ChemoCentryx, Inc.
Form S-1/A
February 06, 2012
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As filed with the Securities and Exchange Commission on February 6, 2012

Registration No. 333-177332

UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

Amendment No. 4

to

FORM S-1

REGISTRATION STATEMENT UNDER THE SECURITIES ACT OF 1933

CHEMOCENTRYX, INC.

(Exact name of Registrant as specified in its charter)

Delaware	**2834**	**94-3254365**
(State or other jurisdiction of incorporation or organization)	*(Primary Standard Industrial Classification Code Number)*	*(I.R.S. Employer Identification Number)*

850 Maude Avenue

Mountain View, CA 94043

(650) 210-2900

(Address, including zip code, and telephone number, including area code, of Registrant s principal executive offices)

Thomas J. Schall, Ph.D.

President and Chief Executive Officer

ChemoCentryx, Inc.

850 Maude Avenue Mountain View, CA 94043

(650) 210-2900

(Name, address, including zip code, and telephone number, including area code, of agent for service)

Copies to:

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Davis Polk & Wardwell LLP
1600 El Camino Real
Menlo Park, CA 94025
(650) 752-2000

Approximate date of commencement of proposed sale to the public:

As soon as practicable after the effective date of this Registration Statement.

If any of the securities being registered on this Form are to be offered on a delayed or continuous basis pursuant to Rule 415 under the Securities Act of 1933, check the following box. ¨

If this Form is filed to register additional securities for an offering pursuant to Rule 462(b) under the Securities Act, please check the following box and list the Securities Act registration statement number of the earlier effective registration statement for the same offering. ¨

If this Form is a post-effective amendment filed pursuant to Rule 462(c) under the Securities Act, check the following box and list the Securities Act registration statement number of the earlier effective registration statement for the same offering. ¨

If this Form is a post-effective amendment filed pursuant to Rule 462(d) under the Securities Act, check the following box and list the Securities Act registration statement number of the earlier effective registration statement for the same offering. ¨

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 the definitions of large accelerated filer, accelerated filer and smaller reporting company in Rule 12b-2 of the Exchange Act.

Large accelerated filer ¨	Accelerated filer ¨	Non-accelerated filer x (Do not check if a smaller reporting company)	Smaller reporting company ¨

The Registrant hereby amends this Registration Statement on such date or dates as may be necessary to delay its effective date until the Registrant shall file a further amendment which specifically states that this Registration Statement shall thereafter become effective in accordance with Section 8(a) of the Securities Act of 1933 or until the Registration Statement shall become effective on such date as the Commission, acting pursuant to said Section 8(a), may determine.

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The information in this preliminary prospectus is not complete and may be changed. These securities may not be sold until the registration statement filed with the Securities and Exchange Commission is effective. This preliminary prospectus is not an offer to sell these securities nor does it seek an offer to buy these securities in any jurisdiction where the offer or sale is not permitted.

Subject to completion, dated February 6, 2012

Prospectus

4,000,000 Shares

Common Stock

This is an initial public offering of common stock by ChemoCentryx, Inc. We are selling 4,000,000 shares of common stock. The estimated initial public offering price is between $14.00 and $16.00 per share.

We have applied for listing of our common stock on the Nasdaq Global Market under the symbol CCXI.

	Per share	Total
Initial public offering price	$	$
Underwriting discounts and commissions	$	$
Proceeds to ChemoCentryx, before expenses	$	$

We have granted the underwriters an option for a period of 30 days to purchase up to 600,000 additional shares of common stock to cover their over-allotment.

Glaxo Group Limited and Techne Corporation, two of our principal stockholders, have agreed to purchase $7.0 million and $5.0 million, respectively, of our common stock in separate private placements concurrent with the completion of this offering at a price per share equal to the initial public offering price. The sale of such shares of common stock will not be registered under the Securities Act of 1933, as amended.

Investing in our common stock involves risks. See Risk Factors beginning on page 11.

Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or determined if this prospectus is truthful or complete. Any representation to the contrary is a criminal offense.

The underwriters expect to deliver the shares of common stock on or about , 2012.

J.P. Morgan **Citigroup**

Cowen and Company

, 2012

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Neither we nor the underwriters have authorized anyone to provide you with any information other than that contained in this prospectus or in any free writing prospectus we may authorize to be delivered or made available to you. We take no responsibility for, and can provide no assurance as to the reliability of, any other information that others may give you. This prospectus may only be used where it is legal to offer and sell shares of our common stock. The information in this prospectus is accurate only as of the date of this prospectus, regardless of the time of delivery of this prospectus or any sale of shares of our common stock. Our business, financial condition, results of operations and prospects may have changed since that date. Neither we nor the underwriters are making an offer of these securities in any jurisdiction where the offer is not permitted.

Until , 2012 (25 days after the date of this prospectus), all dealers that buy, sell or trade shares of our common stock, whether or not participating in this offering, may be required to deliver a prospectus. This delivery requirement is in addition to the dealers obligation to deliver a prospectus when acting as underwriters and with respect to their unsold allotments or subscriptions.

For investors outside the United States: Neither we nor the underwriters have done anything that would permit this offering or possession or distribution of this prospectus in any jurisdiction where action for that purpose is required, other than in the United States. Persons outside the United States who come into possession of this prospectus must inform themselves about, and observe any restrictions relating to, the offering of the shares of common stock and the distribution of this prospectus outside the United States.

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PROSPECTUS SUMMARY

This summary does not contain all of the information you should consider before buying our common stock. You should read the entire prospectus carefully, especially the Risk Factors section beginning on page 11 and our financial statements and the related notes appearing at the end of this prospectus, before deciding to invest in our common stock.

Overview

ChemoCentryx is a biopharmaceutical company focused on discovering, developing and commercializing orally-administered therapeutics to treat autoimmune diseases, inflammatory disorders and cancer. Our approach has been to target the chemokine system, a network of molecules including chemokine ligands and their associated receptors, as well as related chemo-attractant receptors, all of which are known to drive inflammation. Chemokine ligands concentrate at the site of an inflammatory event, serving as signals that attract and guide inflammatory cells to the tissue, where, based on the chemokine ligand and receptor combination, a specific inflammatory response is initiated. In certain diseases, discrete chemokine receptors that play a specific role in the pathology of interest have been identified, and the therapeutic goal is to specifically inhibit that receptor to provide clinical benefit. Accordingly, each of our drug candidates is a small molecule designed to target a specific chemokine or chemo-attractant receptor, thereby blocking the inflammatory response driven by that particular chemokine while leaving the rest of the immune system unaffected. Using our pioneering insights and proprietary technologies designed to better understand the chemokine system, we believe that we have established the broadest pipeline of novel drugs targeting chemokine receptors. Our compounds are designed to be highly potent, selective to minimize the risk of off-target effects and orally-available for improved patient compliance. As small molecules, they are also easier and less costly to manufacture than protein therapeutics, or biologics.

We currently have four drug candidates in clinical development and expect to advance one additional drug candidate into clinical development in 2012. The following table summarizes the status of our drug candidates and preclinical programs:

Our drug candidates include: Traficet-EN (CCX282 or GSK 786), our most advanced drug candidate, currently in three pivotal Phase III clinical trials being conducted by our partner Glaxo Group Limited, or GSK,

an affiliate of GlaxoSmithKline, for the treatment of patients with moderate-to-severe Crohn s disease; CCX140, our lead independent drug candidate, which successfully completed a Phase II clinical trial in type 2 diabetics and is currently in two Phase II clinical trials in patients with diabetic nephropathy, a form of kidney disease; CCX354, which successfully completed a Phase II proof-of-concept clinical trial for the treatment of rheumatoid arthritis, or RA; CCX168, currently in a Phase II proof-of-concept clinical trial for the treatment of anti-neutrophil cytoplasmic antibody, or ANCA-associated vasculitis, or AAV; and CCX662, our independent drug candidate for the treatment of glioblastoma multiforme, or GBM, which is expected to enter a Phase I clinical trial in the second half of 2012. CCX140 and CCX662 are wholly owned and are being developed independently by us, while Traficet-EN, CCX354, and CCX168 are subject to our collaboration agreement with GSK. We are also advancing several additional independent drug candidates through preclinical development. In addition, our strategy has been to identify next generation compounds related to our drug candidates. All of our drug candidates, including those under our collaboration agreement with GSK, have been internally discovered.

Traficet-EN is intended to control the inflammatory response underlying inflammatory bowel disease, or IBD, by targeting the chemokine receptor known as CCR9. In adults, CCR9 is found primarily on a population of T cells, a subset of the body s inflammatory cells, that migrate selectively to the digestive tract. It is believed that when CCR9 s ligand, CCL25 (also known as TECK), is over-expressed, the migration of T cells to the small and large intestine causes persistent inflammation that may result in Crohn s disease or ulcerative colitis, the two forms of IBD. We have completed nine clinical trials with Traficet-EN in a total of 785 subjects, including five Phase I clinical trials, one Thorough QT study (an assessment of cardiovascular safety which is required for regulatory approval), and three Phase II clinical trials. We completed our PROTECT-1 Phase II clinical trial in 436 patients with moderate-to-severe Crohn s disease in 2009. Results from this clinical trial indicated that Traficet-EN was effective in inducing a clinical response over a 12-week treatment period. The results also indicated that Traficet-EN was effective in maintaining clinical remission over a 36-week treatment period. Traficet-EN was safe and well tolerated in all clinical trials completed to date. In December 2009, GSK exercised its option to obtain an exclusive license to further develop and commercialize Traficet-EN. To date, GSK has initiated three pivotal Phase III clinical trials with Traficet-EN in Crohn s disease. If approved, Traficet-EN would be the first oral agent with a novel mechanism of action introduced for the treatment of Crohn s disease since the introduction of corticosteroids and oral immunosuppressants.

CCX140, our lead independent drug candidate, targets the chemokine receptor known as CCR2. CCX140 is a potent and selective antagonist of CCR2 that is found on subsets of monocytes and macrophages, which are cells of the immune system believed to play an important role in inflammatory processes. Blocking CCR2 is intended to reduce the abnormal monocyte and macrophage driven inflammatory response implicated in renal disease. In addition, we have shown that levels of CCL2, the main ligand for CCR2, are elevated in the kidneys of patients with diabetic nephropathy, which is characterized by a persistent and usually progressive decline in renal function. Current treatments of patients with diabetic nephropathy primarily focus on treatment of the underlying type 2 diabetes and hypertension. Given that the current standard of care does not halt or reverse the progression of diabetic patients with impaired kidney function to end-stage renal disease, we believe that an unmet medical need persists for the treatment of diabetic nephropathy. As a precursor to our clinical trials in patients with diabetic nephropathy, in January 2011, we completed a 159-patient randomized Phase II clinical trial to assess the safety and tolerability of CCX140 in patients with type 2 diabetes, the most common cause of diabetic nephropathy. CCX140 was safe and well tolerated in this trial. In addition, CCX140 demonstrated biological activity through a dose-dependent decrease in fasting plasma glucose. The highest dose of 10mg CCX140 administered once-daily also lowered hemoglobin A1c, or HbA1c, with statistical significance over a four-week period. CCX140 is currently in two Phase II clinical trials in patients with diabetic nephropathy and we expect to complete these clinical trials by the end of 2012, provided that we do not increase the sample size of, or add additional dose groups to, the larger of these clinical trials.

CCX354 targets the chemokine receptor known as CCR1. Synovial fluid from the joints of RA patients contains high levels of activated CCR1 chemokine ligands. Blocking CCR1 is intended to reduce inflammation

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and prevent subsequent joint destruction by suppressing the infiltration of inflammatory cells into the arthritic joint. We successfully completed two Phase I clinical trials in a total of 84 healthy subjects, followed by a Phase I/II clinical trial in 24 patients with stable RA and a Phase II proof-of-concept clinical trial in 160 patients with moderate-to-severe RA. Results from this clinical trial demonstrated that patients who met inclusion criteria at the start of dosing had an ACR20 response at Week 12 of 56% in patients receiving 200mg of CCX354 once-daily compared to 44% in patients receiving 100mg of CCX354 twice-daily and 30% in patients receiving placebo. ACR20, ACR50 and ACR70 responses refer to patients who achieve a 20%, 50% and 70% improvement, respectively, according to criteria set by the American College of Rheumatology, or ACR. The ACR20 response difference between the 200mg CCX354 once-daily and placebo groups was statistically significant (p=0.014). The decrease in C-reactive protein, or CRP, a marker of inflammation, was statistically significant in the 200mg CCX354 once-daily group compared to placebo at Week 12 (p=0.023). CCX354 was well tolerated by patients in this clinical trial. This successful Phase II proof-of-concept clinical trial triggered GSK s option rights under our collaboration agreement. GSK exercised its option to further develop and commercialize CCX354 in November 2011 and has an exclusive right to initiate a Phase IIb clinical trial for CCX354 in RA.

CCX168 targets the chemo-attractant C5a receptor, or C5aR, which binds to a biologically activated fragment of the complement protein known as C5. Chemo-attractant receptors are related to the chemokine receptor family and similarly regulate the migration of certain types of inflammatory cells. C5aR is thought to play a role in a range of inflammatory and autoimmune diseases such as AAV, lupus, and psoriasis. We completed a Phase I clinical trial for CCX168, which showed that CCX168 was well tolerated at doses up to 100mg. We initiated a Phase II proof-of-concept clinical trial in AAV in the fourth quarter of 2011 and expect to complete this clinical trial by the end of 2012. If this clinical trial is successfully completed, GSK may exercise its option to further develop and commercialize CCX168 under our collaboration agreement.

CCX662 is our independent drug candidate that is a highly potent and selective small molecule compound which targets CXCR7, a novel chemokine receptor that we believe plays a key role in the survival of certain tumor cells. Of particular interest is the role that CXCR7 appears to play in the development of GBM, the deadliest of all brain cancers. In animal studies, CCX662 demonstrated safety and efficacy against GBM. We are in preclinical development with this drug candidate and anticipate initiation of Phase I clinical trials in patients with GBM in the second half of 2012.

We have developed a suite of proprietary technologies, which we call the EnabaLink drug discovery engine, to better understand the chemokine system and to accelerate the identification of small molecule lead compounds that target and inhibit the function of specific chemokine receptors. We believe this platform provides us with an advantage in the rapid identification of highly specific drug candidates. An important element of this platform is our thorough map of the chemokine network which allows us to better understand how a given chemokine-chemokine receptor interaction impacts the migration of cells in a given disease. With this understanding, we can apply our advanced screening methodologies, including a purpose-built high-throughput robotic screening technology, known as the Reverse Activation of Migration, or RAM, Assay, to identify small molecule antagonists for the chemokine receptor most closely associated with a specific disease. The RAM Assay is designed to markedly reduce or eliminate non-specific inhibitors and toxic inhibitors of cell migration, resulting in highly specific lead candidates. This technology allows us to screen against targets that are not accessible with traditional technologies, providing us with what we believe to be a competitive advantage in drug discovery. We have used our EnabaLink drug discovery engine in our drug candidate programs and continue to apply these powerful research tools in our early stage drug discovery efforts.

Thomas Schall, Ph.D., our founder, President and Chief Executive Officer, has more than 25 years of research experience in the field of chemokine biology and has contributed broadly to the understanding of chemokines and their receptors in human disease. Since our founding, we have raised $384.9 million, of which $175.0 million has been in the form of convertible debt and equity financings and $209.8 million in the form of collaboration funding and government contracts and grants. As of September 30, 2011, we had $81.2 million of

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cash, cash equivalents and investments. In December 2011, we received a $25.0 million option exercise payment under our strategic alliance with GSK (described below) with respect to CCX354 which is reflected in the collaboration funding described above. We believe that our broad pipeline of oral drug candidates, our ability to advance unique, highly specific compounds into and through clinical development across diverse indications and our proprietary drug discovery technologies provide us with distinct advantages that will enable us to continue to exploit the extensive pharmacologic potential of the chemokine system.

Strategic Alliance with GSK

In August 2006, we entered into our strategic alliance with GSK. We have received $245.7 million from GSK, consisting of up-front and milestone payments, equity investments, research funding and option exercise fees. Under the terms of our agreement with GSK, we are responsible for the discovery and development of small molecule antagonists targeting four defined chemokine and chemo-attractant receptor targets (CCR9, CCR1, C5aR and ChemR23) and for advancing them through clinical proof-of-concept. If we demonstrate successful clinical proof-of-concept, GSK is entitled to options to exclusively license drug candidates that are subject to the collaboration and two defined back-up compounds for each drug candidate for further development and commercialization on a worldwide basis. Upon exercising any of its options to drug candidates under the collaboration, GSK is solely responsible for all further clinical development and commercialization expenditures worldwide with respect to that drug candidate and its two designated back-up compounds. In exchange for the rights granted to GSK upon the exercise of its options, we are also entitled to receive regulatory and commercial milestone payments, as earned under the terms of our agreement, and royalties on the net sales of licensed drugs. The agreement contemplated up to six drug options, each of which covers a drug candidate against the four defined targets, including Traficet-EN (CCR9), CCX354 (CCR1), CCX168 (C5aR) and CCX832 (ChemR23), and their associated back-up compounds. The other two drug options were for second generation drug candidates and their associated back-up compounds. However, we and GSK chose not to nominate second generation drug candidates against any of the four defined targets during the agreement s research term, which has expired. In addition, in February 2012, based on unblinded data from a recently completed Phase I clinical trial of CCX832, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds, although both we and GSK continue to have interest in further discussing possible strategic opportunities with respect to ChemR23. GSK has already exercised its options to Traficet-EN and CCX354 and each of their two respective defined back-up compounds. Thus, GSK s only remaining option is to CCX168 and its associated back-up compounds. If GSK does not exercise its option to CCX168, we will evaluate our alternatives for further development of this drug candidate, which may entail internally developing it or identifying other collaboration partners for its development.

Strategy

Our strategy includes the following key elements:

- Collaborate with GSK in the Phase III clinical development and commercialization of Traficet-EN for IBD and in the further development and commercialization of CCX354;
- Forward integrate into a commercial biopharmaceutical company by driving the development and commercialization of our lead independent drug candidate, CCX140, currently in Phase II clinical development for diabetic nephropathy;
- Advance CCX168 under our collaboration with GSK;
- Expand our clinical stage portfolio of internally discovered, independent drug candidates;
- Leverage our expertise and proprietary technologies to continue discovering and developing a broad pipeline of novel chemokine-based therapeutics; and

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Commercialize our drug candidates in specialty markets in North America and partner outside North America and in primary care markets worldwide.

Risks Related to Our Business

Our ability to implement our current business strategy is subject to numerous risks, as more fully described in the section entitled Risk Factors immediately following this prospectus summary. These risks include, among others:

our dependence on the success of Traficet-EN, CCX140 and our other drug candidates;

delays in obtaining, or a failure to obtain, regulatory approval for our drug candidates;

our dependence on GSK for the success of the drug candidates that it licenses from us;

failure of any approved product to achieve significant commercial acceptance in the medical community or receive reimbursement by third-party payors;

unfavorable clinical trial results;

our dependence upon third parties under our licensing, collaboration, contract research and manufacturing agreements;

delays in product launch;

failure to maintain and protect our proprietary intellectual property assets; and

failure to acquire licenses necessary to commercialize any of our drug candidates, including Traficet-EN and CCX140.

In addition, all of our drug candidates are subject to regulatory approval by the Food and Drug Administration, or FDA, and comparable agencies in other countries. Traficet-EN, CCX140, CCX354 and CCX168 are our only drug candidates currently in clinical trials and none of them has received regulatory approval. We cannot give any assurance that they, or any other drug candidates we may develop or acquire, will receive regulatory approval or be successfully commercialized.

We have not generated any revenue to date from product sales and have incurred significant operating losses since our inception in 1997 (other than during the year ended December 31, 2009, when we generated net income of $15.6 million). We incurred net losses of $18.5 million in 2008, $3.1 million in 2010, and $22.8 million for the nine months ended September 30, 2011. As of September 30, 2011, we had an accumulated deficit of approximately $112.5 million and we expect to incur losses for the foreseeable future. We are unable to predict the extent of future losses or when we will become profitable, if at all. Even if we succeed in developing and commercializing one or more of our drug candidates, we may never generate sufficient revenue to achieve and sustain profitability.

Concurrent Private Placements

GSK and Techne Corporation, or Techne, have agreed to purchase $7.0 million and $5.0 million, respectively, of our common stock in separate private placements concurrent with the completion of this offering at a price per share equal to the initial public offering price. The sale of such shares of common stock will not be registered under the Securities Act of 1933, as amended, or the Securities Act.

Corporate Information

We commenced operations in 1997. Our principal offices are located at 850 Maude Avenue, Mountain View, California 94043, and our telephone number is (650) 210-2900. Our website address is

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http://www.chemocentryx.com. The information contained in, or that can be accessed through, our website is not part of this prospectus. We have a wholly owned subsidiary, ChemoCentryx Limited, organized under the laws of the United Kingdom that is currently inactive. Unless the context requires otherwise, in this prospectus the terms ChemoCentryx, we, us and our refer to ChemoCentryx, Inc., a Delaware corporation, a our subsidiary taken as a whole unless otherwise noted.

ChemoCentryx®, the ChemoCentryx logo, Traficet and Traficet-EN are our trademarks in the United States, the European Community, Australia and Japan. EnabaLink® and RAM® are our trademarks in the United States. Each of the other trademarks, trade names or service marks appearing in this prospectus belongs to its respective holder.

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THE OFFERING

Common stock offered by us in this offering	4,000,000 Shares (or 4,600,000 shares if the underwriters over-allotment option is exercised in full)
Common stock to be sold by us to GSK in the concurrent private placement (assuming an initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus))	466,666 Shares
Common stock to be sold by us to Techne in the concurrent private placement (assuming an initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus))	333,333 Shares
Common stock to be outstanding after this offering and the concurrent private placements to GSK and Techne and the automatic conversion of the convertible note held by Techne	34,020,576 Shares
Use of proceeds	We estimate that the net proceeds from this offering will be approximately $53.3 million, or approximately $61.7 million if the underwriters exercise their over-allotment option in full, at an assumed initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus), after deducting the underwriting discounts and commissions and estimated offering expenses payable by us. We will also receive $12.0 million from the sale of 799,999 shares of common stock in the concurrent private placements to GSK and Techne, at a price per share equal to the initial public offering price. We intend to use the net proceeds from this offering and the concurrent private placements to GSK and Techne to further develop our lead independent drug candidate CCX140, to advance CCX168 and CCX662 further in clinical development, for the further exploration of our ChemR23 program, for the research and development of additional drug candidates and for working capital and general corporate purposes.
Proposed Nasdaq Global Market symbol	CCXI

The number of shares of common stock to be outstanding after (1) this offering, (2) the concurrent private placements to GSK and Techne and (3) the automatic conversion of the convertible note held by Techne (see Certain Relationships and Related Party Transactions Relationships with Techne), is based on 28,551,901 shares of common stock outstanding as of September 30, 2011 (assuming conversion of all of our outstanding shares of preferred stock), and an assumed initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus) and excludes the following:

- 4,172,318 shares of common stock issuable upon the exercise of outstanding stock options having a weighted-average exercise price of $4.72 per share;

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159,500 shares of common stock issuable upon the exercise of outstanding warrants having an exercise price of $5.20 per share;

59,009 shares of common stock reserved for issuance pursuant to future option grants under our 2002 Equity Incentive Plan and our 1997 Stock Option/Stock Issuance Plan;

150,000 shares of common stock issuable upon the exercise of warrants, with an exercise price per share equal to 200% of the initial public offering price of our common stock, which warrants will be issued to Techne upon the completion of this offering.

3,000,000 shares of common stock reserved for issuance pursuant to future option grants under our 2012 Equity Incentive Award Plan;

300,000 shares of common stock reserved for issuance under our 2012 Employee Stock Purchase Plan; and

the issuance of an additional 12,044 shares of our common stock upon the automatic conversion of the convertible note held by Techne, assuming a conversion date of February 10, 2012, at a conversion price equal to the assumed public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus).

Except as otherwise indicated, all information contained in this prospectus:

reflects the conversion of all of our outstanding shares of preferred stock into an aggregate of 24,332,186 shares of common stock prior to the completion of this offering;

assumes the adoption of our amended and restated certificate of incorporation and amended and restated bylaws upon the completion of this offering;

assumes that the underwriters do not exercise their over-allotment option;

reflects the issuance and sale of 799,999 shares of common stock in the concurrent private placements to GSK and Techne at the assumed initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus);

reflects the issuance of 668,676 shares of common stock upon the completion of this offering, as a result of the automatic conversion of the convertible note held by Techne, based upon the outstanding principal and interest under this note as of September 30, 2011, at a conversion price equal to the assumed initial public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus); and

reflects a one-for-two reverse stock split of our common stock to be effected before the completion of this offering.

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SUMMARY FINANCIAL DATA

The following summary consolidated financial data for the years ended December 31, 2008, 2009 and 2010 are derived from our audited consolidated financial statements appearing elsewhere in this prospectus. The summary financial data for the nine months ended September 30, 2010 and 2011 and as of September 30, 2011 are derived from our unaudited consolidated financial statements appearing elsewhere in this prospectus and are not indicative of results to be expected for the full year. You should read this data together with our audited and unaudited consolidated financial statements and related notes appearing elsewhere in this prospectus and the information under the captions Selected Financial Data and Management s Discussion and Analysis of Financial Condition and Results of Operations. Our historical results are not necessarily indicative of our future results.

The pro forma as adjusted consolidated balance sheet data reflects (1) the conversion of all outstanding shares of our preferred stock into an aggregate of 24,332,186 shares of common stock prior to the completion of this offering, (2) the issuance and sale by us of 4,000,000 shares of our common stock in this offering, after deducting the underwriting discounts and commissions and estimated offering expenses payable by us, (3) the concurrent private placements to GSK and Techne of $7.0 million and $5.0 million of our common stock, respectively, and (4) the automatic conversion of the convertible note held by Techne at a conversion price equal to the initial public offering price, in each case at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus.

	Years Ended December 31,			Nine Months Ended September 30,	
	2008	2009	2010	2010	2011
				(unaudited)	
	(in thousands, except share and per share data)				
Consolidated Statement of Operations Data:					
Revenues:					
Collaborative research and development revenue from related party	$ 23,551	$ 49,744	$ 34,861	$ 21,746	$ 5,621
Grant revenue	536				
Total revenues:	24,087	49,744	34,861	21,746	5,621
Operating expenses:					
Research and development	35,056	27,474	33,527	25,385	22,914
General and administrative	9,157	6,575	7,292	5,363	5,721
Total operating expenses	44,213	34,049	40,819	30,748	28,635
Income (loss) from operations	(20,126)	15,695	(5,958)	(9,002)	(23,014)
Interest income	1,762	297	436	322	319
Interest expense	(129)	(76)	(81)	(60)	(170)
Other income			2,434	490	16
Income (loss) before provision for income taxes	(18,493)	15,916	(3,169)	(8,250)	(22,849)
Income tax benefit (expense)	23	(293)	73	73	
Net income (loss)	$ (18,470)	$ 15,623	$ (3,096)	$ (8,177)	$ (22,849)
Basic net income (loss) per share[(1)]	$ (4.52)	$ 0.56	$ (0.76)	$ (2.01)	$ (5.49)
Diluted net income (loss) per share[(1)]	$ (4.52)	$ 0.53	$ (0.76)	$ (2.01)	$ (5.49)
Shares used to compute basic net income (loss) per share	4,087,181	3,961,640	4,081,648	4,077,347	4,162,309
Shares used to compute diluted net income (loss) per share	4,087,181	29,256,423	4,081,648	4,077,347	4,162,309

Pro forma basic and diluted net income (loss) per share (unaudited)[1]	$ (0.11)	$ (0.80)
Shares used to compute pro forma basic and diluted net income (loss) per share	28,210,296	28,471,126

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	As of September 30, 2011	
	Actual	Pro Forma As Adjusted[2]
	(in thousands)	
Consolidated Balance Sheet Data		
Cash, cash equivalents and investments	$ 81,182	$ 146,482
Working capital	64,964	130,264
Total assets	85,365	150,665
Non-current equipment financing obligations	1,040	1,040
Convertible note from related party	10,060	
Accumulated deficit	(112,530)	(112,530)
Total stockholders equity	56,955	132,315

(1) See Note 2 within the notes to our consolidated financial statements which are included elsewhere in this prospectus for a description of the method used to compute basic and diluted loss per share.

(2) Each $1.00 increase or decrease in the assumed initial public offering price of $15.00 would increase or decrease, respectively, the amount of cash, cash equivalents and investments, working capital, total assets and total stockholders equity by $3.7 million, assuming the number of shares offered by us, as set forth on the cover page of this prospectus, remains the same and after deducting the underwriting discounts and commissions and estimated offering expenses payable by us.

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RISK FACTORS

Investing in our common stock involves a high degree of risk. You should carefully consider the risks and uncertainties described below and all information contained in this prospectus before deciding to invest in our common stock. If any of the following risks actually occur, our business, financial condition, results of operations and growth prospects would likely be materially and adversely affected. In that event, the trading price of our common stock could decline, and you could lose all or part of your investment.

Risks Related to Our Business

We anticipate that we will continue to incur significant losses for the foreseeable future, and if we are unable to achieve and sustain profitability, the market value of our common stock will likely decline.

We are a clinical-stage biopharmaceutical company. We do not currently have any products approved for sale, and we continue to incur significant research and development and general and administrative expenses related to our operations. Our net loss for the years ended December 31, 2008 and 2010 and the nine months ended September 30, 2011 was $18.5 million, $3.1 million and $22.8 million, respectively (we generated net income of $15.6 million for the year ended December 31, 2009). As of September 30, 2011, we had an accumulated deficit of $112.5 million. We expect to continue to incur significant losses for the foreseeable future. We expect these losses and our cash utilization to increase in the near term as we continue to conduct clinical trials for CCX140, CCX168 and CCX662 and conduct research and development of our other drug candidates. Glaxo Group Limited, or GSK, an affiliate of GlaxoSmithKline, has assumed all funding obligations for the further clinical development and commercialization of Traficet-EN and CCX354. If GSK exercises its option for further development and commercialization of our remaining drug candidate subject to the agreement, it will assume all funding obligations with respect to further clinical development of such drug candidate, but if it does not exercise such option, we will be responsible for such funding obligations. All of our products are in development and none has been approved for sale. To date, we have derived all of our revenues from up-front fees and milestone payments, other payments pursuant to our collaboration agreements and government grants and contracts for research and development. We do not anticipate that we will generate revenue from the sale of our products for the foreseeable future. In addition, if approved, we expect to incur significant costs to commercialize our drug candidates and our drugs may never gain market acceptance. If our drug candidates fail to demonstrate safety and efficacy in clinical trials, do not gain regulatory approval, or do not achieve market acceptance, we may never become profitable. Even if we achieve profitability in the future, we may not be able to sustain profitability in subsequent periods. If we are unable to achieve and sustain profitability, the market value of our common stock will likely decline. Because of the numerous risks and uncertainties associated with developing pharmaceutical products, we are unable to predict the extent of any future losses or whether we will become profitable.

The commercial success of Traficet-EN depends, in large part, on the development and marketing efforts of GSK, and if GSK is unable to perform in accordance with the terms of our agreement, or is unable to obtain the required regulatory approvals for Traficet-EN, our potential to generate future revenue from this drug candidate would be significantly reduced and our business would be materially and adversely harmed.

Since inception, we have invested a significant portion of our time and financial resources in the development of our most advanced drug candidate, Traficet-EN. We currently have three other drug candidates in clinical trials, but we anticipate that our ability to generate significant product revenues in the near term will depend primarily on the successful development, regulatory approval, marketing and commercialization of Traficet-EN by us or by GSK, which is subject to significant uncertainty. In particular, we rely on GSK to fund and conduct the current pivotal Phase III trials with respect to Traficet-EN. Any of the following events or factors could have a material adverse effect on our ability to generate revenue from the commercialization of Traficet-EN:

- GSK may be unable to successfully complete the clinical development of Traficet-EN;
- GSK must comply with additional requests and recommendations from the FDA, including additional clinical trials;

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GSK may not obtain all necessary approvals from the FDA and similar foreign regulatory agencies;

GSK may not commit sufficient resources to the development, regulatory approval, marketing and distribution of Traficet-EN;

Traficet-EN must be manufactured in compliance with requirements of the FDA and similar foreign regulatory agencies and in commercial quantities sufficient to meet market demand;

Traficet-EN may not achieve market acceptance by physicians, patients and third party payors;

Traficet-EN may not compete successfully against alternative products and therapies; and

We, GSK or any other pharmaceutical organization may independently develop products that compete with Traficet-EN.

In order to obtain approval from the FDA of a new drug application, or NDA, for Traficet-EN, GSK will need to demonstrate through evidence from adequate and well-controlled clinical trials that Traficet-EN is safe and effective for each proposed indication. However, Traficet-EN may not be approved even though it achieved its specified endpoints in the current and/or future pivotal Phase III clinical trials intended to support an NDA which may be conducted by GSK. The FDA may disagree with the trial design and the interpretation of data from clinical trials, may ask GSK to conduct additional costly and time consuming clinical trials in order to obtain marketing approval or approval to enter into an advanced phase of development, or may change the requirements for approval even after it has reviewed and commented on the design for our clinical trials. The FDA may also approve Traficet-EN for fewer or more limited indications than GSK may request, or may grant approval contingent on the performance of costly post-approval clinical trials. In addition, the FDA may not approve the labeling claims that we believe are necessary or desirable for the successful commercialization of Traficet-EN.

If GSK or any of our future collaboration partners does not perform in the manner we expect or fulfill its responsibilities in a timely manner, or at all, the clinical development, regulatory approval, and commercialization efforts related to Traficet-EN could be delayed or terminated. It may be necessary for us to assume the responsibility at our own expense for the development of Traficet-EN. In that event, we would likely be required to limit the size and scope of one or more of our programs or increase our expenditures and seek additional funding and our potential to generate future revenue from Traficet-EN would be significantly reduced and our business would be materially and adversely harmed.

If clinical proof-of-concept is not achieved with respect to our remaining drug candidate under our strategic alliance with GSK, if GSK does not exercise its option thereunder, if the further development and commercialization efforts of GSK are not successful with respect to drug candidates for which it does exercise its options thereunder, or if GSK terminates the alliance or a particular program thereunder, we will not receive any additional revenue under the alliance with respect to such programs and our results of operations and financial condition will be materially adversely affected.

In August 2006, we entered into our strategic alliance with GSK. Under the terms of our agreement, we are responsible for the discovery and development of small molecule antagonists targeting four defined chemokine and chemo-attractant receptor targets (CCR9, CCR1, C5aR and ChemR23) and taking them through clinical proof-of-concept. If we demonstrate successful clinical proof-of-concept, GSK is entitled to options to exclusively license drug candidates that are subject to the collaboration and two defined back-up compounds for each drug candidate for further development and commercialization on a worldwide basis. The agreement contemplated up to six drug options, each of which covers a drug candidate against four defined targets, including Traficet-EN (CCR9), CCX354 (CCR1), CCX168 (C5aR) and CCX832 (ChemR23), and their associated back-up compounds. The other two drug options were for second generation drug candidates and their associated back-up compounds that would target any of the four collaboration targets. However, we and GSK chose not to nominate second generation drug candidates against any of the four defined targets during the agreement s research term, which has expired. In addition, in February 2012, based on unblinded data from a recently completed Phase I clinical trial of CCX832, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds, although both we and GSK continue to have

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interest in further discussing possible strategic opportunities with respect to ChemR23. GSK has already exercised its options to Traficet-EN and CCX354 and each of their two respective defined back-up compounds. Thus, GSK s only remaining option is to CCX168 and its associated back-up compounds.

In December 2009, GSK exercised its option under the agreement to obtain an exclusive license for the further development and commercialization of Traficet-EN, our CCR9 drug candidate, and two identified back-up compounds. As a result of GSK s exercise of this option, we are entitled to receive (x) up to $82.0 million, in the aggregate, consisting of (1) a non-refundable option exercise fee of $35.0 million and (2) up to $47.0 million in the aggregate for the filing of an NDA in the United States and comparable filings in other territories, (y) up to $75.0 million in the aggregate for the regulatory approval of products for commercial sale in the United States and other territories and (z) up to $250.0 million in sales milestones. In January 2010, after GSK obtained Hart-Scott-Rodino clearance for its option exercise, it paid us the option exercise fee of $35.0 million and assumed sole responsibility for the further development and commercialization of Traficet-EN and its two designated back-up compounds, at its expense, subject to our specified co-development and commercial participation rights.

In November 2011, GSK exercised its option under the agreement to obtain an exclusive license for the further development and commercialization of CCX354, our CCR1 drug candidate, and two identified back-up compounds. As a result of GSK s exercise of this option, we are entitled to receive (x) up to $72.0 million, in the aggregate, consisting of (1) a non-refundable option exercise fee of $25.0 million and (2) up to $47.0 million in the aggregate for the filing of an NDA in the United States and comparable filings in other territories, (y) up to $75.0 million in the aggregate for the regulatory approval of products for commercial sale in the United States and other territories and (z) up to $125.0 million in sales milestones. In December 2011, GSK paid us the option exercise fee of $25.0 million and assumed sole responsibility for the further development and commercialization of CCX354 and its two designated back-up compounds, at its expense. There is no assurance that GSK will be successful in its further development and commercialization of CCX354 or that the relevant regulatory filing or approval or sales milestones can be achieved such that we will receive the related milestone payments.

If a given proof-of-concept trial for our CCX168 drug candidate is successfully completed and GSK elects to exercise its option to such drug candidate, we would be entitled to receive, as with CCX354, (x) up to $72.0 million, in the aggregate, consisting of (1) an option exercise fee of $25.0 million and (2) up to $47.0 million in the aggregate for the filing of an NDA in the United States and comparable filings in other territories, (y) up to $75.0 million in the aggregate for the regulatory approval of products for commercial sale in the United States and other territories and (z) up to $125.0 million in sales milestones. We cannot assure you that we will be able to successfully complete a proof-of-concept trial for CCX168 or that the relevant regulatory filing or approval milestones can be achieved for any our programs so that we will receive the related option exercise fees and milestone payments. In addition, even if proof-of-concept trials for any of our drug candidates result in positive outcomes, GSK is under no obligation to exercise its option and we cannot assure you that GSK will exercise its remaining option, or that GSK will obtain Hart-Scott-Rodino clearance with respect to such option, to the extent that such approval is required.

GSK may terminate the entire collaboration agreement or any collaboration program on a program-by-program basis for any reason upon 90 days prior written notice to us. The agreement or any program under the agreement may also be terminated for cause under certain circumstances, including material breach and insolvency. In addition, GSK may terminate its rights with respect to the licensed product if it determines in good faith, for any reason, to cease the development and commercialization of such product and provides us with a written notice of such intent.

If GSK does not exercise its option with respect to our other development candidate, terminates its rights with respect to a licensed product, or terminates the agreement:

- we would not be entitled to receive the relevant option exercise fee or milestone payments;

- we would owe GSK up to 5% royalties with respect to drug candidates covered by the agreement which we elected to subsequently commercialize, depending upon the stage of development at which such product commercialization rights reverted back to us;

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- the development of our drug candidates subject to the agreement may be terminated or significantly delayed;
- we may be required to hire additional employees and allocate scarce resources to the development and commercialization of drug candidates that were previously the subject of the GSK agreement and as a result our cash expenditures could increase significantly;
- we would bear all of the risks and costs related to the further development and commercialization of drug candidates that were previously the subject of the GSK agreement, including the reimbursement of third parties; and
- we may need to establish alternative collaboration arrangements, and we may not be able to do so, or may not be able to do so on terms which are acceptable to us, in which case we would likely be required to limit the size or scope of one or more of our programs or increase our expenditures and seek substantial additional funding.

Any of these events would have a material adverse effect on our results of operations and financial condition.

The development of new drugs is a highly risky undertaking which involves a lengthy process, and our drug discovery and development activities therefore may not result in products that are approved by the applicable regulatory authorities on the time schedule we have planned, or at all.

Our drug candidates are in the early stages of drug discovery or clinical trials and are prone to the risks of failure inherent in drug development. As of the date of this prospectus, only four of our current drug candidates, Traficet-EN, CCX140, CCX354 and CCX168 have been tested in human beings. We will need to conduct significant additional preclinical studies and clinical trials before we can demonstrate that any of our drug candidates is safe and effective to the satisfaction of the FDA and other regulatory authorities. Preclinical studies and clinical trials are expensive and uncertain processes that take years to complete. For example, we incurred significant expenses related to the IND filing and the completed single ascending dose Phase I clinical trial for CCX915, our first generation CCR2 drug candidate, which did not advance into Phase II clinical trials because its pharmacokinetic properties in humans did not meet our expectations. Failure can occur at any stage of the process, and we cannot assure you that any of our drug candidates will result in commercially successful products.

We cannot assure you that our ongoing clinical trials or any future clinical trial of any of our other drug candidates, will be completed on schedule, or at all, or whether our planned clinical trials will start in a timely manner. The commencement of our planned clinical trials could be substantially delayed or prevented by a number of factors, including:

- delays or failures in obtaining sufficient quantities of the active pharmaceutical ingredient, or API, and/or drug product;
- delays or failures in reaching agreement on acceptable clinical trial agreement terms or clinical trial protocols with prospective sites;
- delays or failures in obtaining institutional review board, or IRB, approval to conduct a clinical trial at a prospective site;
- the need to successfully complete, on a timely basis, preclinical safety pharmacology studies;
- the limited number of, and competition for, suitable sites to conduct the clinical trials;
- the limited number of, and competition for, suitable patients for enrollment in the clinical trials; and

delays or failures in obtaining regulatory approval to commence a clinical trial.

The completion of our clinical trials could also be substantially delayed or prevented by a number of factors, including:

slower than expected rates of patient recruitment and enrollment;

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failure of patients to complete the clinical trials;

failure of our third party vendors to timely or adequately perform their contractual obligations relating to the clinical trials;

inability or unwillingness of patients or medical investigators to follow our clinical trial protocols;

inability to monitor patients adequately during or after treatment;

termination of the clinical trials by one or more clinical trial sites;

unforeseen safety issues;

lack of efficacy demonstrated during clinical trials;

lack of adequate funding to continue the clinical trials;

the need for unexpected discussions with the FDA or other foreign regulatory agencies regarding the scope or design of our clinical trials or the need to conduct additional trials;

unforeseen delays by the FDA or other foreign regulatory agencies after submission of our results;

an unfavorable FDA inspection of our contract manufacturers of API or drug product; and

inspection of the clinical trial operations or trial sites by the FDA or other regulatory authorities resulting in the imposition of a clinical hold.

Any failure or significant delay in completing clinical trials for our drug candidates would harm the commercial prospects for our drug candidates and adversely affect our financial results.

Additionally, changes in regulatory requirements and guidance may occur and we may need to amend clinical trial protocols to reflect these changes. Amendments may require us to resubmit our clinical trial protocols to IRBs for reexamination, which may impact the costs, timing or successful completion of a clinical trial. If we experience delays in completion of, or if we terminate, any of our clinical trials, the commercial prospects for our drug candidates may be harmed and our ability to generate product revenues will be delayed. In addition, many of the factors that cause, or lead to, a delay in the commencement or completion of clinical trials may also ultimately lead to the denial of regulatory approval of a drug candidate.

If we are required to suspend or discontinue clinical trials due to side effects or other safety risks, or if we are required to conduct studies on the long-term effects associated with the use of our drug candidates, our efforts to commercialize our products could be delayed or halted.

Our clinical trials may be suspended or terminated at any time for a number of safety-related reasons. For example, we may voluntarily suspend or terminate our clinical trials if at any time we believe that our drug candidates present an unacceptable safety risk to the clinical trial patients. In addition, IRBs or regulatory agencies may order the temporary discontinuation or termination of our clinical trials at any time if they believe that the clinical trials are not being conducted in accordance with applicable regulatory requirements, including if they present an unacceptable

safety risk to patients. Administering any drug candidate to humans may produce undesirable side effects. The existence of undesirable side effects resulting from our drug candidates could cause us or regulatory authorities, such as the FDA, to interrupt, delay or halt clinical trials of our drug candidates and could result in the FDA or other regulatory agencies denying further development or approval of our drug candidates for any or all targeted indications. This, in turn, could affect whether GSK exercises its remaining license option under our strategic alliance and could prevent us from commercializing our drug candidates.

Further, chemokine receptors and chemo-attractant receptors are a novel class of targets. As a result, we may experience unforeseen adverse side effects with our existing and future drug candidates, including Traficet-EN and CCX140. As of the date of this prospectus, four of our current drug candidates have been tested in human beings. Although we have not observed significant harmful side effects in prior studies of Traficet-EN, CCX140 or our other drug candidates, later trials could reveal such side effects. The pharmacokinetic profile of preclinical studies may not be indicative of results in any clinical trial. For example, prior to commencing our preclinical studies of our

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CCX140 drug candidate, we studied another drug candidate that targeted CCR2, which we abandoned after pharmacokinetic results were not as favorable in humans as in earlier preclinical animal studies. We have not conducted studies on the long-term effects associated with the use of our drug candidates. Studies of these long-term effects may be required for regulatory approval and would delay our introduction of Traficet-EN, CCX140 or our other drug candidates into the market. These studies could also be required at any time after regulatory approval of any of our drug candidates. Absence of long-term data may also limit the approved uses of our products, if any, to short-term use. Some or all of our drug candidates may prove to be unsafe for human use.

Even if our drug candidates do obtain regulatory approval they may never achieve market acceptance or commercial success.

Even if we obtain FDA or other regulatory approvals, our drug candidates may not achieve market acceptance among physicians, patients and third party payors and, ultimately, may not be commercially successful. Market acceptance of our drug candidates for which we receive approval depends on a number of factors, including:

- the efficacy and safety as demonstrated in clinical trials;
- the clinical indications for which the drug is approved;
- acceptance by physicians, major operators of clinics and patients of the drug as a safe and effective treatment;
- the potential and perceived advantages of our drug candidates over alternative treatments;
- the safety of drug candidates seen in a broader patient group, including its use outside the approved indications;
- the cost of treatment in relation to alternative treatments;
- the availability of adequate reimbursement and pricing by third parties and government authorities;
- relative convenience and ease of administration;
- the prevalence and severity of adverse side effects; and
- the effectiveness of our sales and marketing efforts.

Any failure by our drug candidates that obtain regulatory approval to achieve market acceptance or commercial success would adversely affect our financial results.

The commercial success of CCX140 depends, in part, on our ability to develop and market the drug in North America and to find partners to co-develop and commercialize the drug outside North America, and if we fail in these initiatives, our ability to generate future revenue could be significantly reduced.

If we successfully complete the Phase II program for our lead independent drug candidate, CCX140, we plan to initiate Phase III clinical trials either alone or together with a co-development partner. We plan to retain commercial rights to CCX140 in North America and find partners for co-development and commercialization outside North America. We have invested a significant amount of our time and financial resources in the

development of CCX140 and our ability to generate future revenue will depend, in part, on our ability to identify a co-development partner and the development, regulatory approval, marketing and commercialization of CCX140 by us and any future partners. Any of the following events or factors could have a material adverse effect on our ability to generate revenue from the commercialization of CCX140:

We may be unable to successfully complete the clinical development of CCX140;

Our lack of experience in commercializing and marketing drug products;

We may not have or be able to obtain sufficient financial resources to develop and commercialize CCX140;

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We may not be able to identify a suitable co-development partner;

We or any of our future partners may fail to fulfill our responsibilities in a timely manner or fail to commit sufficient resources to the development, regulatory approval, and commercialization efforts related to CCX140;

We or any of our future partners must comply with additional requests and recommendations from the FDA, including additional clinical trials;

We or any of our future partners may not obtain all necessary approvals from the FDA and similar foreign regulatory agencies;

CCX140 must be manufactured in compliance with requirements of FDA and similar foreign regulatory agencies and in commercial quantities sufficient to meet market demand;

CCX140 may not achieve market acceptance by physicians, patients and third party payors;

CCX140 may not compete successfully against alternative products and therapies; and

We or any pharmaceutical company may independently develop products that compete with CCX140.

We rely on third parties to conduct all our preclinical studies and clinical trials. If these third parties do not successfully carry out their contractual duties or meet expected deadlines, we may be unable to obtain regulatory approval for or commercialize any of our drug candidates.

We currently do not have the ability to independently conduct preclinical studies or clinical trials. We rely on medical institutions, clinical investigators, contract laboratories, collaborative partners and other third parties, such as clinical research organizations, or CROs, to conduct clinical trials on our drug candidates. The third parties with which we contract for execution of our clinical trials play a significant role in the conduct of these trials and the subsequent collection and analysis of data. These third parties are not our employees, and except for contractual duties and obligations, we have limited ability to control the amount or timing of resources that they devote to our programs. In particular, we rely on GSK to fund and conduct the current pivotal Phase III trials with respect to Traficet-EN. Although we rely on these third parties to conduct our preclinical studies and clinical trials, we remain responsible for ensuring that each of our preclinical studies and clinical trials is conducted in accordance with its investigational plan and protocol. Moreover, the FDA and foreign regulatory authorities require us to comply with regulations and standards, commonly referred to as current good clinical practices, or cGCPs, for conducting, monitoring, recording and reporting the results of clinical trials to ensure that the data and results are scientifically credible and accurate and that the trial subjects are adequately informed of the potential risks of participating in clinical trials.

In addition, the execution of preclinical studies and clinical trials, and the subsequent compilation and analysis of the data produced, requires coordination among various parties. In order for these functions to be carried out effectively and efficiently, it is imperative that these parties communicate and coordinate with one another. Moreover, these third parties may also have relationships with other commercial entities, some of which may compete with us. In most cases, these third parties may terminate their agreements with us upon 30 days prior written notice of a material breach by us that is not cured within 30 days. Many of these agreements may also be terminated by such third parties under certain other circumstances, including our insolvency or our failure to comply with applicable laws. In general, these agreements require such third parties to reasonably cooperate with us at our expense for an orderly winding down of services of such third parties under the agreements. If the third parties conducting our clinical trials do not perform their contractual duties or obligations, experience work stoppages, do not meet expected deadlines, terminate their agreements with us or need to be replaced, or if the quality or accuracy of the clinical data they obtain is compromised due to the failure to adhere to our clinical trial protocols or cGCPs, or for any other reason, we may need to enter into new arrangements with alternative third parties, which could be costly, and our clinical trials may be extended, delayed or terminated or may need to be repeated, and we may not be able to obtain regulatory approval for or commercialize the drug candidate being tested in such trials.

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If any of our drug candidates receives marketing approval and we or others later identify undesirable side effects caused by the drug candidate, our ability to market and derive revenue from the drugs could be compromised.

If we or others identify undesirable side effects caused by one of our drugs, any of the following adverse events could occur:

- regulatory authorities may withdraw their approval of the drug or seize the drug;
- we may be required to recall the drug or change the way the drug is administered;
- additional restrictions may be imposed on the marketing of the particular drug or the manufacturing processes;
- we may be subject to fines, injunctions or the imposition of civil or criminal penalties;
- regulatory authorities may require the addition of labeling statements, such as a black box warning or a contraindication;
- we may be required to create a Medication Guide outlining the risks of such side effects for distribution to patients;
- we could be sued and held liable for harm caused to patients;
- the drug may become less competitive; and
- our reputation may suffer.

Any of these could result in the loss of significant revenues, which would materially and adversely affect our results of operations and business.

We will need additional financing and may be unable to raise capital on acceptable terms, or at all, when needed, which would force us to delay, reduce or eliminate our research and development programs and other operations or commercialization efforts.

