Harbor BioSciences, Inc.
Form 10-K
January 20, 2012

UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

WASHINGTON, D.C. 20549

FORM 10-K

x **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934**
For the fiscal year ended December 31, 2011

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 001-34584

HARBOR BIOSCIENCES, INC.

(Exact name of Registrant as specified in its charter)

Delaware	**13-3697002**
(State or other jurisdiction of incorporation or organization)	**(I.R.S. Employer Identification No.)**

9191 Towne Centre Drive, Suite 409

San Diego, CA **92122**
(Address of principal executive offices) **(Zip Code)**
Registrant s telephone number, including area code: (858) 587-9333

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

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

Common Stock, par value $0.01 per share

Title of Class

Indicate by check mark if the Registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act.

YES ¨ NO x

Indicate by check mark if the Registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Act.

YES ¨ NO x

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 requirement for the past 90 days. YES x NO ¨

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files).

YES x NO ¨

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

Indicate by check mark whether the Registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer or a smaller reporting company. See definitions of large accelerated filer, accelerated filer and smaller reporting company in Rule 12b-2. (Check One).

Large accelerated filer ¨ Accelerated filer ¨

Non-accelerated filer ¨ Smaller reporting company x

Indicate by check mark whether the Registrant is a shell company (as defined in Rule 12b-2 of the Act). YES ¨ NO x

The aggregate market value of the voting stock held by nonaffiliates of the Registrant as of June 30, 2011, the end of the Company s most recently completed second fiscal quarter, was approximately $7,372,544 based on the closing stock price of $0.21 for the Registrant s common stock as reported by the OTC Bulletin Board*.

As of January 20, 2012, there were outstanding 35,422,140 shares of the Registrant s common stock, $.01 par value per share.

*Excludes the common stock held by executive officers, directors and stockholders whose ownership exceeded 10% of the Registrant s common stock outstanding at June 30, 2011. This calculation does not reflect a determination that such persons are affiliates for any other purposes.

DOCUMENTS INCORPORATED BY REFERENCE

None

Harbor BioSciences, Inc.

Form 10-K

For the Fiscal Year Ended December 31, 2011

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FORWARD-LOOKING STATEMENTS

This Annual Report on Form 10-K, and the information incorporated herein, contains forward-looking statements that involve and are subject to risks and uncertainties. In particular, statements about our expectations, beliefs, plans, objectives, assumptions or future events or performance are contained or incorporated by reference in this Annual Report on Form 10-K. We have based these forward-looking statements on our current expectations about future events. While we believe these expectations reflected in this Annual Report on Form 10-K are reasonable, the inclusion of any forward-looking statements should not be regarded as a representation that any of our plans will be achieved and such forward-looking statements are inherently subject to risks and uncertainties, many of which are beyond our control. Such forward-looking statements include, but are not limited to, statements preceded by, followed by or that otherwise include the words, believe, may, might, can, could, will, would , should, estimate, continue, anticipate, intend, seek, plan, project, expect, or similar expressions. The actual future results for Harbor BioSciences, Inc. may differ materially from those discussed here for various reasons, including those discussed in this Annual Report in Part 1, Item 1A under the heading Risk Factors, Part II, Item 7 entitled Management s Discussion and Analysis of Financial Condition and Results of Operations and elsewhere throughout this Annual Report on Form 10-K. Given these risks and uncertainties, you are cautioned not to place undue reliance on such forward-looking statements. The forward-looking statements included in this Annual Report are made only as of the date hereof. We do not undertake any obligation to update any such statements or to publicly announce the results of any revisions to any of such statements as a result of new information, to reflect future events or developments. When used in this Annual Report, unless otherwise indicated, we, our and us refers to Harbor BioSciences, Inc.

PART I

EXPLANATORY NOTE

On August 15, 2011, we filed a Form 15 (the Original Form 15) with the Securities and Exchange Commission (the SEC) certifying that, as of such date, there were fewer than 300 holders of record of our common stock. The Original Form 15 had the effect of terminating the registration of our common stock under the Securities Exchange Act of 1934, as amended (the Exchange Act), which was the first step in suspending our obligation to file current and periodic reports with the SEC. However, our duty to file current and periodic reports with the SEC was not suspended immediately upon filing the Original Form 15, due to the prior filing of certain registration statements under the Securities Act of 1933, as amended (the Securities Act) that were deemed to have been made effective during the fiscal year ended December 31, 2011 by the filing of our Annual Report on Form 10-K for the fiscal year ended December 31, 2010, which we filed with the SEC on March 30, 2011. As a result, following the filing of the Original Form 15, on October 28, 2011we filed a Current Report on Form 8-K, and on November 7, 2011 we filed a Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2011. In addition, we are filing this Annual Report on Form 10-K for the fiscal year ended December 31, 2011. However, we have taken certain actions required by SEC rules to ensure that we are not required by the Exchange Act to file current or periodic reports with the SEC with respect to the fiscal year ending December 31, 2012, which actions have included the filing of post-effective amendments to each of our registration statements filed under the Securities Act to terminate such registration statements (which post-effective amendments have been declared effective by the SEC), and the filing, on January 12, 2012, of a Form 15 to notify the SEC that we had fewer than 300 holders of record of our common stock as of December 31, 2011. In addition, we do not expect that we will be required to file current or periodic reports with the SEC with respect to any fiscal year following the fiscal year ending December 31, 2012.

Item 1. Business

GENERAL OVERVIEW

Harbor BioSciences, Inc. (Harbor BioSciences or the Company), a clinical-stage pharmaceutical company, is engaged in the discovery and development of products for the treatment of diseases that typically onset with age. Our current development efforts are primarily focused on a novel series of hormone-related sterols that are derived from the human adrenal metabolome.

We are a development-stage company with two product candidates which recently completed Phase I/IIa clinical trials: Apoptone® (HE3235) in patients with late-stage prostate cancer, and Triolex® (HE3286) in obese type-2 diabetes mellitus patients. Apoptone and Triolex represent two of the lead candidates from Harbor BioSciences technology platform based on endogenous human sterols and their metabolites.

Drawn from our unique and proprietary platform, our research program has identified additional lead candidates that are active in preclinical models of cancer, metabolic conditions, autoimmune conditions, lung, ocular and neuro-inflammation, bone degeneration and organ regeneration.

Our principal executive offices are located at 9191 Towne Centre Drive, Suite 409, San Diego, California 92122, and our telephone number is (858) 587-9333. We incorporated in Delaware in 1992.

On March 26, 1997, Hollis-Eden, Inc., a Delaware corporation, was merged with and into us, then known as Initial Acquisition Corp. (IAC), a Delaware corporation. Upon consummation of the merger of Hollis-Eden, Inc. with IAC, Hollis-Eden, Inc. ceased to exist and IAC changed its name to Hollis-Eden Pharmaceuticals, Inc.

Effective February 9, 2010, we changed our name from Hollis-Eden Pharmaceuticals, Inc. to Harbor BioSciences, Inc. The name change was effected pursuant to Section 253 of the General Corporation Law of the State of Delaware by the merger of our wholly owned subsidiary into us. We were the surviving corporation and, in connection with the merger, we amended our Amended and Restated Certificate of Incorporation to change our name to Harbor BioSciences, Inc.

Effective February 18, 2010, our common stock began trading under a new NASDAQ symbol, HRBR and CUSIP number 41150V 103 . Our common stock was then delisted from the NASDAQ Stock Market at the opening of business on September 23, 2010. Our shares then traded on the OTC Bulletin Board (OTCBB), until August 17, 2011, when our common stock was delisted from the OTCBB as a result of our filing a Form 15 pursuant to SEC Rule 12g-4(a)(i), and subsequently became available for trading on the OTC Markets Group, Inc., informally known as the Pink Sheets® under the trading symbol HRBR.PK and CUSIP number 41150V 202 . On July 28, 2011, we sold an aggregate of 2,000,000 shares of our Series A Preferred Stock (the Redeemable Preferred Shares) to Amun, LLC, a Delaware limited liability company (the Investor) pursuant to the terms of a Stock Purchase Agreement (the Purchase Agreement) and related Stockholders Agreement (the Stockholders Agreement). The Redeemable Preferred Shares represent approximately a 28% of the economic interest in the Company and also entitle the Investor to a number of votes equal to 38.28% of the total number of votes entitled to be cast by holders of all shares of the Company s capital stock (including the Common Stock and Series A Preferred Stock) voting together as single class. Under the terms of these and other related agreements between us and the Investor, the Investor placed $2.825 million in cash into an escrow account (Escrow), which amount is available under certain circumstances to pay certain Company related expenses and to fund our working capital needs. The Stockholder Agreement provides that the Investor will have the right to put the Redeemable Preferred Shares acquired pursuant to the Purchase Agreement back to us in return for the remaining cash held in Escrow at the time of the put, upon the occurrence of certain events.

As contemplated by the Purchase Agreement and the Stockholders Agreement, the Investor intends to bring an offer to us for us to acquire a controlling interest in a profitable entity, which transaction would provide to the Company at least $5,000,000 in cash plus an amount equal to the costs and expenses incurred by the Company in connection with such transaction (not to exceed $200,000), which amounts, together with any operating cash held by us immediately prior to closing such transaction, would be transferable, together with any and all (i) intellectual property and (ii) other assets of the Company related to our biotechnology business, to a newly formed subsidiary of the Company, Harbor Therapeutics Inc., which subsidiary will assume all liabilities of the Company as of immediately prior to such closing (a Qualifying Transaction). The Company expects that the closing of a Qualifying Transaction would provide access to capital and the continuation of its existing business. In addition, the acquisition of a controlling interest in a profitable entity would provide diversification for its shareholders.

On October 26, 2011, we completed the reverse and forward stock splits which were approved by our shareholders at our annual meeting. As a result, the Company purchased the 43,698 common shares that were cancelled, at the previous ten-day average closing price of $0.142 for a total of $6,205. In addition, as a result of the stock splits, the investors in our June 2010 registered direct offering of common stock and warrants became eligible to exercise a put right under the warrants, which entitled them to put the warrants back to us in return for a cash payment equal to the fair value of the warrants as determined by reference to a formula set forth in such warrants. All of the warrant holders exercised their put right and the Company purchased the warrants at a price of $0.0955 for each underlying share for a total of $337,679. An amount equals to the cost of the cancelled common shares and the warrants purchased was distributed to us from the Escrow account established with the funds from the sale of

Preferred Shares to Amun. Harbor BioSciences, Triolex, Apoptone, and the Harbor BioSciences stylized logo are trademarks of Harbor BioSciences, Inc. This filing also includes trademarks owned by other parties. All other trademarks mentioned are the property of their respective owners. Use or display by us of other parties trademarks or products is not intended to and does not imply a relationship with, or endorsements or sponsorship of, us by the trademark or product owners.

Our periodic and current reports that we filed with the SEC are available free of charge, on our website. Our internet address is www.harborbiosciences.com. The reference to our website does not constitute incorporation by reference of the information contained on our website.

Harbor BioSciences Approach

Under conditions of stress, chronic infections or systemic inflammation, it is believed that changes in the profile of adrenal products, and the metabolism of these products, perturb signaling pathways in peripheral tissues to drive the growth of certain tumors and are causative to diseases of advancing age. These age-related diseases include metabolic syndrome, autoimmune diseases, immune-mediated inflammatory diseases and an impaired ability to fight infections. Our development strategy is based on the hypothesis that hormone-derived products are critical to the regulation of the body s complex biological systems. We believe that in young, healthy adults, adrenal products, such as cortisol, progesterone, dehydroepiandrosterone (DHEA), and its metabolome, which includes estrogen and testosterone, provide important signals for proper engagement and regulation of human biological processes.

Today, most drug developers take a *ground up* approach by first striving to intellectualize and identify critical components in the intricate functional biochemical cascades, and then attempting to design drugs that can successfully block or stimulate those specific pathways. This approach presumably results in validated molecular targets for specific diseases. While this approach has resulted in a number of successful drugs, uses of these drugs are often limited by serious side effects due to unanticipated off-target activities. In contrast, ours is a *top down* forward pharmacology approach that begins with the identification of previously unappreciated members of the human sterol metabolome. Then, by applying sophisticated drug development methodology, we develop novel compounds that modify critical endocrine pathways intrinsic to the activities of the native endogenous molecules. Our top-down methodology is based on a successful historical approach to drug discovery as applied to the early development of human hormones. We continue to apply this approach, which has the potential to produce new pharmaceutical product candidates to treat a myriad of diseases associated with advancing age, to our new discoveries in the vast, unexplored human metabolome. We believe that by reapplying this previously successful, but now neglected, top-down drug development principle the potential exists to produce pharmaceuticals that should address numerous important markets, including many with unmet medical needs.

TECHNOLOGY

Platform

Our primary technology is focused on developing novel series of hormone-related sterols that are useful for treating a wide variety of medical conditions. Many of these compounds are either depleted or elevated during advancing age, which are processes accelerated by infectious diseases and chronic inflammatory disorders. In certain indications, high plasma concentrations of these hormones are positively correlated with attenuated disease and the maintenance of good health.

The chemistry and biochemistry of steroids have been extensively studied and utilized in the development of various drugs, especially for treating hormonal imbalances, infections, and cancer, as well as inflammatory conditions. Harbor BioSciences chemical inventory of greater than 700 sterol compounds represents a targeted chemical library derived from components of the mammalian metabolome. Many of the library compounds were previously unknown metabolome components. Other library members include compounds having novel structures based upon those metabolic products with potentially improved pharmaceutical properties. We believe this library contains many unique chemical structures with diverse biological properties and represents the largest sample of compounds associated with the DHEA metabolome.

Our targeted chemical library contains drug-like molecules since they were designed to have useful pharmaceutical properties, including improved oral bioavailability and metabolic and chemical stability. The library members further provide lead compounds for additional new chemical compositions that will expand our intellectual property portfolio. In addition, the library compounds are selected for facile and cost-effective syntheses of those newly derived chemical compositions by taking into consideration future commercialization.

OUR DRUG CANDIDATES IN DEVELOPMENT

We are currently focused on the development of proprietary synthetic sterol derivatives derived from the human 19-carbon steroid scaffold of the mammalian sterol metabolome. We have conducted clinical trials with our lead drug development candidates: Apoptone (HE3235), for late-stage prostate cancer; Triolex (HE3286), for the treatment of obese type-2 diabetes, other metabolic disorders and autoimmune conditions; Neumune (HE2100), for the treatment of sepsis, which is a condition that arises with excess radiation exposure; and HE2000, for the prevention of opportunistic infections in immune-suppressed patients. Each of those compounds is described in more detail below. In addition, our research program focuses on the identification and characterization of new members of the sterol hormone metabolome that may result in future pharmaceutical candidates or nutraceutical products.

Apoptone (HE3235)

Prostate Cancer

Apoptone is a second-generation compound that we selected for clinical development in the area of hormone-associated cancers, such as prostate cancer. Approximately 234,000 patients are diagnosed with prostate cancer each year and global sales for leading prostate cancer drugs range approximately from $500 million to $1 billion annually. Apoptone was discovered by screening our proprietary chemical library against the LNCaP prostate cancer cell line. Its selection was based on a combination of its activity against tumor cells and desirable pharmaceutical properties. Apoptone has been tested in a number of preclinical cancer models and has shown indications of activity in controlling the incidence, growth and development of new tumors in these models. We believe that Apoptone is a disease-modifying agent that may directly induce apoptosis, or cell death, in tumor cells and differs from traditional hormone blockade therapies that interrupt the tumor cell growth signaling through direct androgen or estrogen receptor-mediated mechanisms. While hormone-blockade therapy can effectively control prostate cancer for a period of time, it often fails and the cancer growth resumes by spreading to other organs: usually the bone.

In 2008, we initiated a Phase I/IIa clinical trial with Apoptone in late-stage castrate resistant prostate cancer (CRPC) patients who have failed hormone therapy and at least one cycle of cytotoxic chemotherapy. In December 2009, the trial was amended to include a group of CRPC patients with progressive disease that have not been previously treated with chemotherapy. The open-label dose ranging clinical trial was conducted in various clinical sites including some within the Prostate Cancer Clinical Trial Consortium (PCCTC). The clinical trial is complete. Safety, tolerance, pharmacokinetics and potential activity of Apoptone was evaluated when the drug was administered twice daily in late-stage prostate cancer patients. The potential activity of the drug was measured by its effect on time to disease progression, as determined by prostate-specific antigen (PSA) blood tests, computerized tomography (CT), magnetic resonance imaging (MRI), or bone scintigraphy, and its effect on circulating tumor cells (CTC). Biological activity was found at the lowest dose studied (10 mg) and no dose-limiting toxicity was observed during a 70-fold dose-ranging study with the exception that at the highest dose tested (700 mg) a patient s concomitant medication produced a drug-drug interaction that presented safety concerns with the use of yet higher doses. Apoptone is now staged for Phase IIb clinical trials.

Breast Cancer

We are also exploring the potential for Apoptone to treat breast cancer. In the MNU-induced pre-clinical models of breast cancer, Apoptone successfully treated established tumors and prevented the formation of new tumors. It appeared to be synergistic when given in combination with concurrent taxane chemotherapy. A report on the pre-clinical activity in breast cancer recently published in the peer-reviewed scientific literature.

Apoptone Development Status

Apoptone is manufactured starting from androsterone. The active pharmaceutical ingredient is formulated to an oral dosage form using standard excipients approved for oral dosage products. Non-clinical toxicology studies have been done that enable the use of Apoptone in clinical studies in late-stage prostate cancer and breast cancer patients using 28-day cycles of therapy. Encouraging data were first reported from the Phase I/IIa clinical trial in castration resistant prostate cancer also referred to as hormone resistant prostate cancer at the ASCO Genitourinary Cancers Symposium in San Francisco, March 6, 2010. Preliminary results from the study, conducted in part with participating member sites of the PCCTC, were first reported on November 16, 2009. The phase I/IIa trial was an open-label study with the primary objectives of assessing safety, tolerability, pharmacokinetics and activity of Apoptone in men with CRPC having an ECOG performance status score of less than or equal to 2 (ambulatory and capable of at least self-care). Patient cohorts are defined by oral daily doses of 10 mg, 20 mg, 30 mg, 50 mg, 100 mg, 200 mg, 350 mg and 700 mg. Subjects were treated until toxicity or disease progression, as assessed by CT and bone scans obtained every two cycles. Based on encouraging signs of activity, the PCCTC recommended an extension of the current trial into CRPC patients that had not been treated with cytotoxic chemotherapy. Accordingly, the subject eligibility criteria were amended to include earlier-stage, chemotherapy-naïve patients in 100 mg and 350 mg expansion cohorts. The clinical trial is now complete.

There were 68 patients enrolled in the trial on an intent-to-treat basis. There were 42 taxane-resistant prostate cancer patients entered into the clinical trial at 7 dose levels. Of these 28 (67%) reached their first reassessment (two 28-day cycles), 15 (58%) of these had stable disease on scans or imaging and have received up to 9 additional treatment cycles before disease progression. The Kaplan-Meier estimate for the median time to progression is 15.9 weeks (range 8-24) for this trial. Due to early signs of activity, the 20 mg dose group was expanded to include 14 taxane resistant patients. Eleven of these were evaluable with an actual median time to progression of 19.7 weeks (range 8-24).

In order to gain information on the healthier pre-chemotherapy patients and the tolerability of Apoptone at higher dose levels, twenty six chemotherapy naïve patients were enrolled to the protocol. The 100 mg and 350 mg dose groups were each expanded with 11 additional pre-chemotherapy patients and 4 were enrolled into the 700 mg group. Twenty two of the 26 chemotherapy naive patients (85%) completed more than one 28-day treatment cycle. These patients all had a re-assessment at the completion of the second cycle. Sixteen (73%) had stable disease and received up to 12 additional cycles of therapy and 6 (27%) had progressive disease. The Kaplan-Meier estimate for the median time to progression (TTP) in the chemotherapy naive cohort is > 24 weeks. In the individual 100 and 350 mg dose expansion cohorts, 10 of the 11 patients in each arm completed 2 or more 28-day treatment cycles. The Kaplan-Meier estimate for the median time to progression for the 10 patients that completed 2 or more cycles in each cohort was 24 weeks in the 100 mg cohort; (21, > 24) and 24 weeks (16, 35) in the 350 mg cohort. One patient in the 350 mg group achieved a sustained partial clinical response (100% decrease) of limited nodal (11 mm) disease. The non-target lesions were stable. The response was observed on the first re-evaluation and continued through study closure (8 completed treatment cycles).

Circulating tumor cells (CTC) were evaluated in fifty patients at baseline. Twenty nine (29) patients had < 5 cells per 7.5 mL (favorable) and 21 with ³ 5 cells (unfavorable). After Apoptone treatment, 25 (86%) of those with favorable counts maintained those levels at 4 weeks, and 7 (33%) of those with ³ 5 cells had a decrease to < 5 cells at 4 weeks. Twenty eight patients had CTC enumeration performed at 12 weeks. Of these 16 (84%) maintained < 5 cells and 6 (66%) patients with baseline CTC ³ 5 cells converted to < 5 cells. These results indicate that the majority of patients either maintained a stable disease state or improved in disease status through the 12 week evaluation period. Although the number of patients is small, CTC count declines from ³ 5 to < 5 have been previously associated with improved overall survival.

Changes in PSA levels were consistent with the properties of a tumor-differentiating agent. PSA declines were anticipated to be rare based on data from *in vitro* and pre-clinical studies, which demonstrated PSA expression generally increased concurrent with tumor growth suppression. In this study, Apoptone rarely induced PSA declines, with less than 10% of patients having a greater than 50% decline in PSA at some time during the course of their treatment. Therefore, PSA is not an appropriate surrogate marker for the evaluation of disease status in Apoptone treated patients.

In summary, Apoptone was well-tolerated and no overt dose-limiting toxicities were reported. The Kaplan-Meier estimate for the median time to progression is 15.9 weeks (range 8-24) for this trial. The Kaplan- Meier estimate for the median time to progression in the chemotherapy naive cohort is > 24 weeks. The eleven evaluable taxane chemotherapy-resistant patients had a median time to progression of 19.7 weeks (range 8-24). There was one sustained partial response observed, and by CTC evaluation, a majority of the patients either maintained a stable disease state or improved in disease status through the 12 week evaluation period. The mechanism of action has been partially elucidated with biochemical molecular points of interaction identified.

Apoptone is a molecular entity that represents a new therapeutic approach for the treatment of hormone-associated cancers and is expected to have a more favorable side effect profile than found with presently approved treatments. Several patents have been obtained for pharmaceutical formulations of Apoptone and its use for the treatment of prostate cancer, breast cancer and benign prostate hypertrophy.

Competition

Two forms of taxane chemotherapy are presently approved to treat castrate-resistant prostate cancer. Despite these and other current treatments, there is an ongoing need for novel oral agents that can control prostate cancer progression after conventional therapies or hormone treatments fail. Recently, PROVENGE®, produced by Dendreon, Inc., an autologous immune cell therapy that primes the patient s cells against prostate cancer, was approved. Many forms of prostate cancer are dependent on androgen receptor signaling and are responsive to low levels of androgens that remain after hormone ablation therapy. Abiraterone® produced by Cougar Biotechnology, Inc. (acquired by Johnson & Johnson), also a recently approved agent, impedes the synthesis of androgens by inhibition of an enzyme that transforms a precursor molecule into androgenic and estrogenic hormones. MDV-3100, produced by Medivation, Inc., is in clinical development and is an agent that strongly inhibits the action of androgens by interfering with the androgen receptor thereby inhibiting tumor growth. In addition, there are a number of companies with drug candidates in Phase III clinical trials that are targeting the late-stage castration-resistant prostate cancer patient.

