the U.S. Senate.

Board of Directors

Board Composition

Our restated certificate of incorporation and restated bylaws provide that the authorized number of directors may be changed only by resolution of the board of directors. In accordance with our restated certificate of incorporation and restated bylaws, our board of directors is divided into three classes with staggered three-year terms. At each annual meeting of stockholders commencing with the meeting in 2008, the successors to the directors whose terms then expire will be elected to serve until the third annual meeting following the election. Our directors are divided among the three classes as follows:

- The Class I directors are Dr. Chen and Mr. Reardon, and their terms will expire at the annual meeting of stockholders to be held in 2008;
- The Class II directors are Messrs. Gollust and Wilson, and their terms will expire at the annual meeting of stockholders to be held in 2009; and
- The Class III directors are Drs. Bahcall and Folkman and Mr. Kovner, and their terms will expire at the annual meeting of stockholders to be held in 2010.

Any additional directorships resulting from an increase in the number of directors will be distributed among the three classes so that, as nearly as possible, each class will consist of one-third of the directors.

Audit Committee of the Board of Directors

Our audit committee is composed of Messrs. Gollust, Reardon (chairman) and Wilson. All members of the audit committee satisfy the current independence standards promulgated by the Securities and Exchange Commission and by The Nasdaq Stock Market, LLC, as such standards apply

specifically to members of audit committees. Our board of directors has determined that Mr. Reardon is an "audit committee financial expert," as the Securities and Exchange Commission has defined that term in Item 407 of Regulation S-K. Our audit committee's role and responsibilities are set forth in the audit committee's written charter and include the authority to:

- approve and retain the independent auditors to conduct the annual audit of our books and records;
- review the proposed scope and results of the audit;
- review and pre-approve the independent auditor's audit and non-audit services rendered;
- approve the audit fees to be paid;
- review accounting and financial controls with the independent auditors and our financial and accounting staff;
- review and approve transactions between us and our directors, officers and affiliates;
- recognize and prevent prohibited non-audit services;
- establish procedures for complaints received by us regarding accounting matters;
- oversee internal audit functions, if any; and
- prepare the report of the audit committee that rules of the Securities and Exchange Commission require to be included in our annual meeting proxy statement.

Nominating and Governance Committee of the Board of Directors

Our nominating and governance committee is composed of Messrs. Gollust (chairman), Kovner and Wilson. All members of the nominating and governance committee qualify as independent under the current definition promulgated by The Nasdaq Stock Market, LLC. Our nominating and governance committee's role and responsibilities are set forth in the nominating and governance committee's written charter and include the authority to:

- identify and nominate members of the board of directors;
- develop and recommend to the board of directors a set of corporate governance principles applicable to our company; and
- oversee the evaluation of the board of directors and management.

The text of the nominating and governance committee's charter is posted on our website at *www.syntapharma.com* and will be made available to stockholders without charge, upon request, in writing to the Corporate Secretary at 45 Hartwell Avenue, Lexington, Massachusetts 02421.

Our nominating and governance committee may consider candidates recommended by stockholders as well as from other sources such as other directors or officers, third-party search firms or other appropriate sources. For all potential candidates, our nominating and governance committee may consider all factors it deems relevant, such as a candidate's personal integrity and sound judgment, business and professional skills and experience, independence, knowledge of the industry in which we operate, possible conflicts of interest, diversity, the extent to which the candidate would fill a present need on the board of directors, and concern for the long-term interests of the stockholders. In general, persons recommended by stockholders will be considered on the same basis as candidates from other sources. For each annual meeting, our nominating and governance committee will consider only one recommended nominee from any stockholder or group of affiliated stockholders, and such recommending stockholder or group must have held at least 5% of our common stock for at least one year. All stockholder recommendations for proposed director nominees must be in writing to the

nominating and governance committee, care of our Corporate Secretary at 45 Hartwell Avenue, Lexington, Massachusetts 02421, no later than 120 calendar days prior to the first anniversary of the date of the proxy statement for the prior annual meeting of stockholders or, in certain circumstances, a reasonable time in advance of the mailing of our proxy statement for the annual meeting of stockholders for the current year. The recommendation must be accompanied by the following information concerning the recommending stockholder:

- name, address and telephone number of the recommending stockholder;
- the number of shares of our common stock owned by the recommending stockholder and the time period for which such shares have been held;
- if the recommending stockholder is not a stockholder of record, a statement from the record holder verifying the holdings of the recommending stockholder and a statement from the recommending stockholder of the length of time such shares have been held (alternatively the recommending stockholder may furnish a current Schedule 13D, Schedule 13G, Form 3, Form 4 or Form 5 filed with the Securities and Exchange Commission, together with a statement of the length of time that the shares have been held); and
- a statement from the recommending stockholder as to a good faith intention to continue to hold such shares through the date of the next annual meeting.

The recommendation must also be accompanied by the following information concerning the proposed nominee:

- the information required by Items 401, 403 and 404 of Regulation S-K under the Securities Act of 1933, as amended;
- a description of all relationships between the proposed nominee and the recommending stockholder, including any agreements or understandings regarding the nomination;
- a description of all relationships between the proposed nominee and any of our competitors, customers, suppliers, labor unions or other persons with special interests regarding Synta; and
- the contact information of the proposed nominee.

The recommending stockholder must also furnish a statement supporting a view that the proposed nominee possesses the minimum qualifications as set forth below for director nominees and describing the contributions that the proposed nominee would be expected to make to the board of directors and to the governance of Synta and must state whether, in its view, the proposed nominee, if elected, would represent all stockholders and not serve for the purpose of advancing or favoring any particular stockholder or other constituency of Synta. The recommendation must also be accompanied by the written consent of the proposed nominee (i) to be considered by the nominating and governance committee and interviewed if the committee chooses to do so in its discretion, and (ii) if nominated and elected, to serve as a director.

For all potential candidates, the nominating and governance committee may consider all factors it deems relevant, including the following threshold criteria:

- candidates should possess the highest personal and professional standards of integrity and ethical values;
- candidates must be committed to promoting and enhancing the long-term value of Synta for its stockholders;
- candidates must be able to represent fairly and equally all stockholders without favoring or advancing any particular stockholder or other constituency of Synta;

- candidates must have demonstrated achievement in one or more fields of business, professional, governmental, community, scientific or educational endeavor, and possess mature and objective business judgment and expertise;

- candidates are expected to have sound judgment, derived from management or policy making experience that demonstrates an ability to function effectively in an oversight role; and

- candidates must have, and be prepared to devote, adequate time to the board of directors and its committees.

In addition, the nominating and governance committee will also take into account the extent to which the candidate would fill a present need on the board of directors, including the extent to which a candidate meets the independence and experience standards promulgated by the Securities and Exchange Commission and by The Nasdaq Stock Market, LLC.

Section 16(a) Beneficial Ownership Reporting Compliance

Section 16(a) of the Securities Exchange Act of 1934, as amended, requires our directors and executive officers, and persons who own more than ten percent of a registered class of our equity securities, to file with the Securities and Exchange Commission initial reports of ownership and reports of changes in ownership of our common stock and other of our equity securities. Officers, directors and greater than ten percent stockholders are required by regulations of the Securities and Exchange Commission to furnish us with copies of all Section 16(a) forms they file.

Our records reflect that all reports required to be filed pursuant to Section 16(a) of the Securities Exchange Act of 1934, as amended, by our executive officers and directors have been filed on a timely basis.

Code of Conduct and Ethics

We have adopted a code of conduct and ethics that applies to all of our employees, including our principal executive officer and principal accounting and financial officer, and our directors. The text of the code of conduct and ethics is posted on the Corporate Governance section of our website at *www.syntapharma.com* and will be made available to stockholders without charge, upon request, in writing to the Corporate Secretary at 45 Hartwell Avenue, Lexington, Massachusetts 02421. Disclosure regarding any amendments to, or waivers from, provisions of the code of conduct and ethics that apply to our directors, principal executive and financial and accounting officers will be included in a Current Report on Form 8-K within four business days following the date of the amendment or waiver, unless website posting of such amendments or waivers is then permitted by the rules of The Nasdaq Stock Market, LLC.

Item 11. EXECUTIVE COMPENSATION

Compensation Discussion and Analysis

The primary objectives of the compensation committee of our board of directors with respect to executive compensation are to attract, retain, and motivate the best possible executive talent. The focus is to tie short and long-term cash and equity incentives to achievement of measurable corporate and individual performance objectives, and to align executives' incentives with stockholder value creation. To achieve these objectives, the compensation committee has maintained, and expects to further implement, compensation plans that tie a substantial portion of executives' overall compensation to our research, clinical, regulatory, commercial, and operational performance.

Management develops our compensation plans by utilizing publicly available compensation data and subscription compensation survey data for national and regional companies in the biopharmaceutical industry. We believe that the practices of this group of companies provide us with appropriate compensation benchmarks, because these companies have similar organizational structures and tend to compete with us for executives and other employees. For benchmarking executive compensation, we typically review the compensation data we have collected from the complete group of companies, as well as a subset of the data from those companies that have a similar number of employees as our company. We have also engaged experienced consultants to help us analyze these data and to compare our compensation programs with the practices of the companies represented in the compensation data we review. In 2007, we intend to engage a qualified compensation consultant with experience in evaluating public biopharmaceutical companies to assist with a review of all components of our compensation program. Certain changes in our compensation program may be implemented based on recommendations made by the compensation consultant.

Based on management's analyses and recommendations, the compensation committee has approved a pay-for-performance compensation philosophy, which is intended to bring base salaries and total executive compensation in line with approximately the fiftieth percentile of the companies with a similar number of employees represented in the compensation data we review.

We work within the framework of this pay-for-performance philosophy to determine each component of an executive's initial compensation package based on numerous factors, including:

- the individual's particular background and circumstances, including training and prior relevant work experience;
- the individual's role with us and the compensation paid to similar persons in the companies represented in the compensation data that we review;
- the demand for individuals with the individual's specific expertise and experience at the time of hire;
- performance goals and other expectations for the position;
- comparison to other executives within our company having similar levels of expertise and experience; and
- uniqueness of industry skills.

The compensation committee has also implemented an annual performance management program, under which annual performance goals are determined and set forth in writing at the beginning of each calendar year for the corporation as a whole, each corporate department, and each individual employee. Annual corporate goals are proposed by management and approved by the board of directors at the end of each calendar year for the following year. These corporate goals target the achievement of specific research, clinical, regulatory, and operational milestones. Annual department and individual goals focus on contributions which facilitate the achievement of the corporate goals and

are set during the first quarter of each calendar year. Department goals are proposed by each department head and approved by the Chief Executive Officer. Individual goals are proposed by each employee and approved by his or her direct supervisor. The Chief Executive Officer approves the goals proposed by our other executive officers. The Chief Executive Officer's goals are approved by the compensation committee of the board of directors. Annual salary increases, annual bonuses, and annual stock option awards granted to our employees are tied to the achievement of these corporate, department, and each individual's performance goals.

We perform an interim assessment of the written goals in the third quarter of each calendar year to determine individual, department and corporate progress against the previously established goals and to make any adjustments to the goals for the remainder of the year based on changing circumstances.

During the first calendar quarter, we evaluate individual, department, and corporate performance against the written goals for the recently completed year. Consistent with our compensation philosophy, each employee's evaluation begins with a written self-assessment, which is submitted to the employee's supervisor. The supervisor then prepares a written evaluation based on the employee's self-assessment, the supervisor's own evaluation of the employee's performance, and input from others within the company. This process leads to a recommendation for annual employee salary increases, annual stock option awards, and bonuses, if any, which is then reviewed and approved by the compensation committee. Our executive officers, other than the Chief Executive Officer, submit their self-assessments to the Chief Executive Officer, who performs the individual evaluations and submits recommendations to the compensation committee for salary increases, bonuses, and stock option awards. In the case of the Chief Executive Officer, his individual performance evaluation is conducted by the compensation committee, which determines his compensation changes and awards. For all employees, including our executive officers, annual base salary increases, annual stock option awards, and annual bonuses, to the extent granted, are implemented during the first calendar quarter of the year.

Compensation Components

The components of our compensation package are as follows:

Base Salary

Base salaries for our executives are established based on the scope of their responsibilities and their prior relevant background, training, and experience, taking into account competitive market compensation paid by the companies represented in the compensation data we review for similar positions and the overall market demand for such executives at the time of hire. As with total executive compensation, we believe that executive base salaries should generally target the fiftieth percentile of the range of salaries for executives in similar positions and with similar responsibilities in the companies of similar size to us represented in the compensation data we review. An executive's base salary is also evaluated together with other components of the executive's other compensation to ensure that the executive's total compensation is in line with our overall compensation philosophy.

Base salaries are reviewed annually as part of our performance management program and increased for merit reasons, based on the executive's success in meeting or exceeding individual performance objectives and an assessment of whether significant corporate goals were achieved. If necessary, we also realign base salaries with market levels for the same positions in the companies of similar size to us represented in the compensation data we review, if we identify significant market changes in our data analysis. Additionally, we adjust base salaries as warranted throughout the year for promotions or other changes in the scope or breadth of an executive's role or responsibilities.

In February 2007, the compensation committee approved base salary increases for our executives effective March 1, 2007, including increasing the base salary for each of our Chief Executive Officer and other named executive officers to the following:

Name	Base Salary ($)
Safi R. Bahcall, Ph.D	355,000
Keith S. Ehrlich, C.P.A.	230,000
James G. Barsoum, Ph.D	240,000
Eric W. Jacobson, M.D	280,000
Keizo Koya, Ph.D	270,000

To align our executives' base salaries with market levels for the same positions in companies of similar size to us, base salaries for the Chief Executive Officer and the other named officers, except Dr. Jacobson, were increased by approximately 4-5%. Dr. Jacobson's base salary increase of approximately 8% was provided to recognize Dr. Jacobson's contributions in 2006 and align his base salary with the competitive market level for his position.

Annual Bonus

Our compensation program includes eligibility for an annual performance-based cash bonus in the case of all executives and certain senior, non-executive employees. The amount of the cash bonus depends on the level of achievement of the stated corporate, department, and individual performance goals, with a target bonus generally set as a percentage of base salary. Currently, all executives, other than our Chief Executive Officer, and certain senior non-executive employees are eligible for annual performance-based cash bonuses in amounts ranging from 10%-20% of their base salaries, as set forth in their employment offer letters. In its discretion, the compensation committee may, however, award bonus payments to our executives above or below the amounts specified in their respective offer letters. As provided in his employment agreement, our Chief Executive Officer is eligible for an annual performance-based bonus, the amount of which, if any, is determined by the board of directors or the compensation committee in their sole discretion.

In February 2007, the compensation committee approved the design of an expanded cash bonus plan. The plan is intended to increase the cash element of our annual compensation program in relation to stock-based compensation to more closely track the compensation programs of publicly traded biotechnology companies with which we compete for executives and other employees. However, the compensation committee decided to defer setting the parameters for the expanded cash bonus program until after receiving the recommendations of the compensation expert engaged to evaluate our overall compensation program.

In February 2007, the compensation committee approved our executives' cash bonus awards for 2006 performance, including the following awards to our Chief Executive Officer and other named executive officers:

Name	Bonus ($)
Safi R. Bahcall, Ph.D.	100,000
Keith S. Ehrlich, C.P.A.	44,000
James G. Barsoum, Ph.D.	46,000
Eric W. Jacobson, M.D.	65,000
Keizo Koya, Ph.D.	52,000

The Chief Executive Officer's cash bonus equaled 30% of his base salary and the other named officers were awarded cash bonus amounts equaling 20-25% of their respective base salaries. These percentages were generally consistent with the cash bonuses awarded in the prior year. The

compensation committee believed that these cash bonus awards were competitive with other public biotechnology companies of similar size to us.

Long-Term Incentives

We believe that long-term performance is achieved through an ownership culture that encourages long-term participation by our executive officers in equity-based awards. Our 2006 Stock Plan allows the grant to executive officers of stock options, restricted stock, and other equity-based awards. We typically make an initial equity award of stock options to new employees and annual equity grants as part of our overall compensation program. An option committee appointed by our board of directors is currently authorized to make initial equity grants within certain parameters, beyond which compensation committee approval is required. Annual grants of options to all of our employees are approved by the compensation committee. We expect that all future equity awards to our executive officers will be approved by the compensation committee or our board of directors.

Initial stock option awards. Executives who join us are awarded initial stock option grants. These grants have an exercise price equal to the fair market value of our common stock on the grant date and a vesting schedule of 25% on the first anniversary of the date of hire and quarterly thereafter for the next three years. The amount of the initial stock option award is determined based on the executive's position with us and analysis of the competitive practices of the companies similar in size to us represented in the compensation data that we review. The initial stock option awards are calculated to have a total face value (calculated by multiplying the number of shares subject to the option by the exercise price thereof) equal to a percentage of the executive's base salary, and are intended to provide the executive with incentive to build value in the organization over an extended period of time. The amount of the initial stock option award is also reviewed in light of the executive's base salary and other compensation to ensure that the executive's total compensation is in line with our overall compensation philosophy. Typically, we grant our executives initial stock option awards with a total face value ranging from one to four times the executive's base salary.

Restricted stock awards. We have made grants of restricted stock to executive officers and certain high ranking non-executive employees to provide additional long-term incentive to build stockholder value. Restricted stock awards are made in anticipation of contributions that will create value in the company and are subject to a lapsing repurchase right by the company over a period of time. Because the shares have a defined value at the time the restricted stock grants are made, restricted stock grants are often perceived as having more immediate value than stock options, which have a less calculable value when granted. However, we generally grant fewer shares of restricted stock than the number of stock options we would grant for a similar purpose. In 2004 and 2005, we awarded certain executive officers and senior non-executive employees restricted stock grants that are subject to a lapsing repurchase right as to the first 50% of the shares after two years and the remaining 50% of the shares after the earlier of four years or approval of an NDA with the FDA. The second vesting tranche of these restricted stock grants was structured in this way to recognize the significance of the approval of an NDA to us and to award the executive's role in achieving such a milestone. In December 2006, the compensation committee approved amendments to the restricted stock agreements with vesting in January 2007, which provided the option of withholding from each executive the number of shares of common stock necessary in order to satisfy the statutory minimum tax withholding obligations incurred in January 2007 with respect to the vesting of the initial 50% of these particular awards. See "—Grants of Plan-Based Awards—Amendment of Restricted Stock Agreements" below. The compensation committee approved these amendments to provide the executives with a method to satisfy the statutory minimum tax withholding obligations with respect to the vesting of shares at a time when no public market was expected to exist that would allow the executives to sell their vested shares to obtain the cash funds necessary to remit to Synta.

Annual stock option awards. Our practice is to make annual stock option awards as part of our overall performance management program. The compensation committee believes that stock options provide management with a strong link to long-term corporate performance and the creation of stockholder value. We intend that the annual aggregate value of these awards will be set near competitive median levels for companies represented in the compensation data we review. As is the case when the amounts of base salary and initial equity awards are determined, a review of all components of the executive's compensation is conducted when determining annual equity awards to ensure that an executive's total compensation conforms to our overall philosophy and objectives. A pool of options is reserved for executives and non-officers based on setting a target grant level for each employee category, with the higher ranked employees being eligible for a higher target grant.

In 2006, the compensation committee agreed in principle that annual stock option awards would be based on a multiple of base salary and reflect individual performance levels. In February 2007, the compensation committee determined specific target amounts and made individual grants to our executives, including the following grants to our Chief Executive Officer and our other named executive officers:

Name	Number of Options(#)(1)	Exercise Price Per Share ($)
Safi R. Bahcall, Ph.D	46,000	8.53(2)
Keith S. Ehrlich, C.P.A.	22,000	8.75(3)
James G. Barsoum, Ph.D.	23,000	8.75(3)
Eric W. Jacobson, M.D.	32,500	8.75(3)
Keizo Koya, Ph.D.	26,000	8.75(3)

(1) All options vest as to 25% of the shares on the first anniversary of the date of grant and 6.25% of the shares on the last day of each successive three-month period thereafter.

(2) Exercise price is equal to the closing price on the date of grant, February 28, 2007.

(3) Exercise price is equal to the closing price on the date of grant, February 26, 2007.

Option repricing. In February 2006, our board of directors approved the March 1, 2006 repricing of options issued under our 2001 Stock Plan having an exercise price at or above $16.00 per share to $14.00 per share, including options held by our executives. See "—Grants of Plan-Based Awards—Stock Option Repricing" below. In deciding to approve this repricing, our board of directors considered the fact that:

- we issued the options outstanding under our 2001 Stock Plan (i) to provide our employees an opportunity to acquire or increase an equity interest in the company, thereby creating a stronger incentive to expend maximum effort for our growth and success and (ii) to encourage our employees to continue their service to us; and
- approximately 54% of our outstanding options, whether or not they were exercisable at the time of the repricing, had exercise prices that were significantly higher than the fair market value of our common stock at the date of the repricing.

Our board of directors believed these options were unlikely to be exercised in the foreseeable future because of the disparity that existed at the time of the repricing between the exercise price of the repriced options and the fair market value of our common stock at such time. By approving a one-time repricing and creating options with an exercise price equal to the fair market value of our common stock on the repricing date, our board of directors intended to provide our option holders with the benefit of owning options that over time may have a greater potential to increase in value, create better performance incentives, and thereby more effectively promote stockholder value.

Other Compensation

We maintain broad-based benefits that are provided to all employees, including health insurance, life and disability insurance, dental insurance, and a 401(k) plan with a matching company contribution. In particular circumstances, we also utilize cash signing bonuses when certain executives and senior non-executives join us. Such cash signing bonuses are typically repayable in full to the company if the employee recipient voluntarily terminates employment with us prior to the first anniversary of the date of hire. Whether a signing bonus is paid and the amount thereof is determined on a case-by-case basis under the specific hiring circumstances. For example, we will consider paying signing bonuses to compensate for amounts forfeited by an executive upon terminating prior employment, to assist with relocation expenses, and/or to create additional incentive for an executive to join our company in a position where there is high market demand. We also provide our Chief Executive Officer with a company apartment in Massachusetts free of charge and reimburse him for commuting costs for travel from his residence in New York to our offices in Lexington, Massachusetts. Our board of directors and compensation committee believe that these payments facilitate the Chief Executive Officer's travel between Massachusetts and New York, where our Chief Executive Officer is required to conduct significant business activities on behalf of the company.

Termination Based Compensation

Severance. Upon termination of employment, most executive officers are entitled to receive severance payments under their employment offer letters. In determining whether to approve and setting the terms of such severance arrangements, the compensation committee recognizes that executives, especially highly ranked executives, often face challenges securing new employment following termination. Severance for termination without cause for executive officers, other than our Chief Executive Officer, ranges between zero and three months of base salary. Our Chief Executive Officer's employment agreement provides severance of 24 months of base salary if his employment is terminated without cause. After reviewing the practices of companies represented in the compensation data we obtained, the compensation committee negotiated our Chief Executive Officer's severance package to provide him an amount equal to his base salary for the length of his non-competition arrangement with us. We believe that our Chief Executive Officer's severance package is generally in line with severance packages offered to chief executive officers of the companies of similar size to us represented in the compensation data we reviewed.