We are advancing multiple drug candidates through discovery and development and will require substantial funds to conduct development, including preclinical studies and clinical trials, of our drug candidates. Commercialization of any drug candidate will also require substantial expenditures. While we currently expect GSK to assist us in our development and commercialization efforts with respect to those of our drug candidates for which GSK exercises an option under our agreement, we may also need additional financing to the extent that we are required to hire additional employees to co-promote drug candidates or to commercialize drug candidates that may not be covered by our collaboration agreement.

As of September 30, 2011, we had approximately $81.2 million in cash, cash equivalents and investments. Subsequent to September 30, 2011, we received a $25.0 million option exercise payment under our strategic alliance with GSK with respect to CCX354. We believe that our available cash, cash equivalents and investments, together with the net proceeds of this offering, will be sufficient to fund our anticipated level of operations for at least the next 12 months. Our future financing requirements will depend on many factors, some of which are beyond our control, including:

the rate of progress and cost of our clinical trials, preclinical studies and other discovery and research and development activities;

the timing of, and costs involved in, seeking and obtaining FDA and other regulatory approvals;

the continuation and success of our strategic alliance with GSK and future collaboration partners;

the exercise of the remaining option under the GSK agreement;

the costs of preparing, filing, prosecuting, maintaining and enforcing any patent claims and other intellectual property rights, including litigation costs and the results of such litigation;

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our ability to enter into additional collaboration, licensing, government or other arrangements and the terms and timing of such arrangements;

potential acquisition or in-licensing of other products or technologies; and

the emergence of competing technologies or other adverse market developments.

Future capital requirements will also depend on the extent to which we acquire or invest in additional complementary businesses, products and technologies. We currently have no understandings, commitments or agreements relating to any of these types of transactions.

Until we can generate a sufficient amount of product revenue to finance our cash requirements, which we may never do, we expect to finance future cash needs primarily through public or private equity offerings, debt financings, government grants and contracts and/or strategic collaborations. Additional financing may not be available to us when we need it or it may not be available on favorable terms, if at all. If we are unable to obtain adequate financing when needed, we may have to delay, reduce the scope of or eliminate one or more of our clinical trials or research and development programs or our commercialization efforts. We may be required to enter into collaborative partnerships for one or more of our drug candidate programs at an earlier stage of development or on less favorable terms, which may require us to relinquish rights to some of our drug candidates that we would otherwise have pursued on our own.

We may form additional strategic alliances in the future with respect to our independent programs, and we may not realize the benefits of such alliances.

We may form strategic alliances, create joint ventures or collaborations or enter into licensing arrangements with third parties with respect to our independent programs that we believe will complement or augment our existing business. For example, we plan to find a partner for co-development and commercialization of CCX140 and CCX662 outside North America upon completion of clinical development of CCX140 for the treatment of patients with diabetic nephropathy and CCX662 for the treatment of glioblastoma multiforme, or GBM. We face significant competition in seeking appropriate strategic partners and the negotiation process is time-consuming and complex. Moreover, we may not be successful in our efforts to establish a strategic partnership or other alternative arrangements for any future product candidates and programs because our research and development pipeline may be insufficient, our product candidates and programs may be deemed to be at too early of a stage of development for collaborative effort and/or third parties may not view our product candidates and programs as having the requisite potential to demonstrate safety and efficacy. We cannot be certain that, following a strategic transaction or license, we will achieve the revenues or specific net income that justifies such transaction. Any delays in entering into new strategic partnership agreements related to our product candidates could also delay the development and commercialization of our product candidates and reduce their competitiveness even if they reach the market.

Key elements of our proprietary suite of drug discovery technologies, known as EnabaLink, including our RAM screening technology, are new approaches to the discovery and development of new drug candidates and may not result in the discovery of any small molecule compounds of commercial value.

We must continue to identify and develop compounds that target the chemokine network and expand our portfolio of drug candidates. Research programs to identify new disease targets and drug candidates require substantial technical, financial and human resources. We have limited resources to study the more than 50 known chemokine ligands, as described in a February 2006 article in the New England Journal of Medicine, and approximately 25 identified chemokine receptors. EnabaLink represents a new approach to the development of new drug candidates (see Business Our Proprietary Drug Discovery Platform, EnabaLink) and we cannot assure you that EnabaLink will result in the discovery of new drug candidates. EnabaLink has only resulted in a limited number of clinical and preclinical stage programs to date, and we may not identify any therapeutic small molecule compounds of commercial value using EnabaLink or other commercially available drug discovery technologies.

If our Reverse Activation of Migration, or RAM, screening technology or any other screening technologies fail to identify highly specific hits that lead to the development of new drug candidates, our business may be

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materially and adversely affected. Our scientists may be unable to optimize the chemical hits identified by our RAM screening technology and develop the identified starting material into a candidate for further development that meets the desired product criteria. Our research and development programs may initially show promise in identifying chemokine receptors and their impact on the body s immune system, yet fail to yield drug candidates that are suitable for preclinical and clinical development. We cannot assure you that our current efforts will be successful or that we will not abandon any of our efforts in the future related to a particular chemokine receptor or small molecule program.

We rely on third party contract manufacturing organizations to manufacture and supply our drug candidates for us, other than Traficet-EN and CCX354 for which GSK has manufacturing responsibility. If one of our suppliers or manufacturers fails to perform adequately or fulfill our needs, we may be required to incur significant costs and devote significant efforts to find new suppliers or manufacturers. We may also face delays in the development and commercialization of our drug candidates.

Following GSK s exercise of its options for the further development of Traficet-EN and CCX354, it assumed sole manufacturing responsibility for those drug candidates and each of their two respective back-up compounds and we are no longer involved in their manufacture. We currently have limited experience in, and we do not own facilities for, manufacturing our other drug candidates. We rely upon third party contract manufacturing organizations to manufacture and supply larger quantities of these other drug candidates. The manufacture of pharmaceutical products in compliance with cGMPs requires significant expertise and capital investment, including the development of advanced manufacturing techniques and process controls. Manufacturers of pharmaceutical products often encounter difficulties in production, including difficulties with production costs and yields, quality control, including stability of the drug candidate and quality assurance testing, shortages of qualified personnel, as well as compliance with strictly enforced FDA current good manufacturing practice, or cGMP, requirements, other federal and state regulatory requirements, and foreign regulations. If our manufacturers were to encounter any of these difficulties or otherwise fail to comply with their obligations to us or under applicable regulations, our ability to provide study drugs in our preclinical studies and clinical trials would be jeopardized. Any delay or interruption in the supply of preclinical study or clinical trial materials could delay the completion of our preclinical studies and clinical trials, increase the costs associated with maintaining our preclinical study and clinical trial programs and, depending upon the period of delay, require us to commence new trials at significant additional expense or terminate the studies and trials completely.

All manufacturers of our drug candidates must comply with cGMP requirements enforced by the FDA through its facilities inspection program. These requirements include, among other things, quality control, quality assurance and the maintenance of records and documentation. Manufacturers of our component materials may be unable to comply with these cGMP requirements and with other FDA, state and foreign regulatory requirements. The FDA or similar foreign regulatory agencies at any time may also implement new standards, or change their interpretation and enforcement of existing standards for manufacture, packaging or testing of products. We have little control over our manufacturers compliance with these regulations and standards. A failure to comply with these requirements may result in fines and civil penalties, suspension of production, suspension or delay in product approval, product seizure or recall, or withdrawal of product approval. If the safety of any product supplied is compromised due to our manufacturers failure to adhere to applicable laws or for other reasons, we may not be able to obtain regulatory approval for or successfully commercialize our products, and we may be held liable for any injuries sustained as a result. Any of these factors could cause a delay of clinical trials, regulatory submissions, approvals or commercialization of our drug candidates or entail higher costs or impair our reputation.

We currently rely on a single source supplier for API for each of our drug candidates, other than Traficet-EN and CCX354 for which the responsibility for supplying the API and drug product has been assumed by GSK. IRIX Pharmaceuticals, Inc., currently manufactures the API for CCX140 and CCX168. Our current agreements with our suppliers do not provide for the entire supply of the API necessary for additional clinical trials or for full-scale commercialization. We have agreements with the University of Iowa Pharmaceuticals to manufacture the drug product for CCX140 and GSK to manufacture the drug product for CCX168. In the event that we and our suppliers cannot agree to the terms and conditions for them to provide some or all of our API clinical and

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commercial supply needs, or if any single source supplier terminates the agreement in response to a breach by us, we would not be able to manufacture the API on a commercial scale until a qualified alternative supplier is identified, which could also delay the development of, and impair our ability to commercialize, drug candidates.

Although alternative sources of supply exist, the number of third-party suppliers with the necessary manufacturing and regulatory expertise and facilities is limited, and it could be expensive and take a significant amount of time to arrange for alternative suppliers, which could have a material adverse effect on our business. New suppliers of any API would be required to qualify under applicable regulatory requirements and would need to have sufficient rights under applicable intellectual property laws to the method of manufacturing such ingredients. Obtaining the necessary FDA approvals or other qualifications under applicable regulatory requirements and ensuring non-infringement of third-party intellectual property rights could result in a significant interruption of supply and could require the new manufacturer to bear significant additional costs which may be passed on to us.

We currently have no sales and marketing staff or distribution organization. If we are unable to develop a sales and marketing and distribution capability on our own or through collaborations with GSK or other marketing partners, we will not be successful in commercializing our future products.

We currently have no sales, marketing or distribution capabilities or experience. If our products are approved for sale, we intend to rely on GSK to assist us in the marketing and distribution of our products for which GSK has exercised an option under our agreement, but there can be no assurance it will elect to market and distribute our products or that it will not terminate our collaboration arrangement. If GSK does not exercise its remaining option, we may need to enter into distribution or co-marketing arrangements with other third parties. To the extent we rely on third parties for marketing and distributing our approved products, any revenue we receive will depend upon the efforts of third parties, which may not be successful and are only partially within our control and our product revenue is likely to be lower than if we directly marketed or sold our products. GSK or other future collaborators may fail to develop or effectively commercialize our drug candidates because they cannot obtain necessary regulatory approvals, development or commercialization is not commercially reasonable, they elect to pursue competitive products outside of the collaboration; or for other reasons. If we are unable to enter into arrangements with third parties to commercialize the approved products on acceptable terms or at all, we may not be able to successfully commercialize our future products or we will have to market these products ourselves, which will be expensive and require us to build our own sales force, which we do not have experience doing. For example, we plan to retain commercial rights to CCX140 in North America and intend to build a small specialty sales force calling on nephrologists in North America. In addition, under our collaboration agreement with GSK, we have co-promotion rights with respect to certain drugs, but we do not have experience managing a sales force, selling drugs or marketing drugs. We cannot assure you we will be successful in any of these initiatives. If we are not successful in commercializing our future products, either on our own or through collaborations with GSK or one or more third parties, or co-promoting drugs with GSK, any future product revenue will be materially adversely affected.

We will need to increase the size of our organization, and we may experience difficulties in managing growth.

As of September 30, 2011, we had 64 full-time employees. We will need to continue to expand our managerial, operational, financial and other resources in order to manage our operations and clinical trials, continue our development activities and commercialize our drug candidates. Our management and personnel, systems and facilities currently in place may not be adequate to support this future growth. Our need to effectively execute our growth strategy requires that we:

- manage our clinical trials effectively, including our Phase II clinical trials for CCX140 and CCX168, which are being conducted at numerous trial sites throughout the world;

- manage our internal development efforts effectively while carrying out our contractual obligations to licensors, contractors, collaborators, government agencies and other third parties;

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continue to improve our operational, financial and management controls, reporting systems and procedures; and

identify, recruit, maintain, motivate and integrate additional employees.

We face substantial competition and our competitors may discover, develop or commercialize products faster or more successfully than us.

The biotechnology and pharmaceutical industries are highly competitive, and we face significant competition from companies in the pharmaceutical, biotechnology and other related markets that are researching and marketing products designed to address IBD, chronic kidney disease, including diabetic nephropathy, rheumatoid arthritis, other autoimmune diseases and inflammatory disorders, and cancer. Established pharmaceutical companies that currently sell or are developing drugs in our markets of interest include, for example, Abbott, Amgen, AstraZeneca, Biogen Idec, Bayer, Elan, GSK, Johnson & Johnson, Merck, Merck Serono, Takeda, Novartis, Pfizer, Reata, Sanofi-aventis and Teva. Many or all of these established competitors are also involved in research and drug development regarding various chemokine receptors. Pharmaceutical and biotechnology companies which are known to be involved in chemokine research and related drug development include Pfizer, GSK, Bristol-Myers Squibb, Merck, Takeda, Sanofi-aventis, Incyte, and UCB Pharma among others.

We are developing small molecule therapeutics that will compete with other drugs and alternative therapies that are currently marketed or are being developed to treat IBD, chronic kidney disease and diabetic nephropathy, rheumatoid arthritis, other autoimmune diseases and inflammatory disorders, and cancer. If approved for marketing by the FDA, Traficet-EN, our lead IBD drug candidate, would compete against existing IBD treatments such as Remicade, Humira, and other TNF-a inhibitors, immunomodulatory drugs and corticosteroids and potentially against other novel IBD drug candidates that are currently in development. Similarly, other future drug candidates we are pursuing would compete against numerous existing and established drugs and potentially against other novel drugs and therapies that are currently in development. See Business Competition. We also anticipate that we will face increased competition in the future as new companies enter into our target markets and scientific developments surrounding the chemokine system continue to develop.

Many of our competitors have materially greater name recognition and financial, manufacturing, marketing, research and drug development resources than we do. Additional mergers and acquisitions in the biotechnology and pharmaceutical industries may result in even more resources being concentrated in our competitors. Large pharmaceutical companies in particular have extensive expertise in preclinical and clinical testing and in obtaining regulatory approvals for drugs. In addition, academic institutions, government agencies, and other public and private organizations conducting research may seek patent protection with respect to potentially competitive products or technologies. These organizations may also establish exclusive collaborative or licensing relationships with our competitors.

We may be subject to costly product liability claims related to our clinical trials and drug candidates and, if we are unable to obtain adequate insurance or are required to pay for liabilities resulting from a claim excluded from, or beyond the limits of, our insurance coverage, a material liability claim could adversely affect our financial condition.

Because we conduct clinical trials with human patients, we face the risk that the use of our drug candidates may result in adverse side effects to patients and to otherwise healthy volunteers in our clinical trials. We face even greater risks upon any commercialization of our drug candidates. Although we have product liability insurance for clinical trials for up to $10.0 million, our insurance may be insufficient to reimburse us for any expenses or losses we may suffer, and we will be required to increase our product liability insurance coverage for our advanced clinical trials that we plan to initiate. We do not know whether we will be able to continue to obtain product liability coverage and obtain expanded coverage on acceptable terms, or at all. We may not have sufficient resources to pay for any liabilities resulting from a claim excluded from, or beyond the limits of, our

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insurance coverage. There is also a risk that third parties that we have agreed to indemnify could incur liability. An individual may bring a product liability claim against us if one of our drug candidates or products causes, or is claimed to have caused, an injury or is found to be unsuitable for consumer use. Any product liability claim brought against us, with or without merit, could result in:

- withdrawal of clinical trial volunteers, investigators, patients or trial sites;
- the inability to commercialize our drug candidates;
- decreased demand for our drug candidates;
- regulatory investigations that could require costly recalls or product modifications;
- loss of revenues;
- substantial costs of litigation;
- liabilities that substantially exceed our product liability insurance, which we would then be required to pay ourselves;
- an increase in our product liability insurance rates or the inability to maintain insurance coverage in the future on acceptable terms, if at all;
- the diversion of management s attention from our business; and
- damage to our reputation and the reputation of our products.

Our business involves the use of hazardous materials and we and our third-party manufacturers must comply with environmental laws and regulations, which may be expensive and restrict how we do business.

Our third-party manufacturers activities and our own activities involve the controlled storage, use and disposal of hazardous materials, including the components of our pharmaceutical products, test samples and reagents, biological materials and other hazardous compounds. We and our manufacturers are subject to federal, state and local and foreign laws and regulations governing the use, generation, manufacture, storage, handling and disposal of these hazardous materials. We currently carry no insurance specifically covering environmental claims relating to the use of hazardous materials. Although we believe that our safety procedures for handling and disposing of these materials and waste products comply with the standards prescribed by these laws and regulations, we cannot eliminate the risk of accidental injury or contamination from the use, storage, handling or disposal of hazardous materials. In the event of an accident, state or federal or other applicable authorities may curtail our use of these materials and/or interrupt our business operations. In addition, if an accident or environmental discharge occurs, or if we discover contamination caused by prior operations, including by prior owners and operators of properties we acquire, we could be liable for cleanup obligations, damages and fines. The substantial unexpected costs we may incur could significantly harm our financial condition and results of operations.

Future financings may adversely affect our stockholders or impose restrictions on our assets or operations, which may harm our business.

If we raise additional capital by issuing equity securities or convertible debt securities, then our existing stockholders ownership will be diluted and the terms of any new equity securities may have preferences over our common stock. If we raise additional capital through the issuance of debt securities, the debt will have rights senior to the holders of our common stock and may contain covenants that restrict our operational flexibility or impose liens or other restrictions on our assets. In addition, the terms of future financings may restrict our ability to raise additional capital, which would delay or prevent the further development or commercialization of our drug candidates.

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If we raise additional funds through collaboration, licensing or other similar arrangements, it may be necessary to relinquish potentially valuable rights to our current drug candidates, potential products or proprietary technologies, or grant licenses on terms that are not favorable to us. If adequate funds are not available, our ability to achieve profitability or to respond to competitive pressures would be significantly limited and we may be required to delay, significantly curtail or eliminate the development of one or more of our drug candidates.

We are highly dependent on the services of our founder, President and Chief Executive Officer, Dr. Thomas J. Schall, and if we are not able to retain Dr. Schall or other members of our management or recruit additional management, clinical and scientific personnel, our business will suffer.

We may not be able to attract or retain qualified management and scientific and clinical personnel in the future due to the intense competition for qualified personnel among biotechnology, pharmaceutical and other businesses, particularly in the San Francisco Bay area. Our industry has experienced a high rate of turnover of management personnel in recent years. If we are not able to attract, retain and motivate necessary personnel to accomplish our business objectives, we may experience constraints that will significantly impede the achievement of our development objectives, our ability to raise additional capital and our ability to implement our business strategy.

We are highly dependent on the principal members of our management and scientific staff. The loss of service of any of our management could harm our business. In addition, we are dependent on our continued ability to attract, retain and motivate highly qualified additional management, clinical and scientific personnel. The competition for qualified personnel in the pharmaceutical industry is intense. Due to our limited resources, we may not be able to effectively attract and recruit additional qualified personnel. If we are not able to retain our management, particularly our founder, President and Chief Executive Officer, Dr. Schall, and attract, on acceptable terms, additional qualified personnel necessary for the continued development of our business, we may not be able to sustain our operations or grow. Although we have executed employment agreements with each member of our current executive management team, including Dr. Schall, these agreements are terminable at will with or without notice and, therefore, we may not be able to retain their services as expected. In addition to the competition for personnel, the San Francisco Bay area in particular is characterized by a high cost of living. As such, we could have difficulty attracting experienced personnel to our company and may be required to expend significant financial resources in our employee recruitment and retention efforts.

In addition, we have scientific and clinical advisors who assist us in formulating our product development and clinical strategies. These advisors are not our employees and may have commitments to, or consulting or advisory contracts with, other entities that may limit their availability to us, or may have arrangements with other companies to assist in the development of products that may compete with ours.

Requirements associated with being a public company will increase our costs significantly, as well as divert significant company resources and management attention.

Prior to this offering, we have not been subject to the reporting requirements of the Securities Exchange Act of 1934, as amended, or the Exchange Act, or the other rules and regulations of the SEC or any securities exchange relating to public companies. We are working with our legal, independent accounting and financial advisors to identify those areas in which changes should be made to our financial and management control systems to manage our growth and our obligations as a public company. These areas include corporate governance, corporate control, internal audit, disclosure controls and procedures and financial reporting and accounting systems. We have made, and will continue to make, changes in these and other areas. However, the expenses that will be required in order to adequately prepare for being a public company could be material. Compliance with the various reporting and other requirements applicable to public companies will also require considerable time and attention of management. In addition, the changes we make may not be sufficient to allow us to satisfy our obligations as a public company on a timely basis.

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In addition, being a public company could make it more difficult or more costly for us to obtain certain types of insurance, including directors and officers liability insurance, and we may be forced to accept reduced policy limits and coverage or incur substantially higher costs to obtain the same or similar coverage. The impact of these events could also make it more difficult for us to attract and retain qualified persons to serve on our board of directors, our board committees or as executive officers.

If we are not able to implement the requirements of Section 404 of the Sarbanes-Oxley Act of 2002 in a timely manner or with adequate compliance, we may be subject to sanctions by regulatory authorities.

Section 404 of the Sarbanes-Oxley Act of 2002 requires that we evaluate and determine the effectiveness of our internal controls over financial reporting and beginning with our annual report for fiscal 2013 provide a management report on the internal control over financial reporting. If we have a material weakness in our internal controls over financial reporting, we may not detect errors on a timely basis and our consolidated financial statements may be materially misstated. We will be evaluating our internal controls systems to allow management to report on, and our independent auditors to attest to, our internal controls. We will be performing the system and process evaluation and testing (and any necessary remediation) required to comply with the management certification and auditor attestation requirements of Section 404 of the Sarbanes-Oxley Act of 2002. We cannot be certain as to the timing of completion of our evaluation, testing and remediation actions or the impact of the same on our operations. If we are not able to implement the requirements of Section 404 in a timely manner or with adequate compliance, we may be subject to sanctions or investigation by regulatory authorities, such as the Securities and Exchange Commission, or SEC, or The Nasdaq Stock Market LLC, or Nasdaq. Any such action could adversely affect our financial results or investors confidence in us and could cause our stock price to fall. Moreover, if we are not able to comply with the requirements of Section 404 in a timely manner, or if we or our independent registered public accounting firm identifies deficiencies in our internal controls that are deemed to be material weaknesses, we could be subject to sanctions or investigations by Nasdaq, the SEC or other regulatory authorities, which would entail expenditure of additional financial and management resources and could materially adversely affect our stock price.

We may be adversely affected by the current economic environment.

Our ability to attract and retain collaboration partners or customers, invest in and grow our business and meet our financial obligations depends on our operating and financial performance, which, in turn, is subject to numerous factors, including the prevailing economic conditions and financial, business and other factors beyond our control, such as the rate of unemployment, the number of uninsured persons in the United States and inflationary pressures. We cannot anticipate all the ways in which the current economic climate and financial market conditions could adversely impact our business.

We are exposed to risks associated with reduced profitability and the potential financial instability of our collaboration partners or customers, many of which may be adversely affected by volatile conditions in the financial markets. For example, unemployment and underemployment, and the resultant loss of insurance, may decrease the demand for healthcare services and pharmaceuticals. If fewer patients are seeking medical care because they do not have insurance coverage, our collaboration partners or customers may experience reductions in revenues, profitability and/or cash flow that could lead them to reduce their support of our programs or financing activities. If collaboration partners or customers are not successful in generating sufficient revenue or are precluded from securing financing, they may not be able to pay, or may delay payment of, accounts receivable that are owed to us. This, in turn, could adversely affect our financial condition and liquidity. In addition, if economic challenges in the United States result in widespread and prolonged unemployment, either regionally or on a national basis, prior to the effectiveness of certain provisions of the Patient Protection and Affordable Care Act, as amended by the Health Care and Education Affordability Reconciliation Act, collectively known as the Affordable Care Act, a substantial number of people may become uninsured or underinsured. To the extent economic challenges result in fewer individuals pursuing or being able to afford our products once commercialized, our business, results of operations, financial condition and cash flows could be adversely affected.

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Our internal computer systems, or those of our CROs or other contractors or consultants, may fail or suffer security breaches, which could result in a material disruption of our drug development programs.

Despite the implementation of security measures, our internal computer systems and those of our CROs and other contractors and consultants are vulnerable to damage from computer viruses, unauthorized access, natural disasters, terrorism, war and telecommunication and electrical failures. While we have not experienced any such system failure, accident or security breach to date, if such an event were to occur and cause interruptions in our operations, it could result in a material disruption of our drug development programs. For example, the loss of clinical trial data from completed or ongoing clinical trials for any of our drug candidates could result in delays in our regulatory approval efforts and significantly increase our costs to recover or reproduce the data. To the extent that any disruption or security breach were to result in a loss of or damage to our data or applications, or inappropriate disclosure of confidential or proprietary information, we could incur liability and the further development of our drug candidates could be delayed.

Our employees may engage in misconduct or other improper activities, including noncompliance with regulatory standards and requirements.

We are exposed to the risk of employee fraud or other misconduct. Misconduct by employees could include intentional failures to comply with FDA regulations, provide accurate information to the FDA, comply with manufacturing standards we have established, comply with federal and state healthcare fraud and abuse laws and regulations, report financial information or data accurately or disclose unauthorized activities to us. In particular, sales, marketing and business arrangements in the healthcare industry are subject to extensive laws and regulations intended to prevent fraud, kickbacks, self-dealing and other abusive practices. These laws and regulations may restrict or prohibit a wide range of pricing, discounting, marketing and promotion, sales commission, customer incentive programs and other business arrangements. Employee misconduct could also involve the improper use of information obtained in the course of clinical trials, which could result in regulatory sanctions and serious harm to our reputation. It is not always possible to identify and deter employee misconduct, and the precautions we take to detect and prevent this activity may not be effective in controlling unknown or unmanaged risks or losses or in protecting us from governmental investigations or other actions or lawsuits stemming from a failure to be in compliance with such laws or regulations. If any such actions are instituted against us, and we are not successful in defending ourselves or asserting our rights, those actions could have a significant impact on our business, including the imposition of significant fines or other sanctions.

Our ability to utilize our net operating loss carryforwards and certain other tax attributes may be limited.

Under Section 382 of the Internal Revenue Code, if a corporation undergoes an ownership change (generally defined as a greater than 50% change, by value, in its equity ownership over a three year period), the corporation s ability to use its pre-change net operating loss carryforwards and other pre-change tax attributes to offset its post-change taxable income and taxes may be limited. We previously determined that we had ownership changes that occurred in July 1999 and June 2004, which limit our ability to use our then existing tax attributes. We have not yet determined whether, as a result of our initial public offering and other transactions that have occurred over the past three years, we have experienced or may, upon completion of this offering, experience an additional ownership change. In addition, future changes in our stock ownership, many of the causes of which are outside our control, could result in an ownership change. Any such ownership changes could further limit our ability to use net operating loss carryforwards and other pre-change tax attributes.

Business disruptions could seriously harm our future revenues and financial condition and increase our costs and expenses.

Our operations could be subject to earthquakes, power shortages, telecommunications failures, floods, hurricanes, typhoons, fires, extreme weather conditions, medical epidemics and other natural or manmade disasters or business interruptions. The occurrence of any of these business disruptions could seriously harm our operations and financial condition and increase our costs and expenses. Our corporate headquarters is located in California and certain clinical sites for our drug candidates, operations of our existing and future partners and

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suppliers are or will be located in California near major earthquake faults and fire zones. The ultimate impact on us, our significant partners, suppliers and our general infrastructure of being located near major earthquake faults and fire zones and being consolidated in certain geographical areas is unknown, but our operations and financial condition could suffer in the event of a major earthquake, fire or other natural or manmade disaster.

Risks Related to Intellectual Property

We may have to license rights from Millennium Pharmaceuticals, Inc. or engage in patent litigation in order to secure the rights necessary to commercialize Traficet-EN. Patent litigation could absorb significant management time and financial resources, and, if we do not prevail, could have a material adverse effect on our ability to derive revenues from our agreement with GSK.

Millennium Pharmaceuticals, Inc., or Millennium, has obtained certain U.S. patents which include claims to small molecules that modulate CCR9, compositions thereof, and methods of using them to treat conditions such as IBD. We became aware of Millennium s CCR9-related patent applications during our own routine patent and patent literature review. Millennium, which was acquired by Takeda Pharmaceutical Company Limited, or Takeda, in May 2008 and is currently a wholly owned subsidiary of Takeda, may contend that the claims of these patents cover our patented Traficet-EN drug candidate. We believe that our activities related to Traficet-EN are currently exempt from patent infringement liability because these activities are strictly limited to obtaining information for regulatory approval. However, if and when our Traficet-EN related activities extend beyond those related to seeking regulatory approval, such as, for example, if and when we commercialize Traficet-EN, Millennium might then commence an infringement action against us based on these patents and/or other related patents that it may be granted in the future. If Millennium elects to sue us, we believe that we may have viable defenses to any such infringement suit. However, we cannot assure you that the relevant court would find in our favor with respect to such defenses. Intellectual property litigation and patent litigation in particular, is expensive, complex and lengthy and its outcome is difficult to predict. A court could enter orders that temporarily, preliminarily or permanently enjoin us or our strategic partners from using, selling, offering to sell or importing out current or future drug candidates or could enter an order mandating that we undertake certain remedial activities. During 2005, we did engage in preliminary discussions with Millennium regarding potentially collaborating with respect to CCR9, given that both we and Millennium have patents relating to CCR9. However, these discussions were general in nature and did not progress beyond the preliminary stage. Other than these preliminary discussions, we have not had any conversations or contacts with Millennium relating to CCR9. Under our agreement with GSK, GSK has the right, but not the obligation, to defend against third party patent infringement claims for licensed drugs. If GSK elects to defend against any such claims, it has the sole right to direct the defense of such claims and settle such claims at its own cost and expense. If GSK elects not to defend against such claims, we have the right, but not the obligation, to defend against such claims.

We may also be subject to negative publicity due to litigation. Pending or future patent litigation against us or any strategic partners by Millennium or anyone else may force us or any strategic partners to stop or delay developing, manufacturing or selling potential drug candidates that are claimed to infringe a third party s intellectual property, unless that party grants us or any strategic partners rights to use its intellectual property. If Millennium is able to obtain an injunction and neither we nor our strategic partners are able to obtain a license, both we and our strategic partners would be precluded from the manufacture and sale of Traficet-EN. U.S. patents are entitled to a presumption of validity and the burden of proving invalidity would be heavily weighted against us. Specifically, we would be required to show by clear and convincing evidence that Millennium s patents are invalid. Such decisions on patent validity often favor the patent owner because of the presumption of validity. If we or our strategic partners are unable to show that Millennium s patent is invalid and neither we nor our strategic partners are able to obtain a license from Millennium for the use of their intellectual property at all or on commercially acceptable terms, this would preclude both us and our strategic partners from the manufacture and sale of Traficet-EN or related candidate compounds found to be covered by Millennium s patent claims. If we are able to obtain a license from Millennium, we will be solely responsible for all fees required to be paid to Millennium in connection with such license and GSK will bear no responsibility for such license fees. See Business Intellectual Property.

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The cost to us of any patent litigation or other proceedings, such as interference proceedings, which are meant to determine who first invented any of the claims covered by the patent even if resolved in our favor, could be substantial. Some of our competitors may be able to sustain the costs of such litigation or proceedings more effectively than we can because of their substantially greater financial resources. Discovery proceedings in the United States might lead to the disclosure of some of our proprietary confidential information. Uncertainties resulting from the initiation and continuation of patent litigation or other proceedings could have a material adverse effect on our ability to compete in the marketplace. Patent litigation and other proceedings may also absorb significant management and technical staff s time which may materially and adversely impact our financial position and results of operations.

Our proprietary rights may not adequately protect our technologies and drug candidates. If we are unable to protect our drug candidates and our intellectual property rights, it may materially adversely affect our position in the market.

Our commercial success will depend on our ability to obtain patents and maintain adequate protection for our technologies, intellectual property and drug candidates in the United States and other countries. Our patent estate, on a worldwide basis, includes approximately 390 issued or allowed patents and approximately 325 pending patent applications, with claims relating to all of our current clinical stage drug candidates. With respect to our lead drug candidates in the CCR1, CCR2 and CCR9 programs, we have approximately 100 issued or allowed patents worldwide relating to their chemical composition or use thereof. There are approximately 40 patent applications pending for our other clinical stage compounds in the C5aR and ChemR23 programs. We have approximately 180 issued patents relating to other small molecule compounds and approximately 60 issued patents relating to our novel biological discoveries. We also have approximately 50 issued patents relating to our proprietary screening and drug development technologies. We cannot assure you that any of our patent applications will result in issued patents. We will be able to protect our proprietary rights from unauthorized use by third parties only to the extent that our proprietary technologies and future products are covered by valid and enforceable patents or are effectively maintained as trade secrets. If third parties disclose or misappropriate our proprietary rights, it may materially and adversely impact our position in the market.

We apply for patents covering both our technologies and drug candidates, as we deem appropriate. However, we may fail to apply for patents on important technologies or drug candidates in a timely fashion, or at all. Our existing patents and any future patents we obtain may not be sufficiently broad to prevent others from using our technologies or from developing competing products and technologies. Moreover, the patent positions of numerous biotechnology and pharmaceutical companies are highly uncertain and involve complex legal and factual questions for which important legal principles remain unresolved. As a result, the validity and enforceability of our patents cannot be predicted with certainty. In addition, we cannot assure you that:

- we were the first to make the inventions covered by each of our issued patents and pending patent applications;
- we were the first to file patent applications for these inventions;
- others will not independently develop similar or alternative technologies or duplicate any of our technologies by inventing around our claims;
- any of our pending patent applications will result in issued patents;
- a third party will not challenge our proprietary rights or that a court will hold that our patents are valid and enforceable;
- any patents issued to us or our collaboration partners will provide us with any competitive advantages, or will not be challenged by third parties;
- we will develop additional proprietary technologies that are patentable; or

the patents of others will not have an adverse effect on our business.

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In addition, there are numerous recent changes to the patent laws and proposed changes to the rules of the U.S. Patent and Trademark Office, or USPTO, which may have a significant impact on our ability to protect our technology and enforce our intellectual property rights. For example, on September 16, 2011, President Obama signed the America Invents Act which codifies several significant changes to the U.S. patent laws, including, among other things, changing from a first to invent to a first inventor to file system, limiting where a patentee may file a patent suit, requiring the apportionment of patent damages, replacing interference proceedings with derivation actions, and creating a post-grant opposition process to challenge patents after they have issued. The effects of these changes are currently unclear as the USPTO must still implement various regulations, the courts have yet to address any of these provisions, and the applicability of the act and new regulations on specific patents discussed herein have not been determined and would need to be reviewed.

We rely upon unpatented trade secrets, unpatented know-how and continuing technological innovation to develop and maintain our competitive position, which we seek to protect, in part, by confidentiality agreements with our employees and our collaborators and consultants. We also have agreements with our employees and selected consultants that obligate them to assign their inventions to us. However, it is possible that technology relevant to our business will be independently developed by a person that is not a party to such an agreement. Furthermore, if the employees and consultants that are parties to these agreements breach or violate the terms of these agreements, we may not have adequate remedies for any such breach or violation, and we could lose our trade secrets through such breaches or violations. Further, our trade secrets could otherwise become known or be independently discovered by our competitors. In addition, intellectual property laws in foreign countries may not protect our intellectual property to the same extent as the laws of the United States.

We may become subject to third parties claims alleging infringement of patents and proprietary rights or seeking to invalidate our patents or proprietary rights, which would be costly, time consuming and, if successfully asserted against us, delay or prevent the development and commercialization of our products.

There has been substantial litigation and other proceedings regarding patent and other intellectual property rights in the pharmaceutical and biotechnology industries. We may be subject to third-party claims in the future against us or our collaborators that would cause us to incur substantial expenses and, if successful against us, could cause us to pay substantial damages, including treble damages and attorney s fees if we are found to be willfully infringing a third party s patents. Further, if a patent infringement suit were brought against us or our collaborators, we or they could be forced to stop or delay research, development, manufacturing or sales of the product or drug candidate that is the subject of the suit. As a result of patent infringement claims, or in order to avoid potential claims, we or our collaborators may choose to seek, or be required to seek, a license from the third party and would most likely be required to pay license fees or royalties or both. These licenses may not be available on acceptable terms, or at all. Even if we or our collaborators were able to obtain a license, the rights may be nonexclusive, which would give our competitors access to the same intellectual property. Ultimately, we could be prevented from commercializing a product, or forced to redesign it, or to cease some aspect of our business operations if, as a result of actual or threatened patent infringement claims, we or our collaborators are unable to enter into licenses on acceptable terms. This could harm our business significantly.

In addition to infringement claims against us, if third parties prepare and file patent applications in the United States that also claim technology to which we have rights, we may have to participate in interference proceedings in the USPTO to determine the priority of invention. We may also become involved in similar opposition proceedings in the European Patent Office regarding our intellectual property rights with respect to our products and technology.

Restrictions on our patent rights relating to our drug candidates may limit our ability to prevent third parties from competing against us.

Our success will depend, in part, on our ability to obtain and maintain patent protection for our drug candidates, preserve our trade secrets, prevent third parties from infringing upon our proprietary rights and operate without infringing upon the proprietary rights of others. Composition-of-matter patents on APIs are

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generally considered to be the strongest form of intellectual property protection for pharmaceutical products as they apply without regard to any method of use. Entirely new individual chemical compounds, often referred to as new chemical entities, are typically entitled to composition-of-matter coverage. However, we cannot be certain that the current law will remain the same, or that our drug candidates will be considered novel and non-obvious by the USPTO and courts.

In addition to composition-of-matter patents and patent applications, we also have filed method-of-use patent applications. This type of patent protects the use of the product only for the specified method. However, this type of patent does not prevent a competitor from making and marketing a product that is identical to our product for an indication that is outside the scope of the patented method. Moreover, even if these competitors do not actively promote their product for our targeted indication, physicians may prescribe these products off-label. Although off-label prescriptions may infringe or contribute to the infringement of method-of-use patents, the practice is common and such infringement is difficult to prevent or prosecute.

Patent applications in the United States and most other countries are confidential for a period of time until they are published, and publication of discoveries in scientific or patent literature typically lags actual discoveries by several months or more. As a result, we cannot be certain that we and the inventors of the issued patents and applications that we may in-license were the first to conceive of the inventions covered by such patents and pending patent applications or that we and those inventors were the first to file patent applications covering such inventions. Also, we have numerous issued patents and some patent applications pending before the USPTO and the patent protection may lapse before we manage to obtain commercial value from them, which might result in increased competition and materially affect our position in the market.

We may be subject to claims that we or our employees or consultants have wrongfully used or disclosed alleged trade secrets of our employees or consultants former employers or their clients. These claims may be costly to defend and if we do not successfully do so, we may be required to pay monetary damages and may lose valuable intellectual property rights or personnel.

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

Some of our intellectual property which is discovered through government funded programs is subject to federal regulation such as march-in rights, certain reporting requirements, and a preference for U.S. industry. Compliance with such regulations may limit our exclusive rights, subject us to expenditure of resources with respect to reporting requirements, and limit our ability to contract with foreign manufacturers.

Some of our existing drug candidates, including CCX140, and some of our research and development work were funded, at least in part, by the U.S. government and are therefore subject to certain federal regulations. For example, some of our research and development work on vaccine adjuvants and immunomodulation for biothreat applications was funded by government research grants. In addition, as noted on several of our patents including U.S. Patent Nos. 7,884,110; 7,622,583; 7,776,877; and 7,683,176, inventions covering various CCR9 and CCR2 antagonists were supported at least in part by NIH funding (U19-AI056690-01). Under the march-in provisions of the Bayh-Dole Act, the government may have the right under limited circumstances to require us to grant exclusive, partially exclusive or non-exclusive rights to third parties for intellectual property discovered through the government funded program. The government can exercise its march-in rights if it determines that action is necessary because we fail to achieve practical application of the new invention or because action is necessary to alleviate health or safety needs of the

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public. Intellectual property discovered under the government funded program is also subject to certain reporting requirements, compliance with which may require us to expend substantial resources. Such intellectual property is also subject to a preference for U.S. industry, which may limit our ability to contract with foreign product manufacturers for products covered by such intellectual property. We plan to apply for additional U.S. government funding, and it is possible that we may discover compounds or drug candidates as a result of such funding. Intellectual property under such discoveries would be subject to the applicable provisions of the Bayh-Dole Act.

Risks Related to Government Regulation

The regulatory approval process is expensive, time consuming and uncertain and may prevent us or our collaboration partners from obtaining approvals for the commercialization of some or all of our drug candidates.

The research, testing, manufacturing, labeling, approval, selling, import, export, marketing and distribution of drug products are subject to extensive regulation by the FDA and other regulatory authorities in the United States and other countries, which regulations differ from country to country. Neither we nor our collaboration partners are permitted to market our drug candidates in the United States until we receive approval of an NDA from the FDA. Neither we nor our collaboration partners have submitted an application for or received marketing approval for any of our drug candidates. Obtaining approval of an NDA can be a lengthy, expensive and uncertain process. In addition, failure to comply with FDA and other applicable U.S. and foreign regulatory requirements may subject us to administrative or judicially imposed sanctions, including:

warning letters;

civil and criminal penalties;

injunctions;

withdrawal of approved products;

product seizure or detention;

product recalls;

total or partial suspension of production; and

refusal to approve pending NDAs or supplements to approved NDAs.

Prior to receiving approval to commercialize any of our drug candidates in the United States or abroad, we and our collaboration partners must demonstrate with substantial evidence from well controlled clinical trials, and to the satisfaction of the FDA and other regulatory authorities abroad, that such drug candidates are safe and effective for their intended uses. Results from preclinical studies and clinical trials can be interpreted in different ways. Even if we and our collaboration partners believe the preclinical or clinical data for our drug candidates are promising, such data may not be sufficient to support approval by the FDA and other regulatory authorities. Administering any of our drug candidates to humans may produce undesirable side effects, which could interrupt, delay or halt clinical trials of our drug candidates and result in the FDA or other regulatory authorities denying approval of our drug candidates for any or all targeted indications.

Regulatory approval of an NDA or NDA supplement is not guaranteed, and the approval process is expensive and may take several years. The FDA also has substantial discretion in the approval process. Despite the time and expense exerted, failure can occur at any stage, and we could encounter problems that cause us to abandon or repeat clinical trials, or perform additional preclinical studies and clinical trials. The number of preclinical studies and clinical trials that will be required for FDA approval varies depending on the drug candidate, the disease or condition that

the drug candidate is designed to address, and the regulations applicable to any particular drug candidate. The FDA can delay, limit or deny approval of a drug candidate for many reasons, including, but not limited to, the following:

a drug candidate may not be deemed safe or effective;

FDA officials may not find the data from preclinical studies and clinical trials sufficient;

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the FDA might not approve our or our third party manufacturer s processes or facilities; or

the FDA may change its approval policies or adopt new regulations.

If any of our drug candidates fails to demonstrate safety and efficacy in clinical trials or does not gain regulatory approval, our business and results of operations will be materially and adversely harmed.

Even if we receive regulatory approval for a drug candidate, we will be subject to ongoing regulatory obligations and continued regulatory review, which may result in significant additional expense and subject us to penalties if we fail to comply with applicable regulatory requirements.

Once regulatory approval has been granted, the approved product and its manufacturer are subject to continual review by the FDA and/or non-U.S. regulatory authorities. Any regulatory approval that we or our collaboration partners receive for our drug candidates may be subject to limitations on the indicated uses for which the product may be marketed or contain requirements for potentially costly post-marketing follow-up studies to monitor the safety and efficacy of the product. In addition, if the FDA and/or non-U.S. regulatory authorities approve any of our drug candidates, we will be subject to extensive and ongoing regulatory requirements by the FDA and other regulatory authorities with regard to the labeling, packaging, adverse event reporting, storage, advertising, promotion and recordkeeping for our products. In addition, manufacturers of our drug products are required to comply with current cGMP regulations, which include requirements related to quality control and quality assurance as well as the corresponding maintenance of records and documentation. Further, regulatory authorities must approve these manufacturing facilities before they can be used to manufacture our drug products, and these facilities are subject to continual review and periodic inspections by the FDA and other regulatory authorities for compliance with cGMP regulations. If we or a regulatory authority discovers previously unknown problems with a product, such as adverse events of unanticipated severity or frequency, or problems with the facility where the product is manufactured, a regulatory authority may impose restrictions on that product, the manufacturer or us, including requiring withdrawal of the product from the market or suspension of manufacturing. If we, our drug candidates or the manufacturing facilities for our drug candidates fail to comply with regulatory requirements of the FDA and/or other non-U.S. regulatory authorities, we could be subject to administrative or judicially imposed sanctions, including:

warning letters;

civil or criminal penalties;

injunctions;

suspension of or withdrawal of regulatory approval;

suspension of any ongoing clinical trials;

voluntary or mandatory product recalls and publicity requirements;

refusal to approve pending applications for marketing approval of new drugs or supplements to approved applications filed by us;

restrictions on operations, including costly new manufacturing requirements; or

seizure or detention of our products or import bans.

The regulatory requirements and policies may change and additional government regulations may be enacted for which we may also be required to comply. We cannot predict the likelihood, nature or extent of government regulation that may arise from future legislation or administrative action, either in the United States or in other countries. If we are not able to maintain regulatory compliance, we will not be permitted to market our future products and our business will suffer.

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The availability of adequate third-party coverage and reimbursement for newly approved drugs is uncertain, and failure to obtain adequate coverage and reimbursement from third-party payors could impede our ability to market any future products we may develop and could limit our ability to generate revenue.

There is significant uncertainty related to the third-party payor coverage and reimbursement of newly approved drugs. The commercial success of our future products in both domestic and international markets depends on whether such third-party coverage and reimbursement is available for our future products. Governmental payors, including Medicare and Medicaid, health maintenance organizations and other third-party payors are increasingly attempting to manage their healthcare expenditures by limiting both coverage and the level of reimbursement of new drugs and, as a result, they may not cover or provide adequate reimbursement for our future products. These payors may not view our future products as cost-effective, and coverage and reimbursement may not be available to our customers or may not be sufficient to allow our future products to be marketed on a competitive basis. Third-party payors are exerting increasing influence on decisions regarding the use of, and coverage and reimbursement levels for, particular treatments. Such third-party payors, including Medicare, are challenging the prices charged for medical products and services, and many third-party payors limit or delay coverage and reimbursement for newly approved healthcare products. In particular, third-party payors may limit the covered indications. Cost-control initiatives could cause us to decrease the price we might establish for products, which could result in lower than anticipated product revenues. If the prices for our drug candidates decrease or if governmental and other third-party payors do not provide adequate coverage or reimbursement, our prospects for revenue and profitability will suffer.

Failure to obtain regulatory approvals in foreign jurisdictions will prevent us from marketing our products internationally.

We intend to seek a distribution and marketing partner for CCX140 outside North America and may market future products in international markets. In order to market our future products in the EEA (which is comprised of the 27 Member States of the EU plus Norway, Iceland and Liechtenstein) and many other foreign jurisdictions, we must obtain separate regulatory approvals. More concretely, in the EEA, medicinal products can only be commercialized after obtaining a Marketing Authorization, or MA. There are two types of marketing authorizations:

- The Community MA, which is issued by the European Commission through the Centralized Procedure, based on the opinion of the Committee for Medicinal Products for Human Use of the European Medicines Agency, or EMA, and which is valid throughout the entire territory of the EEA. The Centralized Procedure is mandatory for certain types of products, such as biotechnology medicinal products, orphan medicinal products, and medicinal products indicated for the treatment of AIDS, cancer, neurodegenerative disorders, diabetes, auto-immune and viral diseases. The Centralized Procedure is optional for products containing a new active substance not yet authorized in the EEA, or for products that constitute a significant therapeutic, scientific or technical innovation or which are in the interest of public health in the EU.

- National MAs, which are issued by the competent authorities of the Member States of the EEA and only cover their respective territory, are available for products not falling within the mandatory scope of the Centralized Procedure. Where a product has already been authorized for marketing in a Member State of the EEA, this National MA can be recognized in another Member States through the Mutual Recognition Procedure. If the product has not received a National MA in any Member State at the time of application, it can be approved simultaneously in various Member States through the Decentralized Procedure.

Under the above described procedures, before granting the MA, the EMA or the competent authorities of the Member States of the EEA make an assessment of the risk-benefit balance of the product on the basis of scientific criteria concerning its quality, safety and efficacy.

We have had limited interactions with foreign regulatory authorities, and the approval procedures vary among countries and can involve additional clinical testing, and the time required to obtain approval may differ

from that required to obtain FDA approval. Clinical trials conducted in one country may not be accepted by regulatory authorities in other countries. Approval by the FDA does not ensure approval by regulatory authorities in other countries, and approval by one or more foreign regulatory authorities does not ensure approval by regulatory authorities in other foreign countries or by the FDA. However, a failure or delay in obtaining regulatory approval in one country may have a negative effect on the regulatory process in others. The foreign regulatory approval process may include all of the risks associated with obtaining FDA approval. We may not obtain foreign regulatory approvals on a timely basis, if at all. We may not be able to file for regulatory approvals and even if we file we may not receive necessary approvals to commercialize our products in any market.

Healthcare reform measures could hinder or prevent our drug candidates commercial success.

In the United States, there have been and we expect there will continue to be a number of legislative and regulatory changes to the healthcare system in ways that could affect our future revenues and profitability and the future revenues and profitability of our potential customers. Federal and state lawmakers regularly propose and, at times, enact legislation that would result in significant changes to the healthcare system, some of which are intended to contain or reduce the costs of medical products and services. For example, in March 2010, the President signed one of the most significant healthcare reform measures in decades, the Affordable Care Act. It contains a number of provisions, including those governing enrollment in federal healthcare programs, reimbursement changes and fraud and abuse measures, all of which will impact existing government healthcare programs and will result in the development of new programs. The Affordable Care Act, among other things:

- imposes a non-deductible annual fee on pharmaceutical manufacturers or importers who sell branded prescription drugs, effective 2011;
- increases the minimum level of Medicaid rebates payable by manufacturers of brand-name drugs from 15.1% to 23.1%;
- requires collection of rebates for drugs paid by Medicaid managed care organizations;
- requires manufacturers to participate in a coverage gap discount program, under which they must agree to offer 50% point-of-sale discounts off negotiated prices of applicable brand drugs to eligible beneficiaries during their coverage gap period, as a condition for the manufacturer s outpatient drugs to be covered under Medicare Part D, beginning January 2011; and
- mandates a further shift in the burden of Medicaid payments to the states.

A number of states have challenged the constitutionality of certain provisions of the Affordable Care Act, and many of these challenges are still pending final adjudication in several jurisdictions as well as the United States Supreme Court. Congress has also proposed a number of legislative initiatives, including possible repeal of the Affordable Care Act. At this time, it remains unclear whether there will be any changes made to the Affordable Care Act, whether to certain provisions or its entirety. We cannot assure you that the Affordable Care Act, as currently enacted or as amended in the future, will not adversely affect our business and financial results and we cannot predict how future federal or state legislative or administrative changes relating to healthcare reform will affect our business.

In addition, other legislative changes have been proposed and adopted since the Affordable Care Act was enacted. Most recently, on August 2, 2011, the President signed into law the Budget Control Act of 2011, which, among other things, creates the Joint Select Committee on Deficit Reduction to recommend proposals in spending reductions to Congress. In the event that the Joint Select Committee is unable to achieve a targeted deficit reduction of at least $1.2 trillion for the years 2013 through 2021, or Congress does not act on the committee s recommendation, without amendment, by December 23, 2011, an automatic reduction is triggered. These automatic cuts would be made to several government programs and, with respect to Medicare, would include aggregate reductions to Medicare payments to providers of up to 2% per fiscal year, starting in 2013. More recently, on September 19, 2011, President Obama presented his Plan for Economic Growth and Deficit Reduction to the Joint Select Committee, which includes $248 billion in Medicare savings ($240 billion of which comes from reducing and collecting Medicare payments incorrectly paid) and $72 billion in Medicaid

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savings. Beginning in 2017, the President s proposal also shifts more of the Medicare costs to newly enrolled beneficiaries, including an increase in patient deductibles under Medicare Part B for certain beneficiaries, and increases Part B and Part D premiums for higher-income beneficiaries.