Apoptone is believed to be a disease-modification agent with a novel mechanism of action that distinguishes it from competitive drug candidates. Unlike presently approved and in-development therapies, Apoptone is believed to induce apoptosis by forcing catastrophic differentiation of tumor cells using the same signaling pathways these cell have hijacked for proliferation and survival. Thus, emergence of resistance mechanism may be less likely.

Triolex (HE3286)

Inflammatory Processes in Chronic Diseases

Another primary focus is on diseases that result from chronic inflammatory processes. Properly regulated, inflammation is a protective, life-saving response to invading pathogens. However, chronic and unproductive inflammation (also termed para-inflammation or sterile inflammation) can cause devastating tissue damage and loss of organ function. Chronic inflammation often arises from over-stimulation or dysregulation of the immune system, often resulting in the release of destructive products such as reactive oxygen species and proteolytic enzymes as well as additional pro-inflammatory mediators. The over-production of these dangerous biochemical products may be due to the presence of persistent low-grade infections that promote conditions in which the body s surveillance system is unable to differentiate between itself and invasion of foreign substances or to biochemical dysregulation. Chronic inflammation has been implicated in the pathogenesis of many diseases ranging from autoimmune conditions, such as arthritis and psoriasis, to infectious diseases, including human immunodeficiency virus (HIV), malaria and tuberculosis, lung inflammation conditions, such as asthma, chronic obstructive pulmonary disease and cystic fibrosis, macular degeneration, and neuroinflammatory conditions, such as Parkinson s and Alzheimer s disease, to metabolic diseases, including diabetes and cardiovascular diseases, as well as a number of different cancers.

Current Treatments for Chronic Inflammation

Some of the most widely used drugs for reducing inflammation belong to the corticosteroid class of compounds, which are also derived from the mammalian metabolome. Market research indicates that U.S. physicians issue tens of millions of new prescriptions for corticosteroids each year for a wide range of conditions. While these drugs are highly effective, chronic use leads to immune suppression, bone loss, tissue necrosis, and other serious side effects including mental depression.

Over the last decade, a number of new drugs have been introduced that are focused on inhibiting specific components of the pro-inflammatory cascade, including agents that bind and neutralize specific inflammatory cytokines, such as TNF-a and IL-1ß, as well as drugs that inhibit specific enzymes, such as COX-2, that produce pro-inflammatory mediators. While these drugs have demonstrated significant activities in a number of clinical trials involving chronic inflammatory diseases, such as arthritis, inflammatory bowel disease and psoriasis, most have also demonstrated safety limitations. Many cause dangerous immune suppression and other serious side effects that limit their utility. Most focus on a specific inflammatory mediator, which means they may not remain perpetually effective due to redundancies and compensatory effects in biological pathways. Our goal has been to develop compounds that mediate homeostasis to regain control of the inflammatory process and restore homeostasis.

Obesity, Chronic Inflammation, Insulin Resistance and Diabetes

Diabetes is a disease of aberrant insulin signaling that is comprised of a constellation of syndromes. Insulin is a hormone needed to regulate the transport of glucose from the blood into cells, where it can either be stored or converted to the energy needed to perform cellular processes. When insulin is insufficient or when insulin signaling functions improperly, the result is high blood glucose levels, which over time can lead to a host of severe medical conditions including nerve disease, blindness, limb amputation, heart attack, stroke and death. There are two forms of diabetes: type-1: a chronic condition in which little or no insulin is produced, and type-2 diabetes: a condition in which the body becomes resistant to the effects of insulin or the body produces some, but not enough, insulin to maintain a normal blood sugar level.

Epidemiological studies have clearly defined risk factors for the development or progression of type-2 diabetes, including genetics, and prenatal and postnatal environmental factors, including low birth weight, obesity, nutrient excess, inactivity, gestational diabetes, metabolic dysregulation with advancing age and obesity. Obesity in some individuals, through recently elucidated mechanisms, can lead to insulin resistance, hyper-glycemia, beta-cell dysfunction and ultimately overt diabetes. In turn, diabetes-related hyperglycemia and associated metabolic abnormalities can further alter signal transduction and gene-expression; thus contributing to a forward feeding cycle that results in disease progression.

The need for new classes of agents to treat type-2 diabetes is significant. There are over 25 million Americans with type-2 diabetes, 92 million in China and 220 million worldwide. Obese diabetes is a syndrome that is increasing rapidly as a result of advancing age and the rising incidence of obesity. Clinical data indicates only 36% of type-2 diabetics are currently able to maintain the American Diabetes Association maximum recommended HbA1c level of less than 7.0 % (a form of hemoglobin that is primarily used to identify the average plasma glucose concentration over a prolonged period of time). Large clinical studies have shown that failure to achieve these glucose targets, especially in obese patients, can progressively lead to severe health consequences including neuropathy, blindness, amputation, heart attack, stroke and death. Patients in large clinical trials consistently have a median BMI of 32 indicating that over half the population of type-2 diabetics is obese (BMI > 30).

Academic researchers have increasingly linked obesity-induced chronic inflammation with type-2 diabetes and elucidated its potential role in potentiating insulin resistance. In the setting of type-2 diabetes, evidence suggests that the pathology may arise through perturbations in NFkB signaling, particularly *via* the TLR4 and TNFa receptors. TLR4 is a receptor expressed on the surface of macrophages and other cells and is stimulated by dietary fatty acids as well as certain pathogens such as bacteria from the gut flora. Stimulation of the TLR4 receptor induces a cascade of pro-inflammatory signals including the production of TNFa. Elevated TNFa causes activation events that stimulate a complex network of signaling pathways culminating in the activation of NFkB and the expression of a number of genes under its control. Those gene products are involved in mediating inflammation and the cellular stress response. Persistent stimulation can lead to a chronic inflammatory state that produces the associated pathologies typifying the metabolic syndrome condition.

Current Treatments for Type-2 Diabetes

There are several pharmaceutical approaches to treating obese type-2 diabetes. Metformin is usually the first intervention prescribed by physicians when an individual is diagnosed with type-2 diabetes. Often metformin control begins to fail and frequently clinicians will combine additional drugs that assert different metabolic effects in order to control the disease. These include drugs designed to increase insulin production by the pancreas and reduce glucose production by the liver, and drugs, referred to as insulin sensitizers which are designed to increase the body s sensitivity to insulin and thereby improve glucose disposal from the bloodstream.

Triolex to Treat Chronic Inflammation in Type-2 Diabetes

Triolex is a next-generation compound that we are developing for the treatment of individuals diagnosed with certain chronic inflammatory processes.

In the setting of obese type-2 diabetes, evidence suggests that the mechanism of action for Triolex may be through regulation of the MAPK and NFkB pathways, particularly when these pathways are stimulated through the TLR4 and TNFa receptors. These pathways are a major component of the type-2 diabetes syndrome that is characterized by the presence of a chronic inflammatory state. Triolex is believed to be the first in a new class of insulin sensitizers to target obesity-mediated dysregulated metabolism through re-regulation of these pathways. Our scientists believe that re-regulation by Triolex of the MAPK and NFkB pathways regains control over genes whose products, which includes TNFa and IL-6, are involved in the inflammatory signaling pathway. These cytokines are also thought to be critically involved in the pathogenesis of other metabolic diseases, such as non-alcoholic steatohepatitis, cardiovascular disorders, neuroinflammatory disease, certain autoimmune diseases, such as ulcerative colitis and rheumatoid arthritis, and are also implicated in the pathogenesis of cancer all of which are, in general, diseases associated with advancing age.

Based on biochemical experiments, Triolex has been shown to act on the NFkB pathway in a manner that is independent of the PPARg pathway, which is targeted by other insulin sensitizers. Instead, the action of Triolex is associated with down-regulation of the pro-inflammatory JNK, IKK and p38 kinase pathways that cross-over into the NFkB pathway. Chronic activation of these kinase pathways leads to impairment of the insulin receptor substrate-1 protein (IRS-1) function, which is an important cellular mediator of insulin signaling and glucose transport.

A single-dose Phase I clinical trial conducted in healthy volunteers during 2007 demonstrates that Triolex is orally bioavailable in humans and provides significant drug concentrations in the blood at even the lowest dose tested. The findings also demonstrate that all doses of Triolex tested appear to be safe and well-tolerated with no reported drug-related serious adverse side effects.

A Phase I/II double-blind, placebo-controlled, multi-dose ranging clinical trial with Triolex in obese insulin-resistant subjects was initiated in 2007, and the safety, tolerance and pharmacokinetics of Triolex was evaluated when administered for 28 days. The potential for Triolex to decrease insulin resistance was also assessed. In addition, an open-label cohort of six patients with type-2 diabetes mellitus was studied.

Triolex was found to be safe and improved insulin sensitivity in insulin-resistant subjects. There was no trend in adverse events to differentiate between placebo- and treated-subjects, nor was there an increase in adverse events with dose escalation. Baseline and day 29 hyperinsulinemic-euglycemic clamp studies were performed on 36 subjects dosed twice daily. To test the hypothesis that Triolex would improve insulin sensitivity in insulin-resistant subjects, these subjects were stratified by the median baseline M value of 5 (a glucose disposal rate of 5 mg per minute per kg body weight): 21 subjects had M values < 5 (operationally defined as insulin-resistant); and 13 had M values of > 5 (operationally defined as insulin-sensitive). Pretreatment, insulin-resistant subjects had significantly higher fasting insulin levels, HOMA2 %B, HOMA2 IR values and LPS-stimulated PBMC MCP-1, TNFa and IL-6 protein levels, and trended for an increased IL-1ß protein level confirming greater insulin resistance, inflammatory responses and beta cell function than insulin-sensitive subjects.

After 29 days of treatment there were significant differences in changes from baseline for M values and C-reactive protein levels between Triolex-treated subjects compared to placebo-treated subjects To test the hypothesis that Triolex would benefit insulin-resistant but not insulin-sensitive subjects, the day 29 M-value changes from baseline were compared in the Triolex-treated subjects. In the insulin-resistant group, M values increased and in the insulin-sensitive group M values decreased. That difference was highly significant. When compared to placebo, insulin-resistant Triolex-treated subjects also showed significant improvement in M and trended for a decreased C-reactive protein level, whereas insulin-sensitive subjects did not show these changes. We concluded that Triolex was active in obese, insulin-resistant, pre-diabetic subjects but had no effect in insulin-sensitive, pre-diabetic subjects. That outcome is consistent with an insulin-sensitizing drug.

During 2008, a Phase IIa clinical trial was initiated with Triolex seeking early signs of activity in type-2 diabetes patients. The clinical trial proceeded in two stages. Stage 1 was a double-blinded, placebo controlled 12-week dosing trial that was exploratory in nature and enrolled 96 patients who were on a stable dose of metformin

with hemoglobin A1c (HbA1c) level in excess of 7.5 percent. The primary objectives of the study were to evaluate the change in HbA1c from baseline to week 12 and to evaluate the safety and tolerance of Triolex given 10 mg per day (5 mg BID) as compared to placebo. A final analysis for activity (HbA1c) in the clinical study of unaudited data was performed on all subjects that completed day 84 of the study (72 patients). There was no statistical difference between treatment and placebo for HbA1c in the overall patient population. However, a retrospective analysis of unaudited data was performed on the subpopulation of patients that represented the inflamed, obese, insulin-resistant, diabetic subgroup, in accordance with FDA guidance. That group is reflective of the impaired glucose tolerance subjects that responded to treatment in the company s Phase I pre-diabetes study. The analysis included patients divided into two strata with baseline values either less than or greater than (or equal to) the following criteria: BMI at least 27.3; fasting plasma insulin levels at least 3 µU/mL; and serum monocyte chemotactic protein-1 (MCP-1) levels at least 400 pg/mL. This phenotype represented 42% of all subjects (90 patients) with values for these parameters at baseline. Twenty-seven individuals in the high BMI strata completed 84 days of dosing. Those patients treated with Triolex (13) were showed improvements in their clinical parameters compared to the corresponding placebo patients (14). The improvements included significantly decreased HbA1c (-0.53 %, $p = 0.01$) values and fasting plasma glucose (-26.80 mg/dL, $p < 0.02$) levels, decreased body weight (-2.0 kg, $p = 0.0005$) and significantly increased anhydroglucitol (+0.7 µg/mL, $p = 0.03$) levels signifying decreased post-prandial glucose excursions**.** More Triolex subjects decreased weight (12/13 vs. 8/14, Fisher s Exact Test $p < 0.08$) and increased 1,5 anhydroglucitol levels (8/9 vs. 4/10, Fisher s Exact Test $p < 0.04$). The low BMI strata had a significant increase in HBA1c (+0.7%, $p < 0.005$) levels but with no detectable changes in any other parameter. There were significant differences between the high BMI and low BMI patients in their response to Triolex. The strata differed significantly in HbA1c (1.15 %, $p < 0.002$) and glucose (26.8 mg/dL, $p < 0.02$) levels, body weight (2.2 kg, $p < 0.0001$) and cholesterol (23.5 mg/dL, $p < 0.006$) levels. Significant trends were detected for differences in LDL cholesterol (14.8 mg/dL, $p = 0.08$), triglycerides (21.8 mg/dL, $p < 0.09$) levels and HOMA2 %B (25 %, $p < 0.06$) values. We conclude Triolex demonstrates signs of activity in chronically-inflamed, obese diabetes patients when this drug is taken in combination with metformin.

Stage 2 of the Phase IIa clinical trial was in treatment-naïve diabetic patients (no metformin) with inclusion criteria that restricted the lower limit of BMI to 28, insulin ³ 4 µU/mL, C-peptide ³ 2 ng/mL and MCP-1 ³ 400 pg/mL. There was no significant overall treatment effect on day 84 HbA1c in Cohort 2 treatment-naïve subjects, despite restrictive inclusion criteria. Subjects were again stratified by BMI. Higher BMI subjects were defined as BMI ³ 31.3 kg/m^2. At baseline, higher BMI subjects (32 of 69 subjects, 46%) had significantly higher resistin levels and statistical trends for higher CRP, C-peptide, HOMA2 %B, leptin and lower fructosamine levels when compared to the low BMI subjects. High BMI Triolex-treated subjects showed a statistically significant percent decrease in HbA1c levels at day 112 when compared to the corresponding placebo group (-1.1 %, $p < 0.05$). In the placebo group there was a higher proportion of subjects with decreased HbA1c levels (8/9 vs. 6/13, Fisher s Exact Test $p < 0.08$), and a higher portion with > 1% decrease (5/9 vs. 2/13, Fisher s Exact Test $p < 0.08$). The Triolex-treated subjects had a significantly greater frequency in decreased CRP levels (8/9 vs. 5/14, Fisher s Exact Test $p < 0.03$), and a statistical trend for a day 84 decrease (-1.1 mg/L, Fisher s Exact Test $p = 0.08$). Post prandial glucose excursions were decreased as evidenced by the greater frequency of subjects with increased day 84 1,5-anhydroglucitol (median, + 1.4 µg/mL; 7/8 vs. 6/14 placebos, Fisher s Exact Test $p < 0.03$). In contrast, low BMI patients (28 kg/m^2 31.3 kg/m^2) treated with Triolex had a significantly higher day 84 HbA1c percent change ($p < 0.005$) from baseline (+0.18 %) when compared to the placebo group (-0.93 %) due to a large placebo effect in these patients. There was also a statistical trend for decreased day 84 fructosamine levels (-11.75 µmol, $p < 0.08$) in the placebo but with an absence of a significant difference in day 84 fasting plasma glucose levels. We conclude Triolex demonstrates signs of activity in treatment naïve, chronically inflamed obese diabetes patients.

Triolex shows no consistent pattern of adverse events associated with its use. Actos and Avandia are two widely prescribed insulin sensitizers used in combination with metformin. Recently, Avandia was withdrawn after concerns were raised about an increased risk of cardiac events. The Actos cardiovascular safety profile compares favorably with Avandia and remains on the market with a black-box warning. Subsequently, it was found to be associated with bladder tumors and in some countries has also been withdrawn from the market. The side effects associated with the use of currently approved insulin sensitizers have not been observed with Triolex.

In summary, the Triolex studies have demonstrated a good safety profile for the drug with no consistent pattern of adverse events associated with its use. It is active in obese insulin-resistant, pre-diabetic subjects but has not demonstrated an effect in insulin-sensitive, pre-diabetic subjects, which is an outcome consistent with an insulin-sensitizing drug. We conclude Triolex demonstrates signs of activity in chronically inflamed obese diabetic patients as a single agent and in combination with metformin. The mechanism of action has been partially elucidated with biochemical molecular points of interaction identified.

Competition in Diabetes

Given the large market opportunities for products that treat the indications for which we are currently developing our compounds, most major pharmaceutical companies and many biotechnology companies have programs directed toward finding drugs to treat the indications that we are exploring. In metabolism and type-2 diabetes, there are a number of drugs, such as Actos from Takeda Pharmaceuticals (already approved for improving insulin sensitivity), glucagon-like peptide-1 (such as Victoza® by Novo Nordisk), dipeptidyl peptidase-4 inhibitors (such as Januvia® by Merck & Cp., Inc. and Onglyza® by Bristol Myers Squibb) and numerous other drugs in various stages of development. While Actos currently accounts for a significant share of the market for insulin sensitizers to treat type-2 diabetes, it is known to cause the unwanted side effects of weight gain and edema and was recently either removed from the market, as was the case for Avandia, or given a black-box warning (Actos) by the FDA because of increased treatment-related heart failure and bladder cancer risk associated with the use of the medication.

Autoimmune Disease and Chronic Inflammation

Current Treatments for Autoimmune Diseases

Immune modulators that correct immune dysregulation and chronic inflammatory conditions by inhibition or enhancement of single cytokine targets such as TNFa and IL-1ß or their receptors have been developed by a number of companies. For example, Amgen s Enbrel® targets TNFa as does Johnson & Johnson s Remicade®. Other immune-modulating drugs such as Celebrex® from Pfizer target COX-2. While these targeted approaches have shown clinical benefit and have generated significant sales volumes, redundancy in the immune system can limit their effectiveness. In addition, side effects, health care costs and reimbursement issues are limiting their long-term global utility. We have shown our compounds affect cytokine cascades through direct interactions in the endocrine system. That may potentially make them more attractive drug candidates than those currently available as they directly interact through the endocrine system. Triolex may represent the first in a new class of agents to treat those diseases, assuming it is successfully developed and commercialized.

Rheumatoid Arthritis

Rheumatoid arthritis is a type of chronic arthritis that occurs in joints on the extremities of the body (such as hands, wrists or knees). In rheumatoid arthritis, the immune system attacks the joints and sometimes other organs. According to the Centers for Disease Control and Prevention, or (CDCP), an estimated 50 million people were treated for some form of arthritis and other rheumatic conditions in 2009, 22% of the US adult population.

Based upon positive results with Triolex in published rodent models of collagen-induced and collagen antibody-induced arthritis, a Phase I clinical trial was initiated in rheumatoid arthritis patients in 2008. A 28-day oral dose-ranging study assessed the safety, pharmacokinetics and potential for drug-drug interactions in stable rheumatoid arthritis patients also receiving methotrexate. Triolex was found to be safe and well-tolerated. No drug-drug interaction with methotrexate was found. Triolex is now positioned to enter clinical studies in patients with active rheumatoid arthritis.

Ulcerative Colitis and Other Autoimmune Diseases

Inflammatory bowel disease is comprised of ulcerative colitis, a chronic inflammation of the large intestine, or colon, and Crohn s disease, a condition of inflammation of the small intestines. Ulcerative colitis and Crohn s disease together affect approximately 500,000 to 2 million people in the United States.

Based upon published observations with Triolex in preclinical models widely used by both the pharmaceutical industry and academia to test agents as potential treatments for ulcerative colitis, we commenced a Phase I/II clinical trial in ulcerative colitis patients in 2008. This Phase I/II dose ranging study evaluated the safety, tolerance, pharmacokinetics and activity of Triolex when administered orally for 28 days to patients with active, mild-to-moderate ulcerative colitis. Triolex at the doses studied was found to be safe and well-tolerated but interpretation of the results was confounded by the high frequency of spontaneous colitis flare resolution. There was no indication of a treatment advantage in this acute inflammatory setting when compared to placebo. Triolex is staged for long-term clinical trials directed towards the control of the chronic inflammatory processes associated with this disease, a clinical setting believed to be consistent with the pharmacological properties of the compound.

Triolex has also shown activity in pre-clinical models of multiple sclerosis and lupus erythematosus, which represent additional candidate indications for clinical trials.

Neuroinflammation

Neuroinflammation plays a major role in the pathophysiology of many of the most socially and economically significant diseases of first world nations. These diseases include Alzheimer s disease, Parkinson s disease, epilepsy, amyotrophic lateral sclerosis (ALS), autism, and multiple sclerosis. Recent scientific publications suggest these diseases share a common inflammatory mediator, the ubiquitous intranuclear protein, high mobility group box 1 protein (HMGB1), which is released to the extracellular environment from necrotic cells. HMGB1 acts through at least one pathway known to be regulated by Triolex. Because Triolex readily penetrates the blood-brain-barrier, it has the potential to treat a broad spectrum of diseases with a neuroinflammatory etiology. The Company is investigating the use of Triolex as a treatment for Parkinson s disease with funding from The Michael J. Fox Foundation. The terms of the collaboration call for MJFF to fund up to approximately $150,000 toward pre-clinical development of Triolex in rodents. Our work with MJFF has shown that in a rodent model of MPTP induced neuronal excitotoxicity (a model that closely mimics Parkinson s disease in humans), Triolex significantly reduced motor impairment and production of inflammatory cytokines, which was associated with significantly greater numbers of surviving neurons in the brains of Triolex treated animals. Further funding of preclincal and clinical development is pending a decision by The MJFF. Similarities between the molecular pathophysiology of Parkinson s disease and epilepsy suggest that Triolex may also be active against epilepsy, which affects approximately 5 million people in the U.S. and Europe and 10 million in China, one-third of which are refractory to currently approved anti-seizure drugs. The company intends to initiate Triolex product development activities for neuroinflammation through corporate partners or investment financing.

Ophthalmology

Diseases of the eye are common and frequently have an inflammatory etiology. The associated temporary or permanent loss of vision is socially and economically important. The most common treatments for ocular inflammation are glucocorticoids and cyclosporins, which are associated with serious side effects that include glaucoma, cataract, and increased susceptibility to infection. The development of ophthalmic pharmaceuticals is increasingly popular because cost of clinical trials and product approval for certain ocular conditions can be substantially less than most systemic indications because of the acute nature of many ocular diseases, and the limited systemic drug exposure from topical administration. Inflammatory ocular diseases should be amenable to Triolex treatment based on Triolex s mechanism of action and disease molecular pathophysiology. Importantly, Triolex s mechanism of action cannot cause any of the deleterious side effects of currently approved medications. The results of recent preclinical studies of Triolex in rodent models of uveitis indicate potent anti-inflammatory activity. The Company intends to continue investigating the potential breadth of this opportunity, and to initiate ophthalmic product development through corporate partners or investment financing. Ocular conditions of interest include anterior and posterior uveitis, idiopathic dry eye, Sjogren s dry eye disease (an autoimmune condition related SLE), conjunctivitis, blepharitis, and post surgical inflammation.