Acceleration of vesting of equity-based awards. In the event of a change of control as defined in our 2001 Stock Plan, certain provisions of our 2001 Stock Plan allow for acceleration of equity awards in case an employee is terminated for certain reasons after a change of control, which we refer to as "double trigger" acceleration. See "—Potential Payments Upon Termination or Change of Control—Change of Control Arrangements Under Our 2001 Stock Plan" below for a detailed discussion of these provisions. In addition, our 2006 Stock Plan, under which we will grant future equity awards, provides for a similar "double trigger" acceleration mechanism. See Item 12 "Security Ownership of Certain Beneficial Owners and Management and Related Stockholder Matters—Summary Description of our Stockholder Approved Equity Compensation Plans" below for a discussion of the change of control provisions of the 2006 Stock Plan. We believe a "double trigger" requirement maximizes shareholder value because it prevents an unintended windfall to management in the event of a friendly (non-hostile) change of control. Under this structure, unvested equity awards under our 2001 Stock Plan and 2006 Stock Plan would continue to incentivize our executives to remain with the company after a friendly change of control. If, by contrast, our 2001 Stock Plan and 2006 Stock Plan had only a "single trigger," and if a friendly change of control occurred, management's equity awards would all vest immediately, creating a windfall, and the new owner would then likely find it necessary to replace the compensation with new unvested equity awards in order to retain management. This rationale is why we believe a

"double-trigger" equity vesting acceleration mechanism is more stockholder-friendly, and thus more appropriate for our company, than a "single trigger" acceleration mechanism.

Conclusion

Our compensation policies are designed to retain and motivate our senior executive officers and to ultimately reward them for outstanding individual and corporate performance.

Compensation Committee Report

The compensation committee of our board of directors has reviewed and discussed the compensation discussion and analysis required by Item 402(b) of Regulation S-K, which appears above, with our management. Based on this review and discussion, the compensation committee has recommended to the board of directors that the compensation discussion and analysis be included in our Annual Report on Form 10-K.

Members of the Synta Pharmaceuticals Corp.
Compensation Committee:

Robert N. Wilson (Chairman)
Keith R. Gollust
Bruce Kovner

Summary Compensation Table

The following table shows the compensation paid or accrued during the fiscal year ended December 31, 2006 to (1) our President and Chief Executive Officer, (2) our Chief Financial Officer and (3) our three most highly compensated executive officers, other than our President and Chief Executive Officer and our Chief Financial Officer.

Name and Principal Position	Year	Salary ($)	Bonus ($)	Stock Awards ($)(1)	Option Awards ($)(2)	All Other Compensation ($)	Total ($)
Safi R. Bahcall, Ph.D. President and Chief Executive Officer	2006	340,000	100,000	343,256(3)	243,309(4)	38,610(5)	1,065,175
Keith S. Ehrlich, C.P.A. Vice President, Finance and Administration, Chief Financial Officer	2006	215,334	44,000	137,498(6)	93,101(7)	5,241(8)	495,174
James G. Barsoum, Ph.D. Senior Vice President, Research	2006	228,334	46,000	219,996(9)	115,477(10)	5,339(8)	615,146
Eric W. Jacobson, M.D. Senior Vice President, Clinical Research and Regulatory Affairs, Chief Medical Officer	2006	249,166	65,000	87,498(11)	149,811(12)	5,700(8)	557,175
Keizo Koya, Ph.D. Senior Vice President, Drug Development	2006	258,334	52,000	219,996(13)	122,693(14)	4,078(15)	657,101

(1) See Notes 2 and 6 to our audited consolidated financial statements included elsewhere in this Annual Report on Form 10-K for details as to the assumptions used to determine the fair value of the stock awards. See also our discussion of stock-based compensation under Item 7 "Management's Discussion and Analysis of Financial Condition and Results of Operations—Critical Accounting Policies and Estimates."

(2) See Note 2 to our audited consolidated financial statements included elsewhere in this Annual Report on Form 10-K for details as to the assumptions used to determine the fair value of the option awards. Our executive officers will not realize the value of these awards in cash until these awards are exercised and the underlying shares are subsequently sold. See also our discussion of stock-based compensation under Item 7 "Management's Discussion and Analysis of Financial Condition and Results of Operations—Critical Accounting Policies and Estimates."

(3) Consists of $274,995 and $68,261, representing the compensation expense incurred by us in fiscal year 2006 in connection with grants to Dr. Bahcall of 50,000 shares of restricted common stock on December 21, 2004, calculated in accordance with APB Opinion No. 25, and 4,875 shares of common stock on April 14, 2006, calculated using a $14.00 per share price, the fair market value of the common stock on such date.

(4) Consists of $131,063 and $112,246, representing the compensation expense incurred by us in fiscal year 2006 in connection with option grants to Dr. Bahcall to purchase 37,500 shares of common stock on February 15, 2005 and 50,000 shares of common stock on February 15, 2006, respectively, calculated in accordance with SFAS 123 and SFAS 123R, respectively.

(5) Consists of $28,474 of rental payments for a company apartment for Dr. Bahcall's use and $10,136 in commuting costs for Dr. Bahcall's travel from his home in New York to our offices in Lexington, Massachusetts.

(6) Represents the compensation expense incurred by us in fiscal year 2006 in connection with a grant of 25,000 shares of restricted common stock to Mr. Ehrlich on December 21, 2004, calculated in accordance with APB Opinion No. 25.

(7) Consists of $46,920 and $46,181, representing the compensation expense incurred by us in fiscal year 2006 in connection with option grants to Mr. Ehrlich to purchase 13,425 shares of common stock on February 15, 2005 and 20,571 shares of common stock on February 15, 2006, respectively, calculated in accordance with SFAS 123 and SFAS 123R, respectively.

(8) Represents matching contributions made under our 401(k) plan.

(9) Represents the compensation expense incurred by us in fiscal year 2006 in connection with a grant of 40,000 shares of restricted common stock to Dr. Barsoum on December 21, 2004, calculated in accordance with APB Opinion No. 25.

(10) Consists of $62,561 and $52,916, representing the compensation expense incurred by us in fiscal year 2006 in connection with option grants to Dr. Barsoum to purchase 17,900 shares of common stock on February 15, 2005 and 23,571 shares of common stock on February 15, 2006, respectively, calculated in accordance with SFAS 123 and SFAS 123R, respectively.

(11) Represents the compensation expense incurred by us in fiscal year 2006 in connection with a grant of 25,000 shares of restricted common stock to Dr. Jacobson on December 12, 2005, calculated in accordance with SFAS 123.

(12) Consists of $87,575 and $62,236, representing the compensation expense incurred by us in fiscal year 2006 in connection with option grants to Dr. Jacobson to purchase 25,000 shares of common stock on April 11, 2005 and 27,723 shares of common stock on February 15, 2006, respectively, calculated in accordance with SFAS 123 and SFAS 123R, respectively.

(13) Represents the compensation expense incurred by us in fiscal year 2006 in connection with a grant of 40,000 shares of restricted common stock to Dr. Koya on December 21, 2004, calculated in accordance with APB Opinion No. 25.

(14) Consists of $62,561 and $60,132, representing the compensation expense incurred by us in fiscal year 2006 in connection with option grants to Dr. Koya to purchase 17,900 shares of common stock on February 15, 2005 and 26,785 shares of common stock on February 15, 2006, respectively, calculated in accordance with SFAS 123 and SFAS 123R, respectively.

(15) Consists of $2,792 in lease payments for an automobile for Dr. Koya's use, which expired in 2006, and $1,286 in matching contributions made under our 401(k) plan.

Grants of Plan-Based Awards

The following table shows information regarding grants of equity awards and the repricing of certain previously granted option awards during the fiscal year ended December 31, 2006 held by the executive officers named in the Summary Compensation Table.

Name	Grant Date	All Other Stock Awards: Number of Shares of Stock or Units(#)	All Other Option Awards: Number of Securities Underlying Options(#)	Exercise or Base Price of Option Awards($/Sh)	Grant Date Fair Value of Stock and Option Awards
Safi R. Bahcall, Ph.D.	2/15/06	—	50,000	14.00	$ 489,800
President and Chief Executive Officer	3/01/06(1)	—	37,500	14.00(1)	$ 46,350(1)
	4/14/06	4,875(2)	—		$ 68,261
Keith S. Ehrlich, C.P.A.	2/15/06	—	20,571	14.00	$ 201,518
Vice President, Finance and	3/01/06(1)	—	13,425	14.00(1)	$ 16,593(1)
Administration, Chief Financial Officer	3/01/06(3)	—	37,500	14.00(3)	$ 17,400(3)
James G. Barsoum, Ph.D.	2/15/06	—	23,571	14.00	$ 230,906
Senior Vice President,	3/01/06(1)	—	17,900	14.00(1)	$ 22,124(1)
Research	3/01/06(3)	—	10,000	14.00(3)	$ 4,640(3)
Eric W. Jacobson, M.D	2/15/06	—	27,723	14.00	$ 271,577
Senior Vice President, Clinical Research and Regulatory Affairs, Chief Medical Officer	3/01/06(4)	—	25,000	14.00(4)	$ 30,200(4)
Keizo Koya, Ph.D.	2/15/06	—	26,785	14.00	$ 262,393
Senior Vice President,	3/01/06(1)	—	17,900	14.00(1)	$ 22,124(1)
Drug Development	3/01/06(3)	—	10,000	14.00(3)	$ 4,640(3)

(1) This option was originally granted on February 15, 2005 with an exercise price of $22.00 per share and was repriced effective March 1, 2006 to an exercise price of $14.00 per share. The referenced dollar figure is the incremental fair value associated with this modification, calculated in accordance with SFAS 123R.

(2) Dr. Bahcall received a $100,000 bonus in 2006 for his performance during the fiscal year ended December 31, 2005, which was paid in shares of common stock. On April 14, 2006, the compensation committee granted Dr. Bahcall 4,875 shares of common stock calculated using a $14.00 per share price, the fair market value of the common stock on such date. The remaining $31,739 represents the amount of taxes required to be withheld on the bonus. This stock grant was not issued pursuant to an equity plan.

(3) This option was originally granted on May 27, 2004 with an exercise price of $16.00 per share and was repriced effective March 1, 2006 to an exercise price of $14.00 per share. The referenced dollar figure is the incremental fair value associated with this modification, calculated in accordance with SFAS 123R.

(4) This option was originally granted on April 11, 2005 with an exercise price of $22.00 per share and was repriced effective March 1, 2006 to an exercise price of $14.00 per share. The referenced dollar figure is the incremental fair value associated with this modification, calculated in accordance with SFAS 123R.

The terms of each executive officer's compensation are derived from our employment agreement, in the case of Dr. Bahcall, and our letter agreements, in the case of our other executive officers, entered into between us and them and annual performance reviews conducted by the compensation committee, in the case of Dr. Bahcall, and by Dr. Bahcall for the other executive officers. Annual base salary increases, annual stock option awards and cash bonuses, if any, for Dr. Bahcall are determined by the compensation committee. Dr. Bahcall recommends annual base salary increases, annual stock option awards and cash bonuses, if any, for the other executive officers, which are reviewed and approved by the compensation committee.

Employment Agreement with Dr. Safi Bahcall

Pursuant to a letter agreement effective as of April 18, 2005 between us and Dr. Bahcall, we agreed to employ Dr. Bahcall as our President and Chief Executive Officer on an at-will basis. We also agreed that so long as Dr. Bahcall continues to serve as our President and Chief Executive Officer, he will be nominated by the board of directors for election as a director at each annual meeting preceding which his term as director expires. Under this agreement, Dr. Bahcall's current base salary is $355,000 per year, subject to adjustment from time to time at the discretion of the board of directors or the compensation committee. Dr. Bahcall is also eligible to receive annual performance-based bonuses and grants of stock options under our stock plans at the discretion of the board of directors or the compensation committee. Pursuant to the terms of this agreement, we may apply for and purchase key person life insurance on Dr. Bahcall in an amount determined by Synta and with Synta as the beneficiary and one or more other policies of insurance insuring Dr. Bahcall's life. To date, we have not purchased any life insurance on Dr. Bahcall. As a condition of employment, Dr. Bahcall has entered into a non-competition/non-solicitation agreement pursuant to which he has agreed not to compete with Synta or to solicit customers or employees of Synta for a period of 24 months after the termination of his employment.

Offer Letters

We do not have formal employment agreements with any of our other executive officers named in the Summary Compensation Table, however certain elements of the executive officers' compensation and other employment arrangements are set forth in letter agreements that we executed with each of them at the time their employment with us commenced. The letter agreements provide, among other things, the executive officer's initial annual base salary, eligibility to receive annual performance-based bonuses for fully meeting and exceeding expectations in the 10%—20% range, with a full target level of 20%, such bonus, if any, being at the discretion of the board of directors, and initial stock option award. Each letter agreement provides that the executive officer's employment with us is on an at-will basis. As a condition to their employment, each executive officer has entered into a non-competition/non-solicitation agreement pursuant to which each officer has agreed not to compete with Synta or to solicit customers or employees of Synta for a period of 12 months after the termination of employment. These letter agreements are further described below. Since the date of the letter agreements, the compensation paid to each of these executive officers has been increased, additional equity awards have been awarded and the amount of bonuses has increased.

Keith S. Ehrlich, C.P.A. Pursuant to a letter agreement dated February 19, 2004 between us and Mr. Ehrlich, we agreed to employ Mr. Ehrlich as Vice President, Finance and Administration, beginning on March 1, 2004. In October 2006, Mr. Ehrlich began serving as our Chief Financial Officer. Mr. Ehrlich's annual base salary is currently $230,000 per year.

James G. Barsoum, Ph.D. Pursuant to a letter agreement dated January 22, 2003 between us and Dr. Barsoum, we agreed to employ Dr. Barsoum as Vice President, Biology, beginning on February 26, 2003. In October 2006, Dr. Barsoum began serving as our Senior Vice President, Research. Dr. Barsoum's annual base salary is currently $240,000.

Eric W. Jacobson, M.D. Pursuant to a letter agreement dated March 23, 2005 between us and Dr. Jacobson, we agreed to employ Dr. Jacobson as Vice President, Medical Research, beginning on April 11, 2005. In connection with the execution of the letter agreement, we paid Dr. Jacobson a lump sum bonus of $25,000. Since January 2006, Dr. Jacobson has served as our Chief Medical Officer and since October 2006, as our Senior Vice President, Clinical Research and Regulatory Affairs. Dr. Jacobson's annual base salary is currently $280,000 per year.

Keizo Koya, Ph.D. Pursuant to a letter agreement dated October 1, 2002 between us and Dr. Koya, we agreed to employ Dr. Koya as Senior Vice President of Drug Development, beginning on October 1, 2002. Dr. Koya's annual base salary is currently $270,000 per year.

For a description and quantification of benefits payable to the executive officers named in our Summary Compensation Table in connection with a termination of employment or a change of control, see "—Potential Payments Upon Termination or Change of Control."

Fiscal Year 2006 Equity Awards and Award Modifications

All of the stock option awards disclosed in the Grants of Plan-Based Awards table were issued under our 2001 Stock Plan and were granted with an exercise price per share equal to the fair market value of our common stock on the date of grant, as determined by our board of directors. Subject to the terms of the 2001 Stock Plan and the option agreements issued in connection with these grants, all of the options originally granted in 2006 vest as to 25% of the shares on the first anniversary of the grant date and as to an additional 6.25% of the shares on the last day of each calendar quarter thereafter. Our stock option awards vest in full upon a termination following a change of control as discussed below under "—Potential Payments Upon Termination or Change of Control".

Annual Stock Option Grants

On February 15, 2006, the compensation committee awarded our executive officers option awards as part of the compensation committee's annual option award grants to all of our officers and employees. These awards represent compensation for performance in 2005.

Stock Option Repricing

In February 2006, our board of directors approved the repricing of options issued under our 2001 Stock Plan having an exercise price at or above $16.00 per share to $14.00 per share, the fair market value of our common stock on the date of the repricing, as determined by our board of directors. This repricing applied to all outstanding options under this plan held by our active employees and others having an ongoing relationship with us at the time of the repricing. The repricing was effective on March 1, 2006 and impacted outstanding stock options held by each of our current executive officers named in the Summary Compensation Table.

Amendment of Restricted Stock Agreements

In December 2004, we granted shares of restricted stock to certain of our executive officers, including Drs. Bahcall, Barsoum and Koya and Mr. Ehrlich under our 2001 Stock Plan and pursuant to restricted stock agreements executed in connection therewith. Pursuant to the terms of the restricted stock agreements, 50% of the shares subject to each grant vested on January 4, 2007. In December 2006, our compensation committee approved amendments to these restricted stock agreements, pursuant to which we have agreed, as permitted by the 2001 Stock Plan, to withhold from each officer such number of shares of common stock as is necessary in order to satisfy our statutory minimum tax withholding obligations that were incurred on January 4, 2007 with respect to each officer in connection with the vesting of the shares.

Outstanding Equity Awards at Fiscal Year-End

The following table shows grants of stock options and grants of unvested stock awards outstanding on December 31, 2006, the last day of our fiscal year, to each of the executive officers named in the Summary Compensation Table.

	Option Awards				Stock Awards	
Name	Number of Securities Underlying Unexercised Options (#) Exercisable	Number of Securities Underlying Unexercised Options (#) Unexercisable	Option Exercise Price ($)	Option Expiration Date	Number of Shares or Units of Stock That Have Not Vested (#)	Market Value of Shares or Units of Stock That Have Not Vested ($)(1)
Safi R. Bahcall, Ph.D.	—	—	—	—	50,000(2)	500,000
President and Chief	18,750	18,750(3)	14.00(4)	2/15/15	—	—
Executive Officer	—	50,000(5)	14.00	2/15/16	—	—
Keith S. Ehrlich, C.P.A.	28,646	8,854(6)	14.00(7)	5/27/14	—	—
Vice President, Finance	—	—	—	—	25,000(8)	250,000
and Administration, Chief	6,712	6,712(3)	14.00(4)	2/15/15	—	—
Financial Officer	—	20,571(5)	14.00	2/15/16	—	—
James G. Barsoum,	70,312	4,687(9)	10.843	4/3/13	—	—
Ph.D.	7,500	2,500(10)	14.00(7)	5/27/14	—	—
Senior Vice President,	—	—	—	—	40,000(11)	400,000
Research	8,950	8,950(3)	14.00(4)	2/15/15	—	—
	—	23,571(5)	14.00	2/15/16	—	—
Eric W. Jacobson, M.D.	10,937	14,062(12)	14.00(4)	2/15/15	—	—
Senior Vice President,	—	—	—	—	25,000(13)	250,000
Clinical Research and Regulatory Affairs, Chief Medical Officer	—	27,723(5)	14.00	2/15/16	—	
Keizo Koya, Ph.D.	125,000(14)	—	10.843	12/13/12	—	—
Senior Vice President,	46,875	3,125(15)	10.843	6/17/13	—	—
Drug Development	7,500	2,500(10)	14.00(7)	5/27/14	—	—
	—	—	—	—	40,000(11)	400,000
	8,950	8,950(3)	14.00(4)	2/15/15	—	—
	—	26,785	14.00	2/15/16	—	—

(1) The market value of the stock awards was determined by multiplying the number of shares times $10.00, the initial public offering price of our common stock on February 6, 2007.

(2) 25,000 shares vest on each of January 4, 2007 and January 4, 2009, provided that, if prior to January 4, 2009 we receive approval of an NDA, the 25,000 shares vesting on January 4, 2009 will vest at the time such approval is received.

(3) The option vested as to 25% of the shares on February 15, 2006 and as to an additional 6.25% of the shares on the last day of each calendar quarter thereafter.

(4) These options were originally granted at an exercise price of $22.00 per share and were repriced effective March 1, 2006 to $14.00 per share.

(5) The option vested as to 25% of the shares on February 15, 2007 and as to an additional 6.25% of the shares on the last day of each calendar quarter thereafter.

(6) The option vested as to 10,938 of the shares on March 1, 2005 and as to an additional 2,213 shares on the last day of each calendar quarter thereafter.

(7) These options were originally granted at an exercise price of $16.00 per share and were repriced effective March 1, 2006 to $14.00 per share.

(8) 12,500 shares vest on each of January 4, 2007 and January 4, 2009, provided that, if prior to January 4, 2009 we receive approval of an NDA, the 12,500 shares vesting on January 4, 2009 will vest at the time such approval is received.

(9) The option vested as to 25% of the shares on April 3, 2004 and as to an additional 6.25% of the shares on the last day of each calendar quarter thereafter.

(10) The option vested as to 25% of the shares on March 1, 2005 and as to an additional 6.25% of the shares at the end of each calendar quarter thereafter.

(11) 20,000 shares vest on each of January 4, 2007 and January 4, 2009, provided that, if prior to January 4, 2009 we receive approval of an NDA, the 20,000 shares vesting on January 4, 2009 will vest at the time such approval is received.

(12) The option vested as to 25% of the shares on April 11, 2006 and as to an additional 6.25% of the shares on the last day of each calendar quarter thereafter.

(13) 12,500 shares vest on each of January 4, 2008 and January 4, 2010, provided that, if prior to January 4, 2010 we receive approval of an NDA, the 12,500 shares vesting on January 4, 2010 will vest at the time such approval is received.

(14) The option vested as to 37,500 shares on October 1, 2002 and as to an additional 6.25% of the shares at the end of each calendar quarter thereafter and is fully vested.

(15) The option vested as to 25% of the shares on April 1, 2004 and as to an additional 6.25% of the shares at the end of each calendar quarter thereafter.

Option Exercises and Stock Vested

There were no exercises of stock options or vesting of shares of restricted stock held by the executive officers named in the Summary Compensation Table during the fiscal year ended December 31, 2006.

Pension Benefits

We do not have any qualified or non-qualified defined benefit plans.

Nonqualified Deferred Compensation

We do not have any non-qualified defined contribution plans or other deferred compensation plans.

Potential Payments Upon Termination or Change of Control

We have entered into certain agreements and maintain certain plans that may require us to make certain payments and/or provide certain benefits to the executive officers named in the Summary Compensation Table in the event of a termination of employment or a change of control. The following tables summarize the potential payments to each named executive officer assuming that one of the following events occurs. The tables assume that the event occurred on December 31, 2006, the last day of our fiscal year. We have assumed a price per share of our common stock of $10.00, which represents the initial public offering price of our common stock on February 6, 2007.

Termination of Employment and Change of Control Arrangements

Change of Control Arrangements Under Our 2001 Stock Plan

Under our 2001 Stock Plan, in the event of a termination of our outstanding options in connection with a corporate transaction, where outstanding options are not assumed or substituted, all outstanding options shall become fully exercisable immediately prior to their termination. In addition, in the event of a change of control, as defined below, where outstanding options are assumed or substituted or in the event of a change of control that does not constitute a corporate transaction under our 2001 Stock Plan, all outstanding options will become immediately exercisable in full and all rights of repurchase with respect to outstanding stock grants shall terminate if on or prior to the date that is six months after the date of the change of control event (i) a participant's service with us or our succeeding corporation is terminated by us or the succeeding corporation without cause, as defined below; (ii) a participant terminates his or her service with us as a result of being required to change the principal location where he or she renders services to a location more than 50 miles from his or her location of service immediately prior to the change of control event; or (iii) the participant terminates his or her service after there occurs a material adverse change in a participant's duties, authority or responsibilities which cause such participant's position with us to become of significantly less responsibility or authority than such participant's position was immediately prior to the change of control.

