CATALYST PHARMACEUTICAL PARTNERS, INC.
Form 10-K
March 30, 2012
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UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-K

[Mark One]

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

For the Fiscal Year Ended December 31, 2011

OR

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

Commission File No. 001-33057

CATALYST PHARMACEUTICAL

PARTNERS, INC.

(Exact name of registrant as specified in its charter)

Delaware	**76-0837053**
(State of jurisdiction of	**(IRS Employer**

incorporation or organization)	**Identification No.)**

355 Alhambra Circle, Suite 1500

Coral Gables, Florida	**33134**
(Address of principal executive offices)	**(Zip Code)**

Registrant s telephone number, including area code: (305) 529-2522

Securities Registered Pursuant to Section 12(b) of the Act.

Common Stock, par value $0.001 per share	Nasdaq Capital Market
(Title of each class)	**(Name of exchange on which registered)**

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

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

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

Indicate by check mark whether the registrant: (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such report(s), and (2) has been subject to such filing requirements for the past 90 days. Yes x No ¨

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate web site, if any, every Interactive Data File required to be submitted and posted pursuant to rule 405 of Regulation S-T ((§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes x No ¨

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K (§229.405 of this chapter) is not contained herein, and will not be contained, to the best of registrant s knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. ¨

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, 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 (Check one):

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

As of June 30, 2011, the last business day of the Registrant s most recently completed second quarter, the aggregate market value of all voting, and non-voting common equity held by non-affiliates was $30,212,440.

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

Indicate the number of shares outstanding of each of the issuer s classes of common stock, as of the latest practicable date: 24,741,520 shares of common stock, $0.001 par value per share, were outstanding as of March 23, 2012.

Part III incorporates certain information by reference from the registrant s definitive proxy statement for the 2012 annual meeting of stockholders. The proxy statement with respect to the 2012 annual meeting of stockholders will be filed no later than 120 days after the close of the registrant s fiscal year ended December 31, 2011.

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PART I

You are urged to read this Annual Report on Form 10-K (Form 10-K) in its entirety. This Form 10-K contains forward-looking statements that involve risks and uncertainties. Our actual results may differ significantly from the projected results discussed in these forward-looking statements. Factors that may cause such a difference include, but are not limited to, those discussed below and in Item 1A, Risk Factors. We, our, ours, us, Catalyst, or the Company, when used herein, refers to Catalyst Pharmaceutical Partners, Inc., a Delaware corporation.

Forward-Looking Statements

Some of the statements in this Annual Report on Form 10-K are forward-looking statements, as that term is defined in the Private Securities Litigation Reform Act of 1995. Forward-looking statements do not relate strictly to historical or current matters. Rather, forward-looking statements are predictive in nature and may depend upon or refer to future events, activities or conditions. Although we believe that these statements are based upon reasonable assumptions, we cannot provide any assurances regarding future results. We undertake no obligation to revise or update any forward-looking statements, or to make any other forward-looking statements, whether as a result of new information, future events or otherwise. Because forward-looking statements relate to matters that have not yet occurred, these statements are inherently subject to risks and uncertainties. Many factors could cause our actual activities or results to differ materially from the activities and results anticipated in forward-looking statements. Factors that might cause such differences include, but are not limited to, those discussed in the section entitled Item 1A Risk Factors and Item 7 Management s Discussion and Analysis of Financial Condition and Results of Operations Caution Concerning Forward-Looking Statements.