Pulmonary Diseases and other Autoimmune Diseases

Triolex has shown signs of activity in pre-clinical models of lung inflammation. Accordingly, the Company is exploring the potential for Triolex in a variety of pulmonary diseases with academic collaborators. These conditions include cystic fibrosis, chronic pulmonary disease and asthma.

Triolex Development Status

Triolex is manufactured economically using a multi-step organic synthesis from the widely abundant and inexpensive starting material, DHEA. It is formulated for oral administration with excipients approved for oral dosage products. Diseases associated with chronic inflammation are thought to require drug exposures of extended duration to observe definitive treatment effects. Long term toxicology studies have been completed that qualify Triolex for use in clinical studies of 6 month s duration or longer. There were no untoward side effects detected. Allowed and issued patents claim the compound itself, pharmaceutical formulations and methods of use to treat a variety of inflammatory diseases including type-2 diabetes and autoimmune conditions such as rheumatoid arthritis and ulcerative colitis in the United States, Europe and elsewhere. Applications with pending claims filed to extend patent coverage in additional regions of economic interest including China, Japan and Korea.

NEUMUNE (HE2100)

Neumune as treatment for acute radiation exposure; an ERß selective agonist.

In December 2010, The Company reported on the safety, tolerability and signs of hematologic activity in four double-blinded, randomized, placebo-controlled studies of NEUMUNE in healthy human subjects, published in *The Journal of Radiological Protection.* Those studies demonstrated that Neumune has the potential to directly enhance innate immunity in humans and defined the compound as a highly selective ERß ligand. ERß ligand treatment has been suggested as a potentially safe anti-inflammatory and neuroprotective strategy in multiple sclerosis and other neurodegenerative diseases. In May of 2010, the Company reported that Neumune could ameliorate neuroinflammation in mice and has the potential to limit relapses in patients with multiple sclerosis. The Company is actively soliciting partnerships to develop an orally bioavailable, metabolically stable second generation derivative.

HE2000

HE2000 in Infectious Disease

The Company conducted clinical trials in HIV, AIDS and malaria from the late 1990 s until early 2002. While the primary market opportunities for pharmaceuticals have traditionally been in the U.S., Europe and Japan, our adrenal hormones have a number of attributes that make them potentially globally useful. Included are the potential broad-spectrum activity in multiple infectious diseases, an attractive safety profile to date, a low likelihood of resistance and the relative ease of manufacture. Increasing focus on the infectious disease crises around the world such as those represented by HIV, malaria and tuberculosis has led to a number of recent third party initiatives designed to provide funding for effective approaches to these diseases.

HE2000 has been tested in a series of Phase I/II and Phase II clinical trials in HIV/AIDS patients in the U.S. and South Africa. In all of these studies, HE2000 treatment appeared to be generally well-tolerated with mild to moderate pain at the injection site as the most common adverse event. In addition to assessing the safety profile of HE2000 in clinical trials, we have also assessed the effect of HE2000 on a wide variety of immune and inflammatory markers that are associated with HIV disease progression.

In a South Africa study, HE2000 treatment of HIV patients that received no other therapy resulted in long-lasting, statistically significant declines in a number of key inflammatory mediators including TNFa, IL-1ß and IL-6. In this placebo-controlled study, we also observed significant durable increases in a wide variety of immune cell subsets associated with innate and cell-mediated immunity following treatment with HE2000. In addition, patients that received HE2000 in this trial experienced a significant decline in blood virus levels over the course of the study, which correlated with an increase in HIV specific T-cell mediated immunity. HE2000 was then tested as a monotherapy in late-stage AIDS patients. During this study, patients experienced a statistically significant reduction in the number of opportunistic infections compared to those treated with placebo and the life-threatening tuberculosis infections were completely quelled after 4-months of treatment.

The ability of HE2000 to reduce pro-inflammatory mediators while stimulating innate and cell-mediated immunity has potential implications for the treatment of a number of other infectious diseases, including parasitic infections such as malaria. Based on multiple pre-clinical studies performed by collaborators at the Walter Reid Naval Hospital and the University of Vermont, we performed two Phase II clinical studies in malaria patients at Mahidol University in Bangkok, Thailand. Results indicated that HE2000 was effective in reducing malarial parasite count and cleared blood-borne malarial parasites in most patients within seven days.

A series of tuberculosis animal model studies have also shown that HE2000 is effective when given as a monotherapy in either the acute or chronic phase of this bacterial infection and it appears to have a synergistic effect when combined with the current three-drug regimen considered the standard of care for antibiotic treatment of tuberculosis.

Government Regulation

General

The manufacturing and marketing of our proposed drug candidates and our research and development activities are, and will continue to be, subject to regulation by federal, state and local governmental authorities in the U.S. and other countries. In the U.S., pharmaceuticals are subject to rigorous regulation by the Food and Drug Administration, (FDA), which reviews and approves the marketing of drugs. The Federal Food, Drug and Cosmetic Act, the regulations promulgated thereunder, and other federal and state statutes and regulations govern, among other things, the testing, manufacturing, labeling, storage, record keeping, advertising and promotion of our potential products.

Approval Process

The International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH). Since its inception in 1990, ICH has evolved, through its ICH Global Cooperation Group, to respond to the increasingly global face of drug development, so that the benefits of international harmonization for better global health can be realized. Harmonization ensures that safe, effective, and high quality medicines are developed and registered in the most resource-efficient manner worldwide. Specifically, China has been steadily improving its regulatory regime governing food and pharmaceutical industries in recent years, aligning the country with international standards of practice. Regional Harmonization Initiatives (RHI) include Asia-Pacific Economic Cooperation (APEC), Association of Southeast Asian Nations (ASEAN), Gulf Cooperation Council (GCC), Pan American Network on Drug Regulatory Harmonization (PANDRH) and the Southern African Development Community (SADC). Development in each of these regions may or may not require bridge studies, depending on the genetic diversity within each population, but in any event, costs for such studies would be borne by each regional partner.

The process of obtaining FDA approval for a new drug may take many years and generally involves the expenditure of substantial resources. The steps required before a new drug can be produced and marketed for human use include clinical trials and the approval of a New Drug Application.

Preclinical Testing: In the preclinical phase of development, the promising compound is subjected to extensive laboratory and animal testing to determine if the compound is biologically active and safe.

Investigational New Drug or IND: Before human tests can start, the drug sponsor must file an IND application with the FDA, showing how the drug is made and the results of animal testing. An IND becomes effective 30 days following receipt by the FDA.

Human Clinical Testing: The human clinical testing program usually involves three phases which generally are conducted sequentially, but which, particularly in the case of anti-cancer and other life-saving drugs, may overlap or be combined. Clinical trials are conducted in accordance with protocols that detail the objectives of the study, the parameters to be used to monitor safety and the efficacy criteria to be evaluated. Each protocol is submitted to the FDA as part of the IND filing. Each clinical study is conducted under the auspices of an independent Institutional Review Board, or IRB, for each institution at which the study will be conducted. The IRB will consider, among other things, the design of the clinical trial, ethical factors, the risk to human subjects and the potential benefits of therapy relative to the risk.

Phase I clinical trials: Studies usually are conducted on healthy volunteers or, in the case of certain terminal illnesses such as AIDS or cancer patients with disease that have failed to respond to other treatment, to determine the maximum tolerated dose, side effects and pharmacokinetics of a product.

Phase II clinical studies: These are conducted on a small number of patients having a specific disease to determine initial efficacy in humans for that specific disease, the most effective doses and schedules of administration, and possible adverse effects and safety risks. Phase II/III differs from Phase II in that the trials involved may include more patients and, at the sole discretion of the FDA, be considered the pivotal trials, or trials that will form the basis for FDA approval.

Phase III clinical studies: These are normally the pivotal drug trials consisting of broad scope of studies on diseased patients, in order to evaluate the overall benefits and risks of the drug for the treated disease compared with other available therapies. The FDA continually reviews the clinical trial plans and results and may suggest design changes or may discontinue the trials at any time if significant safety or other issues arise.

New Drug Application or NDA: Upon successful completion of Phase III clinical trials, the drug sponsor files an NDA with the FDA for approval containing details of the chemistry, manufacture and quality control information that has been developed, nonclinical data, results of human tests, and proposed labeling.

Post Approval. If the FDA approves an NDA, the drug sponsor is required to submit reports periodically to the FDA containing adverse reactions, production, and quality control and distribution records. The FDA may also require post-marketing testing to support the conclusion of efficacy and safety of the product, which can involve significant expense. After FDA approval is obtained for initial indications, further clinical trials may be necessary to gain approval for the use of the product for additional indications.

The testing and approval process is likely to require substantial time, from several months to years, and there can be no assurance that any FDA approval will be granted on a timely basis, if at all. The approval process is affected by a number of factors, primarily the side effects of the drug (safety) and its therapeutic benefits (efficacy). Additional preclinical or clinical trials may be required during the FDA review period and may delay marketing approval. The FDA may also deny an NDA if applicable regulatory criteria are not met.

Outside the U.S., we will be subject to foreign regulatory requirements governing human clinical trials and marketing approval for our products. The requirements governing human clinical trials ex-US usually follow International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH) Good Clinical Practices (GCP) or country-specific GCPs which are based on the ICH GCPs. Regulatory approval outside the U.S. typically includes the risks and costs associated with obtaining FDA approval but may also include additional risks and costs.

Manufacturing

We do not have plans to establish manufacturing facilities to produce our drug candidates or any future products. We plan to control our capital expenditures by using contract manufacturers to make our products. We believe that there are a sufficient number of high-quality FDA-approved contract manufacturers available, and we have had discussions, and in some cases established relationships, to fulfill our near-term production needs for both clinical and commercial applications.

The manufacture of our drug candidates or any future products will be subject to rigorous regulations, including the need to comply with the FDA s current Good Manufacturing Practice regulations. As part of obtaining FDA approval for each product, each of the manufacturing facilities must be inspected, approved by and registered with the FDA. In addition to obtaining FDA approval of the prospective manufacturer s manufacturing and quality control procedures, domestic and foreign manufacturing facilities are subject to periodic inspection by the FDA and/or foreign regulatory authorities.

Patents

We currently own or have obtained licenses to a large estate of U.S. and foreign patents and patent applications. We consider the protection of our technology, whether owned or licensed, to the exclusion of use by others, to be vital to our business. While we intend to focus primarily on patented or patentable technology, we may also rely on trade secrets, unpatented property, know-how, regulatory exclusivity, patent extensions and continuing technological innovation to develop our competitive position. In the U.S. and certain foreign countries, the exclusivity period provided by patents covering pharmaceutical products may be extended by a portion of the time required to obtain regulatory approval for a product.

In certain countries, some pharmaceutical-related technology such as disease treatment methods are not patentable or only recently have become patentable and enforcement of intellectual property rights in many countries has been limited or non-existent. Future enforcement of patents and proprietary rights in many countries can be expected to be problematic or unpredictable. We cannot guarantee that any patents issued or licensed to us will provide us with competitive advantages or will not be challenged by others. Furthermore, we cannot be certain that others will not independently develop similar products or will not design around patents issued or licensed to us.

In most cases, patent applications in the U.S. are maintained in secrecy until 18 months after the earliest filing or priority date. Publication of discoveries in the scientific or patent literature, if made, tends to lag behind actual discoveries by at least several months. U.S. patent applicants can elect to prevent publication until the application issues by agreeing not to file the application outside the U.S. This option is rarely used for pharmaceutical patent applications. Consequently, we cannot be certain that a patent owner or licensor of its intellectual property was the first to invent the technology or compounds covered by pending patent applications or issued patents or that it was the first to file patent applications for such inventions. In addition, the patent positions of biotechnology and pharmaceutical companies, including our own, are generally uncertain, partly because they involve complex legal and factual questions.

In addition to the considerations discussed above, companies that obtain patents claiming products, uses or processes that are necessary for, or useful to, the development of our drug candidates or future products could bring legal actions against us claiming infringement. Patent litigation is typically costly and time consuming, and if such an action were brought against us, it could result in significant cost and diversion of our attention. We may be required to obtain licenses to other patents or proprietary rights, and we cannot guarantee that licenses would be made available on terms acceptable to us. If we do not obtain such licenses, we could encounter delays in product market introductions while we attempt to develop or license technology designed around such patents, or we could find that the development, manufacture or sale of products requiring such licenses is foreclosed.

Further, we cannot guarantee that patents that are issued will not be challenged, invalidated or infringed upon or designed around by others, or that the claims contained in such patents will not interfere with the patent claims of others, or provide us with significant protection against competitive products, or otherwise be commercially valuable. To the extent that we are unable to obtain patent protection for our products or technology, our business may be materially adversely affected by competitors who develop substantially equivalent technology.

For a discussion of the risks associated with patent and proprietary rights, see below under Item 1A Risk Factors .

Technology Agreements

Pharmadigm

In August 2002, we entered into a Sublicense Agreement with Pharmadigm, Inc. (currently known as Inflabloc Pharmaceuticals, Inc.). Under the agreement, we obtained exclusive worldwide rights to certain intellectual property of Pharmadigm and the University of Utah and we agreed to make aggregate payments of $0.9 million in cash or in shares of our common stock, at our option, over the following year. We elected to make such payments in equity and have issued a total of 168,921 shares of our common stock in complete satisfaction of this requirement. We will also make additional milestone and royalty payments to Pharmadigm if we meet specified development and commercialization milestones for products covered by its patents. No such milestones have been met to date. The principal patents licensed under the agreement, originally licensed to Pharmadigm from the University of Utah, relate to inventions by Dr. Raymond Daynes and Dr. Barbara A. Areneo. Dr. Daynes served as a scientific consultant for the Company from 1999 to mid-2003.

Aeson Therapeutics

In October 2000, we acquired a 21% equity stake in Aeson Therapeutics Inc. (Aeson) and an exclusive worldwide sublicense to three issued patents in the area of adrenal sterols in exchange for $2.0 million in cash and 208,672 shares of our common stock valued at $2 million. As part of the transaction, Aeson and its stockholders granted us an exclusive option to acquire the remainder of Aeson at a predetermined price. In March 2002, we amended certain aspects of our agreements with Aeson. Under the amendments, we paid Aeson $1.2 million, which extended the initial date by which we could exercise our option to acquire the remainder of Aeson to September 30, 2002. We also received additional equity securities of Aeson as a result of this payment. We elected not to exercise the option to acquire the remainder of Aeson by September 30, 2002. On June 7, 2006, we acquired substantially all of the assets of Aeson. As consideration for Aeson s assets, we agreed (i) to issue a total of 35,000 shares of our common stock to Aeson at the closing of the acquisition and (ii) to issue to Aeson s stockholders up to a total of 165,000 additional shares of our common stock if certain development milestones are achieved. We have not achieved any of the development milestones to date.

China State Institute of Pharmaceutical Industry

In January 2011, the Company announced that it had licensed the research and development and commercialization rights for three of its products, exclusively in the People s Republic of China and Hong Kong, to the China State Institute of Pharmaceutical Industry (CIPI). Harbor BioSciences retains the rights to these products in the U.S. and the rest of the world, and CIPI will make available to the company all pre-clinical and clinical data it generates.

CIPI was recently formed by a merger of the Shanghai Institute of Pharmaceutical Industry and other institutes and companies. CIPI s research and development (R&D) focus has been in the areas of cancer, infectious diseases, cardiovascular, autoimmune disorders, endocrinology and central nervous system (CNS). CIPI is a subsidiary of the China National Pharmaceutical Group Corporation (Sinopharm Group), China s largest pharmaceutical and health industrial group under the state-owned Assets Supervision and Administration Commission of the State Council. Sinopharm Group s core businesses include R&D, manufacturing, distribution and retail sales. Its products are manufactured in more than 10 pharmaceutical and biological production facilities. Sinopharm Group has more than 20 joint ventures with global pharmaceutical companies and through trade and cooperative relations, has a presence in more than 100 countries and regions. Sinopharm Group reported 2010 revenues of approximately ¥80 billion ($12 billion US).

CIPI is a major supplier of both generic drugs and traditional Chinese medicines in China and Hong Kong. The three clinical drug development candidate license agreements cover Harbor BioSciences compounds HE2000, Apoptone and Triolex for any clinical use in the People s Republic of China and Hong Kong. Triolex, has completed Phase IIa clinical trials in patients with Type-2 diabetes and is in early stage development for ulcerative colitis and rheumatoid arthritis; Apoptone, which demonstrated activity in Phase I/IIa trials of prostate cancer; and HE2000, has been shown to limit opportunistic infections, including tuberculosis, in humans infected with the HIV-1 virus, to reduce parasite levels in patients with uncomplicated malaria. CIPI plans to develop the Harbor BioSciences compounds for major indications including diabetes, cancer, chronic inflammatory conditions and infectious diseases.

The Company believes these are the first drug development agreements between a western pioneer drug company and a government-owned Chinese drug developer for pharmaceutical development to be conducted in the People s Republic of China. CIPI, a low cost drug manufacturer, has agreed to supply the licensed products to Harbor BioSciences for use in clinical studies and sales outside of China and Hong Kong. The Company can also elect to distribute these compounds in countries that accept the State Food and Drug Administration s (SFDA) drug approval process.

The Company will receive milestone payments for Triolex, Apoptone and HE2000, excluding infectious diseases, at the completion of Phase II and III clinical studies and upon approval by the SFDA. The Company will also receive royalties based on net profits for the life of each agreement. No milestones have been met to date. The term of each agreement runs until the latter of (1) the expiration of the last licensed patent or any Company, CIPI or joint improvement patent and (2) the first documented third party sale of a competing generic product in the licensed territory. In addition, the Company is CIPI s sole agent with commercial development and sales rights to all of CIPI s improvements that are sold outside the licensed territory. Sales of licensed drugs that are covered by CIPI s improvements outside the territory bear a royalty to Harbor BioSciences.

We have delivered to CIPI in excess of 20,000 pages of pre-clinical, non-clinical and chemistry, manufacture and control data for all three projects. These pages have been translated and evaluated by the dedicated project teams, one for each compound. Our historical data has been evaluated against the People s Republic of China s State Food and Drug Administration criteria with their expert advisors.

Late in the second quarter, we met with CIPI in China to review CIPI s project plans for each compound. We have been informed that CIPI intends to commence Phase I clinical trials for Apoptone during the first half of 2012 and for Triolex and HE2000 during the second half of 2012. According to CIPI, they have developed a small-scale synthesis method for each compound and intend to scale-up each synthesis into a larger batch processes that will be used for clinical trials materials. We were told by CIPI that the process will then be scaled-up to

manufacture the final active pharmaceutical ingredients in quantities to meet market demands. CIPI also informed us that they are evaluating multiple formulations to identify and use an optimized formulation intended to be the final finished product to initiate their clinical trials. Our agreements with CIPI provide that these products will be made available to us for clinical trials outside of the licensed territory if these activities are successfully completed by CIPI. CIPI further told us that reproductive toxicology and certain other tox studies are planned for the first half of 2012.

During the fourth quarter of 2011 and the first quarter of 2012, we intend to deliver the detailed clinical data from its clinical studies for the licensed compounds for translation and evaluation by CIPI s clinical experts. In cooperation with CIPI s advisors and the SFDA, a detailed clinical development strategy for each project with estimated timelines is expected during the first half of 2012.

The Chinese clinical trial strategy differs from the western world. In the western world, the clinical timelines and scope of the trials become longer and more involved as a compound progresses through the clinical development process. In China, the SFDA has a different clinical trial design than generally practiced in the west. Chinese Phase I safety studies typically are much larger in scope and longer in duration to insure the development program does not become halted for safety concerns during the more expensive downstream stages of a program. For similar reasons, a Phase I study does not commence until all of the safety and toxicology studies required by the SFDA have been completed and evaluated. Consequently, Phase II and Phase III studies are typically smaller in scope and faster than the western world.

Pursuant to our agreements with CIPI, as a matter of course, we are to receive data periodically from CIPI s development efforts to supplement the existing data for each project. We intend to use this data for further partnering discussions in territories outside of China and Hong Kong.

The Company announced in June 2011, the signing of an umbrella generic drugs distribution agreement with CIPI. This new agreement provides that Harbor BioSciences and CIPI will select one or more of CIPI s drugs or other products for distribution outside China under separately negotiated sub agreements. This agreement provides that the Company is to receive commercial development, sales and sublicense rights for products under executed sub agreements. Sales of CIPI s products will bear a royalty to Harbor BioSciences. The company has been in discussions with potential partners to market, distribute and to work with Harbor BioSciences to monetize this agreement. We intend to continue with these activities during 2012.

China Business Strategy

We believe that our agreement with CIPI places Harbor Biosciences on the forefront of a rapidly evolving global pharmaceutical industry. China offers relatively rapid product development, especially in the context of Phase II and III studies, diluted risk (defrayed costs), low cost supply and validation of technology, as well as huge, expanding and increasingly affluent markets for our specific indications. As the R&D arm of the Sinopharm Group, CIPI is uniquely qualified and positioned to develop our small molecules. By the terms of our agreement, CIPI will bear all development costs for each candidate compound and Harbor BioSciences will retain the rights to all its products in the U.S. and the rest of the world. This will afford us several exciting opportunities to capitalize on regional licensing and partnership agreements as well as agreements with Health Ministries of individual governments, including socialized medicine countries. Our partnership with CIPI includes a low cost supply agreement by which China will supply API or finished products into the rest of the world, in accordance with specific agreements and regulatory requirements. Such agreements may include payments of up front fees to Harbor Biosciences with additional payments for milestones achieved in China. Concurrently, Harbor Biosciences is actively attempting to develop partnerships and/or licensing agreements in order to monetize its other assets, including Neumune and several novel, naturally occurring sterol hormones that may be suitable for either pharmaceutical or nutraceutical development. All together, Harbor Biosciences seeks to capitalize on at least six compounds in various stages of development, including Apoptone, Triolex, HE2000, Neumune, HE3413 and HE3177. In addition, our pipeline contains potential pre-clinical candidates with improved pharmaceutical properties aimed at pharmaceutically accepted targets including ERß. It is the Company s intention to reduce its burn rate to match revenue recognition from milestones achieved in China and the execution of potential licenses and partnerships with the rest of the world.

Employees

As of January 15, 2012, we had 8.5 full-time equivalent, non-union employees. We believe that our relations with our employees are good.