Under our 2001 Stock Plan, a "*change of control*" means the occurrence of any of the following events:

(i) Ownership. Any "Person" (as such term is used in Sections 13(d) and 14(d) of the Securities Exchange Act of 1934, as amended) becomes the "Beneficial Owner" (as defined in Rule 13d-3 under said Act), directly or indirectly, of our securities representing 50% or more of the total voting power represented by our then outstanding voting securities (excluding for this purpose any such voting securities held by us or our affiliates or by any of our employee benefit plans) pursuant to a transaction or a series of related transactions which the board of directors does not approve; or

(ii) Merger/Sale of Assets. (A) A merger or consolidation of us whether or not approved by the board of directors, other than a merger or consolidation which would result in our voting securities outstanding immediately prior thereto continuing to represent (either by remaining outstanding or by being converted into voting securities of the surviving entity or the parent of such corporation) at least 50% of the total voting power represented by our voting securities or such surviving entity or parent of such corporation, as the case may be, outstanding immediately after such merger or consolidation; (B) or our stockholders approve an agreement for the sale or disposition by us of all or substantially all of our assets; or

(iii) Change in Board Composition. A change in the composition of the board of directors, as a result of which fewer than a majority of the directors are Incumbent Directors. "Incumbent Directors" shall mean directors who either (A) are directors as of January 11, 2005, or (B) are elected, or nominated for election, to the board of directors with the affirmative votes of at least a majority of the Incumbent Directors at the time of such election or nomination (but shall not include an individual whose election or nomination is in connection with an actual or threatened proxy contest relating to the election of directors).

Under our 2001 Stock Plan, "*cause*" shall include (and is not limited to) dishonesty with respect to us or any affiliate, insubordination, substantial malfeasance or non-feasance of duty, unauthorized disclosure of confidential information, and conduct substantially prejudicial to our business or any affiliate. "Cause" is not limited to events which have occurred prior to a participant's termination of service, nor is it necessary that the finding of "cause" occur prior to termination. If it is determined,

subsequent to a participant's termination of service but prior to the exercise of an option, that either prior or subsequent to the participant's termination the participant engaged in conduct which would constitute "cause", then the right to exercise any option is forfeited. Any definition in an agreement between the participant and us or an affiliate, which contains a conflicting definition of "cause" for termination and which is in effect at the time of such termination, shall supersede this definition with respect to such participant.

The tables below reflect the acceleration of options and the lapsing of repurchase rights for unvested options and shares of restricted common stock outstanding as of December 31, 2006.

Dr. Safi Bahcall, President and Chief Executive Officer

Executive Benefits and Payments Upon Termination	Termination in Connection with a Change of Control	Involuntary Not for Cause Termination
Base Salary	$680,000	$680,000
Acceleration of Vesting of Equity	100%	—
Number of Stock Options and Value upon Termination(1)	68,750 shares $—	—
Number of Shares of Vested Stock Received and Value upon Termination(1)	50,000 shares $499,980	—
Total:	$1,179,980	$680,000

(1) Value upon termination is calculated using a value for our common stock of $10.00 per share, the initial public offering price of our common stock on February 6, 2007.

Pursuant to our employment agreement with Dr. Bahcall, in the event of termination without cause, Dr. Bahcall is entitled to continue to receive his then-current base salary for a period of 24 months. As a condition to the receipt of the aforementioned severance payments, Dr. Bahcall will be required to execute and deliver a written release of Synta from any and all claims arising in connection with his employment. Dr. Bahcall has also entered into a non-competition/non-solicitation agreement pursuant to which he has agreed not to compete with Synta or to solicit customers or employees of Synta for a period of 24 months after the termination of his employment.

Pursuant to the terms of the employment agreement, a termination of Dr. Bahcall "*without cause*" shall include, but not be limited to, Dr. Bahcall's resignation following a significant and material diminution in title, salary, duties or responsibilities by us. The preceding sentence notwithstanding, "cause" shall include (but is not limited to): (i) any substantial malfeasance or non-feasance of duty, (ii) any material breach by Dr. Bahcall of any of the terms of the Confidential Information Agreement and Non-Competition Agreement between him and us, (iii) any attempt by Dr. Bahcall to secure any improper personal profit in connection with our business or any of our affiliates, (iv) Dr. Bahcall's conviction, or the entry of a pleading of guilty or nolo contendre to, any crime involving moral turpitude or any felony, or (v) any conduct substantially injurious or prejudicial to our business or that of our affiliates.

Our Other Named Executive Officers

Payments for Termination in Connection with a Change of Control Under Our 2001 Stock Plan. Pursuant to our 2001 Stock Plan, the other executive officers named in the Summary Compensation Table would receive the following in the event of a termination in connection with a change of control, as defined above:

	Keith S. Ehrlich, C.P.A.	James G. Barsoum, Ph.D.	Eric W. Jacobson, M.D.	Keizo Koya, Ph.D.
Acceleration of Vesting of Equity:	100%	100%	100%	100%
Number of Stock Options and Value upon Termination(1)	36,137 shares $ —	39,708 shares $ —	41,785 shares $ —	41,360 shares $ —
Number of Shares of Vested Stock Received and Value upon Termination(1)	25,000 shares $ 249,990	40,000 shares $ 399,984	25,000 shares $ 249,990	40,000 shares $ 399,984
Total:	$ 249,990	$ 399,984	$ 249,990	$ 399,984

(1) Value upon termination is calculated using a value for our common stock of $10.00 per share, the initial public offering price of our common stock on February 6, 2007.

Payment to Dr. Barsoum for Termination Without Cause. Pursuant to our letter agreement with Dr. Barsoum, in the event of termination without cause, Dr. Barsoum is entitled to a one-time severance payment on the date of termination equal to three months of base pay, which as of December 31, 2006, would equal $57,000. Pursuant to the terms of our letter agreement with Dr. Barsoum, " *cause*" means (i) an act of dishonesty demonstrating lack of integrity or moral turpitude, (ii) willful or persistent inattention to the services and duties required in connection with his employment, including failure to comply with all applicable laws and regulations after notice and failure to cure within 30 days or (iii) conviction of any felonious criminal act.

Director Compensation

The following table sets forth a summary of the compensation earned by our directors and/or paid to certain of our directors pursuant to certain agreements we have with them in 2006, other than Dr. Bahcall:

Name	Fees Earned or Paid in Cash ($)	Stock Awards ($)(1)	Option Awards ($)(2)	All Other Compensation ($)	Total ($)
Keith R. Gollust(3)	20,000(4)	40,000(5)	48,345(6)	—	108,345
Lan Bo Chen, Ph.D.(7)	—	—	—	400,000(8)	400,000
Judah Folkman, M.D.(9)	20,000(10)	20,000(11)	52,710(12)	25,000(13)	117,710
Bruce Kovner(14)	10,000(4)	40,000(5)	—	—	50,000
William S. Reardon, C.P.A.(15)	45,000(16)	10,000(17)	—(18)	—	55,000
Robert N. Wilson(19)	20,000(4)	40,000(5)	—	—	60,000

(1) See Notes 2 and 6 to our audited consolidated financial statements included elsewhere in this Annual Report on Form 10-K for details as to the assumptions used to determine the fair value of the stock awards. See also our discussion of stock-based compensation under Item 7 "Management's Discussion and Analysis of Financial Condition and Results of Operations—Critical Accounting Policies and Estimates."

(2) See Note 2 to our audited consolidated financial statements included elsewhere in this Annual Report on Form 10-K for details as to the assumptions used to determine the fair value of the option awards. Our non-employee directors will not realize the value of these awards in cash until these awards are exercised and the underlying shares are subsequently sold. See also our discussion of stock-based compensation under Item 7 "Management's Discussion and Analysis of Financial Condition and Results of Operations —Critical Accounting Policies and Estimates."

(3) As of December 31, 2006, the last day of our fiscal year, there are outstanding 5,584 shares of restricted stock, 4,155 of which are vested, and options for the purchase of 200,000 shares of common stock, all of which are vested, issued to Mr. Gollust.

(4) Represents fees paid for committee service during the fiscal year ended December 31, 2006.

(5) Consists of $20,000 and $20,000, representing the compensation expense incurred by us in fiscal year 2006 in connection with issuances of 1,818 shares of restricted common stock on October 14, 2005 as the elected form of payment for board service from July 1, 2005 through June 30, 2006, calculated in accordance with SFAS 123, all of which are vested, and 2,857 shares of restricted common stock on November 17, 2006 as the elected form of payment for board service from July 1, 2006 through June 30, 2007, calculated in accordance with SFAS 123R, 50% of which are vested and the remainder of which are subject to our repurchase right, which lapses as to 25% of the shares on each of March 31, 2007 and June 30, 2007, respectively. Each non-employee director paid $.0004 per share, the par value of our common stock. The grant date fair value of the restricted stock award on November 17, 2006 was $40,000, calculated in accordance with SFAS 123R. The number of shares issued was calculated by dividing $40,000 by the fair value of our common stock of $14.00 per share as of July 1, 2006.

(6) Represents the compensation expense incurred by us in fiscal year 2006, calculated in accordance with APB Opinion No. 25, in connection with an option grant to Mr. Gollust on May 27, 2004 for the purchase of 75,000 shares of common stock at an exercise price of $10.843 per share, which was below the then present fair market value of $16.00 per share.

(7) As of December 31, 2006, the last day of our fiscal year, we have not granted Dr. Chen any stock or option awards.

(8) Consists of $300,000 in fees paid in 2006 for consulting services pursuant to our consulting agreement with Dr. Chen and $100,000 in payments remitted in 2006 pursuant to our agreement and release with Dr. Chen, both of which agreements are further described below.

(9) As of December 31, 2006, the last day of our fiscal year, there are outstanding 2,857 shares of restricted stock, 1,428 of which are vested, and options for the purchase of 40,000 shares of common stock, 25,937 of which are vested, issued to Dr. Folkman. Dr. Fokman has transferred all right, title and interest in these options to Children's Medical Center Corporation pursuant to stock option transfer agreements in which Children's Medical Center Corporation has agreed to be subject to all of the conditions and restrictions under the options.

(10) Represents the elected form of payment for board service from January 1, 2006 through June 30, 2006.

(11) Represents the compensation expense incurred by us in fiscal year 2006 in connection with the issuance of 2,857 shares of restricted common stock on November 17, 2006 as the elected form of payment for board service from July 1, 2006 through June 30, 2007, calculated in accordance with SFAS 123R, 50% of which are vested and the remainder of which are subject to our repurchase right, which lapses as to 25% of the shares on each of March 31, 2007 and June 30, 2007. Dr. Folkman paid $.0004 per share, the par value of our common stock. The grant date fair value of the restricted stock award on November 17, 2006 was $40,000, calculated in accordance with

SFAS 123R. The number of shares issued was calculated by dividing $40,000 by the fair value of our common stock of $14.00 per share as of July 1, 2006.

(12) Represents the compensation expense incurred by us in fiscal year 2006 in connection with an option grant to Dr. Folkman to purchase 15,000 shares of common stock on September 15, 2005 at an original exercise price of $22.00 per share, calculated in accordance with SFAS 123. Effective March 1, 2006, this option was repriced to an exercise price of $14.00. The incremental fair value associated with this modification, in the amount of $17,280, was calculated in accordance with SFAS 123R. On October 17, 2005, Dr. Folkman transferred all right, title and interest in these options to Children's Medical Center Corporation pursuant to a stock option transfer agreement in which Children's Medical Center Corporation has agreed to be subject to all of the conditions and restrictions under the options.

(13) Represents fees paid in 2006 for consulting services pursuant to our scientific advisory board agreement with Dr. Folkman, further described below.

(14) As of December 31, 2006, the last day of our fiscal year, there are outstanding 5,584 shares of restricted stock, 4,155 of which are vested, and options for the purchase of 54,687 shares of common stock, all of which are vested, issued to Mr. Kovner.

(15) As of December 31, 2006, the last day of our fiscal year, there are outstanding 1,622 shares of restricted stock, 1,265 of which are vested, and options for the purchase of 15,000 shares of common stock, 8,437 of which are vested, issued to Mr. Reardon.

(16) Consists of $15,000 in fees paid for committee service during the fiscal year ended December 31, 2006 and $30,000 as the elected form of payment for board service from January 1, 2006 through December 31, 2006.

(17) Consists of $5,000 and $5,000, representing the compensation expense incurred by us in fiscal year 2006 in connection with issuances of 454 shares of restricted common stock on October 14, 2005 as the elected form of payment for board service from July 1, 2005 through June 30, 2006, calculated in accordance with SFAS 123, all of which are vested, and 714 shares of restricted common stock on November 17, 2006 as the elected form of payment for board service from July 1, 2006 through June 30, 2007, calculated in accordance with SFAS 123R, 50% of which are vested and the remainder of which are subject to our repurchase right, which lapses as to 25% of the shares on each of March 31, 2007 and June 30, 2007, respectively. Mr. Reardon paid $.0004 per share, the par value of our common stock. The grant date fair value of the restricted stock award on November 17, 2006 was $10,000, calculated in accordance with SFAS 123R. The number of shares issued was calculated by dividing $10,000 by the fair value of our common stock of $14.00 per share as of July 1, 2006.

(18) On August 25, 2004, we granted Mr. Reardon an option to purchase 15,000 shares of common stock at an original exercise price of $16.00 per share. Effective March 1, 2006, this option was repriced to an exercise price of $14.00. The incremental fair value associated with this modification, in the amount of $4,920, was calculated in accordance with SFAS 123R.

(19) As of December 31, 2006, the last day of our fiscal year, there are outstanding 5,584 shares of restricted stock, 4,155 of which are vested, and options for the purchase of 62,500 shares of common stock, 54,687 of which are vested, issued to Mr. Wilson.

Director Compensation Policy

We reimburse each member of our board of directors who is not an employee for reasonable travel and other expenses in connection with attending meetings of the board of directors.

In January 2005, our board of directors approved our Director Compensation Policy. Pursuant to this policy, each non-employee director receives an option to purchase 15,000 shares of our common stock upon his or her initial appointment to our board of directors. These options vest as to 25% of such grant on the first anniversary of the grant date and as to an additional 6.25% of such grant on the last day of each calendar quarter thereafter, subject to the non-employee director's continued service as a director. However, in the event of termination of service of a non-employee director, such option will vest to the extent of a pro rata portion through the non-employee director's last day of service based on the number of days accrued in the applicable period prior to his or her termination of service. Each non-employee director stock option will terminate on the earlier of ten years from the date of grant and three months after the recipient ceases to serve as a director, except in the case of death or disability, in which event the option will terminate one year from the date of the director's death or disability. The exercise price of these options is equal to the fair market value of our common stock on the date of grant.

Under this policy, each non-employee director is compensated on an annual basis for providing services to Synta. Director compensation is paid for the period from July 1 through June 30 of each year. Each non-employee director receives compensation consisting of one of the following combinations of cash and/or a grant of our common stock, at the election of each non-employee director, as follows:

- $40,000 cash;
- $30,000 cash and such number of shares of restricted common stock with a value of $10,000 on the date of grant of the shares;
- $20,000 cash and such number of shares of restricted common stock with a value of $20,000 on the date of grant of the shares;
- $10,000 cash and such number of shares of restricted common stock with a value of $30,000 on the date of grant of the shares; or
- such number of shares of restricted common stock with a value of $40,000 on the date of grant of the shares.

The number of shares to be received by a non-employee director is calculated by dividing the total dollar amount that the non-employee director has elected to be paid in shares of common stock by the fair market value of the shares of our common stock on the last business day prior to the date of grant of the shares. Shares granted are subject to a lapsing repurchase right such that the shares are subject to forfeiture to us if a non-employee director does not continue to serve as a member of the board of directors as of the end of the applicable quarter as follows: the repurchase right lapses as to 25% of each such grant on each of September 30, December 31, March 31 and June 30 thereafter, provided such non-employee director continues to serve as a member of the board of directors as of the applicable date.

The option and restricted stock awards disclosed in the above Director Compensation Table and accompanying footnotes that were granted prior to March 15, 2006, were granted under our 2001 Stock Plan, with the exception of the option grant to Mr. Gollust on May 27, 2004, which was not granted pursuant to an equity plan. On March 15, 2006, our board of directors adopted and our stockholders approved our 2006 Stock Plan and at the same time terminated our 2001 Stock Plan. The restricted stock awards granted on November 17, 2006 were granted under our 2006 Stock Plan.

Pursuant to the Director Compensation Policy, each non-employee director also receives an annual fee of $5,000 for each committee of the board of directors on which such individual serves. However, the chairman of each committee, other than the audit committee, receives an annual fee of $10,000, and the chairman of the audit committee receives an annual fee of $15,000 for services as chairman.

Consulting Agreement with Dr. Lan Bo Chen

In 2002, we entered into an oral consulting agreement with Dr. Chen pursuant to which Dr. Chen provided consulting services as mutually determined by us and Dr. Chen from time to time. This consulting agreement had no definitive term. Under the terms of the agreement, we provided compensation to Dr. Chen of $25,000 per month. Dr. Chen was paid $75,000, $300,000, $300,000 and $87,500 in 2002, 2003, 2004 and 2005, respectively, under this arrangement. In April 2005, we entered into a written consulting agreement with Dr. Chen pursuant to which he has agreed to provide consulting services to us and to serve as the chairman and/or a member of our scientific advisory board. This written agreement supersedes the aforementioned oral agreement. Under the terms of this agreement, we pay Dr. Chen $25,000 per month for these services. We are currently in discussions with Dr. Chen to amend this agreement to reduce the amount payable to Dr. Chen to $10,000 per month. This written agreement has no definitive term and may be terminated by us or Dr. Chen upon 15 days advance written notice. The agreement also contains a one-year post termination non-competition and non-solicitation provision. We paid Dr. Chen $212,500 in 2005 and $300,000 in 2006 under this agreement.

Agreement and Release with Dr. Lan Bo Chen

In January 2005, we entered into an Agreement and Release with Dr. Chen whereby we resolved all outstanding matters regarding various oral understandings and arrangements between Dr. Chen and Synta, including arrangements relating to (1) the assignment by Dr. Chen of the benefit of his interests, if any, resulting from our acquisition of the assets of Cancer Genomics, Inc., Kava Pharmaceuticals, Inc., and SinglePixel Biomedical, Inc., (2) Dr. Chen's assignment of inventions, non-competition, non-solicitation and confidentiality agreements with us, and (3) a general release by Dr. Chen of any and all claims that Dr. Chen may have had against us. Pursuant to this agreement we will pay Dr. Chen $500,000 payable in $25,000 installments quarterly for five years. We paid Dr. Chen $100,000 in each of 2005 and 2006 under this agreement.

Scientific Advisory Board Agreement with Dr. Judah Folkman

In September 2003, we entered into a scientific advisory board agreement with Dr. Folkman pursuant to which Dr. Folkman provides consulting services as mutually determined by us and Dr. Folkman from time to time, up to a maximum of five days per month. This agreement had an initial term of one year and provides for automatic one-year extensions. The agreement may be terminated by us or Dr. Folkman for any reason upon 60 days advance written notice and may be immediately terminated in the event of a breach or threatened breach of certain provisions contained in the agreement. Under the terms of this agreement, we agreed to pay Dr. Folkman a consulting fee of $50,000 per year payable in quarterly installments and reimburse his reasonable expenses incurred in connection with his performance under the agreement. Pursuant to this agreement, Dr. Folkman was paid $25,000, $50,000, $50,000 and $25,000 in 2003, 2004, 2005 and 2006, respectively. Under this agreement, Dr. Folkman has also been granted a non-qualified stock option to purchase 25,000 shares of common stock at an exercise price of $10.843 per share. This option vests as to 25% of the shares on the first anniversary of the grant date and an additional 6.25% of the shares at the end of each successive three-month period thereafter, provided that the scientific advisory board agreement remains in effect on the date of vesting. Immediately following the grant of this option, Dr. Folkman transferred all right, title and interest in this option to Children's Medical Center Corporation pursuant to a stock

option transfer agreement in which Children's Medical Center Corporation has agreed to be subject to all of the conditions and restrictions under the option. Pursuant to the terms of the scientific advisory board agreement, we have agreed to indemnify Dr. Folkman and Children's Hospital Boston, its corporate affiliates, current or future directors, trustees, officers, faculty, medical and professional staff, employees, students and agents and their respective successors, heirs and assigns against liability incurred in connection with claims arising out of the agreement, except to the extent caused by Dr. Folkman's misconduct or negligence. Pursuant to an oral agreement between Dr. Folkman and us entered into on or about September 30, 2006, and in connection with Dr. Folkman's appointment to our board of directors in September 2005, Dr. Folkman will no longer receive compensation under this agreement.

Compensation Committee Interlocks and Insider Participation

Our compensation committee is composed of Messrs. Gollust, Kovner and Wilson. No member of our compensation committee has at any time been an employee of ours. None of our executive officers serve as a member of the board of directors or compensation committee of any entity that has one or more executive officers serving as a member of our board of directors or compensation committee.

Each of Messrs. Gollust, Kovner and Wilson and affiliates of theirs have participated in transactions with us. For a detailed description of these transactions, see Item 13 "Certain Relationships and Related Transactions and Director Independence."

Item 12. SECURITY OWNERSHIP OF CERTAIN BENEFICIAL OWNERS AND MANAGEMENT AND RELATED STOCKHOLDER MATTERS

The following table sets forth certain information regarding the beneficial ownership of our common stock as of March 15, 2007 by:

- the executive officers named in the summary compensation table;
- each of our directors;
- all of our current directors and executive officers as a group; and
- each stockholder known by us to own beneficially more than five percent of our common stock.

Beneficial ownership is determined in accordance with the rules of the Securities and Exchange Commission and includes voting or investment power with respect to the securities. Shares of common stock that may be acquired by an individual or group within 60 days of March 15, 2007, pursuant to the exercise of options or warrants, are deemed to be outstanding for the purpose of computing the percentage ownership of such individual or group, but are not deemed to be outstanding for the purpose of computing the percentage ownership of any other person shown in the table. Percentage of ownership is based on 33,816,532 shares of common stock outstanding on March 15, 2007.

Except as indicated in footnotes to this table, we believe that the stockholders named in this table have sole voting and investment power with respect to all shares of common stock shown to be beneficially owned by them, based on information provided to us by such stockholders. Unless otherwise indicated, the address for each director and executive officer listed is: c/o Synta Pharmaceuticals Corp., 45 Hartwell Avenue, Lexington, Massachusetts 02421.

Beneficial Owner	Number of Shares Beneficially Owned	Percentage of Common Stock Beneficially Owned
Directors and Executive Officers		
Safi R. Bahcall, Ph.D.(1)	2,282,243	6.7%
Keith S. Ehrlich, C.P.A.(2)	65,265	*
Keizo Koya, Ph.D.(3)	234,609	*
James G. Barsoum, Ph.D.(4)	140,559	*
Eric W. Jacobson, M.D.(5)	46,463	*
Keith R. Gollust(6)	1,615,327	4.7%
Lan Bo Chen, Ph.D.(7)	3,462,892	10.2%
Judah Folkman, M.D.(8)	2,857	*
Bruce Kovner(9)	8,648,005	25.5%
William S. Reardon, C.P.A.(10)	10,997	*
Robert N. Wilson(11)	404,650	1.1%
All current executive officers and directors as a group (14 persons)(12)	17,124,328	49.3%

Five Percent Stockholders		
CxSynta LLC(13) c/o Caxton Corporation Princeton Plaza, Building 2 731 Alexander Road Princeton, NJ 08540	7,761,716	22.9%
Lin-Huey Chen(14) 184 East Emerson Road Lexington, MA 02420	3,462,892	10.2%
Luxor Capital Group, LP(15) 767 Fifth Avenue, 19th Floor New York, NY 10153	2,119,889	6.3%

* Represents beneficial ownership of less than 1% of the shares of common stock.