Item 1. Business

Catalyst Pharmaceutical Partners, Inc. is a development-stage specialty pharmaceutical company focused on the development and commercialization of prescription drugs targeting diseases and disorders of the central nervous system with a focus on the treatment of addiction and epilepsy. We have two products in development. We are currently evaluating our lead drug candidate, CPP-109 (our formulation of vigabatrin, a GABA aminotransferase inhibitor) for the treatment of cocaine addiction. CPP-109 and CPP-115 have both been granted Fast Track status by the FDA for the treatment of cocaine addiction, which indicates that the FDA has recognized that CPP-109 and CPP-115 are intended for the treatment of a serious or life-threatening condition for which there is no effective treatment and which demonstrates the potential to address unmet medical needs. We also hope to evaluate CPP-109 for the treatment of other addictions and other selected central nervous system indications. Further, we are in the early stages of developing CPP-115, another GABA aminotransferase inhibitor that, based on our pre-clinical studies to date, we believe is more potent than vigabatrin but may have reduced side effects (e.g., visual field defects, or VFDs) from those associated with vigabatrin. We are planning to develop CPP-115 for several indications, including drug addiction, epilepsy (initially infantile spasms) and other selected central nervous disease indications. We believe that we control all current intellectual property for drugs that have a mechanism of action related to inhibition of GABA aminotransferase.

The successful development of CPP-109, CPP-115, or any other product we may acquire, develop or license in the future, is highly uncertain. We cannot reasonably estimate or know the nature, timing, or estimated expenses of the efforts necessary to complete the development of, or the period in which material net cash inflows are expected to commence, or if any net cash inflows will actually commence, due to the numerous risks and uncertainties associated with developing such products, including the uncertainty of:

- the results of our pre-clinical studies and clinical studies and trials, and the number of clinical trials (and the scope of such trials) that will be required for us to seek and obtain approval of New Drug Applications (NDAs) for CPP-109 and CPP-115; and

- the expense of filing, prosecuting, defending and enforcing any patent claims and other intellectual property rights.

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We are currently involved in the following product development activities: (i) the FDA has accepted our Investigational New Drug Application (IND) for CPP-115 (ii) we have commenced an initial Phase I(a) clinical study evaluating the safety of CPP-115 in healthy volunteers; and (iii) we are jointly conducting with the National Institute on Drug Abuse (NIDA) and the Veterans Administration (VA) a U.S. Phase II(b) clinical trial of CPP-109 and, based on current information, we expect to obtain top line results from this trial early in the first quarter of 2013.

Based on an analysis of our current financial condition and forecasts of available cash, we believe that we have sufficient resources to: (i) complete the above-described Phase I(a) clinical study of CPP-115 and the Phase II(b) clinical trial of CPP-109 and (ii) support our operations through the first quarter of 2013. However, there can be no assurance that we will actually have sufficient funds for these purposes. We will require additional funding to complete any other pre-clinical studies and trials that may be required to submit NDAs for and commercialize CPP-109 and CPP-115 and to support our operations beyond the first quarter of 2013. There can be no assurance that we will obtain additional funding or ever be able to commercialize either of our product candidates. See Item 7. Management s Discussion and Analysis of Financial Condition and Results of Operations Liquidity and Capital Resources below.

Our Drug Candidates

The following table summarizes key information regarding our drug candidates:

Drug Candidate	Indications	Current Status
CPP-109	Addiction	Conducting a Phase II(b) clinical trial in conjunction with NIDA and the VA for cocaine addiction.
CPP-115	Addiction, Epilepsy	Conducting a Phase I(a) human safety study

Mechanism of Action

We believe that our drug candidates, CPP-109 and CPP-115, will be effective treatments for addiction and that our drug candidate, CPP-115, will be an effective treatment for both addiction and epilepsy because they increase endogenous GABA levels in the brain through the inhibition of GABA-aminotransferase (GABA-AT). GABA-AT is responsible for the eventual breakdown of GABA and helps to balance its inhibitory effects.

GABA, the most abundant inhibitory neurotransmitter in the brain, inhibits over-excitation of neurons. When GABA binds to a GABA receptor, it raises the action potential threshold of that neuron and inhibits the post-synaptic neuron from firing and triggering the release of neurotransmitters that send a signal to subsequent neurons. This is the mechanism explaining the efficacy of CPP-109 vigabatrin as a treatment for complex partial seizures. In the case of addiction, increased GABA reduces the perception of pleasure and reward by dampening levels of dopamine release brought about by all drugs of abuse, but most notably by stimulants like cocaine and methamphetamine. Addictive drugs have been shown to block or overwhelm mechanisms involved in the removal of dopamine from synaptic clefts in the mesolimbic pathways of the brain, resulting in highly elevated levels of dopamine available to stimulate receptors and a dramatically heightened sense of pleasure or reward. GABA also helps induce relaxation and sleep, and contributes to functions such as motor control and vision.