Executive Officers and Senior Management

Our executive officers and senior management and their ages as of January 15, 2012 are as follows:

Name	Age	Position
James M. Frincke, Ph.D.	61	Chief Executive Officer
Christopher L. Reading, Ph.D.	64	Chief Scientific Officer
Robert W. Weber	61	Chief Financial Officer and Secretary
Former Officer		
Dwight R. Stickney, M.D.	68	Chief Medical Officer

James M. Frincke, Ph.D. joined Harbor BioSciences as Vice President, Research and Development in November 1997, was promoted to Executive Vice President in March 1999, to Chief Scientific Officer in December 2001, to Chief Operating Officer in February 2008 and to Chief Executive Officer in 2009. Dr. Frincke joined Harbor BioSciences, Inc. from Prolinx, Inc., where he served as Vice President, Therapeutics Research and Development from 1995 to 1997. During his 30 years in the biotechnology industry, Dr. Frincke has managed major development programs including drugs, biologicals, and cellular and gene therapy products aimed at the treatment of cancer, infectious diseases, organ transplantation, autoimmune disease and type 2 diabetes. Since joining the biotechnology industry, Dr. Frincke has held vice president, research and development positions in top tier biotechnology companies including Hybritech/Eli Lilly and SyStemix Inc. (acquired by Novartis). In various capacities, he has been responsible for all aspects of pharmaceutical development including early stage research programs, product evaluation, pharmacology, manufacturing, and the management of regulatory and clinical matters for lead product opportunities. Dr. Frincke has authored or co-authored more than 100 scientific articles, abstracts and regulatory filings. Dr. Frincke received his B.S. in Chemistry and his Ph.D. in Chemistry from the University of California, Davis. Dr. Frincke performed his postdoctoral work at the University of California, San Diego.

Christopher L. Reading, Ph.D. joined Harbor BioSciences as Vice President of Scientific Development in January 1999, was promoted to Executive Vice President, Scientific Development in March 2002 and to Chief Scientific Officer in February 2008. Before Harbor BioSciences, Inc., Dr. Reading was Vice President of Product and Process Development at Novartis Inc.-owned SyStemix Inc. During this time, he successfully filed three investigational new drug applications (INDs) in the areas of stem cell therapy technology and stem cell gene therapy for HIV/AIDS. Prior to joining SyStemix, Dr. Reading served on the faculty of the M.D. Anderson Cancer Center in Houston for nearly 13 years. His positions there included Associate and Assistant Professor of Medicine in the Departments of Hematology and Tumor Biology. During his career, Dr. Reading has given more than 25 national and international scientific presentations, published more than 100 peer-reviewed journal articles and 15 invited journal articles as well as written nearly 20 book chapters, and received numerous grants and contracts which supported his research activities. Dr. Reading has served on the National Science Foundation Advisory Committee for Small Business Innovative Research Grants (SBIR) as well as on the editorial boards of *Journal of Biological Response Modifiers* and *Molecular Biotherapy*. He holds a number of patents for his work with monoclonal antibodies and devices. Dr. Reading received his Ph.D. in Biochemistry at the University of California at Berkeley and completed postdoctoral study in tumor biology at The University of California at Irvine. He earned his B.A. in cell biology at the University of California at San Diego.

Dwight R. Stickney, M.D. joined Harbor BioSciences as Medical Director, Oncology in May 2000, was appointed Vice President, Medical Affairs in March 2003 and was promoted to Chief Medical Officer in February 2008. Dr. Stickney joined Harbor BioSciences, Inc. from the Radiation Oncology Division of Radiological Associates of Sacramento Medical Group, Inc., in Sacramento, California, where he served as a Radiation

Oncologist since 1993. While at Radiological Associates, he served as Chairman of the Radiation Oncology Division from 1997 to 1999 and was a member of the Radiation Study section of the National Institute of Health s Division of Research Grants from 1993 to 1997. He also served as the Director of Radiation Research for Scripps Clinic and Research Foundation in La Jolla, California. Dr. Stickney has taught in medical academia as Associate Professor of Radiation Medicine at Loma Linda University School of Medicine and has served as Director of the International Order of Forresters Cancer Research Laboratory and on the Board of Directors of the California Division of the American Cancer Society. Earlier in his career, Dr. Stickney held positions with Burroughs Wellcome and the Centers for Disease Control, and academic teaching appointments at The University of California at Los Angeles and The University of California at Riverside. He has also served as a consultant for a number of biotechnology companies on the design and conduct of clinical trials. Dr. Stickney has authored or co-authored over 80 scientific articles, abstracts and book chapters. He is named inventor on numerous issued patents and patent applications. Dr. Stickney holds a Bachelor of Science in Microbiology, a Masters of Science in Immunology, and a M.D. from Ohio State University. In addition, he is certified as a Diplomat of the American Board of Internal Medicine and Hematology and a Diplomat of the American Board of Radiology, Therapeutic Radiology. Dr. Stickney s employment with the Company terminated effective March 31, 2011.

Robert W. Weber joined Harbor BioSciences in March 1996 and currently serves as the Chief Financial Officer and Secretary. Mr. Weber has over thirty years of experience in financial management. He has been employed at executive levels by multiple start-up companies and contributed to the success of several turnaround situations. He previously served as Vice President of Finance at Prometheus Products, a subsidiary of Sierra Semiconductor (now PMC Sierra), from 1994 to 1996, and Chief Financial Officer for Amercom, a personal computer telecommunications software publishing company, from 1993 to 1994. From February 1988 to August 1993, Mr. Weber served as Chief Financial Officer of Instromedix, a company that develops and markets medical devices and software. Mr. Weber brings a broad and expert knowledge of many aspects of financial management. In various capacities, he has been responsible for all aspects of finance and accounting including cost accounting, cash management, SEC filings, treasury, investor relations, private and venture financing, corporate legal matters, acquisitions/divestitures as well as information technology, human resources and facilities. Mr. Weber received a B.S. from GMI Institute of Technology (now Kettering University) and a MBA from the Stanford Graduate School of Business.

Item 1A. Risk Factors

In evaluating our business, you should consider the following discussion of risks, in addition to other information contained in this Annual Report on Form 10-K, as well as our other public filings with the Securities and Exchange Commission. Any of the following risks could materially adversely affect our business, financial condition, results of operations and prospects and, as a result, the market price of our common stock could decline, and you may lose all or part of the money you paid to buy our common stock.

We are still a development stage company.

We have never had any revenues from sales of products. None of our drug candidates has been approved for commercial sale and we do not expect that any of our present or future drug candidates will be commercially available for a number of years, if at all. We have incurred losses since our inception and we expect to continue to incur significant additional operating losses for the foreseeable future as we fund clinical trials and other expenses in support of regulatory approval of our drug candidates.

We need to raise additional money before we achieve profitability; if we fail to raise additional money, it could be difficult or impossible to continue our business.

As of December 31, 2011, our unrestricted cash and cash equivalents totaled approximately $1.5 million. In addition, subject to certain limitations, beginning January 1, 2012, and continuing until the Qualifying Transaction closes or until Amun exercises its put right, $200,000 per month will be disbursed to us from the Escrow account. Based on our current plans, these disbursements should be sufficient to meet our monthly operating expenses and capital requirements until such funds are depleted. As of January 15, 2012, the balance of the Escrow account was approximately $2.28 million. Further, if the Qualifying Transaction is consummated, we believe that our capital resources, together with interest thereon, would be sufficient to meet our operating expenses and capital requirements through 2013. If the Escrow account is not available to us, based on our current plans, we believe our unrestricted cash and cash equivalents, and interest earned thereon, would be sufficient to meet our operating

expenses and capital requirements for several months. However, changes in our research and development plans or other events affecting our operating expenses could result in the expenditure of such cash before that time. We will require substantial additional funds in order to finance our drug discovery and development programs, fund operating expenses, pursue regulatory clearances, develop manufacturing, marketing and sales capabilities, and prosecute and defend our intellectual property rights. We may seek additional funding through public or private financing or through collaborative arrangements with strategic partners.

You should be aware that in the future:

- we may not obtain additional financial resources when necessary or on terms favorable to us, if at all; the escrow funds may not be available to us; the Qualifying Transaction may not be consummated; and
- any available additional financing may not be adequate.

If we cannot raise additional funds when needed, or on acceptable terms, we will not be able to continue to develop our drug candidates.

We may need to liquidate the Company in a voluntary dissolution under Delaware law or seek protection under the provisions of the U.S. Bankruptcy Code, and in either event, it is unlikely that stockholders would receive any value for their shares.

We have not generated any revenues from product sales, and have incurred losses in each year since our inception in 1994. If a Qualifying Transaction is not consummated, we expect that it will be very difficult to raise capital to continue our operations and our independent registered public accounting firm has issued an opinion with an explanatory paragraph to the effect that there is substantial doubt about our ability to continue as a going concern in the annual report for the period ended December 31, 2011. If a Qualifying Transaction is not consummated, we do not believe that we could succeed in raising additional capital needed to sustain our operations without some strategic transaction, such as a partnership or merger. If we are unable to consummate such a transaction, we expect that we would need to cease all operations and wind down. Although we are currently evaluating our strategic alternatives with respect to all aspects of our business, we cannot assure you that any actions that we take would raise or generate sufficient capital to fully address the uncertainties of our financial position. As a result, we may be unable to realize value from our assets and discharge our liabilities in the normal course of business. If we are unable to settle our obligations to our creditors or if we are unable to consummate a strategic transaction, we would likely need to liquidate the Company in a voluntary dissolution under Delaware law or seek protection under the provisions of the U.S. Bankruptcy Code. In that event, we, or a trustee appointed by the court, may be required to liquidate our assets. In either of these events, we might realize significantly less from our assets than the values at which they are carried on our financial statements. The funds resulting from the liquidation of our assets would be used first to satisfy obligations to creditors before any funds would be available to pay our stockholders, and any shortfall in the proceeds would directly reduce the amounts available for distribution, if any, to our creditors and to our stockholders. In the event we are required to liquidate under Delaware law or the federal bankruptcy laws, it is highly unlikely that stockholders would receive any value for their shares. See Liquidity and Capital Resources in Part II, Item 7, Management s Discussion and Analysis of Financial Condition and Results of Operations and Note 1 to our financial statements.

We may be unable to obtain a quorum for meetings of our stockholders or obtain necessary stockholder approvals and therefore be unable to take certain actions

Our bylaws require that a quorum, consisting of a majority of the outstanding shares of voting stock, be present in person or by proxy in order to transact business at a meeting of our stockholders. In addition, amendments to our amended and restated certificate of incorporation, such as an amendment to increase our authorized capital stock, require the approval of a majority of our outstanding shares. Under Rule 452 of the New York Stock Exchange, the U.S. broker-dealer may vote shares absent direction from the beneficial owner on certain matters, such as an amendment to our amended and restated articles of incorporation to increase authorized shares that are to be used for general corporate purposes and the ratification of our auditors. As a result, unless more stockholders elect to be present in person or by proxy in future annual or special meetings of stockholders, we may be unable to obtain a quorum at such meetings or obtain stockholder approval of proposals when needed.

If we are unable to obtain a quorum at a meeting of our stockholders and thus fail to get stockholder approval of corporate actions, such failure could have a materially adverse effect on us. In addition, brokers may only vote on those matters for which broker discretionary voting is allowed under Rule 452 of the New York Stock Exchange, and we may not be able to obtain the required number of votes to approve certain proposals that require a majority of all outstanding shares to approve the proposal due to our reliance on broker discretionary voting. Therefore, it is possible that even if we are able to obtain a quorum for our meetings of the stockholders we still may not receive enough votes to approve proxy proposals presented at such meeting and, depending on the proposal in question, such failure could have a material adverse effect on us.

If we do not obtain government regulatory approval for our products, we cannot sell our products and we will not generate revenues.

Our principal efforts are currently centered on a proprietary class of small compounds that we believe shows promise for the treatment of several diseases and disorders. However, all drug candidates require approval by the United States Food and Drug Administration (FDA) before they can be commercialized in the United States as well as approval by various foreign government agencies before they can be commercialized in other countries. These regulations change from time to time and new regulations may be adopted. While limited clinical trials of our drug candidates have been conducted to date, significant additional trials are required, and we may not be able to demonstrate that our drug candidates are safe or effective. In addition, success in early development does not mean that later development will be successful because, for example, drug candidates in later-stage clinical trials may fail to demonstrate sufficient safety and efficacy despite having progressed through initial clinical testing. Our clinical experience with our drug candidates is limited, and to date our drug candidates have been tested in less than the number of patients that will likely need to be studied to gain regulatory approval. The data collected from clinical trials with larger patient populations may not demonstrate sufficient safety and efficacy to support regulatory approval of these drug candidates. In addition, we do not know whether early results from any of our ongoing clinical trials will be predictive of final results of any such trial. If we are unable to demonstrate the safety and effectiveness of a particular drug candidate to the satisfaction of regulatory authorities, the drug candidate will not obtain required government approval and we will experience potentially significant delays in, or be required to abandon development of the drug candidate. If we do not receive FDA or foreign approvals for our drug candidates, we will not be able to sell products and will not generate revenues. If we receive regulatory approval of one of our drug candidates, such approval may impose limitations on the indicated uses for which we may market the resulting product, which may limit our ability to generate significant revenues. Further, U.S. or foreign regulatory agencies could change existing, or promulgate new, regulations at any time, which may affect our ability to obtain approval of our drug candidates or require significant additional costs to obtain such approvals. In addition, if regulatory authorities determine that we or a partner conducting research and development activities on our behalf have not complied with regulations in the research and development of one of our drug candidates, then they may not approve the drug candidate and we will not be able to market and sell it. If we were unable to market and sell our drug candidates, our business and results of operations would be materially and adversely affected.

Recent publicity concerning the safety of certain drug products has resulted in heightened scrutiny by the FDA in the process of approving new drugs, which could delay or limit any regulatory approvals we may obtain for our drug candidates.

In light of widely publicized events concerning the safety risk of certain drug products, the FDA, members of Congress, the Government Accountability Office, medical professionals and the general public have raised concerns about potential drug safety issues. These events have resulted in the withdrawal of drug products, revisions to drug labeling that further limit use of the drug products and establishment of risk management programs that may, for instance, restrict distribution of drug products after approval. In addition, the Food and Drug Administration Amendments Act of 2007, or FDAAA, grants significant expanded authority to the FDA, much of which is aimed at improving the safety of drug products before and after approval. In particular, the new law authorizes the FDA to, among other things, require post-approval studies and clinical trials, mandate changes to drug labeling to reflect new safety information and require risk evaluation and mitigation strategies for certain drugs, including certain currently approved drugs. It also significantly expands the federal government s clinical trial registry and results databank, which we expect will result in significantly increased government oversight of clinical trials. Under the FDAAA, companies that violate these and other provisions of the new law are subject to substantial civil monetary penalties, among other regulatory, civil and criminal penalties. The increased attention to drug safety issues may result in a more cautious approach by the FDA in its review of data from our clinical trials. Data from clinical trials may receive greater scrutiny, particularly with respect to safety, which may make the FDA or other regulatory authorities more likely to require additional preclinical studies or clinical trials by drug development companies. As a result, the FDA may require us to conduct additional preclinical studies or clinical trials during the clinical development of one or more of our drug candidates as a condition precedent to approval which could potentially delay our development plans, limit the indications for which our drug candidates are ultimately approved, and otherwise adversely impact us.

If we do not successfully commercialize our products, we may never achieve profitability.

We have experienced significant operating losses to date because of the substantial expenses we have incurred to acquire and fund development of our drug candidates. We have never had significant operating revenues and have never commercially introduced a product. Our accumulated deficit was approximately $262 million as of December 31, 2011. Our net losses for fiscal years 2011, 2010 and 2009 were approximately $3.8 million, $6.6 million and $15.6 million, respectively. Many of our research and development programs are at an early stage. Potential drug candidates are subject to inherent risks of failure. These risks include the possibilities that no drug candidate will be found safe or effective, meet applicable regulatory standards or receive the necessary regulatory clearances. Even if we were ultimately to receive regulatory approval for one or more of our drug candidates, we may be unable to commercialize them successfully for a variety of reasons. These include, for example, the availability of alternative treatments, lack of cost effectiveness, the cost of manufacturing the product on a commercial scale, the effect of competition with other drugs, or because we may have inadequate financial or other resources to pursue one or more of our drug candidates through commercialization. If we are unable to develop safe, commercially viable drugs, we may never achieve profitability. If we become profitable, we may not remain profitable.

As a result of our intensely competitive industry, we may not gain enough market share to be profitable.

The biotechnology and pharmaceutical industries are intensely competitive. We have numerous competitors in the U.S. and elsewhere. Because we are pursuing potentially large markets, our competitors include major multinational pharmaceutical companies, specialized biotechnology firms as well as academic institutions, government agencies and private and public research institutions. Several of these entities have already successfully marketed and commercialized products that will compete with our drug candidates, assuming that our drug candidates gain regulatory approval. A large number of companies including Merck & Co., Inc., GlaxoSmithKline, Takeda Pharmaceuticals, Amylin Pharmaceuticals, Inc., AstraZeneca, Novartis, Novo Nordisk, Pfizer Inc., Sanofi-Aventis and Eli Lilly and Co. are developing and marketing new drugs for the treatment of type 2 diabetes. Similarly, a large number of companies, including Merck & Co., Inc., Pfizer Inc., Johnson & Johnson Inc. and Amgen Inc., are developing and marketing new drugs for the treatment of chronic inflammatory conditions. In addition, there are also a number of other companies with drug candidates in development targeting late-stage prostate cancer, including compounds already in Phase 3 clinical trials. One or more such compounds may be approved before any of our drug candidates could potentially be approved. Many, if not all, of these competing drug development programs are being conducted by pharmaceutical and biotechnology companies with considerably greater financial resources, human resources and experience than ours.

All of these competitors have greater financial and other resources, larger research and development staffs and more effective marketing and manufacturing organizations than we do. In addition, academic and government institutions have become increasingly aware of the commercial value of their research findings. These institutions are now more likely to enter into exclusive licensing agreements with commercial enterprises, including our competitors, to develop and market commercial products.

Our competitors may succeed in developing or licensing technologies and drugs that are more effective, or less costly than any we are developing. Our competitors may succeed in obtaining FDA or other regulatory approvals for drug candidates before we do. If competing drug candidates prove to be more effective or less costly or better-marketed than our drug candidates, our drug candidates, even if approved for sale, may not be able to compete successfully with our competitors existing products or new products under development. Similarly, we cannot predict whether any of our drug candidates, if approved, will have sufficient advantages to cause healthcare professionals to adopt our products over competing products. If we are unable to compete successfully, we may never be able to sell enough products at a price sufficient to permit us to generate profits.

Our common stock has a very limited trading market

Our common stock was recently delisted from the NASDAQ Stock Market and the OTC Bulletin Board (OTCBB) and now trades on the OTC Markets Group, Inc., informally known as Pink Sheets®. The Company s common stock was delisted from the NASDAQ Stock Market at the opening of business on September 23, 2010 at which time the common stock became available for trading on the OTCBB. On August 17, 2011, our common stock

was delisted from the OTCBB as a result of our filing a Form 15 pursuant to Rule 12g-4(a)(i), and subsequently became available for trading on the Pink Sheets® under the trading symbol HRBR.PK. The Pink Sheets® is not a stock exchange or a regulated entity. Price quotations are provided by over-the-counter market makers and company information is provided by the over-the-counter companies. The Pink Sheets® provide significantly less liquidity than the NASDAQ stock market or any other national securities exchange. The quotation of our common stock on the Pink Sheets® may reduce the price of our common stock. Further, the quotation of our common stock on the Pink Sheets® may materially adversely affect our ability to raise capital on terms acceptable to us or at all. In addition, trading in our common stock has historically been extremely limited. Further, our common stock may be subject to manipulation because of the thinness of the market for our stock. This limited trading may adversely affect the liquidity of our common stock, not only in terms of the number of shares that can be bought and sold at a given price, but also through delays in the timing of transactions and reduction in security analysts and the media s coverage of us. As a result, there could be a larger spread between the bid and the ask prices of our common stock and you may not be able to sell shares of our common stock when or at prices you desire.

We have deregistered our common stock under the Exchange Act, which could negatively affect the liquidity and trading prices of our common stock and result in less disclosure about the Company.

We have deregistered our common stock under the Exchange Act. By deregistering, our obligations to file reports with the SEC (including periodic reports, proxy statements, and tender offer statements) ceased and we expect that there will be a substantial decrease in the liquidity in our common stock even though stockholders may still continue to trade our common stock on the Pink Sheets®. In addition, companies with common stock quoted on the Pink Sheets® are not required to meet the reporting requirements set forth under the Exchange Act. There can be no assurances that we will provide financial information to allow for public trading of our common stock on the Pink Sheets®, or that market makers will continue to make a market in our common stock. As a result, investors may find it more difficult to dispose of or obtain accurate quotations as to the market value of our common stock, and the ability of our stockholders to sell our securities in the secondary market may be materially limited.

Substantial sales of our stock may impact the market price of our common stock.

As evidenced by the completion of our registered direct offering completed in June 2010, future sales of substantial amounts of our common stock, including shares that we may issue upon exercise of options and warrants or conversion of convertible securities, could adversely affect the market price of our common stock. Further, if we raise additional funds through the issuance of common stock or securities convertible into or exercisable for common stock, the percentage ownership of our stockholders will be reduced and the price of our common stock may fall.

Issuing preferred stock with rights senior to those of our common stock could adversely affect holders of common stock.

Our charter documents give our board of directors the authority to issue shares of preferred stock without a vote or action by our stockholders. The board also has the authority to determine the terms of preferred stock, including price, preferences and voting rights. The rights granted to holders of preferred stock may adversely affect the rights of holders of our common stock. For example, a series of preferred stock may be granted the right to receive a liquidation preference a pre-set distribution in the event of liquidation that would reduce the amount available for distribution to holders of common stock. In addition, the issuance of the Series A Preferred Stock makes it more difficult for a third party to acquire a majority of our outstanding voting stock. As a result, common stockholders could be prevented from participating in transactions that would offer an optimal price for their shares.

If we were to lose the services of members of our management team, or fail to attract or retain qualified personnel in the future, our business objectives would be more difficult to implement, adversely affecting our operations.

Our ability to successfully implement our business strategy depends upon the continued services of our management team. If we lose the services of one or more of these individuals, replacement could be difficult and may take an extended period of time and could impede significantly the achievement of our business objectives.

Our results of operations and liquidity needs could be materially negatively affected by market fluctuations and economic downturn.

Our results of operations could be materially negatively affected by economic conditions generally, both in the U.S. and elsewhere around the world. Continuing concerns over inflation, energy costs, geopolitical issues, the availability and cost of credit, the U.S. mortgage market and a declining residential real estate market in the U.S. have contributed to increased volatility and diminished expectations for the economy and the markets going forward. These factors, combined with volatile oil prices, declining business and consumer confidence and increased unemployment, precipitated an economic recession from which the global economy is in stages of recovery. Domestic and international equity markets continue to experience heightened volatility and turmoil. These events and the continuing market upheavals may have an adverse effect on us. In the event of a market downturn, our results of operations could be adversely affected by those factors in many ways, including making it more difficult for us to raise funds if necessary and our stock price may further decline.

Failure to protect our proprietary technology could impair our competitive position.