(1) Consists of 2,245,525 shares of common stock owned of record by and 36,718 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Dr. Bahcall. The amount excludes an aggregate of 110,000 shares of common stock of which 15,000 shares are owned of record by the Safi R. Bahcall Irrevocable Trust, the trustee of which is Dr. Bahcall's mother and of which Dr. Bahcall is the beneficiary; 24,250 shares are owned of record by the 2004 Neta Bahcall Grantor Retained Annuity Trust, the trustee of which is Dr. Bahcall and of which Dr. Bahcall is a beneficiary; 40,750 shares are owned of record by the 2006 Neta Bahcall Grantor Retained Annuity Trust, the trustee of which is Dr. Bahcall's sister and of which Dr. Bahcall is a beneficiary; 15,000 shares are owned of record by the Dan O. Bahcall Irrevocable Trust, the trustee of which is Dr. Bahcall's mother and of which Dr. Bahcall's brother is the beneficiary; and 15,000 shares are owned of record by the Orli G. Bahcall Irrevocable Trust, the trustee of which is Dr. Bahcall's mother and of which Dr. Bahcall's sister is the beneficiary. Dr. Bahcall disclaims beneficial ownership of the shares held by these trusts except to the extent of any pecuniary interest therein.

(2) Consists of 20,426 shares of common stock owned of record by and 44,839 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Mr. Ehrlich.

(3) Consists of 33,045 shares of common stock owned of record by and 201,564 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Dr. Koya.

(4) Consists of 40,000 shares of common stock owned of record by and 100,559 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Dr. Barsoum.

(5) Consists of 25,300 shares of common stock owned of record by and 21,163 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Dr. Jacobson.

(6) Consists of 34,357 shares of common stock owned of record by and 200,000 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Mr. Gollust; 50,000 shares of common stock owned of record by the Gollust Trust II, a trust established for the benefit of Mr. Gollust's minor children; and 1,330,970 shares of common stock owned of record by Wyandanch Partners, L.P. Mr. Gollust is the president and sole stockholder of Gollust Management, Inc., which is the general partner of Wyandanch Partners, L.P.

(7) Consists of 742,024 shares of common stock owned of record by Dr. Chen; 55,071 shares of common stock owned of record by the Lan Bo Chen 2004 GRAT, the beneficiaries of which are Dr. Chen's two daughters; 142,223 shares of common stock owned of record by LAJ Holdings LLC, the co-managers of which are Dr. Chen and his spouse; 2,004,016 shares of common stock owned of record by the Wisteria Trust, the trustee of which is Dr. Chen's spouse; 243,481 shares of common stock owned of record by the Ann Chen Trust, a co-trustee of which is Dr. Chen's spouse; 243,481 shares of common stock owned of record by the Jane Chen Trust, a co-trustee of which is Dr. Chen's spouse; 12,946 shares of common stock owned of record by the Chen Grandchildren's Trust, a co-trustee of which is Dr. Chen's spouse; 6,950 shares of common stock owned of record by the Alexander Chen Wu 2002 Irrevocable Trust, a co-trustee of which is Dr. Chen's spouse; an aggregate of 9,900 shares of common stock owned of record by Dr. Chen's two daughters and their husbands; and 2,800 shares of common stock owned of record by the Allison Chen Wu 2004 Irrevocable Trust, a co-trustee of which is Dr. Chen's spouse. See note 14.

(8) Represents shares of common stock owned of record by Dr. Folkman.

(9) Consists of 831,602 shares of common stock owned of record by and 54,687 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Mr. Kovner; and 7,761,716 shares of common stock owned of record by CxSynta LLC. Caxton Corporation is the managing member of CxSynta LLC and Bruce Kovner is the chairman of Caxton Corporation. See note 13.

(10) Consists of 1,622 shares of common stock owned of record by and 9,375 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Mr. Reardon.

(11) Consists of 346,057 shares of common stock owned of record by and 58,593 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by Mr. Wilson.

(12) Consists of the shares of common stock set forth in footnotes 1 through 11 and 41,052 shares of common stock owned of record by and 169,409 shares of common stock issuable upon the exercise of options exercisable within 60 days of March 15, 2007 held by three executive officers not named in the table.

(13) Represents shares of common stock owned of record by CxSynta LLC. Caxton Corporation is the managing member of CxSynta LLC and Bruce Kovner is the chairman of Caxton Corporation. See note 9.

(14) Consists of 742,024 shares of common stock owned of record by Ms. Chen's spouse, Dr. Chen; 55,071 shares of common stock owned of record by the Lan Bo Chen 2004 GRAT, the granter of which is Ms. Chen's spouse and the beneficiaries of which are Dr. Chen's two daughters; 142,223 shares of common stock owned of record by LAJ Holdings LLC, of which Ms. Chen is a manager; 2,004,016 shares of common stock owned of record by the Wisteria Trust, of which Ms. Chen is the trustee; 243,481 shares of common stock owned of record by the Ann Chen Trust, of which Ms. Chen is a co-trustee; 243,481 shares of common stock owned of record by the Jane Chen Trust, of which Ms. Chen is a co-trustee; 12,946 shares of common stock owned of record by the Chen Grandchildren's Trust, of which Ms. Chen is a co-trustee; 6,950 shares of common stock owned of record by the Alexander Chen Wu 2002 Irrevocable Trust, of which Ms. Chen is a co-trustee; an aggregate of 9,900 shares of common stock owned of record by Ms. Chen's two daughters and their husbands; and 2,800 shares of common stock owned of record by the Allison Chen Wu 2004 Irrevocable Trust, of which Ms. Chen is a co-trustee. See note 7.

(15) Consists of 953,591 shares held of record by Luxor Capital Partners, LP ("Luxor Onshore") and 1,166,298 shares held of record by Luxor Capital Partners Offshore, Ltd. ("Luxor Offshore").

Luxor Capital Group is a registered investment advisor and acts as the investment manager of Luxor Onshore and Luxor Offshore. Luxor Management, LLC is the General Partner of Luxor Capital Group, and LCH Holdings, LLC is the General Partner of Luxor Onshore. Christian Leone is the managing member of both Luxor Management, LLC and LCG Holdings, LLC.

Equity Compensation Plan Information

The following table provides certain aggregate information with respect to all of the Company's equity compensation plans in effect as of December 31, 2006:

Plan Category	Number of Securities to be Issued Upon Exercise of Outstanding Options (1)	Weighted Average Exercise Price of Outstanding Options ($)	Number of Securities Remaining Available for Future Issuance Under Equity Compensation Plans (excluding securities reflected in first column)
Equity Compensation Plans Approved by Securityholders			
2001 Stock Plan	2,900,318	11.87	0
2006 Stock Plan	67,750	14.00	2,326,356
Equity Compensation Plans not Approved by Securityholders(2)	75,000	10.84	0
Total	3,043,068	11.88	2,326,356

(1) Includes options to purchase 21,239 shares of our common stock issued under our 2001 Stock Plan and 2006 Stock Plan that were cancelled after December 31, 2006.

(2) Represents a stand alone option agreement we have with our current director Keith Gollust.

Summary Description of Our Stockholder Approved Equity Compensation Plans

2001 Stock Plan

Our 2001 Stock Plan was adopted by our board of directors and approved by our stockholders in July 2001. In August 2002 and December 2003, our board of directors and stockholders approved amendments to our 2001 Stock Plan and in January 2005 and May 2005, our board of directors amended our 2001 Stock Plan. Under this plan, we granted incentive stock options, nonqualified stock options and restricted and unrestricted stock awards. A maximum of 3,750,000 shares of common stock were authorized for issuance under our 2001 Stock Plan. In March 2006, we terminated the 2001 Stock Plan. All outstanding stock options granted and restricted stock issued under the 2001 Stock Plan as of the date of termination remained outstanding and subject to their respective terms and the terms of the 2001 Stock Plan. No shares are available for future grant under this plan.

In accordance with the terms of the 2001 Stock Plan, our board of directors has authorized our compensation committee to administer our 2001 Stock Plan. In February 2005, the board of directors delegated authority to an option committee of the board of directors comprised of our President and Chief Executive Officer, Safi Bahcall, to grant options to purchase up to a total of 125,000 shares of our common stock. The board of directors intended that this share pool would be used primarily to grant options to new hires and that the number of shares the option committee had authority to grant would be periodically replenished. Due to administrative error, the board of directors did not take action to replenish the pool of options the option committee had authority to grant, and the option committee granted options to purchase a total of 225,750 shares of common stock. The grant of options

in excess of the shares the option committee had authority to grant were ratified by the compensation committee in November 2006.

Our board of directors or any committee to which the board of directors delegates authority may, with the consent of the affected plan participants, amend outstanding awards consistent with the terms of the 2001 Stock Plan.

Upon a merger or other reorganization event, our board of directors, may, in their sole discretion, take any one or more of the following actions pursuant to our 2001 Stock Plan, as to some or all outstanding options:

- provide that all options shall be assumed or substituted by the successor corporation;
- upon written notice to a participant, provide that the participant's unexercised options will become exercisable in full and will terminate immediately prior to the consummation of such transaction unless exercised by the participant;
- in the event of a merger pursuant to which holders of our common stock will receive a cash payment for each share surrendered in the merger, make or provide for a cash payment to the participants equal to the difference between the merger price times the number of shares of our common stock subject to such outstanding options (at prices not in excess of the merger price), and the aggregate exercise price of all such outstanding options (all options being made fully vested and immediately exercisable prior to their termination), in exchange for the termination of such options; and
- provide that outstanding awards shall be assumed or substituted by the successor corporation, become realizable or deliverable, or restrictions applicable to an award will lapse, in whole or in part, prior to or upon the merger or reorganization event.

In addition, in the event of a change in control under our 2001 Stock Plan where outstanding options are assumed or substituted or in the event of a change in control that does not constitute a corporate transaction under our 2001 Stock Plan, options will become immediately exercisable in full if on or prior to the date that is six months after the date of the change in control (i) an option holder's service with us or our succeeding corporation is terminated by us or the succeeding corporation without cause, as defined in our 2001 Stock Plan; (ii) a participant terminates his or her service with us as a result of being required to change the principal location where he or she renders services to a location more than 50 miles from his or her location of service immediately prior to the change in control; or (iii) the participant terminates his or her service after there occurs a material adverse change in a participant's duties, authority or responsibilities which cause such participant's position with us to become of significantly less responsibility or authority than such participant's position was immediately prior to the change in control. Our 2001 Stock Plan provides similar change in control vesting provisions for restricted stock under the plan.

In February 2006, our board of directors approved the repricing of options issued under our 2001 Stock Plan having an exercise price at or above $16.00 per share to $14.00 per share, the fair market value of our common stock on the date of the repricing, as determined by our board of directors. This repricing applied to all outstanding options under this plan held by our active employees and others having an ongoing relationship with us at the time of the repricing. The repricing was effective on March 1, 2006.

2006 Stock Plan

Our 2006 Stock Plan was adopted by our board of directors in March 2006 and approved by our stockholders in March 2006 and amended in January 2007. The 2006 Stock Plan provides for the grant of incentive stock options, nonqualified stock options, restricted and unrestricted stock awards and

other stock-based awards. There are 2,500,000 shares of common stock currently reserved for issuance under the 2006 Stock Plan. In addition, the 2006 Stock Plan contains an "evergreen provision" which allows for an annual increase in the number of shares available for issuance under the plan on the first day of each of our fiscal years during the period beginning in fiscal year 2008 and ending on the second day of fiscal year 2016. The annual increase in the number of shares shall be equal to the lowest of

- 1,300,000 shares;
- 5% of our outstanding shares on the first day of the fiscal year; and
- an amount determined by our board of directors.

Under this provision, no annual increase shall be made to the extent that the number of shares of common stock available for issuance under the 2006 Stock Plan and all other employee or director stock plans would exceed 25% of our outstanding shares on the first day of the applicable fiscal year.

In accordance with the terms of the 2006 Stock Plan, our board of directors has authorized our compensation committee to administer our 2006 Stock Plan however, our full board of directors shall retain authority to make grants to our executive officers and members of our board of directors. In accordance with the provisions of the 2006 Stock Plan, our board of directors or compensation committee will determine the terms of options and other awards, including:

- the determination of which employees, directors and consultants will be granted options and other awards;
- the number of shares subject to options and other awards;
- the exercise price of each option which may not be less than fair market value on the date of grant;
- the schedule upon which options become exercisable;
- the termination or cancellation provisions applicable to options; the terms and conditions of other awards, including conditions for repurchase, termination or cancellation, issue price and repurchase price; and
- all other terms and conditions upon which each award may be granted in accordance with the 2006 Stock Plan.

No participant may receive awards for over 125,000 shares of common stock in any fiscal year.

In addition, our board of directors or any committee to which the board of directors delegates authority may, with the consent of the affected plan participants, reprice or otherwise amend outstanding awards consistent with the terms of the 2006 Stock Plan.

Upon a merger or other reorganization event, our board of directors, may, in their sole discretion, take any one or more of the following actions pursuant to our 2006 Stock Plan, as to some or all outstanding awards:

- provide that all options shall be assumed or substituted by the successor corporation;
- upon written notice to a participant, provide that the participant's unexercised options will become exercisable in full and will terminate immediately prior to the consummation of such transaction unless exercised by the participant;
- in the event of a merger pursuant to which holders of our common stock will receive a cash payment for each share surrendered in the merger, make or provide for a cash payment to the participants equal to the difference between the merger price times the number of shares of our common stock subject to such outstanding options (at prices not in excess of the merger price),

and the aggregate exercise price of all such outstanding options (all options being made fully vested and immediately exercisable prior to their termination), in exchange for the termination of such options; and

- provide that outstanding awards shall be assumed or substituted by the successor corporation, become realizable or deliverable, or restrictions applicable to an award will lapse, in whole or in part, prior to or upon the merger or reorganization event.

In addition, in the event of a change in control under our 2006 Stock Plan where outstanding options are assumed or substituted or in the event of a change in control that does not constitute a corporate transaction under our 2006 Stock Plan, options will become immediately exercisable in full if on or prior to the date that is six months after the date of the change in control (i) an option holder's service with us or our succeeding corporation is terminated by us or the succeeding corporation without cause, as defined in our 2006 Stock Plan; (ii) a participant terminates his or her service with us as a result of being required to change the principal location where he or she renders services to a location more than 50 miles from his or her location of service immediately prior to the change in control; or (iii) the participant terminates his or her service after there occurs a material adverse change in a participant's duties, authority or responsibilities which cause such participant's position with us to become of significantly less responsibility or authority than such participant's position was immediately prior to the change in control. Our 2006 Stock Plan provides similar change in control vesting provisions for restricted stock under the plan and allows the board of directors to make appropriate adjustments for other stock-based awards.

Summary Description of Our Non-Stockholder Approved Equity Compensation Plans

On May 24, 2004, we granted our current director Keith Gollust a non-qualified option to purchase 75,000 shares of our common stock at an exercise price of $10.843 per share. Pursuant to the option agreement, the option vested as to 50% of the shares upon grant and as to 6.25% of the shares at the end of each successive three-month period thereafter.

Item 13. CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS AND DIRECTOR INDEPENDENCE

Certain Relationships and Related Transactions

The following is a description of the transactions we have engaged in since January 1, 2006 with our directors and officers and beneficial owners of more than five percent of our voting securities and their affiliates.

Private Placement of Our Series A Convertible Preferred Stock

In June 2006, we issued an aggregate of 8,000,000 shares of our Series A convertible preferred stock to 42 investors at a purchase price of $5.00 per share, including an aggregate of 2,551,731 shares to the following directors, officers, and beneficial owners of more than five percent of our voting securities, and their affiliates:

Name	Number of Shares of Series A Preferred Stock	Aggregate Purchase Price
Robert N. Wilson	67,495	$ 337,475
Bruce Kovner	30,131	150,655
CxSynta, LLC	2,154,105	10,770,525
Wyandanch Partners, L.P.	300,000	1,500,000

Robert N. Wilson is one of our directors. Bruce Kovner is one of our directors. CxSynta, LLC is a beneficial owner of more than five percent of our voting securities and an affiliated investment vehicle

of the Caxton Corporation. Mr. Kovner is the Chairman of the Caxton Corporation. Keith R. Gollust, one of our directors, is the president and sole stockholder of Gollust Management, Inc., which is the general partner of Wyandanch Partners, LP. The purchase price per share was the fair market value as determined by arms-length negotiations between sophisticated investors and Synta's management and board of directors, based on factors such as our stage of development and valuations of similarly situated private biopharmaceutical companies.

Each share of Series A convertible preferred stock issued in our June 2006 private placement was automatically convertible upon the closing of an initial public offering into that number of shares of our common stock determined by dividing (i) the Series A per share purchase price of $5.00 plus accumulated dividends of 8% per year (which were calculated in arrears on the last day of each fiscal quarter with respect to the prior quarterly period but without prorating for any partially completed fiscal quarter) by (ii) a conversion price equal to the lesser of (a) $20.00 or (b) 66.6667% of the initial public offering price per share of our common stock. Upon closing of our initial public offering on February 9, 2007, the following directors, officers and beneficial owners of more than five percent of our voting securities, and their affiliates, received the following shares of our common stock as a result of the conversion of their shares of Series A convertible preferred stock and accrued dividends thereon:

Name	Number of Shares of Series A Preferred Stock Converted	Amount of Accrued Dividends Converted	Aggregate Number of Shares of Common Stock Received
Robert N. Wilson	67,495	$ 15,681	52,973
Bruce Kovner	30,131	7,000	23,648
CxSynta, LLC	2,154,105	500,461	1,690,646
Wyandanch Partners, L.P.	300,000	69,699	235,454

Participation in Initial Public Offering

In February 2007, we issued an aggregate of 5,000,000 shares of our common stock in connection with our initial public offering at an initial public offering price of $10.00 per share, including an aggregate of 1,000,000 shares to the following directors and beneficial owners of more than five percent of our voting securities, and their affiliates:

Name	Number of Shares of Common Stock	Aggregate Purchase Price
Robert N. Wilson	100,000	$ 1,000,000
Bruce Kovner	720,000	7,200,000
Wyandanch Partners, L.P.	180,000	1,800,000

The initial public offering price of $10.00 per share was determined through negotiations between us and the representatives of the underwriters of the offering based on several factors.

Investor Rights Agreement

Pursuant to an Amended and Restated Investor Rights Agreement dated December 31, 2002 by and among Synta and certain stockholders, as amended on January 11, 2005 and January 31, 2007, certain of our stockholders are entitled to registration rights with respect to the shares of common stock held by them. These shareholders include the following directors, beneficial owners of more than five percent of our voting securities, and their affiliates: CxSynta, LLC, Gollust Trust II, Wyandanch Partners, LP, Keith R. Gollust and Bruce Kovner.

Indemnification Arrangements

Our restated certificate of incorporation and restated bylaws to be effective upon completion of this offering provide that we will indemnify our directors and officers to the fullest extent permitted by Delaware law. In addition, we have entered into indemnification agreements with each of our directors and executive officers.

Policy for Approval of Related Person Transactions

Pursuant to the written charter of our audit committee as amended in January 2007, the audit committee is responsible for reviewing and approving, prior to our entry into any such transaction, all transactions in which we are a participant and in which any of the following persons has or will have a direct or indirect material interest:

- our executive officers;
- our directors;
- the beneficial owners of more than 5% of our securities;
- the immediate family members of any of the foregoing persons; and
- any other persons whom the board of directors determines may be considered related persons.

For purposes of these procedures, "immediate family members" means any child, stepchild, parent, stepparent, spouse, sibling, mother-in-law, father-in-law, son-in-law, daughter-in-law, brother-in-law, or sister-in-law, and any person (other than a tenant or employee) sharing the household with the executive officer, director or 5% beneficial owner.

In reviewing and approving such transactions, the audit committee shall obtain, or shall direct our management to obtain on its behalf, all information that the committee believes to be relevant and important to a review of the transaction prior to its approval. Following receipt of the necessary information, a discussion shall be held of the relevant factors if deemed to be necessary by the committee prior to approval. If a discussion is not deemed to be necessary, approval may be given by written consent of the committee. This approval authority may also be delegated to the chairman of the audit committee in some circumstances. No related person transaction shall be entered into prior to the completion of these procedures.

The audit committee or its chairman, as the case may be, shall approve only those related person transactions that are determined to be in, or not inconsistent with, the best interests of us and our stockholders, taking into account all available facts and circumstances as the committee or the chairman determines in good faith to be necessary. These facts and circumstances will typically include, but not be limited to, the benefits of the transaction to Synta; the impact on a director's independence in the event the related person is a director, an immediate family member of a director or an entity in which a director is a partner, shareholder or executive officer; the availability of other sources for comparable products or services; the terms of the transaction; and the terms of comparable transactions that would be available to unrelated third parties or to employees generally. No member of the audit committee shall participate in any review, consideration or approval of any related person transaction with respect to which the member or any of his or her immediate family members is the related person.

Director Independence

Our board of directors has reviewed the materiality of any relationship that each of our directors has with Synta, either directly or indirectly. Based on this review, the board of directors has determined that the following directors are "independent directors" as defined by The Nasdaq Stock Market, LLC: Messrs. Gollust, Kovner, Reardon and Wilson.

Item 14. PRINCIPAL ACCOUNTING FEES AND SERVICES

Accounting Fees and Services

The following table presents fees for professional audit services rendered by KPMG LLP, independent registered public accountants, for the audit of our annual financial statements for the years ended December 31, 2006 and December 31, 2005 and fees billed for other services rendered by KPMG LLP during those periods.

	2005	2006
Audit fees	$ 376,332	$ 365,000
Audit-related fees	—	15,000
Tax fees	13,900	15,000
All other fees	—	—
Total	$ 390,232	$ 395,000

Audit Fees

KPMG's fees for audit services totaled $365,000 and $376,332 for 2006 and 2005, respectively. Audit services were comprised of services associated with the 2006 and 2005 annual audits and registration statements.

Audit-Related Fees

KPMG's fees for audit-related services totaled $15,000 and $0 for 2006 and 2005, respectively. Audit-related services were comprised of an employee benefit plan audit.

Tax Fees

KPMG's fees for tax services totaled $15,000 and $13,900 for 2006 and 2005, respectively. Tax services were comprised of tax compliance, tax advice and tax planning services.

All Other Fees

KPMG did not have any fees for any other services for 2006 or 2005.

Policy on Audit Committee Pre-Approval of Audit and Permissible Non-audit Services of Independent Auditors

Consistent with policies of the Securities and Exchange Commission regarding auditor independence, the audit committee has responsibility for appointing, setting compensation and overseeing the work of the independent auditor. In recognition of this responsibility, the audit committee has established a policy to pre-approve all audit and permissible non-audit services provided by the independent auditor.

Prior to engagement of the independent auditor for the next year's audit, management will submit an aggregate of services expected to be rendered during that year for each of four categories of services to the audit committee for approval.