CPP-109 and CPP-115 are GABA analogs that are readily absorbed and promptly available to the central nervous system, producing effects that last for many hours after a single dose. Due to the fact that these drugs are not receptor active , their administration does not appear to affect the baseline levels of dopamine, nor those variations in dopamine levels caused by normal stimuli. We believe that the similarities between CPP-115 and the well characterized drug, CPP-109, will simplify the development of CPP-115 because potential development risks can be predicted and managed.

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History and Side Effect Profile of Vigabatrin

Vigabatrin has been marketed for decades in over 30 countries by Sanofi-Aventis and its predecessors under the brand names Sabril®, Sabrilex® and Sabrilan® (hereinafter referred to as Sabril®) as an adjunct (add-on) treatment for adult epilepsy and as a primary treatment for the management of infantile spasms. The composition of matter patents for Sabril® in the U.S. expired more than ten years ago. On August 21, 2009, the FDA approved two NDAs for Sabril® for the treatment of infantile spasms and as an adjunctive therapy for adult patients with refractory complex partial seizures who have failed treatments with several other anti-epileptic drugs. The NDAs are for different formulations of Sabril® and both NDAs are held by Lundbeck Inc. (Lundbeck). Due to the risks of visual field damage associated with vigabatrin, Sabril® was approved under an FDA-mandated Risk Evaluation and Mitigation Strategy (REMS) program and is only available through a special restricted distribution program approved by the FDA.

In chronic use for the treatment of epilepsy, vigabatrin has been generally well tolerated with lower than average neurological side effects compared to other approved epilepsy therapies. The most common side effects reported have been drowsiness and fatigue. However, one clearly established adverse side effect is the development, of peripheral visual field defects, or VFDs. VFDs occur in approximately 33% of patients when cumulative dosage levels of vigabatrin approach approximately 1,500 grams. These VFDs are manifest as a constriction of the peripheral field of vision (i.e. tunnel vision).

Based on available information as described above and our clinical trial experience to date, we believe that VFDs occur at cumulative doses far higher than the total dosage amount we anticipate will be used for addiction treatment. To date, we believe that no subjects treated in the trial conducted in Mexico, or in our previously completed U.S. Phase II(a) cocaine trial or our methamphetamine human proof-of-concept study, have shown any evidence of VFDs.

CPP-115 is structurally similar to vigabatrin. Due to these similarities, we believe that these two drugs will share a number of biochemical features related to absorption, metabolism, and elimination, and our pre-clinical studies of CPP-115 to date support our expectations. However, based upon our pre-clinical studies of CPP-115 to date, we expect that there will be a significant reduction, and possibly elimination, of VFDs from the use of CPP-115 compared to vigabatrin. However, there can be no assurance that this will ultimately prove to be the case.

CPP-109 (Vigabatrin) To Treat Addiction

In 2002, we obtained from Brookhaven National Laboratory (Brookhaven) an exclusive license for several patent and patent applications to develop vigabatrin as a treatment for cocaine and other addictions. We have been granted Fast Track status for CPP-109 from the FDA for cocaine addiction. Under the Federal Food, Drug, and Cosmetic Act, or FDCA, the FDA is directed to facilitate the development and expedite review of drugs and biologics intended to treat serious or life-threatening conditions and that demonstrate the potential to address unmet medical needs. Fast Track designation emphasizes communication between us and the FDA and affords us benefits that may help to expedite the approval process. For example, Fast Track designation affords us the potential to submit an NDA for CPP-109 on a rolling, or modular, basis, allowing the FDA to review sections of the NDA in advance of receiving our full submission. The designation also means that we may have increased communications with the FDA regarding the design of our clinical studies, which we hope will expedite the development and review of our application for the approval of CPP-109 for cocaine addiction and provide greater certainty overall in the regulatory pathway. However, there can be no assurance that our receipt of Fast Track status will assist us in the regulatory process for CPP-109.