There likely will continue to be legislative and regulatory proposals at the federal and state levels directed at containing or lowering the cost of health care. We cannot predict the initiatives that may be adopted in the future or their full impact. The continuing efforts of the government, insurance companies, managed care organizations and other payors of healthcare services to contain or reduce costs of health care may adversely affect:

- our ability to set a price we believe is fair for our products;
- our ability to generate revenues and achieve or maintain profitability; and
- the availability of capital.

A number of states have challenged the constitutionality of certain provisions of the Affordable Care Act, and many of these challenges are still pending final adjudication in several jurisdictions. Congress has also proposed a number of legislative initiatives, including possible repeal of the Affordable Care Act. At this time, it remains unclear whether there will be any changes made to the Affordable Care Act, whether to certain provisions or its entirety. We cannot assure you that the Affordable Care Act, as currently enacted or as amended in the future, will not adversely affect our business and financial results.

Further, changes in regulatory requirements and guidance may occur and we may need to amend clinical trial protocols to reflect these changes. Amendments may require us to resubmit our clinical trial protocols to IRBs for reexamination, which may impact the costs, timing or successful completion of a clinical trial. In light of widely publicized events concerning the safety risk of certain drug products, regulatory authorities, members of Congress, the Governmental Accounting 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. The increased attention to drug safety issues may result in a more cautious approach by the FDA to clinical trials and the drug approval process. Data from clinical trials may receive greater scrutiny with respect to safety, which may make the FDA or other regulatory authorities more likely to terminate clinical trials before completion, or require longer or additional clinical trials that may result in substantial additional expense and a delay or failure in obtaining approval or approval for a more limited indication than originally sought.

Heightened Congressional scrutiny on the adequacy of the FDA s drug approval process and the agency s efforts to assure the safety of marketed drugs resulted in the enactment of legislation addressing drug safety issues, the FDA Amendments Act of 2007. This legislation provided the FDA with expanded authority over drug products after approval, and the FDA s exercise of this authority has resulted in delays or increased costs during the period of product development, clinical trials and regulatory review and approval, and increased costs to assure compliance with new post-approval regulatory requirements. Given the serious public health risks of high profile adverse safety events with certain drug products, the FDA may require, as a condition of approval, costly risk management programs which may include safety surveillance, restricted distribution and use, patient education, enhanced labeling, special packaging or labeling, expedited reporting of certain adverse events, preapproval of promotional materials and restrictions on direct-to-consumer advertising.

If we fail to comply with healthcare regulations, we could face substantial penalties and our business, operations and financial condition could be adversely affected.

Even though we do not and will not control referrals of healthcare services or bill directly to Medicare, Medicaid or other third-party payors, certain federal and state healthcare laws and regulations pertaining to fraud and abuse and patients rights are and will be applicable to our business. We could be subject to healthcare fraud and abuse and patient privacy regulation by both the federal government and the states in which we conduct our business. The regulations that may affect our ability to operate include, without limitation:

the federal healthcare program Anti-Kickback Statute, which prohibits, among other things, any person from knowingly and willfully offering, soliciting, receiving or providing remuneration, directly or

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indirectly, to induce either the referral of an individual, for an item or service or the purchasing or ordering of a good or service, for which payment may be made under federal healthcare programs such as the Medicare and Medicaid programs;

the federal False Claims Act, which prohibits, among other things, individuals or entities from knowingly presenting, or causing to be presented, false claims, or knowingly using false statements, to obtain payment from the federal government, and which may apply to entities like us which provide coding and billing advice to customers;

federal criminal laws that prohibit executing a scheme to defraud any healthcare benefit program or making false statements relating to healthcare matters;

the federal Health Insurance Portability and Accountability Act of 1996, as amended by the Health Information Technology for Economic and Clinical Health Act, which governs the conduct of certain electronic healthcare transactions and protects the security and privacy of protected health information; and

state law equivalents of each of the above federal laws, such as anti-kickback and false claims laws which may apply to items or services reimbursed by any third-party payor, including commercial insurers.

The recently enacted Affordable Care Act, among other things, amends the intent requirement of the Federal Anti-Kickback Statute and criminal healthcare fraud statutes. A person or entity no longer needs to have actual knowledge of this statute or specific intent to violate it. In addition, the Affordable Care Act provides that the government may assert that a claim including items or services resulting from a violation of the Federal Anti-Kickback Statute constitutes a false or fraudulent claim for purposes of the False Claims Act.

If our operations are found to be in violation of any of the laws described above or any other governmental regulations that apply to us, we may be subject to penalties, including civil and criminal penalties, damages, fines and the curtailment or restructuring of our operations. Any penalties, damages, fines, curtailment or restructuring of our operations could adversely affect our ability to operate our business and our financial results. Any action against us for violation of these laws, even if we successfully defend against it, could cause us to incur significant legal expenses and divert our management s attention from the operation of our business. Moreover, achieving and sustaining compliance with applicable federal and state privacy, security and fraud laws may prove costly.

Risks Related To This Offering

The price of our common stock may be volatile, and you may not be able to resell your shares at or above the initial public offering price.

The initial public offering price for the shares of our common stock sold in this offering has been determined by negotiation between the underwriters and us. This price may not reflect the market price of our common stock following this offering. Prior to this offering, there has been no public market for our common stock. An active and liquid trading market for our common stock may not develop or be sustained after this offering. You may be unable to sell your shares of common stock at or above the initial public offering price due to fluctuations in the market price of our common stock. Factors that could cause volatility in the market price of our common stock include, but are not limited to:

results from, and any delays in, clinical trial programs relating to our drug candidates, including the ongoing and planned clinical trials for Traficet-EN, CCX140, CCX354, CCX168 and other drug candidates;

announcements of regulatory approvals or disapprovals of our drug candidates, including Traficet-EN and CCX140, or delays in any regulatory agency review or approval processes;

failure or discontinuation of any of our research programs;

announcements relating to future collaborations or our existing collaboration with GSK;

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general economic conditions in the United States and abroad;

acquisitions and sales of new products, technologies or business;

delays in the commercialization of any of our drug candidates;

market conditions in the pharmaceutical, biopharmaceutical and biotechnology sectors;

the issuance of new or changed securities analysts reports or recommendations regarding us, our competitors or our industry in general;

actual and anticipated fluctuations in our quarterly operating results;

disputes concerning our intellectual property or other proprietary rights;

introduction of technological innovations or new products by us or our competitors;

manufacturing issues related to our drug candidates for clinical trials or future products for commercialization;

market acceptance of our future products;

deviations in our operating results from the estimates of analysts, or other analyst comments;

third party payor coverage and reimbursement policies;

new legislation in the United States relating to the sale or pricing of pharmaceuticals;

FDA or other U.S. or foreign regulatory actions affecting us or our industry;

product liability claims or other litigation or public concern about the safety of our drug candidates or future drugs;

our ability to obtain necessary intellectual property licenses including, if necessary, those relating to Traficet-EN and other CCR9 drug candidates;

the outcome of any future legal actions to which we are party;

sales of our common stock by our officers, directors or significant stockholders;

additions or departures of key personnel; and

external factors, including natural disasters and other crises.

In addition, the stock markets in general, and the markets for pharmaceutical, biopharmaceutical and biotechnology stocks in particular, have experienced extreme volatility that have been often unrelated to the operating performance of the issuer. These broad market fluctuations may adversely affect the trading price or liquidity of our common stock. In the past, when the market price of a stock has been volatile, holders of that stock have sometimes instituted securities class action litigation against the issuer. If any of our stockholders were to bring such a lawsuit against us, we could incur substantial costs defending the lawsuit and the attention of our management would be diverted from the operation of our business.

The ownership of our common stock will continue to be highly concentrated, and these stockholders could delay or prevent a change of control.

After this offering and the concurrent private placements to GSK and Techne and the automatic conversion of the convertible note held by Techne, our officers and directors, together with holders of 5% or more of our outstanding common stock and their respective affiliates, will beneficially own approximately 75% of our common stock (assuming no exercise of the underwriters over-allotment option). Accordingly, these stockholders, acting as a group, will continue to have significant influence over the outcome of corporate actions requiring stockholder approval, including the election of directors, any merger, consolidation or sale of all or substantially all of our assets or any other significant corporate transaction. The interests of these stockholders

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may not be the same as or may even conflict with your interests. For example, these stockholders could delay or prevent a change of control of our company, even if such a change of control would benefit our other stockholders, which could deprive our stockholders of an opportunity to receive a premium for their common stock as part of a sale of our company or our assets and might affect the prevailing market price of our common stock. The significant concentration of stock ownership may adversely affect the trading price of our common stock due to investors perception that conflicts of interest may exist or arise.

Future sales of our common stock or securities convertible or exchangeable for our common stock may depress our stock price.

If our existing stockholders or holders of our convertible notes, options or warrants sell, or indicate an intention to sell, substantial amounts of our common stock in the public market after the lock-up and legal restrictions on resale discussed in this prospectus lapse, the trading price of our common stock could decline. The perception in the market that these sales may occur could also cause the trading price of our common stock to decline. Based on shares of common stock outstanding as of September 30, 2011, upon (1) the completion of this offering, (2) the conversion of all of our preferred stock into 24,332,186 shares of common stock prior to the completion of this offering, (3) the concurrent private placements to GSK and Techne of $7.0 million and $5.0 million of our common stock, respectively, and (4) the automatic conversion of the convertible note held by Techne at a conversion price equal to the initial public offering price, in each case at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus, we will have outstanding a total of 34,020,576 shares of common stock, assuming no exercise of the underwriters overallotment option. Of these shares, only the shares of common stock sold by us in this offering, plus any shares sold upon exercise of the underwriters overallotment option will be freely tradable, without restriction, in the public market immediately following this offering. Our underwriters may, however, in their sole discretion, permit our officers, directors and other stockholders and the holders of our outstanding options and warrants who are subject to these lock-up agreements to sell shares prior to the expiration of the lock-up agreements.

We expect that the lock-up agreements pertaining to this offering will expire 180 days from the date of this prospectus (subject to extension upon the occurrence of specified events). After the lock-up agreements expire, up to an additional 30,020,576 shares of common stock, and up to approximately 309,500 shares of common stock issuable upon exercise of our outstanding warrants, including warrants to purchase up to 150,000 shares of our common stock that will be issued to Techne upon completion of this offering, will be eligible for sale in the public market, subject to volume limitations under Rule 144 under the Securities Act of 1933, as amended, or the Securities Act, with respect to shares held by directors, executive officers and other affiliates. In addition, shares of common stock that are either subject to outstanding options or reserved for future issuance under our employee benefit plans will become eligible for sale in the public market to the extent permitted by the provisions of various vesting schedules, the lock-up agreements and Rule 144 and Rule 701 under the Securities Act and, in any event, we plan to file a registration statement permitting shares of common stock issued on exercise of options to be freely sold in the public market. If these additional shares of common stock are sold, or if it is perceived that they will be sold, in the public market, the trading price of our common stock could decline.

Certain holders of shares of our common stock, warrants to purchase our capital stock and the shares of common stock issuable upon exercise of those warrants will be entitled to rights with respect to the registration of their shares under the Securities Act, subject to the lock-up agreements described above. See Description of Capital Stock Registration Rights. Registration of these shares under the Securities Act would result in the shares becoming freely tradable without restriction under the Securities Act, except for shares purchased by affiliates. In addition, after the lock-up agreements described above expire, our directors may and we expect that our executive officers will establish programmed selling plans under Rule 10b5-1 of the Exchange Act, for the purpose of effecting sales of our common stock. Any sales of securities by these stockholders, or the perception that those sales may occur, including the entry into such programmed selling plans, could have a material adverse effect on the trading price of our common stock.

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If there is no viable public market for our common stock, you may not be able to sell your shares at or above the initial public offering price.

Prior to this offering, there has been no public market for our common stock, and there can be no assurance that a regular trading market will develop and continue after this offering or that the market price of our common stock will not decline below the initial public offering price. The initial public offering price was determined through negotiations between us and the underwriters and may not be indicative of the market price of our common stock following this offering. Among the factors considered in such negotiations were prevailing market conditions, certain of our financial information, market valuations of other companies that we and the underwriters believe to be comparable to us, estimates of our business potential, the present state of our development and other factors deemed relevant. See Underwriting for additional information.

Investors in this offering will suffer immediate and substantial dilution of their investment.

If you purchase common stock in this offering, you will pay more for your shares than our pro forma as adjusted net tangible book value per share. Based upon an assumed initial public offering price of $15.00 per share, the midpoint of the range on the cover page of this prospectus, you will incur immediate and substantial dilution of $11.11 per share, representing the difference between our assumed initial public offering price and our pro forma as adjusted net tangible book value per share. Based upon an assumed initial public offering price of $15.00 per share, the midpoint of the range on the cover page of this prospectus, purchasers of common stock in this offering will have contributed approximately 24.1% of the aggregate purchase price paid by all purchasers of our stock but will own only approximately 11.8% of our common stock outstanding after this offering. In addition, if you choose to invest in our common stock, you will pay a price per share that substantially exceeds the value of our assets after subtracting our liabilities. As of September 30, 2011, we had options outstanding under our equity compensation plans to purchase an aggregate of 4,172,318 shares of common stock at a weighted-average exercise price of $4.72 per share and had warrants outstanding to purchase an aggregate of 159,500 shares of our preferred stock at an exercise price of $5.20 per share. To the extent these outstanding options or warrants are exercised, you will incur further dilution.

If we sell shares of our common stock in future financings, common stockholders may experience immediate dilution and, as a result, our stock price may decline.

We may from time to time issue additional shares of common stock at a discount from the current trading price of our common stock. As a result, our common stockholders would experience immediate dilution upon the purchase of any shares of our common stock sold at such discount. In addition, as opportunities present themselves, we may enter into financing or similar arrangements in the future, including the issuance of debt securities, preferred stock or common stock. For example, upon completion of this offering, we will issue Techne a warrant with a ten-year term to purchase up to 150,000 shares of our common stock at an exercise per share equal to 200% of the initial public offering price of a share of our common stock and such warrant, if exercised, would likely be exercised at a time when the exercise price of such warrant represented a discount to the trading price of our common stock. If we issue common stock or securities convertible into common stock, our common stockholders would experience additional dilution and, as a result, our stock price may decline.

Our quarterly operating results may fluctuate significantly or may fall below the expectations of investors or securities analysts, each of which may cause our stock price to fluctuate or decline.

We expect our operating results to be subject to quarterly fluctuations. Our net loss and other operating results will be affected by numerous factors, including:

- variations in the level of expenses related to our drug candidates or future development programs;
- if any of our drug candidates receives regulatory approval, the level of underlying demand for these drug candidates and wholesalers buying patterns.

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addition or termination of clinical trials or funding support;

our execution of any collaborative, licensing or similar arrangements, and the timing of payments we may make or receive under these arrangements;

any intellectual property infringement lawsuit in which we may become involved;

regulatory developments affecting our drug candidates or those of our competitors;

ability to secure new government contracts and allocation of our resources to or away from performing work under government contracts; and

If our quarterly operating results fall below the expectations of investors or securities analysts, the price of our common stock could decline substantially. Furthermore, any quarterly fluctuations in our operating results may, in turn, cause the price of our stock to fluctuate substantially. We believe that quarterly comparisons of our financial results are not necessarily meaningful and should not be relied upon as an indication of our future performance.

We will have broad discretion in the use of the net proceeds of this offering and the concurrent private placements to GSK and Techne and may not use them effectively.

Our management will have broad discretion over the use of the net proceeds from this offering and the concurrent private placements to GSK and Techne. Because of the number and variability of factors that will determine our use of such proceeds, you may not agree with how we allocate or spend the proceeds from this offering and the concurrent private placements. We may pursue collaborations or clinical trials that do not result in an increase in the market value of our common shares and that may increase our losses. Our failure to allocate and spend the net proceeds from this offering and the concurrent private placements effectively would have a material adverse effect on our financial condition and business. Until the net proceeds are used, they may be placed in investments that do not produce significant investment returns or that may lose value.

Provisions of our charter documents or Delaware law could delay or prevent an acquisition of our company, even if the acquisition would be beneficial to our stockholders, and could make it more difficult for you to change management.

Provisions of our amended and restated certificate of incorporation and amended and restated bylaws may discourage, delay or prevent a merger, acquisition or other change in control that stockholders may consider favorable, including transactions in which stockholders might otherwise receive a premium for their shares. In addition, these provisions may frustrate or prevent any attempt by our stockholders to replace or remove our current management by making it more difficult to replace or remove our board of directors. These provisions include:

a classified board of directors so that not all directors are elected at one time;

a prohibition on stockholder action through written consent;

a requirement that special meetings of stockholders be called only by the chairman of the board of directors, the chief executive officer, the president or by the board of directors;

limitation of our stockholders entitled to call special meetings of stockholders;

an advance notice requirement for stockholder proposals and nominations;

the authority of our board of directors to issue preferred stock with such terms as our board of directors may determine; and

a requirement of approval of not less than 66 2/3% of all outstanding shares of our capital stock entitled to vote to amend any bylaws by stockholder action, or to amend specific provisions of our certificate of incorporation.

In addition, Delaware law prohibits a publicly held Delaware corporation from engaging in a business combination with an interested stockholder, generally a person who, together with its affiliates, owns or within the

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last three years has owned 15% of our voting stock, for a period of three years after the date of the transaction in which the person became an interested stockholder, unless the business combination is approved in a prescribed manner. Accordingly, Delaware law may discourage, delay or prevent a change in control of our company.

Provisions in our charter and other provisions of Delaware law could limit the price that investors are willing to pay in the future for shares of our common stock.

Our employment agreements with our named executive officers may require us to pay severance benefits to any of those persons who are terminated in connection with a change of control of us, which could harm our financial condition or results.

Our named executive officers are parties to employment agreements providing for aggregate cash payments of up to approximately $3.8 million for severance and other benefits and acceleration of vesting of stock options with a value of approximately $7.5 million (as of December 31, 2011) in the event of a termination of employment in connection with a change of control of us. The accelerated vesting of options could result in dilution to our existing stockholders and harm the market price of our common stock. The payment of these severance benefits could harm our financial condition and results. In addition, these potential severance payments may discourage or prevent third parties from seeking a business combination with us.

We do not anticipate paying any cash dividends on our capital stock in the foreseeable future, therefore capital appreciation, if any, of our common stock will be your sole source of gain for the foreseeable future.

We have never declared or paid cash dividends on our capital stock. We do not anticipate paying any cash dividends on our capital stock in the foreseeable future. We currently intend to retain all available funds and any future earnings to fund the development and growth of our business. In addition, our ability to pay cash dividends is currently prohibited by our loan and security agreement with Silicon Valley Bank, and any future debt financing arrangement may contain terms prohibiting or limiting the amount of dividends that may be declared or paid on our common stock. As a result, capital appreciation, if any, of our common stock will be your sole source of gain for the foreseeable future.

If securities or industry analysts do not publish research, or publish inaccurate or unfavorable research, about our business, our stock price and trading volume could decline.

The trading market for our common stock will depend, in part, on the research and reports that securities or industry analysts publish about us or our business. Securities and industry analysts do not currently, and may never, publish research on our company. If no securities or industry analysts commence coverage of our company, the trading price for our stock would likely be negatively impacted. In the event securities or industry analysts initiate coverage, if one or more of the analysts who cover us downgrade our stock or publish inaccurate or unfavorable research about our business, our stock price would likely decline. In addition, if our operating results fail to meet the forecast of analysts, our stock price would likely decline. If one or more of these analysts cease coverage of our company or fail to publish reports on us regularly, demand for our stock could decrease, which might cause our stock price and trading volume to decline.

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CAUTIONARY NOTE REGARDING FORWARD-LOOKING STATEMENTS

This prospectus contains forward-looking statements that involve risks and uncertainties. All statements other than statements of historical facts contained in this prospectus are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as may, could, will, would, should, expect, plan, anticipate, believe, estimate, intend, predict, seek, contemplate negative of these terms or other comparable terminology. These forward-looking statements include, but are not limited to, statements about:

- the initiation, timing, progress and results of our preclinical studies and clinical trials, and our research and development programs;
- our ability to advance drug candidates into, and successfully complete, clinical trials;
- our collaborator s exercise of its license option;
- the commercialization of our drug candidates;
- the implementation of our business model, strategic plans for our business, drug candidates and technology;
- the scope of protection we are able to establish and maintain for intellectual property rights covering our drug candidates and technology;
- estimates of our expenses, future revenues, capital requirements and our needs for additional financing;
- the timing or likelihood of regulatory filings and approvals;
- our ability to maintain and establish collaborations or obtain additional government grant funding;
- our use of proceeds from this offering and the concurrent private placements to GSK and Techne;
- our financial performance; and
- developments relating to our competitors and our industry.

These statements relate to future events or to our future financial performance and involve known and unknown risks, uncertainties and other factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by these forward-looking statements. Factors that may cause actual results to differ materially from current expectations include, among other things, those listed under Risk Factors and elsewhere in this prospectus.

Any forward-looking statement in this prospectus reflects our current views with respect to future events and is subject to these and other risks, uncertainties and assumptions relating to our operations, results of operations, industry and future growth. Given these uncertainties, you should not place undue reliance on these forward-looking statements. Except as required by law, we assume no obligation to update or revise these

forward-looking statements for any reason, even if new information becomes available in the future.

This prospectus also contains estimates, projections and other information concerning our industry, our business, and the markets for certain drugs, including data regarding the estimated size of those markets, their projected growth rates, the incidence of certain medical conditions, statements that certain drugs, classes of drugs or dosages are the most widely prescribed in the United States or other markets, the perceptions and preferences of patients and physicians regarding certain therapies and other prescription, prescriber and patient data, as well as data regarding market research, estimates and forecasts prepared by our management. Information that is based on estimates, forecasts, projections, market research or similar methodologies is inherently subject to uncertainties and actual events or circumstances may differ materially from events and circumstances reflected in this information. Unless otherwise expressly stated, we obtained this industry, business, market and other data from reports, research surveys, studies and similar data prepared by market research firms and other third parties, industry, medical and general publications, government data and similar sources. In particular, unless otherwise specified, all prescription, prescriber and patient data in this prospectus is from Datamonitor. In some cases, we do not expressly refer to the sources from which this data is derived. In that regard, when we refer to one or more sources of this type of data in any paragraph, you should assume that other data of this type appearing in the same paragraph is derived from the same sources, unless otherwise expressly stated or the context otherwise requires.

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USE OF PROCEEDS

We estimate that the net proceeds from the sale of 4,000,000 shares of common stock in this offering will be approximately $53.3 million at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus, after deducting the underwriting discounts and commissions and estimated offering expenses payable by us. We will also receive $7.0 million from the sale of shares of common stock in the concurrent private placement to GSK and $5.0 million from the sale of shares of common stock in the concurrent private placement to Techne, each at a price per share equal to the initial public offering price. If the underwriters exercise their over-allotment option in full, we estimate that the net proceeds will be approximately $61.7 million after deducting the underwriting discounts and commissions and estimated offering expenses payable by us. Each $1.00 increase or decrease in the assumed initial public offering price of $15.00 would increase or decrease, respectively, our net proceeds by $3.7 million, assuming the number of shares offered by us, as set forth on the cover page of this prospectus, remains the same and after deducting the underwriting discounts and commissions and estimated offering expenses payable by us.

We currently expect to use our net proceeds from this offering, together with the proceeds from the sale of our concurrent private placements to GSK and Techne as follows:

- Approximately $25.0 million to further develop CCX140, including the completion of the two Phase II clinical trials in diabetic nephropathy and additional Phase I clinical trials and related activities in anticipation of conducting end-of-Phase II meetings with the FDA and EMA;

- Approximately $10.0 million to advance CCX168 and CCX662 through Phase II clinical proof-of-concept and to further explore our ChemR23 program, including the possible optimization of ChemR23 antagonist leads;

- Approximately $10.0 million to fund our research and drug discovery activities related to additional drug candidates; and

- The remainder for working capital and general corporate purposes, including hiring of additional personnel and expenses associated with being a public company.

However, due to the uncertainties inherent in the product development process, it is difficult to estimate with certainty the exact amounts of the net proceeds from this offering and the concurrent private placements to GSK and Techne that may be used for the above purposes. Our management will have broad discretion over the use of the net proceeds from this offering and the concurrent private placements. The amounts and timing of our expenditures will depend upon numerous factors including the results of our research and development efforts, the timing and success of preclinical studies and any ongoing clinical trials or clinical trials we may commence in the future, the timing of regulatory submissions and the amount of cash, if any, generated by our collaboration agreement.

Pending the use of the proceeds from this offering and the concurrent private placement, we intend to invest the net proceeds in short-term, interest-bearing, investment-grade securities, certificates of deposit or government securities.

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DIVIDEND POLICY

We have never declared or paid cash dividends on our capital stock. We intend to retain all available funds and any future earnings, if any, to fund the development and expansion of our business and we do not anticipate paying any cash dividends in the foreseeable future. In addition, unless waived, the terms of our credit facility with Silicon Valley Bank prohibit us from paying cash dividends. Any future determination related to dividend policy will be made at the discretion of our board of directors.

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CAPITALIZATION

The following table sets forth our cash, cash equivalents and investments and capitalization as of September 30, 2011:

- on an actual basis;
- on a pro forma basis to reflect (1) conversion of all outstanding shares of our preferred stock into an aggregate of 24,332,186 shares of common stock prior to the completion of this offering, and (2) the automatic conversion of the convertible note held by Techne at a conversion price equal to the initial public offering price, in each case at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus; and
- on a pro forma as adjusted basis to additionally reflect (1) the concurrent private placements to GSK and Techne of $7.0 million and $5.0 million of our common stock, respectively, and (2) the issuance and sale by us of 4,000,000 shares of our common stock in this offering, after deducting the underwriting discounts and commissions and estimated offering expenses payable by us, at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover of this prospectus.

You should read this information together with our audited financial statements and related notes appearing elsewhere in this prospectus and the information set forth under the heading Selected Financial Data and Management s Discussion and Analysis of Financial Condition and Results of Operations.

	As of September 30, 2011		
	Actual	Pro Forma (unaudited)	Pro Forma As Adjusted[1]
	(in thousands, except share and per share data)		
Cash, cash equivalents and investments[2]	$ 81,182	$ 81,182	$ 146,482
Equipment financing obligations	$ 1,581	$ 1,581	$ 1,581
Convertible note from related party	10,060		
Convertible preferred stock, $0.001 par value; 48,989,914 shares authorized, 48,664,392 shares issued and outstanding, actual; no shares authorized, issued and outstanding, pro forma and pro forma as adjusted	49		
Common stock, $0.001 par value; 68,000,000 shares authorized, 4,219,715 shares issued and outstanding, actual; 200,000,000 shares authorized, 29,220,577 shares issued and outstanding, pro forma; 34,020,576 shares issued and outstanding, pro forma as adjusted	8	29	34
Preferred stock, $0.001 par value; no shares issued and outstanding, actual; 10,000,000 shares authorized, no shares issued and outstanding, pro forma and pro forma as adjusted			
Additional paid-in capital	169,485	179,573	244,868
Employee note receivable	(16)	(16)	(16)
Accumulated other comprehensive income (loss)	(41)	(41)	(41)
Accumulated deficit	(112,530)	(112,530)	(112,530)
Total stockholders equity	56,955	67,015	132,315
Total capitalization	$ 68,596	$ 68,596	$ 133,896

(1) Each $1.00 increase or decrease in the assumed initial public offering price of $15.00 would increase or decrease, respectively, the amount of cash, cash equivalents and investments, additional paid-in capital and total capitalization by $3.7 million, assuming the number of shares offered by us, as set forth on the cover page of this prospectus, remains the same and after deducting the underwriting discount and estimated offering expenses payable by us.

(2) Subsequent to September 30, 2011, we received a $25.0 million option exercise payment under our strategic alliance with GSK with respect to CCX354.

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The actual and pro forma as adjusted outstanding shares information in the table above excludes the following:

- 4,172,318 shares of common stock issuable upon the exercise of outstanding stock options having a weighted-average exercise price of $4.72 per share;
- 159,500 shares of common stock issuable upon the exercise of outstanding warrants having an exercise price of $5.20 per share;
- 59,009 shares of common stock reserved for issuance pursuant to future option grants under our 2002 Equity Incentive Plan and our 1997 Stock Option/Stock Issuance Plan;
- 150,000 shares of common stock issuable upon the exercise of warrants, with an exercise price per share equal to 200% of the initial public offering price of our common stock, which warrants will be issued to Techne upon the completion of this offering.
- 3,000,000 shares of common stock reserved for issuance pursuant to future option grants under our 2012 Equity Incentive Award Plan;
- 300,000 shares of common stock reserved for issuance under our 2012 Employee Stock Purchase Plan; and
- the issuance of an additional 12,044 shares of our common stock upon the automatic conversion of the convertible note held by Techne, assuming a conversion date of February 10, 2012, at a conversion price equal to the assumed public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus).

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DILUTION

If you invest in our common stock, your interest will be diluted to the extent of the difference between the initial public offering price per share of our common stock in this offering and the pro forma as adjusted net tangible book value per share of our common stock after this offering. As of September 30, 2011, we had a historical net tangible book value of $57.0 million, or $1.99 per share of common stock, taking into account the expected conversion of our outstanding preferred stock into common stock prior to the completion of this offering. Without giving effect to the conversion of our outstanding preferred stock into common stock, we had a historical net tangible book value of $57.0 million, or $13.50 per share of common stock, as of September 30, 2011. Historical net tangible book value per share is equal to our total tangible assets, less total liabilities, divided by the number of outstanding shares of our common stock. Investors participating in this offering will incur immediate and substantial dilution. After giving effect to (1) the conversion of all of our preferred stock into 24,332,186 shares of common stock prior to the completion of this offering, (2) the sale of 4,000,000 shares of common stock in this offering, after deducting the underwriting discounts and commissions and estimated offering expenses payable by us, (3) the concurrent private placements to GSK and Techne of $7.0 million and $5.0 million of our common stock, respectively, and (4) the automatic conversion of the convertible note held by Techne at a conversion price equal to the initial public offering price, in each case at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus, our pro forma as adjusted net tangible book value as of September 30, 2011 was approximately $132.3 million, or approximately $3.89 per share of common stock. This represents an immediate increase in pro forma as adjusted net tangible book value of $1.26 per share to our existing stockholders and an immediate dilution of $11.11 per share to investors participating in this offering. The following table illustrates this per share dilution:

Assumed initial public offering price per share		$ 15.00
Historical net tangible book value per share as of September 30, 2011 assuming the conversion of the preferred stock into common stock	$ 1.99	
Pro forma increase in net tangible book value per share attributable to pro forma transactions described in the preceding paragraph other than the offering	0.64	
Pro forma net tangible book value per share as of September 30, 2011	$ 2.63	
Pro forma increase in net tangible book value per share attributable to new investors	1.26	
Pro forma as adjusted net tangible book value per share after this offering		3.89
Dilution per share to new investors participating in this offering		$ 11.11

Each $1.00 increase or decrease in the assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus, would increase or decrease, respectively, our pro forma as adjusted net tangible book value by approximately $3.7 million, the pro forma as adjusted net tangible book value per share by approximately $0.11 per share and the dilution to investors purchasing shares in this offering by approximately $0.11 per share, assuming the number of shares offered by us, as set forth on the cover page of this prospectus, remains the same and after deducting the underwriting discounts and commissions and estimated offering expenses payable by us.

The following table summarizes, on a pro forma as adjusted basis as of September 30, 2011, the differences between the number of shares of common stock purchased from us, the total consideration and the average price per share paid by existing stockholders (giving effect to each of the pro forma transactions described in the first paragraph of this section other than the offering) and by investors participating in this offering, after deducting estimated underwriting discounts and commissions and estimated offering expenses, at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus.

	Shares Purchased		Total Consideration		Average Price
	Number	Percent	Amount	Percent	Per Share
Existing stockholders	30,020,576	88.2%	$ 188,495,547	75.9%	$ 6.28
Investors participating in this offering	4,000,000	11.8	60,000,000	24.1	15.00
Total	34,020,576	100.0%	$ 248,495,547	100.0%	

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The number of shares of common stock to be outstanding after this offering and the concurrent private placements to GSK and Techne is based on the number of shares outstanding as of September 30, 2011 and excludes the following:

- 4,172,318 shares of common stock issuable upon the exercise of outstanding stock options having a weighted-average exercise price of $4.72 per share;
- 159,500 shares of common stock issuable upon the exercise of outstanding warrants having an exercise price of $5.20 per share;
- 59,009 shares of common stock reserved for issuance pursuant to future option grants under our 2002 Equity Incentive Plan and our 1997 Stock Option/Stock Issuance Plan;
- 150,000 shares of common stock issuable upon the exercise of warrants, with an exercise price per share equal to 200% of the initial public offering price of our common stock, which warrants will be issued to Techne upon the completion of this offering.
- 3,000,000 shares of common stock reserved for issuance pursuant to future option grants under our 2012 Equity Incentive Award Plan;
- 300,000 shares of common stock reserved for issuance under our 2012 Employee Stock Purchase Plan; and
- the issuance of an additional 12,044 shares of our common stock upon the automatic conversion of the convertible note held by Techne, assuming a conversion date of February 10, 2012, at a conversion price equal to the assumed public offering price of $15.00 per share (the midpoint of the range set forth on the cover page of this prospectus).

If the underwriters exercise their over-allotment option in full, pro forma as adjusted net tangible book value as of September 30, 2011 will increase to $140.7 million, or $4.06 per share, representing an increase to existing stockholders of $2.08 per share, and there will be an immediate dilution of an additional $10.94 per share to new investors.

Furthermore, we may choose to raise additional capital through the sale of equity or convertible debt securities due to market conditions or strategic considerations even if we believe we have sufficient funds for our current or future operating plans. New investors will experience further dilution if any of our outstanding options or warrants are exercised, new options are issued and exercised under our equity incentive plans or we issue additional shares of common stock, other equity securities or convertible debt securities in the future.

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SELECTED FINANCIAL DATA

The selected consolidated statement of operations data for the years ended December 31, 2006 and 2007 and the consolidated balance sheet data as of December 31, 2006, 2007 and 2008 are derived from our audited financial statements not included in this prospectus. We derived the consolidated statement of operations data for the years ended December 31, 2008, 2009 and 2010 and the consolidated balance sheet data as of December 31, 2009 and 2010 from our audited financial statements appearing elsewhere in this prospectus.

The consolidated statement of operations data for the nine months ended September 30, 2010 and 2011 and the selected consolidated balance sheet data as of September 30, 2011, have been derived from our unaudited consolidated financial statements appearing elsewhere in this prospectus. The unaudited interim consolidated financial information has been prepared on the same basis as the annual consolidated financial information and, in the opinion of management, reflect all adjustments, which include only normal recurring adjustments, necessary to present fairly our consolidated financial position as of September 30, 2011 and the consolidated results of operations for the nine months ended September 30, 2010 and 2011. Interim results are not necessarily indicative of results to be expected for the full year. You should read this data together with our audited consolidated financial statements and related notes appearing elsewhere in this prospectus and the information under the caption Management s Discussion and Analysis of Financial Condition and Results of Operations.

We have presented pro forma net loss per share information for the year ended December 31, 2010 and the nine months ended September 30, 2011 to reflect (1) the conversion of all outstanding shares of our preferred stock into an aggregate of 24,332,186 shares of common stock prior to the completion of this offering, (2) the issuance and sale by us of 4,000,000 shares of our common stock in this offering, after deducting the underwriting discounts and commissions and estimated offering expenses payable by us (3) the concurrent private placements to GSK and Techne of $7.0 million and $5.0 million of our common stock, respectively, and (4) the automatic conversion of the convertible note held by Techne at a conversion price equal to the initial public offering price, in each case at an assumed initial public offering price of $15.00 per share, the midpoint of the range set forth on the cover page of this prospectus.

	Years Ended December 31,					Nine Months Ended September 30,	
	2006	2007	2008	2009	2010	2010	2011
	(in thousands, except share and per share data)						
						(unaudited)	
Consolidated Statement of Operations Data:							
Revenues:							
Collaborative research and development revenue from related party	$ 4,950	$ 18,149	$ 23,551	$ 49,744	$ 34,861	$ 21,746	$ 5,621
Grant revenue	3,158	2,588	536				
Total revenues:	8,108	20,737	24,087	49,744	34,861	21,746	5,621
Operating expenses:							
Research and development	21,946	33,193	35,056	27,474	33,527	25,385	22,914
General and administrative	5,300	6,680	9,157	6,575	7,292	5,363	5,721
Total operating expenses	27,246	39,873	44,213	34,049	40,819	30,748	28,635
Income (loss) from operations	(19,138)	(19,136)	(20,126)	15,695	(5,958)	(9,002)	(23,014)
Interest income	1,932	3,930	1,762	297	436	322	319
Interest expense	(138)	(93)	(129)	(76)	(81)	(60)	(170)
Other income					2,434	490	16
Income (loss) before provision for income taxes	(17,344)	(15,299)	(18,493)	15,916	(3,169)	(8,250)	(22,849)
Income tax benefit (expense)		(395)	23	(293)	73	73	
Net income (loss)	$ (17,344)	$ (15,694)	$ (18,470)	$ 15,623	$ (3,096)	$ (8,177)	$ (22,849)
Basic net income (loss) per share[1]	$ (4.83)	$ (4.03)	$ (4.52)	$ 0.56	$ (0.76)	$ (2.01)	$ (5.49)
Diluted net income (loss) per share[1]	$ (4.83)	$ (4.03)	$ (4.52)	$ 0.53	$ (0.76)	$ (2.01)	$ (5.49)

Shares used to compute basic net income (loss) per share	3,592,805	3,892,924	4,087,181	3,961,640	4,081,648	4,077,347	4,162,309
Shares used to compute diluted net income (loss) per share	3,592,805	3,892,924	4,087,181	29,256,423	4,081,648	4,077,347	4,162,309
Pro forma basic and diluted net income (loss) per share (unaudited)[1]					$ (0.11)		$ (0.80)
Shares used to compute pro forma basic and diluted net income (loss) per share					28,210,296		28,471,126

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	As of December 31,					As of September 30,
	2006	2007	2008	2009	2010	2011
			(in thousands)			(unaudited)
Consolidated Balance Sheet Data:						
Cash, cash equivalents and investments	$ 82,227	$ 65,435	$ 90,158	$ 65,316	$ 82,836	$ 81,182
Working capital	70,730	49,029	76,598	79,125	82,712	64,964
Total assets	87,780	74,631	97,924	103,469	98,133	85,365
Non-current equipment financing obligations		1,163	455	7	945	1,040
Convertible note from related party						10,060
Accumulated deficit	(68,044)	(83,738)	(102,208)	(86,585)	(89,681)	(112,530)
Total stockholders equity	44,426	30,024	60,960	77,302	76,773	56,955

(1) See Note 2 within the notes to our consolidated financial statements which are included elsewhere in this prospectus for a description of the method used to compute basic and diluted loss per share.

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MANAGEMENT S DISCUSSION AND ANALYSIS OF

FINANCIAL CONDITION AND RESULTS OF OPERATIONS

You should read the following discussion and analysis of financial condition and results of operations together with the section entitled Selected Financial Data and our financial statements and related notes included elsewhere in this prospectus. This discussion and other parts of this prospectus contain forward-looking statements that involve risk and uncertainties, such as statements of our plans, objectives, expectations and intentions. Our actual results could differ materially from those discussed in these forward-looking statements. Factors that could cause or contribute to such differences include, but are not limited to, those discussed in the Risk Factors section.

Overview

ChemoCentryx is a biopharmaceutical company focused on discovering, developing and commercializing orally-administered therapeutics to treat autoimmune diseases, inflammatory disorders and cancer. We currently have four drug candidates in clinical development, and expect to advance one additional drug candidate into clinical development in 2012. Our drug candidates include: Traficet-EN (CCX282 or GSK 786), our most advanced drug candidate, currently in three pivotal Phase III clinical trials being conducted by our partner Glaxo Group Limited, or GSK, an affiliate of GlaxoSmithKline, for the treatment of patients with moderate-to-severe Crohn s disease; CCX140, our lead independent drug candidate, which successfully completed a Phase II clinical trial in type 2 diabetics and is currently in two Phase II clinical trials in patients with diabetic nephropathy, a form of kidney disease; CCX354, which successfully completed a Phase II proof-of-concept clinical trial for the treatment of rheumatoid arthritis, or RA; CCX168, currently in a Phase II proof-of-concept clinical trial for the treatment of anti-neutrophil cytoplasmic antibody, or ANCA-associated vasculitis, or AAV; and CCX662, our independent drug candidate for the treatment of glioblastoma multiforme, or GBM, which is expected to enter a Phase I clinical trial in the second half of 2012. CCX140 and CCX662 are wholly owned and are being developed independently by us, while Traficet-EN, CCX354 and CCX168 are subject to our collaboration agreement with GSK. In December 2009 and November 2011, GSK exercised its options to obtain exclusive licenses for the further development and commercialization of Traficet-EN and CCX354, respectively. Upon exercise of these options, GSK assumed sole responsibility for the further development and commercialization of these drug candidates and each of their two respective back-up compounds. We are also advancing several additional independent drug candidates through preclinical development. In addition, our strategy has been to identify next generation compounds related to our drug candidates. All of our drug candidates, including those under our collaboration agreement with GSK, have been internally discovered.

In August 2006, we entered into our strategic alliance with GSK. We have received $245.7 million from GSK, consisting of up-front and milestone payments, equity investments, research funding and option exercise fees. Under the terms of our agreement with GSK, we are responsible for the discovery and development of small molecule antagonists targeting four defined chemokine and chemo-attractant receptor targets (CCR9, CCR1, C5aR and ChemR23) and for advancing them through clinical proof-of-concept. If we demonstrate successful clinical proof-of-concept, GSK is entitled to options to exclusively license drug candidates that are subject to the collaboration and two defined back-up compounds for each drug candidate for further development and commercialization on a worldwide basis. Upon exercising any of its options to drug candidates under the collaboration, GSK is solely responsible for all further clinical development and commercialization expenditures worldwide with respect to that drug candidate and its two designated back-up compounds. In exchange for the rights granted to GSK upon the exercise of its options, we are also entitled to receive regulatory and commercial milestone payments, as earned under the terms of our agreement, and royalties on the net sales of licensed drugs. The agreement contemplated up to six drug options, each of which covers a drug candidate against the four defined targets, including Traficet-EN (CCR9), CCX354 (CCR1), CCX168 (C5aR) and CCX832 (ChemR23), and their associated back-up compounds. The other two drug options were for second generation drug candidates and their associated back-up compounds. However, we and GSK chose not to nominate second generation drug candidates against any of the four defined targets during the agreement s research term, which has expired. In addition, based on unblinded data from a recently completed Phase I clinical trial of CCX832, in February 2012 we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds, although both we and GSK continue to have interest in further discussing possible strategic

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opportunities with respect to ChemR23. GSK has already exercised its options to Traficet-EN and CCX354 and each of their two respective defined back-up compounds. Thus, GSK s only remaining option is to CCX168 and its associated back-up compounds. If GSK does not exercise its option to CCX168, we will evaluate our alternatives for further development of this drug candidate, which may entail internally developing it or identifying other collaboration partners for its development.

Since commencing our operations in 1997, our efforts have focused on research, development and the advancement of our drug candidates into and through clinical trials. As a result, we have incurred significant losses. We have funded our operations primarily through the sale of convertible preferred and common stock, contract revenue under our collaborations, government contracts and grants and borrowings under equipment financing arrangements. As of September 30, 2011, we had an accumulated deficit of $112.5 million. We expect to continue to incur net losses as we develop our drug candidates, expand clinical trials for our drug candidates currently in clinical development, expand our research and development activities, expand our systems and facilities, seek regulatory approvals and engage in commercialization preparation activities in anticipation of Food and Drug Administration, or FDA, approval of our drug candidates. In addition, if a product is approved for commercialization, we will need to expand our organization. Significant capital is required to launch a product and many expenses are incurred before revenues are received. We are unable to predict the extent of any future losses or when we will become profitable, if at all.

Financial Operations Overview

Revenues

We have not generated any revenue from product sales. Since our inception, our revenue has been derived from two primary sources: (1) contract revenue, up-front payments and development milestone payments from GSK and (2) government contracts and grants. The following table summarizes our revenue for each of the years ended December 31, 2008, 2009 and 2010 and the nine months ended September 30, 2010 and 2011.

	Year Ended December 31,			Nine Months Ended September 30,	
	2008	2009	2010	2010	2011
			(in thousands)		
GSK					
Contract revenue	$ 7,251	$ 4,100	$ 4,721	$ 2,513	$ 2,900
Recognition of up-front payments	6,300	5,644	5,140	4,233	2,721
Milestones	10,000	40,000	25,000	15,000	
Government contracts and grants	536				
Total revenues	$ 24,087	$ 49,744	$ 34,861	$ 21,746	$ 5,621

Research and Development Expenses

Research and development expenses represent costs incurred to conduct basic research, such as the discovery and development of our understanding of the chemokine system; the discovery and development of novel small molecule therapeutics, such as Traficet-EN and CCX140; the development of our suite of proprietary drug discovery technologies, known collectively as EnabaLink, which includes our proprietary Reverse Activation of Migration, or RAM, screening technology and preclinical studies and clinical trials of our drug candidates. We expense all research and development expenses as they are incurred. These expenses consist primarily of salaries and related benefits, including stock-based compensation, third-party contract costs relating to research, formulation, manufacturing, preclinical study and clinical trial activities, laboratory consumables, and allocated facility costs.

The following table summarizes our research and development expenses for each of the years ended December 31, 2008, 2009 and 2010 and the nine months ended September 30, 2010 and 2011. The project specific expenses summarized in the following table reflect costs directly attributable to our clinical development

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candidates and preclinical candidates nominated and selected for further development. Such project specific expenses include third-party contract costs relating to formulation, manufacturing, preclinical studies and clinical trial activities. Unlike with respect to our early stage research and drug discovery programs, we allocate research and development salaries, benefits or indirect costs to our development candidates and we have included such costs in the project specific expenses. All remaining research and development expenses are reflected in Research and drug discovery which represents early stage drug discovery programs. Such expenses include allocated employee salaries and related benefits, stock-based compensation, consulting and contracted services to supplement our in-house laboratory activities, laboratory consumables and allocated facility costs associated with these earlier stage programs.

At any given time, we typically have several active early stage research and drug discovery projects. Our internal resources, employees and infrastructure are not directly tied to any individual research or drug discovery project and are typically deployed across multiple projects. As such, we do not maintain information regarding these costs incurred for our early stage research and drug discovery programs on a project specific basis.

	Year Ended December 31,			Nine Months Ended September 30,	
	2008	2009	2010	2010	2011
			(in thousands)		
Development Candidate (Target)					
Traficet-EN (CCR9)	$ 16,804	$ 3,487	$	$	$
CCX140 (CCR2)	820	2,257	5,214	6,712	5,687
CCX354 (CCR1)	735	3,538	6,106	4,229	3,230
CCX168 (C5aR)		1,527	2,870	3,381	3,317
CCX832 (ChemR23)[(1)]			1,398	2,003	1,520
Research and drug discovery	16,697	16,665	17,939	9,060	9,160
Total research and development	$ 35,056	$ 27,474	$ 33,527	$ 25,385	$ 22,914

(1) In February 2012, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds.

We expect our research and development expenses to increase as we advance our development programs further and increase the number and size of our clinical trials. The process of conducting preclinical studies and clinical trials necessary to obtain regulatory approval is costly and time consuming. We or our partners may never succeed in achieving marketing approval for any of our drug candidates. The probability of success for each drug candidate may be affected by numerous factors, including preclinical data, clinical data, competition, manufacturing capability and commercial viability. For the remaining product option covered under our strategic alliance with GSK, for which we receive milestone payments, we are responsible for development of drug candidates through clinical proof-of-concept, after which time GSK has an option to an exclusive license on a compound by compound basis. Our strategy includes entering into additional partnerships with third parties for the development and commercialization of some of our independent drug candidates that are not subject to our alliance with GSK.

Most of our product development programs are at an early-to-mid-stage; therefore the successful development of our drug candidates is highly uncertain and may not result in approved products. Completion dates and completion costs can vary significantly for each drug candidate and are difficult to predict for each product. Given the uncertainty associated with clinical trial enrollments and the risks inherent in the development process, we are unable to determine the duration and completion costs of the current or future clinical trials of our drug candidates or if, or to what extent, we will generate revenues from the commercialization and sale of any of our drug candidates. We anticipate we will make determinations as to which programs to pursue and how much funding to direct to each program on an ongoing basis in response to the scientific and clinical success of each drug candidate, as well as ongoing assessment as to each drug candidate s commercial potential. We will need to raise additional capital or may seek additional strategic alliances in the future in order to complete the development and commercialization of our drug candidates, including CCX140, our lead independent drug candidate.

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General and Administrative Expenses

General and administrative expenses consist primarily of salaries and related benefits, including stock-based compensation and travel expenses, in executive, finance, business and corporate development and other administrative functions. Other general and administrative expenses include allocated facility-related costs not otherwise included in research and development expenses, legal costs of pursuing patent protection of our intellectual property, and professional fees for auditing, tax, and legal services. We expect that general and administrative expenses will increase in the future as we expand our operating activities and incur additional costs associated with being a public company. These public company related increases will likely include legal fees, accounting fees, directors and officers liability insurance premiums and investor relations related fees.

Other Income

Other income, net, consists primarily of income or expenses which are non-recurring in nature. For instance, in 2010, we were awarded $1.9 million from the United States Department of Treasury for eight projects under the Qualitative Therapeutic Discovery Project Program under the Patient Protection and Affordable Care Act of 2010 to support research with the potential to produce new therapies and reported such amount in other income.

Critical Accounting Policies and Significant Judgments and Estimates

The preparation of our consolidated financial statements requires us to make estimates and assumptions that affect the reported amounts of assets and liabilities and the disclosure of contingent assets and liabilities at the date of our financial statements as well as the reported revenues and expenses during the reported periods. We base our estimates on historical experience and on various other factors that we believe are reasonable under the circumstances, the results of which form the basis for making judgments about the carrying value of assets and liabilities that are not apparent from other sources. Actual results may differ from these estimates under different assumptions or conditions.

While our significant accounting policies are described in the Notes to our consolidated financial statements appearing at the end of this prospectus, we believe that the following critical accounting policies relating to revenue recognition, clinical trial expenses and stock-based compensation are most important to understanding and evaluating our reported financial results.

Revenue Recognition

We generate revenue from two principal sources: (1) collaborative research and development agreements with pharmaceutical companies and (2) government contracts and grants. We recognize revenue in accordance with the criteria outlined in the Securities and Exchange Commission s Topic 13 and Accounting Standards Codification, or ASC, 605-25 and by the Financial Accounting Standards Board, or FASB. Following these accounting pronouncements, revenue is recognized when the following criteria have been met:

- persuasive evidence of an arrangement exists;
- delivery has occurred and risk of loss has passed;
- the seller s price to the buyer is fixed or determinable; and
- collectibility is reasonably assured.