Audit services include audit work performed in the preparation of financial statements, as well as work that generally only the independent auditor can reasonably be expected to provide, including comfort letters, statutory audits, and attest services and consultation regarding financial accounting and/or reporting standards.

Audit-Related services are for assurance and related services that are traditionally performed by the independent auditor, including due diligence related to mergers and acquisitions, employee benefit plan audits, and special procedures required to meet certain regulatory requirements.

Tax services include all services performed by the independent auditor's tax personnel except those services specifically related to the audit of the financial statements, and includes fees in the areas of tax compliance, tax planning, and tax advice.

Other Fees are those associated with services not captured in the other categories. We generally do not request such services from the independent auditor.

Prior to engagement, the audit committee pre-approves these services by category of service. The fees are budgeted and the audit committee requires the independent auditor and management to report actual fees versus the budget periodically throughout the year by category of service. During the year, circumstances may arise when it may become necessary to engage the independent auditor for additional services not contemplated in the original pre-approval. In those instances, the audit committee requires specific pre-approval before engaging the independent auditor.

The audit committee may delegate pre-approval authority to one or more of its members. The member to whom such authority is delegated must report, for informational purposes only, any pre-approval decisions to the audit committee at its next scheduled meeting.

PART IV

Item 15. EXHIBITS, FINANCIAL STATEMENT SCHEDULES

Item 15(a) The following documents are filed as part of this Annual Report on Form 10-K:

Item 15(a)(1) and (2) See Item 8 to this Annual Report on Form 10-K. Other financial statement schedules have not been included because they are not applicable or the information is included in the financial statements or notes thereto.

Item 15(a)(3) Exhibits
The following is a list of exhibits filed as part of this Annual Report on Form 10-K.

Exhibit Number	Description of Exhibit
3.1(1)	Restated Certificate of Incorporation of the Registrant.
3.2(1)	Restated Bylaws of the Registrant.
4.1(1)	Form of Common Stock Certificate.
4.2.1(1)	Amended and Restated Investor Rights Agreement, dated December 13, 2002, by and among the Registrant and certain stockholders of the Registrant.
4.2.2(1)	First Amendment, dated January 11, 2005, to the Amended and Restated Investor Rights Agreement, dated December 13, 2002, by and among the Registrant and certain stockholders of the Registrant.
4.2.3(1)	Second Amendment, dated January 31, 2007, to the Amended and Restated Investor Rights Agreement, dated December 13, 2002, by and among the Registrant and certain stockholders of the Registrant.
10.1(1)*	2001 Stock Plan.
10.2(1)*	2006 Stock Plan.
10.2(a)(1)*	Form of incentive stock option agreement under 2006 Stock Plan.
10.2(b)(1)*	Form of nonqualified stock option agreement under 2006 Stock Plan.
10.2(c)(1)*	Form of restricted stock agreement under 2006 Stock Plan.
10.2(d)(1)*	Form of nonqualified stock option agreement for directors under 2006 Stock Plan.
10.2(e)(1)*	Form of restricted stock agreement for directors under 2006 Stock Plan.
10.3(1)*	Director Compensation Policy.
10.4(1)*	Non-Qualified Stock Option Agreement, dated May 27, 2004, by and between the Registrant and Keith R. Gollust.
10.5(1)	Duffy Hartwell Limited Partnership Commercial Lease, dated November 4, 1996, by and between Duffy Hartwell Limited Partnership and Shionogi BioResearch Corp., as amended by First Amendment to Commercial Lease, dated August 30, 2006.

10.6(1)	Lease of 125 Hartwell Avenue, Lexington, MA, dated October 26, 1992, by and between Fuji ImmunoPharmaceuticals Corp. and 125 Hartwell Trust, as amended by First Amendment dated January 31, 1993, Second Amendment dated October 1, 1997, Third Amendment dated November 1, 2002, Assignment and Assumption of Lease and Consent of Release by Landlord and Fourth Amendment of Lease, dated July 9, 2004, Fifth Amendment, dated October 22, 2004 and Sixth Amendment, dated August 1, 2005.
10.7(1)	Lease, dated January 13, 2005, by and between the Registrant and Mortimer B. Zuckerman and Edward H. Linde, Trustees of 91 Hartwell Avenue Trust, as amended on August 14, 2006.
10.8(1)	Master Lease Agreement, dated November 10, 2004, by and between the Registrant and General Electric Capital Corporation, as amended by Letter Agreement, dated June 24, 2005, and as extended by Letter Agreement, dated November 29, 2006.
10.9(1)	Stock Exchange Agreement, dated September 9, 2002, by and among the Registrant, Principia Associates, Inc. and certain stockholders of Principia Associates, Inc.
10.10(1)	Agreement of Merger, dated December 27, 2002, by and among the Registrant, DGN Genetics Acquisition Corp., Diagon Genetics, Inc. and certain stockholders of Diagon Genetics, Inc.
10.11(1)**	Asset Purchase Agreement, dated December 17, 2003, by and among the Registrant, Cancer Genomics, Inc., Kava Pharmaceuticals, Inc., SinglePixel Biomedical, Inc. and CMAC, LLC.
10.12(1)*	Letter Agreement, dated April 18, 2005, by and between the Registrant and Safi R. Bahcall, Ph.D.
10.13(1)*	Letter Agreement, dated October 12, 2002, by and between the Registrant and Dr. Keizo Koya.
10.14(1)*	Letter Agreement, dated January 22, 2003, by and between the Registrant and Dr. James Barsoum.
10.15(1)*	Letter Agreement, dated April 15, 2004, by and between the Registrant and Dr. Jeremy Chadwick.
10.16(1)*	Letter Agreement, dated February 19, 2004, by and between the Registrant and Keith Ehrlich.
10.17(1)*	Letter Agreement, dated January 14, 2003, by and between the Registrant and Wendy E. Rieder.
10.18(1)*	Letter Agreement, dated March 24, 2005, by and between the Registrant and Eric W. Jacobson.
10.19(1)*	Letter Agreement, dated February 27, 2006, by and between the Registrant and Martin D. Williams.
10.20(1)*	Scientific Advisory Board Agreement, dated September 1, 2003, by and between the Registrant and Judah Folkman, M.D.
10.21(1)*	Agreement and Release, dated January 14, 2005, by and between the Registrant and Lan Bo Chen, Ph.D.

10.22(1)*	Consulting Agreement, dated April 18, 2005, by and between the Registrant and Lan Bo Chen, Ph.D.
10.23(1)*	Severance Agreement, dated January 27, 2006, by and between the Registrant and Dr. Matthew Sherman.
10.24(1)*	Consulting Agreement, dated January 30, 2006, by and between the Registrant and Dr. Matthew Sherman.
10.25(1)*	Form of Indemnification Agreement between the Registrant and its directors and executive officers.
10.26(1)	Lease Agreement, dated December 14, 2006, by and between ARE-MA Region No. 24, LLC and the Registrant.
10.27*	Summary of compensation arrangements applicable to the Registrants Named Executive Officers (2006 bonus and 2007 salary increases).
21.1	List of Subsidiaries.
31.1	Certification of Principal Executive Officer under Section 302 of the Sarbanes-Oxley Act of 2002.
31.2	Certification of Principal Accounting and Financial Officer under Section 302 of the Sarbanes-Oxley Act of 2002.
32.1	Certification of the Principal Executive Officer and the Principal Accounting and Financial Officer under Section 906 of the Sarbanes-Oxley Act of 2002.

* Management contract, compensatory plan or arrangement.

** Confidential portions of these documents have been filed separately with the Securities and Exchange Commission pursuant to a request for confidential treatment.

(1) Incorporated by reference from the Registrant's Registration Statement on Form S-1, as amended (Registration No. 333-138894), initially filed with the Securities and Exchange Commission on November 22, 2006.

SIGNATURES

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

SYNTA PHARMACEUTICALS CORP.

Date: March 28, 2007

By: /s/ SAFI R. BAHCALL

Safi R. Bahcall, Ph.D.
President and Chief Executive Officer

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

Signature	Title	Date
/s/ SAFI R. BAHCALL Safi R. Bahcall, Ph.D.	President, Chief Executive Officer and Director (principal executive officer)	March 28, 2007
/s/ KEITH S. EHRLICH Keith S. Ehrlich, C.P.A.	Vice President, Finance and Administration, Chief Financial Officer (principal accounting and financial officer)	March 28, 2007
/s/ KEITH R. GOLLUST Keith R. Gollust	Chairman of the Board	March 28, 2007
/s/ LAN BO CHEN Lan Bo Chen, Ph.D.	Director	March 28, 2007
/s/ JUDAH FOLKMAN Judah Folkman, M.D.	Director	March 28, 2007
/s/ BRUCE KOVNER Bruce Kovner	Director	March 28, 2007
/s/ WILLIAM REARDON William Reardon, C.P.A.	Director	March 28, 2007
/s/ ROBERT N. WILSON Robert N. Wilson	Director	March 28, 2007

INDEX TO FINANCIAL STATEMENTS

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)

Years ended December 31, 2006, 2005, and 2004 and the period from inception (March 10, 2000) through December 31, 2006

Report of Independent Registered Public Accounting Firm

The Board of Directors
Synta Pharmaceuticals Corp.:

We have audited the accompanying consolidated balance sheets of Synta Pharmaceuticals Corp. (the Company), a development-stage company, as of December 31, 2006 and 2005, and the related consolidated statements of operations, stockholders' equity (deficit) and comprehensive loss, and cash flows for each of the years in the three-year period ended December 31, 2006 and the period from inception (March 10, 2000) through December 31, 2006. These consolidated financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these consolidated financial statements based on our audits.

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

In our opinion, the consolidated financial statements referred to above present fairly, in all material respects, the financial position of Synta Pharmaceuticals Corp. as of December 31, 2006 and 2005, and the results of their operations and their cash flows for each of the years in the three-year period ended December 31, 2006, and the period from inception (March 10, 2000) through December 31, 2006, in conformity with U.S. generally accepted accounting principles.

As discussed in Note 2 to the consolidated financial statements, the Company adopted Statement of Financial Accounting Standard (SFAS) No.123R, *Share-Based Payment*, effective January 1, 2006.

/s/ KPMG LLP

Boston, Massachusetts
March 27, 2007

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)

Consolidated Balance Sheets

(in thousands, except share and per share amounts)

	December 31, 2006	Pro forma December 31, 2006 (note 15 (unaudited))	December 31, 2005
Assets			
Current assets:			
Cash and cash equivalents	$ 33,687	$ 78,387	$ 23,809
Restricted cash	540	540	457
Marketable securities available-for-sale	13,137	13,137	38,248
Prepaid expenses and other current assets	263	263	436
Total current assets	47,627	92,327	62,950
Property and equipment, net	6,067	6,067	8,127
Deferred offering costs	963	—	—
Other assets	132	132	133
Total assets	$ 54,789	$ 98,526	$ 71,210
Liabilities and Stockholders' Equity (Deficit)			
Current liabilities:			
Accounts payable	$ 2,632	1,892	$ 3,361
Accrued expenses	6,127	5,904	8,741
Capital lease obligations—current	2,330	2,330	1,915
Deferred revenue	457	457	457
Total current liabilities	11,546	10,583	14,474
Capital lease obligations—long-term	3,170	3,170	4,259
Total liabilities	14,716	13,753	18,733
Convertible preferred stock, at redemption value:			
Series A convertible preferred stock, $0.0001 par value per share. Authorized: 8,000,000 shares at December 31, 2006 and no shares at December 31, 2005. Issued and outstanding: 8,000,000 shares at December 31, 2006 (actual), no shares at December 31, 2006 (pro forma) and no shares at December 31, 2005	41,820	—	—
Commitments and contingencies (note 10)			
Stockholders' equity (deficit):			
Common stock, par value $0.0001 per share. Authorized 158,000,000 shares at December 31, 2006 (actual), 100,000,000 shares at December 31, 2006 (pro forma) and 150,000,000 shares at December 31, 2005; 22,564,068 shares issued and outstanding at December 31, 2006 (actual), 33,842,833 shares issued and outstanding at December 31, 2006 (pro forma) and 22,674,426 shares issued and outstanding at December 31, 2005	2	3	2
Additional paid-in capital	234,807	321,326	239,029
Deferred compensation	—	—	(7,225)
Accumulated other comprehensive income (loss)	2	2	(41)
Deficit accumulated during the development stage	(236,558)	(236,558)	(179,288)

Total stockholders' equity (deficit)	(1,747)	84,773	52,477
Total liabilities and stockholders' equity (deficit)	$ 54,789	$ 98,526	$ 71,210

See accompanying notes to consolidated financial statements.

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)

Consolidated Statements of Operations

(in thousands, except share and per share amounts)

	Years ended December 31			Period from inception (March 10, 2000) through December 31, 2006
	2006	2005	2004	
Research grant revenue	$ —	—	$ 173	$ 1,477
Operating expenses:				
Research and development	50,503	59,901	38,136	180,446
In-process research and development	—	—	1,583	19,671
General and administrative	8,648	11,279	7,383	34,342
Other compensation expense(1)	—	—	—	9,315
Total operating expenses	59,151	71,180	47,102	243,774
Loss from operations	(59,151)	(71,180)	(46,929)	(242,297)
Other income:				
Investment income, net	1,881	2,317	995	5,739
Net loss	(57,270)	(68,863)	(45,934)	(236,558)
Convertible preferred stock dividends	1,859	—	—	1,859
Net loss attributable to common stockholders	$ (59,129)	$ (68,863)	$ (45,934)	$ (238,417)
Basic and diluted weighted average common shares outstanding	22,265,242	22,253,423	18,703,899	
Basic and diluted net loss attributable to common stockholders per share	$ (2.66)	$ (3.09)	$ (2.46)	

(1) Excluded from general and administrative expense.

See accompanying notes to consolidated financial statements.

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)
Consolidated Statements of Stockholders' Equity (Deficit) and Comprehensive Loss
(in thousands, except share amounts)

	Common stock								
	Shares	Amount	Additional paid-in capital	Deferred compensation	Stock subscription receivable	Accumulated other comprehensive income (loss)	Deficit accumulated during the development stage	Total stockholders' equity (deficit)	Comprehensive loss
Balance at inception	—	$ —	$ —	$ —	$ —	$ —	$ —	$ —	$
Net loss	—	—	—	—	—	—	(78)	(78)	(78)
Balance at December 31, 2000	—	—	—	—	—	—	(78)	(78)	$ (78)
Issuance of common shares to founders	5,100,000	1	1	—	—	—	—	2	
Issuance of common shares	1,700,000	—	3,400	—	(225)	—	—	3,175	
Issuance and remeasurement of stock options for services	—	—	120	(120)	—	—	—	—	
Compensation expense related to stock options for services	—	—	—	26	—	—	—	26	
Net loss	—	—	—	—	—	—	(381)	(381)	(381)
Balance at December 31, 2001	6,800,000	1	3,521	(94)	(225)	—	(459)	2,744	$ (381)
Issuance of common shares	3,563,059	—	38,635	—	—	—	—	38,635	
Issuance of common stock and warrants for Principia	1,234,875	—	15,860	—	—	—	—	15,860	
Proceeds from stock subscription	—	—	—	—	225	—	—	225	
Issuance of common stock for licenses	96,111	—	1,042	—	—	—	—	1,042	
Issuance of common stock for Diagon	786,463	—	8,525	—	—	—	—	8,525	
Issuance and remeasurement of stock options for services	—	—	851	(851)	—	—	—	—	
Compensation expense related to stock options for services	—	—	—	274	—	—	—	274	
Net loss	—	—	—	—	—	—	(36,154)	(36,154)	(36,154)
Balance at December 31, 2002	12,480,508	1	68,434	(671)	—	—	(36,613)	31,151	$ (36,154)
Issuance of common shares, net	5,116,790	1	70,479	—	—	—	—	70,480	
Amount due from stock subscription	—	—	500	—	(500)	—	—	—	
Issuance of common stock for licenses	18,444	—	200	—	—	—	—	200	
Exercise of stock warrants	143,869	—	288	—	—	—	—	288	
Exercise of stock options	39,062	—	423	—	—	—	—	423	
Modification of employee stock options	—	—	1,289	—	—	—	—	1,289	
Issuance and remeasurement of stock options for services	—	—	2,541	(2,541)	—	—	—	—	
Compensation expense related to stock options for services	—	—	—	905	—	—	—	905	
Unrealized gain on marketable securities	—	—	—	—	—	33	—	33	33
Net loss	—	—	—	—	—	—	(27,878)	(27,878)	(27,878)
Balance at December 31, 2003	17,798,673	2	144,154	(2,307)	(500)	33	(64,491)	76,891	$ (27,845)

See accompanying notes to consolidated financial statements.

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)
Consolidated Statements of Stockholders' Equity (Deficit) and Comprehensive Loss (Continued)
(in thousands, except share amounts)

	Common stock								
	Shares	Amount	Additional paid-in capital	Deferred compensation	Stock subscription receivable	Accumulated other comprehensive income (loss)	Deficit accumulated during the development stage	Total stockholders' equity (deficit)	Comprehensive loss
Issuance of common shares under stock subscription	187,500	$ —	$ 2,493	$ —	$ 500	$ —	$ —	$ 2,993	$
Issuance of common shares, net	3,999,996	—	79,900	—	—	—	—	79,900	
Issuance of common stock in connection with acquisition	138,336	—	2,213	—	—	—	—	2,213	
Issuance of restricted common shares	365,000	—	8,030	(8,030)	—	—	—	—	
Issuance of stock options at less than fair value	—	—	471	(471)	—	—	—	—	
Exercise of stock options	32,421	—	352	—	—	—	—	352	
Exercise of stock warrants	28,773	—	58	—	—	—	—	58	
Issuance and remeasurement of stock options for services	—	—	1,259	(1,259)	—	—	—	—	
Compensation expense related to stock options for services	—	—	—	1,331	—	—	—	1,331	
Compensation expense related to issuance of stock options and restricted stock below fair value	—	—	—	301	—	—	—	301	
Unrealized loss on marketable securities	—	—	—	—	—	(149)	—	(149)	(149)
Net loss	—	—	—	—	—	—	(45,934)	(45,934)	(45,934)
Balance at December 31, 2004	22,550,699	2	238,930	(10,435)	—	(116)	(110,425)	117,956	$ (46,083)
Issuance of restricted common shares	96,589	—	1,425	(1,425)	—	—	—	—	
Forfeitures of restricted common shares	(40,000)	—	(881)	743	—	—	—	(138)	
Exercise of stock warrants	67,138	—	134	—	—	—	—	134	
Issuance of stock options for services	—	—	201	(201)	—	—	—	—	
Forfeitures of stock options for services	—	—	(329)	329	—	—	—	—	
Remeasurement of stock options for services	—	—	(451)	451	—	—	—	—	
Compensation expense related to stock options for services	—	—	—	1,142	—	—	—	1,142	
Compensation expense related to issuance of stock options and restricted stock below fair value	—	—	—	2,171	—	—	—	2,171	
Unrealized gains on marketable securities	—	—	—	—	—	75	—	75	75
Net loss	—	—	—	—	—	—	(68,863)	(68,863)	(68,863)
Balance at December 31, 2005	22,674,426	$ 2	$ 239,029	$ (7,225)	$ —	$ (41)	$ (179,288)	$ 52,477	$ (68,788)
Eliminate deferred stock compensation	—	—	(7,225)	7,225	—	—	—	—	
Convertible preferred stock dividends	—	—	(1,859)	—	—	—	—	(1,859)	
Forfeitures of restricted common shares	(127,500)	—	—	—	—	—	—	—	
Issuance of common shares for services	4,875	—	69	—	—	—	—	69	
Issuance of restricted common shares	12,142	—	—	—	—	—	—	—	
Exercise of stock options	125	—	2	—	—	—	—	2	
Compensation expense related to stock options for services	—	—	4,791	—	—	—	—	4,791	
Unrealized gains on marketable securities	—	—	—	—	—	43	—	43	43
Net loss	—	—	—	—	—	—	(57,270)	(57,270)	(57,270)
Balance at December 31, 2006	22,564,068	$ 2	$ 234,807	$ —	$ —	$ 2	$ (236,558)	$ (1,747)	$ (57,227)

See accompanying notes to consolidated financial statements.

SYNTA PHARMACEUTICALS CORP.
(A Development-Stage Company)

Consolidated Statements of Cash Flows

(in thousands)

	Years ended December 31			Period from inception (March 10, 2000) through December 31, 2006
	2006	2005	2004	
Cash flows from operating activities:				
Net loss	$ (57,270)	$ (68,863)	$ (45,934)	$ (236,558)
Adjustments to reconcile net loss to net cash used in operating activities:				
In-process research and development	—	—	1,583	19,671
Common stock issued for licenses	—	—	—	1,242
Expense deferred offering costs	—	1,085	—	1,085
Other stock-related compensation expense	4,791	3,175	1,632	21,407
Depreciation and amortization	3,655	2,455	1,547	8,957
Changes in operating assets and liabilities, net of acquisitions:				
Restricted cash	(83)	—	(112)	(540)
Prepaid expenses and other current assets	173	161	(108)	(3)
Other assets	1	(17)	(33)	(64)
Accounts payable	(729)	476	2,041	2,052
Accrued expenses	(3,523)	(354)	5,477	3,289
Deferred revenue	—	—	112	457
Net cash used in operating activities	(52,985)	(61,882)	(33,795)	(179,005)
Cash flows from investing activities:				
Cash paid for acquisitions, net of cash acquired	—	—	—	(5,586)
Advances issued to related parties	—	—	—	(1,630)
Purchases of marketable securities	(118,204)	(184,365)	(124,711)	(475,196)
Sales and maturities of marketable securities	143,358	228,424	82,494	462,061
Repayment of advances from related parties	—	—	—	1,630
Purchases of property and equipment	(1,580)	(4,883)	(1,594)	(9,074)
Net cash provided by (used in) investing activities	23,574	39,176	(43,811)	(27,795)
Cash flows from financing activities:				
Proceeds from issuance of common stock and exercise of common stock warrants, net	—	134	82,951	195,890
Proceeds from issuance of convertible preferred stock, net	39,961	—	—	39,961
Proceeds from exercise of stock options	2	—	352	778
Proceeds from sale—leaseback of property and equipment	1,412	4,745	1,317	7,474
Payment of capital lease obligation	(2,086)	(1,100)	(153)	(3,429)
Payment of deferred offering costs	—	—	(187)	(187)
Net cash provided by financing activities	39,289	3,779	84,280	240,487
Net increase (decrease) in cash and cash equivalents	9,878	(18,927)	6,674	33,687
Cash and cash equivalents at beginning of period	23,809	42,736	36,062	—
Cash and cash equivalents at end of period	$ 33,687	$ 23,809	$ 42,736	$ 33,687
Supplemental disclosure of noncash investing and financing activities:				
Purchase of equipment under capital lease	$ 1,412	5,549	$ 1,878	$ 8,839

Convertible preferred stock dividends	$ 1,859	—	—	$	1,859
Cash paid for interest	$ 574	274	$ 19	$	867

See accompanying notes to consolidated financial statements.

Notes to Consolidated Financial Statements

(1) Nature of Business

Synta Pharmaceuticals Corp. (the Company) was incorporated in March 2000 and commenced operations in July 2001. The Company is a biopharmaceutical company focusing on discovering, developing and commercializing small molecule drugs that address severe medical conditions, including cancer and chronic inflammatory diseases.