We own or have obtained a license to a number of U.S. and foreign patents and patent applications. Our success depends in part on our ability to obtain and defend patent rights and other intellectual property rights that are important to our ability to commercialize our drug candidates, if approved and our ability to operate our business without infringing the proprietary rights of third parties. We place considerable importance on obtaining patent protection for significant new technologies, products and processes. Legal standards relating to the validity of patents covering pharmaceutical and biotechnology inventions and the scope of claims made under such patents are still developing. In some of the countries in which we intend to market our drug candidates, if approved, pharmaceuticals are either not patentable or have only recently become patentable. Past enforcement of intellectual property rights in many of these countries has been limited or non-existent. Future enforcement of patents and proprietary rights in many other countries may be problematic or unpredictable. Moreover, the issuance of a patent in one country does not assure the issuance of a similar patent in another country. Claim interpretation and infringement laws vary by nation, so the extent of any patent protection is uncertain and may vary in different jurisdictions. Our domestic patent position is also highly uncertain and involves complex legal and factual questions. The applicant or inventors of subject matter covered by patent applications or patents owned by or licensed to us may not have been the first to invent or the first to file patent applications for such inventions. Due to uncertainties regarding patent law and the circumstances surrounding our patent applications, the pending or future patent applications we own or have licensed may not result in the issuance of any patents. Existing or future patents owned by or licensed to us may be challenged, infringed upon, invalidated, found to be unenforceable or circumvented by others. Further, any rights we may have under any issued patents may not provide us with sufficient protection against similar competitive products or technologies that do not infringe our patents or otherwise cover commercially valuable products or processes.

Litigation or other disputes regarding patents and other proprietary rights may be expensive, cause delays in bringing products to market and harm our ability to operate.

The manufacture, use or sale of our drug candidates may infringe on the patent rights of others. If we are unable to avoid infringement of the patent rights of others, we may be required to seek a license, defend an infringement action or challenge the validity of the patents in court. Patent litigation is costly and time consuming and can preclude, delay or suspend commercialization of our drug candidates. We may not have sufficient resources to bring these actions to a successful conclusion. In addition, if we do not obtain a license, develop or obtain non-infringing technology, or fail to successfully defend an infringement action or have the patents we are alleged to infringe declared invalid, we may:

- incur substantial money damages;
- encounter significant delays in bringing our drug candidates to market;
- be precluded from participating in the manufacture, use or sale of our drug candidates or methods of treatment without first obtaining licenses to do so; and/or
- not be able to obtain any required license on favorable terms, if at all.

In addition, if another party claims the same subject matter or subject matter overlapping with the subject matter that we have claimed in a U.S. patent application or patent, we may decide or be required to participate in interference proceedings in the U.S. Patent and Trademark Office in order to determine the priority of invention. Loss of such an interference proceeding would deprive us of patent protection sought or previously obtained and could prevent us from commercializing our products. Participation in such proceedings could result in substantial costs, whether or not the eventual outcome is favorable. These additional costs could adversely affect our financial results.

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Litigation may be expensive and time consuming and may adversely affect our operations.

From time to time, we may be involved in litigation relating to claims arising out of our operations in the normal course of business. Participation in such proceedings is time consuming and could result in substantial costs, whether or not the eventual outcome is favorable. These additional costs could adversely affect our financial results.

Confidentiality agreements with employees and others may not adequately prevent disclosure of trade secrets and other proprietary information.

In order to protect our proprietary technology and processes, we also rely in part on confidentiality agreements with our employees, consultants, outside scientific collaborators and sponsored researchers and other advisors. 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 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.

Existing and/or future pricing regulations and reimbursement limitations may limit our potential profits from the sale of our products.

The requirements governing product licensing, pricing and reimbursement vary widely from country to country. Some countries require approval of the sale price of a drug before it can be marketed. In many countries, the pricing review period begins after product-licensing approval is granted. As a result, we may obtain regulatory approval for a drug candidate in a particular country, but then be subject to price regulations that reduce our profits from the sale of the product. In some foreign markets pricing of prescription pharmaceuticals is subject to continuing government control even after initial marketing approval. In addition, certain governments may grant third parties a license to manufacture our product without our permission. Such compulsory licenses may be on terms that are less favorable to us and would likely have the effect of reducing our revenues.

Varying price regulation between countries can lead to inconsistent prices and some re-selling by third parties of products from markets where products are sold at lower prices to markets where those products are sold at higher prices. Any practice of exploiting price differences between countries could undermine our sales in markets with higher prices and reduce the sales of our future products, if any.

While we do not have any applications for regulatory approval of our drug candidates currently pending, any decline in the size of the markets in which we may in the future sell commercial products, assuming our receipt of the requisite regulatory approvals, could cause the perceived market value of our business and the price of our common stock to decline.

Our ability to commercialize our drug candidates successfully also will depend in part on the extent to which reimbursement for the cost of our drug candidates and related treatments will be available from government health administration authorities, private health insurers and other organizations. Third-party payers are increasingly challenging the prices charged for medical products and services. If we succeed in bringing any of our drug candidates to the market, such drug candidates may not be considered cost effective and reimbursement may not be available or sufficient to allow us to sell such drug candidates on a profitable or competitive basis.

Delays in the conduct or completion of preclinical or clinical studies or the analysis of the data from preclinical or clinical studies may result in delays in our planned filings for regulatory approvals, or adversely affect our ability to enter into collaborative arrangements.

The current status of our two lead drug candidates is set forth below. We have completed:

Phase I and I/II clinical trials with Triolex in the United States under an IND, for the treatment of metabolic disorders;

Phase IIa clinical trial with Triolex in the United States in type 2 diabetes patients under an IND for the treatment of metabolic disorders;

Phase I/II clinical trial with Triolex in the United States under an IND for the treatment of gastrointestinal inflammatory conditions;

Phase I clinical trial with Triolex in the United States in rheumatoid arthritis patients under an IND for the treatment of inflammatory conditions; and

Phase I/IIa clinical trial with Apoptone in the United States in late-stage prostate cancer patients who have failed hormone therapy and at least one round of chemotherapy treatment or have not received chemotherapy under an IND for the treatment of hormone-sensitive cancers including prostate cancer.

Any of the following reasons, among others, could delay or suspend the completion of our future studies:

delays in enrolling volunteers;

interruptions in the manufacturing of our drug candidates or other delays in the delivery of materials required for the conduct of our studies;

we may not be able to enter collaborative arrangements besides the CIPI agreements;

we can not control the uncertainties and lack direct control over the developments of our licensed compounds in China;

lower than anticipated retention rate of volunteers in a clinical trial;

unfavorable efficacy results;

serious side effects experienced by study participants relating to the drug candidate;

reaching agreement on acceptable terms with prospective contract research organizations and clinical trial sites;

failure to conduct a clinical trial in accordance with regulatory requirements or clinical protocols;

inspection of a clinical trial operations or clinical trial site by regulatory authorities resulting in the imposition of a clinical hold;

new communications from regulatory agencies about how to conduct these studies; or

failure to raise additional funds resulting in lack of adequate funding to continue a clinical trial or study.

If the manufacturers of our drug candidates do not comply with current Good Manufacturing Practices regulations, or cannot produce sufficient quantities of our drug candidates to enable us to continue our development, we will fall behind on our business objectives.

Manufacturers producing our drug candidates must follow current Good Manufacturing Practices regulations enforced by the FDA and foreign equivalents. If a manufacturer of our drug candidates does not conform to current Good Manufacturing Practices regulations and cannot be brought up to such a standard, we will be required to find alternative manufacturers that do conform. This may be a long and difficult process, and may delay our ability to receive FDA or foreign regulatory approval of our drug candidates.

We also rely on our manufacturers to supply us with a sufficient quantity of our drug candidates to conduct clinical trials. If we have difficulty in the future with obtaining our required quantity and quality of supply, we could experience significant delays in our development programs and regulatory process.

Our ability to achieve any significant revenue may depend on our ability to establish effective sales and marketing capabilities.

Our efforts to date have focused on the development and evaluation of our drug candidates. As we continue preclinical and clinical studies and seek to commercialize our drug candidates, we may need to build a sales and marketing infrastructure. As a company, we have no experience in the sales and marketing of pharmaceutical products. If we fail to establish a sufficient marketing and sales force or to make alternative arrangements to have our drug candidates marketed and sold by others on attractive terms, it will impair our ability to commercialize our drug candidates and to enter new or existing markets. Our inability to effectively enter these markets would materially and adversely affect our ability to generate significant revenues.

We may face product liability claims related to the use or misuse of our drug candidates, which may cause us to incur significant losses.

We are currently exposed to the risk of product liability claims due to administration of our drug candidates in clinical trials, since the use or misuse of our drug candidates during a clinical trial could potentially result in injury or death. If we are able to commercialize our products, we will also be subject to the risk of losses in the future due to product liability claims in the event that the use or misuse of our commercial products results in injury or death. We currently maintain liability insurance on a claims-made basis. Because we cannot predict the magnitude or the number of claims that may be brought against us in the future, we do not know whether the insurance policies coverage limits are adequate. The insurance is expensive, difficult to obtain and may not be available in the future on acceptable terms, or at all. Any claims against us, regardless of their merit, could substantially increase our costs and cause us to incur significant losses.

Our securities could be subject to extreme price fluctuations that could adversely affect your investment.

The market prices for securities of life sciences companies are highly volatile particularly those that are not profitable. Publicized events and announcements, most of which we cannot control, may have a significant impact on the market price of our common stock, which has been, and is likely to continue to be, volatile. For example:

biological or medical discoveries by competitors;

public concern about the safety of our drug candidates;

delays in the conduct or analysis of our preclinical or clinical studies;

unfavorable results from preclinical or clinical studies;

delays in obtaining or failure to obtain purchase orders of our drug candidates;

announcements in the scientific and research community;

changes in the potential commercial markets for our drug candidates;

unfavorable developments concerning patents or other proprietary rights;

unfavorable domestic or foreign regulatory or governmental developments or actions;

broader economic, industry and market trends unrelated to our performance;

issuances of new equity securities by us, pursuant to our effective shelf registration statement or otherwise;

discussion of us or our stock price by the financial and scientific press and in online investor communities; or

additions or departures of key personnel

may have the effect of temporarily or permanently driving down the price of our common stock. In addition, the stock market from time to time experiences extreme price and volume fluctuations which particularly affect the market prices for emerging and life sciences companies, such as ours, and which are often unrelated to the operating performance of the affected companies. For example, our stock price has ranged from $0.05 to $0.37 between July 1, 2010 and December 31, 2011.

These broad market fluctuations may adversely affect the ability of a stockholder to dispose of his shares at a price equal to or above the price at which the shares were purchased. In addition, in the past, following periods of volatility in the market price of a company s securities, securities class-action litigation has often been instituted against that company. Any litigation against the Company, including this type of litigation, could result in substantial costs and a diversion of management s attention and resources, which could materially adversely affect our business, financial condition and results of operations.

Item 1B. Unresolved Staff Comments

None.

Item 2. Properties

Our corporate headquarters are currently located at 9191 Towne Centre Drive, Suite 409, San Diego, CA 92122, where we have leased approximately 4,291 square feet of office space through January 2012. We believe that our facilities are adequate for our current operations.

Item 3. Legal Proceedings

From time to time, we may be involved in litigation relating to claims arising out of our operations in the normal course of business. While it is impossible to predict accurately or to determine the eventual outcome of these matters, as of the date of this report, we do not believe that we are engaged in any legal proceedings that are expected, individually or in the aggregate, to have a material adverse effect on our business, financial condition or operating results.

Item 4. Removed and Reserved.

PART II

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

Our common stock was recently delisted from the NASDAQ Stock Market and the OTCBB and now trades on the Pink Sheets® under the symbol HRBR.PK. Our common stock was delisted from the NASDAQ Capital Market on September 23, 2010 at which time the stock became available for trading on the OTCBB under the symbol HRBR.OB. On August 17, 2011, our common stock was delisted from the OTCBB as a result of our filing a Form 15 pursuant to Rule 12g-4(a)(i), and subsequently became available for trading on the Pink Sheets®.

The following table sets forth the quarterly high and low sales prices for our common stock from January 1, 2010 through December 31, 2011.

	High	Low
2010		
First Quarter	$ 0.82	$ 0.41
Second Quarter	0.70	0.26
Third Quarter	0.37	0.16
Fourth Quarter	0.22	0.12
2011		
First Quarter	$ 0.26	$ 0.13
Second Quarter	0.24	0.12
Third Quarter	0.26	0.10
Fourth Quarter	0.19	0.05

On December 31, 2011, the closing price of our common stock as reported by the Pink Sheets® was $0.13 share. There were approximately 120 stockholders of record and approximately 7,400 beneficial stockholders of our common stock as of such date. We have not paid cash dividends on our common stock and do not intend to do so in the foreseeable future.

On July 28, 2011, we sold an aggregate of 2,000,000 shares of our Series A Preferred Stock (the Preferred Shares) to Amun, LLC, a Delaware limited liability company (the Investor) pursuant to the terms of a Stock Purchase Agreement (the Purchase Agreement) and related Stockholders Agreement (the Stockholders Agreement). The Preferred Shares represent approximately a 28% of the economic interest in the Company and also entitle the Investor to a number of votes equal to 38.28% of the total number of votes entitled to be cast by holders of all shares of the Company s capital stock (including the Common Stock and Series A Preferred Stock) voting together as single class. Under the terms of these and other related agreements between the Company and the Investor, the Investor placed $2.825 million in cash into an escrow account, which amount is available under certain circumstances to pay certain Company related expenses and to fund the Company s working capital needs. Amounts received are included as restricted cash. The Stockholder Agreement provides that the Investor will have the right to put the Preferred Shares acquired pursuant to the Purchase Agreement back to the Company in return for the remaining cash held in escrow at the time of the put, upon the occurrence of certain events.

On October 26, 2011, we completed the reverse and forward stock splits which were approved by our shareholders at the annual meeting. As a result, the Company purchased the 43,698 common shares that were cancelled, at the previous ten-day average closing price of $0.142 for a total of $6,205. In addition, as a result of the stock splits, the investors in our June 2010 registered direct offering of common stock and warrants became eligible to exercise a put right under the warrants which entitled them to receive a cash payment in an amount equal to the fair value of the warrants as determined by reference to a formula set forth in the warrants. All of the warrant holders exercised the put right and the Company purchased the warrants at a price of $0.0955 for each underlying share for a total of $337,679. We were reimbursed for the cost of the cancelled common shares and the warrants purchased from the escrow account established with the funds from the sale of Preferred Shares to Amun.

Item 6. Selected Financial Data

The following data summarizes certain selected financial data for each of the five years ended December 31, 2011 through 2007 and the period from inception (August 15, 1994) to December 31, 2011. The information presented should be read in conjunction with the financial statements and related notes included elsewhere in this report (in thousands, except per share amounts).

	2011	2010	2009	2008	2007	Period from Inception (Aug. 15, 1994) to December 31, 2011
Statement of Operations Data:						
Contract revenues	$ 146	$ 0	$ 0	$ 0	$ 645	$ 1,354
Research and development	1,867	3,759	10,555	16,070	18,319	176,880(1)
General and administrative	2,102	2,771	5,140	6,537	8,150	95,790(1)
Total operating expenses	3,969	6,530	15,695	22,607	26,469	272,670
Interest income (expense)	6	16	138	1,048	2,781	17,385
Other income (expense)	14	(83)	(69)	(6)	(78)	(8,301)
Net loss	$ (3,803)	$ (6,597)	$ (15,626)	$ (21,565)	$ (23,121)	$ (262,232)
Net loss per share, basic and diluted	$ (0.11)	$ (0.20)	$ (0.53)	$ (0.74)	$ (0.80)	
Weighted average number of common Shares outstanding, basic and diluted	35,459	32,803	29,319	29,060	28,955	
Balance Sheet Data:						
Cash and equivalents	$ 1,470	$ 5,923	$ 9,738	$ 24,152	$ 43,215	
Total assets	4,025	6,096	10,286	25,157	45,123	
Total current liabilities	2,853	1,235	1,286	1,952	3,018	
Stockholders equity	$ 1,172	$ 4,861	$ 9,000	$ 23,205	$ 42,105	

(1) Share-Based Payment (ASC 718), expense was not included in financial results for any of the previous years prior to 2006. (See ASC 718, Share-Based Payments in the Notes to Financial Statements).

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

The following discussion contains forward-looking statements that involve risks and uncertainties. See Forward-Looking Statements elsewhere in this Annual Report on Form 10-K. This discussion and analysis should be read in conjunction with the financial statements and notes included elsewhere in this Annual Report.

General

We are a development-stage pharmaceutical company engaged in the discovery and development of drug candidates for the treatment of diseases and disorders in which the body is unable to mount an appropriate immune or metabolic response due to disease or the process of aging. Our initial technology development efforts are primarily focused on a series of adrenal steroid hormones and hormone analogs that are derived from our Hormonal Signaling Technology Platform.

We have been unprofitable since our inception. As of December 31, 2011, we had an accumulated deficit of approximately $262 million. We expect to incur substantial additional operating losses for the foreseeable future as we allocate resources to activities in support of the development of our drug candidates. In addition, in the future, we may have to meet the substantial new challenge of developing the capability to market products if we are successful in obtaining regulatory approval for any of our current or future drug candidates. Accordingly, our activities to date are not as broad in depth or scope as the activities we may undertake in the future, and our historical operations and financial information are not indicative of the future operating results or financial condition or ability to operate profitably as a commercial enterprise when and if we succeed in bringing any drug candidates to market.

On March 26, 1997, Hollis-Eden, Inc., a Delaware corporation, was merged with and into us, then known as Initial Acquisition Corp. (IAC), a Delaware corporation. Upon consummation of the merger of Hollis-Eden, Inc. with IAC, Hollis-Eden, Inc. ceased to exist, and IAC changed its name to Hollis-Eden Pharmaceuticals, Inc.

Effective February 9, 2010, we changed our name from Hollis-Eden Pharmaceuticals, Inc. to Harbor BioSciences, Inc. The name change was effected pursuant to Section 253 of the General Corporation Law of the State of Delaware by the merger of our wholly owned subsidiary into us. We were the surviving corporation and, in connection with the merger, we amended our Amended and Restated Certificate of Incorporation to change our name to Harbor BioSciences, Inc.

Effective February 18, 2010, our common stock began trading under a new NASDAQ symbol, HRBR and CUSIP number 41150V 103 . Our common stock was delisted from NASDAQ in September, 2010, at which time the stock became available for trading on the OTCBB under the symbol HRBR.OB. On August 17, 2011, our common stock was delisted from the OTCBB as a result of our filing a Form 15 pursuant to Rule 12g-4(a)(i), and subsequently became available for trading on the Pink Sheets® with the symbol HRBR.PK and CUSIP number 41150V 202 .

Results of Operations

We have devoted substantially all of our resources to the payment of research and development expenses and general and administrative expenses. From inception through December 31, 2011, we have incurred approximately $176.9 million in research and development expenses, $95.8 million in general and administrative expenses. From inception, (August 15, 1994), through December 31, 2011 we have generated approximately $1.4 million in revenues (which resulted from providing research and development services under our Study Funding Agreements with Cystic Fibrosis Foundation Therapeutics, Inc., (CFFT) and the Michael J. Fox Foundation (MJFF). We have earned $9.1 million in other income. The other income and expense is comprised of $7.6 million in deemed discount expense, $0.4 million in interest expense and a $0.3 million loss on disposal of assets. These expenses have been offset by $17.4 million in interest income. The combination of these resulted in a net loss of $262.2 million for the period from inception, (August 15, 1994), until December 31, 2011.

Research and development and general and administrative expenses include the expense for ASC 718 share-based payments for all fiscal years starting with 2006, (See ASC 718 Share-Based Payments in the Notes to Financial Statements).

Research and development expenses were $1.9 million, $3.8 million and $10.6 million in 2011, 2010 and 2009, respectively. The research and development expenses relate primarily to the ongoing development, preclinical testing and clinical trials for our drug candidates. Research and development expenses decreased $1.9 million in 2011 compared to 2010; $6.8 million in 2010 compared to 2009, due primarily to a decrease in general clinical and preclinical research and development projects resulting from reduced personnel and a decline in stock option compensation expense.

General and administrative expenses were $2.1 million, $2.8 million and $5.1 million in 2011, 2010 and 2009, respectively. General and administrative expenses relate to salaries and benefits, facilities, patent fees, legal, accounting/auditing, investor relations, consultants, insurance and travel. General and administrative expenses decreased $0.7 million in 2011 compared to 2010; $2.3 million in 2010 compared to 2009 due mainly to a decreases in salaries expense resulting from reduced personnel, patent fees, consulting fees, Directors and Officers insurance and stock option compensation expense.

Other income and expenses were $20 thousand, ($67) thousand and $69 thousand in 2011, 2010 and 2009, respectively. The increase in income of $87 thousand for 2011 compared to 2010 was due to a gain on sale of assets in 2011 compared to a loss on sale of assets in 2010. The increase in expense of $136 thousand for 2010 compared to 2009 was due mainly to the disposal of assets combined with lower interest rates and lower cash balances. Included in the 2010 loss was $83 thousand related to the sale of our laboratory equipment and $69 thousand for 2009.

Liquidity and Capital Resources

Our operations to date have consumed substantial capital without generating any revenues other than the amount received under our collaboration with CFFT and MJFF. We have financed our operations since inception primarily through the sale of our equity securities raising a total of $205 million, net of expenses. In addition, we have received a total of $18 million from the exercise of warrants and stock options from inception. As of December 31, 2011, our unrestricted cash and cash equivalents totaled approximately $1.5 million and a $2.48 million balance in the restricted cash account. The Amun investors have a put right on the restricted cash account and thus these funds may be returned to these investors. In addition, subject to certain limitations, beginning January 1, 2012, and continuing until the Qualifying Transaction closes or until Amun exercises it s put right, $200,000 per month will be disbursed to us from the Escrow account. Based on our current plans, these disbursements should be sufficient to meet our monthly operating expenses and capital requirements until such funds are depleted. As of January 15, 2012, the balance of the Escrow account was approximately $2.28 million. Further, if the Qualifying Transaction is consummated, we believe that our capital resources, together with interest thereon, would be sufficient to meet our operating expenses and capital requirements through 2013. If the Escrow account is not available to us, based upon our current plans, we believe that our unrestricted cash and cash equivalents, and interest thereon, would be sufficient to meet our operating expenses and capital expenditures for several months. However, changes in our research and development plans or other events affecting our operating expenses may result in the expenditure of such cash before that time. However, changes in our research and development plans or other events affecting our operating expenses could result in the expenditure of such cash before that time.

Our future capital requirements will depend upon many factors, including progress with preclinical testing and clinical trials, the number and breadth of our programs, the time and costs involved in preparing, filing, prosecuting, maintaining and enforcing patent claims and other proprietary rights, the time and costs involved in obtaining regulatory approvals, competing technological and market developments, and our ability to establish collaborative arrangements, effective commercialization, marketing activities and other arrangements. We may incur negative cash flows and net losses for the foreseeable future.