As a result, we recognize revenue under the government grants when the work is performed or the expenses are incurred. Any amounts received in advance of performance are recorded as deferred revenue until earned.

Under collaboration agreements, we may receive payments for non-refundable up-front fees, reimbursement for research and development services, milestone payments and royalties. In assessing the appropriate revenue recognition related to a collaboration agreement, we first determine whether an arrangement includes multiple elements, such as the delivery of intellectual property rights and research and development

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property rights generally are not separable from the activity of providing research and development services because the intellectual property right does not have stand-alone value separate from the research and development services provided or evidence of fair value does not exist for the undelivered research and development services. Accordingly, we account for our collaboration agreements as a combined unit of accounting. The revenue from up-front payments is recognized on a straight-line basis over the estimated term of the research and development obligations covered under the research and development collaboration agreement. We periodically review the basis for our estimates, and we may change the estimates if circumstances change. These changes can significantly increase or decrease the amount of revenue recognized. As we applied our policy to our collaboration arrangements we made judgments which affected the pattern of revenue recognition. For instance, in our arrangement with GSK, we are obligated to provide research and development services. We are recognizing revenue over the estimated period of our performance of the research and development services, which was estimated to end in March 2014, the expected completion date of the proof-of-concept trial for the last of the drug candidates to be developed under the GSK alliance. In 2010 we increased our estimate for the remaining estimated research and development period under our arrangement with GSK by approximately 1.25 years. This change in estimate was accounted for prospectively and reduced the annualized revenue recognition by approximately $2.0 million per year. In February 2012, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds and we will revise the estimated period of performance prospectively to end by December 2012.

In addition to up-front payments and research and development funding, we may also be entitled to milestone payments that are contingent upon our achieving a predefined objective. Milestone payments are recorded as revenue upon achievement if there is substantive uncertainty at the date the arrangement is entered into that the event will be achieved and the achievement of the milestone is based on our performance.

Clinical Trial Accruals and Related Expenses

We accrue and expense costs for clinical trial activities performed by third parties, including clinical research organizations, or CROs, and clinical investigators, based upon estimates made as of the reporting date of the work completed over the life of the individual study in accordance with agreements established with CROs and clinical trial sites. Some CROs invoice us on a monthly basis, while others invoice upon milestones achieved and the expense is recorded as services are rendered. We determine the estimates of clinical activities incurred at the end of each reporting period through discussion with internal personnel and outside service providers as to the progress or stage of completion of trials or services, as of the end of each reporting period, pursuant to contracts with numerous clinical trial centers and CROs and the agreed upon fee to be paid for such services. The significant factors considered in estimating accruals include the number of patients enrolled and the percentage of work completed to date. Costs of setting up clinical trial sites for participation in the trials that are paid for in advance are expensed over the estimated set-up period. While the set-up periods vary from one arrangement to another, such set-up periods generally take from two to six months. Such set-up activities include clinical site identification, local ethics committee submissions, regulatory submissions, clinical investigator kick-off meetings and pre-study site visits. Clinical trial site costs related to patient enrollments are accrued as patients are entered into the trial.

To date, we have not experienced significant changes in our estimates of clinical trial accruals after a reporting period. However, due to the nature of estimates, we cannot assure you that we will not make changes to our estimates in the future as we become aware of additional information about the status or conduct of our clinical trials.

Stock-Based Compensation

Stock-based compensation cost is measured at the grant date, based on the fair value of the award, and is recognized as an expense over the employee s requisite service period on a straightline basis. We recorded non-cash stock-based compensation expense of $0.7 million, $1.8 million and $2.3 million for the years ended December 31, 2008, 2009 and 2010 and $1.7 million and $2.0 million during the nine months ended September 30, 2010 and 2011, respectively. At December 31, 2010 and September 30, 2011, we had $5.2 million and $4.9 million, respectively, of total unrecognized stock-based compensation expense, net of estimated

forfeitures, related to stock option plans that will be recognized over a weighted-average period of 2.76 years and 2.51 years, respectively. We expect to continue to grant stock options in the future, and to the extent that we do, our actual stock-based compensation expense recognized in future periods will likely increase.

Significant Factors, Assumptions and Methodologies Used in Determining Fair Value

Our board of directors, with the assistance of management and independent consultants, performed fair value analyses for the valuation of our common stock as of December 2009, December 2010 and June 2011. For grants made on dates for which there was no contemporaneous valuation to utilize in setting the exercise price of our common stock, and given the absence of an active market for our common stock, our board of directors determined the fair value of our common stock on the date of grant based on several factors, including:

- important developments in our operations, most significantly related to the clinical development of our lead drug candidates, Traficet-EN and CCX140;
- equity market conditions affecting comparable public companies;
- the likelihood of achieving a liquidity event for the shares of common stock, such as an initial public offering or an acquisition of us, given prevailing market conditions; and
- that the grants involved illiquid securities in a private company.

In determining the fair value of our common stock, we used a combination of the market multiple approach and the initial public offering, or IPO, value approach to estimate the enterprise value of our company. The per share common stock value was estimated by allocating the enterprise value using the probability-weighted expected return method, or PWERM, at each valuation date.

The market multiple approach estimates the value of a business by comparing a company to similar publicly-traded companies. When selecting the comparable companies to be used for the market multiple approaches, we focused on companies within the biopharmaceutical industry. Some of the specific criteria used to select the comparable companies included those discovering and developing small molecule drugs in the therapeutic areas of autoimmune or inflammatory disorders and cancer and whose product pipeline was comprised of lead candidates in a pre-commercial stage and/or pre-Phase III clinical development. The mix of comparable companies was reviewed at each valuation date to assess whether to add or delete companies; however, following each review, the comparable companies remained largely unchanged from those used in prior valuation analysis.

A group of comparable publicly-traded companies is selected and market multiples are calculated using each company s stock price and other financial data. An estimate of value for our company is completed by applying selected market multiples based on forecasted financial results for both the comparable companies and the subject company. Given that we are several years away from generating product revenue and we were unable to develop reliable long-term forecasts, our analysis applied the market approach based on our research and development spending results, which was deemed to be the most relevant financial measure.

The IPO value approach estimates the value of a business by estimating a future IPO value based on pre-money valuations of biopharmaceutical IPOs of similar stage over approximately the preceding two to three year period, discounted to the present value (as further discussed below in each valuation discussion). Given that both the market multiple approach and the IPO value approach provide relevant estimates of fair value, which did not differ significantly, we applied equal weighting to each of these approaches to determine an initial estimated enterprise value. The initial estimated enterprise value was then allocated to the common stock using the PWERM for the periods described below.

The PWERM is a scenario-based analysis that estimates the value per share based on the probability-weighted present value of expected future investment returns, considering each of the possible outcomes available to us, as well as the rights of each share class. The PWERM estimates the common stock value to our stockholders under each of four possible future scenarios IPO, sale, stay private and liquidation. The value per share under each scenario was then probability weighted and the resulting weighted values per share were summed to determine the fair value per share of our common stock. In the liquidation, sale and stay private

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scenarios, the value per share was allocated taking into account the liquidation preferences and participation rights of our convertible preferred stock consistent with the method outlined in the AICPA Practice Guide, *Valuation of Privately-Held Company Equity Securities Issued as Compensation*. In the IPO scenario, it was assumed that all outstanding shares of our convertible preferred stock would convert into common stock. Over time, as we achieved certain company related milestones, the probability of each scenario was evaluated and adjusted accordingly, with the probability of a liquidity event such as an IPO or sale remaining in the range of 55-60% and 15-20%, respectively. The probability of remaining a private company or liquidating remained in the range from 20-25% and 0-5%, respectively. In addition, our previously filed registration statement which was withdrawn in 2008 influenced the probability weighting.

We also considered the fact that our stockholders cannot freely trade our common stock in the public markets. The estimated fair value of our common stock at each grant date reflected a non-marketability discount partially based on the anticipated likelihood and timing of a future liquidity event.

In the contemporaneous valuations leading up to the filing of our prior registration statement on Form S-1 in November 2007, the non-marketability discount rate decreased over time as the perceived risk of completing an IPO was reduced. From December 2009 to June 2011 the contemporaneous valuations used to establish the fair value of our common stock assumed the expected length of time until, and probability of, an IPO remained the same based on overall market conditions at the time of each of the valuations and the non-marketability discount remained unchanged during that period.

There is inherent uncertainty in these forecasts and projections and if we had made different assumptions and estimates than those described above, the amount of our stock-based compensation expense, net loss and net loss per share amounts could have been materially different.

In connection with the preparation of our consolidated financial statements, we reassessed the fair value of our common stock for financial reporting purposes at interim dates between the contemporaneous valuations where there were stock option grants. For these interim periods we adjusted the fair value based on market conditions, progress made in our development programs and whether we achieved company milestones, when we deemed appropriate. Since December 2009, we had a number of developments in our business that we believe contributed to increase in the fair value of our common stock as discussed below.

Common stock valuations

Information regarding our stock option grants to our employees and non-employees along with the exercise price, which equals the estimated fair value of the underlying common stock for stock options issued since January 1, 2010 is summarized as follows:

Grant Date	Shares Subject to Options Granted	Exercise Price per Common Share	Fair Value per Common Share	Intrinsic Value per Common Share
March 4, 2010	46,450	$ 6.30	$ 6.30	
May 27, 2010	6,250	6.30	6.30	
August 10, 2010	423,628	6.30	6.30	
August 11, 2010	279,166	6.30	6.30	
November 17, 2010	9,000	6.30	6.30	
February 9, 2011 (unaudited)	32,062	6.60	6.60	
May 11, 2011 (unaudited)	1,500	6.60	6.60	
August 4, 2011 (unaudited)	346,617	6.90	6.90	
November 9, 2011 (unaudited)	10,500	6.90	6.90	

December 2009: As of December 2009, the results of our Phase II clinical trial of Traficet-EN demonstrated clinical efficacy in patients with moderate-to-severe Crohn s disease with a favorable safety and tolerability profile in both induction and maintenance periods of the study. Based on a confluence of data, GSK exercised its option to obtain an exclusive license for further development and worldwide commercialization of Traficet-EN. The option exercised by GSK constituted a material change in our business and financial position; we therefore conducted a contemporaneous valuation as of December 31, 2009. The valuation used a risk-adjusted discount of 18%, a

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non-marketability discount of 24% and an estimated time to a liquidity event of 12-18 months. Given that a potential liquidity event was estimated to be in the 12-18 month time horizon, we utilized the PWERM allocation model. The expected outcomes were weighted more toward an IPO (55-60%), with a lower weight for remaining private (20-25%), and a lower weight for a sale (15-20%) and with the lowest weights given to liquidation (0-5%). The valuation indicated a fair value of $6.30 per share for our common stock.

March 2010, May 2010, August 2010, and November 2010: During these periods, there were no material changes to our business, and therefore we did not adjust the fair value of our common stock as of March 2010, May 2010, August 2010 and November 2010.

December 2010: As of December 2010, we continued to make progress in our preclinical and clinical product portfolio. In December 2010, we completed a Phase II clinical trial for CCX140 for the treatment of type 2 diabetes which demonstrated that the compound was safe and well tolerated. In addition, in December 2010, we initiated a Phase I clinical trial of CCX832 and as a result, earned a milestone payment from GSK. The advancement of our clinical trials and the earned milestone payment constituted a significant change in our business and financial position, and we therefore conducted a contemporaneous valuation as of December 31, 2010. The valuation used a risk-adjusted discount of 18%, a non-marketability discount of 24% and an estimated time to a liquidity event of 12 months. The expected outcomes were weighted more toward an IPO (55-60%), with a lower weight for remaining private (20-25%), and a lower weight for a sale (15-20%), with the lowest weights given to liquidation (0-5%). The valuation indicated a fair value of $6.60 per share for our common stock.

February 2011 and May 2011: During these periods, we continued to advance our clinical pipeline consisting of several drug candidates including our lead compound under the GSK alliance, Traficet-EN, which entered pivotal Phase III clinical trials in January 2011 for the treatment of patients with moderate-to-severe Crohn s disease. We advanced our other clinical trial programs further into the clinic, however we did not expect data from these trials to be available at this time. As a result, there were no material changes to our business, and therefore we did not adjust the fair value of our common stock as of February 2011 or May 2011.

June 2011: As of June 2011, we completed enrollment of our Phase II study of CCX354 for the treatment of rheumatoid arthritis with data expected in the second half of 2011. We also initiated pre-study activities for our Phase II proof-of-concept clinical trial for CCX168 in patients with AAV. We conducted a contemporaneous valuation as of June 30, 2011. At that time, our board of directors had not approved moving forward with an IPO. The valuation used a risk-adjusted discount of 18%, a non-marketability discount of 24% and an estimated time to a liquidity event of 12-18 months. The expected outcomes were weighted more toward an IPO (55-60%), with a lower weight for remaining private (20-25%), and a lower weight for a sale (15-20%), with the lowest weights given to liquidation (0-5%). The valuation indicated a fair value of $6.90 per share for our common stock.

August 2011 and November 2011: During these periods, we continued to advance our clinical pipeline further in development and completed our Phase II study of CCX354 for the treatment of rheumatoid arthritis. Although GSK did exercise its option to obtain an exclusive license to further develop and commercialize CCX354 in November 2011, such exercise occurred subsequent to our November 9, 2011 stock option grants and therefore did not affect the fair market value we ascribed to our common stock in determining the option exercise price with respect to such grants. While we did not conduct any contemporaneous valuations of our common stock during this period, in November 2011 one of our executive officers sold 50,000 shares of our common stock at a price of $6.90 per share to an officer of one of our shareholders in a transaction negotiated at arms length.

Assuming an initial offering price of our common stock of $15.00 per share, which is the midpoint of the range set forth on the cover page of this prospectus, the public offering price of our common stock will exceed the exercise price of options issued by us on November 9, 2011 by $8.10 per share. We believe that this increase in the fair value of our common stock was primarily attributable to the following developments during the period:

- GSK s exercise of its option on November 28, 2011 to further develop and commercialize CCX354;
- GSK s $25.0 million payment to us in December 2011, with respect to exercising its option to further develop and commercialize CCX354;

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- the commencement, in December 2011, of dosing of patients with diabetic nephropathy in a Phase II clinical trial for CCX140, our lead independent drug candidate;
- the increased likelihood that we would complete our initial public offering in the first quarter of 2011;
- the decrease in the discount for a lack of marketability of our common stock based on the expected time to a liquidity event; and
- improving market conditions.

The exercise by GSK of its option to obtain a license to further develop and commercialize CCX354 contributed to the increase in the value of our common stock in several respects, including due to the following factors:

- GSK s exercise of its option with respect to CCX354, as it constitutes the second of three potential option exercises under our strategic alliance with GSK, helps to validate the viability both of our drug discovery platform and CCX354 s clinical efficacy in the treatment of rheumatoid arthritis;
- GSK s exercise of this option creates the potential that material milestone payments will be received with respect to this drug candidate;
- the exercise of an option to a second drug candidate provides the potential for a second revenue stream under our strategic alliance with GSK, thereby increasing the total revenue we may potentially realize under this strategic alliance;
- the exercise of an option to a second drug candidate decreases the likelihood that we will be completely dependent on a single drug candidate for our future revenues and that we will be subject to the risks associated with being dependent on a single drug; and
- as a result of GSK s exercise of its option to CCX354, GSK has become solely responsible for all further clinical development and commercialization expenditures worldwide with respect to CCX354 and its two designated back-up compounds, thereby freeing up resources for application to our other programs.

With respect to the commencement of dosing of patients with diabetic nephropathy in a Phase II clinical trial for CCX140, this development contributed to the increase in the fair market value of our stock during this period by moving this drug candidate closer to commercialization. As this drug candidate is not subject to our strategic partnership with GSK and we currently have exclusive development and commercialization rights, we could potentially retain a larger portion of any revenues generated by this drug candidate than we could with respect to drug candidates that our subject to our strategic collaboration with GSK.

Net Operating Loss Carryforwards

As of December 31, 2010, we had net operating loss and research and development tax credit carryforwards for federal income tax purposes of approximately $68.2 million and $4.5 million, respectively. The federal net operating loss and tax credit carryforwards will expire at various dates beginning in 2019 if not utilized. We also had net operating loss and research and development tax credit carryforwards for state income tax purposes of approximately $67.8 million and $2.5 million respectively. The state net operating loss carryforwards will expire at various dates beginning in 2014 if not utilized. The state research and development tax credits can be carried forward indefinitely.

Utilization of the net operating losses and credits may be subject to a substantial annual limitation due to ownership change limitations provided by the Internal Revenue Code of 1986 as amended. The annual limitation may result in the expiration of our net operating losses and credits before they can be used. We have recorded a valuation allowance for the full amount of the portion of the deferred tax asset related to our net operating loss and research and development tax credit carryforwards.

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Results of Operations

Comparison of Nine Months Ended September 30, 2010 and 2011

	Nine Months Ended September 30,		Change	
	2010	2011	2011 vs. 2010	%
	(in thousands)			
Revenue	$ 21,746	$ 5,621	$ (16,125)	(74%)
Research and development expenses	25,385	22,914	(2,471)	(10%)
General and administrative expenses	5,363	5,721	358	7%
Interest income	322	319	(3)	(1%)
Interest expense	(60)	(170)	(110)	(83%)
Other income	490	16	(474)	(97%)
Income tax benefit	73		(73)	(100%)

Revenue. We recognized revenue of $5.6 million in the nine months ended September 30, 2011 and $21.7 million in the same period in 2010. This decrease was primarily due to milestone payments received in connection with our GSK alliance during the nine months ended September 30, 2010 for the development candidate nomination of CCX832 and Phase I clinical trial initiation of CCX168. Total milestone payments recognized in the nine months ended September 30, 2010 were $15.0 million. No milestone payments were recognized in the same period in 2011.

Research and development expenses. Research and development expenses were $22.9 million in the nine months ended September 30, 2011 and $25.4 million in the same period in 2010. This decrease was primarily due to the completion of our Phase II clinical trial of CCX140 in type 2 diabetes in December 2010.

General and administrative expenses. General and administrative expenses were $5.7 million in the nine months ended September 30, 2011 and $5.4 million for the same period in 2010. This increase was primarily related to higher professional fees for legal and financial consulting services in connection with intellectual property and business development related activities, respectively.

Interest income, net. Interest income, net of interest expense, was $0.1 million in the nine months ended September 30, 2011 and $0.3 million in the same period in 2010.

Other income. Other income was $0.5 million for the nine months ended September 30, 2010. No other income was recognized in 2011. This decrease was due to the receipt of an insurance claim in the nine months ended September 30, 2010.

Comparison of Years Ended December 31, 2009 and 2010

	Year Ended December 31,		Change	
	2009	2010	2010 vs. 2009	%
	(in thousands)			
Revenue	$ 49,744	$ 34,861	$ (14,883)	(30%)
Research and development expenses	27,474	33,527	6,053	22%
General and administrative expenses	6,575	7,292	717	11%
Interest income	297	436	139	47%
Interest expense	(76)	(81)	(5)	(7%)
Other income		2,434	2,434	N/A
Income tax benefit (expense)	(293)	73	366	125%

Revenue. We recognized revenue of $34.9 million for the year ended December 31, 2010 and $49.7 million for the same period in 2009. This decrease was primarily due to higher milestone payments in 2009. In December 2009, GSK exercised its option to obtain an exclusive license for further development and worldwide commercialization of Traficet-EN. The associated option exercise fee of $35.0 million was recognized as revenue in full in the year ended December 31, 2009. Total milestones payments recognized in 2010 and 2009 were $25.0 million and $40.0 million, respectively.

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Research and development expenses. Research and development expenses were $33.5 million in 2010 and $27.5 million in 2009. This increase was primarily due to higher clinical expenses for CCX140, CCX354 and CCX168, reflecting further patient enrollment in the associated clinical trials for these drug candidates and for CCX832 for which Phase I clinical trials were initiated in 2010.

General and administrative expenses. General and administrative expenses were $7.3 million in 2010 and $6.6 million in 2009. This increase was primarily due to higher stock based compensation resulting from additional stock option grants and an increase in the fair value per share of our common stock during 2010.

Interest income, net. Interest income, net of interest expense, was $0.4 million in 2010 and $0.2 million in 2009. This increase was a result of higher cash and investment balances in 2010 primarily due to the receipt of milestone payments from GSK in 2010.

Other income. Other income was $2.4 million for the year ended December 31, 2010. No other income was recognized in 2009. This increase was due to the $1.9 million in government grants awarded from the United States Department of Treasury for eight projects under the Qualitative Therapeutic Discovery Project Program under the Patient Protection and Affordable Care Act of 2010 to support research with the potential to produce new therapies and $0.5 million for the receipt of an insurance claim in 2010.

Income tax benefit (expense). Income tax expense was $0.3 million in 2009. State income tax expense resulting from the state of California s temporary suspension of the use of net operating loss carrybacks in 2009 was partially offset by a federal income tax credit in the same period.

Comparison of Years Ended December 31, 2008 and 2009

	Year Ended December 31,		Change	
	2008	2009	2009 vs. 2008	%
		(in thousands)		
Revenue	$ 24,087	$ 49,744	$ 25,657	107%
Research and development expenses	35,056	27,474	(7,582)	(22%)
General and administrative expenses	9,157	6,575	(2,582)	(28%)
Interest income	1,762	297	(1,465)	(83%)
Interest expense	(129)	(76)	53	41%
Income tax benefit (expense)	23	(293)	(316)	(1,374%)

Revenue. We recognized revenue of $49.7 million in 2009 and $24.1 million in 2008. This increase was primarily due to higher milestone payments in 2009. In December 2009, GSK exercised its option to obtain an exclusive license for further development and worldwide commercialization of Traficet-EN. The associated option exercise fee of $35.0 million was recognized as revenue in full in the year ended December 31, 2009. Total milestones payments recognized in 2009 and 2008 were $40.0 million and $10.0 million, respectively.

Grant revenue was $0.5 million in 2008, reflecting the completion of efforts related to a grant from the National Institute of Allergy and Infectious Diseases. No grant revenue was recognized in 2009.

Research and development expenses. Research and development expenses were $27.5 million in 2009 and $35.1 million in 2008. This decrease was primarily due to lower medical and regulatory expenses of $7.6 million as a result of the completion of our Phase II trial for Traficet-EN, partially offset by higher clinical expenses for CCX140, CCX354 and CCX168, reflecting further patient enrollment in associated clinical trials for these drug candidates and for CCX832 for which Phase I clinical trials were initiated in 2010.

General and administrative expenses. General and administrative expenses were $6.6 million in 2009 and $9.2 million in 2008, representing a decrease of $2.6 million. This decrease was primarily a result of higher legal and financial professional fees associated with our previously filed registration statement which was withdrawn.

Interest income, net. Interest income, net of interest expense, was $0.2 million in 2009 and $1.6 million in 2008. This decrease was primarily due to a significantly lower interest rate environment in 2009.

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Income tax benefit (expense). Income tax expense was $0.3 million in 2009. State income tax expense resulting from the state of California s temporary suspension of the use of net operating loss carrybacks in 2009 was partially offset by a federal income tax credit in the same period.

Liquidity and Capital Resources

Since our inception, we have raised $384.9 million to fund our operations primarily through the private placement of equity, contract revenue under our collaborations, government contracts and grants and borrowings under equipment financing arrangements. As of September 30, 2011, we had approximately $81.2 million in cash, cash equivalents and investments. In December 2011, we received a $25.0 million option exercise payment under our strategic alliance with GSK with respect to CCX354 which is reflected in the collaboration funding described above. The following table shows a summary of our cash flows for each of the three years ended December 31, 2008, 2009 and 2010 and nine months ended September 30, 2010 and 2011.

	Years Ended December 31,			Nine Months Ended September 30,	
	2008	2009	2010	2010	2011
			(in thousands)		
				(unaudited)	
Cash provided by (used in)					
Operating activities	$ (22,758)	$ (22,961)	$ 17,664	$ 22,437	$ (12,904)
Investing activities	23,777	(29,915)	(27,629)	(33,923)	(3,295)
Financing activities	47,890	(1,639)	945	1,053	11,484

Operating activities. Net cash used in operating activities was $12.9 million for the nine months ended September 30, 2011, compared to net cash provided by operating activities of $22.4 million for the same period in 2010. This decrease was primarily due to the receipt by us in the nine months ended September 30, 2010 of a $35.0 million milestone payment from GSK in connection with the exercise of its option to obtain an exclusive license for further development and worldwide commercialization of Traficet-EN.

Net cash provided by operations was $17.7 million for the year ended December 31, 2010, compared to net cashed used in operations of $23.0 million in 2009. This increase was primarily due to the receipt of the $35.0 million milestone payment from GSK in 2010.

Net cash used in operations was $23.0 million for the year ended December 31, 2009, compared to $22.8 million for the year ended December 31, 2008. The use of cash in these periods was primarily due to the funding of our drug discovery and development efforts adjusted for non-cash charges and changes in components of working capital.

Investing activities. Net cash used in or provided by investing activities for periods presented primarily relate to the purchase, sale and maturity of investments used to fund the day-to-day needs of our business.

Financing activities. Net cash provided by financing activities was $11.5 million for the nine months ended September 30, 2011, compared to $1.1 million for the same period in 2010. This increase was primarily due to $10.0 million in proceeds from the issuance of a convertible note and $1.1 million in proceeds from the issuance of Series B convertible preferred stock in connection with the exercise of an associated warrant.

Net cash provided by financing activities was $0.9 million for the year ended December 31, 2010 compared to cash used in financing activities of $1.6 million for the same period in 2009. This increase was due to $1.5 million in proceeds received from the drawdown of our equipment line of financing in 2010. In addition, we repurchased $1.1 million in common stock from our Chief Executive Officer in 2009.

Net cash used in financing activities was $1.6 million for the year ended December 31, 2009, compared to net cash provided by financing activities of $47.9 million in 2008. The decrease in cash provided by financing activities was due to $48.7 million in net proceeds from the 2008 private placement of our Series E convertible preferred stock.

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We believe that our existing cash, cash equivalents and investments as of September 30, 2011, along with the estimated net proceeds from this offering, will be sufficient to meet our anticipated cash requirements for at least the next 12 months. However, our forecast of the period of time through which our financial resources will be adequate to support our operations is a forward-looking statement that involves risks and uncertainties, and actual results could vary materially.

Our future capital requirements are difficult to forecast and will depend on many factors, including:

- the achievement of milestones under our agreement with GSK;
- the terms and timing of any other collaborative, licensing and other arrangements that we may establish;
- the initiation, progress, timing and completion of preclinical studies and clinical trials for our drug candidates and potential drug candidates;
- the number and characteristics of drug candidates that we pursue;
- the progress, costs and results of our clinical trials;
- the outcome, timing and cost of regulatory approvals;
- delays that may be caused by changing regulatory requirements;
- the cost and timing of hiring new employees to support our continued growth;
- the costs involved in filing and prosecuting patent applications and enforcing and defending patent claims;
- the costs and timing of procuring clinical and commercial supplies of our drug candidates;
- the costs and timing of establishing sales, marketing and distribution capabilities; and
- the extent to which we acquire or invest in businesses, products or technologies.

Contractual Obligations and Commitments

The following is a summary of our long-term contractual cash obligations as of December 31, 2010.

	Payments Due by Period	
	Total	

		Less than One Year	1-3 Years	3-5 Years	More than 5 Years
			(in thousands)		
Operating lease[1]	$ 2,975	$ 844	$ 1,816	$ 315	$
Equipment financing obligations (including interest)[2]	1,493	407	815	271	
Total contractual obligations	$ 4,468	$ 1,251	$ 2,631	$ 586	$

(1) We lease our facility in Mountain View, California. The lease expires in 2014.

(2) In February 2007, we entered into a $2.5 million credit facility with Silicon Valley Bank to finance equipment purchases and other related expenses. In April 2010, we modified this credit facility to increase its limit to $4.0 million until March 31, 2011. As of September 30, 2011 we had drawn $2.2 million under this modified credit facility. The loans under this credit facility are secured by certain of our assets and are being repaid over 48 months. Interest rates are fixed at the time of drawdown, with effective rates ranging from 6.3%-7.0%.

In September 2011, we entered into a convertible note loan agreement with Techne pursuant to which we issued a convertible note with a principal amount of $10.0 million, bearing interest at a rate of 5.0% per annum and maturing in September 2021. Upon completion of this offering, all outstanding principal and interest under this note will automatically convert into shares of our common stock at a conversion price equal to the initial public offering price of our common stock. See Certain Relationships and Related Party Transactions.

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We enter into contracts in the normal course of business with CROs for clinical trials and clinical supply manufacturing and with vendors for preclinical research studies, research supplies and other services and products for operating purposes. These contracts generally provide for termination on notice, and therefore are cancelable contracts and not included in the table of contractual obligations and commitments.

Off-Balance Sheet Arrangements

We do not have any off-balance sheet arrangements (as defined by applicable SEC regulations) that are reasonably likely to have a current or future material effect on our financial condition, results of operations, liquidity, capital expenditures or capital resources, except warrants and stock options.

Quantitative and Qualitative Disclosure About Market Risk

The primary objectives of our investment activities are to ensure liquidity and to preserve principal while at the same time maximizing the income we receive from our marketable securities without significantly increasing risk. Some of the securities that we invest in may have market risk. This means that a change in prevailing interest rates may cause the principal amount of the marketable securities to fluctuate. To minimize the risk in the future, we intend to maintain our portfolio of cash equivalents and short term investments in a variety of securities, including commercial paper, money market funds, government and non-government debt securities and corporate obligations. Because of the short-term maturities of our cash equivalents and marketable securities, we do not believe that an increase in market rates would have any significant impact on the realized value of our marketable securities.

Recent Accounting Pronouncements

In October 2009, the FASB issued Accounting Standard Update No. 2009-13, *Revenue Recognition Multiple Deliverable Revenue Arrangement*, or ASU 2009-13. ASU 2009-13 provides application guidance on whether multiple deliverables exist, how the deliverables should be separated and how the consideration should be allocated to one or more units of accounting. This update establishes a selling price hierarchy for determining the selling price of a deliverable. The selling price used for each deliverable will be based on vendor-specific objective evidence, if available, third-party evidence if vendor-specific objective evidence is not available, or estimated selling price if neither vendor-specific or third-party evidence is available. We adopted this guidance prospectively beginning on January 1, 2011 and there was no cumulative effect upon adoption. Under ASU 2009-13, we may be required to exercise considerable judgment in determining the estimated selling price of delivered items under new agreements and our revenue under new agreements may be more accelerated as compared to the prior accounting standard. As such, the adoption of ASU 2009-13 could have a material impact on our financial statements going forward.

In April 2010, the FASB issued Accounting Standard Update No. 2010-17, *Revenue Recognition Milestone Method*, or ASU 2010-17. ASU 2010-17 provides guidance in applying the milestone method of revenue recognition to research or development arrangements. Under this guidance, companies may recognize revenue contingent upon the achievement of a milestone in its entirety, in the period in which the milestone is achieved, only if the milestone meets all the criteria within the guidance to be considered substantive. ASU 2010-17 is effective on a prospective basis for research and development milestones achieved in fiscal years beginning on or after June 15, 2010. We adopted this standard on a prospective basis on January 1, 2011 with no impact.

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BUSINESS

Overview

ChemoCentryx is a biopharmaceutical company focused on discovering, developing and commercializing orally-administered therapeutics to treat autoimmune diseases, inflammatory disorders and cancer. Our approach has been to target the chemokine system, a network of molecules including chemokine ligands and their associated receptors, as well as related chemo-attractant receptors, all of which are known to drive inflammation. Chemokine ligands concentrate at the site of an inflammatory event, serving as signals that attract and guide inflammatory cells to the tissue, where, based on the chemokine ligand and receptor combination, a specific inflammatory response is initiated. In certain diseases, discrete chemokine receptors that play a specific role in the pathology of interest have been identified, and the therapeutic goal is to specifically inhibit that receptor to provide clinical benefit. Accordingly, each of our drug candidates is a small molecule designed to target a specific chemokine or chemo-attractant receptor, thereby blocking the inflammatory response driven by that particular chemokine while leaving the rest of the immune system unaffected. Using our pioneering insights and proprietary technologies designed to better understand the chemokine system, we believe that we have established the broadest pipeline of novel drugs targeting chemokine receptors. Our compounds are designed to be highly potent, selective to minimize the risk of off-target effects and orally-available for improved patient compliance. As small molecules, they are also easier and less costly to manufacture than protein therapeutics, or biologics.

We currently have four drug candidates in clinical development, and expect to advance one additional drug candidate into clinical development in 2012. Two of these drug candidates are wholly owned and are being developed independently by us while three are subject to our collaboration agreement with Glaxo Group Limited, or GSK, an affiliate of GlaxoSmithKline. Under this agreement, GSK has exercised its options to obtain exclusive licenses to further develop and commercialize Traficet-EN and CCX354 and each of their two respective defined back-up compounds and will have a similar option right to CCX168 if we establish clinical proof-of-concept.

All of our drug candidates have been internally discovered and include:

- Traficet-EN (CCX282 or GSK 786) Our most advanced drug candidate, currently in three pivotal Phase III clinical trials being conducted by our partner GSK for the treatment of patients with moderate-to-severe Crohn s disease;

- CCX140 Our lead independent drug candidate successfully completed a Phase II clinical trial in type 2 diabetics and is currently in two Phase II clinical trials in patients with diabetic nephropathy, a form of kidney disease;

- CCX354 Successfully completed a Phase II proof-of-concept clinical trial for the treatment of rheumatoid arthritis, or RA, and GSK exercised its option to obtain an exclusive license for the further development and commercialization of this drug candidate and its two identified back-up compounds in November 2011;

- CCX168 Currently in a Phase II proof-of-concept clinical trial for the treatment of anti-neutrophil cytoplasmic antibody, or ANCA-associated vasculitis, or AAV, and is subject to GSK s option; and

- CCX662 Our independent drug candidate for the treatment of glioblastoma multiforme, or GBM, is expected to enter a Phase I clinical trial in the second half of 2012.

We are also advancing several additional independent drug candidates through preclinical development, the most advanced of which target chemokine receptors involved in atopic dermatitis, inflammatory bowel disease, or IBD, including ulcerative colitis, liver inflammation, psoriasis, and RA.

Traficet-EN, our most advanced drug candidate, is intended to control the inflammatory response underlying IBD by targeting the chemokine receptor known as CCR9. In adults, CCR9 is found primarily on a population of T cells, a subset of the body s inflammatory cells, that migrate selectively to the digestive tract. It is believed that

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when CCR9 s ligand, CCL25 (also known as TECK), is over-expressed, the migration of T cells to the small and large intestine causes persistent inflammation that may result in Crohn s disease or ulcerative colitis, the two forms of IBD. We have completed nine clinical trials with Traficet-EN in a total of 785 subjects, including five Phase I clinical trials (three in the United States and two in the United Kingdom), one Thorough QT study in the United States (an assessment of cardiovascular safety which is required for regulatory approval), and three Phase II clinical trials (one in the Netherlands, the United Kingdom, and the United States, one in Finland and one (PROTECT-1) in Australia, Austria, Belgium, Brazil, Bulgaria, Canada, the Czech Republic, Denmark, France, Germany, Hungary, Israel, the Netherlands, Poland, South Africa, Sweden and the United Kingdom). We completed our PROTECT-1 Phase II clinical trial in 436 patients with moderate-to-severe Crohn s disease in 2009. Results from this clinical trial indicated that Traficet-EN was effective in inducing a clinical response over a 12-week treatment period. The results also indicated that Traficet-EN was effective in maintaining clinical remission over a 36-week treatment period. Traficet-EN was safe and well tolerated in all clinical trials completed to date. In December 2009, GSK exercised its option to obtain an exclusive license to further develop and commercialize Traficet-EN. To date, GSK has initiated three pivotal Phase III clinical trials with Traficet-EN in Crohn s disease. These studies are currently being conducted in Australia, Belgium, Canada, the Czech Republic, Denmark, France, Germany, Japan, Poland, the Netherlands, Norway, Sweden, the United Kingdom and the United States. If approved, Traficet-EN would be the first oral agent with a novel mechanism of action introduced for the treatment of Crohn s disease since the introduction of corticosteroids and oral immunosuppressants.

CCX140, our lead independent drug candidate, targets the chemokine receptor known as CCR2. CCX140 is a potent and selective antagonist of CCR2 that is found on subsets of monocytes and macrophages, which are cells of the immune system believed to play an important role in inflammatory processes. Blocking CCR2 is intended to reduce the abnormal monocyte and macrophage driven inflammatory response implicated in renal disease. In addition, it has been shown that levels of CCL2, the main ligand for CCR2, are elevated in the kidneys of patients with diabetic nephropathy, which is characterized by a persistent and usually progressive decline in renal function. Current treatments of patients with diabetic nephropathy primarily focus on treatment of the underlying type 2 diabetes and hypertension. Given that the current standard of care does not halt or reverse the progression of diabetic patients with impaired kidney function to end-stage renal disease, we believe that an unmet medical need persists for the treatment of diabetic nephropathy. As a precursor to our clinical trials in patients with diabetic nephropathy, in January 2011, we completed a 159-patient randomized Phase II clinical trial, conducted in Australia, the Czech Republic, Germany, Hungary and New Zealand, to assess the safety and tolerability of CCX140 in patients with type 2 diabetes, the most common cause of diabetic nephropathy. CCX140 was safe and well tolerated in this trial. In addition, CCX140 demonstrated biological activity through a dose-dependent decrease in fasting plasma glucose. The highest dose of 10mg CCX140 administered once-daily also lowered hemoglobin A1c, or HbA1c, with statistical significance compared to placebo over a four-week period. CCX140 is currently in two Phase II clinical trials in patients with diabetic nephropathy and we expect to complete these clinical trials by the end of 2012, provided that we do not increase the sample size of, or add additional dose groups to, the larger of these clinical trials. One trial is being conducted in Belgium, the Czech Republic, Germany, Hungary and the United Kingdom, and the other is being conducted in the Netherlands.

CCX354 targets the chemokine receptor known as CCR1. Synovial fluid from the joints of RA patients contains high levels of activated CCR1 chemokine ligands. Blocking CCR1 is intended to reduce inflammation and prevent subsequent joint destruction by suppressing the infiltration of inflammatory cells into the arthritic joint. We successfully completed two Phase I clinical trials in a total of 84 healthy subjects, conducted in Switzerland followed by a Phase I/II clinical trial in 24 patients with stable RA, conducted in Belgium and Romania, and a Phase II proof-of-concept clinical trial in 160 patients with moderate-to-severe RA, conducted in Belgium, the Czech Republic, Germany, Hungary, Poland, Romania and the Ukraine. Results from the Phase II proof-of-concept clinical trial demonstrated that CCX354 was safe and well tolerated by patients with RA in this trial, and demonstrated clinical and biological activity at a dose of 200mg of CCX354 once-daily. This successful clinical trial triggered GSK s option rights under our collaboration agreement. GSK exercised its option to further develop and commercialize CCX354 in November 2011 and has an exclusive right to initiate a Phase IIb clinical trial for CCX354 in RA.

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CCX168 targets the chemo-attractant C5a receptor, or C5aR, which binds to a biologically activated fragment of the complement protein known as C5. Chemo-attractant receptors are related to the chemokine receptor family and similarly regulate the migration of certain types of inflammatory cells. C5aR is thought to play a role in a range of inflammatory and autoimmune diseases such as AAV, lupus and psoriasis. We completed a Phase I clinical trial for CCX168, conducted in Switzerland, which showed that CCX168 was well tolerated at doses up to 100mg. We initiated a Phase II proof-of-concept clinical trial in AAV in the fourth quarter of 2011 and expect to complete this clinical trial by the end of 2012. This clinical trial is being conducted in Belgium, Czech Republic, Germany, Hungary, the Netherlands, Poland, Sweden and the United Kingdom. If this clinical trial is successfully completed, GSK may exercise its option to further develop and commercialize CCX168.

With the exception of PROTECT-1, our Crohn s Disease trial for Traficet-EN, we have conducted the majority of our Phase I and Phase II clinical trials in Europe. At this time no decision has been made regarding where future Phase I and Phase II clinical trials will be conducted. Our planned future Phase III clinical trials for CCX140 will be conducted in the United States, Europe and possibly other countries outside of the United States and Europe which have not yet been determined.

For any drug candidate for which GSK exercises its option under the collaboration agreement, GSK would be solely responsible for all further clinical development and commercialization expenditures worldwide with respect to that drug candidate and its two designated back-up compounds. Upon the exercise of any of these options, we would receive an option exercise fee and would become eligible to receive regulatory and commercial milestone payments, as earned under the terms of our agreement, and royalties on the net sales of licensed drugs. GSK has already exercised its option to Traficet-EN and CCX354. If GSK does not exercise its option to CCX168, we will evaluate our alternatives for further development of this drug candidate, which may entail internally developing it or identifying other collaboration partners for its development.

CCX662 is our independent drug candidate that is a highly potent and selective small molecule compound which targets CXCR7, a novel chemokine receptor that we believe plays a key role in the survival of certain tumor cells. Of particular interest is the role that CXCR7 appears to play in the development of GBM, the deadliest of all brain cancers. In animal studies, CCX662 demonstrated safety and efficacy against GBM. We are in preclinical development with this drug candidate and anticipate initiation of Phase I clinical trials in patients with GBM in the second half of 2012.

We have developed a suite of proprietary technologies, which we call the EnabaLink drug discovery engine, to better understand the chemokine system and to accelerate the identification of small molecule lead compounds that target and inhibit the function of specific chemokine receptors. We believe this platform provides us with an advantage in the rapid identification of highly specific drug candidates. An important element of this platform is our thorough map of the chemokine network, which allows us to better understand how a given chemokine-chemokine receptor interaction impacts the migration of cells in a given disease. With this understanding, we can apply our advanced screening methodologies, including a purpose-built high-throughput robotic screening technology, known as the Reverse Activation of Migration, or RAM, Assay, to identify small molecule antagonists for the chemokine receptor most closely associated with a specific disease. The RAM Assay is designed to markedly reduce or eliminate non-specific inhibitors and toxic inhibitors of cell migration, resulting in highly specific lead candidates. This technology allows us to screen against targets that are not accessible with traditional technologies, providing us with what we believe to be a competitive advantage in drug discovery. We have used our EnabaLink drug discovery engine in our drug candidate programs and continue to apply these powerful research tools in our early stage drug discovery efforts.

Thomas Schall, Ph.D., our founder, President and Chief Executive Officer, has more than 25 years of research experience in the field of chemokine biology and has contributed broadly to the understanding of chemokines and their receptors in human disease. Since our founding, we have raised $384.9 million, of which $175.0 million has been in the form of convertible debt and equity financings and $209.8 million in the form of collaboration funding and government contracts and grants. As of September 30, 2011, we had $81.2 million of cash, cash equivalents and investments. In December 2011, we received a $25.0 million option exercise payment under our strategic alliance with GSK with respect to CCX354 which is reflected in the collaboration funding

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described above. We believe that our broad pipeline of oral drug candidates, our ability to advance unique, highly specific compounds into and through clinical development across diverse indications and our proprietary drug discovery technologies provide us with distinct advantages that will enable us to continue to exploit the extensive pharmacologic potential of the chemokine system.

Strategy

Our strategy includes the following key elements:

Collaborate with our partner GSK in the Phase III clinical development and commercialization of Traficet-EN and in the further development and commercialization of CCX354. Prior to the exercise of GSK s option to Traficet-EN, our most advanced drug candidate, we conducted all of the preclinical and clinical trials for Traficet-EN, including a 436-patient Phase II clinical trial called PROTECT-1 in patients with moderate-to-severe Crohn s disease. We will continue to collaborate with GSK, in the clinical development and commercialization of Traficet-EN in this and other indications, and in the further development and commercialization of CCX354, for which GSK exercised its option in November 2011.

Forward integrate into a commercial biopharmaceutical company by driving the development and commercialization of our lead independent drug candidate, CCX140. We have initiated two Phase II clinical trials in patients with diabetic nephropathy. If we successfully complete the Phase II program for CCX140, we plan to initiate Phase III clinical trials either alone or together with a co-development partner. We plan to retain commercial rights to CCX140 in North America and seek a partner for co-development and commercialization outside North America.

Advance our other drug candidate under our collaboration with GSK. We also plan to continue advancing our other clinical program under our collaboration with GSK, CCX168, currently in a Phase II proof-of-concept clinical trial in patients with AAV.

Expand our clinical stage portfolio of internally discovered, independent drug candidates. We intend to advance CCX662, currently in preclinical development, into clinical trials in patients with GBM, in the second half of 2012. We will also explore new therapeutic applications for other independent programs currently in preclinical development, including those targeting the CCR4, CCR6, CXCR6 and CCR9 receptors in various inflammatory diseases and cancer.

Leverage our expertise and proprietary technologies to continue discovering and developing a broad pipeline of novel chemokine-based therapeutics. We intend to use our RAM Assay to screen against additional chemokine receptors and chemo-attractant receptors and expect to continue to discover and validate novel chemokine receptors by using our proprietary EnabaLink suite of drug discovery technologies. We plan to use our proprietary technologies to internally discover and develop a balanced portfolio of independent and partnered compounds.

Commercialize our drug candidates in specialty markets in North America and partner outside North America and in primary care markets worldwide. We intend to retain significant rights to our independent drug candidates which address specialty market opportunities such as renal disease and certain cancers. We plan to build capabilities to market our drugs to physician specialists in North America and seek commercialization partners for our drugs marketed to physician specialists outside of North America and to primary care physicians worldwide.

Focusing on the Chemokine System

Understanding Inflammation

The human immune system serves to protect the body against infections and injuries. It recognizes these threats and quickly mounts a defensive response. Inflammation is a key component of the immune response and

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serves as one line of defense to infection, irritation or injury as immune system cells attempt to suppress and control an infectious agent, such as bacteria, or to break down and carry away damaged tissue, as in the case of injury. Specialized white blood cells known as antigen presenting cells such as macrophages and lymphocytes are mobilized to the affected tissue and work in concert to recognize, neutralize and eliminate the perceived threat. Macrophages and other antigen presenting cells pick up and ingest foreign materials and present the threatening antigens to lymphocytes, also known as T cells and B cells. T cells in turn destroy infected cells or coordinate other inflammatory cells, such as B cells, which produce antibodies, or proteins with the ability to neutralize antigens, to bind with the antigen leading to the destruction of the foreign agent. Macrophages then dispose of dead cells and debris.

Acute inflammation is characterized by the rapid onset of pain, heat, redness, swelling and loss of function. When inflammation is long-term, or chronic, and is directed at the body s own tissues, this can result in various forms of autoimmune disease. Different autoimmune diseases tend to affect different tissues or organs. For example, in Crohn s disease certain inflammatory cells attack tissues predominantly in the digestive tract, while in RA a different set of inflammatory cells is involved in attacking the tissues that make up the joints between bones. While the cause of autoimmune diseases is not known, we and others have demonstrated that the self-perpetuating, tissue-damaging inflammation associated with these conditions is in part characterized by dysregulation of the chemokine system.

IBD, RA, AAV, lupus and skin inflammatory diseases such as psoriasis and atopic dermatitis, are all examples of chronic conditions in which an inappropriate inflammatory response underlies disease. In recent years, conditions that were not previously considered to be the result of inflammation, such as type 2 diabetes, chronic kidney disease and cancer, have joined the long list of human diseases thought to be the result, at least in part, of uncontrolled and chronic inflammation. Many autoimmune diseases are highly debilitating, creating a significant social and financial burden. According to the National Institutes of Health, there were more than 80 clinically distinct autoimmune diseases which collectively affected as many as 23.5 million people in the United States in 2010.

Role of Chemokines in Disease

Inflammation involves a complex series of cellular events that rely on chemical messengers known as chemokines, or ***chemo***-attractant cyto***kines***, which send out signals to attract inflammatory cells, or leukocytes, to the site of disease or injury. Chemokines bind to chemokine receptors found on the surface of leukocytes, and the specific combination of various chemokines and chemokine receptors serve to precisely coordinate the inflammatory response.

The chemokine system, including chemokines and chemo-attractants, directs inflammatory responses, serving to precisely coordinate immune system cell movement. The human chemokine network is made up of more than 50 known chemokine ligands and approximately 25 identified chemokine receptors. Some chemokines are known to bind to more than one chemokine receptor. Certain receptors can bind to multiple chemokines, while other chemokine receptors only bind to a single ligand. Different chemokines are made in different tissues at different times and different chemokine receptors are expressed on the surface of different types of inflammatory cells. Those cells can only respond to a chemokine in a given organ or tissue if the cell possesses a receptor that specifically recognizes the chemokine that is present in the local environment. In this way, each chemokine-chemokine receptor combination may direct a different inflammatory response and this response can be tailored by the body based on the type of injury, irritation or other threat.

Inappropriate activity of the chemokine network is at the core of numerous autoimmune and inflammatory conditions. For example, in Crohn s disease dysregulation of either the chemokine CCL25 or the chemokine receptor to which it binds, CCR9, is thought to selectively attract inflammatory T cells to and subsequently attack tissues in the digestive tract. As drivers of the inflammatory response, chemokines and their receptors present opportunities for the development of new therapies. By selectively blocking a given chemokine-chemokine receptor combination, and largely leaving other chemokine-chemokine receptor interactions unaffected, we believe even aggressive forms of chronic inflammation and autoimmune diseases can potentially be brought under control in a safe, effective manner.

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Chemokines are also involved in the causes of diseases that were not historically classified as inflammatory. For example, CCR2 is responsible for the recruitment of monocytes from blood into the adipose tissue and liver of obese, insulin-resistant individuals. The monocytes mature into inflammatory macrophages within these tissues and interfere with the biochemical signals involved in the regulation of glucose levels, often resulting in type 2 diabetes.

Research also indicates that chemokines may contribute to inflammatory damage by direct activation of non-inflammatory cells that are part of the affected tissue. For example, evidence indicates that certain cells within diabetic kidneys begin to express CCR2 on their surface and become impaired due to the resulting increased levels of the chemokine CCL2 found in such kidneys.

In addition to its central role in autoimmune and inflammatory conditions, the chemokine system plays an important role in other diseases, such as cancer. It is known that tumors induce the expression of chemokines that are involved in promoting the growth of blood vessels that feed tumors, providing a link to the chemokine system s role in the establishment and spread of cancer. In addition, certain chemokine ligands and their corresponding receptors have been implicated in the survival, growth and metastasis of human cancer. The chemokine system is likely a more recent evolutionary branch of other chemo-attractant systems in the body such as the complement system. The complement system includes the protein C5a, which under certain conditions has pro-inflammatory effects.

Our Drug Candidates and Preclinical Programs

Traficet-EN for Inflammatory Bowel Disease

Understanding Crohn s Disease

IBD refers to two diseases, Crohn s disease and ulcerative colitis, both characterized by inflammation of the gastrointestinal tract. Both Crohn s disease and ulcerative colitis are chronic and recurring autoimmune conditions. Researchers believe that these conditions occur when the body s inflammatory cells become overreactive to microbes in the gastrointestinal tract, such as bacteria normally found in the intestines for digestion, and mount a destructive inflammatory response.

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According to the Crohn s and Colitis Foundation of America, or CCFA, Crohn s disease is estimated to affect as many as 700,000 Americans. According to National Digestive Diseases Information Clearinghouse, men and women are affected equally by the disease. While patients may be of any age, Crohn s disease is primarily a disease that commences in adolescents and young adults, with onset between the ages of 15 and 35.