The Company is subject to risks common to emerging companies in the drug development and pharmaceutical industry including, but not limited to, uncertainty of product development and commercialization, lack of marketing and sales history, dependence on key personnel, uncertainty of market acceptance of products, product liability, uncertain protection of proprietary technology, potential inability to raise additional financing and compliance with FDA and other government regulations.

The Company has incurred significant operating losses since its inception and, as a result, at December 31, 2006 had a deficit accumulated during the development stage of $236.6 million. Operations have been funded principally through the sale of common stock, convertible preferred stock and capital leases. At December 31, 2006, the Company had approximately $46.8 million in cash and marketable securities. In February 2007, the Company sold 5,000,000 shares of its common stock at $10.00 per share in an initial public offering, resulting in net proceeds of approximately $44.7 million (see Note 15). Based on the Company's current operating plans, it expects proceeds of this offering together with its existing resources to be sufficient to fund its planned operations through at least mid-2008. Over the long-term the Company will need to raise additional capital to further its drug development efforts and its clinical trials. The Company is currently seeking corporate partners to enter into collaboration arrangements as part of its overall strategy to develop and commercialize its products. However, no assurances can be made that future capital will be available on terms acceptable to the Company to support its long-term liquidity needs.

(2) Summary of Significant Accounting Policies

Basis of Presentation

Since its inception, the Company has devoted its efforts to research, product development, and securing financing. Although the Company's planned principal operations have commenced, it has not earned significant revenue. Accordingly, the consolidated financial statements are presented in accordance with Statement of Financial Accounting Standards (SFAS) No. 7, *Accounting and Reporting by Development-Stage Enterprises.*

Principles of Consolidation

The consolidated financial statements include the financial statements of Synta Pharmaceuticals Corp. and its wholly owned subsidiaries. All significant intercompany balances and transactions have been eliminated in consolidation.

Use of Estimates

The preparation of financial statements in conformity with accounting principles generally accepted in the United States of America requires management to make estimates and assumptions that affect certain reported amounts of assets and liabilities, disclosure of contingent assets and liabilities at the

date of the financial statements, and the reported amounts of revenues and expenses during the reporting periods. Significant items subject to such estimates and assumptions include long-term contract accruals, recoverability of long-lived and deferred tax assets, valuation of acquired in-process research and development, measurement of stock-based compensation, and the fair value of the Company's common stock. The Company bases its estimates on historical experience and various other assumptions that management believes to be reasonable under the circumstances. Changes in estimates are recorded in the period in which they become known. Actual results could differ from those estimates.

Cash and Cash Equivalents

Cash equivalents include money market funds and marketable securities. The Company considers all highly liquid investments with original maturities of three months or less at the date of purchase to be cash equivalents. Changes in cash and cash equivalents may be affected by shifts in investment portfolio maturities, as well as actual cash disbursements.

Marketable Securities

The Company considers its marketable securities available-for-sale in accordance with SFAS No. 115, *Accounting for Certain Investments in Debt and Equity Securities*. Marketable securities consist of investments in high-grade corporate, government and government agency obligations that are classified as available-for-sale. Since these securities are available to fund current operations they are classified as current assets on the consolidated balance sheets. Marketable securities are stated at fair value, including accrued interest, with their unrealized gains and losses included as a component of accumulated other comprehensive income (loss), which is a separate component of stockholders' equity, until such gains and losses are realized. The fair value of these securities is based on quoted market prices. If a decline in value is considered other-than-temporary, based on available evidence, the unrealized loss is transferred from other comprehensive income (loss) to the consolidated statement of operations. Realized gains and losses are determined on the specific identification method.

During the years ended December 31, 2006, 2005 and 2004, the Company recorded no realized gains and losses on marketable securities. There were no charges to write down marketable securities in 2006 and 2005.

Credit Risk and Concentrations

Financial instruments that potentially subject the Company to a concentration of credit risk consist of money market funds and marketable securities. Deposits with banks may exceed the amount of insurance provided on such deposits. Generally, these deposits may be redeemed upon demand and, therefore, bear minimal risk. Marketable securities consist of investments in high-grade corporate, government and government agency obligations. The Company's policy for investments in marketable securities, approved by the board of directors, establishes guidelines relating to diversification and maturities that allows the Company to manage risk.

Fair Value of Financial Instruments

The carrying amounts of the Company's financial instruments, which include cash equivalents, marketable securities, and capital lease obligations, approximate their fair values.

Property and Equipment

Property and equipment is carried at cost and depreciated using the straight-line method over the estimated useful lives of the related assets, which range from three to five years. Leasehold improvements are amortized over the lesser of the lease term or estimated useful life.

Research and Development Costs

Research and development costs are expensed as incurred in accordance with SFAS No. 2, *Accounting for Research and Development Costs.* Research and development costs are comprised of costs incurred in performing research and development activities, including salaries, benefits, facilities, research-related overhead, clinical trial costs, contracted services, technology acquisition license fees, and other external costs.

Patents

Costs to secure and defend patents are expensed as incurred and are classified as general and administrative expense in the Company's consolidated statements of operations. Patent expenses were approximately $1,561,000, $1,598,000, $1,605,000 and $5,550,000 for the years ended December 31, 2006, 2005, 2004, and for the period from inception (March 10, 2000) through December 31, 2006, respectively.

Income Taxes

The Company accounts for income taxes in accordance with SFAS No. 109, *Accounting for Income Taxes*. Deferred tax assets and liabilities are determined based on differences between financial reporting and income tax basis of assets and liabilities, as well as net operating loss carryforwards, and are measured using the enacted tax rates and laws that are expected to be in effect when the differences reverse. Deferred tax assets may be reduced by a valuation allowance to reflect the uncertainty associated with their ultimate realization.

Impairment of Long-Lived Assets

The Company accounts for the impairment and disposition of long-lived assets in accordance with SFAS No. 144, *Accounting for the Impairment or Disposal of Long-Lived Assets* (SFAS 144). In accordance with SFAS 144, management assesses the potential impairments of its long-lived assets whenever events or changes in circumstances indicate that an asset's carrying value may not be recoverable. If the carrying value exceeds the undiscounted future cash flows estimated to result from the use and eventual disposition of the asset, the Company writes down the asset to its estimated fair value. Management believes that no long-lived assets were impaired as of December 31, 2006 and 2005.

Revenue Recognition

Revenues to date have been generated by research grant contracts and, accordingly, the Company recognizes revenue in accordance with the Securities and Exchange Commission's (SEC) Staff Accounting Bulletin No. 101, *Revenue Recognition in Financial Statements* (SAB 101), as amended by Staff Accounting Bulletin No. 104, *Revenue Recognition* (SAB 104). Revenues from research contracts are recognized in the period the related services are performed and the reimbursable costs are incurred. The Company is a development-stage enterprise, and no revenues have been derived to date from its principal operations.

Stock-Based Compensation

(i) Stock-Based Compensation under APB No. 25

Prior to January 1, 2006, the Company applied the intrinsic-value-based method of accounting prescribed by Accounting Principles Board (APB) Opinion No. 25, *Accounting for Stock Issued to Employees,* and related interpretations including Financial Accounting Standards Board (FASB) Interpretation No. 44, *Accounting for Certain Transactions involving Stock Compensation, an Interpretation of APB Opinion No. 25,* in accounting for its employee stock options. Under this method, compensation expense is generally recorded on the date of grant only if the estimated fair value of the underlying stock exceeds the exercise price. Given the absence of an active market for the Company's common stock, the board of directors historically has determined the estimated fair value of common stock on the dates of grant based on several factors, including progress against regulatory, clinical and product development milestones, sales of common stock to outside investors and the likelihood of achieving a liquidity event such as an initial public offering or sale of the Company. As a result, the Company recorded deferred compensation charges for the difference between the estimated fair value of the common stock and the exercise price of options granted at the date of grant. Compensation expense is recognized over the vesting period on a straight-line basis.

The Company accounts for stock options issued to nonemployees in accordance with the provisions of SFAS No. 123, *Accounting for Stock-Based Compensation,* and Emerging Issues Task Force (EITF) No. 96-18, *Accounting for Equity Instruments that are Issued to Other than Employees, or in Conjunction with Selling Goods or Services,* which requires valuing the stock options using a Black-Scholes option pricing model and remeasuring such stock options to the current fair value until the performance date has been reached.

SFAS No. 123 and SFAS No. 148, *Accounting for Stock-Based Compensation—Transition and Disclosure, an amendment of FASB Statement No. 123,* established accounting and disclosure requirements using a fair-value-based method of accounting for stock-based employee compensation plans. As permitted by existing accounting standards, the Company elected to continue to apply the intrinsic-value-based method of accounting described above, for options granted through December 31, 2005. The following table illustrates the effect on net loss attributable to common stockholders as if the fair-value-based method had been applied to all outstanding and unvested awards for the years ended December 31, 2005 and 2004 and the period from inception (March 10, 2000) through December 31,

2005, prior to the adoption of SFAS No. 123(R), *Share-Based Payment* on January 1, 2006 (in thousands, except per share amounts).

	Years ended December 31,		Period from inception (March 10, 2000) through December 31, 2005
	2005	2004	
Net loss attributable to common stockholders, as reported	$ (68,863)	$ (45,934)	$ (179,288)
Add: stock-based employee compensation expense determined under the fair value method	(4,172)	(1,099)	(8,248)
Deduct: stock-based employee compensation expense included in reported net loss	2,034	301	3,754
Pro forma net loss attributable to common stockholders	$ (71,001)	$ (46,732)	$ (183,782)
Basic and diluted net loss attributable to common stockholders per common share, as reported	$ (3.09)	$ (2.46)	—
Basic and diluted net loss attributable to common stockholders per common share, pro forma	$ (3.19)	$ (2.50)	—

For the years ended December 31, 2006, 2005 and 2004, the fair value of each employee stock option award was estimated on the date of grant based on the fair value method using the Black-Scholes option pricing valuation model with the following weighted average assumptions:

	Years ended December 31,			Period from inception (March 10, 2000) through December 31, 2006
	2006	2005	2004	
Risk-free interest rate	4.63%	3.91%	3.78%	3.61%
Expected life in years	6.25 years	5 years	5 years	5.23 years
Volatility	75%	70%	—	25%
Expected dividend yield	—	—	—	—
Weighted average grant-date fair value	$9.80	$13.40	$10.08	$5.32

Prior to January 17, 2005, the Company utilized the minimum value method and therefore did not consider volatility in estimating the fair value of its stock options.

(ii) Stock Based Compensation under SFAS No. 123(R):

Effective January 1, 2006, the Company adopted SFAS No. 123(R) using the modified prospective method of transition for employee stock option awards granted after January 17, 2005 (valued using the fair value method), and using the prospective method for awards granted prior to January 17, 2005 (valued using the minimum value method). Therefore, compensation cost recognized in the year ended

December 31, 2006 includes: (a) compensation costs related to the vesting of employee stock options granted after January 17, 2005 but prior to January 1, 2006, based on the grant date fair value method estimated in accordance with the provisions of SFAS 123 adjusted for estimated forfeitures (b) compensation costs related to the continued vesting of nonvested restricted stock awards granted prior to January 1, 2006, and (c) compensation costs for all share-based payments granted or modified subsequent to January 1, 2006, based on the provisions of SFAS No. 123(R).

Prior to the adoption of SFAS No. 123(R), the Company presented its unamortized portion of deferred compensation cost for nonvested stock options in the consolidated statement of stockholders' equity (deficit) and comprehensive loss with a corresponding credit to additional paid-in capital. Upon the adoption of SFAS No. 123(R), these amounts were offset against each other. Under SFAS No. 123(R), an equity instrument is not considered to be issued until the instrument vests. As a result, compensation costs are recognized over the requisite service period with an offsetting credit to additional paid-in capital, and the deferred compensation balance of $7,225,000 at January 1, 2006 was netted against additional paid-in capital during the first quarter of 2006.

The Company continued to use the Black-Scholes option pricing model as the most appropriate valuation method for its option grants. The Black-Scholes model requires inputs for risk-free interest rate, dividend yield, volatility and expected lives of the options. Since the Company has a limited history of stock activity, expected volatility is based on historical data from several public companies similar in size and value to the Company. The Company will continue to use a weighted average approach using historical volatility and other similar public entity volatility information until historical volatility of the Company is relevant to measure expected volatility for future option grants. The Company estimates the forfeiture rate based on historical data. Based on an analysis of historical forfeitures, the Company has applied a forfeiture rate of 10% to all options vesting in the year ended December 31, 2006. The analysis will be re-evaluated at least annually and the forfeiture rate will be adjusted as necessary. The risk-free rate for periods within the contractual life of the option is based on the U.S. Treasury yield curve in effect at the time of the grant. The expected lives for options granted represents the period of time that options granted are expected to be outstanding. Since January 1, 2006 the Company has used the simplified method for determining the expected lives of options.

For awards with graded vesting, the Company allocates compensation costs under SFAS No. 123(R) on a straight-line basis over the requisite service period. The Company amortized the fair value of each option over each option's service period, which is generally the vesting period.

As a result of adopting SFAS No. 123(R) on January 1, 2006, the Company's net loss for the year ended December 31, 2006 was approximately $3,159,000 higher than if it had continued to account for share-based compensation under APB Opinion No. 25.

The Company's net loss for the year ended December 31, 2006 includes $4,791,000 of compensation costs and no income tax benefit related to the Company's stock-based compensation arrangements for employee and nonemployee awards. As of December 31, 2006, the total amount of unrecognized stock-based compensation expense is $13,331,000 and will be recognized over a weighted average period of 3.6 years.

The following table outlines the details of recognized and unrecognized expense for these stock-based compensation arrangements (in thousands):

	Stock compensation expense for the year ended December 31, 2006	Unrecognized stock compensation expense as of December 31, 2006
Employee stock options	$ 2,752	$ 8,196
Repriced employee stock options	407	313
Employee options issued below fair value	60	29
Non-employee stock options	272	240
Restricted stock	1,300	4,553
	$ 4,791	$ 13,331

Stock-based compensation expense is allocated as follows (in thousands):

	Years ended December 31,		
	2006	2005	2004
Research and development	$ 3,372	$ 2,397	$ 1,204
General and administrative	1,419	778	428
Total	$ 4,791	$ 3,175	$ 1,632

Certain of the employee stock options granted by the Company are structured to qualify as incentive stock options (ISOs). Under current tax regulations, the Company does not receive a tax deduction for the issuance, exercise or disposition of ISOs if the employee meets certain holding requirements. If the employee does not meet the holding requirements, a disqualifying disposition occurs, at which time the Company will receive a tax deduction. The Company does not record tax benefits related to ISOs unless and until a qualifying disposition occurs. In the event of a disqualifying disposition, the entire tax benefit is recorded as a reduction of income tax expense. The Company has not recognized any income tax benefit for the share-based compensation arrangement due to the fact that the Company does not believe it is more likely than not it will recognize any deferred tax assets from such compensation cost recognized in the current period.

Comprehensive Income (Loss)

SFAS No. 130, *Reporting Comprehensive Income*, requires that all components of comprehensive income (loss) be disclosed in the consolidated financial statements. Comprehensive income (loss) is defined as the change in equity of a business enterprise during a period from transactions, and other events and circumstances from non-owner sources. Changes in unrealized gains and losses on marketable securities represents the only difference between the Company's net loss and comprehensive loss.

Segment Reporting

The Company has adopted SFAS No. 131, *Disclosure About Segments of an Enterprise and Related Information,* which requires companies to report selected information about operating segments, as well as enterprise-wide disclosures about products, services, geographical area, and major customers. Operating segments are determined based on the way management organizes its business for making operating decisions and assessing performance. The Company has only one operating segment, the discovery, development and commercialization of drug products.

Basic and Diluted Net Loss Per Common Share

Net loss per share is computed based on the guidance of SFAS No. 128, *Earnings Per Share*, requiring companies to report both basic net loss per common share, which is computed using the weighted average number of common shares outstanding during the period, and diluted net loss per common share, which is computed using the weighted average number of common shares outstanding and the weighted average dilutive potential common shares outstanding using the treasury stock method. However, for all periods presented, diluted net loss per share is the same as basic net loss per share as the inclusion of weighted average shares of unvested restricted common stock and common stock issuable upon the exercise of stock options and warrants and conversion of convertible preferred stock would be anti-dilutive.

The following table summarizes securities outstanding as of each year-end which were not included in the calculation of diluted net loss per share as their inclusion would be anti-dilutive.

	December 31		
	2006	2005	2004
Common stock options	3,043,068	2,947,683	2,512,112
Common stock warrants	—	—	67,138
Nonvested restricted common stock	291,069	421,589	365,000
Convertible preferred stock	2,092,931	—	—

The convertible preferred stock and accrued dividends have been reflected as being converted into common stock using a $20.00 per share conversion factor. The convertible preferred stock has several different conversion rights that are discussed in note 6 to these audited consolidated financial statements.

Recent Accounting Pronouncements

In May 2005, the FASB issued SFAS No. 154, *Accounting Changes and Error Correction* (SFAS No. 154). SFAS No. 154 is a replacement of APB Opinion No. 20, *Accounting Changes* (APB Opinion No. 20), and SFAS No. 3, *Reporting Accounting Changes in Interim Financial Statements.* This statement applies to all voluntary changes in accounting principle, and changes the accounting for, and reporting of, a change in accounting principle. SFAS No. 154 requires retrospective application to prior periods' financial statements of a voluntary change in accounting principle unless it is impracticable to do so. APB Opinion No. 20 previously required that most voluntary changes in accounting principle be recognized by including in net income of the period of the change the cumulative effect of changing to

the new accounting principle. SFAS No. 154 carries forward many provisions of APB Opinion No. 20 without change, including the provisions related to the reporting of a change in accounting, a change in the reporting entity, and the correction of an error. SFAS No. 154 does not change the transition provisions of any existing account pronouncements, including those that are in a transition phase as of the effective date of the statement. The Company adopted the provisions of SFAS No. 154 on January 1, 2006, and the adoption of the new standard did not have a material impact on the Company's consolidated financial position or consolidated statement of operations.

In June 2005, the FASB issued FSP 150-5. The FSP clarifies that freestanding warrants and similar instruments on shares that are redeemable should be accounted for as liabilities under SFAS No. 150, regardless of the timing of the redemption feature or price, even though the underlying shares may be classified as permanent or temporary equity. The FSP was effective for the first reporting period beginning after June 30, 2005. The Company adopted FSP 150-5 in 2006 and the impact was not material to the Company's consolidated financial position or consolidated statement of operations.

In July 2006, the FASB issued FASB Interpretation No. 48, *Accounting for Uncertainty in Income Taxes—an interpretation of FAS 109*. This interpretation clarifies the accounting for uncertainty in income taxes recognized in a company's financial statements in accordance with FASB Statement No. 109, *Accounting for Income Taxes.* This interpretation prescribes a recognition threshold and measurement attribute for the financial statement recognition and measurement of a tax position taken in a tax return. It also provides guidance on derecognition, classification, interest and penalties, accounting in interim periods, disclosure and transition. Interpretation No. 48 is effective for fiscal years beginning after December 15, 2006. Earlier application is encouraged if the company has not yet issued financial statements, including interim financial statements, in the period Interpretation No. 48 is adopted. The Company is currently evaluating the impact the adoption of this interpretation will have on its consolidated results of operations and financial position.

In September 2006, the FASB issued SFAS No. 157, *Fair Value Measurements* (SFAS No. 157), which provides guidance for using fair value to measure assets and liabilities. The pronouncement clarifies (1) the extent to which companies measure assets and liabilities at fair value; (2) the information used to measure fair value; and (3) the effect that fair value measurements have on earnings. SFAS No. 157 will apply whenever another standard requires (or permits) assets or liabilities to be measured at fair value. SFAS No. 157 will be applicable to us as of January 1, 2008. We do not believe the adoption of SFAS No. 157 will have a material impact on our overall financial position or results of operations.

In September 2006, the U.S. Securities and Exchange Commission issued Staff Accounting Bulletin No. 108, *Considering the Effects of Prior Year Misstatements when Quantifying Misstatements in Current Year Financial Statements* (SAB No. 108). SAB No. 108 provides guidance on the consideration of the effects of prior year misstatements in quantifying current year misstatements in determining whether the current year's financial statements are materially misstated. SAB No. 108 is effective for fiscal years ending after November 15, 2006. We have adopted SAB No. 108 as of December 31, 2006 and its adoption did not have a material impact on our consolidated financial statements.

In February 2007, the FASB issued SFAS No. 159, *The Fair Value Option for Financial Assets and Financial Liabilities* including an amendment of SFAS No. 115, which permits entities to choose to

measure many financial instruments and certain other items at fair value that are not currently required to be measured at fair value. SFAS No. 159 is effective for the Company beginning in fiscal 2009. The Company is currently evaluating SFAS No. 159 and the impact that it may have on its results of operations or financial position.

(3) Acquisitions

Principia Associates, Inc.

In September 2002, the Company acquired all of the outstanding capital stock of Principia Associates, Inc. (Principia) and its wholly-owned subsidiary, SBR Pharmaceuticals Corp. (formerly Shionogi BioResearch Corp.) (SBR) in exchange for an aggregate of 1,234,875 shares of common stock of the Company together with warrants to purchase an aggregate of 239,780 shares of common stock of the Company, forgiveness of $1.0 million in short-term promissory notes receivable and cash of approximately $268,000. Total value of consideration paid was approximately $16.9 million. Principia was formed and held by three stockholders of the Company. On July 31, 2002, Principia and members of the Company's board of directors, together with their respective affiliates, acquired a majority of the common stock of SBR. The Company's scientific founder, a member of the board of directors and major shareholder of the Company, previously owned approximately 20% of SBR.

The common stock of the Company was valued at $10.84 per share, its fair value as determined by the Company's board of directors, for an aggregate value of approximately $13.4 million. The common stock purchase warrants, which expired in 2005, had an exercise price of $2.00 per share. The warrants were valued at approximately $2.2 million using the Black-Scholes valuation pricing model, with the following assumptions: risk-free interest rate of 2.3%, volatility of 75%, and a life of three years.

The following table summarizes the estimated fair value of the assets acquired and liabilities assumed at the date of acquisition (in thousands):

Current assets, including cash of $922	$	995
In-process research and development		13,888
Property and equipment		3,527
Other assets		67
Total assets acquired		18,477
Liabilities assumed		1,617
Net assets acquired	$	16,860

For accounting purposes, the transaction was treated as an acquisition of assets and not a business combination because Principia did not meet the definition of a business under EITF 98-3, *Determination Whether a Nonmonetary Transaction Involves Receipt of Productive Assets or of a Business*. The purchase price was allocated to assets acquired and liabilities assumed based on management's analysis and estimates of fair values. Management's estimates of fair value are based on assumptions believed to be reasonable, but which are inherently uncertain and unpredictable. The acquired in-process research and development (IPR&D) was valued at $11.7 million. The remaining excess purchase price over the identified tangible and intangible assets and liabilities assumed was

approximately $2.2 million. The excess amount was allocated to the acquired intangible assets, resulting in approximately $13.9 million being assigned to IPR&D assets that were written off at the date of acquisition in accordance with FASB Interpretation No. 4, *Applicability of FASB Statement No. 2 to Business Combinations Accounted for by the Purchase Method*.