We expect that it will be very difficult to raise capital to continue our operations and our independent registered public accounting firm has issued an opinion with an explanatory paragraph to the effect that there is substantial doubt about our ability to continue as a going concern. We do not believe that we could succeed in raising additional capital needed to sustain our operations without some strategic transaction, such as a partnership in addition to the China license agreements or a merger. If we are unable to consummate such a transaction, we expect that we would need to cease all operations and wind down. Although we are currently evaluating our strategic alternatives with respect to all aspects of our business, we cannot assure you that any actions that we take would raise or generate sufficient capital to fully address the uncertainties of our financial position. As a result, we may be unable to realize value from our assets and discharge our liabilities in the normal course of business. If we are unable to settle our obligations to our creditors or if we are unable to consummate a strategic transaction, we would likely need to liquidate the Company in a voluntary dissolution under Delaware law or seek protection under the provisions of the U.S. Bankruptcy Code. In that event, we, or a trustee appointed by the court, may be required to liquidate our assets. In either of these events, we might realize significantly less from our assets than the values at which they are carried on our financial statements. The funds resulting from the liquidation of our assets would be used first to satisfy obligations to creditors before any funds would be available to pay our stockholders, and any shortfall in the proceeds would directly reduce the amounts available for distribution, if any, to our creditors and to our stockholders. In the event we are required to liquidate under Delaware law or the federal bankruptcy laws, it is highly unlikely that stockholders would receive any value for their shares.

Off-Balance Sheet Arrangements

Harbor BioSciences, Inc. currently does not have any off-balance sheet arrangements.

Contractual Obligations:

As of December 31, 2011, we had the following contractual obligations

		Payments Due by Period			
Contractual Obligations	Total	Less than one year	One to three years	Three to five years	More than Five years
Operating Leases	$ 9	$ 9	$	$	$

We may also be required to make substantial milestone or royalty payments in cash based on the terms of some of our agreements (See Note 6 to the Financial Statements).

Our operations to date have consumed substantial capital without generating any revenues other than the amount received under our collaborations with CFFT and MJFF. We will continue to require substantial and increasing amounts of funds to conduct necessary research and development and preclinical and clinical testing of our drug candidates, and to market any drug candidates that receive regulatory approval. We do not expect to generate revenue from operations for the foreseeable future, and our ability to meet our cash obligations as they become due and payable may depend for at least the next several years on our ability to sell securities, borrow funds or some combination thereof.

Our future capital requirements will depend upon many factors, including progress with preclinical testing and clinical trials, the number and breadth of our programs, the time and costs involved in preparing, filing, prosecuting, maintaining and enforcing patent claims and other proprietary rights, the time and costs involved in obtaining regulatory approvals, competing technological and market developments, and our ability to establish collaborative arrangements, effective commercialization, marketing activities and other arrangements. We may incur increasing negative cash flows and net losses for the foreseeable future. We are seeking additional funding through public or private financing or through collaborative arrangements with strategic partners. Our auditor has stated in the opinion that there is substantial doubt about the Company s ability to continue as a going concern.

Critical Accounting Policies

Certain of our accounting policies require the application of judgment and estimates by management, which may be affected by different assumptions and conditions. These estimates are typically based on historical experience, terms of existing contracts, trends in the industry and information available from other outside sources, as appropriate. We believe the estimates and judgments associated with our reported amounts are appropriate in the circumstances. Actual results could materially vary from those estimates under different assumptions or conditions.

All research and development costs are expensed as incurred. The value of acquired in-process research and development is charged to expense on the date of acquisition. Research and development expenses include, but are not limited to, acquired in-process technology deemed to have no alternative future use, license fees related to license agreements, preclinical and clinical trial studies, payroll and personnel expense, and lab supplies, consulting and research-related overhead. Research and development expenses paid in the form of cash and our stock to related parties aggregated $11.5 million for the period from inception (August 15, 1994) to December 31, 2003 (see Note 6, Colthurst, Edenland and Mr. Prendergest and Aeson Therapeutics). No such related party expenses were incurred in 2011, 2010 or 2009.

As of January 1, 2006, we account for share-based payments in accordance with ASC 718. Under the fair value recognition provisions of this statement, share-based payments cost is measured at the grant date based on the value of the award and is recognized as expense over the vesting period. Determining the fair value of share-based awards at the grant date requires judgment, including estimating our stock price volatility and employee stock option exercise behavior. If actual results differ significantly from these estimates, stock-based compensation expense and our results of operations could be materially impacted.

Our expected volatility is based upon the historical volatility of our stock. Our expected life for our options is based on historical stock option activity. Because share-based payments expense is recognized in our statement of operations based on awards ultimately expected to vest, the amount of expense has been reduced for estimated forfeitures. ASC 718 requires forfeitures to be estimated at the time of grant and revised, if necessary, in subsequent periods if actual forfeitures differ from those estimates. Forfeitures were estimated based on historical experience. If factors change and we employ different assumptions in the application of ASC 718, the compensation expense that we record in future periods may differ significantly from what we have recorded in the current period.

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On July 13, 2006, ASC 740-10, Accounting for Uncertainty in Income Taxes, which is effective for fiscal years beginning after December 15, 2006, which establishes recognition and measurement thresholds that must be met before a tax benefit can be recognized in the financial statements. The Company has adopted ASC 740-10 on January 1, 2007, and it has had no material impact on its financial statements.

Item 7A. Quantitative and Qualitative Disclosures about Market Risk

At December 31, 2011, our investment portfolio included only cash and money market accounts and did not contain fixed-income securities. There would be no material impact to our investment portfolio, in the short term, associated with any change in interest rates, and any decline in interest rates over time will reduce our interest income, while increases in interest rates over time will increase our interest income.

Item 8. Financial Statements and Supplementary Data

Harbor BioSciences, Inc. (A Development Stage Company)

Harbor BioSciences, Inc.

(A Development Stage Company)

Balance Sheets

(In thousands, except par value)	**December 31,**	
	2011	**2010**
ASSETS:		
Current assets:		
Cash and cash equivalents	$ 1,470	$ 5,923
Prepaid expenses	60	100
Deposits	10	28
Other receivables	0	1
Total current assets	1,540	6,052
Property and equipment, net of accumulated depreciation of $66 and $273	7	44
Restricted cash	2,478	0
Total assets	$ 4,025	$ 6,096
LIABILITIES AND STOCKHOLDERS EQUITY:		
Current liabilities:		
Accounts payable	$ 7	$ 201
Accrued expenses	346	1,005
Redeemable preferred stock	2,478	0
Other current liabilities	22	29
Total current liabilities	2,853	1,235
Commitments and contingencies (Notes 6, 11, 12)		
Stockholders equity: (Notes 3, 7, 8, 9, 10)		
Preferred stock, $.01, 10,000 shares authorized; no shares issued or outstanding	0	0
Common stock, $.01 par value, 100,000 shares authorized; 35,481 and 35,525 shares issued and 35,422 and 35,466 outstanding respectively	355	355
Paid-in capital	263,395	263,281
Cost of treasury stock (59 shares)	-346	-346
Deficit accumulated during development stage	-262,232	-258,429
Total stockholders equity	1,172	4,861
Total liabilities and stockholders equity	$ 4,025	$ 6,096

The accompanying notes are an integral part of these financial statements.

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Operations

	For the year ended December 31,			Period from Inception (Aug.15, 1994) to December 31,
	2011	2010	2009	2011
	(In thousands, except per share amounts)			
Revenue:				
Contract R&D revenue	$ 146	$ 0	$ 0	$ 1,354
Operating expenses:				
Research and development	1,867	3,759	10,555	176,880
General and administrative	2,102	2,771	5,140	95,790
Total operating expenses	3,969	6,530	15,695	272,670
Other income (expense):				
Gain / (loss) on disposition of assets	14	(83)	(69)	(286)
Non-cash amortization of deemed discount and deferred issuance costs on convertible debentures	0	0	0	(7,627)
Interest income	6	16	138	17,385
Interest expense	0	0	0	(388)
Total other income, net	20	(67)	69	9,084
Net loss	-$ 3,803	-$ 6,597	-$ 15,626	-$ 262,232
Net loss per share, basic and diluted	-$ 0.11	-$ 0.20	-$ 0.53	
Weighted average number of common shares outstanding, basic and diluted	35,459	32,803	29,319	

The accompanying notes are an integral part of these financial statements

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Stockholders Equity

(In thousands)	Preferred stock at par value		Common stock at par value		Capital in excess of par value	Cost of Repurchased Common Stock		Deficit accumulated during development stage	Total
	Shares	Amount	Shares	Amount		Shares	Amount		
Contribution by stockholder	0	$ 0	0	$ 0	$ 103	0	$ 0	$ 0	$ 103
Common stock issued for cash	0	0	2,853	0	25	0	0	0	25
Common stock issued as consideration for the license agreements (Note 6)	0	0	543	0	5	0	0	0	5
Net loss	0	0	0	0	0	0	0	-1,277	-1,277
Balance at December 31, 1994	0	0	3,396	0	133	0	0	-1,277	-1,144
Common stock issued for cash	0	0	679	0	250	0	0	0	250
Common stock issued as consideration for amendments to the license agreements (Note 6)	0	0	76	0	28	0	0	0	28
Net loss	0	0	0	0	0	0	0	-672	-672
Balance at December 31, 1995	0	0	4,151	0	411	0	0	-1,949	-1,538
Common stock issued in conversion of debt (Note 7)	0	0	165	0	371	0	0	0	371
Common stock issued for cash, net of expenses (Note 7)	0	0	580	0	1,234	0	0	0	1,234
Common stock issued as consideration for termination of a finance agreement	0	0	15	0	34	0	0	0	34
Warrants issued to consultants for services rendered	0	0	0	0	24	0	0	0	24
Net loss	0	0	0	0	0	0	0	-692	-692
Balance at December 31, 1996	0	0	4,911	0	2,074	0	0	-2,641	-567
Recapitalization of Company upon the merger with Initial Acquisition Corp. (Note 3)	0	0	883	58	6,213	0	0	0	6,271
Warrants issued to a certain director upon the successful closure of the merger (Note 3)	0	0	0	0	570	0	0	0	570
Exercise of warrants, net of expenses	0	0	978	10	5,619	0	0	0	5,629
Amortization of deferred compensation	0	0	0	0	282	0	0	0	282
Exercise of stock options	0	0	0	0	1	0	0	0	1
Net loss	0	0	0	0	0	0	0	-5,253	-5,253
Balance at December 31, 1997	0	0	6,772	68	14,759	0	0	-7,894	6,933
Exercise of warrants	0	0	399	4	1,196	0	0	0	1,200
Exercise of stock options	0	0	53	1	155	0	0	0	156
Private Placement, net of expenses (Note 7)	4	0	1,329	13	19,877	0	0	0	19,890
Warrants issued for services in lieu of cash (Note 10)	0	0	0	0	408	0	0	0	408
Stock issued for license fee (Note 6)	0	0	33	0	500	0	0	0	500
Stock issued for services in lieu of cash	0	0	6	0	95	0	0	0	95
	0	0	0	0	240	0	0	0	240

Options issued for services in lieu of cash (Note 9)									
Amortization of deferred compensation	0	0	0	0	308	0	0	0	308
Net loss	0	0	0	0	0	0	0	-5,427	-5,427
Balance at December 31, 1998	4	0	8,592	86	37,538	0	0	-13,321	24,303

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Stockholders Equity

(In thousands)	Preferred stock at par value		Common stock at par value		Capital in excess of par value	Cost of Repurchased Common Stock		Deficit accumulated during development stage	Total
	Shares	Amount	Shares	Amount		Shares	Amount		
Exercise of warrants	0	0	755	8	5,136	0	0	0	5,144
Exercise of stock options	0	0	10	0	75	0	0	0	75
Private Placement, net of expenses (Note 7)	0	0	1,368	14	24,759	0	0	0	24,773
Preferred Stock Conversion (Note 7,8)	(4)	0	346	3	-3	0	0	0	0
Deferred compensation-Options forfeited (Note 9)	0	0	0	0	51	0	0	0	51
Amortization of non-employee options	0	0	0	0	559	0	0	0	559
Warrants issued for services in lieu of cash (Note 10)	0	0	0	0	2,140	0	0	0	2,140
Options accelerated vesting (Note 9)	0	0	0	0	4,900	0	0	0	4,900
Net loss	0	0	0	0	0	0	0	-15,320	-15,320
Balance at December 31, 1999	0	0	11,071	111	75,155	0	0	-28,641	46,625
Exercise of warrants	0	0	133	2	758	0	0	0	760
Exercise of stock options	0	0	1	0	5	0	0	0	5
Common Stock issued for 401(k)/401(m) plan	0	0	6	0	63	0	0	0	63
Common Stock issued for In-Process R&D (Note 6)	0	0	209	2	1,998	0	0	0	2,000
Options granted for license fee	0	0	38	0	598	0	0	0	598
Amortization of non-employee options	0	0	0	0	79	0	0	0	79
Common Stock issued for purchase of technology	0	0	132	1	1,847	0	0	0	1,848
Net loss	0	0	0	0	0	0	0	-19,515	-19,515
Balance at December 31, 2000	0	0	11,590	116	80,503	0	0	-48,156	32,463
Exercise of stock options	0	0	10	0	22	0	0	0	22
Common Stock issued for 401(k)/401(m) plan	0	0	16	0	96	0	0	0	96
Private Placement, net of expenses (Note 7)	0	0	1,280	13	10,644	0	0	0	10,657
Warrants issued for services in lieu of cash (Note 10)	0	0	0	0	80	0	0	0	80
Amortization of non-employee options	0	0	0	0	96	0	0	0	96
Warrants issued for services	0	0	0	0	208	0	0	0	208
Net loss	0	0	0	0	0	0	0	-15,762	-15,762
Balance at December 31, 2001	0	0	12,896	129	91,649	0	0	-63,918	27,860
Exercise of stock options	0	0	0	0	2	0	0	0	2
Common Stock issued for 401(k)/401(m) plan	0	0	26	0	137	0	0	0	137
Common Stock issued for sublicense agreement (Note 6)	0	0	50	1	204	0	0	0	205
Common Stock issued to consultants	0	0	0	0	17	0	0	0	17
Amortization of non-employee options	0	0	0	0	66	0	0	0	66
Warrants issued for services	0	0	0	0	247	0	0	0	247
Net loss	0	0	0	0	0	0	0	-17,502	-17,502
Balance at December 31, 2002	0	0	12,972	130	92,322	0	0	-81,420	11,032

Common Stock issued for 401(k)/401(m) plan	0	0	32	0	223	0	0	0	223
Exercise of warrants	0	0	467	5	3,323	0	0	0	3,328
Exercise of stock options	0	0	85	1	955	0	0	0	956
Stock options issued	0	0	0	0	561	0	0	0	561
Private Placement, net of expenses	0	0	1,283	13	14,290	0	0	0	14,303
Common Stock issued for sublicense agreement (Note 6)	0	0	119	1	644	0	0	0	645
Common Stock issued for milestone payment	0	0	50	1	281	0	0	0	282

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Stockholders Equity

(In thousands)	Preferred stock at par value		Common stock at par value		Capital in excess of par value	Cost of Repurchased Common Stock		Deficit accumulated during development stage	Total
	Shares	Amount	Shares	Amount		Shares	Amount		
Debt Conversion	0	0	1,755	17	9,983	0	0	0	10,000
Common Stock issued in lieu of cash / interest	0	0	9	0	142	0	0	0	142
Public Offering, net of expenses	0	0	2,500	25	58,576	0	0	0	58,601
Deemed discount on convertible debentures	0	0	0	0	6,470	0	0	0	6,470
Warrants issued for services	0	0	0	0	1,398	0	0	0	1,398
Amortization of non-employee options	0	0	0	0	128	0	0	0	128
Purchase of treasury stock	0	0	0	0	0	-59	-346	0	-346
Net loss	0	0	0	0	0	0	0	-25,671	-25,671
Balance at December 31, 2003	0	0	19,272	193	189,296	-59	-346	-107,091	82,052
Common Stock issued for 401(k) plan	0	0	17	0	147	0	0	0	147
Exercise of warrants	0	0	6	0	11	0	0	0	11
Exercise of stock options	0	0	4	0	16	0	0	0	16
Common Stock issued for In-Process R&D (Note 6)	0	0	48	0	629	0	0	0	629
Amortization of non-employee options	0	0	0	0	136	0	0	0	136
Net loss	0	0	0	0	0	0	0	-24,757	-24,757
Balance at December 31, 2004	0	0	19,347	193	190,235	-59	-346	-131,848	58,234
Common Stock issued for 401(k) plan	0	0	25	0	151	0	0	0	151
Exercise of warrants	0	0	42	1	260	0	0	0	261
Exercise of stock options	0	0	35	1	123	0	0	0	124
Public Offering, net of expenses (Note 7)	0	0	1,333	13	9,502	0	0	0	9,515
Amortization of non-employee options	0	0	0	0	30	0	0	0	30
Net loss	0	0	0	0	0	0	0	-29,441	-29,441
Balance at December 31, 2005	0	0	20,782	208	200,301	-59	-346	-161,289	38,874
Common Stock issued for 401(k) plan	0	0	45	1	224	0	0	0	225
Exercise of warrants	0	0	10	0	1	0	0	0	1
Warrants issued to consultants	0	0	0	0	226	0	0	0	226
Exercise of stock options	0	0	34	0	86	0	0	0	86
Private Placements, net of expenses	0	0	8,000	80	48,697	0	0	0	48,777
Stock-Based Compensation Expense	0	0	0	0	3,534	0	0	0	3,534
Amortization of non-employee warrants	0	0	0	0	13	0	0	0	13
Restricted stock grant, net of forfeitures	0	0	65	1	401	0	0	0	402
Common Stock issued for In-Process R&D	0	0	35	0	180	0	0	0	180
Deferred Compensation	0	0	0	0	-309	0	0	0	-309
Net loss	0	0	0	0	0	0	0	-30,231	-30,231
Balance at December 31, 2006	0	0	28,971	290	253,354	-59	-346	-191,520	61,778
Common Stock issued for 401(k) plan	0	0	96	1	192	0	0	0	193
Exercise of stock options	0	0	9	0	20	0	0	0	20
Stock-Based Compensation Expense	0	0	0	0	3,128	0	0	0	3,128

Restricted Stock Forfeitures	0	0	-12	0	-33	0	0	0	-33
Amortization of non-employee warrants	0	0	0	0	17	0	0	0	17
Deferred Compensation	0	0	0	0	123	0	0	0	123
Net loss	0	0	0	0	0	0	0	-23,121	-23,121
Balance at December 31, 2007	0	0	29,064	291	256,801	-59	-346	-214,641	42,105

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Stockholders Equity

(In thousands)	Preferred stock at par value		Common stock at par value		Capital in excess of	Cost of Repurchased Common Stock		Deficit accumulated during development stage	Total
	Shares	Amount	Shares	Amount	par value	Shares	Amount		
Common Stock issued for 401(k) plan	0	0	164	1	174	0	0	0	175
Stock-Based Compensation Expense	0	0	0	0	2,404	0	0	0	2,404
Deferred Compensation	0	0	0	0	86	0	0	0	86
Net loss	0	0	0	0	0	0	0	-21,565	-21,565
Balance at December 31, 2008	0	0	29,228	292	259,465	-59	-346	-236,206	23,205
Common Stock issued for 401(k) plan	0	0	271	2	96	0	0	0	98
Restricted Stock Forfeitures	0	0	-6	0	-3	0	0	0	-3
Stock-Based Compensation Expense	0	0	0	0	1,240	0	0	0	1,240
Deferred Compensation	0	0	0	0	86	0	0	0	86
Net loss	0	0	0	0	0	0	0	-15,626	-15,626
Balance at December 31, 2009	0	0	29,493	294	260,884	-59	-346	-251,832	9,000
Common Stock issued for 401(k) plan	0	0	137	2	41	0	0	0	43
Net Proceeds from Financing	0	0	5,895	59	1,730	0	0	0	1,789
Stock-Based Compensation Expense	0	0	0	0	612	0	0	0	612
Deferred Compensation	0	0	0	0	14	0	0	0	14
Net loss	0	0	0	0	0	0	0	-6,597	-6,597
Balance at December 31, 2010	0	0	35,525	355	263,281	-59	-346	-258,429	4,861
Purchase of Fractional Shares as a result of Reverse Stock Split	0	0	-44	0	-6	0	0	0	-6
Stock-Based Compensation Expense	0	0	0	0	114	0	0	0	114
Warrant Liability	0	0	0	0	-338	0	0	0	-338
Release of Redemption of Preferred Stock	0	0	0	0	344	0	0	0	344
Net loss	0	0	0	0	0	0	0	-3,803	-3,803
Balance at December 31, 2011	0	0	35,481	$ 355	$ 263,395	-59	-$ 346	-$ 262,232	$ 1,172

The accompanying notes are an integral part of these financial statements.

Harbor BioSciences, Inc.

(A Development Stage Company)

Statements of Cash Flows

(In thousands)	2011	2010	2009	Period from Inception (Aug. 15, 1994) to December 31, 2011
Cash flows from operating activities:				
Net loss	-3,803	-6,597	-15,626	-262,232
Adjustments to reconcile net loss to net cash used in operating activities:				
Depreciation	2	21	208	2,245
Disposal of assets	-14	83	69	286
Compensation expense related to equity awards	114	626	1,323	11,398
Amortization of deemed discount on convertible debentures	0	0	0	6,470
Amortization of deferred issuance cost	0	0	0	1,157
Common stock issued for 401k/401m plan	0	43	98	1,550
Common stock and options issued as consideration for license fees and amendments, milestone payment, interest, note repayment and services	0	0	0	2,926
Expense related to warrants issued as consideration to consultants	0	0	0	4,369
Expense related to warrants issued to a director for successful closure of merger	0	0	0	570
Expense related to stock options issued	0	0	0	5,718
Expense related to common stock issued for the purchase of technology	0	0	0	1,848
Common stock issued as consideration for In-Process R&D	0	0	0	2,809
Deferred compensation expense related to options issued	0	0	0	1,210
Changes in assets and liabilities:				
Prepaid expenses	40	109	53	-60
Deposits	18	20	20	-10
Other receivable	1	80	-81	0
Accounts payable	-194	67	-187	714
Accrued expenses	-659	-145	-479	300
Other Liabilities	-7	29	0	22
Net cash used in operating activities	-4,502	-5,664	-14,602	-218,710
Cash flows provided by investing activities:				
Proceeds from sale of property and equipment	49	26	197	272
Purchase of property and equipment	0	0	-9	-2,825
Net cash provided by (used in) investing activities	49	26	188	-2,553
Cash flows from financing activities:				
Contributions from stockholder	0	0	0	104
Restricted Cash	-2,825	34	0	-2,825
Redeemable Preferred Stock	2,478	0	0	2,478
Restricted Cash used in settlement of warrant liability	338	0	0	338
Restricted Cash used in settlement of stock related to reverse stock split	6	0	0	6
Restricted Cash used for bank fees	3	0	0	3
Net proceeds from sale of preferred stock	0	0	0	4,000
Net proceeds from sale of common stock	0	1,789	0	185,323
Net proceeds from issuance of convertible debentures and warrants	0	0	0	9,214
Purchase of treasury stock	0	0	0	-346
Proceeds from issuance of debt	0	0	0	371

Net proceeds from recapitalization	0	0	0	6,271
Net proceeds from warrants/options exercised	0	0	0	17,796
Net cash provided by financing activities	0	1,823	0	222,733
Net increase (decrease) in cash and equivalents	-4,453	-3,815	-14,414	1,470
Cash and equivalents at beginning of period	5,923	9,738	24,152	0
Cash and equivalents at end of period	1,470	5,923	9,738	1,470

(In thousands)	2011	2010	2009	Period from Inception (Aug. 15, 1994) to December 31, 2011
Supplemental disclosure of cash flow information:				
Interest paid	0	0	0	388
Conversion of debt to equity	0	0	0	10,371
Warrants issued to consultants in lieu of cash, no vesting	0	0	0	559
Warrants issued in lieu of cash, commissions on private placement	0	0	0	733
Warrants issued in connection with convertible debentures	0	0	0	371

The accompanying notes are an integral part of these financial statements.