Crohn s disease is chronic; patients suffer periods of flare-ups or periods characterized by intense symptoms, interspersed with periods of remission where symptoms decrease or disappear. Symptoms may range from mild to severe and can include persistent diarrhea, abdominal pain, fever, and rectal bleeding, as well as loss of appetite and subsequent weight loss. Crohn s disease patients will experience ulcerations that penetrate deeply into the mucosal tissues that line the walls of the bowel. Crohn s disease may involve the entire length of the gastrointestinal tract from the mouth to the anus, but the most typical areas of involvement are in the small intestine and colon. Complications of Crohn s disease include obstruction or blockage of the intestine due to scar tissue build-up and nutritional deficiencies. Ulcerative lesions associated with the disease can, on occasion, completely penetrate the bowel wall, leading to painful fistula formation, or an abnormal break or opening in the bowel wall, which can cause infectious complications that require surgical intervention.

Patients with Crohn s disease are typically referred to and treated by gastroenterologists. Prior to referral and accurate diagnosis, the condition may go undiagnosed or misdiagnosed as a variety of gastrointestinal ailments. Crohn s disease often mimics other conditions and symptoms may vary widely, presenting challenges for an accurate diagnosis. Crohn s disease is diagnosed through a colonoscopy, mucosal biopsy and small bowel x-ray to rule out other conditions such as ulcerative colitis and to ascertain which parts of the digestive tract are involved and establish a baseline for monitoring treatment and management. The primary goals for drug therapy are to induce and maintain significant clinical improvement or remission, resulting in improvement of active symptoms.

We believe that Crohn s disease represents a significant underserved clinical problem with high medical and economic costs and a large impact on quality of life. The disease is strongly linked to work disability and unemployment, as 48% of Crohn s disease patients were employed full-time at the time of diagnosis of their disease, and 39% of patients were unemployed, according to a 2005 publication in the Journal of Clinical Gastroenterology. Healthcare costs are significant, as patients typically require daily drug therapy over many years to control symptoms and maintain remissions, and a number of patients require multiple hospitalizations and, in some cases, surgeries over the course of their illness. In addition, currently available treatments for Crohn s disease are often associated with adverse reactions that may require frequent monitoring and impact patient compliance.

Limitations of Current Therapies

Crohn s disease is a chronic condition, and consequently patients continue on a given therapeutic regimen over the course of a lifetime. Current treatments for Crohn s disease are directed toward bringing a patient s active disease, or acute flare-ups, under control or into remission. The initial induction therapy is often followed by chronic maintenance therapy to preserve the remission or to keep disease manifestations at a minimal level. If the disease continues to progress, patients continue on a given therapeutic regimen from the time of diagnosis forward, adding additional therapies as flare-ups recur or persist. Over their lifetimes, Crohn s disease patients will typically use a broad array of drugs, often in combination, to seek improvement of their symptoms. Many patients also often require one or more surgical procedures over the course of their lifetimes to treat the disease.

In spite of the existence of a number of medications for the treatment of Crohn s disease, according to a 2001 publication in Alimentary Pharmacology Therapeutics, a study showed that 25% of patients had active disease every year, while another 53% fluctuated between years in remission and years in relapse. Only 22% of patients experienced long-term remissions. When medications can no longer control symptoms, patients have few or no options beyond surgery. According to the CCFA, as of the end of 2009, an estimated 75% of Crohn s disease patients eventually required one or more hospitalizations or surgeries to repair a fistula or fissure, to remove an intestinal obstruction or to treat an intestinal abscess, and approximately 50% of adult patients had a recurrence of symptomatic Crohn s disease within five years after having surgery. There is no known cure for Crohn s disease.

For patients with mild-to-moderate disease, aminosalicylates, a type of oral non-steroidal anti-inflammatory drug, are considered a first-line therapeutic approach. These drugs, also called 5-ASAs, may be administered for years after a patient receives an initial diagnosis, though there is limited clinical evidence demonstrating their efficacy in inducing remission of the disease. Oral antibiotics are also used as a primary therapy, as some researchers believe that these drugs may be useful in reducing intestinal bacteria and indirectly suppressing the patient s immune system. If patients symptoms progress after these treatments, they are often given a multi-week course of oral steroid therapy to treat the flare-up.

For patients with moderate-to-severe disease, immunosuppressants and corticosteroids, in some cases administered intravenously, are prescribed for active disease. Corticosteroids are often used in conjunction with immunosuppressants as they may provide a more immediate impact and have shown some efficacy in treating patients with active Crohn s disease. Immunosuppressants can take up to four months to induce remission and are associated with increased susceptibility to serious infections. Individual immunosuppressive agents have other serious toxic effects, including chronic liver, lung, and kidney damage, or acute inflammation of the pancreas. In addition, profound side effects are known to occur with long-term corticosteroids use, including weight gain, elevated blood sugar, high blood pressure, vision problems such as glaucoma and cataracts, fragile bones and increased susceptibility to infections. Once the flare-up subsides, the patient may be maintained on immunosuppressants, or in some cases with additional oral corticosteroids therapies.

For patients whose active Crohn s disease cannot be controlled with immunosuppressants or corticosteroids, TNF-a inhibitors such as Remicade (infliximab), sold by Johnson & Johnson, are now commonly used to treat severe cases of Crohn s disease. TNF-a inhibitors block the natural activity of tumor necrosis factor-alpha, or TNF-a, a cytokine present in large quantities as part of the downstream inflammatory response triggered by chemokines in Crohn s disease. Other TNF-a inhibitors marketed in the United States for the treatment of Crohn s disease include Humira (adalimumab), sold by Abbott Laboratories, and Cimzia (certolizumab pegol), sold by UCB. Tysabri (natalizumab), an anti-alpha4 integrin antibody, sold by Biogen Idec, that blocks alpha4 integrin, which is present on several inflammatory cell types, is also used. Remicade and Tysabri are administered by intravenous infusion. The other therapies are given by subcutaneous injection.

Patients who experience acute flare-ups are generally treated with three infusions of Remicade at zero, two and six weeks and the patient is generally continued indefinitely on a maintenance regimen of infusions every eight weeks. The formation of anti-infliximab antibodies to infliximab treatment is associated with a loss of clinical response, infusion reactions and discontinuation of treatment. This may occur in these patients due to the development of neutralizing antibodies against the therapy as a result of its nonhuman components. Humira is typically administered once every other week. Cimzia is given subcutaneously at zero, two and four weeks, and every four weeks thereafter, if the patient responds favorably to the drug. Tysabri is given every four weeks by intravenous infusion. Amgen s Enbrel (etanercept) has also been studied in IBD and is sometimes prescribed off-label for its treatment.

While TNF-a inhibitors as a class and Tysabri represent clear advances in the treatment of Crohn s disease, they act by down-regulating the body s inflammatory response in a generalized manner and are not tissue-specific. Due at least in part to their broad activity, TNF-a inhibitors are associated with serious adverse side effects including an increased risk of life-threatening infections, such as sepsis. TNF-a inhibitors have also been associated with such serious risks as drug-induced lupus, reactivation of latent tuberculosis, and increased risk of developing lymphoma or other blood or neurological disorders. Treatment with TNF-a inhibitors is also expensive, with the combined costs of the drug and its administration often exceeding $20,000 per year per patient, according to The Pain Practitioner, *Spring 2011 Pharmacologic Approaches to Rheumatoid Arthritis*. Tysabri has been associated with the rapidly progressive, often fatal complication of progressive multifocal encephalopathy, which arises from an often latent virus that a compromised immune system can no longer suppress.

For Crohn s disease patients, one or more surgeries are a radical, but unfortunately common treatment of last resort. Crohn s disease patients often undergo repeated procedures for the removal of sections of diseased bowel to treat complications such as severe bleeding, inflammatory obstruction of the flow of bowel contents, or for removal of fistulous tracts through which bowel contents drain into the abdominal space or externally to the body surface.

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The ChemoCentryx Solution: Traficet-EN A Novel CCR9 Antagonist

Traficet-EN is being developed as a first-in-class oral anti-inflammatory agent for the treatment of IBD, including Crohn s disease and ulcerative colitis. Traficet-EN is orally administered, which we believe would be an important improvement in patient convenience and potentially patient compliance as compared to existing intravenous and subcutaneous treatments with biologics. We believe that the combination of Traficet-EN s specificity and the convenience of oral administration may make this a treatment of choice for IBD patients. In addition, as a synthetic small molecule, the drug may have significant cost-of-goods advantages over protein therapeutics, or biologics, such as TNF-a inhibitors.

Traficet-EN Mechanism of Action in IBD

Traficet-EN is designed to prevent the migration of inflammatory cells to the digestive tract by blocking the function of CCR9, which is found primarily on a subset of inflammatory T cells that plays a key role in the development of both forms of IBD. T cell migration into the digestive tract is guided by a chemokine called CCL25 found in the intestines. CCL25-mediated activation of CCR9 draws T cells into the intestinal tissue, where they release inflammatory mediators that can ultimately lead to tissue damage. In adults, CCL25 only signals CCR9-expressing T cells to migrate to the gastrointestinal tract, and blocking CCR9 is not thought to interfere with cell migration elsewhere. Therefore, by blocking CCR9, Traficet-EN may be able to halt the inflammatory response underlying IBD without otherwise impacting the patient s immune system. In addition, by interrupting CCR9 signaling, we believe Traficet-EN may hasten the elimination of inflammatory T cells from the intestines, thus potentially speeding recovery by reducing the longevity of flare-ups associated with IBD. We believe this mechanism of action would allow Traficet-EN, if approved, to be a highly effective treatment with significantly fewer side effects than currently available immunosuppressive therapies, including TNF-a inhibitors.

Traficet-EN Drug Development Strategy and Clinical Trials

We have completed nine clinical trials with Traficet-EN in a total of 785 subjects, including five Phase I clinical trials in a total of 151 subjects, one Thorough QT study in 57 subjects demonstrating cardiovascular safety, two Phase II clinical trials in 510 patients with Crohn s disease and one Phase II clinical trial in 67 patients with celiac disease. The largest of these Phase II clinical trials, PROTECT-1, was conducted in 436 patients with moderate-to-severe Crohn s disease. Results from this clinical trial indicated that Traficet-EN was effective in inducing a clinical response over a 12-week treatment period in these patients. Furthermore, the results indicated that Traficet-EN was also effective in maintaining clinical remission over a 36-week treatment period. Traficet-EN was safe and well tolerated in all clinical trials completed to date. In December 2009, GSK exercised its option to obtain an exclusive license to Traficet-EN and its two designated back-up compounds and is now solely responsible for all further clinical development and commercialization expenditures worldwide.

To date, three pivotal Phase III clinical trials have been initiated by GSK with Traficet-EN in Crohn s disease. The pivotal Phase III clinical trials are designed to support the use of Traficet-EN to induce clinical response or remission of Crohn s disease, and to provide maintenance of remission for Crohn s disease.

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PROTECT-1 Crohn s Disease Clinical Trial

PROTECT-1 was designed to demonstrate Traficet-EN s efficacy as an oral treatment capable of inducing and maintaining clinical response or remission among patients with moderate-to-severe Crohn s disease. This was a double-blind, randomized, placebo-controlled clinical trial, and enrolled 436 patients with an active Crohn s disease flare-up and Crohn s Disease Activity Index, or CDAI, scores of 250 to 450 and C-reactive protein, or CRP, levels of at least 7.5mg/L (CRP being a more objective, but non-specific, marker of inflammation). The clinical trial was conducted in 17 countries at more than 100 study centers. The clinical trial included four separate study periods:

1. A 12-week induction period during which all patients received either placebo, 250mg once-daily (QD), 500mg once-daily or 250mg twice-daily (BID) of Traficet-EN orally. The purpose of this study period was to determine Traficet-EN s ability to induce clinical response or remission in patients with active Crohn s disease.

2. A 4-week active treatment period during which all patients received 250mg of Traficet-EN twice-daily orally.

3. A 36-week maintenance period during which all patients with a 70-point-or-greater drop in CDAI scores at the end of the 4-week active treatment period relative to their baseline entry level criteria were re-randomized to either 250mg of Traficet-EN twice-daily or placebo given orally. The purpose of this study period was to determine Traficet-EN s ability to maintain clinical response or remission in patients with Crohn s disease.

4. A 4-week follow-up period during which all patients were followed for safety after the conclusion of dosing of Traficet-EN.

The CDAI is a research tool used for distinguishing a patient s level of disease from inactive to severely active disease. CDAI scores can range from zero to 600, and a patient experiencing an active Crohn s disease flare-up can be expected to have a CDAI score of 220 to 450 for moderate-to-severe disease and above 450 for severe to very severe disease. Clinical remission is considered to be a CDAI score of less than 150. For a treatment to be regarded as effective, researchers look for a drop in CDAI of at least 70 to 100 points. CDAI is currently the only measure regarded by regulatory agencies as an appropriate endpoint to assess the efficacy of a given drug in Crohn s disease.

Overall, the clinical trial demonstrated evidence of efficacy in both the induction of treatment response as well as the maintenance of remission for patients with moderate-to-severe Crohn s disease when treated with Traficet-EN. Furthermore, Traficet-EN was shown to be safe and well tolerated over the one-year course of this clinical trial.

More specifically, data reported from the 12-week induction period of the PROTECT-1 clinical trial showed that the 500mg once-daily oral dose of Traficet-EN in patients with small bowel and/or colonic Crohn s disease was consistently superior to placebo across key efficacy endpoints, including CDAI, normalization of CRP, and Crohn s Disease Endoscopic Index of Severity, or CDEIS.

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500mg Once-Daily Traficet-EN Showed Statistically Significant CDAI ³ 100-Point Response at Week 12

As shown below, the difference in clinical response rate between the 500mg once-daily Traficet-EN group and placebo increased at each timepoint during the 12-week induction period: 2% at Week 4; 7% at Week 8; and 15% at Week 12. The CDAI ³ 100-point response was 55% in the 500mg once-daily group versus 40% for placebo at Week 12 (p=0.029). At Week 12, the CDAI ³ 70-point response was 61% in the 500mg once-daily group versus 47% for placebo (p=0.039).

Approximately 26% of patients had previously received anti-TNF agents and 53 of these were non-responsive to one or more of these anti-TNF agents based on data collected during screening. An exploratory analysis showed that in these patients CDAI ³ 70-point and CDAI ³ 100-point responses at Week 12 occurred in 8 of 14 (57%) patients in the 500mg once-daily group compared to 5 of 18 (28%) patients in the placebo group. Remission was observed in 3 of 14 (21%) patients in the 500mg once-daily group and 1 of 18 (6%) patients in the placebo group.

500mg Once-Daily Traficet-EN Significantly Reduced Endoscopic Lesions of Crohn s Disease

A decrease in CRP confirmed the effect of 500mg once-daily Traficet-EN and, as shown below, colonoscopic evidence of improvement based on CDEIS was observed in the 500mg once-daily Traficet-EN group compared to placebo (p=0.049). Only a subset of the patients in the clinical trial consented to serial invasive endoscopy and that subset is presented in the chart below.

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Traficet-EN, But Not Placebo, Showed a CDAI Remission Rate of Approximately 50% Over the Course of the Maintenance Period

Maintenance of clinical remission (CDAI £150) showed statistically significant separation between the 250mg twice-daily Traficet-EN and placebo groups. As shown below, data from the maintenance period of PROTECT-1 showed that Traficet-EN treatment was able to maintain the clinical remission rate at 47% to 50%, whereas the remission rate dropped progressively from 50% to 31% in the placebo group over the course of the maintenance period. At week 36 of the maintenance period, which was week 52 of the clinical trial overall, 47% of the patients in the Traficet-EN group were in remission compared to 31% in the placebo group (p=0.011). Furthermore, 41% of patients in the Traficet-EN group were in corticosteroid-free remission compared to 28% in the placebo group (p=0.041).

We also demonstrated that Traficet-EN may reduce the need for corticosteroids. Approximately 11% of patients in the Traficet-EN group compared to 21% in the placebo group started or increased corticosteroid treatment during the maintenance period (p=0.04) and 57% of patients receiving Traficet-EN were able to stop their corticosteroid treatment compared to 43% of patients receiving placebo. CRP normalized in 19% of patients in the Traficet-EN group compared to 10% in the placebo group (p=0.041).

Traficet-EN was safe and well tolerated with no evidence of immune system compromise when administered orally for up to 12 consecutive months to patients in the PROTECT-1 clinical trial. More specifically, in the induction period, a similar proportion of patients in the placebo and Traficet-EN overall groups, 62.5% and 59.8% of patients, respectively, reported treatment-emergent adverse events, or TEAEs, during the clinical trial. Also, a similar proportion of patients in the placebo and Traficet-EN overall groups, 10.4% and 8.6% of patients, respectively, reported treatment-emergent serious adverse events, or SAEs, during the induction period of the clinical trial. In the maintenance period, a similar proportion of patients in the placebo and Traficet-EN groups reported TEAEs and SAEs leading to discontinuation of the clinical trial treatment. One patient died more than one month after discontinuing early from the clinical trial. This was considered by the clinical investigator to be related to worsening of underlying Crohn s disease and not related to treatment with Traficet-EN. The patient received 250mg twice-daily of Traficet-EN in the induction and active treatment periods. No serious or opportunistic infections were observed in patients receiving Traficet-EN and there were no safety concerns based on the other safety evaluations including laboratory data, vital signs and ECGs.

Phase III Clinical Program

GSK has initiated three pivotal Phase III clinical trials intended to obtain the clinical results necessary to apply for marketing approval for Traficet-EN in Crohn s disease. In general, the development approach of the Phase III program is modeled after the design of our PROTECT-1 clinical trial. The following pivotal Phase III clinical trials are currently ongoing:

SHIELD-1 is a multi-national, randomized, double-blind, placebo-controlled clinical trial to evaluate the efficacy and safety of two doses, 500mg once-daily and 500mg twice-daily, of Traficet-EN over 12 weeks of treatment in approximately 600 adult patients with moderate-to-severe Crohn s disease. Patient recruitment was initiated in December 2010.

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SHIELD-2 is a multi-national, randomized, double-blind, placebo-controlled clinical trial to evaluate the efficacy and safety of two doses, 500mg once-daily and 500mg twice-daily, of Traficet-EN in maintaining disease remission over 52 weeks in approximately 750 adult patients with Crohn s disease. Eligible patients will have achieved disease improvement and/or remission in SHIELD-1. Patient recruitment was initiated in April 2011.

SHIELD-3 is a multi-national, open-label clinical trial to evaluate the safety and effectiveness of 500mg twice-daily of Traficet-EN over 108 weeks in approximately 800 adult patients with Crohn s disease. Patients completing previous clinical trials with the drug or patients who withdraw early from the SHIELD-2 maintenance clinical trial may be eligible to participate. Patient recruitment was initiated in April 2011.

Traficet-EN Commercialization Strategy

Following the exercise of its option, GSK became solely responsible for all further clinical development and commercialization of Traficet-EN and its two designated back-up compounds worldwide. However, we have the option, which we can exercise at our sole discretion, to co-promote Traficet-EN to physician specialists in the United States, subject to our payment of 35% of GSK s development costs. Under the terms of the agreement, our promotional efforts could be as high as 50% of all promotional efforts to physician specialists in the United States. As consideration for our promotional efforts, GSK would be required to pay us an amount similar to what it would pay a third-party contract sales force.

Additional Indications for Traficet-EN

Ulcerative colitis is an additional indication for which Traficet-EN could be developed. Preclinical studies showed that Traficet-EN can prevent the onset of and treat large bowel inflammation in mice, and may have the potential to treat ulcerative colitis in humans. We have also recently demonstrated that, contrary to the prevailing assumption regarding the absence of CCL25 from the colon, increased levels of CCL25 are present in colonic tissue from human patients with IBD, which further supports our belief that Traficet-EN has the potential for treating ulcerative colitis.

CCX140 for Diabetic Nephropathy and Other Renal Diseases

Understanding Diabetic Nephropathy and Limitations of Current Therapies

Diabetic nephropathy is a common disease among patients with diabetes and hypertension. It is characterized by a persistent and usually progressive decline in renal function, as measured by glomerular filtration rate, a measure of the rate of fluid filtration in the kidney, and/or albuminuria, a condition where elevated protein levels are present in the urine, which can be an indicator of kidney damage.

Given the rise in the incidence of obesity, type 2 diabetes and hypertension, the associated incidence of diabetic nephropathy has reached epidemic proportions in industrialized nations. According to the National Kidney Foundation, as of the end of 2010, an estimated 26 million Americans had chronic kidney disease and patients with diabetic nephropathy form the largest segment (approximately 35%) of the renal disease patient population. Diabetic nephropathy is the leading cause of a condition known as end-stage renal disease, or ESRD, the most severe stage of chronic kidney disease. ESRD patients impose a significant economic burden on the United States, constituting approximately 1.3% of Medicare beneficiaries but accounting for approximately 8.1% of Medicare expenditures, according to the 2011 annual report of the United States Renal Data System. Consequently, we believe that a drug which would delay or prevent the onset of ESRD would have significant pharmaco-economic benefits.

Current treatment options for patients with diabetic nephropathy mainly include drugs that treat the underlying conditions of diabetes and hypertension. Angiotensin receptor blockers, or ARBs, and angiotensin converting enzyme, or ACE, inhibitors are commonly prescribed to control hypertension and slow the progression of diabetic nephropathy. Even with these therapies, about 20% of patients with diabetic nephropathy will progress into ESRD, at which point patients must rely on regular dialysis sessions or a kidney transplant in order to survive.

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A number of experimental treatments for diabetic nephropathy are currently being evaluated in clinical trials, although none of them have yet demonstrated clear and convincing benefit in large long-term clinical trials, either in terms of slowing or reversing disease progression. Several of these treatments aim to interfere with the inflammatory or fibrotic processes that are now recognized as central to the disease.

The ChemoCentryx Solution: CCX140 A Novel CCR2 Antagonist

While historically diabetic nephropathy was not considered an inflammatory disease, there is now clear evidence of the role of macrophages in this disease. Kidney biopsies from patients with diabetic nephropathy show elevated numbers of macrophages in the glomeruli, which are the basic filtering elements in the kidney. It has also been shown that the extent of tissue damage in the interstitial areas surrounding the proximal tubules, which are the second component of the filtering apparatus in the kidney, is strongly correlated with the numbers of macrophages present. Experimental studies in preclinical diabetic models have clarified that monocyte and macrophage infiltration occurs at early stages of disease and that this infiltration correlates with renal injury.

In recent years, CCR2 has been identified as the main driver of monocyte and macrophage recruitment into diseased kidneys. Levels of CCL2, the main ligand for CCR2, are elevated in the kidneys of patients with diabetic nephropathy. CCL2 is produced by kidney cells in response to high glucose levels, and levels of CCL2 in the urine are strong indicators of renal damage and correlate well with albuminuria and interstitial macrophage numbers.

We have utilized various animal models to study the relationship between CCR2 inhibition and renal function. Data generated from these models have confirmed and expanded observations made by other independent investigators in preclinical models of diabetic nephropathy indicating that CCR2 inhibition leads to pronounced reduction in albuminuria, as well as improvement in markers of renal function. We have also demonstrated that CCR2 inhibition provides benefit in several models of non-diabetic nephropathy. The following table summarizes the key findings from each of these animal models:

Summary of Findings From CCR2 Inhibition in Animal Models of Nephropathy

Biological Parameter	Effect of CCR2 Inhibition
Albuminuria	Reduced
Hyperglycemia	Reduced
Glomerular Filtration Rate	Decreased hyperfiltration
Serum Markers of Renal Function	Reduced serum creatinine and blood urea nitrogen levels
Histological Improvements	Reduced number of renal interstitial macrophages
	Reduced percentage of glomeruli with mesangiolysis
	Increased podocyte density

Data from preclinical studies indicate that CCX140 is a potent and selective antagonist of CCR2 which is required for monocytes to infiltrate the inflamed kidney, where they differentiate into macrophages. While CCX140 is not the first CCR2 antagonist to advance into clinical trials, we believe that it is unique in a number of ways, including its high selectivity for CCR2 relative to other chemokine receptors such as CCR5. We believe that CCX140 also distinguishes itself from other CCR2 antagonists in that it has been shown preclinically to be free of the cardiovascular safety signals associated with other CCR2 antagonists. CCX140 has been shown in a number of preclinical toxicology studies to be suitable for evaluation in humans for chronic use in diabetic nephropathy. Comparative pre-clinical studies with a compound with a completely different mode of action, such as bardoxolone methyl, have revealed that CCR2 inhibition reduces urinary protein while bardoxolone methyl does not appear to have a meaningful effect on this parameter.

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CCX140 Drug Development Strategy and Clinical Trials

Our clinical development strategy was to first assess the safety and tolerability of CCX140 in patients with type 2 diabetes and normal renal function prior to evaluation of the drug in patients with diabetic nephropathy. As a precursor to our clinical trials in patients with diabetic nephropathy, in January 2011, we completed a 159-patient randomized Phase II clinical trial to assess the safety and tolerability of CCX140 in patients with type 2 diabetes, the most common cause of diabetic nephropathy. CCX140 is currently in two Phase II clinical trials in diabetic nephropathy and we expect to complete these clinical trials by the end of 2012.

CCX140 Phase I Clinical Trials

We conducted a randomized, double-blind, placebo-controlled, single ascending dose Phase I clinical trial in 56 healthy subjects in which single oral doses of 0.05mg, 0.1mg, 0.3mg, 0.6mg, 1mg, 3mg, and 10mg of CCX140 were compared to placebo. CCX140 was well tolerated by most clinical trial subjects and no SAEs were observed. All observed adverse events were either mild or moderate in intensity and no subjects were withdrawn from the clinical trial due to adverse events. There did not appear to be a relationship between CCX140 dose level and the overall subject incidence of adverse events in the clinical trial. The pharmacokinetic, or PK, profile of CCX140 showed dose-proportionality.

In addition, we conducted a randomized, double-blind, placebo-controlled, multiple ascending dose Phase I clinical trial in 32 healthy subjects in which once-daily oral doses of 0.6mg and 2mg of CCX140 for seven days, and 5mg and 10mg of CCX140 for ten days were compared to placebo. CCX140 was well tolerated by most clinical trial subjects and no SAEs were observed. All adverse events were either mild or moderate in intensity and no subjects were withdrawn from the clinical trial due to adverse events. The PK profile was consistent with the single-dose clinical trial.

To enable us to potentially evaluate doses higher than 10mg in our ongoing Phase II clinical trial in patients with diabetic nephropathy, we are currently conducting an additional Phase I clinical trial at doses of 5mg, 10mg, 12.5mg and 15mg once-daily.

CCX140 Phase II Clinical Trial in Type 2 Diabetes

Our Phase II clinical trial was designed to demonstrate safety of CCX140 in patients with type 2 diabetes and normal renal function and the effect of CCX140 on glycemic indices. We conducted a randomized, double-blind, placebo and active controlled clinical trial in 159 patients with type 2 diabetes on a stable dose of metformin for at least eight weeks, with 32 patients receiving placebo, 32 receiving pioglitazone hydrochloride (an approved therapeutic for type 2 diabetes serving as the active control), 63 receiving 5mg of CCX140 and 32 receiving 10mg of CCX140 orally once-daily for 28 days.

The clinical trial met its primary objective by demonstrating the safety and tolerability of CCX140 in these patients. In addition, CCX140 showed encouraging signs of biological activity. For example, patients receiving CCX140 experienced a dose-dependent decrease in fasting plasma glucose, or FPG, from baseline to day 29 of the trial period, or Day 29. The least squares mean change from baseline to Day 29 in FPG was -4.3mg/dL for 5mg CCX140, -16.1mg/dL for 10mg CCX140, -10.7mg/dL for placebo, and -21.4mg/dL for 30mg pioglitazone hydrochloride. The decrease in FPG observed in the active control group receiving 30mg pioglitazone hydrochloride was in line with the anticipated decrease of 25mg/dL as reported in published literature. Decreases in FPG from baseline to Day 29 for the pioglitazone hydrochloride and CCX140 groups were not statistically different when compared to placebo. However, the decrease in FPG in the 10mg CCX140 group was comparable to the 30mg pioglitazone hydrochloride response observed over four weeks of treatment.

Patients in the 10mg CCX140 group also experienced a statistically significant (p=0.045 vs. placebo) decrease from baseline in HbA1c, one of the most important glycemic indices, indicating an improvement in glycemic control which may be an added benefit when treating patients with diabetic nephropathy. The least squares mean change from baseline in HbA1c was -0.09% for 5mg CCX140, -0.23% for 10mg CCX140, -0.09%

for placebo, and -0.13% for 30mg pioglitazone hydrochloride. In addition, the fructosamine levels, another index of glycemic control, trended lower in the CCX140 and pioglitazone hydrochloride groups, but did not reach statistical significance compared to placebo. Plasma CCL2 and circulating monocyte levels were unchanged by CCX140 treatment.

No SAEs were observed in patients receiving CCX140 treatment. The incidence of TEAEs across treatment groups was similar. Two patients discontinued the clinical trial due to a TEAE. One patient, in the 5mg CCX140 group, experienced a TEAE of dyspepsia, which led to discontinuation of clinical trial medication. The TEAE was considered by the clinical investigator to be moderate in severity and possibly related to clinical trial medication. A second patient, in the 10mg CCX140 group, experienced a TEAE of gouty arthritis, which led to discontinuation of clinical trial medication. This patient had a medical history of gout and this adverse event was considered by the clinical investigator to be severe in intensity and probably not related to CCX140. The most commonly observed TEAE in patients receiving CCX140 was hypertension, which occurred in three patients in the 5mg CCX140 group. Review of the blood pressure data from the subjects with TEAEs of hypertension, as well as the overall group blood pressure data, did not reveal any significant worsening trend in blood pressure with CCX140 treatment compared to placebo. No adverse effects of hypertension were observed in patients in the 10mg CCX140 group.

CCX140 treatment did not negatively affect the patients serum lipid profiles (total cholesterol, HDL and LDL cholesterol, triglycerides, and non-esterified fatty acids) over the four-week treatment period. Changes in ECG were not clinically meaningful and there was no detrimental effect on renal function observed.

Current and Future CCX140 Clinical Development in Diabetic Nephropathy

CCX140 is currently in two Phase II clinical trials in patients with diabetic nephropathy. The first randomized, double-blind, placebo-controlled Phase II clinical trial will enroll at least 135 patients. The primary objective of this clinical trial will be to evaluate the safety and tolerability of CCX140 in patients with diabetic nephropathy. Secondary objectives include evaluation of the effect of CCX140 on albuminuria as well as HbA1c. The three treatment groups will consist of placebo, 5mg and 10mg of CCX140 and the treatment duration will be 12 weeks, with a four-week follow-up period. Following an interim analysis for efficacy evaluation, the sample size may be increased to up to 270 patients, and/or additional dose groups may be added. These potential modifications to the dose groups will allow us to increase the probability of obtaining statistically significant results. Patients with residual albuminuria, despite being on a stable therapeutic dose of an ACE inhibitor or ARB will be included in this clinical trial. The key efficacy endpoint is change from baseline in first morning urinary albumin:creatinine ratio, a major indicator of renal function. Provided that we do not increase the sample size or add additional dose groups, we expect to complete this clinical trial by the end of 2012.

The second randomized, double-blind, placebo-controlled Phase II clinical trial we are conducting is in 20 patients with diabetic nephropathy. The primary objective of this clinical trial is to evaluate the effect of CCX140 on 24-hour urinary albumin excretion. The two treatment groups consist of placebo and 10mg of CCX140. The treatment duration is 12 weeks, with a four-week follow-up period. Patients with residual albuminuria, despite being on a stable therapeutic dose of an ACE inhibitor or ARB will be included in this clinical trial. We expect to complete this clinical trial by the end of 2012.

If the Phase II clinical program indicates that CCX140 is safe and efficacious in treating patients with diabetic nephropathy, we plan to conduct end-of-Phase II meetings with the FDA and European Medicines Agency, at which time the clinical results from the CCX140 program will be reviewed and a Phase III clinical program will be discussed. It is anticipated that the Phase III program will include at least 1,500 patients. We expect that the patient population from the first Phase III clinical trial will be large enough to assess the rate of major cardiovascular events in order to exclude the possibility of a cardiovascular safety signal with CCX140.

CCX140 Commercialization Strategy

We plan to retain commercial rights to CCX140 in North America and intend to build a specialty sales force to call on nephrologists who treat diabetic nephropathy patients. There are approximately 8,300 nephrologists in

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the United States. We believe that a moderately sized sales force will be sufficient to call on all key prescribing nephrologists in this market. In addition, we plan to seek partners for co-development and commercialization of CCX140 outside North America.

CCX354 for Rheumatoid Arthritis

Understanding Rheumatoid Arthritis

RA, a chronic inflammatory disease, is among the most debilitating of all forms of arthritis, causing pain, stiffness, swelling and limitation in the motion and function of multiple joints which may eventually become deformed. Sometimes these symptoms make even the simplest activities, such as walking, difficult to manage. The exact cause of RA is unknown, but it is believed to result from the body s immune system attacking the synovium, which is the tissue that lines a patient s joints.

As of 2006, more than two million Americans suffered from RA, according to a report by Data Monitor. RA is two to three times more common in women than in men and generally strikes between the ages of 20 and 50, but it can also affect young children and adults older than age 50.

Limitations of Current Therapies

Although therapy for patients with RA has improved dramatically over the last 25 years, there is still no cure and no single therapy which is effective for all patients. Many patients will need to change treatment strategies during the course of their disease due to lack of efficacy and adverse side effects. People with RA, particularly those whose disease is not well controlled, may have a higher risk of other diseases such as osteoporosis, or thinning of bone resulting in fracture.

Current treatment options for RA consist of corticosteroids such as prednisone, immunosuppressants such as methotrexate, TNF-a inhibitors such as Remicade, Humira and Cimzia, and other biologic agents such as Rituxan (rituximab) and Orencia (abatacept). Corticosteroids may provide relief during a disease flare-up, but often have serious side effects, including high blood pressure, osteoporosis, reduced ability to fight infections, mood swings, diabetes and gastric ulcers. Immunosuppressants have anti-inflammatory and bone-sparing effects, but the general suppression of the immune system leads to increased risk of infection. The use of these drugs may lead to mouth sores, stomach ulcers, and low white blood counts, and can cause severe toxicity of the liver and bone marrow, which require regular monitoring with blood testing.

Biologic agents have been approved by the FDA to treat moderate-to-severe RA that has not responded to an adequate trial of one or more of the traditional courses of treatment. Biologic agents must be given by subcutaneous injection or by intravenous infusion. According to The Pain Practitioner, *Spring 2011 Pharmacologic Approaches to Rheumatoid Arthritis*, annual treatment cost with biologic agents range from $13,000 to $30,000 per patient depending on the drug used and the dose administered. Biologic agents are effective in some patients to treat the signs and symptoms of RA and the bone erosive effects of the disease. However, their use may lead to serious side effects including serious infections, tuberculosis, an increased risk of lymphoma and serious hypersensitivity reactions.

The ChemoCentryx Solution: CCX354 A Novel CCR1 Antagonist

Over 20 years ago, Dr. Schall and his colleagues reported the initial cloning and characterization of the chemokine receptor that came to be known as CCR1. There is strong evidence implicating CCR1 in the pathology of RA; first, CCR1-expressing monocytes and macrophages are consistently found at high levels in the synovium of RA patients, and second, the C6 superagonists of CCR1, which are activated highly potent forms of certain CCR1 ligands, are consistently detected at high levels in synovial fluids from RA patients. Blocking CCR1 is intended to reduce the inflammation and prevent subsequent joint destruction by suppressing the infiltration of inflammatory cells into the arthritic joint.

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We are currently developing a new class of proprietary small molecule CCR1 inhibitors and have selected CCX354 as a drug candidate. CCX354 was designed by optimization of a chemical lead that was discovered using our EnabaLink drug discovery engine and is chemically distinct from all known inhibitors of this receptor. Our preclinical data suggest that the compound selectively inhibits CCR1-mediated migration of monocytes and does not inhibit migration of inflammatory cells mediated by other chemokine receptors, even when the compound is given at high doses. We believe that this high degree of target specificity is an important safety feature that may allow CCX354 to be effective while avoiding unwanted side effects associated with existing injectable biologics and other immunosuppressive agents currently used to treat RA.

Preclinical studies indicate that CCX354 has a favorable safety and PK profile. In animal studies, CCX354 is well absorbed when given orally and the compound is well tolerated at dose levels much higher than those required to cause inhibition of CCR1 function. In *in vitro* and animal studies, because of its potency and selectivity, CCX354 did not appear to have the safety concerns that have hindered others from successfully developing compounds blocking this receptor. In addition, CCX354 does not significantly inhibit the activity of a class of liver protein which is necessary for metabolizing other common drugs that patients may be taking. We believe that this lack of interference suggests that the compound may be safely administered along with other medications used concurrently by RA patients.

CCX354 Drug Development Strategy and Clinical Trials

We have completed two Phase I clinical trials with CCX354 in 84 healthy subjects, a Phase I/II clinical trial in 24 patients with RA, and a Phase II proof-of-concept clinical trial in 160 patients with moderate-to-severe RA partially responsive to methotrexate. In November 2011, GSK exercised its option to obtain an exclusive license to CCX354 and its two designated back-up compounds and is now solely responsible for all further clinical development and commercialization expenditures worldwide.

CCX354 Phase I Clinical Trials

We completed a randomized, double-blind, placebo-controlled, single ascending dose Phase I clinical trial in 48 healthy volunteers in which single oral doses of 1mg, 3mg, 10mg, 30mg, 100mg and 300mg of CCX354 were compared to placebo. CCX354 appeared to be well tolerated by most subjects in this clinical trial. No SAEs were reported and no subjects were withdrawn from the clinical trial due to adverse events. The subject incidence of adverse events was relatively similar across treatment groups. The most common adverse events in subjects receiving CCX354 were dizziness, headache, dizziness postural, nasopharyngitis, and pharyngolaryngeal pain and there was no evidence that the incidence of adverse events was CCX354 dose-dependent. There were no safety concerns of subjects receiving CCX354 based on review of hematology, serum chemistry, urinalysis, vital signs, ECG results, or physical examination results from this clinical trial.

We also completed a multiple ascending dose Phase I clinical trial in healthy volunteers, which included oral dose regimens of 3mg, 30mg and 300mg of CCX354 once-daily and 10mg, 30mg and 100mg of CCX354 twice-daily for seven days. CCX354 was well tolerated by most clinical trial subjects, no SAEs were reported and no subjects were withdrawn from the clinical trial due to adverse events. The subject incidence of adverse events was relatively similar across treatment groups, 70% in the CCX354 group compared to 83% in the placebo group. All adverse effects were either mild or moderate in intensity. The most common adverse events in subjects receiving CCX354 were headache, flatulence, sense of hair loss, ocular hyperemia, abdominal pain, diarrhea, dizziness, and pharyngolaryngeal pain. There was no evidence that the incidence of these most common adverse events was CCX354 dose-dependent.

We evaluated the safety and tolerability of 100mg or 200mg of CCX354 daily in 24 patients with stable RA on a stable dose of methotrexate in a Phase I/II clinical trial. An important secondary objective of this clinical trial was to assess the PK profile of CCX354 and to determine if there was any interaction between CCX354 and methotrexate. Doses of 100mg of CCX354 once-daily, 100mg of CCX354 twice-daily and 200mg of CCX354 once-daily or placebo were given for 14 days. There were no SAEs, TEAEs leading to death, or TEAEs leading

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to discontinuation of treatment and the proportion of patients with TEAEs was the same in the placebo group and the CCX354 group overall. All adverse effects were mild or moderate in severity. The PK profile of CCX354 was not significantly affected by methotrexate co-administration and the PK profile of methotrexate was also not significantly affected by CCX354 co-administration, indicating that there is no negative drug-drug interaction between these two drugs.

CCX354 Phase II Clinical Trial in Rheumatoid Arthritis

We have successfully completed a randomized, double-blind, placebo-controlled Phase II proof-of-concept clinical trial in 160 patients with RA on a stable dose of methotrexate for at least eight weeks. Patients received either placebo, 100mg of CCX354 twice-daily or 200mg of CCX354 once-daily for 84 days, followed by 28 days without treatment. The primary objective of the clinical trial was to evaluate the safety and tolerability of CCX354 in patients with moderate-to-severe RA. Key secondary objectives included assessment of the effect of CCX354 on RA disease activity measured by the ACR response criteria, Disease Activity Score 28-CRP, the ACR components and bone resorption markers. ACR20, ACR50 and ACR70 responses refer to patients who achieve a 20%, 50% and 70% improvement, respectively, according to criteria set by the American College of Rheumatology, or ACR. Patients who met inclusion criteria at the start of dosing demonstrated an ACR20 response at Week 12 of 56% in subjects receiving 200mg of CCX354 once-daily compared to 44% in subjects receiving 100mg of CCX354 twice daily, and 30% in subjects receiving placebo. The ACR20 response difference between the 200mg CCX354 once-daily and placebo groups was statistically significant (p=0.014). Patients who met inclusion criteria at the start of dosing and who did not previously receive biologic agents, such as anti-TNF drugs, demonstrated an ACR20 response at Week 12 of 62% in subjects receiving 200mg of CCX354 once-daily compared to 41% in subjects receiving 100mg of CCX354 twice-daily and 26% in subjects receiving placebo. The ACR20 response difference between the 200mg CCX354 once-daily and placebo groups was statistically significant (p=0.002). The decrease in CRP was statistically significant in the 200mg CCX354 once-daily group compared to placebo at Week 12 (p=0.023). There was a median CRP decrease from baseline to Week 12 of 33% in the 200mg CCX354 once-daily group, 30% in the 100mg twice-daily group and 11% in the placebo group in subjects eligible at the start of dosing. Similarly, there was a median CRP decrease from baseline to Week 12 of 38% in the 200mg CCX354 once-daily group, 30% in the 100mg twice-daily group and 10% in the placebo group in subjects eligible at the start of dosing who also did not previously receive biologic agents. ACR50, ACR70, Disease Activity Score 28-CRP, and ACR component results indicated greatest efficacy in the 200mg CCX354 once-daily group. Decreases in bone turnover markers were more pronounced in the CCX354 groups compared to placebo and reached statistical significance at several time points during the study. Clinical responders had higher plasma CCX354 concentrations than non-responders. CCX354 was safe and well tolerated by study subjects. No SAEs were observed in the 200mg CCX354 once-daily or placebo groups. Four SAEs were reported in the 100mg CCX354 twice-daily group, none considered related to CCX354 use by the study investigators. These were vasovagal reaction, or fainting, following blood draw, non-cardiac related chest pain, myocardial infarction, or heart attack, and psychomotor epilepsy, which was observed one week after stopping study medication. No significant safety issues were observed regarding safety laboratory parameters including hepatic, renal, metabolic and hematologic data.

Future CCX354 Clinical Development in Rheumatoid Arthritis

CCX354 is subject to our collaboration agreement with GSK and in November 2011 it exercised its option to obtain a license to further develop and commercialize CCX354 and its two defined back-up compounds for all indications, including RA. Upon exercising its option, GSK became solely responsible for all further clinical development and commercialization expenditures worldwide with respect to CCX354 and its two designated back-up compounds and has an exclusive right to initiate a Phase IIb clinical trial for CCX354 in RA.

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CCX168 for ANCA-Associated Vasculitis

Understanding ANCA-Associated Vasculitis

AAV is a type of autoimmune disease caused by autoantibodies, which are abnormal antibodies that attack one s own cells and tissues. ANCAs are autoantibodies that attack a certain type of white blood cells called neutrophils. When ANCAs attack these neutrophils, they cause these cells to attack the walls of small blood vessels in different tissues and organs of the body, which causes AAV. AAV encompasses various conditions including:

Renal limited vasculitis or ANCA glomerulonephritis: the blood vessel damage occurs in the kidneys. No other organs are affected.

Microscopic polyangiitis: caused by injury to blood vessels in multiple tissues at the same time. It can be seen in the kidneys, skin, nerves, and lungs.

Granulomatosis with polyangiitis (Wegener s granulomatosis): the blood vessel damage occurs in connection with a process called granulomatous inflammation. This often affects the lung, sinuses, nose, eyes or ears.

In AAV, activation of the complement cascade, an escalating group of inflammatory responses, leads to production of the very potent chemo-attractant factor C5a. This in turn leads to the attraction and activation of neutrophils and other white blood cells, which play a key role in the disease.

Limitations of Current Therapies

AAV currently is treated with high-dose corticosteroids and cyclophosphamide, azathioprine, mycophenolate mofetil, rituximab, and plasma exchange in severe cases. Even though these approaches may induce a treatment response in 70% to 90% of patients, corticosteroids and cyclophosphamide are associated with substantial morbidity and mortality, particularly as a result of serious infections. Cyclophosphamide is also associated with an increased risk of cancer, bladder conditions and infertility in patients. Azathioprine and mycophenolate mofetil are also general immunosuppressive therapies, associated with increased infection risk. Rituximab is a mouse-human chimeric anti-CD20 antibody, which depletes B cells and is associated with increased risk of serious infections, as well as progressive multifocal encephalopathy. Because of the serious adverse effects of these current therapies, we believe that there is an important unmet medical need for therapies that are safe, effective, and corticosteroid-sparing.

The ChemoCentryx Solution: CCX168 A Novel C5a Receptor Antagonist

CCX168 is a potent and highly specific antagonist of the human C5a receptor. The compound displays excellent oral bioavailability in various species and has demonstrated an excellent preclinical safety profile, consistent with its intended chronic use in patients. We have evaluated the pharmacological activity of CCX168 preclinically utilizing models that are relevant to the intended therapeutic use in humans. In the most recent of these models, treatment with an oral dose of CCX168 completely blocked the neutropenia, or removal of neutrophils from blood, produced by an injection of C5a.

The efficacy of CCX168 was demonstrated in a mouse model of ANCA-associated glomerulonephritis which recapitulates many of the histological features of the human disease. In these studies, oral doses of CCX168 completely blocked the glomerulonephritic vasculitis induced by intravenous injection of anti-myeloperoxidase antibodies. Levels of CCX168 in the blood of these mice were comparable to those expected in the blood of patients participating in our ongoing Phase II proof-of-concept clinical trial with CCX168.

CCX168 Drug Development Strategy and Clinical Trials

We have completed a Phase I clinical trial with CCX168 in 40 healthy subjects and we have initiated a Phase II clinical trial.

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CCX168 Phase I Clinical Trial

We completed a Phase I clinical trial in 40 healthy subjects with our C5aR-specific blocker, CCX168. This was a randomized, double-blind, placebo-controlled, two-period clinical trial in which subjects received either CCX168 or placebo, as a single dose in the first period and as multiple once-daily or twice-daily oral doses in the second period. Single oral doses of 1mg, 3mg, 10mg, 30mg, and 100mg of CCX168 were studied. In period two, CCX168 doses of 1mg, 3mg, and 10mg once-daily for seven days, and 30mg and 50mg twice-daily for seven days, were studied. CCX168 appeared to be well tolerated by clinical trial subjects in this clinical trial and no serious adverse events or withdrawals due to adverse events have been observed. The most commonly reported adverse events in subjects receiving CCX168 in the multi-dose period were headache, diarrhea, dizziness, lower abdominal pain, nausea, and oropharyngeal pain.

CCX168 Phase II Clinical Trial

We have initiated a Phase II clinical trial for CCX168. This is a randomized, double-blind, placebo-controlled clinical trial in 60 patients with AAV with mild-to-moderate renal involvement, the aim of which is to optimize the treatment to induce remission for patients with non-life-threatening AAV with mild-to-moderate renal involvement. The intent is to reduce the toxicity of induction therapy by reducing the overall exposure to or eliminating entirely the use of systemic corticosteroids during the induction period with an inhibitor of the complement C5a receptor plus cyclophosphamide. The primary objective of this clinical trial is to evaluate the safety and tolerability of CCX168 in patients with AAV on background cyclophosphamide treatment. The secondary objectives of this clinical trial include assessment of the feasibility of reducing or eliminating the use of corticosteroids in the treatment of patients with AAV without the need for rescue corticosteroid measures; evaluation of the PK profile of CCX168 in patients with AAV; and assessment of changes in renal function based on estimated glomerular filtration rate, hematuria, and proteinuria with CCX168 compared to placebo. The clinical trial will be conducted in three sequential steps with 12, 12, and 36 patients, respectively in each step. 30mg of CCX168 twice-daily given orally will be compared to placebo twice-daily for 84 days, followed by an 84-day follow-up period. We expect to complete this clinical trial by the end of 2012. If this clinical trial is successfully completed, GSK may exercise an option to further develop CCX168. If GSK does not exercise its option, we will evaluate our options for further development of CCX168, which may entail internally developing this drug candidate or identifying another collaboration partner for its development.

CCX662 CXCR7 Antagonist for Glioblastoma Multiforme

Understanding Glioblastoma Multiforme

According to Datamonitor, the incidence of primary brain cancer in 2007 across seven major markets (United States, Japan, France, Germany, Italy, Spain and the United Kingdom) was estimated to be 47,000. GBM is the most common and most aggressive of the primary brain tumors, accounting for 50%-60% of primary brain tumors in adults and it is slightly more common in men than in women. While GBM occurs in all age groups, its incidence is increasing in elderly patients. GBM is the most severe grade of brain tumors and is highly malignant, infiltrates the brain extensively and at times may become enormous before turning symptomatic. GBM, like other brain tumors, produces symptoms such as seizures, cognitive disorders and/or personality changes, among others. Symptoms depend on the location, size, and rate of growth of the tumor. Median life expectancy of GBM patients receiving radiation post-surgery is only 12 months.

Limitations of Current Therapies

Although the prognosis of GBM is uniformly poor, treating patients in an attempt to improve the quality of life is worthwhile. The current standard of care includes maximal safe surgical resection, followed by a combination of radiation and chemotherapy with temozolomide, or Temodar, the most widely used form of chemotherapy in GBM.

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Temodar induces DNA damage resulting in cell death. The current optimized dosing regimen includes six weeks of daily Temodar followed by monthly maintenance doses. This regimen has resulted in a median survival of 14.6 months, and 2-year overall survival of 27% and remains the current standard of care. Temodar was approved on the basis of only a 2.5 month improvement in overall survival. At present, no therapy has been identified which prevents relapse for the majority of patients and the tumor invariably recurs with often debilitating neurological and psychological symptoms, and salvage therapy has thus far met with very limited clinical success. At this stage, probably the most important part of the management of patients with GBM is compassionate and effective supportive care.

The overall prognosis for GBM has changed little in the past two decades, despite major improvements in neuroimaging, neurosurgery, radiation treatment techniques, adjuvant chemotherapy, and supportive care. There are considerable unmet needs in GBM, most notably the relatively low survival benefit offered by the existing standard of care and the lack of effective alternatives to Temodar.

The ChemoCentryx Solution: CCX662 A Novel CXCR7 Antagonist

We are developing drugs that target CXCR7 and combine an anti-angiogenic approach to stopping the blood supply to cancerous cells with anti-tumor activity via direct attack of tumor cells. Our most recent data support the notion that CXCR7 could be causally connected to tumor growth regulation. This is coupled with clear evidence from immunohistochemistry staining of primary human tumor tissues which revealed clear evidence of CXCR7 expression. In contrast to standard chemotherapies which can be highly toxic, our compounds are designed to selectively target CXCR7 in an effort to halt cancer progression while minimizing detrimental effects, such as generalized immunosuppression or cytotoxicity.