The value assigned to IPR&D related to research projects for which technological feasibility had not yet been established and no future alternative uses existed. The fair value was determined using the income approach, which discounts expected future cash flows from projects under development to their net present value using a risk-adjusted rate. Each project was analyzed to determine the utilization of core technology; the complexity, cost and time to complete development; any alternative future use or current technological feasibility; and the stage of completion. Future cash flows were estimated, taking into account the expected life cycles of the product and the underlying technology, relevant market sizes and industry trends. The estimated net cash flows from these products were based on management's estimates of related revenues, cost of goods sold, R&D costs, selling, general and administrative costs, and income taxes. Discount rates ranging from 30% to 40% were utilized based on the nature of the technology of the products, the stage of completion of the projects, the complexity of the development effort and the risks associated with reaching technological feasibility of the projects.

SBR had three products under development at the acquisition date, contributing 63%, 27%, and 10% of the total IPR&D value. The products under development are intended to result in therapeutic products in the areas of oncology and autoimmune disease. Commercialization of any product is not anticipated for several years.

Diagon Genetics, Inc.

In December 2002, the Company acquired all of the outstanding capital stock of Diagon Genetics, Inc. (Diagon). The purchase price of approximately $13.5 million consisted of 786,463 shares of common stock at a per share value of $10.84 and $5.0 million in cash. Diagon was previously owned by the Company's Chief Executive Officer and scientific founder, both of whom are board members and significant shareholders of the Company.

For accounting purposes, the transaction did not constitute a business combination because Diagon did not meet the definition of a business under EITF No. 98-3, *Determining Whether a Nonmonetary Transaction Involves Receipt of Productive Assets or of a Business*. At the time, Diagon's activities consisted of owning the rights to the development of certain intellectual property that might be used to develop therapeutic drug products. Commercialization of any product is not anticipated for several years. The Company allocated the purchase price to the fair value of the acquired assets and liabilities. As a result, the Company recorded in-process research and development of $4.2 million, which was written off at the date of acquisition. As noted above, Diagon was previously owned by the Company's Chief Executive Officer and its scientific founder, both members of the Company's board of directors, therefore the remaining excess purchase price of $9.3 million was charged to operations as other compensation expense in the accompanying consolidated statements of operations.

The value assigned to IPR&D related to research projects for which technological feasibility had not yet been established and no future alternative uses existed. The fair value was determined using the income approach, which discounts expected future cash flows from projects under development to their net present value using a risk-adjusted rate. Each project was analyzed to determine the utilization of core technology; the complexity, cost and time to complete development; any alternative future use or current technological feasibility; and the stage of completion. Future cash flows were estimated, taking into account the expected life cycles of the product and the underlying technology, relevant market sizes and industry trends. The estimated net cash flows from these products were based on management's estimates of related revenues, cost of goods sold, R&D costs, selling, general and administrative costs, and income taxes. A discount rate of 30% was utilized based on the nature of the technology of the products, the stage of completion of the projects, the complexity of the development effort and the risks associated with reaching technological feasibility of the projects.

The Company had three products under development at the acquisition date, contributing 66%, 29%, and 5% of the total IPR&D value. The products under development are intended to result in therapeutic products in the areas of oncology, autoimmune disease, and allergy. Commercialization of any product is not anticipated for several years.

Cancer Genomics, Inc., Kava Pharmaceuticals, Inc. and SinglePixel Biomedical, Inc.

In January 2004, the Company acquired certain assets of Cancer Genomics, Inc., Kava Pharmaceuticals, Inc. (Kava) and SinglePixel Biomedical, Inc. (collectively, CKS) in a single transaction. Direct and indirect shareholders in these companies included the Company's scientific founder, who is also a board member, as well as three current or former Company executives. The purchase price of approximately $2.2 million consisted of 138,336 shares of the Company's common stock. In addition, the Company is required to make cash payments of up to $2.0 million if certain milestones are achieved. If commercialization is achieved, the Company will be required to pay royalties on the gross sales of any payment of service covered by the acquired technology. The intellectual property acquired from Kava is unrelated to our current clinical programs or our programs in development.

The following table summarizes the estimated fair value of the assets acquired and liabilities assumed at the date of acquisition (in thousands):

In-process research and development	$ 1,583
Property and equipment (including capitalized software)	736
Total assets acquired	2,319
Liabilities assumed	(106)
Net assets acquired	$ 2,213

The purchase price was allocated to assets acquired and liabilities assumed based on management's analysis and estimates of fair values. Management's estimates of fair value are based on assumptions believed to be reasonable, but which are inherently uncertain and unpredictable. The acquired IPR&D was initially valued at approximately $0.5 million. The remaining excess purchase price over the identified tangible and intangible assets and liabilities assumed was approximately $1.1 million. The

excess amount was allocated to the acquired intangible assets, resulting in approximately $1.6 million being assigned to IPR&D assets that were written off at the date of acquisition in accordance with FASB Interpretation No. 4. The Kava IPR&D pertained to the small-molecule pharmaceutical for the treatment of anxiety and general pain. The initial value of the Kava IPR&D was based on the cost approach. During 2002, after an initial investment to advance the technology, the Company ceased further funding of the project.

(4) Cash, Cash Equivalents and Marketable Securities

A summary of cash and cash equivalents and available-for-sale marketable securities held by the Company as of December 31, 2006 and 2005 is as follows:

	December 31, 2006			
	Cost	Unrealized gains	Unrealized losses	Fair value
	(in thousands)			
Cash and cash equivalents:				
Cash and money market funds	$ 33,687	—	—	$ 33,687
Marketable securities:				
Corporate bonds:				
Due within 1 year	13,135	2	—	13,137
Total cash, cash equivalents and marketable securities	$ 46,822	$ 2	$ —	$ 46,824

	December 31, 2005			
	Cost	Unrealized gains	Unrealized losses	Fair value
	(in thousands)			
Cash and cash equivalents:				
Cash and money market funds	$ 23,809	$ —	$ —	$ 23,809
Marketable securities:				
Corporate bonds:				
Due within 1 year	38,289	—	(41)	38,248
Total cash, cash equivalents and marketable securities	$ 62,098	$ —	$ (41)	$ 62,057

(5) Property and Equipment

Property and equipment consist of the following at December 31:

	2006	2005
	(in thousands)	
Laboratory equipment	$ 8,724	$ 7,272
Leasehold improvements	3,854	3,824
Office equipment	1,042	954
Furniture and fixtures	677	677
	14,297	12,727
Less accumulated depreciation and amortization	(8,230)	(4,600)
	$ 6,067	$ 8,127

Depreciation and amortization expenses of property and equipment were approximately $3,655,000, $2,455,000, $1,547,000 and $8,957,000 for the years ended December 31, 2006, 2005, 2004, and for the period from inception (March 10, 2000) through December 31, 2006, respectively. The net book value and accumulated depreciation of equipment under capital lease was $4,050,000 and $3,020,000 and $4,609,000 and $1,175,000, at December 31, 2006 and 2005, respectively.

(6) Stockholders' Equity

Capital Stock—Authorized Shares

In June 2006, the Company's stockholders approved an increase in the number of authorized shares of common stock from 150,000,000 shares to 158,000,000 shares and 8,000,000 shares of preferred stock all of which are designated as shares of Series A Convertible Preferred Stock, each share having a $0.0001 par value (See Note 15).

Each common stockholder is entitled to one vote for each share of stock held. The common stock will vote together with all other classes and series of stock of the Company as a single class on all actions to be taken by the Company's stockholders. Each share of common stock is entitled to receive dividends, as and when declared by the Company's board of directors.

The Company has never declared cash dividends on any of its capital stock and does not expect to do so in the foreseeable future.

On December 13, 2002 and as amended on January 11, 2005, the Company entered into an Amended and Restated Investor Rights Agreement (the Investor Rights Agreement) with its three largest stockholders and their affiliates exclusive of the founders (the Investors). The Investors Rights Agreement grants certain rights and privileges to and places certain restrictions upon the Investors, including: (i) grants the Investor a right of first refusal to purchase the Investor's pro rata share of any private securities offering by the Company, so long as such Investor owns at least 5% of the Company's outstanding common stock; (ii) piggyback registration rights with respect to any registration by the Company of its securities in preparation for a public offering, with priority over other Company stockholders; (iii) demand registration rights commencing 180 days after a public offering in which such Investor did not exercise its piggyback registration rights, allowing the Investor to demand that the Company register the Investor's securities so long as the value of such securities equals or exceeds $5.0

million; and (iv) places restrictions upon the Investors' abilities to transfer, contract to transfer, or enter into any swap agreement related to the Company's securities starting from the date of an initial public offering and ending up to 180 days later, provided that all of the Company's directors, executive officers, and 1% or greater shareholders agree to similar restrictions. Finally, the Company bears certain information reporting and indemnification obligations with respect to the Investors and the registration of the Company's securities, and the Investors bear certain indemnification obligations to the Company with respect to the registration of the Investor's Company securities.

Issuance of Common Stock

In July 2001, the Company issued 5,100,000 shares of its common stock to its founding members for $0.0004 per share.

Between July and December 2001, the Company sold 1,700,000 shares of its common stock at $2.00 per share (the A Round Financing) through a stock subscription, resulting in gross proceeds of $3.4 million. As of December 31, 2001, the Company had a stock subscription receivable of $225,000, which was received in 2002.

During 2002, the Company sold 3,563,059 shares of its common stock at $10.8432 per share (the B Round Financing), resulting in gross proceeds of approximately $38.6 million.

In July and December 2002, the Company issued an aggregate of 96,111 shares of its common stock, plus $30,000 of cash, in exchange for exclusive royalty-bearing licenses for certain patent rights. The aggregate value of the stock and cash consideration of $1,072,000 was charged immediately to research and development costs.

Between January and March 2003, the Company completed the B Round Financing by issuing 2,179,292 shares of common stock at $10.8432 per share, which resulted in gross proceeds of approximately $23.6 million.

In March 2003, the Company issued 18,444 shares of its common stock, plus $40,000 cash, in exchange for an exclusive royalty-bearing license for certain patent rights. The total value of the consideration paid of $240,000 was expensed immediately to research and development costs (see note 10).

In September 2003, the Company commenced the sale of 3,125,000 shares of its common stock at $16.00 per share (the C Round Financing). Through December 31, 2003, the Company had issued 2,937,498 shares, resulting in gross proceeds of $47.0 million. In addition, 31,250 shares of common stock were subscribed but unissued. The stock subscription receivable of $500,000 is reflected as a component of stockholders' equity on the accompanying consolidated balance sheet. The remaining 187,500 shares of common stock were issued in January 2004, which resulted in additional gross proceeds of $3.0 million.

In January 2004, the Company received the proceeds under a stock subscription for 187,500 shares of its common stock at $16.00 per share, for net proceeds of $2,993,000 (C Round Financing).

In November 2004, the Company sold 3,999,996 shares of its common stock at $20.00 per share, for net proceeds of $79,900,000.

In April 2006, the Company issued the chief executive officer 4,875 shares of its common stock at $14.00 per share in connection with a partial payment of his annual bonus.

In 2002 and 2004, the Company issued common stock in connection with acquisitions (see note 3).

Convertible Preferred Stock

In June 2006, the Company sold 8,000,000 shares of its Series A Convertible Preferred Stock (the "Preferred Stock") at a price of $5.00 per share resulting in gross proceeds of $40 million. The Preferred Stock accrues a cumulative annual dividend of 8% of its purchase price. (See note 15)

Liquidation Preference

In the event of a liquidating event or deemed liquidating event as defined below, amounts available for distributions to holders of the Company's capital stock will be paid first to holders of the Preferred Stock, on a pro rata basis, equal to the Preferred Stock purchase price plus all accrued or declared but unpaid dividends (the "accrued dividends") and second ratably to holders of common stock.

A merger, acquisition, consolidation or sale of all or substantially all assets or other reorganizations or any transaction or series of transactions resulting in a transfer of more than 50% stock ownership of the Company constitutes a deemed liquidating event. The Preferred Stock is classified outside of permanent equity because the transfer of stock ownership is outside of the Company's control and, accordingly, is treated as redeemable.

Voting rights

The Preferred Stock generally votes together with common stock with the right to that number of votes equal to the number of shares of common stock then issuable upon conversion of the Preferred Stock.

Conversion

Voluntary conversion

The number of shares of common stock into which each share of Preferred Stock may be converted at the holder's option is determined by dividing the Preferred Stock purchase price plus all accrued dividends by $20.00 per share.

Automatic conversion upon an initial public offering

Each share of Preferred Stock will be automatically converted into shares of common stock upon the consummation of a firm committment underwritten public offering of the Company's common stock (an "IPO"). The number of shares of common stock into which each share of Preferred Stock will be converted will be determined by dividing the Preferred Stock purchase price plus all accrued dividends by the lesser of $20.00 or 66.6667% of the offering price to the public of the IPO.

Automatic conversion upon vote

Each share of the Preferred Stock will be automatically converted into shares of common stock upon the affirmative vote of the holders of at least a majority of the Preferred Stock voting as a separate class. The number of shares of common stock into which each share of Preferred Stock will be converted will be determined by dividing the Preferred Stock purchase price plus all accrued dividends by $20.00 per share.

Optional conversion following qualified financing

In the event that, prior to the closing of an IPO, the Company completes a Qualified Financing as defined below, each share of the Preferred Stock will be convertible at any time within the two month period following such Qualified Financing, at the option of the holder, into shares of the same type and class of capital stock of the Company issued in such Qualified Financing (the "Investor Stock"). The number of shares of Investor Stock into which each share of Preferred Stock will be converted will be determined by dividing the Preferred Stock purchase price plus all accrued dividends by the price per share paid by the purchasers of such shares of Investor Stock. A "Qualified Financing" means a transaction, or series of related transactions, entered into by the Company for the primary purpose of raising capital in which the Company issues shares of Investor Stock and receives gross proceeds of at least $5.0 million; provided that a Qualified Financing does not include an IPO nor any sale of equity by the Company entered into by the Company primarily for purposes other than capital raising, as reasonably determined by the Board of Directors. This right of optional conversion shall apply only to the first Qualified Financing occurring after the closing of the sale of the Preferred Stock, and not to any other successive transaction, or series of related transactions, entered into by the Company for the primary purpose of raising capital and may be waived or eliminated by the affirmative vote of the holders of at least a majority of the Preferred Stock voting as a separate class.

Proportional adjustments

The Preferred Stock, including its conversion price, is subject to proportional adjustments for stock dividends, stock splits, combinations or other similar recapitalization affecting to shares of common stock.

Issuance of Restricted Stock

During 2004 and 2005, the Company sold and issued 365,000 and 87,500 restricted shares of common stock, respectively, to its officers and certain employees at par value, of which 40,000 and 127,500 of these restricted shares were forfeited in 2005 and 2006, respectively. Holders of 260,000 of the restricted shares employed by the Company in January 2007 will become vested in 50% of the restricted stock. The remaining 50% vests upon the earlier of January 2009 or the approval of the Company's first New Drug Application (NDA) by the Food and Drug Administration (FDA). Holders of 25,000 shares of the restricted shares employed by the Company in January 2008 will become vested in 50% of the restricted stock. The remaining 50% vests upon the earlier of January 2010 or the approval of the Company's first NDA by the FDA. During 2005 and 2006, the Company sold and issued 9,089 and 12,142 shares of restricted stock, respectively, at par value to certain members of its Board of Directors in connection with their annual director fees. These restricted shares vest over the

service periods. The excess of the fair value over the purchase price of the common stock at the date of issuance, an aggregate of approximately $9.5 million, has been recorded as deferred compensation and is being amortized and expensed ratably over the estimated vesting periods. Compensation expense recognized for restricted shares was approximately $1,300,000, $1,916,000 and $59,000 in 2006, 2005 and 2004, respectively.

Warrants

In September 2002, the Company issued warrants to purchase an aggregate of 239,780 shares of its common stock at an exercise price of $2.00 per share and with an expiration date of September 19, 2005, in connection with its acquisition of Principia (see note 3). In December 2003, warrants to purchase 143,869 shares of the Company's common stock were exercised, resulting in proceeds of $288,000. In November 2004, warrants to purchase 28,773 shares of the Company's common stock were exercised, resulting in proceeds of $58,000. In January 2005, the remaining outstanding warrants to purchase 67,138 shares of the Company's common stock were exercised, resulting in proceeds to the Company of $134,000.

(7) Stock Option Plans

In July 2001, the Company adopted the Synta Pharmaceuticals Corp. 2001 Stock Plan (the 2001 Stock Option Plan). The 2001 Stock Option Plan provided for the grant of incentive stock options, nonstatutory stock options and restricted stock to employees, officers, directors and consultants to the Company. A total of 3,750,000 shares of common stock were reserved for issuance under the 2001 Stock Option Plan. The administration of the 2001 Stock Option Plan is under the general supervision of the board of directors. The exercise price of the stock options was determined by the board of directors, provided that incentive stock options were granted at not less than fair market value of the common stock on the date of grant and expired no later than ten years from the date the option was granted. As of December 31, 2006, the Company had options outstanding to purchase 2,900,343 shares of its common stock, including options to purchase 75,000 shares of the Company's common stock granted outside of the 2001 Stock Option Plan, had outstanding 294,089 restricted shares of common stock and had zero shares available for future issuances under the 2001 Stock Option Plan.

In March 2006, the Company terminated the 2001 Stock Option Plan and adopted the Synta Pharmaceuticals Corp. 2006 Stock Plan (the 2006 Stock Option Plan). The 2006 Stock Option Plan provides for the grant of incentive stock options, nonstatutory stock options and nonvested stock to employees, officers, directors and consultants to the Company. A total of 2,406,248 shares of common stock have been reserved for issuance under the 2006 Stock Option Plan. The administration of the 2006 Stock Option Plan is under the general supervision of the board of directors. The exercise price of the stock options is determined by the board of directors, provided that incentive stock options are granted at not less than fair market value of the common stock on the date of grant and expire no later than ten years from the date the option is granted. Options generally vest over four years. The Company issues stock from its unissued stock pool to satisfy stock option exercises. As of December 31, 2006, the Company had options outstanding to purchase 67,750 shares of its common stock, had outstanding 12,142 restricted shares of common stock, and had 2,326,356 shares available for future issuances under the 2006 Stock Option Plan.

In February 2006, the Company's board of directors authorized the amendment of 933,075 stock options outstanding as of March 1, 2006 for active employees, board of directors and consultants under the 2001 Stock Option Plan having an exercise price of $16.00 and above to provide for such options to have an amended exercise price equal to the then fair value of $14.00 per share. The amendment affected 159 option holders, of which 150 were employees. The amendment was accounted for in the same manner as the cancellation of existing options and the grant of new options. The Company recognized compensation expense, in the amount of approximately $269,000, to reflect the incremental compensation for vested options in connection with the re-pricing and $138,000 of additional compensation in the year ended December 31, 2006 to reflect the amortization of the incremental compensation for the unvested options. As of December 31, 2006, the total amount of unrecognized additional stock-based compensation expense in connection with the amended shares is $313,000 and will be recognized over a weighted average period of 3 years.

Non-Vested ("Restricted") Stock Awards With Service Conditions

The Company's share-based compensation plan provides for awards of restricted shares of common stock to officers, other employees and non-employee directors. Restricted stock awards are subject to forfeiture if employment terminates during the prescribed retention period. The remaining unrecognized compensation expense on restricted stock at December 31, 2006 was $4,553,000. The weighted average period over which the balance is expected to be recognized is 2 years. Vesting may accelerate upon the Food and Drug Administration approval of the Company's first New Drug Application.

General Option Information

The following table summarizes stock option activity during the years ended December 31, 2006, 2005 and 2004:

	2006			2005			2004		
	Options available for Grant	Shares	Weighted average exercise price of shares under plan	Options available for Grant	Shares	Weighted average exercise price of shares under plan	Options available for Grant	Shares	Weighted average exercise price of shares under plan
Outstanding at January 1	384,241	2,947,683	$ 13.92	876,402	2,512,112	$ 11.80	1,894,489	1,923,868	$ 10.20
Granted	(763,129)	750,987	14.00	(801,164)	704,575	22.00	(1,131,593)	766,593	15.72
Exercised	—	(125)	16.00	—	—	—	(32,421)	(32,421)	10.84
Cancelled(1)	298,994	(655,477)	15.84	309,003	(269,004)	15.32	145,927	(145,928)	11.08
Additional shares reserved(2)	2,406,250	—	—	—	—	—	—	—	—
Outstanding at December 31	2,326,356	3,043,068	$ 11.88	384,241	2,947,683	$ 13.92	876,402	2,512,112	$ 11.80
Exercisable at December 31		2,010,830	$ 10.88		1,747,697	$ 10.80		1,234,349	$ 9.88

(1) In March 2006, the Company terminated the 2001 Stock Option Plan and cancelled the then 93,472 shares reserved for future issuance.

Options cancelled subsequent to the March 2006 termination of the 2001 Stock Option Plan do not return to the pool of options available for future issuance.

Includes the effect of stock option cancellations for the period prior to termination of the 2001 Stock Plan of 277,593 shares.

Includes the effect of non-vested restricted stock cancellations for the period prior to termination of the 2001 Stock Plan of 112,500 shares.

Includes the effect of stock option cancellations under the 2006 Stock Plan of 2,375 shares.

(2) In March 2006, the Company adopted the 2006 Stock Option Plan and authorized 2,406,250 shares for future issuance.

Included in the Company's stock options outstanding at December 31, 2006 are 354,681 options issued to non-employee consultants with a weighted average exercise price of $9.72 of which 325,743 are vested. The compensation expense is recorded over the respective vesting periods and is subject to variable accounting treatment prior to vesting, whereby the Company remeasures the fair value of the options at the end of each reporting period. Compensation expense related to these options was approximately $272,000, $1,142,000, $1,331,000 and $3,820,000 for the years ended December 31, 2006, 2005 and 2004, and for the period from inception (March 10, 2000) through December 31, 2006, respectively.

The following table summarizes information about outstanding and exercisable stock options at December 31, 2006:

	Options Outstanding				Options Exercisable			
Exercise price	Number outstanding	Weighted average remaining contractual life (years)	Weighted average exercise price per share	Aggregate intrinsic value	Number exercisable	Weighted average remaining contractual life	Weighted average exercise price per share	Aggregate intrinsic value
$ 2.00	164,762	4.91	$ 2.00	$ 1,977,144	164,762	4.91	$ 2.00	$ 1,977,144
10.84	1,406,537	6.07	10.84	4,444,656	1,373,282	6.07	10.84	4,339,571
14.00	1,471,769	8.38	14.00	—	472,786	7.51	14.00	—
	3,043,068	7.13	$ 11.88	$ 6,421,800	2,010,830	6.31	$ 10.88	$ 6,316,715

The following table summarizes the stock-based payment awards to employees during 2006:

Recipient	Month Granted	Shares	Per Share Exercise/ Price	Per Share Fair Value	Per Share Intrinsic Value
Employees	January 2006	1,150	$ 14.00	$ 14.00	$ —
Employees	February 2006	677,587	14.00	14.00	—
Employees	March 2006	4,500	14.00	14.00	—
Employees	May 2006	17,400	14.00	14.00	—
Employees	June 2006	6,075	14.00	14.00	—
Employees	July 2006	9,625	14.00	14.00	—
Employees	August 2006	10,750	14.00	14.00	—
Employees	September 2006	6,625	14.00	14.00	—
Employees	October 2006	17,275	14.00	14.00	—
Total		750,987			

General Restricted Shares Information

The following table summarizes restricted stock activity during the years ended December 31, 2006, 2005 and 2004:

	2006		2005		2004	
	Shares	Weighted average grant date fair value	Shares	Weighted average grant date fair value	Shares	Weighted average grant date fair value
Outstanding at January 1	421,589	$ 20.32	365,000	$ 22.00	—	—
Granted	12,142	$ 14.00	96,589	$ 14.76	365,000	$ 22.00
Exercised	—	—	—	—	—	—
Cancelled	(127,500)	$ 18.08	(40,000)	$ 22.00	—	—
Outstanding at December 31	306,231	$ 21.04	421,589	$ 20.32	365,000	$ 22.00

In April 2006, stock options to purchase 125 shares of the Company's common stock were exercised, resulting in proceeds of $2,000.