Harbor BioSciences, Inc.

(A Development Stage Company)

Notes to Financial Statements

1. The Company

Harbor BioSciences, Inc., (Harbor BioSciences or the Company), a development stage pharmaceutical company, is engaged in the discovery, development and commercialization of products for the treatment of diseases related to aging. From inception (August 15, 1994) through March 1997, the Company s efforts were directed toward organizing, licensing technology and preparing for offerings of shares of its common stock. Since 1997, the Company has been expanding its intellectual property, developing its lead drug candidates, performing preclinical tests and has entered into and completed multiple clinical studies. Our primary technology development efforts are focused on a series of adrenal steroid hormones and synthetic analogs that may be useful in treating a wide variety of medical conditions, if successfully developed. These adrenal hormones are depleted during advancing age, a process accelerated by infectious diseases and chronic immune system disorders. High plasma concentrations of these hormones are positively correlated with attenuated disease, in certain indications, and their maintenance is often associated with healthy aging.

During the past three years, the Company has devoted substantially all of its research, development and clinical efforts and financial resources toward the development of Apoptone and Triolex. The Company has incurred a net loss of $3.8 million in 2011, has had cumulative net losses of $262.2 million from inception to date and has limited financial resources at December 31, 2011.

These events raise substantial doubt about the Company s ability to continue as a going concern. The accompanying financial statements have been prepared assuming that the Company will continue as a going concern. This basis of accounting contemplates the recovery of the Company s assets and the satisfaction of liabilities in the normal course of business and does not include any adjustments to reflect the possible future effects on the recoverability and classification of assets or the amounts and classification of liabilities that might be necessary should the Company be unable to continue as a going concern. In an effort to preserve cash, the Company initiated steps during 2009 to significantly reduce its operating costs including a substantial reduction in personnel, closure of its laboratory, sale of equipment and reduction of leased space. Expense reduction and cash preservation activities continued during 2010 and 2011 and will continue into 2012.

The Company is seeking to maximize the value of its remaining assets. The Company is currently evaluating its strategic alternatives, which include the following:

- Pursue potential strategic transactions, which could include mergers, license agreements or other collaborations, with third parties;
- Sell or out-license the Company s remaining assets, including the Company s library of compounds; or
- Implement an orderly wind down of the Company if other alternatives are not deemed viable and in the best interests of the Company.

Subject to certain limitations, beginning January 1, 2012, and continuing until the Qualifying Transaction closes or until Amun exercises it s put right, $200,000 per month will be disbursed to us from the Escrow account. Based on our current plans, these disbursements should be sufficient to meet our monthly operating expenses and capital requirements until such funds are depleted. As of January 15, 2012, the balance of the Escrow account was approximately $2.28 million. Further, if the Qualifying Transaction is consummated, we believe that our capital resources, together with interest thereon, would be sufficient to meet our operating expenses and capital requirements through 2013. If the Escrow account is not available to us, based upon our current plans, we believe that our unrestricted cash and cash equivalents, and interest thereon, would be sufficient to meet our operating expenses and capital expenditures for several months

2. Summary of Accounting Policies

Cash Equivalents

The Company considers any liquid investments with maturity of three months or less when purchased to be cash equivalents. At December 31, 2011 the Company s unrestricted cash equivalents are approximately $1.5 million and are deposited primarily in a checking account with a financial institution.

Harbor BioSciences, Inc.

(A Development Stage Company)

Notes to Financial Statements

Property and Equipment

Property and equipment are stated at cost net of accumulated depreciation. The Company provides for depreciation using the straight-line method over the estimated useful lives of the assets. The cost of major additions and improvements is capitalized, while maintenance and repair costs that do not improve or extend the lives of the respective assets are charged to operations as incurred.

Property and equipment balances and corresponding lives were as follows:

	December 31		
	2011	**2010**	**Lives**
	(in thousands)		
Machinery, equipment and information systems	21	110	5-7 years
Equipment held for sale	0	21	
Furniture and fixtures	52	186	5-7 years
Total	73	317	
Less: Accumulated depreciation	(66)	(273)	
	$ 7	$ 44	

Depreciation expense associated with property and equipment was approximately $2000, $21,000 and $208,000 in 2011, 2010 and 2009, respectively.

In accordance with ASC Topic 360, Property, Plant and Equipment, the Company reviews long-lived assets for impairment whenever events or changes in circumstances indicate that the carrying amount of an asset may not be recoverable. Recoverability of assets to be held and used is measured by a comparison of the carrying amount of an asset to future undiscounted net cash flows expected to be generated by the asset. Recoverability measurement and estimating of undiscounted cash flows is done at the lowest possible levels for which there are identifiable assets. If such assets are considered to be impaired, the impairment to be recognized is measured by the amount by which the carrying amount of assets exceeds the fair value of the assets. Assets to be disposed of are reported at the lower of the carrying amount or fair value less costs to sell. The Company had no impairments in 2011, 2010 and 2009.

Accrued Expenses

Accrued expenses include approximately $0.2 million and $0.3 million in accrued vacation expense, $0.1 million and $0.7 million in other research and development / general and administrative expenses as of December 31, 2011 and 2010, respectively.

Revenue Recognition

In December 2003, the Securities and Exchange Commission (SEC) issued ASC Topic 605, Revenue Recognition, which updates and summarizes the Commission s views on the application of generally accepted accounting principles to revenue recognition in financial statements. The Company believes that its revenue recognition policies conform to the requirements of ASC Topic 605.

Contract revenue is recognized as the services are performed on a cost reimbursement basis. Revenue associated with development milestones, if any, is recognized based upon the achievement of the milestones, as defined in the respective agreements. Overall, revenue is considered to be realized or realizable and earned when there is persuasive evidence of a revenue arrangement in the form of a contract or purchase order, the services have been performed, the price is fixed or determinable and collectability is reasonably assured.

Research and Development

All research and development costs are expensed as incurred. The value of acquired in-process research and development is charged to expense on the date of acquisition. Research and development expenses include, but are not

Harbor BioSciences, Inc.

(A Development Stage Company)

Notes to Financial Statements

limited to, acquired in-process technology deemed to have no alternative future use, license fees related to license agreements, preclinical and clinical trial studies, payroll and personnel expense, lab supplies, consulting and research-related overhead. Research and development expenses paid in the form of cash and Company stock to related parties aggregated $11.5 million for the period from inception (August 15, 1994) to December 31, 2003 (see Note 6, Colthurst, Edenland and Mr. Prendergest and Aeson Therapeutics). No such related party expenses were incurred in 2011, 2010 or 2009.

Accounting for Share-Based Payments

The Company has an equity-based incentive compensation plan known as The 2005 Equity Incentive Plan (the Plan). The Plan allows us to grant stock options and other stock or stock-based awards, including stock appreciation rights, stock purchase awards, restricted stock awards and restricted stock units awards. The Plan also allows us to provide equity compensation to non-employee directors and consultants. The exercise price for an option granted under the Plan is typically not less than the fair market value of the common stock subject to such option. The term of any options granted under the Plan may not exceed 10 years from the date of the grant. Options issued to employees generally vest over a four-year period, with 25% vesting on the first anniversary date and the balance vesting monthly during years two, three and four.

Prior to (ASC Topic 718), Compensation-Stock Compensation, all stock options for employees (with the exception of three grants) have been granted at or above the market price where the exercise price of the option equaled or exceeded the market price of the stock on the date of the grant. As a result, under previous rules, there was no stock-based compensation expense for those grants. Compensation expense was taken for the three options granted at below market value (see 2005 Annual Report on Form 10-K, Notes to Financial Statements *No. 9 Stock Options* for more detail). As of December 31, 2011 the Plans have 7,066,940 shares of common stock reserved for issuance.

Effective January 1, 2006, we adopted ASC Topic 718, requiring us to recognize expense related to the fair value of our stock-based compensation awards. We elected the modified prospective transition method as permitted by ASC Topic 718; accordingly, results from prior periods have not been restated. Under this transition method, stock-based compensation expense for the fiscal year ended December 31, 2011, 2010 and 2009 includes:

a) compensation expense for all stock-based compensation awards granted prior to January 1, 2006 but not yet vested, based on the grant date fair value estimated in accordance with the original provisions of ASC Topic 718, and

b) compensation expense for all stock-based compensation awards granted subsequent to December 31, 2005 based on the grant-date fair value estimated in accordance with the provisions of ASC Topic 718.

The fair value of each stock option granted is estimated on the date of grant using the Black-Scholes option-pricing model. The assumptions used to calculate the fair value of options granted are evaluated and revised, as necessary, to reflect the Company s experience. Compensation expense is recognized using the straight-line method for all stock-based awards issued after January 1, 2006. Compensation expense is recognized only for those options expected to vest, with forfeitures estimated at the date of grant based on the Company s historical experience and future expectations. Prior to the adoption of ASC Topic 718, the effect of forfeitures on the pro forma expense amounts was not recognized. ASC Topic 718, requires forfeitures to be estimated at the time of the grant and revised as necessary in subsequent periods if actual forfeitures differ from those estimates.

Black-Scholes Option Valuation Assumptions (1)

	Fiscal Years Ended		
	December 31, 2011 (4)	December 31, 2010	December 31, 2009
Risk-free interest rate	n/a	4.35%	4.5%
Expected dividend yield	n/a	0%	0%
Expected life (2)	n/a	6.32 years	7.04 years

Expected volatility (3)	n/a	88%	85%

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(1) Forfeitures are estimated as 7.78% for 2010 and 7.88% for 2009 for stock options.
(2) The 2010 and 2009 expected life is based on historical experience
(3) The expected stock price volatility is estimated based on historical experience.
(4) There were no stock options granted during 2011.

The Black-Scholes option valuation model was developed for use in estimating the fair value of traded options that have no vesting restrictions and are fully transferable. In addition, option valuation models require the input of highly subjective assumptions including the expected stock price volatility. Because the Company s employee stock options have characteristics significantly different from those of traded options, and because changes in subjective input assumptions can materially affect the fair value estimate, in management s opinion, the existing models do not necessarily provide a reliable single measure of the fair value of the Company s options.

401(k) Matching Contributions

Our Company sponsored a 401(k) savings plan, to which eligible domestic employees may voluntarily contribute a portion of their income, subject to statutory limitations. In addition to the participant s own contributions to these 401(k) savings plans, we matched such contributions up to a designated level. Total matching contributions related to employee savings plans were approximately zero, $76,000 and $120,000 in 2011, 2010 and 2009, respectively. The 401(k) plan was terminated in 2011 and the assets disbursed according to the participants instructions.

Income Taxes

The Company provides for income taxes under the principles of ASC Topic 740, Income Taxes, which requires that provision be made for taxes currently due and for the expected future tax effects of temporary differences between book and tax bases of assets and liabilities.

On July 13, 2006 ASC Topic 740 was issued, Accounting for Uncertainty in Income Taxes, which establishes recognition and measurement thresholds that must be met before a tax benefit can be recognized in the financial statements. The Company adopted ASC Topic 740 on January 1, 2007, and it has had no material impact on its financial statements.

Financial Instruments

The Company s financial instruments consist primarily of cash and accounts payable. These financial instruments are stated at their respective carrying values, which approximate their fair values, due to their short-term nature.

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 the reported amounts of assets and liabilities and disclosure of contingent assets and liabilities at the date of the financial statements and the reported amounts of revenues and expenses during the reporting period. Actual results could materially differ from those estimates.

Concentrations of Risk

Financial instruments, which potentially expose the Company to concentrations of credit risk, consist primarily of cash and cash equivalents. The Company places its cash with high quality financial institutions.

Cash and cash equivalents are maintained at financial institutions and, at times, balances may exceed federally insured limits. We have never experienced any losses related to these balances. All of our non-interest bearing cash balances were fully insured at December 31, 2011 due to a temporary federal program in effect from December 31, 2010 through December 31, 2012. Under the program, there is no limit to the amount of insurance for eligible accounts. Beginning 2013, insurance coverage will revert to $250,000 per depositor at each financial institution, and our

non-interest bearing cash balances may again exceed federally insured limits. At December 31, 2011 the Company had no interest-bearing amounts on deposit in excess of federally insured limits.

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Net Loss per Share

Basic net loss per share is computed by dividing net loss by the weighted average number of common shares outstanding during the period. Diluted net loss per share is computed in a manner consistent with basic net loss per share after giving effect to potentially dilutive securities. Potential common shares of 3,332,168, 7,379,164 and 5,010,334 related to the Company s outstanding stock option and warrants were excluded from the computation of diluted net loss per share for the years ended December 31, 2011, 2010 and 2009 because their effect on net loss per share is anti-dilutive.

3. Recapitalization

During March 1997, Hollis-Eden Inc. was merged (the Merger) with and into the Company (then known as Initial Acquisition Corp. (IAC)). Upon consummation of the Merger, Hollis-Eden Inc. ceased to exist, and IAC changed its name to Hollis-Eden Pharmaceuticals, Inc. IAC (now called Hollis-Eden Pharmaceuticals, Inc.) was the continuing legal entity and registrant for SEC reporting purposes. The IAC Merger was accounted for as a recapitalization of Hollis-Eden Inc. by an exchange of Common Stock of Hollis-Eden Inc., for the net assets of IAC, consisting primarily of $6.5 million in cash and other receivables.

Under the terms of the merger agreement, each share of Hollis-Eden Inc. Common Stock outstanding converted into one share of Common Stock of Hollis-Eden Pharmaceuticals, Inc. Common Stock (Company Common Stock), and all warrants and options to purchase Hollis-Eden Inc. Common Stock outstanding converted into the right to receive the same number of shares of Company Common Stock.

Upon the consummation of the Merger, pursuant to an agreement, the Company issued warrants to purchase an aggregate of 50,000 shares of Company Common Stock at an exercise price of $0.10 per share to a director and former officer. Additional paid-in capital was increased by $570,000 with an offsetting $570,000 charge recorded to operations during the three months ended March 31, 1997.

Effective February 9, 2010, we changed our name from Hollis-Eden Pharmaceuticals, Inc. to Harbor BioSciences, Inc. The name change was effected pursuant to Section 253 of the General Corporation Law of the State of Delaware by the merger of our wholly owned subsidiary into us. We were the surviving corporation and, in connection with the merger, we amended our Amended and Restated Certificate of Incorporation to change our name to Harbor BioSciences, Inc.

Effective February 18, 2010, our common stock began trading under a new NASDAQ symbol, HRBR and CUSIP number 41150V 103 . Our common stock was delisted from NASDAQ in September, 2010, at which time the stock became available for trading on the OTCBB under the symbol HRBR.OB. On August 17, 2011, our common stock was delisted from the OTCBB as a result of our filing a Form 15 pursuant to Rule 12g-4(a)(i), and subsequently became available for trading on the Pink Sheets® with the symbol HRBR.PK and CUSIP number 41150V 202 .

4. Other Receivable from Related Party

On April 23, 2001, the Company entered into a promissory note with a stockholder/officer in the amount of $16,875. Interest was at 4.5% per annum. The promissory note was paid in full prior to the due date of April 23, 2004.

On May 22, 1998, the Company entered into a promissory note with a stockholder/officer in the amount of $200,000. Interest was at 5.5% per annum. The note was repaid in full in May 2003.

On March 21, 2005, the Company entered into a promissory note with an employee with a maximum loan amount of $20,000. Interest was at 6% per annum. The first installment of $10,000 was made on the commencement date. A second installment of $10,000 was made on April 20, 2005. The loan was repaid with a balance of approximately $2,000 forgiven on May 10, 2007.

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5. Income Taxes

The Company has available a federal and state net operating loss carryforward of approximately $215.3 million and $155.8 million at December 31, 2011, respectively, which may be carried forward as an offset to taxable income, if any, in future years through 2031 for both federal and state purposes. The Company has research and development credit carryforwards of approximately $11.1 million and $8.5 million, respectively, for federal and state at December 31, 2011. The Company has a net federal and state deferred tax asset of approximately $86.8 million and $17.7 million at December 31, 2011, respectively, mainly comprised of research and development credits, the net operating loss carryforwards, and capitalized start-up cost, partially offset by IRC section 481(a) adjustment recognizable through 2014. The net deferred tax assets have been fully reserved due to the uncertainty of the Company being able to generate taxable income under the more likely than not criteria of ASC Topic 740.

The difference between the Company s expected Federal tax benefit calculated using a 35% tax rate and the Company s zero effective tax rate is primarily related to a full valuation allowance established against the Company s net deferred tax assets.

If certain substantial changes in the Company s ownership should occur, there would potentially be an annual limitation on the amount of the carryforwards, which could be utilized in a tax year. The Company performed a Section 382 change in control test as of July 2011 and determined that there was an ownership change, as defined under IRS section 382, in March 1997. However, the section 382 annual limitation for the ownership change occurred in March 1997 didn t have a material impact to the Company s ability to use its net operating losses and research and development credit carryforwards during the carryover periods.

On January 1, 2006, the Company adopted the provision of ASC 740, which clarifies the accounting for uncertain tax positions. The provision requires that the Company recognize the impact of a tax position in our financial statement if the position is more likely than not to be sustained upon examination and on the technical merits of the position. The adoption of this provision had no material impact to the Company s financial statement due to its full valuation allowance position. The Company identified in prior year an uncertain tax position with respect to its treatment of certain cost subject to IRC section 195. However, the Company filed a request for change in accounting method in 2011 and the related unrecognized tax benefits were recognized in 2011. As of December 31, 2011, the Company had no material unrecognized tax benefits.

A reconciliation of the beginning and ending balance of unrecognized tax benefits is as follows:

Balance at December 31, 2010	$ 3,021,000
Gross increase	0
Gross decrease	3,021,000
Balance at December 31, 2011	$ 0

The Company does not anticipate any material change in the total amount of unrecognized tax benefits will occur within the next twelve months.

The Company s practice is to recognize interest and/or penalties related to income tax matters in income taxes expense. The Company had no accrual for interest or penalties on the Company s balance sheets at December 31, 2011, and had not recognized interest and/or penalties in the statement of operations for the period ended December 31, 2011.

The Company is subject to examination for tax years after 2007 for federal purposes and after 2006 for California state tax purposes.

6. Related Party Licenses and other Agreements and Contingencies

Colthurst, Edenland and Mr. Prendergast

During 1994, the Company entered into two license agreements and one research, development and option agreement as discussed in the following paragraphs.

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Pursuant to a license agreement dated May 18, 1994 (Colthurst License Agreement) with related parties, Patrick T. Prendergast, a significant stockholder at the time, and with Colthurst Limited, a company controlled by Mr. Prendergast, the Company acquired the exclusive worldwide rights to Mr. Prendergast s patent rights, know-how and background technology relating to the treatment of human/animal immunodeficiency. The agreement was amended on August 11, 1995 to change the license fee payment terms as discussed below in paragraph four of this Note. Per the license agreement, the Company agreed to pay royalties on product revenues.

On August 25, 1994, the Company entered into a license agreement (Edenland License Agreement) with a related party, Edenland Inc., a company controlled by Mr. Prendergast, for the exclusive worldwide rights to Mr. Prendergast s patent rights, know-how and background technology related to the substance tradenamed HE317 and to any other pharmaceutical product that became subject to the license agreement under the research, development and option agreement discussed below. The agreement was amended on August 11, 1995 to change the license fee payment terms as discussed in the following paragraph. Per the Edenland License Agreement, the Company agreed to pay royalties on product revenues.

Effective August 11, 1995, Edenland, Inc., Colthurst Limited and the Company entered into amendments concerning the license fee payment terms to the two agreements described above. Under this amendment, the Company agreed to pay a license fee by April 28, 1996 plus additional license fees within 24 months of April 1996. The balances of these fees were paid in full by May 1997. As consideration for entering into certain amendments, the Company issued 75,472 shares of the Company s common stock to Edenland, Inc. and Colthurst Limited.

Per the amended Colthurst License Agreement, a renewal annual license fee was payable commencing May 1998. The Company paid this fee in 1998 by issuing shares of its common stock and, in 1999, paid in cash.

In August 1994, the Company entered into a Research, Development and Option Agreement, with Edenland, Inc. and Mr. Prendergast. The agreement provided for the development of HE317 to a certain stage of development and granted the Company the right of first option on new products developed by Edenland, Inc. The agreement committed the Company to pay for certain development costs up to the amount of $3.0 million with certain contingencies for funding. In October 1996, the Company and Edenland, Inc. entered into an amendment, which accelerated the date that the $3.0 million payment for HE317 or other product development costs was to be made. The Company paid $2.7 million during 1997 and the remaining $300,000 in April 1998.

On January 20, 2000, the Company reached a settlement regarding various disputes with Mr. Prendergast, Colthurst and Edenland. The parties entered into two new technology agreements, the Technology Assignment Agreement and the Sponsored Research and License Agreement.

The Technology Assignment Agreement (Assignment Agreement) replaced the Colthurst License Agreement. Pursuant to the Assignment Agreement, Mr. Prendergast and Colthurst assigned to the Company ownership of all patents, patent applications and current or future improvements of the technology under the Colthurst License Agreement, including HE2000, the Company s lead clinical compound at the time. The annual license fee of $500,000 and the royalty obligations under the Colthurst License Agreement were eliminated. In consideration for the foregoing, the Company agreed to issue to Colthurst 660,000 shares of Common Stock and a warrant to purchase an aggregate of 400,000 shares of Common Stock at $25 per share. Only 132,000 of such shares of Common Stock were issued in 2000, with the remaining 528,000 shares to be issued over the next four years conditioned on continued compliance with the agreement and, in particular, satisfaction of the Conditions (as defined below). In addition, all of the shares under the warrant vest over four years conditioned on continued compliance with the agreement and, in particular, satisfaction of the Conditions (as defined below). The Sponsored Research and License Agreement replaced the Edenland License Agreement and the Research, Development and Option Agreement. Pursuant to the Sponsored Research and License Agreement, Edenland exclusively licensed to the Company a number of compounds, together with all related patents and patent applications, and the Company funded additional preclinical research projects conducted by Edenland. The Company would also have exclusive license rights to all results of such research and would have royalty obligations to Edenland on sales of new products, if any, resulting from such research. None of the compounds licensed under the Sponsored Research and License Agreement have been developed by the Company and, as described below, this agreement is now terminated.

As stated above, the issuance of the additional shares of Common Stock and the vesting of the warrant was dependent upon the satisfaction of certain conditions (the Conditions). In accordance with ASC Subtopic 505-50 these

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future events could not be determined at the date of the agreements (January 2000). Accordingly, the shares and warrants are accounted for as they vest or are issued. During 2000, the Company recorded a research and development charge for $1.9 million representing the fair value of the 132,000 shares issued under the Assignment Agreement.

Because all of the Conditions were not satisfied, the Company did not issue any additional shares to Colthurst and believed it had no obligation to issue any additional shares and that the warrant would not vest as to any shares of Common Stock.