We believe we were the first to provide clear evidence that CXCR7 expression can directly control tumor growth *in vivo*. We and others have shown that CXCR7 regulates several important biological processes including cell survival, cell adhesion, trans-endothelial migration, tumor development and metastatic growth in secondary organs, in a variety of *in vivo* and *in vitro* models. CXCR7 is expressed on many human tumor cells but not on most healthy cells. In our tumor model systems we found that reduction or inhibition of CXCR7 by genetic and pharmacological means reduces or abolishes tumor formation *in vivo*, and that the introduction of CXCR7 into a naïve background is both necessary and sufficient for that tumor to grow aggressively *in vivo*. CXCR7 is highly expressed in human GBM and in GBM-associated blood vessels.

We and others have demonstrated, using various aggressive rodent GBM models, that CXCR7 inhibition is effective at preventing tumor growth, particularly when utilized in combination with radiation. Recent discoveries support a role for CXCR7 in preventing apoptosis, or programmed cell death, of human GBM cells. Separate observations indicate that recruitment of bone-marrow derived cells from blood into irradiated mouse GBM/brain is critical for tumor re-vascularization after irradiation and is dependent on CXCL12, the main chemokine ligand for CXCR7.

We have discovered several highly potent and selective small molecule compounds for CXCR7. We have selected one of these molecules, CCX662, as a clinical candidate and it is currently in preclinical development. Due to scientific and medical need reasons, our current efforts are primarily focused on the development of CCX662 for the treatment of GBM. CCX662 may qualify for orphan drug status, which may provide a faster path to regulatory approval.

We believe that CCX662 represents a promising novel therapeutic for the treatment of GBM and other types of cancer. Preclinical studies support not only the anti-tumor efficacy of this drug but also an excellent safety profile, a reflection of its highly targeted and specific activity profile, which is fundamentally different from many other cytotoxic drugs in development or on the market.

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Other Preclinical Programs

CCR4 Antagonist for Atopic Dermatitis

CCR4 is expressed primarily on T helper-2, or Th2, cells, which are key drivers of allergic conditions, such as atopic dermatitis, asthma, and allergic rhinitis. Multiple investigators have demonstrated increased levels of CCR4-activating chemokines in skin and lung tissues in connection with atopic dermatitis and asthma, respectively. During the past few years, a number of pharmaceutical companies have tried to develop small molecule CCR4 antagonists but have consistently failed to advance a molecule into clinical trials, primarily due to the inability to identify molecules with sufficient potency and PK properties and with an adequate safety profile, particularly in regards to cardiovascular safety.

Atopic dermatitis is an inflammatory, chronically recurring condition of the skin, which is often first diagnosed in children. Symptoms may vary from person to person but generally include red, inflamed, and itchy areas of rash, which can quickly develop into raised and painful bumps. Atopic dermatitis affects as many as 3% of adults in the United States. In its most severe form, it can be an absolutely debilitating disease.

CCX6239 is a novel, orally administered CCR4 antagonist we are developing for the treatment of atopic dermatitis. We have shown that CCX6239 effectively blocks the mobilization of white blood cells mediated by CCR4 in preclinical models. Potential additional areas of interest for this drug include asthma, allergic rhinitis and other allergic conditions. Based on preclinical data, we expect that CCX6239 will have excellent efficacy and safety, as it interferes selectively with the recruitment to skin of the key population of inflammatory cells that drive dermal allergic reactions, leaving most of the immune system unaltered. Given the young age of many atopic dermatitis patients, safety is a major consideration for drugs for the treatment of this disease and there are safety concerns with several of the currently available treatment options, which can increase the risk of opportunistic infection by broadly suppressing the immune system.

CCR9 Antagonist for IBD

Building on our extensive expertise in the area of CCR9 antagonists and IBD and following the expiration of our target exclusivity obligations with respect to CCR9 under our collaboration agreement with GSK, we started a *de novo* discovery program under which we have designed a series of novel molecules that we believe will represent the next generation of CCR9 antagonists. The lead compound from this series, CCX7034, demonstrates excellent selectivity for CCR9 relative to all other chemokine receptors, excellent oral bioavailability, and a very promising preclinical safety profile. However, molecules such as CCX7034 have been designed to interact with the CCR9 receptor in a unique way which produces molecules with much greater potency towards CCR9 than first-generation molecules. CCX7034 and several related molecules from this program are currently in preclinical development. The first clinical indication that might be pursued for this molecule is in the treatment of ulcerative colitis.

CXCR6 Antagonist for Chronic Hepatitis

Clearance of viral infections is associated with a vigorous T cell response. However, the immune system is often unable to clear hepatic infections. In fact, inflammation, chronic hepatitis and liver damage often result from persistent attempts by the immune system to deal with the underlying infection. Autoimmune hepatitis is a disease of unknown etiology also associated with pronounced T cell mediated liver inflammation and tissue damage. The chemokine receptor known as CXCR6 is expressed on a subset of specialized inflammatory cells, including certain types of T cells, natural killer cells, and natural killer T cells, or NKT cells, and is accepted, based on preclinical work with CXCR6-deficient mice, as a liver homing receptor for those cells. Under inflammatory conditions, various cell types in the liver produce the chemokine ligand that attracts CXCR6-expressing inflammatory cells, particularly NKT cells. We believe that these effects may be blocked by our CXCR6 antagonists without the adverse side effects associated with current method of treatment.

CCX5224 is a new, orally administered, internally developed CXCR6 antagonist that we are developing for the treatment of moderate-to-severe chronic hepatitis associated with viral and autoimmune hepatitis. CCX5224

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has demonstrated excellent preclinical potency, selectivity and safety, and displays high oral bioavailability. CCX5224 is currently in preclinical development.

Research Programs

We are working on several additional research programs, from which additional drug candidates may be identified in the future. The most advanced of these programs centers around a chemokine receptor known as CCR6.

CCR6 Program

One of the most intriguing areas of current research in immunology involves the study of a newly discovered type of helper T cells known as Th17 cells. These highly specialized cells are efficient producers of several highly inflammatory cytokines in the IL-17 family. While Th17 cells most likely play a role in the protection against extracellular pathogens, there is a large amount of preclinical and clinical data that implicate these cells, as well as IL-17, in the development of a large number of autoimmune diseases, including RA, IBD, and psoriasis. Activated Th17 cells isolated from chronically inflamed human tissues produce high levels of TNF-a and other cytokines.

A hallmark of Th17 cells is that they express high levels of CCR6, which is not found on Th1 and Th2 cells. Th17 cells and the newly discovered Th22 cells both express CCR6 and CCL20, which is the only known chemokine ligand for CCR6. High levels of CCL20 have been documented to be present in psoriatic skin, IBD intestinal tissue, RA joint biopsies and asthmatic lungs.

Our RAM screening technique has produced several suitable CCR6 antagonist leads, which are now being optimized via medicinal chemistry approaches. We believe that there are many potential therapeutic applications for a CCR6 antagonist. In general, these areas mirror those identified as being of interest for Th17-targeted therapeutics, including RA, psoriasis, and IBD, among others. We intend to identify a lead CCR6 antagonist for this program during 2012.

ChemR23 Program

In certain skin inflammatory diseases, the movement of plasmacytoid dendritic cells, or pDC, from blood into the skin sets in motion an inflammatory response that involves the recruitment and activation of various other types of inflammatory cells. This movement appears to be largely controlled by the binding of chemerin to its recently discovered chemo-attractant receptor known as ChemR23. This receptor displays a unique expression profile among leukocytes, restricted mainly to subsets of natural killer cells and immature pDC. Chemerin is the natural ligand for ChemR23 and is synthesized as a precursor protein which under inflammatory conditions undergoes activation. Elevated chemerin levels are seen in multiple skin inflammatory diseases, including cutaneous lupus, oral lichen planus and psoriasis.

Applying our EnabaLink suite of technologies we have produced several suitable ChemR23 antagonist leads. We believe that there are many potential therapeutic applications for a ChemR23 antagonist. CCX832, a ChemR23 antagonist, was one of our drug candidates subject to an option we had granted to GSK for further development and commercialization. However, in February 2012, based on unblinded data from a recently completed Phase I clinical trial of CCX832, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds. Both we and GSK continue to have interest in further discussing possible strategic opportunities with respect to ChemR23.

Our Proprietary Drug Discovery Platform, EnabaLink

Since the founding of our company, we have developed a set of proprietary drug discovery tools, known collectively as the EnabaLink technology suite, specifically designed to unlock the chemokine system s

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complexity and to accelerate a productive drug discovery program. Our proprietary EnabaLink drug discovery technologies allow our scientists to accurately predict the specific chemokine receptors implicated in a given condition and to identify and optimize small molecule compounds best suited for treatment of the disease. One of the initial tools that we developed is a thorough functional genomic map of the chemokine system which assists us in our understanding of the role of a given chemokine receptor in the system as well as its likely effect on the migration of inflammatory cells in a given inflammatory disease state.

As part of this platform, we have also developed a proprietary high throughput cell migration-based assay, known as the RAM Assay, capable of identifying chemokine receptor inhibitors while eliminating non-specific inhibitors and cytotoxic inhibitors of cell migration. Our proprietary RAM Assay typically uses cells expressing a given chemokine receptor in its natural environment and enables the screening of small molecule libraries against chemokine receptor targets which are not amenable to traditional screening technologies. This produces additional novel chemical hits with structural diversity, allowing us to expand the number of chemical structures, which serve as starting points for subsequent optimization into drug candidates.

We have used our EnabaLink drug discovery engine to create a broad pipeline of promising chemokine-based drug candidates. The combination of proprietary in-house technologies and internally discovered drug candidates has resulted in an extensive intellectual property estate covering composition of matter and associated method of treatments for our compounds, novel biology-related discoveries, such as unique targets and new drug discovery technologies. We have generated more than six clinical or preclinical-stage programs, each targeting distinct chemokine receptors with different small molecule compounds. Drug candidates emerging from these programs act with high affinity and selectivity *in vitro* by binding to the precise chemokine receptor associated with the essential inflammatory processes underlying a given condition. Our compounds are designed to be highly potent and selective to minimize the risk of off-target effects and orally-available for improved patient compliance. As small molecules, they are also easier and less costly to manufacture than biologics.

Strategic Alliance with GSK

In August 2006, we entered into our strategic alliance with GSK. We have received $245.7 million from GSK, consisting of up-front and milestone payments, equity investments, research funding and option exercise fees. Under the terms of our agreement with GSK, we are responsible for the discovery and development of small molecule antagonists targeting four defined chemokine and chemo-attractant receptor targets (CCR9, CCR1, C5aR and ChemR23) and for advancing them through clinical proof-of-concept. After we demonstrate successful clinical proof-of-concept, GSK is entitled to options to exclusively license drug candidates that are subject to the collaboration and two defined back-up compounds for each drug candidate for further development and commercialization on a worldwide basis. Upon exercising any of its options to drug candidates under the collaboration, GSK is solely responsible for all further clinical development and commercialization expenditures worldwide with respect to that drug candidate and its two designated back-up compounds. In exchange for the rights granted to GSK upon the exercise of its options, we are also entitled to receive regulatory and commercial milestone payments, as earned under the terms of our agreement, and royalties on the net sales of licensed drugs. The agreement contemplated up to six drug options, each of which covers a drug candidate, including Traficet-EN (CCR9), CCX354 (CCR1), CCX168 (C5aR) and CCX832 (ChemR23), and their associated back-up compounds. The other two drug options were for second generation drug candidates and their associated back-up compounds. However, we and GSK chose not to nominate second generation drug candidates against any of the four defined targets during the agreement s research term, which has expired. In addition, in February 2012, based on unblinded data from a recently completed Phase I clinical trial of CCX832, we and GSK determined not to further advance the development of CCX832 or its two designated back-up compounds, although both we and GSK continue to have interest in further discussing possible strategic opportunities with respect to ChemR23. GSK has already exercised its options to Traficet-EN and CCX354 and each of their two respective defined back-up compounds. Thus, GSK s only remaining option is to CCX168 and its associated back-up compounds.

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GSK does not have exclusive rights to a given clinical indication or substitution rights with respect to a given collaboration target. Our proprietary programs around CCR2, CXCR7 or any other receptors are not part of the GSK collaboration.

The Joint Steering Committee, or JSC, established under the collaboration agreement, comprises three representatives from GSK and three of our representatives and has review and oversight responsibilities for all research and development activities performed by us. After an option exercise, the JSC serves as a vehicle to facilitate the information exchange between GSK and us with respect to further clinical development and commercialization activities performed by GSK for such licensed drug candidates. We have final decision making authority with respect to all decisions relating to the research program, the conduct of Phase I clinical trials and Phase II proof-of-concept clinical trials and the manufacture of drug candidates prior to option exercise. We and GSK mutually determine the indication to be studied for each collaboration drug candidate. GSK has the final decision making authority with respect to the criteria for us to establish proof-of-concept and the Phase II proof-of-concept trial design for CCX168, however, the collaboration provides for a limit on the scope and the costs of a given Phase II proof-of-concept trial. GSK also has final decision making authority with respect to all decisions relating to exercising its option, the funding and development of drug candidates after an option exercise and commercialization of the same.

In December 2009, GSK exercised its option to obtain an exclusive license to further develop and commercialize Traficet-EN following our completion of the PROTECT-1 clinical trial. We received an option exercise fee of $35.0 million in January 2010 after GSK obtained Hart-Scott-Rodino clearance. After exercising the option, GSK became solely responsible for all further clinical development and commercialization expenditures for Traficet-EN and its two designated back-up compounds worldwide.

In November 2011, GSK exercised its option to obtain an exclusive license to further develop and commercialize CCX354 following our completion of the proof-of-concept clinical trial for this drug candidate. After exercising this option, GSK became solely responsible for all further clinical development and commercialization expenditures for CCX354 and its two designated back-up compounds worldwide. We received an option exercise fee of $25.0 million in December 2011 with respect to this option exercise.

With respect to CCX168, the remaining drug candidate subject to the agreement, GSK has an option exercisable with respect to such drug candidate upon our demonstration of successful clinical proof-of-concept and, if GSK elects to exercise its option, we will be entitled to an option exercise fee of $25.0 million upon the exercise of such option by GSK. For each of our drug candidates subject to the agreement we would be entitled to receive regulatory filing milestones of up to $47.0 million in the aggregate for the filing of an NDA in the United States and comparable filings in other territories, up to $75.0 million in the aggregate for the regulatory approval of products for commercial sale in the United States and other territories and up to $250.0 million in sales milestones for Traficet-EN and $125.0 million for each of CCX354 and CCX168.

Under the collaboration, even if proof-of-concept clinical trials for any of our drug candidates that are subject to this collaboration result in positive outcomes, GSK may decide not to exercise its option. In the event that GSK elects not to exercise its option upon successful completion of a given proof-of-concept clinical trial, all rights to that program revert back to us, subject to a royalty obligation to GSK in the amount of 3% of annual worldwide net sales of the relevant drug candidate (capped at $50.0 million) if we go on to develop and commercialize it. Upon exercising its remaining option under the collaboration, GSK would be required to commence and pursue a development and commercialization program for such drug candidate from such option exercise promptly after option exercise and would be solely responsible for all further clinical development and commercialization expenditures worldwide with respect to that program. The collaboration agreement requires GSK to use commercially reasonable efforts to further develop and commercialize all licensed drug candidates, including conducting all further clinical trials, developing additional formulations if necessary, preparing and filing all regulatory filings, manufacturing the drug product and marketing the licensed drug worldwide. However, GSK has the right to discontinue the development and commercialization of any licensed drug candidate based on factors or considerations consistent with its diligence efforts. In the event that GSK exercises an option with respect to a drug candidate and later determines in good faith to cease the development and commercialization of the licensed drug,

either in its entirety, or on a country-by-country basis, we can elect to further develop and commercialize such licensed drug under a non-exclusive license grant from GSK. If we so elect, we will be solely responsible for satisfying all obligations to third parties with respect to the development, manufacture or commercialization of such licensed drug including any ongoing obligations of GSK under any third party manufacturing, licensing or other agreements, and we will be obligated to pay GSK 3% to 5% of annual worldwide net sales of such licensed drug depending on the licensed drug s stage of development at the time at which we make such election.

We retain the option to co-develop Traficet-EN for certain gastrointestinal indications. Not later than 12 months before the first expected NDA for Traficet-EN, GSK is required to provide us with a detailed development plan and budget. After the final development plan and budget has been submitted we will have 90 days to decide whether to co-develop Traficet-EN. We can decide, in our sole discretion, whether to exercise the option to co-develop Traficet-EN for Crohn s disease alone or for Crohn s disease and ulcerative colitis, should GSK pursue the development of Traficet-EN in such indications.

If we elect to co-develop Traficet-EN we would be required to pay 35% of GSK s development costs related to the selected gastrointestinal indications. Such co-development rights apply only to drug candidates arising out of the CCR9 program and only for such gastrointestinal indications as Crohn s disease or ulcerative colitis. In return for having co-developed Traficet-EN, we would receive an increase over base line royalties, based on worldwide net sales of Traficet-EN. We also have the option, which we can exercise at our sole discretion, to co-promote Traficet-EN to physician specialists in the United States, subject to our payment of 35% of GSK s development costs. Under the terms of the agreement, our promotional efforts could be as high as 50% of all promotional efforts to physician specialists in the United States. As consideration for our promotional efforts, GSK would be required to pay us an amount similar to what it would pay a third-party contract sales force.

Under the terms of the agreement, we received $63.5 million in the form of cash and equity from GSK in 2006. GSK was also obligated to provide research funding of up to $5.0 million per year during the first three years of our collaboration. The research term expired in August 2009, but was extended by six months for the ChemR23 program and we received an additional $2.5 million in research funding from GSK for the six months extension. Upon nomination of one of the defined back-up compounds in a given non-CCR9 collaboration program, we are entitled to receive a non-refundable, non-creditable milestone payment of $5.0 million for each drug candidate, for a maximum of three such payments for a given collaboration target. We received such pre-clinical milestone payments for CCX354, CCX168 and CCX832, respectively. Upon initiation of a first-in-humans study, we would be entitled to receive a non-refundable, non-creditable milestone payment of $10.0 million per drug candidate. We also received such first-in-humans milestone payments for CCX354, CCX168 and CCX832, respectively.

In addition, we are entitled to receive base royalties on net sales of the licensed drugs. The base royalties for each program differ, but are set at levels commensurate with the development stage of each program at the time we entered into the agreement. With respect to Traficet-EN and its two designated back-up compounds, GSK is obligated to pay us different base royalties on net sales in the United States and outside the United States. Tiered base percentage royalties on net sales in IBD indications in the United States range from the mid teens to the low twenties while tiered percentage royalties on net sales in IBD indications outside the United States range from the low to high teens. If we decide to exercise our co-development option for Traficet-EN, base royalty rates will increase by up to 5%, depending on the sales tier, the region and the indication for which the co-development option is exercised. With respect to CCX354 and CCX168, or any of their designated back-up compounds, GSK is obligated to pay us double-digit tiered percentage royalties with the potential to reach the mid teens on annual worldwide net sales. We are also entitled to receive sales milestones on a per drug basis.

This agreement will expire with respect to each licensed drug and country upon the expiration of the payment obligations of GSK for that licensed drug in that country and would expire in its entirety upon the expiration of the last payment obligation of GSK for the last licensed drug in the last country. Following GSK s exercise of its options, GSK is obligated to pay us royalties on net sales of products that include a compound from such collaboration program in a given country for so long as a valid claim of our independently owned patents or a jointly owned patent covers or claims the composition of matter or a relevant method of use of a

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licensed drug candidate subject to the collaboration agreement. In the absence of such a valid claim on a country-by-country basis, GSK is obligated to pay a reduced royalty rate for a period of 12 years from the date of the first commercial sale of a licensed drug candidate as long as we own or control a pending patent application which covers the composition of matter or a relevant method of use of a licensed drug candidate. In the event that a particular licensed drug candidate is sold in a country in which a generic pharmaceutical product approved in reliance on the prior approval of such licensed drug candidate is also sold, then GSK is no longer obligated to pay us royalties on such licensed products after the end of the first 180-day period in which one or more third parties sell a number of units of the generic product worldwide equal to 25% or more of the aggregate number of units of the licensed drug candidate sold in such country during that same 180-day period. Consequently, it is not possible at this time to determine when GSK s payment obligations under the collaboration agreement will expire.

GSK may terminate the collaboration agreement for any reason upon 90 days prior written notice to us. The agreement and each program under the agreement may also be terminated under certain circumstances, including by either party for material breach or insolvency of the other party. The rights and obligations of the parties that survive termination of the agreement vary depending on the basis of the termination.

We are obligated to use diligent efforts to carry out the early development programs covered by the agreement. If we fail to do so and fail to cure such breach within the specified cure period, we will be required to pay a penalty to GSK based on certain percentages of up-front payments and research funding relating to the relevant collaboration target that we received from GSK. Alternatively, if we fail to cure such breach, at GSK s election, we would be required to grant GSK a worldwide exclusive license to the compounds and their associated back-up compounds which are the subject of the relevant early development program, whether or not proof-of-concept has been achieved at such time and, other than certain royalty obligations, GSK would not be obligated to pay milestone payments, costs and other fees to us in connection with the exercise of such options.

Under the terms of the agreement, with respect to each collaboration target, we are obligated to not, either alone or with a third party, conduct any research or development activities or grant any license or other rights with respect to the identification or optimization of small molecule antagonists or agonists, as applicable, for such collaboration target unless and until either (i) GSK exercises its option to a drug candidate and its two back-up compounds with respect to such collaboration target or (ii) GSK terminates the collaboration program with respect to such collaboration target. Once the target exclusivity with respect to a collaboration target expires, we are free to initiate our own de novo proprietary drug discovery effort with respect to such target free of the exclusivity restrictions of our strategic alliance with GSK. There are no contractual restrictions that would preclude GSK from developing or investing in drug candidates that would compete with drug candidates that are subject to our collaboration with GSK.

Under the terms of the agreement, GSK has the right, but not the obligation, to defend against third party patent infringement claims for licensed drugs. If GSK elects to defend against any such claims, it has the sole right to direct the defense of such claims and settle such claims at its own cost and expense. If GSK elects not to defend against such claims, we have the right, but not the obligation, to defend against such claims. If the development or commercialization of licensed drugs requires use of third party intellectual property, we and GSK will share all license fees, provided however, that in the event that Millennium has valid patents relating to CCR9 and we are required to take a license from Millennium, we will be solely responsible for all fees required to be paid to Millennium in connection with such license. See Intellectual Property.

Intellectual Property

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

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As for the pharmaceutical products we develop and commercialize, as a normal course of business, we intend to pursue composition-of-matter patents, where possible, and dosage and formulation patents, as well as method of use patents on novel indications for known compounds. We also seek patent protection with respect to novel biological discoveries, including new targets and applications as well as adjuvant and vaccine candidates. We have also pursued patents with respect to our proprietary screening and drug development processes and technology. We have sought patent protection, either alone or jointly with our collaborators, as our collaboration agreements may dictate.

Our patent estate, on a worldwide basis, includes approximately 390 issued or allowed patents and approximately 325 pending patent applications, with claims relating to all of our current clinical stage drug candidates. With respect to our lead drug candidates in the CCR1, CCR2 and CCR9 programs, we have approximately 100 issued or allowed patents worldwide relating to their chemical composition or use thereof. There are approximately 40 patent applications pending for our other clinical stage compounds in the C5aR and ChemR23 programs. We have approximately 180 issued patents relating to other small molecule compounds and approximately 65 issued patents relating to our novel biological discoveries. We also have approximately 50 issued patents relating to our proprietary screening and drug development technologies.

Individual patents extend for varying periods depending on the date of filing of the patent application or the date of patent issuance and the legal term of patents in the countries in which they are obtained. Generally, patents issued for applications filed in the United States are effective for twenty years from the earliest effective filing date. In addition, in certain instances, a patent term can be extended to recapture a portion of the term effectively lost as a result of the FDA regulatory review period. However, the restoration period cannot be longer than five years and the total patent term including the restoration period must not exceed 14 years following FDA approval. The duration of foreign patents varies in accordance with provisions of applicable local law, but typically is also twenty years from the earliest effective filing date. Our issued patents will expire on dates ranging from 2020 to 2029. However, the actual protection afforded by a patent varies on a product by product basis, from country to country and depends upon many factors, including the type of patent, the scope of its coverage, the availability of regulatory related extensions, the availability of legal remedies in a particular country and the validity and enforceability of the pate.

Furthermore, the patent positions of biotechnology and pharmaceutical products and processes like those we intend to develop and commercialize are generally uncertain and involve complex legal and factual questions. No consistent policy regarding the breadth of claims allowed in such patents has emerged to date in the United States. The patent situation outside the United States is even more uncertain. Changes in either the patent laws or in interpretations of patent laws in the United States and other countries can diminish our ability to protect our inventions and enforce our intellectual property rights and more generally could affect the value of intellectual property. Accordingly, we cannot predict the breadth of claims that may be allowed or enforced in our patents or in third party patents. The biotechnology and pharmaceutical industries are characterized by extensive litigation regarding patents and other intellectual property rights. Our ability to maintain and solidify our proprietary position for our drugs and technology will depend on our success in obtaining effective claims and enforcing those claims once granted. We do not know whether any of the patent applications that we may file or license from third parties will result in the issuance of any patents. The issued patents that we own or may receive in the future, may be challenged, invalidated or circumvented, and the rights granted under any issued patents may not provide us with proprietary protection or competitive advantages against competitors with similar technology. Furthermore, our competitors may be able to independently develop and commercialize similar drugs or duplicate our technology, business model or strategy without infringing our patents. Because of the extensive time required for clinical development and regulatory review of a drug we may develop, it is possible that, before any of our drugs can be commercialized, any related patent may expire or remain in force for only a short period following commercialization, thereby reducing any advantage of any such patent.

In addition to patents, we rely upon unpatented trade secrets and know-how and continuing technological innovation to develop and maintain our competitive position. We seek to protect our proprietary information, in part, using confidentiality agreements with our commercial partners, collaborators, employees and consultants and invention assignment agreements with our employees. We also have confidentiality agreements or invention

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assignment agreements with our commercial partners and selected consultants. These agreements are designed to protect our proprietary information and, in the case of the invention assignment agreements, to grant us ownership of technologies that are developed through a relationship with a third party. These agreements may be breached, and we may not have adequate remedies for any breach. In addition, our trade secrets may otherwise become known or be independently discovered by competitors. To the extent that our commercial partners, collaborators, employees and consultants use intellectual property owned by others in their work for us, disputes may arise as to the rights in related or resulting know-how and inventions.

Our commercial success will also depend in part on not infringing upon the proprietary rights of third parties. It is uncertain whether the issuance of any third party patent would require us to alter our development or commercial strategies, or our drugs or processes, obtain licenses or cease certain activities. Our breach of any license agreements or failure to obtain a license to proprietary rights that we may require to develop or commercialize our future drugs may have a material adverse impact on us. If third parties prepare and file patent applications in the United States that also claim technology to which we have rights, we may have to participate in interference proceedings in the U.S. Patent and Trademark Office, or USPTO, to determine priority of invention.

Millennium Pharmaceuticals, Inc., or Millennium, has obtained certain United States patents which include claims to small molecules that modulate CCR9, compositions thereof, and methods of using them to treat conditions such as IBD. Millennium may contend that the claims of these patents cover our patented Traficet-EN drug candidate. We believe that our activities related to Traficet-EN are currently exempt from patent infringement liability because these activities are strictly limited to obtaining information for regulatory approval. However, if and when our Traficet-EN related activities extend beyond those related to seeking regulatory approval, such as, for example, if and when we commercialize Traficet-EN, Millennium might then commence an infringement action against us based on these patents and/or other related patents that it may be granted in the future. If Millennium elects to sue us, we believe that we may have viable defenses to any such infringement suit. However, we cannot assure you that the relevant court would find in our favor with respect to such defenses. Intellectual property litigation and patent litigation in particular, is expensive, complex and lengthy and its outcome is difficult to predict. A court could enter orders that temporarily, preliminarily or permanently enjoin us or our partners from using, selling, offering to sell or importing our current or future drug candidates or could enter an order mandating that we undertake certain remedial activities. Pending or future patent litigation against us or any strategic partners by Millennium or anyone else may force us or any strategic partners to stop or delay developing, manufacturing or selling potential drug candidates that are claimed to infringe a third party s intellectual property, unless that party grants us or any strategic partners rights to use its intellectual property. If Millennium is able to obtain an injunction and neither we nor our partners are able to obtain a license, we and our partners will be precluded from the manufacture and sale of Traficet-EN. If we or our partners are unable to show that Millennium s patent is invalid and neither we nor our partners are able to obtain a license from Millennium for the use of their intellectual property, at all or on commercially acceptable terms, this would preclude us and our partners from the manufacture and sale of Traficet-EN or related candidate compounds found to be covered by Millennium s patent claims.

In addition, substantial scientific and commercial research has been conducted for many years in the areas in which we have focused our development efforts, which has resulted in third parties having a number of issued patents and pending patent applications. Patent applications in the United States and elsewhere are published only after eighteen months from the priority date. The publication of discoveries in the scientific or patent literature frequently occurs substantially later than the date on which the underlying discoveries were made. Therefore, patent applications relating to drugs similar to Traficet-EN and any future drugs, discoveries or technologies we might develop may have already been filed by others without our knowledge.

Competition

We compete in the segments of the pharmaceutical, biotechnology and other related markets that address IBD, chronic kidney disease and diabetic nephropathy, rheumatoid arthritis, other autoimmune diseases and inflammatory

disorders, and cancer. We face significant competition from many pharmaceutical and biotechnology companies that are also researching and selling products designed to address these markets. Many of our competitors have materially greater financial, manufacturing, marketing, research and drug development resources than we do. Large pharmaceutical companies in particular have extensive expertise in preclinical and clinical testing and in obtaining regulatory approvals for drugs. In addition, academic institutions, government agencies, and other public and private organizations conducting research may seek patent protection with respect to potentially competitive products or technologies. These organizations may also establish exclusive collaborative or licensing relationships with our competitors.

It is possible that our competitors will develop and market drugs that are less expensive and more effective than our drug candidates, or that will render our drug candidates obsolete. It is also possible that our competitors will commercialize competing drugs before we or our partners can launch any drugs developed from our drug candidates. If approved for marketing by the FDA, Traficet-EN, our lead drug candidate for the treatment of IBD, would compete against existing IBD treatments such as Remicade, Humira, and other TNF-a inhibitors, immunomodulatory drugs and corticosteroids and potentially against other novel IBD drug candidates that are currently in development. Remicade is a humanized monoclonal antibody targeted to TNF-a, indicated for the treatment of Crohn s disease, ulcerative colitis, rheumatoid arthritis, psoriasis, psoriatic arthritis and ankylosing spondylitis. Annual sales for Remicade totaled at least $6.0 billion in 2010. Humira, a similar drug, is also a human monoclonal antibody that acts as a TNF-a inhibitor. Marketed by Abbott Laboratories in the United States and Europe, Humira is approved for the treatment of Crohn s disease, rheumatoid arthritis, psoriatic arthritis and ankylosing spondylitis. Annual worldwide sales for Humira totaled $6.5 billion in 2010.

We believe that Traficet-EN offers three distinct advantages as compared to currently used biologic therapies such as Remicade and Humira. First, unlike Remicade and Humira which are given by infusion or injection, Traficet-EN would be administered orally as a capsule or a tablet. We expect that oral administration of Traficet-EN will have a positive effect on patient compliance. Second, given that Traficet-EN is a small molecule which can be synthesized using standard chemistry processes, the molecule will be cheaper to manufacture than biologic agents which require complex and expensive cell based systems to produce a given biologic agent. The lower cost of goods for Traficet-EN could result in a more favorable pricing structure which, in turn, could lead to pharmaco-economic benefits for patients and healthcare providers. Third, Traficet-EN s mode of action may not lead to the broad suppression of the patient s immune system which is often seen with TNF-a inhibitors.

Our lead independent drug candidate, CCX140, a CCR2 antagonist, if approved for marketing by the FDA for diabetic nephropathy, would compete with treatments commonly used for type 2 diabetes and hypertension patients. ARBs and ACE inhibitors, are commonly prescribed treatments used to reduce blood pressure and increase kidney function, reducing the progression of diabetic nephropathy. Many patients eventually progress to end-stage renal disease and require hemodialysis, peritoneal dialysis, or renal transplant. There are other candidates in Phase III development for diabetic nephropathy including Abbott / Reata s bardoxolone.

CCX354 is our lead CCR1 antagonist candidate in RA. Treatment of RA can be divided into disease-modifying antirheumatic drugs, or DMARDs, anti-inflammatory agents and analgesics. Current treatment options for RA consist of corticosteroids such as prednisone, immunosuppressants such as methotrexate (Rheumatrex and Trexall), azathioprine (such as Imuran), sulfasalazine (Azulfidine), hydroxychloroquine (Plaquenil) and 6-mercaptopurine (Purinethol), leflunomide (Arava), and biologic agents including TNF-a inhibitors (etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), certolizumab (Cimzia), and golimumab (Simponi)), anakinra (Kineret), rituximab (Rituxan), abatacept (Orencia), and tocilizumab (Actemra). There are also several novel, oral kinase inhibitors in Phase III development for RA including AstraZeneca / Rigel s fostamatinib and Pfizer s tasocitinib.

Many of these currently approved treatments have notable and common adverse events including liver and bone marrow toxicity, renal toxicity, pneumonitis, immunosuppression, allergic skin reactions, autoimmune diseases and infections.

We expect that competition among any of our drugs approved for sale will be based on various factors, including drug safety and efficacy, prevalence of negative side effects, reliability, ease of administration, availability, price, insurance coverage and reimbursement status and patent position. We believe that our ability

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to compete depends largely upon our ability to research, develop and commercialize our existing and future drug candidates. Further, we need to continue to attract and retain qualified personnel, obtain patent protection, develop proprietary technology or processes and secure sufficient capital resources for the substantial time period between technological conception and commercial sales of drugs. Our ability to compete will also be affected by the speed at which we are able to identify and develop, conduct clinical testing and obtain regulatory approvals of our drug candidates. Potential competitors may develop treatments that are more effective and/or safer than our drug candidates or that would make our technology and drug candidates obsolete or non-competitive.

Established pharmaceutical companies that currently sell or are developing drugs in our markets of interest include Abbott, Amgen, AstraZeneca, Biogen Idec, Bayer, Elan, GSK, Johnson & Johnson, Merck, Merck Serono, Takeda, Novartis, Pfizer, Reata, Sanofi-aventis and Teva. Many or all of these established competitors are also heavily involved in research and drug development regarding various chemokine receptors. Pharmaceutical and biotechnology companies which are known to be involved in chemokine research and related drug development include Pfizer, GSK, Bristol-Myers Squibb, Merck, Takeda, Sanofi-aventis, Incyte, and UCB Pharma among others. These companies and others also compete with us in recruiting and retaining qualified scientific and management personnel, and in acquiring technologies complementary to, or necessary for, our programs.

Manufacturing

Our current drug candidates are manufactured using common chemical engineering and synthetic processes from readily available raw materials. Following GSK s exercise of its options for the further development of Traficet-EN and CCX354, it assumed sole manufacturing responsibility for these drug candidates and each of their two respective designated back-up compounds. We rely on contract manufacturing organizations to produce our other drug candidates in accordance with the FDA s current good manufacturing practices, or cGMP, regulations for use in our clinical trials. However, we currently rely on a single source supplier for our requirements of the API of each of these other drug candidates. The manufacture of pharmaceuticals is subject to extensive cGMP regulations, which impose various procedural and documentation requirements and govern all areas of record keeping, production processes and controls, personnel and quality control. We expect to rely on contract manufacturers for the manufacture of clinical and commercial supplies of our compounds other than those drug candidates for which GSK has exercised its option.

We purchase quantities of our drug candidates from our contract manufacturers pursuant to purchase orders that we place from time to time. If we were unable to obtain sufficient quantities of drug candidates or receive raw materials in a timely manner, we could be required to delay our ongoing clinical trials and seek alternative manufacturers, which would be costly and time-consuming. We believe we have multiple potential sources for our contract manufacturing.

Government Regulation

The FDA and comparable regulatory authorities in state and local jurisdictions and in other countries impose substantial and burdensome requirements upon companies involved in the clinical development, manufacture, marketing and distribution of drugs. These agencies and other federal, state and local entities regulate research and development activities and the testing, manufacture, quality control, safety, effectiveness, labeling, storage, record keeping, approval, advertising and promotion, export and import of our drug candidates.

In the United States, the FDA regulates drug products under the Federal Food, Drug, and Cosmetic Act, or FFDCA, and the FDA s implementing regulations. If we fail to comply with applicable FDA or other requirements at any time during the drug development process, clinical testing, the approval process or after approval, we may become subject to administrative or judicial sanctions. These sanctions could include the FDA s refusal to approve pending applications, license suspension or revocation, withdrawal of an approval, warning letters, product recalls, product seizures, total or partial suspension of production or distribution, injunctions, fines, civil penalties or criminal prosecution. Any FDA enforcement action could have a material

adverse effect on us. The process required by the FDA before our drug candidates may be marketed in the United States generally involves the following:

- completion of extensive preclinical laboratory tests, preclinical animal studies and formulation studies all performed in accordance with the FDA s current good laboratory practice, or cGLP, regulations;
- submission to the FDA of an investigational new drug, or IND, application which must become effective before human clinical trials in the United States may begin;
- performance of adequate and well-controlled human clinical trials to establish the safety and efficacy of the drug candidate for each proposed indication;
- submission to the FDA of a new drug application, or NDA;
- satisfactory completion of an FDA inspection of the manufacturing facility or facilities at which the drug is produced to assess compliance with current Good Manufacturing Practice, or cGMP, regulations; and
- FDA review and approval of the NDA prior to any commercial marketing, sale or shipment of the drug.

The testing and approval process requires substantial time, effort and financial resources, and we cannot be certain that any approvals for our drug candidates will be granted on a timely basis, if at all.

Once a pharmaceutical drug candidate is identified for development, it enters the preclinical testing stage. Preclinical studies include laboratory evaluations of drug chemistry, formulation and stability, as well as studies to evaluate toxicity in animals. The results of the preclinical studies, together with manufacturing information and analytical data, are submitted to the FDA as part of an IND application. The IND automatically becomes effective 30 days after receipt by the FDA, unless the FDA, within the 30-day time period, raises concerns or questions about the conduct of the clinical trial, including concerns that human research subjects will be exposed to unreasonable health risks. In such a case, the IND sponsor and the FDA must resolve any outstanding concerns before the clinical trial can begin. Submission of an IND may result in the FDA not allowing the clinical trials to commence or not allowing the clinical trials to commence on the terms originally specified in the IND. A separate submission to an existing IND must also be made for each successive clinical trial conducted during drug development, and the FDA must grant permission, either explicitly or implicitly by not objecting, before each clinical trial can begin.

Clinical trials involve the administration of the investigational drug to human subjects under the supervision of qualified investigators. Clinical trials are conducted under protocols detailing, among other things, the objectives of the clinical trial, the parameters to be used in monitoring safety and the effectiveness criteria to be used. Each protocol must be submitted to the FDA as part of the IND. An independent institutional review board, or IRB, for each medical center proposing to conduct a clinical trial must also review and approve a plan for any clinical trial before it can begin at that center and the IRB must monitor the clinical trial until it is completed. The FDA, the IRB, or the sponsor may suspend or discontinue a clinical trial at any time on various grounds, including a finding that the subjects are being exposed to an unacceptable health risk. Clinical testing also must satisfy extensive Good Clinical Practice, or GCP, requirements, including the requirements for informed consent.

All clinical research performed in the United States in support of an NDA must be authorized in advance by the FDA under the IND regulations and procedures described above. However, a sponsor who wishes to conduct a clinical trial outside the United States may, but need not, obtain FDA authorization to conduct the clinical trial under an IND. If a foreign clinical trial is not conducted under an IND, the sponsor may submit data from the clinical trial to FDA in support of an NDA so long as the clinical trial is conducted in compliance with an international guideline for the ethical conduct of clinical research known as the Declaration of Helsinki and/or the laws and regulations of the country or countries in which the clinical trial is performed, whichever provides the greater protection to the participants in the clinical trial. We conducted our PROTECT-1 clinical trial solely at foreign clinical research sites, and we did not have authorization from the FDA under an IND to conduct that clinical trial in the United States. We designed the clinical trial to comply with FDA regulatory requirements for the use of foreign clinical data in support of an NDA, and we intend to submit data from the PROTECT-1 clinical

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trial in support of our future U.S. marketing application for Traficet-EN. We are pursuing a similar development strategy for CCX140 for which we are currently conducting two Phase II clinical trials in patients with diabetic nephropathy in certain European countries. These clinical trials are also designed to comply with FDA regulatory requirements so that the data from these trials can be used to support a regulatory filing in the United States. We plan to include the United States and Europe in our later-stage clinical development program for CCX140 and for other drug candidates we develop independently prior to filing for an NDA with the FDA and the EMA.

Clinical Trials

For purposes of NDA submission and approval, clinical trials are typically conducted in three sequential phases, which may overlap or be combined.

Phase I clinical trials are initially conducted in a limited population of subjects to test the drug candidate for safety, dose tolerance, absorption, metabolism, distribution and excretion in healthy humans or, on occasion, in patients with severe problems or life-threatening diseases to gain an early indication of its effectiveness.

Phase II clinical trials are generally conducted in a limited patient population to:

evaluate dosage tolerance and appropriate dosage;

identify possible adverse effects and safety risks; and

evaluate preliminarily the efficacy of the drug for specific targeted indications in patients with the disease or condition under study.

Phase III clinical trials, commonly referred to as pivotal studies, are typically conducted when Phase II clinical trials demonstrate that a dose range of the drug candidate is effective and has an acceptable safety profile. Phase III clinical trials are generally undertaken with large numbers of patients, such as groups of several hundred to several thousand, to further evaluate dosage, to provide substantial evidence of clinical efficacy and to further test for safety in an expanded and diverse patient population at multiple, geographically-dispersed clinical trial sites.

In some cases, the FDA may condition approval of an NDA on the sponsor s agreement to conduct additional clinical trials to further assess the drug s safety and effectiveness after NDA approval. Such post-approval clinical trials are typically referred to as Phase IV clinical trials.

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

New Drug Applications

The results of preclinical studies and of the clinical trials, together with other detailed information, including extensive manufacturing information and information on the composition of the drug, are submitted to the FDA in the form of an NDA requesting approval to market the drug for one or more specified indications. The FDA reviews an NDA to determine, among other things, whether a drug is safe and effective for its intended use.

Once an NDA has been accepted for filing, by law the FDA has 180 days to review the application and respond to the applicant. However, the review process is often significantly extended by FDA requests for additional information or clarification. Under the Prescription Drug User Fee Act, the FDA has a goal of responding to NDAs within ten months of submission for standard review, but this timeframe is also often extended. The FDA may refer the application to an advisory committee for review, evaluation and

recommendation as to whether the application should be approved. The FDA is not bound by the recommendation of an advisory committee, but it generally follows such recommendations. The FDA may deny approval of an NDA if the applicable statutory and regulatory criteria are not satisfied, or it may require additional clinical data or an additional Phase III clinical trial. Even if such data are submitted, the FDA may ultimately decide that the NDA does not satisfy the criteria for approval. Data from clinical trials are not always conclusive and the FDA may interpret data differently than we interpret data. Once the FDA approves an NDA, or supplement thereto, the FDA may withdraw the approval if ongoing regulatory requirements are not met or if safety problems are identified after the drug reaches the market. Where a withdrawal may not be appropriate, the FDA still may seize existing inventory of such drug or require a recall of any drug already on the market. In addition, the FDA may require testing, including Phase IV clinical trials and surveillance programs to monitor the effect of approved drugs which have been commercialized. The FDA has the authority to prevent or limit further marketing of a drug based on the results of these post-marketing programs.

A sponsor may also seek approval of its drug candidates under programs designed to accelerate FDA s review and approval of NDAs. For instance, a sponsor may seek FDA designation of a drug candidate as a fast track product. Fast track products are those products intended for the treatment of a serious or life-threatening condition and which demonstrate the potential to address unmet medical needs for such conditions. If fast track designation is obtained, the FDA may initiate review of sections of an NDA before the application is complete. This rolling review is available if the applicant provides and the FDA approves a schedule for the remaining information. In some cases, a fast track product may be approved on the basis of either a clinical endpoint or a surrogate endpoint that is reasonably likely to predict clinical benefit under the FDA s accelerated approval regulations. Approvals of this kind typically include requirements for appropriate post-approval Phase IV clinical trials to validate the surrogate endpoint or otherwise confirm the effect of the clinical endpoint. In addition, drug candidates may be eligible for priority review, or review within a six month timeframe from the date a complete NDA is accepted for filing, if a sponsor shows that its drug candidate provides a significant improvement compared to marketed drugs. When appropriate, we intend to seek fast track designation and/or accelerated approval for our drugs. We cannot predict whether any of our drug candidates will obtain a fast track and/or accelerated approval designation, or the ultimate impact, if any, of the fast track or the accelerated approval process on the timing or likelihood of FDA approval of any of our proposed drugs.

Drugs may be marketed only for the FDA approved indications and in accordance with the provisions of the approved labeling. Further, if there are any modifications to the drug, including changes in indications, labeling, or manufacturing processes or facilities, the applicant may be required to submit and obtain FDA approval of a new NDA or NDA supplement, which may require us to develop additional data or conduct additional preclinical studies and clinical trials.

Before approving an application, the FDA will inspect the facility or the facilities at which the finished drug product, and sometimes the active drug ingredient, is manufactured, and will not approve the drug unless cGMP compliance is satisfactory. The FDA may also inspect the sites at which the clinical trials were conducted to assess their compliance, and will not approve the drug unless compliance with GCP requirements is satisfactory.

The testing and approval processes require substantial time, effort and financial resources, and each may take several years to complete. The FDA may not grant approval on a timely basis, or at all. Even if we believe a clinical trial has demonstrated safety and efficacy of one of our drug candidates for the treatment of a disease, the results may not be satisfactory to the FDA. Preclinical and clinical data may be interpreted by the FDA in different ways, which could delay, limit or prevent regulatory approval. We may encounter difficulties or unanticipated costs in our efforts to secure necessary governmental approvals which could delay or preclude us from marketing drugs. The FDA may limit the indications for use or place other conditions on any approvals that could restrict the commercial application of the drugs. After approval, certain changes to the approved drug, such as adding new indications, manufacturing changes, or additional labeling claims are subject to further FDA review and approval. Depending on the nature of the change proposed, an NDA supplement must be filed and approved before the change may be implemented. For many proposed post-approval changes to an NDA, the FDA has up to 180 days to review the application. As with new NDAs, the review process is often significantly extended by the FDA requests for additional information or clarification.

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Other Regulatory Requirements

Any drugs manufactured or distributed by us or our collaborators pursuant to FDA approvals would be subject to continuing regulation by the FDA, including recordkeeping requirements and reporting of adverse experiences associated with the drug. Drug manufacturers and their subcontractors are required to register their establishments with the FDA and certain state agencies, and are subject to periodic unannounced inspections by the FDA and certain state agencies for compliance with ongoing regulatory requirements, including cGMPs, which impose certain procedural and documentation requirements upon us and our third party manufacturers. Failure to comply with the statutory and regulatory requirements can subject a manufacturer to possible legal or regulatory action, such as warning letters, suspension of manufacturing, seizure of product, injunctive action or possible civil penalties. We cannot be certain that we or our present or future third party manufacturers or suppliers will be able to comply with the cGMP regulations and other ongoing FDA regulatory requirements. If we or our present or future third party manufacturers or suppliers are not able to comply with these requirements, the FDA may halt our clinical trials, require us to recall a drug from distribution or withdraw approval of the NDA for that drug.

The FDA closely regulates the post-approval marketing and promotion of drugs, including standards and regulations for direct-to-consumer advertising, off-label promotion, industry-sponsored scientific and educational activities and promotional activities involving the Internet. A company can make only those claims relating to safety and efficacy that are approved by the FDA. Failure to comply with these requirements can result in adverse publicity, warning letters, corrective advertising and potential civil and criminal penalties. Physicians may prescribe legally available drugs for uses that are not described in the product s labeling and that differ from those tested by us and approved by the FDA. Such off-label uses are common across medical specialties. Physicians may believe that such off-label uses are the best treatment for many patients in varied circumstances. The FDA does not regulate the behavior of physicians in their choice of treatments. The FDA does, however, impose stringent restrictions on manufacturers communications regarding off-label use.

Healthcare Reform

In March 2010, the President signed one of the most significant healthcare reform measures in decades. The Patient Protection and Affordable Care Act, as amended by the Health Care and Education Reconciliation Act, collectively known as the Affordable Care Act, substantially changes the way healthcare will be financed by both governmental and private insurers, and significantly impacts the pharmaceutical industry. The comprehensive $940 billion dollar overhaul is expected to extend coverage to approximately 32 million previously uninsured Americans. The Affordable Care Act contains a number of provisions, including those governing enrollment in federal healthcare programs, reimbursement changes and fraud and abuse, which will impact existing government healthcare programs and will result in the development of new programs, including Medicare payment for performance initiatives and improvements to the physician quality reporting system and feedback program. Additionally, the Affordable Care Act:

- mandates a further shift in the burden of Medicaid payments to the states;
- increases the minimum level of Medicaid rebates payable by manufacturers of brand-name drugs from 15.1% to 23.1%;
- requires collection of rebates for drugs paid by Medicaid managed care organizations;
- requires manufacturers to participate in a coverage gap discount program, under which they must agree to offer 50 percent point-of-sale discounts off negotiated prices of applicable brand drugs to eligible beneficiaries during their coverage gap period, as a condition for the manufacturer s outpatient drugs to be covered under Medicare Part D, beginning January 2011; and
- imposes a non-deductible annual fee on pharmaceutical manufacturers or importers who sell branded prescription drugs to specified federal government programs.

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The Affordable Care Act also establishes an Independent Payment Advisory Board, or IPAB, to reduce the per capita rate of growth in Medicare spending. Beginning in 2014, IPAB is mandated to propose changes in Medicare payments if it is determined that the rate of growth of Medicare expenditures exceeds target growth rates. The IPAB has broad discretion to propose policies to reduce expenditures, which may have a negative impact on payment rates for services, including imaging services. A proposal made by the IPAB is required to be implemented by the U.S. government s Centers for Medicare & Medicaid Services unless Congress adopts a proposal with savings greater than those proposed by the IPAB. IPAB proposals may impact payments for physician and free-standing services beginning in 2015 and for hospital services beginning in 2020.

A number of states have challenged the constitutionality of certain provisions of the Affordable Care Act, and many of these challenges are still pending final adjudication in several jurisdictions. Congress has also proposed a number of legislative initiatives, including possible repeal of the Affordable Care Act. At this time, it remains unclear whether there will be any changes made to the Affordable Care Act, whether to certain provisions or its entirety.

In addition, other legislative changes have been proposed and adopted since the Affordable Care Act was enacted. Most recently, on August 2, 2011, the President signed into law the Budget Control Act of 2011, which, among other things, creates the Joint Select Committee on Deficit Reduction to recommend proposals in spending reductions to Congress. In the event that the Joint Select Committee is unable to achieve a targeted deficit reduction of at least $1.2 trillion for the years 2013 through 2021, or Congress does not act on the committee s recommendation, without amendment, by December 23, 2011, an automatic reduction is triggered. These automatic cuts would be made to several government programs and, with respect to Medicare, would include aggregate reductions to Medicare payments to providers of up to 2% per fiscal year, starting in 2013. Further, on September 19, 2011, President Obama presented his Plan for Economic Growth and Deficit Reduction to the Joint Select Committee, which includes $248 billion in Medicare savings ($240 billion of which comes from reducing and collecting Medicare payments incorrectly paid) and $72 billion in Medicaid savings. Beginning in 2017, the President s proposal also shifts more of the Medicare costs to newly enrolled beneficiaries, including an increase in patient deductibles under Medicare Part B for certain beneficiaries, and increases Part B and Part D premiums for higher-income beneficiaries. The full impact on our business of the Affordable Care Act and other new laws is uncertain. Nor is it clear whether other legislative changes will be adopted, if any, or how such changes would affect the demand for our drugs once commercialized.