CPP-115 for the Treatment of Addiction and Epilepsy

In August 2009, we licensed the exclusive worldwide rights to commercialize certain composition of matter patents relating to a new class of novel GABA aminotransferase inhibitors and derivatives of vigabatrin. We intend to develop these compounds for a broad range of central nervous system illnesses that could benefit from the inhibition of GABA aminotransferase. CPP-115 is our lead compound from this group of composition of matter patents.

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The development efforts of CPP-115 were led by Dr. Richard B. Silverman, the John Evans Professor of Chemistry at Northwestern University (Northwestern). Dr. Silverman, who holds 44 patents, is the inventor of pregabalin, also known as Lyrica®, which is marketed by Pfizer. His goal in inventing the compound that became CPP-115 was to mimic the mechanism of action of vigabatrin, while making it both more potent and specific.

CPP-115 works by the same mechanism of action as CPP-109; the inhibition of GABA aminotransferase, which leads to increased brain GABA levels that reduce epileptogenesis or dampen the addiction reinforcing dopamine surge. We believe that CPP-115 and vigabatrin are the only two GABA aminotransferase inhibitors, either under development or marketed at this time, and that our patent estates for CPP-109 and CPP-115 are the only existing, currently in force, intellectual property rights for drugs with this primary mode of action.

Based on testing to date, CPP-115 has been shown to be at least 200 times more potent than CPP-109, our version of vigabatrin, in both in-vitro and animal model studies. The increased potency could enable the development of dosage forms potentially administrable by other routes of administration compared with the marketed oral, immediate release formulations of vigabatrin, Sabril®. Further, based on pre-clinical testing completed to date, CPP-115 has a superior specificity to GABA aminotransferase and we believe, will have a better side effect profile (e.g. less visual field defects) compared with Sabril®.

CPP-115 has been granted Fast Track status by the FDA for the treatment of cocaine addiction and orphan drug designation for the treatment of infantile spasms. CPP-115 has also been granted orphan medicinal product designation in the EU for the treatment of West Syndrome (a form of infantile spasms).

Our Strategy

Our strategy is to become a leading specialty pharmaceutical company focused on the in-licensing and development of proprietary drug candidates for the treatment of selected diseases of the central nervous system. Our near-term strategy is to focus on the regulatory approval of CPP-109 for the treatment of cocaine addiction and to initially demonstrate the safety and efficacy of CPP-115 for the treatment of addiction and epilepsy. Our long-term strategy is to gain approvals for additional indications for CPP-109, including methamphetamine addiction, and to initially gain approval for CPP-115 to treat addiction and epilepsy. Specifically, we intend to:

Focus on CPP-109 for cocaine addiction. A treatment for cocaine addiction addresses a significant unmet medical need, and we believe that our receipt of Fast Track status from the FDA for CPP-109 for cocaine addiction may facilitate the regulatory approval process. Enrollment for our U.S. Phase II(b) clinical trial evaluating CPP-109 for the treatment of cocaine addiction we are conducting with NIDA and the VA began in the first quarter of 2011. This trial is currently ongoing and we expect to receive top-line results from this trial early in the first quarter of 2013. Assuming success, we expect that this trial will serve as one of the adequate and well-controlled trials required to support approval of an NDA.

Develop additional indications for CPP-109. The mechanism of action of CPP-109 and pre-clinical data indicate it to be suitable as a potential treatment for addictions to methamphetamine, nicotine, prescription pain medications, alcohol and marijuana, as well as for obsessive-compulsive disorders including binge eating patterns and compulsive gambling. We hope to develop CPP-109 for one or more of these additional indications, subject to the availability of funding.