After arbitration proceedings during 2004 and 2005, pursuant to which Colthurst sought more than $25 million in damages for the non-issuance of the 528,000 shares of common stock and the warrant to purchase up to 400,000 shares of common stock, in February 2006 the parties agreed to a settlement and release of all issues in dispute between the parties. Under the settlement agreement, (1) the Company agreed to make a payment of $3 million in cash and (2) the parties agreed to terminate the Sponsored Research and License Agreement between the Company and Edenland Inc. The $3.0 million was accrued as an expense as of December 31, 2005. Under the settlement agreement, the Colthurst parties remain prohibited from conducting any further research, development or commercialization activities of any kind relating in any way to the technology (including HE2000) that was assigned to the Company under the Assignment Agreement.

Aeson Therapeutics

In October 2000, the Company acquired a 21% equity stake in Aeson Therapeutics Inc. (Aeson) and an exclusive worldwide sublicense to three issued patents in the area of adrenal steroids in exchange for $2.0 million in cash and 208,672 shares of Common Stock valued at $2 million. The cash and shares were expensed as in-process R&D during the fourth quarter of 2000. As part of the transaction, Aeson and its stockholders granted the Company an exclusive option to acquire the remainder of Aeson at a predetermined price.

In March 2002, the Company amended certain of its agreements with Aeson. Under the amendments, the Company paid Aeson $1.2 million, which extended the initial date by which the Company could exercise its option to acquire the remainder of Aeson to September 30, 2002. The Company also received additional equity securities as a result of its $1.2 million payment. The $1.2 million payment was expensed as in-process R&D. The Company elected not to exercise the option to acquire the remainder of Aeson by September 30, 2002.

On June 7, 2006, the Company acquired substantially all of the assets of Aeson. As consideration for Aeson s assets, the Company agreed (i) to issue a total of 35,000 shares of common stock to Aeson at the closing of the acquisition and (ii) to issue to Aeson s stockholders up to a total of 165,000 additional shares of common stock if certain development milestones are achieved. The acquisition was expensed as in-process research and development. The Company has not achieved any of the development milestones.

Pharmadigm

In August 2002, we entered into a Sublicense Agreement with Pharmadigm, Inc. (currently known as Inflabloc Pharmaceuticals, Inc.). Under the agreement, we obtained exclusive worldwide rights to certain intellectual property of Pharmadigm and the University of Utah and we agreed to make aggregate payments of $0.9 million in cash or in shares of our common stock, at our option, over the next year. This cost was expensed in the third quarter of 2002. We elected to make such payments in equity and have issued a total of 168,921 shares of our common stock in complete satisfaction of this requirement (of which 118,921 shares were issued the quarter ended March 31, 2003). We may also make substantial additional milestone and royalty payments in cash to Pharmadigm if we meet specified development and commercialization milestones for products covered by the patents. To date, no such milestones have been met. The principal patents licensed under the agreement, originally licensed to Pharmadigm from the University of Utah, relate to inventions by Dr. Raymond Daynes and Dr. Barbara A. Areneo. Dr. Daynes served as a scientific consultant to the Company from 1999 to mid-2003.

Congressional Pharmaceutical

In February 2004, the Company acquired Congressional Pharmaceutical Corporation (CPC) and replaced CPC as the exclusive licensee to certain intellectual property rights held by the University of Chicago. These intellectual property rights consist of a series of patents and patent applications that relate to discoveries made by David J. Grdina, Ph.D.,

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Professor of Radiation and Cellular Oncology at the University of Chicago. The patented technology covers a series of compounds that have the potential to protect against DNA mutations that can occur as a result of radiation injury or chemotherapy. In the acquisition the Company issued approximately 50,000 shares of common stock to the former stockholders of CPC valued at approximately $650,000, in accordance with Emerging Issues Task Force No. 99-12. In addition, if the Company achieves certain development milestones, it will be required to issue additional shares of our common stock to the former stockholders of CPC. In the event all of the milestones are achieved, the total number of additional shares that the Company would be required to issue to the former stockholders of CPC is 275,000, more than half of which would be issued only upon FDA approval of CPC s product. No such milestone has been met to date. Furthermore, upon regulatory approval and commercialization of products covered by the licensed intellectual property, the Company may be required to pay royalties to the former stockholders of CPC and the University of Chicago. Following the acquisition, Dr. Grdina agreed to an exclusive consulting arrangement with the Company in the fields of hematopoiesis and radiation and chemotherapy exposure. In March 2007, the Company terminated its consulting agreement with Dr. Grdina. In May 2007, the University of Chicago terminated the license agreement with the Company.

AFRRI Collaboration

The Company performed work on two task orders that were issued under collaboration with the Armed Forces Radiobiology Research Institute (AFRRI). Under these task orders, the Company conducted radiation studies with a subcontractor. The task orders committed AFRRI to reimburse the Company for $2.0 million in subcontractor fees. The reimbursement amounts from AFRRI were recorded in the same timeline as the subcontractor fees, resulting in no impact on the statement of operations. There was no activity during 2007 under the AFRRI collaboration. The Company terminated its collaborative research and development agreement with AFRRI effective August 12, 2007.

Study Funding Agreement

The Company has a Study Funding Agreement with Cystic Fibrosis Foundation Therapeutics, Inc. (CFFT). The agreement commits CFFT to provide a total of $1.7 million to be paid in seven tranches based on the Company s completion of certain agreed-upon events. The agreement also contains a provision indicating that upon termination of this agreement by either party, CFFT shall pay the Company for all work performed through the date of termination, plus reasonable costs of bringing the study to an orderly close.

In return for this funding, the Company has agreed to pay CFFT a minimum royalty over a specified period following regulatory approval in the United States of America. Additional compensation is due to CFFT if net sales of this compound exceed a specified amount over a period of time.

Revenue is recognized under this agreement on a percentage of completion method for each distinct agreed-upon event.

This agreement expired December 31, 2009.

Agreements China State Institute of Pharmaceutical Industry Agreements

In January 2011, the Company announced that it had licensed the research and development and commercialization rights for three of its products, exclusively in the People s Republic of China and Hong Kong, to China State Institute of Pharmaceutical Industry (CIPI). Harbor BioSciences retains the rights to these products in the U.S. and the rest of the world, and CIPI will make available to the company all pre-clinical and clinical data it generates.

CIPI was recently formed by a merger of the Shanghai Institute of Pharmaceutical Industry and other institutes and companies. CIPI s R&D focus has been in the areas of cancer, infectious diseases, cardiovascular, autoimmune disorders, endocrinology and CNS. CIPI is a subsidiary of the China National Pharmaceutical Group Corporation (Sinopharm Group), China s largest pharmaceutical and health industrial group under the state-owned Assets Supervision and Administration Commission of the State Council. Sinopharm Group s core businesses include R&D, manufacturing, distribution and retail sales. Its products are manufactured in more than 10 pharmaceutical and biological production facilities. Sinopharm Group has more than 20 joint ventures with global pharmaceutical companies and through trade and cooperative relations, has a presence in more than 100 countries and regions. Sinopharm Group reported 2010 revenues of approximately $12 billion U.S.

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CIPI is a major supplier of both generic drugs and traditional Chinese medicines in China and Hong Kong. The three license agreements cover Harbor BioSciences compounds HE2000, Apoptone and Triolex for any clinical use in the People s Republic of China and Hong Kong. CIPI plans to develop the Harbor BioSciences compounds for major indications including diabetes, cancer, inflammation and infectious diseases

The Company believes these are the first drug development agreements between a western pioneer drug company and a government-owned Chinese drug developer for pharmaceutical development to be conducted in the People s Republic of China. CIPI, a low cost drug manufacturer, has agreed to supply the licensed products to Harbor BioSciences for use in clinical studies and sales outside of China and Hong Kong. The Company can also elect to distribute these compounds in countries that accept the State Food and Drug Administration s (SFDA) drug approval process.

Clinical drug development candidates licensed to CIPI include Triolex, which has completed Phase IIa clinical trials in patients with Type 2 diabetes and is in early stage development for ulcerative colitis and rheumatoid arthritis; Apoptone, which has demonstrated activity in Phase I/IIa trials of prostate cancer; and HE2000, which has shown to limit opportunistic infections, including tuberculosis, in humans infected with the HIV-1 virus, to reduce parasite levels in patients with uncomplicated malaria and to attenuate non-productive lung inflammation in animal models.

The Company will receive milestone payments for Triolex, Apoptone and HE2000, excluding infectious diseases, at the completion of Phase II and III clinical studies and upon approval by the SFDA. The Company will also receive royalties based on net profits for the life of each agreement. The term of each agreement runs until the latter of (1) the expiration of the last licensed patent or any Company, CIPI or joint improvement patent and (2) the first documented third party sale of a competing generic product in the licensed territory. In addition, the Company is CIPI s sole agent with commercial development and sales rights to all of CIPI s improvements that are sold outside the licensed territory. Sales of licensed drugs that are covered by CIPI s improvements outside the territory bear a royalty to Harbor BioSciences.

7. Common Stock

Reverse Stock Splits

During February 1995, there was a 3-for-5 reverse stock split of the Company s common stock and in March 1996, a 1-for-2.65 reverse stock split of the Company s common stock. Both reverse stock splits have been retroactively reflected for all periods presented.

Reverse/Forward Stock Splits

During October 2011, there was a 1-to-1,000 reverse stock split immediately followed by a 1,000-to-1 forward stock split. A total of 43,698 shares were cancelled. Stockholders whose shares were cancelled received a cash payment of $0.142 per share. The Company paid $6,205 in total for the cancelled shares.

Common Stock Financings

In January 1996, the Company completed a $367,522 round of debt financing with a group of private investors. These notes, with an 8% interest rate, were due on or before the earlier of (i) January 21, 1997 or (ii) the closing of a private or public offering of securities. During April 1996, the debt financing plus accrued interest was converted into 164,962 shares of common stock at a price of $2.25 per share. In April 1996, the Company privately issued 580,005 shares of the Company s common stock at an offering price of $2.25 per share. Total proceeds from this offering aggregated $1,234,499.

During May 1998, the Company completed a private financing totaling $20.6 million in gross proceeds. The Company issued 1,329,201 shares of common stock, (of which 192,061 shares were subject to adjustment based on future average stock price (Adjustable Common Stock)), 4,000 shares of 5% Series A Convertible Preferred Stock and warrants to purchase 1,437,475 shares of common stock in the financing. The warrants entitled the holders to purchase up to a total of 1,437,475 shares of common stock at a price of $17.00 per share.

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The Convertible Preferred Stock had an initial conversion price of $20.30 for the first seven months, after which it could be adjusted, either up or down, based on the future stock prices of the Company s common stock. The Convertible Preferred Stock was converted to common stock in January 1999 (See Note 8).

In January 1999, the Company completed two private placements of an aggregate of 1,367,868 shares of common stock at prices ranging from $18.00 to $18.50 per share. In connection with the private placements, the Company issued warrants to purchase an aggregate of 90,000 shares of the Company s common stock, with an exercise price of $18.25 per share, as a finder s fee. The Company raised approximately $25.0 million in gross proceeds.

During December 2001, the Company raised $11.5 million in gross proceeds from the sale of 1.28 million shares of newly issued common stock in a private placement at a price of $9.00 per share. The investors were a group of qualified institutional buyers and institutional accredited investors. The Company also issued warrants to purchase up to 128,000 shares of common stock having an exercise price of $12.00 per share to investors. As a finders fee, the Company issued to its placement agent two warrants for a total of 112,640 shares of common stock, one warrant with an exercise price of $9.00 and the other with an exercise price of $12.00.

On February 25, 2003, we completed a private placement in which we issued $10.0 million aggregate principal amount of three-year convertible debentures (debentures), bearing interest at 7.5% per year, and warrants to purchase up to 701,760 shares of common stock. The debentures were convertible into common stock at a price of $5.70 per share, which represented a discount from the price of our common stock on the commencement date. Also issued in connection with this private placement were warrants to purchase up to 350,880 shares of common stock which are exercisable at a price per share of $6.17, subject to adjustment, and warrants to purchase up to 350,880 shares of common stock which are exercisable at a price per share of $6.71, subject to adjustment. The warrants were exercisable until February 25, 2007.

In connection with the issuance of the debentures and warrants, we recorded approximately $3.5 million related to the beneficial conversion feature and approximately $3.0 million for the detachable warrants on the debentures. The total amount of the deemed discount on the debentures as a result of the warrant issuance and the beneficial conversion feature amounts to $6.5 million. The beneficial conversion feature and warrant value (deemed discount) were amortized over the term of the debentures and as conversion of the debentures occurred.

On June 20, 2003, convertible debentures with a face value of $0.5 million were converted into 87,720 shares of our common stock leaving a $9.5 million aggregate principle amount of convertible debentures outstanding. On August 11, 2003, the remaining aggregate principal amount of convertible debentures with a face value of $9.5 million was converted into 1,666,680 shares of our common stock with a value of $5.70 per share.

During June 2003, the Company completed a private placement of common stock and warrants, from which it received gross proceeds of $14.7 million. In October 2003 the Company completed a public offering of an aggregate of 2,500,000 shares of common stock at a price of $25.00 per share and received $62.5 million in gross proceeds from this offering.

On June 1, 2005 the Company raised approximately $10.0 million in gross proceeds from the sale of 1,333,333 shares of the Company s common stock at an exercise price of $6.17 per share. Additionally, the Company issued a four-year warrant to purchase up to an additional 266,667 shares of common stock at an exercise price of $10.00 per share. In connection with this transaction, the Company incurred approximately $0.5 million in direct costs and recorded net proceeds of approximately $9.5 million.

On February 6, 2006 the Company raised approximately $26.0 million in gross proceeds from the sale of 4,000,000 shares of the Company s common stock at a price of $6.50 per share. The direct costs related to this financing were $1.7 million, resulting in net proceeds of $24.3 million. Additionally, the Company issued four-year warrants to purchase up to an additional 800,000 shares of common stock at an exercise price of $8.75 per share. The warrants were not exercisable until six months following issuance.

On June 7, 2006 the Company issued 35,000 shares of the Company s common stock to Aeson Therapeutics, Inc. (Aeson) in connection with the purchase of substantially all of Aeson s assets, resulting in an expense of $180,000. Upon certain events, the Company may be obligated to issue an additional 165,000 shares. The acquisition was expensed as in-process research and development. To date, the Company has not achieved any of the development milestones.

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On November 13, 2006 the Company raised approximately $26.0 million in gross proceeds from the sale of 4,000,000 shares of the Company s common stock at a price of $6.50 per share. The direct costs related to this financing were $1.6 million, resulting in net proceeds of $24.4 million. Additionally, the Company issued four-year warrants to purchase up to an additional 800,000 shares of common stock at an exercise price of $8.75 per share. The warrants were not exercisable until six months following issuance.

On June 10, 2010 the Company raised approximately $2.06 million in gross proceeds from the sale of approximately 5.9 million shares of its common stock and warrants to purchase approximately 3.5 million shares of its common stock. The shares of common stock and warrants to purchase common stock were sold in units, with each unit consisting of one share of common stock and a warrant to purchase 0.6 of a share of common stock. The purchase price per unit is $0.35.

8. Preferred Stock

During May 1998, as part of a private placement, the Company issued 4,000 shares of Convertible Preferred Stock for proceeds of $4.0 million. The proceeds of the offering are included in the proceeds to the May 1998 financing described in Note 7, above.

During January 1999, the Company issued 346,127 shares of common stock in connection with the conversion of the Series A Convertible Preferred Stock and additional shares relating to the Adjustable Common Stock. The Adjustable Common Stock was issued during the private placement of May 1998 and was subject to adjustment based on the future average stock price of the Company s common stock as described in Note 7. Upon conversion, all outstanding shares of Preferred stock and Adjustable Common stock were eliminated.

In November 1999, the Company adopted a Shareholders Rights Plan in which Preferred Stock purchase rights (Rights) were distributed as a dividend at the rate of one Right for each share of common stock held as of the close of business on November 29, 1999. Each right entitled stockholders to buy, upon certain events, one one-hundredth of a share of a new Series B junior participating preferred stock of the Company at an exercise price of $100.00. The Rights are designed to guard against partial tender offers and other abusive tactics that might be used in an attempt to gain control of the Company or to deprive stockholders of their interest in the long-term value of the Company. The Rights were exercisable only if a person or group acquires 15% or more of the Company s common stock or announces a tender offer of which the consummation would result in ownership by a person or group of 15% or more of the Company s common stock. The Rights were redeemable for one cent per Right at the option of the Board of Directors prior to this event occurring. The Rights expired on November 14, 2009.

Effective October 19, 2009, the Company executed an Amended and Restated Rights Agreement (the Rights Agreement) between the Company and American Stock Transfer and Trust Company, LLC, as Rights Agent, amending and restating the Rights Agreement dated as of November 15, 1999 (the Original Rights Agreement). The purposes of this amendment of the Original Agreement include, among other things: to extend the expiration date of the Preferred Stock purchase rights issued from November 14, 2009 to November 14, 2019; to change how many new shares of common stock the Rights holders can purchase at a price of $100 per Right (the Purchase Right) after the 15% threshold is crossed from two times the number of the Company s common stock that the Purchase Price is worth to five times the number of Company s common stock that the Purchase Price is worth; to decrease the redemption price for Company-initiated redemption of the Rights from $0.01 to $0.0001.

On July 28, 2011, we sold an aggregate of 2,000,000 shares of our Series A Preferred Stock (the Redeemable Preferred Shares) to Amun, LLC, a Delaware limited liability company (the Investor) pursuant to the terms of a Stock Purchase Agreement (the Purchase Agreement) and related Stockholders Agreement (the Stockholders Agreement). The Redeemable Preferred Shares represent approximately a 28% of the economic interest in the Company and also entitle the Investor to a number of votes equal to 38.28% of the total number of votes entitled to be cast by holders of all shares of the Company s capital stock (including the Common Stock and Series A Preferred Stock) voting together as single class. Under the terms of these and other related agreements between the Company and the Investor, the Investor placed $2.825 million in

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cash into an escrow account, which amount is available under certain circumstances to pay certain Company related expenses and to fund the Company s working capital needs. Amounts received are included as restricted cash as of December 31, 2011. The Stockholder Agreement provides that the Investor will have the right to put the Redeemable Preferred Shares acquired pursuant to the Purchase Agreement back to the Company in return for the remaining cash held in escrow at the time of the put, upon the occurrence of certain events.

9. Stock Options and Restricted Stock

Stock options and restricted stock, which are converted into common stock if exercised, are not considered when calculating earnings per share as the effect is anti-dilutive.

Stock Options

1997 Stock Option Plan

The 1997 Stock Option Plan (the 1997 Option Plan) was approved by the Company s stockholders in 1997. Under the 1997 Option Plan, shares of common stock have been reserved for issuance to employees, officers, directors, and consultants of the Company and provides for the grant of incentive and nonstatutory stock options. The Board of Directors determines terms of the stock option agreements, including vesting requirements. The exercise price of incentive stock options must equal at least the fair market value on the date of grant. The options expire not later than ten years from the date of the grant and generally are exercisable ratably over a three-year or four-year period beginning one-year from the date of the grant.

2005 Equity Incentive Plan

In June 2005, the Company s stockholders approved an amendment and restatement of the 1997 Option Plan to become the 2005 Equity Incentive Plan (the 2005 Equity Plan). Options granted under the 1997 Option Plan prior to its amendment and restatement will continue to be subject to the terms and conditions set forth in the agreements evidencing such options and the terms of the 1997 Option Plan except that the Board may elect to extend one or more of the features of the 2005 Equity Plan to stock awards granted under the 1997 Option Plan. The approval of the 2005 Equity Plan in June 2005 increased the number of shares reserved for issuance beyond those reserved for issuance under the 1997 Option Plan by 350,000 shares for a total of 5,500,000 reserved shares. The 2005 Equity Plan will allow the Company greater flexibility in designing equity incentives, including stock appreciation rights, stock purchase awards, restricted stock awards and restricted stock unit awards. In December 2005, the Board of Directors amended the 2005 Equity Plan to reserve an additional 100,000 shares to be used only for the grant of stock awards to persons not previously employed by the Company, or following a bona fide period of non-employment, as an inducement material to those persons entering into employment with the Company with the meeting of the Rule 4350(i)(1)(A)(iv) of the NASDAQ Marketplace Rules, and to provide that any such inducement grants must be granted either by a majority of the Company s independent directors or a committee comprised of a majority of independent directors.

On March 18, 2006, the Board of Directors amended and restated the 2005 Equity Plan to reserve an additional 500,000 shares for issuance under the 2005 Equity Plan, which was subsequently approved by the Company s stockholders in June 2006.

On March 30, 2007 the Board of Directors amended and restated the 2005 Equity Plan to reserve an additional 1,500,000 shares for issuance, for a total of 7,500,000 reserved shares and 100,000 inducement shares. The amendment was approved by the Company s stockholders in June 2007. The approval of the amendment allows the Company to continue to grant stock options and other awards at levels determined appropriate by our Board of Directors.

On March 28, 2008, the Board of Directors amended and restated the 2005 Equity Plan to reserve an additional 800,000 shares for issuance under the 2005 Equity Plan, which was subsequently approved by the Company s stockholders in June 2008.

The following table summarizes stock option activity under the Plan and the 2005 Equity Plan for 1997 through 2011 (in thousands, except per share amounts):

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	Shares *In Thousands*	Price Per Share Range	Weighted Average
1997			
Granted	518	$ 6.75-8.70	$ 7.13
Outstanding, December 31, 1997	518	$ 6.75-8.70	$ 7.13
1998			
Granted	341	13.25-16.75	14.52
Forfeited	100	8.70	8.70
Outstanding, December 31, 1998	759	$ 6.75-16.75	$ 10.24
1999			
Granted	776	10.56-16.63	12.70
Forfeited	61	14.06-14.63	14.63
Outstanding, December 31, 1999	1,474	$ 6.75-16.75	$ 11.36
2000			
Granted	774	6.50-15.06	8.18
Exercised	1	6.75	6.75
Forfeited	24	6.75-15.13	14.22
Outstanding, December 31, 2000	2,223	$ 6.50-16.75	$ 10.22
2001			
Granted	170	3.53-11.84	6.13
Forfeited	65	5.09-16.63	13.31
Outstanding, December 31, 2001	2,328	$ 3.53-16.75	$ 9.80
2002			
Granted	696	5.15-10.10	9.48
Forfeited	55	5.13-13.13	8.17
Outstanding, December 31, 2002	2,969	$ 3.53-16.75	$ 10.98
2003			
Granted	943	2.25-17.83	6.59
Exercised	85	4.50-13.13	11.25
Forfeited	66	4.00-16.75	12.17
Outstanding, December 31, 2003	3,761	$ 2.25-17.83	$ 8.88

2004			
Granted	596	8.54-15.20	13.69
Exercised	4	3.53-5.29	3.75
Forfeited	46	10.56-17.83	13.66
Outstanding, December 31, 2004	4,307	$ 2.25-17.83	$ 9.50

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2005			
Granted	408	5.22-10.75	9.94
Exercised	13	3.53-6.68	5.67
Forfeited	56	5.29-10.47	8.06
Outstanding, December 31, 2005	4,646	$ 2.25-17.83	$ 9.57
2006			
Granted	965	4.43-7.08	5.67