Third-Party Payor Coverage and Reimbursement

Although none of our drug candidates has been commercialized for any indication, if they are approved for marketing, commercial success of our drug candidates will depend, in part, upon the availability of coverage and reimbursement from third-party payors at the federal, state, and private levels. Government payor programs, including Medicare and Medicaid, private health care insurance companies, and managed-care plans have attempted to control costs by limiting coverage and the amount of reimbursement for particular procedures or drug treatments. The U.S. Congress and state legislatures from time to time propose and adopt initiatives aimed at cost-containment. Ongoing federal and state government initiatives directed at lowering the total cost of health care will likely continue to focus on health care reform, the cost of prescription pharmaceuticals and on the reform of the Medicare and Medicaid payment systems. Examples of how limits on drug coverage and reimbursement in the United States may cause reduced payments for drugs in the future include:

- changing Medicare reimbursement methodologies;
- fluctuating decisions on which drugs to include in formularies;
- revising drug rebate calculations under the Medicaid program; and
- reforming drug importation laws.

Some third-party payors also require pre-approval of coverage for new or innovative devices or drug therapies before they will reimburse health care providers who use such therapies. While we cannot predict

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whether any proposed cost-containment measures will be adopted or otherwise implemented in the future, the announcement or adoption of these proposals could have a material adverse effect on our ability to obtain adequate prices for our drug candidates and operate profitably.

Other Healthcare Laws and Regulations

We are also subject to healthcare regulation and enforcement by the federal government and the states and foreign governments in which we conduct our business. The laws that may affect our ability to operate include:

- the federal healthcare programs Anti-Kickback Statute, which prohibits, among other things, persons from knowingly and willfully soliciting, receiving, offering or paying remuneration, directly or indirectly, in exchange for or to induce either the referral of an individual for, or the purchase, order or recommendation of, any good or service for which payment may be made under federal healthcare programs such as the Medicare and Medicaid programs;

- federal false claims laws which prohibit, among other things, individuals or entities from knowingly presenting, or causing to be presented, claims for payment from Medicare, Medicaid, or other third-party payors that are false or fraudulent;

- federal criminal laws that prohibit executing a scheme to defraud any healthcare benefit program or making false statements relating to healthcare matters;

- the federal Health Insurance Portability and Accountability Act of 1996, as amended by the Health Information Technology for Economic and Clinical Health Act, which governs the conduct of certain electronic healthcare transactions and protects the security and privacy of protected health information;

- state law equivalents of each of the above federal laws, such as anti-kickback and false claims laws which may apply to items or services reimbursed by any third-party payor, including commercial insurers.

If our operations are found to be in violation of any of the laws described above or any other governmental laws and regulations that apply to us, we may be subject to penalties, including civil and criminal penalties, damages, fines, the curtailment or restructuring of our operations, the exclusion from participation in federal and state healthcare programs and imprisonment, any of which could adversely affect our ability to operate our business and impact our financial results.

International Regulation

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

Under European Union regulatory systems, marketing authorizations may be submitted either under a centralized or mutual recognition procedure. The centralized procedure provides for the grant of a single marking authorization that is valid for all European Union member states. The mutual recognition procedure provides for mutual recognition of national approval decisions. Under this procedure, the holder of a national marking authorization may submit an application to the remaining member states. Within 90 days of receiving the applications and assessment report, each member state must decide whether to recognize approval.

In addition to regulations in Europe and the United States, we will be subject to a variety of foreign regulations governing clinical trials and commercial distribution of our future drugs.

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Employees

As of September 30, 2011, we had 64 full-time employees, 32 of whom hold Ph.D.s, M.D.s or both. Of our total workforce, 53 employees are engaged in research and development, and 11 employees are engaged in business development, finance, legal, human resources, facilities, information technology administration and general management. We have no collective bargaining agreements with our employees and we have not experienced any work stoppages. We believe that our relations with our employees are good.

Facilities

Our corporate headquarters are located in Mountain View, California, where we lease 35,755 square feet of office and laboratory space. In April 2004, we entered into a ten-year lease agreement for that facility.

We believe that our existing facilities are adequate for our current needs, as the facility has sufficient laboratory space to house additional scientists to be hired as we expand. When our leases expire, we may exercise our renewal options or look for additional or alternate space for our operations and we believe that suitable additional or alternative space will be available in the future on commercially reasonable terms.

Legal Proceedings

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

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MANAGEMENT

Executive Officers and Directors

The following table sets forth information regarding our executive officers and directors, as of December 31, 2011:

Name	Age	Position(s)
Thomas J. Schall, Ph.D.	52	President, Chief Executive Officer and Director
Markus J. Cappel, Ph.D.	50	Chief Business Officer and Treasurer
Susan M. Kanaya	49	Senior Vice President, Finance, Chief Financial Officer and Secretary
Juan C. Jaen, Ph.D.	54	Senior Vice President, Drug Discovery and Chief Scientific Officer
Petrus (Pirow) Bekker, M.D., Ph.D.	51	Senior Vice President of Medical and Clinical Affairs
Rishi Gupta, J.D.[(1)(2)(3)]	34	Director
Roger C. Lucas, Ph.D.[(3)]	68	Director
Geoffrey M. Parker[(1)(3)]	47	Director
Edward E. Penhoet, Ph.D.[(1)(2)(3)]	71	Director

(1) Member of the audit committee.

(2) Member of the compensation committee.

(3) Member of the nominating and corporate governance committee.

Thomas J. Schall, Ph.D., is the founder of our company and has served as our President, Chief Executive Officer and Director since November 1996. From December 1993 to November 1996, Dr. Schall worked at the DNAX Research Institute, a division of Schering-Plough Corporation, a pharmaceutical company. Prior to his work at the DNAX Research Institute, he worked as a scientist with Genentech, Inc., a pharmaceutical company. Dr. Schall participated in some of the earliest discoveries of chemokine system function and activities. Dr. Schall cloned one of the first chemokines to be discovered, and provided some of the earliest data for the existence of the previously unknown family of molecules which later came to be called the chemokines. Dr. Schall s laboratories have been responsible for the discovery or co-discovery of almost one-third of all known chemokine receptors. Dr. Schall received his B.S. in biology from Northern Illinois University and his Ph.D. in cancer biology from Stanford University. We believe Dr. Schall is qualified to serve on our board of directors because of his extensive executive leadership experience, many years of service as one of our directors and our President and Chief Executive Officer and extensive scientific expertise and knowledge of the chemokine system.

Markus J. Cappel, Ph.D., has served as our Chief Business Officer since February 2007, and Treasurer since August 2004. From March 2003 to February 2007, he served as our Senior Vice President of Corporate and Business Development. From October 2001 to March 2003, Dr. Cappel served as our Vice President of Business Development. Prior to joining us, Dr. Cappel served as Vice President of Business Development at Alkermes, Inc., a biotechnology company, from 1998 to 2001. Prior to this, he served as Director of Business Development with Millennium Pharmaceuticals as well as in various business development roles at Cygnus, Inc., a biotechnology company. Dr. Cappel received his B.S. in pharmacy and his Ph.D. in pharmaceutics from J.W. Goethe University, Frankfurt, Germany, and his M.B.A. from Harvard Business School. Dr. Cappel also completed postdoctoral studies in pharmaceutics at the University of Michigan.

Susan M. Kanaya has served as our Senior Vice President, Finance, and Chief Financial Officer since January 2006, and Secretary since February 2006. Prior to joining us, Ms. Kanaya served as Senior Vice President, Finance, and Chief Financial Officer at Kosan Biosciences Inc., a biotechnology company, from 1999 to 2005. Prior to this, she served in financial management positions at SUGEN, Inc., a biotechnology company, from 1994 to 1999, most recently as Vice President, Finance, and Treasurer. Ms. Kanaya also served as Controller with high technology companies and as a public accountant with KPMG. Ms. Kanaya received her B.S. in business administration from the University of California, Berkeley.

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Juan C. Jaen, Ph.D., has served as our Chief Scientific Officer since February 2010. From January 2007 to February 2010, Dr. Jaen served as our Senior Vice President, Drug Discovery. From 1996 to 2006, Dr. Jaen was employed at Tularik, a biotechnology company, and then Amgen Inc., a pharmaceutical company (following Tularik s acquisition by Amgen in 2004), most recently serving as Vice President of Chemistry. From 1983 to 1996, Dr. Jaen was employed at Parke-Davis/Warner-Lambert, a pharmaceutical company, most recently as Chemistry Director, with responsibility for the neurodegenerative disease research area. Dr. Jaen received his B.S. from the Universidad Complutense in Madrid, Spain and holds a Ph.D. in organic chemistry from the University of Michigan.

Petrus (Pirow) Bekker, M.D., Ph.D., has served as our Senior Vice President of Clinical and Medical Affairs since February 2009. From April 2005 to February 2009, Dr. Bekker served as our Vice President of Clinical and Medical Affairs. Prior to joining us, Dr. Bekker worked at Amgen Inc., where he served as Senior Director of Global Safety from July 2004 to April 2005 and as Senior Director of Clinical Development and Director of Clinical Development from December 1997 to July 2004. Prior to this, he served as a clinical researcher and scientist at Procter & Gamble Pharmaceuticals in various capacities for seven years. Dr. Bekker received his Ph.D. in molecular biology from Pennsylvania State University and his M.D. and medical training in South Africa at the University of Pretoria.

Rishi Gupta, J.D., has served as a member of our board of directors since May 2011. Since 2004, Mr. Gupta has served as Private Equity Principal of OrbiMed Advisors LLC, a healthcare asset management company. From 1999 to 2000, Mr. Gupta served as a corporate finance analyst in healthcare investment banking at Raymond James & Associates and from 2000 to 2001 served as Manager of Corporate Development at Veritas Medicine. Mr. Gupta received his A.B. degree magna cum laude in Biochemical Sciences from Harvard College and holds a J.D. from the Yale Law School. Mr. Gupta also serves on the board of directors of several private companies. We believe Mr. Gupta is qualified to serve on our board of directors because of his experience in venture capital and financial services and investing in life sciences companies.

Roger C. Lucas, Ph.D., has served as a member of our board of directors since September 1997. Since 1995, Dr. Lucas has served as Vice Chairman and a member of the board of directors of Techne Corporation, a biotechnology company. From 1985 to 1995, Dr. Lucas served as the Chief Scientific Officer, Senior Executive Vice President and Secretary of Techne Corporation and the founder of its Biotechnology Division. Prior to this, Dr. Lucas was Vice President of Research at R&D Systems, now a subsidiary of Techne, where he worked for over 10 years. Dr. Lucas received his B.S. in biology and chemistry from St. Mary s College, Minnesota, and his Ph.D. in physiology and cell biology from the Illinois Institute of Technology. Dr. Lucas also presently serves on the board of directors of a number of privately held companies. We believe Dr. Lucas is qualified to serve on our board of directors because of his experience in the healthcare industry as an entrepreneur and a director of a range of public and private companies and his leadership and management experience from his service as an executive for a public life sciences company.

Geoffrey M. Parker, has served as a member of our board of directors since December 2009. Since September 2010, Mr. Parker has served as Senior Vice President and Chief Financial Officer of Anacor Pharmaceuticals, Inc. after serving in a consulting capacity since December 2009. From July 2009 to July 2010, Mr. Parker served in a consulting capacity as Chief Business Officer of InteKrin Therapeutics, Inc., a biotechnology company, and previously served as Managing Director and Partner in the Investment Banking Division of Goldman, Sachs & Co. From 1997 to 2009, Mr. Parker directed Goldman Sachs West Region Healthcare Investment Banking practice. From 1995 to 1997, Mr. Parker was Vice President at Feibusch & Co., a venture capital firm in Larkspur, California. Mr. Parker received his A.B. in Engineering Sciences and Economics from Dartmouth College and his M.B.A. from Stanford University. We believe Mr. Parker is qualified to serve on our board of directors because of his financial sophistication, his experience as the Chief Financial Officer of a public biotechnology company and his management background as an executive in the financial services industry.

Edward E. Penhoet, Ph.D., has served as a member of our board of directors since December 2007. Since August 2000, Dr. Penhoet has served as a director of Alta Partners, a venture capital firm. Dr. Penhoet is currently a member of the President s Council of Advisors for Science and Technology (PCAST). Dr. Penhoet

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was a member of the Independent Citizens Oversight Committee of the California Institute of Regenerative Medicine where he served as the Vice Chairman from 2004 to 2008. From July 1998 to July 2002, Dr. Penhoet served as the Dean of the School of Public Health and as a Professor of Public Health and of Molecular and Cellular Biology at the University of California, Berkeley. Dr. Penhoet was a co-founder of the Chiron Corporation, a biotechnology company, where he served as President, Chief Executive Officer and a director from its formation in 1981 to April 1998. From 1971 to 1981, Dr. Penhoet was a faculty member of the Biochemistry Department at the University of California, Berkeley. From 2004 to 2008, Dr. Penhoet served as President of the Gordon and Betty Moore Foundation. Dr. Penhoet is a member of the Institute of Medicine of the National Academy of Sciences and The American Academy of Arts & Sciences and currently serves, or has during the past five years served, as a director of Chiron, Corcept Therapeutics, Inc., IDM Pharma, Inc. and Renovis, Inc., together with several privately held biotechnology companies. Dr. Penhoet received a B.A. in Biology from Stanford University and a Ph.D. in Biochemistry from the University of Washington. We believe Dr. Penhoet s qualifications to sit on our board of directors include his extensive knowledge of biochemistry and related science, together with his experience as a founder and chief executive officer of a leading biotechnology company and his corporate governance expertise. We believe Dr. Penhoet is qualified to serve on our board of directors because of his extensive leadership experience in the healthcare industry as an entrepreneur, venture capitalist and executive and his service on the boards of directors of a range of public and private life sciences companies.

Board Composition

Our board of directors currently consists of five members with one vacancy. Our amended and restated bylaws will permit the authorized number of directors to be determined by resolution of the board of directors. Each director elected shall hold office until his or her successor is elected and qualified. Effective upon the closing of this offering, we will divide the terms of office of the directors into three classes:

Class I, whose term will expire at the annual meeting of stockholders to be held in 2013;

Class II, whose term will expire at the annual meeting of stockholders to be held in 2014; and

Class III, whose term will expire at the annual meeting of stockholders to be held in 2015.

Upon the closing of this offering, Class I shall consist of Thomas J. Schall, Ph.D. and one vacancy, Class II shall consist of Geoffrey M. Parker and Rishi Gupta, J.D. and Class III shall consist of Edward E. Penhoet, Ph.D. and Roger C. Lucas, Ph.D. At each annual meeting of stockholders after the initial classification, the successors to directors whose terms will then expire shall serve from the time of election and qualification until the third annual meeting following election and until their successors are duly elected and qualified. A resolution of the board of directors may change the authorized number of directors. Any additional directorships resulting from an increase in the number of directors will be distributed among the three classes so that, as nearly as possible, each class will consist of one-third of the directors. This classification of the board of directors may have the effect of delaying or preventing changes in control or management of our company.

Board Committees

Our board of directors has established three standing committees: the audit committee, the compensation committee and the nominating and corporate governance committee.

Audit Committee

The audit committee is composed of Messrs. Parker and Gupta and Dr. Penhoet (our audit committee financial expert), all of whom will be independent, within the meaning of applicable SEC rules and regulations of The Nasdaq Stock Market LLC, or Nasdaq, upon completion of this offering. We believe that the composition and functioning of our audit committee complies with all applicable requirements of the Sarbanes-Oxley Act of 2002, and all applicable SEC and Nasdaq rules and regulations. We intend to comply with future requirements to the extent they become applicable to us.

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Our audit committee is responsible for overseeing our accounting and financial reporting processes and audits of our consolidated financial statements on behalf of our board of directors. Our independent auditor reports directly to the audit committee. The specific powers and responsibilities of our audit committee include:

- appointing, assessing the qualifications of, compensating, retaining, and overseeing the work of our independent auditor, for the purpose of preparing or issuing an auditor s report or performing other audit, review, and attest services;
- reviewing our annual audited consolidated financial statements with management and our independent auditor;
- reviewing the appointment of, replacement of, and meeting with our internal auditor to discuss significant reports to management;
- overseeing and monitoring the integrity of our consolidated financial statements, our compliance with legal and regulatory requirements as they relate to consolidated financial statements or accounting matters, our independent auditor s qualifications, independence and the performance of our internal accounting and financial controls;
- determining whether to recommend to our board of directors that the audited financial statements be included in our annual report for the fiscal year subject to the audit;
- reviewing all related party transactions on an ongoing basis;
- preparing the report that SEC rules require be included in our annual proxy statement;
- providing our board of directors with the results of its monitoring and recommendations;
- providing our board of directors with additional information and materials as it deems necessary to make our board of directors aware of significant financial matters that require the attention of our board of directors; and
- evaluating its own performance on an annual basis.

Compensation Committee

The compensation committee is composed of Dr. Penhoet and Mr. Gupta, both of whom will be independent, within the meaning of applicable SEC and Nasdaq rules, upon completion of this offering. We believe that the composition and functioning of our compensation committee complies with all applicable requirements of the Sarbanes-Oxley Act of 2002, and all applicable SEC and Nasdaq rules and regulations. We intend to comply with future requirements to the extent they become applicable to us.

Our compensation committee is responsible for, among other things:

- reviewing and approving our corporate goals and objectives relating to the compensation of our Chief Executive Officer, evaluating the performance of our Chief Executive Officer in light of those goals and objectives, and determining and approving the compensation of our Chief Executive Officer based on such evaluation;

reviewing and approving the compensation of our officers and certain employees; reviewing and approving general compensation goals and guidelines for employees and the criteria by which bonuses, long-term incentive compensation, stock options, employee pension and welfare benefits plans are determined;

determining our policy with respect to change of control or parachute payments;

managing and reviewing executive officer and director indemnification and insurance matters;

preparing the compensation committee report to be included as part of our annual proxy statement; and

evaluating its own performance on an annual basis.

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Nominating and Corporate Governance Committee

The nominating and corporate governance committee is composed of Messrs. Parker and Gupta and Drs. Penhoet and Lucas, all of whom will be independent, within the meaning of applicable SEC and Nasdaq rules, upon completion of this offering. We believe that the composition and functioning of our nominating and corporate governance committee complies with all applicable requirements of the Sarbanes-Oxley Act of 2002, and all applicable SEC and Nasdaq rules and regulations. We intend to comply with future requirements to the extent they become applicable to us.

Our nominating and corporate governance committee is responsible for, among other things:

- overseeing our board of director s annual review of its performance, composition, and organization, and making recommendations on these matters to our board of directors;
- reviewing, soliciting and making recommendations to our board of directors and stockholders with respect to candidates for election to our board of directors;
- reviewing the performance of each current director and determining whether to recommend the nomination of such director for an additional term; and
- evaluating its own performance on an annual basis.

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EXECUTIVE AND DIRECTOR COMPENSATION

Compensation Discussion and Analysis

Overview

This compensation discussion and analysis provides information about the material elements of our executive compensation program for our named executive officers, consisting of the following persons:

- Thomas J. Schall, Ph.D., our President and Chief Executive Officer;
- Susan M. Kanaya, our Chief Financial Officer, Secretary and Senior Vice President, Finance;
- Markus J. Cappel, Ph.D., our Chief Business Officer and Treasurer;
- Juan C. Jaen, Ph.D., our Chief Scientific Officer and Senior Vice President, Drug Discovery; and
- Petrus J. Bekker, M.D., Ph.D., our Senior Vice President, Medical and Clinical Affairs.

Specifically, this compensation discussion and analysis provides an overview of our executive officer compensation philosophy, the overall objectives of our executive officer compensation program, and each compensation element that we provide. In addition, we explain how and why the compensation committee and our board of directors arrived at specific compensation policies and decisions involving our named executive officers during the year ended December 31, 2011.

Objectives of Our Compensation Program

We recognize that the ability to excel depends on the integrity, knowledge, imagination, skill, diversity and teamwork of our employees. To this end, we strive to create an environment of mutual respect, encouragement and teamwork an environment that rewards commitment and performance and that is responsive to the needs of our employees. The objectives of our compensation and benefits programs for our employees generally, and for our named executive officers specifically, are to:

- attract, engage and retain the workforce that helps ensure our future success;
- motivate and inspire employee behavior that fosters a high-performance culture;
- support a cost-effective and flexible business model;
- reinforce key business objectives; and
- align employee interests with stockholder interests.

Most of our compensation elements simultaneously fulfill one or more of these objectives. These elements consist of:

base salaries;

annual performance bonuses;

long-term equity incentives;

perquisites, health, welfare and retirement benefits; and

post-termination benefits.

We believe that each element aligns the interests of our named executive officers with the interests of our stockholders in different ways, whether through focusing on short-term and long-term performance goals, promoting an ownership mentality toward one s job, linking individual compensation opportunities to our performance or by ensuring healthy employees. This mix of compensation is intended to ensure that total compensation reflects our overall success or failure and to motivate our named executive officers to meet appropriate performance measures. In determining each element of compensation for any given year, our board of directors and our compensation committee consider and determine each element individually and then review the resulting total compensation and determine whether it is reasonable and competitive. We have no

pre-established policy or target for the allocation between either cash and non-cash or short-term and long-term incentive compensation. Each of these compensation elements is described in more detail below.

Compensation Determination Process

The compensation committee of our board of directors develops, reviews and approves each of the elements of the executive officer compensation program of our company as a whole, and for our named executive officers individually, and regularly assesses the effectiveness and competitiveness of the program.

In the first quarter of each year, the compensation committee reviews the performance of each of our named executive officers during the previous year. At this time the compensation committee also reviews our performance relative to the corporate performance objectives set by the board of directors for the previous year and makes the final annual bonus payment determinations based on our performance and the compensation committee s evaluation of each named executive officer s performance for the prior year. In connection with this review, the compensation committee also reviews and adjusts, as appropriate, annual base salaries for our named executive officers and grants, as appropriate, additional equity awards to our named executive officers and certain other eligible employees for the coming fiscal year. During the first quarter of each year, our compensation committee also reviews and recommends to the full board of directors for approval the corporate performance objectives for purposes of our performance bonus programs for that year.

Our Chief Executive Officer, with the assistance and support of the human resources department and the other executive officers, aids the compensation committee by providing annual recommendations regarding the compensation of all of our named executive officers, other than himself. The compensation committee also, on occasion, meets with our Chief Executive Officer to obtain recommendations with respect to our compensation programs and practices generally. The compensation committee considers, but is not bound to accept, the chief executive officer s recommendations with respect to named executive officer compensation. In the beginning of each year, our named executive officers work with our Chief Executive Officer to establish their individual performance goals for the year, based on their respective roles within the company.

Our Chief Executive Officer generally attends all of the compensation committee meetings, but the compensation committee also holds executive sessions that are not attended by any members of management or non-independent directors, as needed from time to time. Any decisions regarding our Chief Executive Officer s compensation package are made without him present.

Role of Compensation Consultant and Comparable Company Information

The compensation committee is authorized to retain the services of third-party compensation consultants and other outside advisors from time to time, as the committee sees fit, in connection with compensation matters. Compensation consultants and other advisors retained by the compensation committee will report directly to the compensation committee which has the authority to select, retain and terminate any such consultants or advisors. During 2011, the compensation committee reviewed our compensation programs and practices in light of certain market comparison information compiled by our management team at the request of the compensation committee. Specifically, for 2011 compensation decisions, the compensation committee referred to a market comparison group, which included comparable companies within our industry, other biotechnology companies of similar size in terms of revenue and market capitalization, and companies which are otherwise relevant. For 2011, these companies included: Affymax, Inc., Array BioPharma, Inc., Cytokinetics, Incorporated, Exelixis, Inc., MAP Pharmaceuticals, Inc., Rigel Pharmaceuticals, Inc. and XenoPort, Inc. These companies were selected because all of them were at a similar stage of development, had a total value/market capitalization or size similar to ours or were otherwise companies which we believe we compete against for executive talent. During 2011, the compensation committee also referred to the 2011 Radford Global Life Sciences Survey, which consists of public companies throughout the United States primarily from the life sciences industry with between 50 and 150 employees.

With respect to Dr. Schall s and Ms. Kanaya s cash compensation, the data from our market comparison group was weighted more heavily by the compensation committee than the Radford survey data, as there were

individuals at our peer companies who held positions with titles that were an exact match for Dr. Schall s and Ms. Kanaya s. For all other named executive officers, the Radford survey data was more heavily weighted by the compensation committee, as there were no exact matches for the titles of such other officers at our peer group companies. With respect to the survey data presented to the compensation committee, the identities of the individual companies included in the survey were not provided to the compensation committee, and the compensation committee did not refer to individual compensation information for such companies.

We expect that the compensation committee will continue to review comparable company survey data in connection with setting the compensation we offer our named executive officers to help ensure that our compensation programs are competitive and fair.

However, our compensation committee does not establish compensation levels based on benchmarking. Our compensation committee has relied instead upon the judgment of its members in making compensation decisions, after reviewing our performance and carefully evaluating a named executive officer s performance during the year against established goals, leadership qualities, operational performance, business responsibilities, career with our company, current compensation arrangements and long-term potential to enhance stockholder value. While competitive market compensation paid by other companies is reviewed by the compensation committee, the compensation committee does not attempt to set compensation at a certain target percentile within a peer group or otherwise rely entirely on that data to determine named executive officer compensation. Instead, the compensation committee incorporates flexibility into our compensation programs and in the assessment process to respond to and adjust for the evolving business environment.

We strive to achieve an appropriate mix between equity incentive awards and cash payments in order to meet our objectives. Any apportionment goal is not applied rigidly and does not control our compensation decisions, and our compensation committee does not have any policies for allocating compensation between long-term and short-term compensation or cash and non-cash compensation. Our mix of compensation elements is designed to reward recent results and motivate long-term performance through a combination of cash and equity incentive awards. We believe the most important indicator of whether our compensation objectives are being met is our ability to motivate our named executive officers to deliver superior performance and retain them to continue their careers with us on a cost-effective basis.

The compensation levels of our named executive officers reflect to a significant degree the varying roles and responsibilities of such executive officers. As a result of the compensation committee s and the board of directors assessment of our Chief Executive Officer s role and responsibilities within our company, there are significant compensation differentials between him and our other named executive officers.

We do not yet have a formal policy to adjust or recover awards or payments if the relevant performance measures upon which they are based are restated or are otherwise adjusted in a manner that would otherwise reduce the size of the initial payment or award.

Executive Compensation Elements

The following describes each element of our executive compensation program, the rationale for each, and how compensation amounts are determined.

Base Salaries

In general, base salaries for our named executive officers are initially established through arm s length negotiation at the time the executive officer is hired, taking into account such executive officer s qualifications, experience and prior salary. We have entered into employment agreements with each of our named executive officers setting forth their initial base salaries, which employment agreements were approved by our compensation committee. Base salaries of our named executive officers are approved and reviewed annually by our compensation committee and adjustments to base salaries are based on the scope of an executive officer s responsibilities, individual contribution, prior experience and sustained performance. Decisions regarding salary increases may take into account the executive officer s current salary, equity ownership and the amounts paid to

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an executive officer s peers inside our company by conducting an internal analysis which compares the pay of an executive officer to other members of the management team. Base salaries are also reviewed in the case of promotions or other significant changes in responsibility. Base salaries are not automatically increased if the compensation committee believes that other elements of the named executive officer s compensation are more appropriate in light of our stated objectives. This strategy is consistent with our intent of offering compensation that is cost-effective, competitive and contingent on the achievement of performance objectives.

Our Chief Executive Officer s base salary is based upon the same policies and criteria used for other named executive officers as described above. Each year the compensation committee reviews the chief executive officer s compensation arrangements and his individual performance for the previous fiscal year, as well as our performance as a whole, and makes adjustments to such compensation, if appropriate.

In February 2011, the compensation committee set annual base salaries for our named executive officers to be in effect until the next annual review. The 2011 base salary for each of the named executive officer represents a 3.5% increase above his or her 2010 base salary. The 2011 annual base salaries of the named executive officers are as follows: Dr. Schall, $465,750; Ms. Kanaya, $359,625; Dr. Cappel, $342,554; Dr. Jaen, $362,250; and Dr. Bekker, $356,891. Such increase was based on the compensation committee s review of comparable company compensation data, as discussed above, and general budget considerations based on the company s financial position. The compensation committee felt that this increase represented a competitive merit-based increase for our named executive officers. The actual base salaries paid to our named executive officers for 2011 are set forth in the Summary Compensation Table below.

Annual Performance Bonuses

Each named executive officer is also eligible for an annual performance bonus based upon the achievement of certain corporate performance goals and objectives approved by our board of directors and individual performance.

Bonuses are set based on the executive officer s base salary as of the end of the bonus year, and are expected to be paid out in the first quarter of the following year. Pursuant to their employment agreements, each of the named executive officers is eligible to receive a target bonus of 25% of his or her base salary, with the exception of Dr. Schall, who is eligible to receive a target bonus of 40% of his base salary. Each named executive officer s bonus is based entirely on performance relative to corporate objectives.

At the beginning of each year, the board of directors (considering the recommendations of the compensation committee and management) sets corporate goals and milestones for the year. These goals and milestones and the proportional emphasis placed on each are set by the board of directors after considering management input and our overall strategic objectives. These goals generally relate to factors such as financial targets, achievement of product development objectives and establishment of new collaborative arrangements. The board of directors, upon recommendation of the compensation committee, determines the level of achievement of the corporate goals for each year. This achievement level is then applied to each named executive officer s target bonus to determine that year s total bonus award.

All final bonus payments to our named executive officers are determined by our compensation committee. The actual bonuses awarded in any year, if any, may be more or less than the target, depending on individual performance and the achievement of corporate objectives and may also vary based on other factors at the discretion of the compensation committee.

2011 Bonuses

For 2011, the corporate performance objectives generally fell into the following categories: objectives related to continued progress in the area of clinical trials and pipeline development; drug discovery efforts and pipeline support; and financial and corporate objectives, including achieving revenue and cash utilization targets for 2011, expansion and implementation of infrastructure to support a public company and general business

development progress. With the exception of the financial targets described above, quantitative measures were not established for the corporate objectives during 2011. Instead these performance objectives and areas of emphasis were used as a guide by the board of directors in subjectively determining overall corporate performance as they represented those areas in which the named executive officers and our employees generally were expected to focus their efforts during the year. The three foregoing areas of emphasis were weighted based on their level of importance to our business plan.

Our board of directors has not yet met to evaluate management s performance relative to corporate performance objectives and no bonuses have been paid to our named executive officers for 2011.

Long-Term Equity Incentives

The goals of our long-term, equity-based incentive awards are to align the interests of our named executive officers and other employees, non-employee directors and consultants with the interests of our stockholders. Because vesting is based on continued service, our equity-based incentives also encourage the retention of our named executive officers through the vesting period of the awards. In determining the size of the long-term equity incentives to be awarded to our named executive officers, we take into account a number of internal factors, such as the relative job scope, the value of existing long-term incentive awards, individual performance history, prior contributions to us and the size of prior grants. Our compensation committee has not historically referred to competitive market data in determining long-term equity incentive awards. Based upon these factors, the compensation committee determines the size of the long-term equity incentives at levels it considers appropriate to create a meaningful opportunity for reward predicated on the creation of long-term stockholder value.

To reward and retain our named executive officers in a manner that best aligns employees interests with stockholders interests, we use stock options as the primary incentive vehicle for long-term compensation. We believe that stock options are an effective tool for meeting our compensation goal of increasing long-term stockholder value by tying the value of the stock options to our future performance. Because employees are able to profit from stock options only if our stock price increases relative to the stock option s exercise price, we believe stock options provide meaningful incentives to employees to achieve increases in the value of our stock over time.

We use stock options to compensate our named executive officers both in the form of initial grants in connection with the commencement of employment and annual retention grants. Annual retention grants of stock options are typically approved by the compensation committee during the summer of each year. While the majority of stock option awards to our employees have been made pursuant to our annual retention grant program, the compensation committee retains discretion to make stock option awards to employees at other times, including in connection with the hiring of an employee, the promotion of an employee, to reward an employee, for retention purposes or for other circumstances recommended by management or the compensation committee. We have not granted any equity awards other than stock options to date.

The exercise price of each stock option grant is the fair market value of our common stock on the grant date, as determined by our board of directors. Since June 2006, the fair market value of our common stock has been determined by an independent third party appraisal. Except as described below, we have never granted stock options with an exercise price that is less than the fair market value of our common stock on the grant date, as determined pursuant to our equity incentive plans. Stock option awards to our named executive officers typically vest over a four-year period as follows: 25% of the shares underlying the option vest on the first anniversary of the vesting commencement date and the remainder of the shares underlying the option vest in equal monthly installments over the remaining 36 months thereafter. From time to time, our compensation committee may, however, determine that a different vesting schedule is appropriate. For a description of certain accelerated vesting provisions applicable to such options, see Summary Compensation Equity Compensation and Other Benefit Plans below. We do not have any security ownership requirements for our named executive officers.

In August 2011, the compensation committee awarded the following options to our named executive officers: Ms. Kanaya, options to purchase 44,180 shares; Dr. Cappel, options to purchase 48,697 shares; Dr. Jaen,

options to purchase 49,425 shares; and Dr. Bekker, options to purchase 39,265 shares. Each of these option awards vests over four years, as described above, and has an exercise price of $6.90 per share, which the board of directors determined was the fair value per share of our common stock on the date of grant. Because we no longer had enough shares remaining available for issuance under our equity plans to grant options commensurate in number with past practice, Dr. Schall recommended to the compensation committee that he wished to be excluded from any grants and that the remaining option shares be divided among the other members of our executive management team. The compensation committee granted options at two-thirds the level recommended by Dr. Schall, leaving approximately 55,000 options available for grant under our equity plans. The compensation committee indicated that it would revisit whether to make additional grants to the executive management team and to Dr. Schall if and when additional shares become available for grant under our equity plans.

As a privately owned company, there has been no active market for our common stock. Accordingly, we have had no program, plan or practice pertaining to the timing of stock option grants to named executive officers coinciding with the release of material non-public information.

Perquisites, Health, Welfare and Retirement Benefits

The establishment of competitive benefit packages for our employees is an important factor in attracting and retaining highly qualified personnel.

Health and Welfare Benefits

Our named executive officers are eligible to participate in all of our employee benefit plans, including our medical, dental, vision, group life and disability insurance plans, in each case on the same basis as other employees. We believe that these health and welfare benefits help ensure that we have a productive and focused workforce through reliable and competitive health and other benefits.

Retirement Savings

All of our full-time employees in the United States, including our named executive officers, are eligible to participate in our 401(k) plan. Pursuant to our 401(k) plan, employees may elect to reduce their current compensation by up to the statutorily prescribed annual limit of $17,000 in 2012 (additional salary deferrals not to exceed $5,500 are available to those employees 50 years of age or older) and to have the amount of this reduction contributed to our 401(k) plan. While we may elect to make matching contributions, no such contributions have been made. The 401(k) Plan currently does not offer the ability to invest in our securities.

Perquisites

We do not provide significant perquisites or personal benefits to our named executive officers. We do, however, pay the premiums for term life insurance for our named executive officers.

Post-Termination Benefits

We have entered into employment agreements which provide for certain severance benefits in the event a named executive officer s employment is involuntarily or constructively terminated. Such severance benefits are intended and designed to alleviate the financial impact of an involuntary termination and maintain a stable work environment through salary continuation and equity award vesting acceleration. We provide severance benefits because they are essential to help us fulfill our objective of attracting and retaining key managerial talent. While these arrangements form an integral part of the total compensation provided to these individuals and are considered by the compensation committee when determining executive officer compensation, the decision to offer these benefits did not influence the compensation committee s determinations concerning other direct compensation or benefit levels. The compensation committee has determined that such arrangements offer

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protection that is competitive within our industry and for our company size and are designed to attract highly qualified individuals and encourage them to maintain their employment with us. In determining the severance benefits payable pursuant to the executive officer employment agreements, the compensation committee considered the input of our executive officers as to what they expected and what level of severance benefits would be sufficient to retain our current executive team and to recruit talented executive officers in the future. For a description of these employment agreements with our named executive officers, see Summary Compensation Employment Agreements below.

As described above, we routinely grant our named executive officers stock options under our equity incentive plans. For a description of the change in control provisions in such equity incentive plans applicable to these stock options, see Summary Compensation Equity Compensation Plans and Other Benefit Plans below.

Tax Deductibility of Executive Compensation

The compensation committee and our board of directors have considered the potential future effects of Section 162(m) of the Internal Revenue Code of 1986 as amended, or the Code, on the compensation paid to our executive officers. Section 162(m) disallows a tax deduction for any publicly held corporation for individual compensation exceeding $1.0 million in any taxable year for our Chief Executive Officer and each of the other named executive officers (other than our chief financial officer), unless compensation is performance based. As we are not currently publicly traded, our board of directors has not previously taken the deductibility limit imposed by Section 162(m) into consideration in setting compensation. Our compensation committee, however, has adopted a policy that, where reasonably practicable, we will seek to qualify the variable compensation paid to our executive officers for an exemption from the deductibility limitations of Section 162(m).

In approving the amount and form of compensation for our executive officers, the compensation committee will continue to consider all elements of the cost to our company of providing such compensation, including the potential impact of Section 162(m).

Accounting for Stock-Based Compensation

We follow Financial Accounting Standards Board Accounting Standards Codification Topic 718 (formerly known as SFAS No. 123(R)), or ASC Topic 718, for our stock-based compensation awards. ASC Topic 718 requires companies to calculate the grant date fair value of their stock-based awards using a variety of assumptions. This calculation is performed for accounting purposes and reported in the compensation tables below, even though recipients may never realize any value from their awards. ASC Topic 718 also requires companies to recognize the compensation cost of their stock-based awards in their income statements over the period that an employee is required to render service in exchange for the award.

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Summary Compensation Table

The following table shows information regarding the compensation of our named executive officers during the fiscal years ended December 31, 2011 and 2010.

Name and Principal Position	Year	Salary	Bonus	Stock Option Awards[1]	Non-Equity Incentive Plan Compensation[2]	All Other Compensation[3]	Total
Thomas J. Schall, Ph.D.	2011	$ 465,750				$ 690	$ 466,440
President and Chief Executive Officer	2010	450,000		$ 1,106,281	$ 162,000	690	1,718,971
Susan M. Kanaya	2011	359,625		205,802		450	565,877
Senior Vice President, Finance and Chief Financial Officer	2010	347,464	$ 4,842[4]	283,994	78,179	450	714,929
Markus J. Cappel, Ph.D.	2011	342,554		226,843		690	570,087
Chief Business Officer	2010	330,970		255,079	74,468	450	660,967
Juan C. Jaen, Ph.D.	2011	362,250		230,232		690	593,172
Senior Vice President, Drug Discovery and Chief Scientific Officer	2010	350,000		418,248	78,750	690	847,688
Petrus J. Bekker, M.D.,Ph.D.	2011	356,891		182,907		690	540,488
Senior Vice President, Medical and Clinical Affairs	2010	344,822		194,356	77,585	690	617,453

(1) Amounts shown represent the aggregate grant date fair value of the option awards granted during 2010 and 2011 computed in accordance with FASB Topic ASC 718. These amounts do not correspond to the actual value that will be recognized by the named executive officer with respect to such awards. The assumptions used in the valuation of these awards are consistent with the valuation methodologies specified in the notes to our financial statements included elsewhere in this prospectus.

(2) Amounts shown represent performance bonuses for 2010, which were each paid in a cash lump sum in the first quarter of 2011. Our board of directors has not yet met to evaluate management s performance relative to corporate performance objectives and no bonuses have been paid to our named executive officers for 2011.

(3) Amounts shown represent term life insurance paid by the Company on behalf of the named executive officers.

(4) In order to avoid potential adverse tax consequences to Ms. Kanaya, in October 2007, our compensation committee approved an increase to the exercise price of options granted on February 9, 2006, from $0.88 per share to $2.00 per share, which represents the exercise price of options that were issued immediately following receipt of our December 31, 2005 appraisal. Our compensation committee further approved periodic cash bonuses to Ms. Kanaya, commencing in January 2008, in the amount of the increased exercise price of $1.12 per share, to be paid as the amended options vest and become exercisable.

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Grants of Plan-Based Awards

The following table sets forth summary information regarding grants of plan-based awards made to our named executive officers during our fiscal year ended December 31, 2011.

Name	Grant Date	Estimated Possible Payouts Under Non-Equity Incentive Plan Awards[1] Threshold	Target	Maximum[2]	Option Awards: Number of Securities Underlying Options[3]	Exercise or Base Price of Option Awards[4]	Grant Date Fair Value of Option Awards[5]
Thomas J. Schall, Ph.D.			$ 186,300				
Susan M. Kanaya	08/04/11		89,906		44,180	$ 6.90	$ 205,802
Markus J. Cappel, Ph.D.	08/04/11		85,639		48,697	6.90	226,843
Juan C. Jaen, Ph.D.	08/04/11		90,563		49,425	6.90	230,232
Petrus J. Bekker, M.D., Ph.D.	08/04/11		89,223		39,265	6.90	182,907

(1) Represents target awards granted under our annual bonus plan, as described above under the heading Bonuses.

(2) There is no maximum possible payout under our annual bonus plan.

(3) The options have a 10-year term and vest at the rate of 25% of the original number of shares on the first anniversary of the vesting commencement date and 1/48th of the original number of shares on each monthly anniversary thereafter, provided that the option holder continues to provide services to the company.

(4) Reflects the fair market value per share of our common stock on the grant date as determined by our board of directors based on an appraisal prepared by an independent valuation firm.

(5) Amounts shown represent the aggregate grant date fair value of the option awards computed in accordance with FASB Topic ASC 718. The assumptions used in the valuation of these awards are consistent with the valuation methodologies specified in the notes to our financial statements included elsewhere in this prospectus.

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Outstanding Equity Awards at December 31, 2011

The following table sets forth specified information concerning unexercised stock options for each of the named executive officers outstanding as of December 31, 2011.

	Option Awards[1]				
	Number of Securities Underlying Unexercised Options				
Name	Grant Date	Exercisable	Unexercisable	Option Exercise Price	Option Expiration Date
Thomas J. Schall, Ph.D.	05/13/04	100,000		$ 0.60	05/13/14
	05/05/05	125,000		0.60	05/05/15
	09/10/08	250,000		6.00	09/10/18
	07/29/09	310,000		6.00	07/29/19
	08/11/10	279,166		6.30	08/11/20
Susan M. Kanaya	02/09/06	207,500		0.88[2]	02/09/16
	09/10/08	75,000		6.00	09/10/18
	07/28/09	92,500		6.00	07/28/19
	08/10/10	71,614		6.30	08/10/20
	08/04/11	44,180		6.90	08/04/21
Markus J. Cappel, Ph.D.	05/13/04	62,500		0.60	05/13/14
	05/05/05	75,000		0.60	05/05/15
	02/06/07	50,000		4.30	02/06/17
	09/10/08	100,000		6.00	09/10/18
	07/28/09	77,500		6.00	07/28/19
	08/10/10	64,323		6.30	08/10/20
	08/04/11	48,697		6.90	08/04/21
Juan C. Jaen, Ph.D.	02/06/07	197,500		4.30	02/06/17
	09/10/08	37,500		6.00	09/10/18
	07/28/09	42,500		6.00	07/28/19
	08/10/10	105,469		6.30	08/10/20
	08/04/11	49,425		6.90	08/04/21
Petrus J. Bekker, M.D., Ph.D.	05/05/05	62,500		0.60	05/05/15
	09/10/08	50,000		6.00	09/10/18
	02/17/09	100,000		6.00	02/17/19
	07/28/09	12,500		6.00	07/28/19
	08/10/10	49,010		6.30	08/10/20
	08/04/11	39,265		6.90	08/04/21

(1) The options have a 10-year term and vest at the rate of 25% of the original number of shares on the first anniversary of the vesting commencement date and 1/48th of the original number of shares on each monthly anniversary thereafter, provided that the option holder continues to provide services to the company. All options are immediately exercisable for shares of restricted stock, to the extent such options are unvested on the date of exercise.

(2) In order to avoid potential adverse tax consequences to Ms. Kanaya, in October 2007, our compensation committee approved an increase to the exercise price of such options to $2.00 per share, which represents the exercise price of options that were issued immediately following receipt of our December 31, 2005 appraisal. Our compensation committee further approved periodic cash bonuses to Ms. Kanaya, commencing in January 2008, in the amount of the increased exercise price of $1.12 per share, to be paid as the amended options vest and become exercisable.

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Option Exercises and Stock Vested

The following table summarizes information regarding the stock options that were exercised during 2011 for each of the named executive officers.

	Option Awards	
Name	Number of Shares Acquired on Exercise	Value Realized on Exercise[1]
Thomas J. Schall, Ph.D.		
Susan M. Kanaya		
Markus J. Cappel, Ph.D.	125,000	$ 797,500
Juan C. Jaen, Ph.D.		
Petrus J. Bekker, M.D., Ph.D.		

(1) Represents the difference between the market value of the option shares on the exercise date and the option exercise price.

Pension Benefits and Nonqualified Deferred Compensation

We do not provide a pension plan for our employees and none of our named executive officers participated in a nonqualified deferred compensation plan during the fiscal year ended December 31, 2011.

Employment Agreements

We have entered into amended and restated employment agreements with each of our named executive officers. Each of the employment agreements has a three-year term, subject to automatic successive one-year renewals unless we provide written notice of our desire to terminate the agreement at least sixty days prior to the expiration of the then-current term. Pursuant to the employment agreements, each executive officer is eligible for a target performance bonus of up to 25% (40% with respect to Dr. Schall) of his or her base salary, based upon the achievement of financial and performance objectives established by the compensation committee. Any final bonus payment shall be determined by our board of directors or compensation committee.

The employment agreements provide for certain severance payments to our named executive officers. All cash severance payments are payable in a lump sum. If we terminate an executive officer s employment without cause or if the executive officer resigns for good reason (unless such termination occurs within 12 months following a change in control), we are obligated to pay such executive officer a lump sum severance payment equal to his or her base salary in effect at the time of termination for a specified number of months. The applicable severance period is 18 months for Drs. Schall, Cappel and Jaen and Ms. Kanaya, and six months for Dr. Bekker. Additionally, each of Drs. Schall, Cappel and Jaen and Ms. Kanaya will receive accelerated vesting and/or exercisability of 100% of his or her outstanding stock awards.

Under each of the employment agreements, if we terminate an executive officer s employment without cause or if the executive officer resigns for good reason, in each case within 12 months following a change in control, we are obligated to pay such executive officer a lump sum severance payment equal to the sum of: (1) 18 months of his or her base salary in effect at the time of termination, (2) one and one-half times the executive officer s target bonus, and (3) 18 months of health benefits continuation at our cost. Furthermore, all of the executive officer s outstanding stock awards will vest upon the date of termination. The foregoing change-in-control severance benefits shall only apply so long as the executive officer is working on a full-time basis. For a further description of the potential compensation payable to our named executive officers under their amended and restated employment agreements, please see Potential Benefits Upon Termination or Change in Control below.

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For purposes of the amended and restated employment agreements, cause means an executive officer (1) has committed an act of fraud, embezzlement or dishonesty in connection with the executive officer s employment, or has intentionally committed some other illegal act that has, or may be reasonably expected to have, a material adverse impact on the company or any successor or parent or subsidiary thereof; (2) has been convicted of, or entered a plea of guilty or no contest to, a felony, or to any crime involving moral turpitude, which causes or may reasonably be expected to cause substantial economic injury to or substantial injury to the reputation of the company or any successor or parent or subsidiary thereof; (3) has made any unauthorized use or disclosure of confidential information or trade secrets of the company or any successor or parent or subsidiary thereof that has, or may reasonably be expected to have, a material adverse impact on any such entity; (4) has materially breached a company policy, materially breached the provisions of the executive officer s employment agreement (if any), or has committed any other intentional misconduct that has, or may be reasonably expected to have, a material adverse impact on the company or any successor or parent or subsidiary thereof, or (5) has intentionally refused or intentionally failed to act in accordance with any lawful and proper direction or order of the board of directors or the appropriate individual to whom the executive officer reports; provided such direction is not materially inconsistent with the executive officer s customary duties and responsibilities.

For purposes of the amended and restated employment agreements, good reason means (1) a material diminution in the executive officer s authority, duties or responsibilities, (2) a material diminution in the executive officer s base compensation unless such a reduction is imposed across-the-board to senior management of the company, (3) a material change in the geographic location at which the executive officer must perform services to us or (4) any other action or inaction that constitutes a material breach by the company or any successor or affiliate of its obligations to the executive officer under the employment agreement. With respect to Drs. Schall, Cappel and Jaen and Ms. Kanaya, good reason also includes a material diminution in the authority, duties or responsibilities of the supervisor to whom the executive officer is required to report.

For purposes of the amended and restated employment agreements, change in control has the same meaning as such term is given under the terms of our 2012 Equity Incentive Award Plan, as described below.

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Potential Benefits Upon Termination or Change in Control

We have agreements with each of our named executive officers which provide for certain benefits in the event of a termination of employment under certain circumstances. The two right-hand columns describe the payments that would apply in two different potential scenarios: (1) a termination of the named executive officer s employment by the company without cause or his or her resignation for good reason; and (2) a termination of the named executive officer s employment by the company without cause or his or her resignation for good reason within 12 months following a change in control. The table assumes that the termination and change in control occurred on December 31, 2011. For purposes of estimating the value of amounts of equity compensation to be received in the event of a termination of employment, we have assumed a price per share of our common stock of $15.00, the midpoint of the range set forth on the front cover of this prospectus, which price the board of directors has determined exceeds the actual fair market value of our common stock on December 31, 2011. For a further description of potential benefits in the event of a termination of employment or change in control, please see Employment Agreements above.

Name and Position	Benefit Type	Payment in the Case of a Termination by the Company Without Cause or by Executive Officer for Good Reason	Payment in the Case of a Termination by the Company Without Cause or by Executive Officer for Good Reason Within 12 Months Following a Change in Control
Thomas J. Schall, Ph.D.	Cash Severance[1]	$ 698,625	$ 978,075
	Benefits Continuation		23,463
	Accelerated Vesting of Stock Options[2]	3,001,070	3,001,070
	Total Value:	3,699,695	4,002,608
Susan M. Kanaya	Cash Severance[1]	539,438	674,297
	Benefits Continuation		23,742
	Accelerated Vesting of Stock Options[2]	1,188,210	1,188,210
	Total Value:	1,727,648	1,886,249
Markus J. Cappel, Ph.D.	Cash Severance[1]	513,831	642,289
	Benefits Continuation		12,934
	Accelerated Vesting of Stock Options[2]	1,163,210	1,163,210
	Total Value:	1,677,041	1,818,433
Juan C. Jaen, Ph.D.	Cash Severance[1]	543,375	679,219
	Benefits Continuation		40,152
	Accelerated Vesting of Stock Options[2]	1,193,589	1,193,589
	Total Value:	1,736,964	1,912,960
Petrus J. Bekker, M.D., Ph.D.	Cash Severance[1]	178,446	669,171
	Benefits Continuation		34,931
	Accelerated Vesting of Stock Options[2]		947,344
	Total Value:	178,446	1,651,446

(1) Payable in a lump sum.