(8) Accrued Expenses

Accrued expenses consist of the following at December 31:

	2006	2005
	(in thousands)	
Contracted research costs	$ 3,052	$ 5,541
Compensation and benefits	1,196	887
Professional fees	1,451	1,537
Other	428	776
	$ 6,127	$ 8,741

(9) Income Taxes

Differences between the actual tax benefit and tax benefit computed using the United States federal income tax rate is as follows:

	Years ended December 31			Period from inception (March 10, 2000) through December 31, 2006
	2006	2005	2004	
	(in thousands)			
Income tax benefit at statutory rate	$ (19,472)	$ (23,414)	$ (15,618)	$ (80,797)
In-process research and development	—	—	—	6,331
Stock-based compensation	579	—	—	4,289
Tax credits	(1,743)	(2,232)	(1,434)	(6,910)
Other	40	33	20	466
Change in valuation allowance	20,596	25,613	17,032	76,621
Income tax benefit	$ —	$ —	$ —	$ —

The effects of temporary differences that give rise to significant portions of deferred tax assets and deferred tax liabilities at December 31, are presented below:

	2006	2005
	(in thousands)	
Deferred tax assets:		
Federal and state net operating loss carryforwards	$ 80,157	$ 60,054
Federal and state research and experimentation credits	8,310	6,422
Licenses	663	725
Depreciation and amortization	1,867	901
Deferred compensation	3,609	2,366
Other	743	536
Deferred tax assets	95,349	71,004
Less valuation allowance	(95,349)	(71,004)
Net deferred tax assets	$ —	$ —

The valuation allowance for deferred tax assets was approximately $95,349,000 and $71,004,000 as of December 31, 2006 and 2005, respectively. The increase in the total valuation allowance for the years ended December 31, 2006 and 2005, and for the period from inception (March 10, 2000) through December 31, 2006 was approximately $24,345,000, $30,367,000 and $95,349,000 respectively. The Company has established valuation allowances against its deferred tax assets because management believes that, after considering all of the available objective evidence, both historical and perspective, the realization of the deferred tax assets does not meet the "more likely than not" criteria under SFAS No. 109.

In 2005 and February 2007, the Company performed analyses to determine if there were changes in ownership, as defined by Section 382 of the Internal Revenue Code, that would limit its ability to utilize certain net operating loss and tax credit carryforwards. The Company determined that it experienced an ownership change, as defined by Section 382, in connection with its acquisition of Principia Associates, Inc. on September 20, 2002, but did not experience a change in ownership upon the effectiveness of the Company's initial public offering. As a result, the utilization of the Company's federal tax net operating loss carryforwards generated prior to the ownership change is limited. As of December 31, 2006, the Company has net operating loss carryforwards for U.S. federal tax purposes of approximately $201,430,000, after taking into consideration net operating losses expected to expire unused as a result of Section 382 limitations, and the remainder will expire in varying amounts through 2026 unless utilized. At December 31, 2006, the Company has state net operating loss carryforwards of approximately $186,136,000, which will expire through 2010 unless utilized. The utilization of these net operating loss carryforwards may be further limited if the Company experiences future ownership changes as defined in Section 382 of the Internal Revenue Code. At December 31, 2006, the Company had approximately $6,950,000 and $1,360,000, respectively, in federal and state research and development credits which expire through 2026 and 2021, respectively.

(10) Commitments and Contingencies

Leases

The Company leases its research and office facilities under non-cancelable operating leases with terms expiring through 2011. Each of these leases contains renewal options ranging from one to five years.

In August 2006, the Company renewed a lease for one of its research and office facilities for one year.

In August 2006, the Company renewed a lease for one of its research and office facilities for a five-year term with a five-year renewal option.

In December 2006, the Company entered into a new lease for a research and office facility. This lease expires in October 2011, but the Company may extend for an additional two years or terminate any time after three years provided the Company repays the unamortized portion of landlord-funded tenant improvements and landlord's legal fees.

The Company subleased laboratory and office space from its scientific founder, who is a major shareholder of the Company, under a tenant-at-will arrangement. This lease was assumed by the Company in May 2005. In January 2007, the Company entered into an early termination agreement for this research and office facility under which the Company was obligated to pay the landlord $68,000 for termination fees and expenses.

In November 2004, the Company entered into an agreement for a revolving property and equipment lease line of credit which was amended in 2005. Under the amended agreement, the Company may periodically directly lease, or sell and lease-back, up to $6.0 million of property and equipment, with payment periods of 36 or 48 months and a $1.00 purchase option at the end of each lease period. The lease rates are based upon a fixed base interest rate plus the respective prevailing 36- or 48-month U.S. Treasury Bill interest rates at the time of each funding. The leases are accounted for as capital leases. In 2006 and 2005, the Company sold and leased back under this agreement an aggregate of approximately $7.5 million of its previously purchased property and equipment, of which approximately $2.8 million and $4.7 million were capitalized and are being paid over 36 and 48 months, respectively. As a result, the Company recorded a net deferred gain of approximately $112,000, which is being amortized over the applicable lease periods. As of December 31, 2006, approximately $1.2 million was available under this lease line for future property and equipment expenditures. The Company also leases certain vehicles and equipment under various other non-cancellable capital and operating leases.

Future minimum payments, excluding operating costs and taxes, under the Company's capital and non-cancellable operating leases, excluding the terminated lease, are approximately as follows (in thousands):

	Capital leases	Operating leases
Years ended December 31,		
2007	$ 2,774	$ 2,079
2008	2,097	1,034
2009	1,207	905
2010	178	890
2011	—	826
Total minimum lease payments	6,256	$ 5,734
Less: amount representing interest	(756)	
Present value of minimum capital lease payments	5,500	
Less current portions of capital lease obligations	(2,330)	
Capital lease obligations—long term	$ 3,170	

Rent expense was approximately $1,914,000, $2,217,000 and $1,338,000 and $6,852,000 for the years ended December 31, 2006, 2005 and 2004, and for the period from inception (March 10, 2000) through December 31, 2006, respectively, including rent paid for the lease from its scientific founder in the amounts of approximately $0, $96,000, $213,000 and $691,000, respectively.

License Agreements

Queen's Medical Center

In March 2003, the Company entered into an exclusive, royalty-bearing license agreement with Queen's Medical Center (QMC) for certain technology related to ion channel technologies. The Company paid QMC cash of $40,000 and issued 18,444 shares of its common stock. The total consideration paid of approximately $240,000 was expensed immediately to research and development costs. Under the terms of the Agreement, if certain milestones are met, the Company is obligated to make cash payments of up to an aggregate of $1.0 million. If commercialization is achieved, the Company will be required to pay royalties to QMC on the net sales of any product using the licensed technologies. In the event the Company grants a sublicense of the licensed technology, the Company is obligated to compensate QMC a percentage of all fees received from the sublicense.

Through December 31, 2006, no milestone, royalty, or sublicense payments had been earned by or paid to QMC.

Beth Israel Deaconess Medical Center

In connection with its acquisition of Diagon in December 2002 (see note 3), the Company acquired two exclusive licenses relating primarily to monoclonal antibodies and ion channel technologies, respectively, in return for payment of cash and 46,111 shares of its common stock to Beth Israel Deaconess Medical Center (Beth Israel). The total value of the stock of $500,000 was expensed immediately by the Company to research and development costs. Under the terms of the licenses, if

certain milestones are met, the Company is required to make cash payments up to an aggregate of $2.0 million. If commercialization is achieved, the Company will be required to pay royalties on the net sales of any product using the licensed technologies. In the event the Company grants a sublicense of the licensed technologies, the Company is obligated to compensate Beth Israel a percentage of all fees received from the sublicense.

As a result of the Diagon acquisition, the Company also assumed an exclusive license with Beth Israel to specific know-how relating to certain calcium channels. Under the terms of the agreement, if certain milestones are met, the Company is required to make cash payments up to an aggregate of $800,000. If commercialization is achieved, the Company will be required to pay royalties on the net sales of any product using the licensed know-how.

Through December 31, 2006, no milestone, royalty or sublicense payments had been earned by or paid to Beth Israel.

Dana-Farber Cancer Institute

In July 2002, the Company entered into an exclusive license agreement with Dana-Farber Cancer Institute (DFCI) for certain patent rights relating to the use of immune system modulators with other agents for use against cancer. The Company paid DFCI cash of approximately $30,000 and issued 50,000 shares of its common stock. The total consideration paid of approximately $572,000 was expensed immediately to research and development costs. Under the terms of the agreement, if certain milestones are met, the Company is required to make cash payments up to an aggregate of $600,000. If commercialization is achieved, the Company will be required to pay nominal royalties on the net sales of any product using the licensed technologies.

Through December 31, 2006, no milestone, royalty or sublicense payments had been earned by or paid to DFCI.

Consulting Agreements

In July 2002, the Company entered into a consulting agreement with a member of its scientific advisory board (SAB), which was amended and restated effective January 1, 2004. The agreement has an initial term of two years from the amendment date and automatically extends for additional one-year terms unless thirty days' written notice is given by either party. In addition to an annual consulting fee, in the event the Company executes a transaction during the first two years of the consulting agreement in which the Company grants a license or other right of certain defined intellectual property, the SAB member is entitled to a one-time bonus payment of $150,000 and a portion of any up-front license fee, milestone payments or equity payments to purchase the Company's common stock over a certain defined amount related to the license transaction. The bonus and milestone payments may be paid in either cash or common stock, at the Company's discretion. In addition, the Company will pay QMC a portion of any committed research payments received by the Company that directly relate to the intellectual property, provided that the research agreement with QMC remains in effect when such payment is received by the Company. The SAB member may be entitled to a retention bonus of $1.0 million in the event the Company is acquired or there is a sale of substantially all of the assets related to the consulting agreement, subject to certain limitations.

In October 2002, the Company entered into a consulting agreement with an SAB member for scientific advisory services which was amended in October 2003. Under the amended consulting agreement, the term is four years from the effective date of the amendment, and for a one-time payment of $400,000, a one-time bonus payment based on the achievement of a certain performance milestone was eliminated. In addition to an annual consulting fee, the consultant is entitled to a bonus payment of a portion of any up-front or milestone payments received by the Company related to calcium channel technology during the four-year term of the amended agreement.

Guarantees

As permitted under Delaware law, the Company's Certificate of Incorporation and Bylaws provide that the Company will indemnify certain of its officers and directors for certain claims asserted against them in connection with their service as an officer or director. The maximum potential amount of future payments that the Company could be required to make under these indemnification provisions is unlimited. However, the Company has purchased a directors' and officers' liability insurance policy that reduces its monetary exposure and enables it to recover a portion of any future amounts paid. The Company believes the estimated fair value of these indemnification arrangements is minimal.

The Company customarily agrees in the ordinary course of its business to indemnification provisions in agreements with clinical trials investigators in its drug development programs, in sponsored research agreements with academic and not-for-profit institutions, in various comparable agreements involving parties performing services for the Company in the ordinary course of business, and in its real estate leases. The Company also expects to agree to certain indemnification provisions in any drug discovery and development collaboration agreements. With respect to the Company's clinical trials and sponsored research agreements, these indemnification provisions typically apply to any claim asserted against the investigator or the investigator's institution relating to personal injury or property damage, violations of law or certain breaches of the Company's contractual obligations arising out of the research or clinical testing of the Company's compounds or drug candidates. With respect to lease agreements, the indemnification provisions typically apply to claims asserted against the landlord relating to personal injury or property damage caused by the Company, to violations of law by the Company or to certain breaches of the Company's contractual obligations. The indemnification provisions appearing in collaboration agreements are similar, but in addition provide some limited indemnification for its collaborator in the event of third-party claims alleging infringement of intellectual property rights. In each of the cases above, the term of these indemnification provisions generally survives the termination of the agreement, although the provision has the most relevance during the contract term and for a short period of time thereafter. The maximum potential amount of future payments that the Company could be required to make under these provisions is generally unlimited. The Company purchased insurance policies covering personal injury, property damage and general liability that reduce its exposure for indemnification and would enable it in many cases to recover a portion of any future amounts paid. The Company has never paid any material amounts to defend lawsuits or settle claims related to these indemnification provisions. Accordingly, the Company believes the estimated fair value of these indemnification arrangements is minimal.

(11) Related Party Transactions

In January 2005, the Company entered into an Agreement and Release with its scientific founder, who is a board member, whereby all outstanding matters regarding various oral understandings and arrangements between the scientific founder and the Company were resolved, including arrangements relating to (1) the assignment by the scientific founder of the benefit of his interests, if any, resulting from the Company's acquisition of the net assets of CKS, (2) the scientific founder's assignment of inventions, non-competition, non-solicitation and confidentiality agreements with the Company, and (3) a release by the scientific founder of any and all claims that the scientific founder may have had against the Company. Pursuant to this agreement, the Company is paying the scientific founder $500,000, payable in $25,000 installments quarterly for five years. The full amount of the obligation was charged to research and development expense in 2005.

The Company pays its scientific founder and a member of the board consulting fees of approximately $25,000 per month. Total consulting fees paid in 2006, 2005 and 2004 were approximately $300,000 each year.

During 2002 and 2001, the Company contracted with a company owned by the Company's scientific founder, board member and significant shareholder to provide drug development testing services. Amounts advanced under this arrangement totaled $500,000 and $1.0 million as of December 31, 2002 and 2001, respectively. During 2003 and 2002, all advances were paid back to the Company as no services were ever performed.

On August 23, 2002 and September 11, 2002, the Company issued two promissory notes receivable of $500,000 each to SBR (a wholly-owned subsidiary of Principia). The promissory notes had a fixed interest rate of 7% and were due on December 31, 2002. The promissory notes were forgiven in connection with the Company's acquisition of Principia (see note 3).

(12) Retirement Plan

In 2003, the Company implemented a 401(k) retirement plan (the Synta 401(k) Plan) in which substantially all of its permanent employees are eligible to participate. Participants may contribute a percentage of their annual compensation to the plan, subject to statutory limitations. The Company may declare discretionary matching contributions to the Synta 401(k) Plan.

In April 2006, the Company began matching participants' contributions up to 50% of the first 6% of the employee's salary. The match is subject to a three-year equally graded vesting schedule and any forfeitures will be applied to reduce the Company's contributions. Company contributions for the year ended December 31, 2006 were approximately $236,000, subject to forfeitures.

(13) Research Grant Contracts

In May 2003, the Company was awarded a $500,000 government contract with DARPA to perform research services associated with performance enhancement. As of December 31, 2003, the Company had recognized approximately $43,000 of research grant revenue for services performed under the terms of the contract, which expired in September 2004. In addition, the Company recorded deferred revenue of approximately $457,000, which represents advance payments received under this contract. The advance payments were deposited in a separate non-interest-bearing account and are recorded as restricted cash as of December 31, 2006 and 2005.

(14) Initial Public Offering Costs

During 2005 and 2004 the Company incurred $2,389,000 of costs in connection with its planned initial public offering of common stock, of which $1,084,000 was deferred at December 31, 2004. Following the Company's filing of its S-1 with the Securities and Exchange Commission in 2005, the Company determined that it would not complete the planned offering and withdrew its filing. The Company did not reactivate and complete its offering within 90 days of the withdrawal of the filing and, accordingly, these costs were expensed in 2005.

(15) Subsequent Events

Restricted Common Stock Buyback

In January 2007, the Company re-purchased 29,051 shares of its restricted common stock from certain officers and non-officer employees in order to fund the minimum statutory tax withholding requirements related to the vesting of 80,000 shares of restricted common stock.

Reverse Stock Split

In January 2007, the Board of Directors and the stockholders of the Company approved (i) a 1-for-4 reverse stock split, which was effected on February 2, 2007, (ii) an adjustment of the authorized common shares to 100,000,000, which became effective upon the completion of an initial public offering of the Company's common stock, and (iii) an adjustment in the number of common shares reserved under the 2006 Stock Option Plan to 2,500,000. All share data shown in the accompanying consolidated financial statements have been retroactively restated to reflect the reverse split.

Initial Public Offering

In February 2007, the Company raised $50.0 million in gross proceeds from the sale of 5,000,000 shares of its common stock in an initial public offering ("IPO") at $10.00 per share. The net offering proceeds after deducting approximately $5.3 million in expenses for underwriters' discounts, fees and commissions, legal, accounting, printing and listing and filing fees, and miscellaneous expenses were approximately $44.7 million. As of December 31, 2006, the Company had approximately $1.0 million in deferred IPO costs related to this offering. All outstanding shares of the Series A convertible preferred stock and $1.9 million in accumulated dividends were converted into 6,278,765 shares of common stock upon the effectiveness of the IPO. The unaudited pro forma balance sheet as of December 31, 2006 reflects the receipt of net offering proceeds of approximately $44.7 million and the conversion of the Series A convertible preferred stock into common stock.

In accordance with EITF No. 98-5, *Accounting for Convertible Securities with Beneficial Conversion Features or Contingently Adjustable Conversion Ratios*, and EITF No. 00-27, *Application of Issue No. 98-5 to Certain Convertible Instruments* , the Company will record a non-cash beneficial conversion charge of approximately $58.6 million in Q1 2007 in connection with the contingent adjustable conversion feature of the Series A convertible preferred stock.

(16) Quarterly Financial Data (unaudited)

The following tables present a summary of quarterly results of operations for 2006 and 2005:

	Three Months Ended			
	March 31, 2006	June 30, 2006	September 30, 2006	December 31, 2006
	(in thousands, except per share data)			
Net loss attributable to common stockholders	$ (16,211)	$ (14,891)	$ (14,733)	$ (13,294)
Basic and diluted net loss attributable to common stockholders per share	$ (0.73)	$ (0.67)	$ (0.66)	$ (0.60)
Basic and diluted weighted average number of common shares outstanding	22,219,025	22,224,772	22,226,964	22,230,033

	Three Months Ended			
	March 31, 2005	June 30, 2005	September 30, 2005	December 31, 2005
	(in thousands, except per share data)			
Net loss attributable to common stockholders	$ (17,857)	$ (19,044)	$ (16,470)	$ (15,492)
Basic and diluted net loss attributable to common stockholders per share	$ (0.80)	$ (0,86)	$ (0.74)	$ (0.70)
Basic and diluted weighted average number of common shares outstanding	22,245,430	22,254,481	22,256,070	22,257,547

Exhibit 10.27

COMPENSATION OF NAMED EXECUTIVE OFFICERS

Named Executive Officer	2006 Bonus	2007 Annual Base Salary*
Safi R. Bahcall, Ph.D. President and Chief Executive Officer	$ 100,000	$ 355,000
Keith S. Ehrlich, C.P.A. Vice President, Finance and Administration, Chief Financial Officer	$ 44,000	$ 230,000
James G. Barsoum, Ph.D. Senior Vice President, Research	$ 46,000	$ 240,000
Eric W. Jacobson, M.D. Senior Vice President, Clinical Research and Regulatory Affairs, Chief Medical Officer	$ 65,000	$ 280,000
Keizo Koya, Ph.D. Senior Vice President, Drug Development	$ 52,000	$ 270,000

* Effective March 1, 2007.

QuickLinks

Exhibit 10.27

Exhibit 21.1

SUBSIDIARIES OF SYNTA PHARMACEUTICALS CORP.

Synta Securities Corp., a Massachusetts securities corporation
Synta Limited Incorporated, a United Kingdom company

QuickLinks

Exhibit 31.1

CERTIFICATIONS UNDER SECTION 302

I, Safi R. Bahcall, Ph.D., certify that:

1. I have reviewed this annual report on Form 10-K of Synta Pharmaceuticals Corp.;

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

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

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

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

b) [reserved] / [paragraph omitted pursuant to SEC Release Nos. 33-8760 and 34-54942];

c) evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

d) disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's fourth fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

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

a) all significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

b) any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: March 28, 2007

/s/ SAFI R. BAHCALL

Safi R. Bahcall, Ph.D.
President and Chief Executive Officer
(principal executive officer)

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Exhibit 31.2

CERTIFICATIONS UNDER SECTION 302

I, Keith S. Ehrlich, certify that:

1. I have reviewed this annual report on Form 10-K of Synta Pharmaceuticals Corp.;

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

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

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

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

b) [reserved] / [paragraph omitted pursuant to SEC Release Nos. 33-8760 and 34-54942];

c) evaluated the effectiveness of the registrant's disclosure controls and procedures and presented in this report our conclusions about the effectiveness of the disclosure controls and procedures, as of the end of the period covered by this report based on such evaluation; and

d) disclosed in this report any change in the registrant's internal control over financial reporting that occurred during the registrant's most recent fiscal quarter (the registrant's fourth fiscal quarter in the case of an annual report) that has materially affected, or is reasonably likely to materially affect, the registrant's internal control over financial reporting; and

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

a) all significant deficiencies and material weaknesses in the design or operation of internal control over financial reporting which are reasonably likely to adversely affect the registrant's ability to record, process, summarize and report financial information; and

b) any fraud, whether or not material, that involves management or other employees who have a significant role in the registrant's internal control over financial reporting.

Date: March 28, 2007

/s/ KEITH S. EHRLICH

Keith S. Ehrlich, C.P.A.
Vice President, Finance and Administration,
Chief Financial Officer
(principal accounting and financial officer)

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Exhibit 31.2

Exhibit 32.1

CERTIFICATIONS UNDER SECTION 906

Pursuant to section 906 of the Sarbanes-Oxley Act of 2002 (subsections (a) and (b) of section 1350, chapter 63 of title 18, United States Code), each of the undersigned officers of Synta Pharmaceuticals Corp., a Delaware corporation (the "Company"), does hereby certify, to such officer's knowledge, that:

The Annual Report for the year ended December 31, 2006 (the "Form 10-K") of the Company fully complies with the requirements of Section 13(a) or 15(d) of the Securities Exchange Act of 1934, and the information contained in the Form 10-K fairly presents, in all material respects, the financial condition and results of operations of the Company.

Dated: March 28, 2007 /s/ SAFI R. BAHCALL

Safi R. Bahcall, Ph.D.
President and Chief Executive Officer
(principal executive officer)

Dated: March 28, 2007 /s/ KEITH S. EHRLICH

Keith S. Ehrlich, C.P.A.
Vice President, Finance and Administration,
Chief Financial Officer
(principal accounting and financial officer)

A signed original of this written statement required by Section 906 has been provided to the Company and will be retained by the Company and furnished to the Securities and Exchange Commission or its staff upon request.

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