Continue clinical and pre-clinical work on CPP-115. During the fourth quarter of 2011, we completed our IND-enabling studies, filed an IND, and began a Phase I(a) human clinical trial for CPP-115 to evaluate its safety. We expect to receive final results from this Phase I(a) human clinical trial during the second quarter of 2012. Subject to the availability of funding, we hope to begin further human clinical trials for CPP-115 during the latter part of 2012 or early 2013.

Identify and initiate strategic partnering discussions for specific indications in the U.S. and Europe. We believe that there may be several potential pharmaceutical partners interested in jointly developing and marketing CPP-109 and CPP-115 in the U.S. and/or Europe. We have held preliminary discussions with several parties regarding potential transactions, but no agreements have been entered into to date.

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Our Potential Markets

Drug Addiction

Historically, individuals suffering from addiction have been treated primarily through behavioral modification and therapy. These treatments have shown a high rate of relapse. We believe that a pharmacological treatment for cocaine addiction and/or other stimulant addictions, including methamphetamine, would complement and significantly improve the effectiveness of counseling programs.

Despite the significant public health implications, there are very few therapies approved for the treatment of addiction, either in the United States or in the rest of the world. Further, there are no therapies currently approved for stimulant addiction to substances, such as cocaine and methamphetamine. We believe that currently approved drugs for addiction treatment, as well as compounds under development (other than CPP-109 and CPP-115), are subject to the following limitations:

- no single compound has broad applicability for treatment of multiple addictions;
- many of these compounds are receptor active , which means they have drug-like effects themselves and have the potential for abuse or addiction;
- increasing dosages over time may be required due to development of tolerance; and
- they are often ineffective at eliminating drug cravings or responding to increasing levels of drug use.

We believe that CPP-109 and CPP-115 do not suffer from these limitations and therefore, if approved, that both will have the potential to become widely prescribed, safe and effective treatments for cocaine, methamphetamine and other addictions.

Addictive drugs are used recreationally because of the transient, pleasurable effect they have on the user. Recent scientific evidence has established that drug abuse can interfere with the brain s normal balance of neurotransmitter release and reuptake, resulting in addiction. If this balance is not restored, addicted individuals, even after significant periods of abstinence, may be incapable of suppressing cravings or quitting through willpower alone, even with the assistance of professional counseling.

Cocaine binds to the dopamine reuptake transporter protein of the pre-synaptic neurons preventing the reuptake and eventual breakdown of dopamine, resulting in enhanced and prolonged stimulation of dopamine on post-synaptic receptors, causing a feeling of prolonged euphoria for the user.

Addiction to cocaine is caused by a neurological process called desensitization. Because the brain senses an unnaturally high level of dopamine, it responds by reducing the amount of dopamine released and the number of dopamine receptors created. Consequently, when the cocaine wears off, the user has a lower amount of dopamine and fewer functioning dopamine receptors, which results in a depressed mood. This desensitization process creates a lowering of mood each time the user takes more of the drug, causing the user to seek additional cocaine to restore normal feelings, and requiring the user to take an increasing amount of cocaine to achieve the same feeling of euphoria as before.

Addiction is a worldwide health problem that affects millions of people and has wide-ranging negative social consequences. According to NIDA, there are no pharmacologic treatments for cocaine addiction currently approved for marketing by the FDA. We believe that other therapies being developed for the treatment of cocaine addiction, but not yet approved for marketing, suffer from the significant limitations discussed earlier which have not been exhibited to date by CPP-109 or CPP-115.

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A June 2011 report of the National Center on Addiction and Substance Abuse at Columbia University titled Adolescent Substance Abuse : America s #1 Public Health Problem found that in annual federal, state and local government spending as a result of substance abuse and addiction was at least $467.7 billion almost $1,500 for every man, woman, and child in America. A 2009 report from the same group found that for every dollar federal and state governments spent on substance abuse and addiction in 2005, 95.7 cents went to shoveling up the wreckage and only 1.9 cents to prevention and treatment and 0.4 cents to research.