(2) Represents the value of those options that would vest as a result of the named executive officer s termination, which is equal to the number of shares that would vest multiplied by the difference between $15.00, the midpoint of the range set forth on the front cover of this prospectus, and the exercise price per share of such option.

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Equity Compensation Plans and Other Benefit Plans

2012 Equity Incentive Award Plan

We have adopted our 2012 Equity Incentive Award Plan, or the Equity Plan. The Equity Plan will become effective on the day prior to the public trading date of our common stock. The material terms of the Equity Plan are summarized below. The Equity Plan is filed as an exhibit to the registration statement of which this prospectus forms a part.

Authorized Shares. A total of 3,000,000 shares of our common stock will initially be reserved for issuance under the Equity Plan. In addition, the number of shares initially reserved under the Equity Plan will be increased by (1) the number of shares that as of the closing of this offering, have been reserved but not issued pursuant to any awards granted under our 2002 Plan and are not subject to any awards granted thereunder, and (2) the number of shares subject to stock options or similar awards granted under the 2002 Plan or the 1997 Plan that expire or otherwise terminate without having been exercised in full and unvested shares issued pursuant to awards granted under the 2002 Plan that are forfeited to or repurchased by us, with the maximum number of shares to be added to the Equity Plan pursuant to clauses (1) and (2) above equal to 6,000,000 shares. In addition, the number of shares available for issuance under the Equity Plan will be annually increased on the first day of each of our fiscal years during the term of the Equity Plan, beginning with the 2012 fiscal year, by an amount equal to the least of:

2,000,000 shares;

4% of the outstanding shares of our common stock as of the last day of our immediately preceding fiscal year; or

such other amount as our board of directors may determine.

The Equity Plan will also provide for an aggregate limit of 29,000,000 shares of common stock that may be issued under the Equity Plan over the course of its ten-year term.

Shares issued pursuant to awards under the Equity Plan that we repurchase or that are forfeited, as well as shares used to pay the exercise price of an award or to satisfy the tax withholding obligations related to an award, will become available for future grant under the Equity Plan. In addition, to the extent that an award is paid out in cash rather than shares, such cash payment will not reduce the number of shares available for issuance under the Equity Plan.

Plan Administration. The compensation committee of our board of directors will administer the Equity Plan (except with respect to any award granted to independent directors (as defined in the Equity Plan), which must be administered by our full board of directors). Following the completion of this offering, to administer the Equity Plan, our compensation committee must consist solely of at least two members of our board of directors, each of whom is a non-employee director for purposes of Rule 16b-3 under the Exchange Act and, with respect to awards that are intended to constitute performance-based compensation under Section 162(m) of the Code, an outside director for purposes of Section 162(m). Subject to the terms and conditions of the Equity Plan, our compensation committee has the authority to select the persons to whom awards are to be made, to determine the type or types of awards to be granted to each person, the number of awards to grant, the number of shares to be subject to such awards, and the terms and conditions of such awards, and to make all other determinations and decisions and to take all other actions necessary or advisable for the administration of the Equity Plan. Our compensation committee is also authorized to establish, adopt, amend or revise rules relating to administration of the Equity Plan. Our board of directors may at any time revest in itself the authority to administer the Equity Plan.

Eligibility. Options, stock appreciation rights, or SARs, restricted stock and other awards under the Equity Plan may be granted to individuals who are then our officers or employees or are the officers or employees of any of our subsidiaries. Such awards may also be granted to our non-employee directors and consultants but only employees may be granted incentive stock options, or ISOs. As of December 31, 2011, there were four

non-employee directors and approximately 64 employees who would have been eligible for awards under the Equity Plan had it been in effect on such date. At such time after the completion of this offering when we are subject to the requirements of Section 162(m) of the Code, the maximum number of shares that may be subject to awards granted under the Equity Plan to any individual in any calendar year cannot exceed 2,000,000 and the maximum amount that may be paid to a participant in cash during any calendar year with respect to one or more cash based awards under the Equity Plan is $5,000,000.

Awards. The Equity Plan provides that our compensation committee (or the board of directors, in the case of awards to non-employee directors) may grant or issue stock options, SARs, restricted stock, restricted stock units, dividend equivalents, stock payments and performance awards, or any combination thereof. Our compensation committee (or the board of directors, in the case of awards to non-employee directors) will consider each award grant subjectively, considering factors such as the individual performance of the recipient and the anticipated contribution of the recipient to the attainment of our long-term goals. Each award will be set forth in a separate agreement with the person receiving the award and will indicate the type, terms and conditions of the award.

Nonqualified stock options, or NQSOs, will provide for the right to purchase shares of our common stock at a specified price which may not be less than the fair market value of a share of common stock on the date of grant, and usually will become exercisable (at the discretion of our compensation committee or our board of directors, in the case of awards to non-employee directors) in one or more installments after the grant date, subject to the participant s continued employment or service with us and/or subject to the satisfaction of performance targets established by our compensation committee (or our board of directors, in the case of awards to non-employee directors). NQSOs may be granted for any term specified by our compensation committee (or our board of directors, in the case of awards to non-employee directors).

ISOs will be designed to comply with the provisions of the Code and will be subject to specified restrictions contained in the Code. Among such restrictions, ISOs must have an exercise price of not less than the fair market value of a share of common stock on the date of grant, may only be granted to employees, must expire within a specified period of time following the optionee s termination of employment, and must be exercised within the ten years after the date of grant. In the case of an ISO granted to an individual who owns (or is deemed to own) more than 10% of the total combined voting power of all classes of our capital stock, the Equity Plan provides that the exercise price must be at least 110% of the fair market value of a share of common stock on the date of grant and the ISO must expire upon the fifth anniversary of the date of its grant.

Restricted stock may be granted to participants and made subject to such restrictions as may be determined by our compensation committee (or our board of directors, in the case of awards to non-employee directors). Typically, restricted stock may be forfeited for no consideration if the conditions or restrictions are not met, and it may not be sold or otherwise transferred to third parties until restrictions are removed or expire. Recipients of restricted stock, unlike recipients of options, may have voting rights and may receive dividends, if any, prior to the time when the restrictions lapse.

Restricted stock units may be awarded to participants, typically without payment of consideration or for a nominal purchase price, but subject to vesting conditions including continued employment or on performance criteria established by our compensation committee (or our board of directors, in the case of awards to non-employee directors). Like restricted stock, restricted stock units may not be sold or otherwise transferred or hypothecated until vesting conditions are removed or expire. Unlike restricted stock, stock underlying restricted stock units will not be issued until the restricted stock units have vested, and recipients of restricted stock units generally will have no voting or dividend rights prior to the time when vesting conditions are satisfied.

SARs granted under the Equity Plan typically will provide for payments to the holder based upon increases in the price of our common stock over the exercise price of the SAR. Except as required by Section 162(m) of the Code with respect to SARs intended to qualify as performance-based

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compensation as described in Section 162(m) of the Code, there are no restrictions specified in the Equity Plan on the exercise of SARs or the amount of gain realizable therefrom. Our compensation committee (or the board of directors, in the case of awards to non-employee directors) may elect to pay SARs in cash or in common stock or in a combination of both.

Dividend equivalents represent the value of the dividends, if any, per share paid by us, calculated with reference to the number of shares covered by the stock options, SARs or other awards held by the participant.

Performance awards may be granted by our compensation committee on an individual or group basis. Generally, these awards will be based upon the attainment of specific performance goals that are established by our compensation committee and relate to one or more performance criteria on a specified date or dates determined by our compensation committee. Any such cash bonus paid to a covered employee within the meaning of Section 162(m) of the Code may be, but need not be, qualified performance-based compensation as described below and will be paid in cash.

Stock payments may be authorized by our compensation committee (or our board of directors, in the case of awards to non-employee directors) in the form of common stock or an option or other right to purchase common stock as part of a deferred compensation arrangement, made in lieu of all or any part of compensation, including bonuses, that would otherwise be payable to employees, consultants or members of our board of directors.

Transferability of Awards. Unless the administrator provides otherwise, our Equity Plan generally does not allow for the transfer of awards and only the recipient of an option or stock appreciation right may exercise such an award during his or her lifetime.

Qualified Performance-Based Compensation. The compensation committee may designate employees as covered employees whose compensation for a given fiscal year may be subject to the limit on deductible compensation imposed by Section 162(m) of the Code. The compensation committee may grant to such covered employees restricted stock, dividend equivalents, stock payments, restricted stock units, cash bonuses and other stock-based awards that are paid, vest or become exercisable upon the attainment of company performance criteria which are related to one or more of the following performance criteria as applicable to our performance or the performance of a division, business unit or an individual: operating or other costs and expenses, improvements in expense levels, cash flow (including, but not limited to, operating cash flow and free cash flow), return on assets, return on capital, stockholders equity, return on stockholders equity, total stockholder return, return on sales, gross or net profit or operating margin, working capital, net earnings (either before or after interest, taxes, depreciation and amortization), gross or net sales or revenue, net income (either before or after taxes), adjusted net income, operating earnings, earnings per share of stock, adjusted earnings per share of stock, price per share of stock, regulatory body approval for commercialization of a product capital raised in financing transactions or other financing milestones, market recognition (including but not limited to awards and analyst ratings), financial ratios, implementation or completion of critical projects, market share, economic value, comparisons with various stock market indices, and implementation, completion or attainment of objectively determinable objectives relating to research, development, regulatory, commercial or strategic milestones or development. These performance criteria may be measured in absolute terms or as compared to performance in an earlier period or as compared to any incremental increase or decrease or as compared to results of a peer group or to market performance indicators or indices.

The compensation committee may provide that one or more objectively determinable adjustments will be made to one or more of the performance goals established for any performance period. Such adjustments may include one or more of the following: items related to a change in accounting principle, items relating to financing activities, expenses for restructuring or productivity initiatives, other non-operating items, items related to acquisitions, items attributable to the business operations of any entity acquired by us during the performance period, items related to the disposal of a business of segment of a business, items related to discontinued operations that do not qualify as a segment of a business under applicable accounting standards, items attributable to any stock dividend, stock split, combination or exchange of shares occurring during the performance period, any other items of significant income or expense which are determined to be appropriate

adjustments, items relating to unusual or extraordinary corporate transactions, events or developments, items related to amortization of acquired intangible assets, items that are outside the scope of our core, on-going business activities, items related to acquired in-process research and development items relating to changes in tax laws, items relating to major licensing or partnership arrangements, items relating to asset impairment charges, items relating to gains and losses for litigation, arbitration or contractual settlements, or items relating to any other unusual or nonrecurring events or changes in applicable laws, accounting principles or business conditions.

Forfeiture, Recoupment and Clawback Provisions. Pursuant to its general authority to determine the terms and conditions applicable to awards under the Equity Plan, the compensation committee has the right to provide, in an award agreement or otherwise, that an award shall be subject to the provisions of any recoupment or clawback policies implemented by us, including, without limitation, any recoupment or clawback policies adopted to comply with the requirements of the Dodd-Frank Wall Street Reform and Consumer Protection Act and any rules or regulations promulgated thereunder.

Adjustments. If there is any stock dividend, stock split, combination or exchange of shares, merger, consolidation or other distribution (other than normal cash dividends) of our assets to stockholders, or any other change affecting the shares of our common stock or the share price of our common stock other than an equity restructuring (as defined in the Equity Plan), the plan administrator may make such equitable adjustments, if any, as the plan administrator in its discretion may deem appropriate to reflect such change with respect to (1) the aggregate number and type of shares that may be issued under the Equity Plan (including, but not limited to, adjustments of the number of shares available under the plan and the maximum number of shares which may be subject to one or more awards to a participant pursuant to the plan during any calendar year), (2) the number and kind of shares, or other securities or property, subject to outstanding awards, (3) the number and kind of shares, or other securities or property, for which automatic grants are to be subsequently made to new and continuing non-employee directors, (4) the terms and conditions of any outstanding awards (including, without limitation, any applicable performance targets or criteria with respect thereto), and (5) the grant or exercise price per share for any outstanding awards under the plan. If there is any equity restructuring, (1) the number and type of securities subject to each outstanding award and the grant or exercise price per share for each outstanding award, if applicable, will be proportionately adjusted, and (2) the plan administrator will make proportionate adjustments to reflect such equity restructuring with respect to the aggregate number and type of shares that may be issued under the Equity Plan (including, but not limited to, adjustments of the number of shares available under the plan and the maximum number of shares which may be subject to one or more awards to a participant pursuant to the plan during any calendar year). Adjustments in the event of an equity restructuring will not be discretionary. Any adjustment affecting an award intended as qualified performance-based compensation will be made consistent with the requirements of Section 162(m) of the Code. The plan administrator also has the authority under the Equity Plan to take certain other actions with respect to outstanding awards in the event of a corporate transaction, including provision for the cash-out, termination, assumption or substitution of such awards.

Corporate Transactions. In the event of a change in control where the acquirer does not assume awards granted under the Equity Plan, awards issued under the Equity Plan will be subject to accelerated vesting such that 100% of the awards will become vested and exercisable or payable, as applicable. Under the Equity Plan, a change in control is generally defined as:

a transaction or series of related transactions (other than an offering of our stock to the general public through a registration statement filed with the Securities and Exchange Commission, or SEC) whereby any person or entity or related group of persons or entities (other than us, our subsidiaries, an employee benefit plan maintained by us or any of our subsidiaries or a person or entity that, prior to such transaction, directly or indirectly controls, is controlled by, or is under common control with, us) directly or indirectly acquires beneficial ownership (within the meaning of Rule 13d-3 under the Exchange Act) of more than 50% of the total combined voting power of our securities outstanding immediately after such acquisition;

during any two-year period, individuals who, at the beginning of such period, constitute our board of directors together with any new director(s) whose election by our board of directors or nomination for election by our stockholders was approved by a vote of at least two-thirds of the directors then still in

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office who either were directors at the beginning of the two-year period or whose election or nomination for election was previously so approved, cease for any reason to constitute a majority of our board of directors;

our consummation (whether we are directly or indirectly involved through one or more intermediaries) of (x) a merger, consolidation, reorganization, or business combination or (y) the sale or other disposition of all or substantially all of our assets in any single transaction or series of transactions or (z) the acquisition of assets or stock of another entity, in each case other than a transaction:

which results in our voting securities outstanding immediately before the transaction continuing to represent (either by remaining outstanding or by being converted into our voting securities or the voting securities of the person that, as a result of the transaction, controls us, directly or indirectly, or owns, directly or indirectly, all or substantially all of our assets or otherwise succeeds to our business (we or such person being referred to as a successor entity)) directly or indirectly, at least a majority of the combined voting power of the successor entity s outstanding voting securities immediately after the transaction; and

after which no person or group beneficially owns voting securities representing 50% or more of the combined voting power of the successor entity; provided, however, that no person or group is treated as beneficially owning 50% or more of combined voting power of the successor entity solely as a result of the voting power held in us prior to the consummation of the transaction; or

our stockholders approve a liquidation or dissolution of the company.

Amendment, Termination. Our board of directors has the authority to amend, suspend or terminate the Equity Plan at any time. However, stockholder approval of any amendment to the Equity Plan will be obtained to the extent necessary to comply with any applicable law, regulation or stock exchange rule. Additionally, stockholder approval is required within 12 months of an increase in the maximum number of shares issuable under the Equity Plan or that may be issued to an individual in any calendar year. Except as necessary to comply with Section 409A of the Code, no amendment, suspension or termination of the Equity Plan will impair the rights or obligations of a holder under an award theretofore granted, unless such award expressly so provides or such holder consents. If not terminated earlier by our board of directors, the Equity Plan will terminate on the tenth anniversary of the date it becomes effective.

Repricing Permitted. Our compensation committee (or the board of directors, in the case of awards to non-employee directors) shall have the authority, without the approval of our stockholders, to authorize the amendment of any outstanding award to reduce its price per share and to provide that an award will be canceled and replaced with the grant of an award having a lesser price per share.

Securities Laws and Federal Income Taxes. The Equity Plan is designed to comply with various securities and federal tax laws as follows:

Securities Laws. The Equity Plan is intended to conform to all provisions of the Securities Act of 1933, as amended, and the Exchange Act and any and all regulations and rules promulgated by the SEC thereunder, including, without limitation, Rule 16b-3. The Equity Plan will be administered, and awards will be granted and may be exercised, only in such a manner as to conform to such laws, rules and regulations.

Federal Income Tax Consequences. The material federal income tax consequences of the Equity Plan under current federal income tax law are summarized in the following discussion, which deals with the general tax principles applicable to the Equity Plan. The following discussion is based upon laws, regulations, rulings and decisions now in effect, all of which are subject to change. Foreign, state and local tax laws, and employment, estate and gift tax considerations are not discussed due to the fact that they may vary depending on individual circumstances and from locality to locality.

Stock Options and Stock Appreciation Rights. An Equity Plan participant generally will not recognize taxable income and we generally will not be entitled to a tax deduction upon the grant of a stock option or stock appreciation right. The tax consequences of exercising a stock option and the subsequent disposition of the shares received upon exercise will depend upon whether the option qualifies as an

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ISO as defined in Section 422 of the Code. The Equity Plan permits the grant of options that are intended to qualify as ISOs as well as options that are not intended to so qualify; however, ISOs generally may be granted only to our employees and employees of our parent or subsidiary corporations, if any. Upon exercising an option that does not qualify as an ISO when the fair market value of our stock is higher than the exercise price of the option, an Equity Plan participant generally will recognize taxable income at ordinary income tax rates equal to the excess of the fair market value of the stock on the date of exercise over the purchase price, and we (or our subsidiaries, if any) generally will be entitled to a corresponding tax deduction for compensation expense, in the amount equal to the amount by which the fair market value of the shares purchased exceeds the purchase price for the shares. Upon a subsequent sale or other disposition of the option shares, the participant will recognize a short-term or long-term capital gain or loss in the amount of the difference between the sales price of the shares and the participant s tax basis in the shares.

Upon exercising an ISO, an Equity Plan participant generally will not recognize taxable income, and we will not be entitled to a tax deduction for compensation expense. However, upon exercise, the amount by which the fair market value of the shares purchased exceeds the purchase price will be an item of adjustment for alternative minimum tax purposes. The participant will recognize taxable income upon a sale or other taxable disposition of the option shares. For federal income tax purposes, dispositions are divided into two categories: qualifying and disqualifying. A qualifying disposition generally occurs if the sale or other disposition is made more than two years after the date the option was granted and more than one year after the date the shares are transferred upon exercise. If the sale or disposition occurs before these two periods are satisfied, then a disqualifying disposition generally will result.

Upon a qualifying disposition of ISO shares, the participant will recognize long-term capital gain in an amount equal to the excess of the amount realized upon the sale or other disposition of the shares over their purchase price. If there is a disqualifying disposition of the shares, then the excess of the fair market value of the shares on the exercise date (or, if less, the price at which the shares are sold) over their purchase price will be taxable as ordinary income to the participant. If there is a disqualifying disposition in the same year of exercise, it eliminates the item of adjustment for alternative minimum tax purposes. Any additional gain or loss recognized upon the disposition will be recognized as a capital gain or loss by the participant.

We will not be entitled to any tax deduction if the participant makes a qualifying disposition of ISO shares. If the participant makes a disqualifying disposition of the shares, we should be entitled to a tax deduction for compensation expense in the amount of the ordinary income recognized by the participant.

Upon exercising or settling a SAR, an Equity Plan participant will recognize taxable income at ordinary income tax rates, and we should be entitled to a corresponding tax deduction for compensation expense, in the amount paid or value of the shares issued upon exercise or settlement. Payments in shares will be valued at the fair market value of the shares at the time of the payment, and upon the subsequent disposition of the shares the participant will recognize a short-term or long-term capital gain or loss in the amount of the difference between the sales price of the shares and the participant s tax basis in the shares.

Restricted Stock and Restricted Stock Units. An Equity Plan participant generally will not recognize taxable income at ordinary income tax rates and we generally will not be entitled to a tax deduction upon the grant of restricted stock or restricted stock units. Upon the termination of restrictions on restricted stock or the payment of restricted stock units, the participant will recognize taxable income at ordinary income tax rates, and we should be entitled to a corresponding tax deduction for compensation expense, in the amount paid to the participant or the amount by which the then fair market value of the shares received by the participant exceeds the amount, if any, paid for them. Upon the subsequent disposition of any shares, the participant will recognize a short-term or long-term capital gain or loss in

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the amount of the difference between the sales price of the shares and the participant s tax basis in the shares. However, an Equity Plan participant granted restricted stock that is subject to forfeiture or repurchase through a vesting schedule such that it is subject to a risk of forfeiture (as defined in Section 83 of the Code) may make an election under Section 83(b) of the Code to recognize taxable income at ordinary income tax rates, at the time of the grant, in an amount equal to the fair market value of the shares of common stock on the date of grant, less the amount paid, if any, for such shares. We will be entitled to a corresponding tax deduction for compensation, in the amount recognized as taxable income by the participant. If a timely Section 83(b) election is made, the participant will not recognize any additional ordinary income on the termination of restrictions on restricted stock, and we will not be entitled to any additional tax deduction.

Dividend Equivalents, Stock Payment Awards and Cash-Based Awards. An Equity Plan participant will not recognize taxable income and we will not be entitled to a tax deduction upon the grant of dividend equivalents, stock payment awards or cash-based awards until cash or shares are paid or distributed to the participant. At that time, any cash payments or the fair market value of shares that the participant receives will be taxable to the participant at ordinary income tax rates and we should be entitled to a corresponding tax deduction for compensation expense. Payments in shares will be valued at the fair market value of the shares at the time of the payment, and upon the subsequent disposition of the shares, the participant will recognize a short-term or long-term capital gain or loss in the amount of the difference between the sales price of the shares and the participant s tax basis in the shares.

Section 409A of the Code. Certain types of awards under the Equity Plan may constitute, or provide for, a deferral of compensation under Section 409A. Unless certain requirements set forth in Section 409A are complied with, holders of such awards may be taxed earlier than would otherwise be the case (*e.g.*, at the time of vesting instead of the time of payment) and may be subject to an additional 20% federal income tax (and, potentially, certain interest penalties). To the extent applicable, the Equity Plan and awards granted under the Equity Plan will be structured and interpreted to comply with Section 409A and the Department of Treasury regulations and other interpretive guidance that may be issued pursuant to Section 409A.

Section 162(m) Limitation. In general, under Section 162(m) of the Code, income tax deductions of publicly held corporations may be limited to the extent total compensation (including base salary, annual bonus, stock option exercises and non-qualified benefits paid) for certain executive officers exceeds $1 million (less the amount of any excess parachute payments as defined in Section 280G of the Code) in any one year. However, under Section 162(m), the deduction limit does not apply to certain performance-based compensation if an independent compensation committee determines performance goals and if the material terms of the performance-based compensation are disclosed to and approved by our stockholders. In particular, stock options and SARs will satisfy the performance-based compensation exception if the awards are made by a qualifying compensation committee, the plan sets the maximum number of shares that can be granted to any person within a specified period and the compensation is based solely on an increase in the stock price after the grant date. Specifically, the option exercise price must be equal to or greater than the fair market value of the stock subject to the award on the grant date. Under a Section 162(m) transition rule for compensation plans of corporations which are privately held and which become publicly held in an initial public offering, certain awards under the Equity Plan will not be subject to Section 162(m) until a specified transition date, which is the earlier of (1) the material modification of the Equity Plan, (2) the issuance of all employer stock and other compensation that has been allocated under the Equity Plan, or (3) the first annual meeting of stockholders at which directors are to be elected that occurs after the close of the third calendar year following the calendar year in which the initial public offering occurs. After the transition date, rights or awards granted under the Equity Plan, other than options and SARs, will not qualify as performance-based compensation for purposes of Section 162(m) unless such rights or awards are granted or vest upon pre-established objective performance goals, the material terms of which are disclosed to and approved by our stockholders.

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We have attempted to structure the Equity Plan in such a manner that, after the transition date, the compensation attributable to stock options and SARs which meet the other requirements of Section 162(m) will not be subject to the $1 million limitation. We have not, however, requested a ruling from the Internal Revenue Service or an opinion of counsel regarding this issue.

Amended and Restated 2002 Equity Incentive Plan

Our Amended and Restated 2002 Equity Incentive Plan, or the 2002 Plan, was initially adopted by our board of directors in May 2002 and approved by our stockholders in September 2002. As amended to date, we have reserved a total of 4,650,000 shares of common stock for issuance under the 2002 Plan. As of December 31, 2011, options to purchase 753,614 shares of common stock had been exercised (net of repurchases), options to purchase 3,847,878 shares of common stock were outstanding and 48,508 shares of common stock remained available for grant. As of December 31, 2011, the outstanding options were exercisable at a weighted average exercise price of approximately $4.93 per share. The material terms of the 2002 Plan are summarized below. The 2002 Plan is filed as an exhibit to the registration statement of which this prospectus is a part.

No Further Grants. After the effective date of the Equity Plan, no additional awards will be granted under the 2002 Plan, and all awards granted under the 2002 Plan that are repurchased, forfeited, expire or are cancelled will become available for grant under the Equity Plan.

Administration. The compensation committee of our board of directors administers the 2002 Plan. Subject to the terms and conditions of the 2002 Plan, our compensation committee has the authority to select the persons to whom awards are to be made, to determine the number of shares to be subject thereto and the terms and conditions thereof, and to make all other determinations and to take all other actions necessary or advisable for the administration of the 2002 Plan. Our compensation committee is also authorized to adopt, amend or rescind rules relating to administration of the 2002 Plan. Our board of directors may at any time abolish the compensation committee and revest in itself the authority to administer the 2002 Plan. The full board of directors administers the 2002 Plan with respect to awards to non-employee directors.

Eligibility. Options and restricted stock under the 2002 Plan may be granted to individuals who are then our officers or employees or are the officers or employees of any of our subsidiaries. Such awards may also be granted to our non-employee directors or consultants, but only employees may be granted ISOs.

Awards. The 2002 Plan provides that our compensation committee may grant or issue stock options and restricted stock or any combination thereof. Each award will be set forth in a separate agreement with the person receiving the award and will indicate the type, terms and conditions of the award.

NQSOs provide for the right to purchase shares of our common stock at a specified price and usually will become exercisable at the discretion of our compensation committee or, in the case of awards to non-employee directors, the board of directors, in one or more installments after the grant date, subject to the participant s continued employment or service with us and/or subject to the satisfaction of performance targets established by our compensation committee or, in the case of awards to non-employee directors, the board of directors.

ISOs are designed to comply with the provisions of the Internal Revenue Code and will be subject to specified restrictions contained in the Internal Revenue Code and as further described above in connection with our 2002 Plan.

Restricted stock may be granted to participants and made subject to such restrictions as may be determined by our compensation committee or, in the case of awards to non-employee directors, the board of directors. Typically, restricted stock and stock bonus awards may be forfeited for no consideration or repurchased by us at the original purchase price if the conditions or restrictions are not met and they may not be sold, or otherwise transferred to third parties until restrictions are removed or expire. Purchasers of restricted stock, unlike recipients of options, will have voting rights and will receive dividends, if any, prior to the time when the restrictions lapse.

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Corporate Transactions. In the event of an acquisition where the acquirer does not assume awards granted under the 2002 Plan or substitute similar awards, awards issued under the 2002 Plan and held by participants in the plan whose status as a service provider has not terminated prior to such acquisition will be subject to accelerated vesting such that 100% of such award will become vested and exercisable or payable, as applicable, and all other outstanding options under the 2002 Plan will be terminated if not exercised prior to the closing of the acquisition. Under the 2002 Plan, an acquisition is generally defined as:

- the sale, transfer or other disposition of all or substantially all of our assets or our complete liquidation or dissolution; or
- a merger or consolidation in which securities possessing more than 50% of the total combined voting power of our outstanding securities are transferred to a person or persons different from the persons holding those securities immediately prior to such transaction.

Amendment and Termination of the 2002 Plan. Our board of directors may terminate, amend or modify the 2002 Plan. However, stockholder approval of any amendment to the 2002 Plan will be obtained in order to increase the maximum number of shares issuable under the 2002 Plan, to extend the term of the 2002 Plan and, to the extent necessary and desirable to comply with any applicable law, regulation, or stock exchange rule. If not terminated earlier by the board of directors, the 2002 Plan will terminate on the tenth anniversary of the date of its initial adoption by our board of directors.

Securities Laws and Federal Income Taxes. The 2002 Plan is also designed to comply with various securities and federal tax laws as described above in connection with the Equity Plan.

Amended and Restated 1997 Stock Option/Stock Issuance Plan

Our amended and restated 1997 Stock Option/Stock Issuance Plan, or the 1997 Plan, was initially adopted by our board of directors in October 1997 and approved by our stockholders in October 1997. We have reserved a total of 1,350,000 shares of common stock for issuance under the 1997 Plan. As of December 31, 2011, options to purchase 1,181,483 shares of common stock had been exercised, options to purchase 168,515 shares of common stock were outstanding and two shares of common stock remained available for grant. As of December 31, 2011, the outstanding options were exercisable at a weighted average exercise price of approximately $3.81 per share. The material terms of the 1997 Plan are summarized below. The 1997 Plan is filed as an exhibit to the registration statement of which this prospectus is a part.

No Further Grants. No additional awards will be granted under the 1997 Plan after the expiration of the 1997 Plan in May 2012, and all awards granted under the 1997 Plan that are repurchased, forfeited, expire or are cancelled will become available for grant under the Equity Plan.

Administration. The compensation committee of our board of directors administers the 1997 Plan. Subject to the terms and conditions of the 1997 Plan, our compensation committee has the authority to select the persons to whom awards are to be made, to determine the number of shares to be subject thereto and the terms and conditions thereof, and to make all other determinations and to take all other actions necessary or advisable for the administration of the 1997 Plan. Our compensation committee is also authorized to adopt, amend or rescind rules relating to administration of the 1997 Plan. Our board of directors may at any time abolish the compensation committee and revest in itself the authority to administer the 1997 Plan. The full board of directors administers the 1997 Plan with respect to awards to non-employee directors.

Eligibility. Options under the 1997 Plan may be granted to individuals who are then our officers or employees or are the officers or employees of any of our parent or subsidiary corporations. Options may also be granted to our non-employee directors or consultants, but only employees may be granted ISOs.

Awards. The 1997 Plan provides that our compensation committee may grant or issue stock options and restricted stock or any combination thereof. Each award will be set forth in a separate agreement with the person receiving the award and will indicate the type, terms and conditions of the award.

NQSOs provide for the right to purchase shares of our common stock at a specified price, which for purposes of the 1997 Plan may be no less than 85 percent of the fair market value on the date of grant,

and usually will become exercisable, at the discretion of our compensation committee or the board of directors, in the case of awards to non-employee directors, in one or more installments after the grant date, subject to the participant s continued employment or service with us and/or subject to the satisfaction of performance targets established by our compensation committee, or the board of directors, in the case of awards to non-employee directors. Under the 1997 Plan, in the case of an NQSO granted to an individual who owns or is deemed to own at least 10% of the total combined voting power of all classes of our capital stock, the 1997 Plan provides that the exercise price must be at least 110% of the fair market value of a share of common stock on the date of grant. NQSOs may be granted for a maximum ten-year term.

ISOs are designed to comply with the provisions of the Internal Revenue Code and are subject to specified restrictions contained in the Internal Revenue Code and as further described above in connection with our Equity Plan.

Restricted stock may be granted to participants and made subject to such restrictions as may be determined by our compensation committee, or the board of directors, in the case of awards to non-employee directors. Typically, restricted stock and stock bonus awards may be forfeited for no consideration or repurchased by us at the original purchase price if the conditions or restrictions are not met and they may not be sold, or otherwise transferred to third parties until restrictions are removed or expire. Purchasers of restricted stock, unlike recipients of options, will have voting rights and will receive dividends, if any, prior to the time when the restrictions lapse.

Corporate Transactions. In the event of a corporate transaction where the acquirer does not assume or replace awards granted under the 1997 Plan, awards issued under the 1997 Plan will be subject to accelerated vesting such that 100% of such award will become vested and exercisable or payable, as applicable. Under the 1997 Plan, a corporate transaction is generally defined as:

the sale, transfer or other disposition of all or substantially all of our assets or a complete liquidation or dissolution; or

a merger or consolidation in which securities possessing more than 50% of the total combined voting power of our outstanding securities are transferred to a person or persons different from the persons holding those securities immediately prior to such transaction.

Amendment and Termination of the 1997 Plan. Our board of directors may terminate, amend or modify the 1997 Plan. However, stockholder approval of any amendment to the 1997 Plan will be obtained to the extent necessary and desirable to comply with any applicable law, regulation, or stock exchange rule. If not terminated earlier by the board of directors, the 1997 Plan will terminate in May 2012.

Securities Laws and Federal Income Taxes. The 1997 Plan is also designed to comply with various securities and federal tax laws as described above in connection with the Equity Plan.

2012 Employee Stock Purchase Plan

We have adopted our 2012 Employee Stock Purchase Plan, or the ESPP. The ESPP will become effective on the business day prior to the day on which the Registration Statement, of which this prospectus is a part, becomes effective. Our executive officers and all of our other employees will be allowed to participate in our ESPP, subject to the eligibility requirements described below. The material terms of the ESPP are summarized below. The ESPP is filed as an exhibit to the registration statement of which this prospectus forms a part.

A total of 300,000 shares of our common stock will initially be reserved for issuance under our ESPP. In addition, the number of shares available for issuance under the ESPP will be annually increased on the first day of each fiscal year during the term of the ESPP, beginning with the 2012 fiscal year, by an amount equal to the least of:

300,000 shares;

1% of the outstanding shares of our common stock as of the last day of our immediately preceding fiscal year; or

such other amount as may be determined by our board of directors.

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The ESPP will also provide for an aggregate limit of 3,300,000 shares of common stock that may be issued under the ESPP during the term of the ESPP.

Our board of directors or its committee has full and exclusive authority to interpret the terms of the ESPP and determine eligibility. Our compensation committee will be the initial administrator of the ESPP.

Our employees are eligible to participate in the ESPP if they are customarily employed by us or any participating subsidiary for at least 20 hours per week and more than five months in any calendar year. However, an employee may not be granted rights to purchase stock under our ESPP if such employee, immediately after the grant, would own (directly or through attribution) stock possessing 5% or more of the total combined voting power or value of all classes of our common or other class of stock.

Our ESPP is intended to qualify under Code Section 423 and stock will be offered under the ESPP during offering periods. The length of the offering periods under the ESPP will be determined by our compensation committee and may be up to 27 months long. Employee payroll deductions will be used to purchase shares on each purchase date during an offering period. The purchase dates will be determined by the compensation committee for each offering period, but will generally be the last day in each offering period. Offering periods under the ESPP will commence when determined by our compensation committee. The compensation committee may, in its discretion, modify the terms of future offering periods.

Our ESPP permits participants to purchase common stock through payroll deductions of up to 15% of their eligible compensation, which includes a participant s gross base compensation for services to the company, excluding overtime payments, sales commissions, incentive compensation, bonuses, expense reimbursements, fringe benefits and other special payments. A participant may purchase a maximum of 20,000 shares of common stock during each offering period. In addition, no employee will be permitted to accrue the right to purchase stock under the ESPP at a rate in excess of $25,000 worth of shares during any calendar year during which such a purchase right is outstanding (based on the fair market value per share of our common stock as of the first day of the offering period).

On the first trading day of each offering period, each participant automatically is granted an option to purchase shares of our common stock. The option expires at the end of the offering period or upon termination of employment, whichever is earlier, but is exercised at the end of each purchase period to the extent of the payroll deductions accumulated during such purchase period. The purchase price of the shares will be 85% of the lower of the fair market value of our common stock on the first trading day of the offering period or on the applicable purchase date. Participants may end their participation at any time during an offering period, and will be paid their accrued payroll deductions that have not yet been used to purchase shares of common stock. Participation ends automatically upon termination of employment with us.

A participant may not transfer rights granted under the ESPP other than by will, the laws of descent and distribution or as otherwise provided under the ESPP.

In the event of certain significant transactions or a change in control (as defined in the ESPP), the compensation committee may provide for (1) either the replacement or termination of outstanding rights in exchange for cash, (2) the assumption or substitution of outstanding rights by the successor or survivor corporation or parent or subsidiary thereof, if any, (3) the adjustment in the number and type of shares of stock subject to outstanding rights, (4) the use of participants accumulated payroll deductions to purchase stock on a new purchase date prior to the next purchase date and termination of any rights under ongoing offering periods or (5) the termination of all outstanding rights. Under the ESPP, a change in control has the same definition as given to such term in the Equity Plan.

The compensation committee may amend, suspend or terminate the ESPP. However, stockholder approval of any amendment to the ESPP will be obtained for any amendment which changes the aggregate number or type of shares that may be sold pursuant to rights under the ESPP, changes the corporations or classes of corporations whose employees are eligible to participate in the ESPP or changes the ESPP in any manner that would cause the ESPP to no longer be an employee stock purchase plan within the meaning of Section 423(b) of the Code. The ESPP will terminate no later than the tenth anniversary of the ESPP s initial adoption by our board of directors.

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Securities Laws. The ESPP has been designed to comply with various securities laws in the same manner as described above in the description of the Equity Plan.

Federal Income Taxes. The material federal income tax consequences of the ESPP under current federal income tax law are summarized in the following discussion, which deals with the general tax principles applicable to the ESPP. The following discussion is based upon laws, regulations, rulings and decisions now in effect, all of which are subject to change. Foreign, state and local tax laws, and employment, estate and gift tax considerations are not discussed due to the fact that they may vary depending on individual circumstances and from locality to locality.

The ESPP, and the right of participants to make purchases thereunder, is intended to qualify under the provisions of Section 423 of the Code. Under the applicable Code provisions, no income will be taxable to a participant until the sale or other disposition of the shares purchased under the ESPP. This means that an eligible employee will not recognize taxable income on the date the employee is granted an option under the ESPP (*i.e.*, the first day of the offering period). In addition, the employee will not recognize taxable income upon the purchase of shares. Upon such sale or disposition, the participant will generally be subject to tax in an amount that depends upon the length of time such shares are held by the participant prior to disposing of them. If the shares are sold or disposed of more than two years from the first day of the offering period during which the shares were purchased and more than one year from the date of purchase, or if the participant dies while holding the shares, the participant (or his or her estate) will recognize ordinary income measured as the lesser of (1) the excess of the fair market value of the shares at the time of such sale or disposition over the purchase price or (2) an amount equal to 15% of the fair market value of the shares as of the first day of the offering period. Any additional gain will be treated as long-term capital gain. If the shares are held for the holding periods described above but are sold for a price that is less than the purchase price, there is no ordinary income and the participating employee has a long-term capital loss for the difference between the sale price and the purchase price.

If the shares are sold or otherwise disposed of before the expiration of the holding periods described above, the participant will recognize ordinary income generally measured as the excess of the fair market value of the shares on the date the shares are purchased over the purchase price and we will be entitled to a tax deduction for compensation expense in the amount of ordinary income recognized by the employee. Any additional gain or loss on such sale or disposition will be long-term or short-term capital gain or loss, depending on how long the shares were held following the date they were purchased by the participant prior to disposing of them. If the shares are sold or otherwise disposed of before the expiration of the holding periods described above but are sold for a price that is less than the purchase price, the participant will recognize ordinary income equal to the excess of the fair market value of the shares on the date of purchase over the purchase price (and we will be entitled to a corresponding deduction), but the participant generally will be able to report a capital loss equal to the difference between the sales price of the shares and the fair market value of the shares on the date of purchase.

Incentive Plan

We have adopted a cash incentive plan, which we refer to as the Incentive Plan, under which we will provide cash incentives to our executive officers and other key employees following the consummation of this offering. The purpose of the Incentive Plan is to enable the Company and its subsidiaries to attract, retain, motivate and reward the best qualified executive officers and key employees by providing them with the opportunity to earn competitive compensation directly linked to the Company s performance. The Incentive Plan will become effective on the business day prior to the public trading date of our common stock. The material terms of the Incentive Plan are summarized below. The Incentive Plan is filed as an exhibit to the registration statement of which this prospectus forms a part.

Administration. The Incentive Plan will be administered by our compensation committee, which may delegate its authority under the Incentive Plan to any of its duly constituted subcommittees.

Performance Criteria. The compensation committee may establish the performance objective or objectives that must be satisfied in order for a participant to receive an award under the Incentive Plan or may make discretionary payments from the plan. Performance objectives under the Incentive Plan may be based upon

the relative or comparative achievement of performance criteria, as determined by the compensation committee for the applicable performance period, which performance criteria may include: operating or other costs and expenses, improvements in expense levels, cash flow (including, but not limited to, operating cash flow and free cash flow), return on assets, return on capital, stockholders equity, return on stockholders equity, return on sales, total stockholder return, gross or net profit or operating margin, working capital, net earnings (either before or after interest, taxes, depreciation and amortization), gross or net sales or revenue, net income (either before or after taxes), adjusted net income, operating earnings, earnings per share of stock, adjusted earnings per share of stock, price per share of stock, capital raised in financing transactions or other financing milestones, regulatory body approval for commercialization of a product, market share, economic value, market recognition (including but not limited to awards and analyst ratings), financial ratios, implementation or completion of critical projects, comparisons with various stock market indices, and implementation, completion or attainment of objectively determinable objectives relating to research, development, regulatory, commercial or strategic milestones or development. These performance criteria may be measured in absolute terms or as compared to any incremental increase or decrease or as compared to results of a peer group or to market performance indicators or indices. The compensation committee may exclude any or all extraordinary, unusual or non-recurring items and the cumulative effects of accounting changes from performance objectives for a performance period and may also adjust performance objectives in its discretion.

Payment. Payment of awards will be made as soon as practicable after the compensation committee certifies that one or more of the applicable performance criteria have been attained or determines the payable amount of an award. The compensation committee will determine whether an award will be paid in cash, stock (including restricted stock or restricted stock units) or other awards under the Equity Plan, or in a combination of cash, stock and other awards, and may impose whatever additional conditions on such shares or other awards as it deems appropriate, including conditioning the vesting of such shares or other awards on the performance of additional service.

Maximum Award; Discretion. The maximum award amount payable to a participant in cash per fiscal year under the Incentive Plan is $5,000,000. The compensation committee may, in its discretion, increase, reduce or eliminate awards otherwise payable under the Incentive Plan for any reason.

Termination of Employment. Unless otherwise determined by the compensation committee in its discretion, any participant whose employment terminates will forfeit all rights to any and all unpaid awards under the Incentive Plan.

Forfeiture; Disgorgement. If we are required to prepare an accounting restatement due to material noncompliance with any financial reporting requirement under the securities laws, and a participant knowingly or grossly negligently engaged in the misconduct or knowingly or grossly negligently failed to prevent the misconduct, or if the participant is one of the individuals subject to automatic forfeiture under section 304 of the Sarbanes-Oxley Act of 2002, then the participant must forfeit and disgorge any awards received during the twelve months following the filing of the financial document embodying such financial reporting requirement and any other awards earned based on the materially non-complying financial reporting. In addition, any award paid to a current or former executive officer during the three-year period preceding the date on which the restatement is required, based on erroneous data, must be forfeited and disgorged to us to the extent the award is in excess of what would have been paid to the officer under the restated data. Participants must also forfeit and disgorge any awards to the extent required by applicable law or regulations in effect on or after the effective date of the Incentive Plan.

Director Compensation

We compensate certain non-employee members of the board of directors for their service. Directors who are also employees do not receive cash or equity compensation for service on the board of directors in addition to compensation payable for their service as our employees. The non-employee members of our board of directors are also reimbursed for travel, lodging and other reasonable expenses incurred in attending board of directors or committee meetings.

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We do not currently provide any cash compensation to our non-employee directors. Our board of directors has historically granted to certain of our non-employee directors options to purchase shares of our common stock under our 1997 Plan and our 2002 Plan. None of our non-employee directors received an equity award during 2011.

Following the completion of this offering, we intend to provide cash compensation in the form of an annual retainer of $30,000 for each non-employee director. We will also pay an additional annual retainer of $15,000 to the chairman of our audit committee, $10,000 to other non-employee directors who serve on our audit committee, $7,500 to the chair of our compensation committee, $5,000 to other non-employee directors who serve on our compensation committee, $5,000 to the chair of our nominating and corporate governance committee and $2,500 to other non-employee directors who serve on our nominating and corporate governance committee. We have reimbursed and will continue to reimburse our non-employee directors for their reasonable expenses incurred in attending meetings of our board of directors and committees of the board of directors.

Following the completion of this offering, any non-employee director who is first elected to the board of directors will be granted an option to purchase 25,000 shares of our common stock on the date of his or her initial election to the board of directors. Such options will have an exercise price per share equal to the fair market value of our common stock on the date of grant. In addition, on the date of each annual meeting of our stockholders following this offering, each non-employee director will be eligible to receive an option to purchase 12,500 shares of common stock.

The initial options granted to non-employee directors described above will vest and become exercisable in 36 equal monthly installments over the three-year period following the date of grant, subject to the director s continuing service on our board of directors on those dates. The annual options granted to non-employee directors described above will vest and/or become exercisable in 12 equal monthly installments over the first year following the date of grant, subject to the director s continuing service on our board of directors (and, with respect to grants to a chairman of the board of directors or board committee, service as chairman of the board of directors or a committee) on those dates. The term of each option granted to a non-employee director shall be ten years. The terms of these options are described in more detail under Equity Compensation Plans and Other Benefit Plans 2012 Equity Incentive Award Plan.

During 2011, we did not pay any director for service as a member of the board of directors or any board committee, either in cash compensation or with equity awards. Outstanding vested and nonvested options held by members of our board of directors at December 31, 2011, were:

	Shares Underlying Options Outstanding At December 31, 2011	
	Vested	Unvested
Geoffrey M. Parker	25,000	25,000
Thomas J. Schall, Ph.D.	724,704	339,462
Roger C. Lucas, Ph.D.		
Edward E. Penhoet, Ph.D.		
Samuel P. Wertheimer, Ph.D.[1]		
Rishi Gupta[2]		

(1) Dr. Wertheimer resigned from the board of directors on May 11, 2011.
(2) Mr. Gupta was appointed to the board of directors on May 11, 2011.

Risk Assessment of Compensation Program

In September and October 2011, management assessed our compensation program for the purpose of reviewing and considering any risks presented by our compensation policies and practices that are reasonably likely to have a material adverse effect on us. As part of that assessment, management reviewed the primary elements of our compensation program, including base salary, short-term incentive compensation and long-term incentive compensation. Management s risk assessment included a review of the overall design of each primary

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element of our compensation program, and an analysis of the various design features, controls and approval rights in place with respect to compensation paid to management and other employees that mitigate potential risks to us that could arise from our compensation program. Following the assessment, management determined that our compensation policies and practices did not create risks that were reasonably likely to have a material adverse effect on us and reported the results of the assessment to our compensation committee.

COMPENSATION COMMITTEE INTERLOCKS AND INSIDER PARTICIPATION

None of the members of our compensation committee has at any time been one of our officers or employees. None of our executive officers currently serves, or in the past year has served, as a member of the board of directors or compensation committee of any entity that has one or more executive officers on our board of directors or compensation committee.

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CERTAIN RELATIONSHIPS AND RELATED PARTY TRANSACTIONS

The following is a description of transactions since January 1, 2008 to which we have been a party, in which the amount involved exceeds $120,000, and in which any of our directors, executive officers or holders of more than 5% of our capital stock, or an affiliate or immediate family member thereof, had or will have a direct or indirect material interest. We believe the terms obtained or consideration that we paid or received, as applicable, in connection with the transactions described below were comparable to terms available or the amounts that would be paid or received, as applicable, from unaffiliated third parties.

Sales and Purchases of Securities

In August 2008, Glaxo Group Limited, one of our principal stockholders, purchased 6,793,478 shares of our Series E Preferred Stock at a price of $7.36 per share for an aggregate purchase price of $50.0 million.

In January 2009, we repurchased 183,333 shares of our common stock from Dr. Thomas J. Schall, our President and Chief Executive Officer, for a total purchase price of $1.1 million.

Relationships with GSK

In August 2006, we entered into a collaboration agreement with Glaxo Group Limited, or GSK, an affiliate of GlaxoSmithKline. See Business Strategic Alliance with GSK. Pursuant to the terms of this agreement we received research funding, upfront payments and milestone payments aggregating $23.6 million, $49.7, million and $34.9 million, in the years ended December 31, 2008, 2009 and 2010, respectively. In addition, GSK has agreed to purchase $7.0 million of our common stock in a private placement concurrent with this offering at a price per share equal to the initial public offering price. The sale of such shares of common stock will not be registered.

Relationships with Techne

In September 2011, we entered into a convertible note loan agreement with Techne, one of our principal stockholders, pursuant to which we issued a convertible note to Techne with a principal amount of $10.0 million, bearing interest at a rate of 5.0% per annum and maturing in September 2021. Upon completion of this offering, all outstanding principal and interest under this note will automatically convert into shares of our common stock at a conversion price equal to the initial public offering price of our common stock. Upon the conversion of this note in connection with this offering, we will issue Techne warrants with a ten-year term to purchase 150,000 shares of our common stock at an exercise price per share equal to 200% of the initial public offering price of our common stock. In addition, Techne has also agreed, pursuant to the terms of the convertible note loan agreement, to purchase $5.0 million of our common stock in a private placement concurrent with this offering at a price per share equal to the initial public offering price. The sale of such shares of common stock will not be registered.

During the years ended December 31, 2010, 2009 and 2008, we paid Techne Corporation, $0.1 million, $0.1 million and $0.2 million, respectively, for research materials. During the nine months ended September 30, 2011, we paid Techne Corporation approximately $43,000 for research materials.

Director and Executive Officer Compensation

Please see Executive and Director Compensation Director Compensation for a discussion of options granted to our non-employee directors. Please see Executive and Director Compensation, Management Outstanding Equity Awards at Fiscal Year End, and Management Summary Compensation for additional information regarding compensation of executive officers.

Employment Agreements

We have entered into employment agreements with our executive officers. For more information regarding these agreements, see Executive and Director Compensation Employment Agreements.

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Indemnification Agreements and Directors and Officers Liability Insurance

We intend to enter into indemnification agreements with each of our executive officers and directors.

Registration Rights Agreements

We and the holders of our Series A-1, Series A-2, Series A-3, Series B, Series C, Series D and Series E Preferred Stock, and Dr. Schall, as well as holders of certain warrants, have entered into agreements pursuant to which these stockholders will have, among other things, registration rights under the Securities Act with respect to their common shares following this offering. Prior to the completion of this offering, all outstanding shares of our preferred stock will be converted into common stock. GSK and Techne will also be entitled to registration rights with respect to shares of our common stock purchased by GSK and Techne in the concurrent private placements. See Description of Capital Stock Registration Rights for a further description of the terms of these agreements.

Procedures for Related Party Transactions

Our board of directors reviews and approves transactions with directors, officers and holders of 5% or more of our voting securities and their affiliates, each, a related party. Prior to this offering, the material facts as to the related party s relationship or interest in the transaction were disclosed to our board of directors and the transaction was not considered approved by our board of directors unless a majority of the directors who were not interested in the transaction approved the transaction. Our current policy with respect to approval of related party transactions is not in writing.

Following this offering, any request for us to enter into a related party transaction with an officer, director, principal stockholder or any of their immediate family members or affiliates, in which the