In 2010, an estimated 22.6 million people in the United States aged 12 or over were current users of illicit drugs (defined as usage in the past month), according to the National Survey on Drug Use and Health, published by SAMHSA, which we refer to as the SAMHSA survey. This represents 8.9% of the total population aged 12 or older. This rate was higher than the rate in 2009 (8.7%), 2008 (8.0%), 2007 (8.0%), 2005 (8.1%) and 2004 (7.9%).

According to the most recent SAMHSA survey, an estimated 1.5 million people, or 0.6% of the population aged 12 or over, had used cocaine in the month preceding the survey. Additionally, in 2010, approximately 637,000 people aged 12 or over had used cocaine for the first time within the preceding 12 months, an average of approximately 1,700 new users per day. In addition, approximately 699,000 patients received treatment for cocaine abuse in 2010.

According to the same survey, the number and percentage of past-month nonmedical users of stimulants decreased slightly from 1.3 million (0.5%) in 2009 to 1.1 million (0.4%) in 2010, based on a decrease in methamphetamine users, from 502,000 (0.2%) to 353,000 (0.1%). These numbers are similar to those seen in 2008 and represent the resumption of a trend that had seen methamphetamine use fall from 2006 to 2008, but increase in 2009.

In addition, approximately 5.1 million people in 2010, or 2.0% of the population aged 12 or over took prescription pain relievers for non-medical purposes in the month preceding the survey. This remained substantially unchanged from 2009, when 5.2 million people, or 2.1% of the population aged 12 or over, took prescription pain relievers for non-medical purposes in the month preceding the survey. Further, approximately 16.9 million people aged 12 or over in the United States were classified as heavy drinkers in 2010. Additionally, there are approximately 17.4 million persons aged 12 or over who used marijuana in the month preceding the survey and approximately 1.0 million people sought treatment in 2010. Finally, obsessive-compulsive disorders such as compulsive gambling have been shown to have similar dopamine-related mechanisms of action to drug addiction and affect millions of persons in the United States and around the world.

Addiction is not only a U.S. health problem. In 2007, according to the United Nations Office on Drugs and Crime, there were between 4.3 million and 4.6 million users of cocaine and between 2.4 million and 3.1 million users of amphetamine-type stimulants between the ages of 15 and 64 across Europe who had used these drugs within the past year. We believe that the direct and indirect costs of cocaine and methamphetamine use are indicative of a significant global public health problem, representing a significant unmet medical need for which no adequate pharmaceutical therapies exist.

Epilepsy

Epilepsy is a brain disorder in which clusters of nerve cells, or neurons, in the brain sometimes signal abnormally. In epilepsy, the normal pattern of neuronal activity becomes disturbed, causing strange sensations, emotions, and behavior or sometimes convulsions, muscle spasms, and loss of consciousness. Epilepsy is a disorder with many possible causes. Anything that disturbs the normal pattern of neuron activity from illness to brain damage to abnormal brain development can lead to seizures. Epilepsy may develop because of an abnormality in brain wiring, an imbalance of nerve signaling chemicals called neurotransmitters, imbalance of sensitivity to neurotransmitters, or some combination of these factors.

We intend to focus our development efforts for CPP-115 on its use as a treatment for infantile spasms and adult complex partial seizures. Although vigabatrin (CPP-109) is one of the drugs in our development pipeline, we have no plans to develop CPP-109 for the treatment of epilepsy.

An infantile spasm (IS) is a specific type of seizure seen in an epilepsy syndrome of infancy and childhood. The onset of infantile spasms is usually in the first year of life, typically between 4-8 months. The seizures primarily consist of a sudden bending forward of

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the body with stiffening of the arms and legs; some children arch their backs as they extend their arms and legs. Spasms tend to occur upon awakening or after feeding, and often occur in clusters of up to 100 spasms at a time. Infants may have dozens of clusters and several hundred spasms per day. Infantile spasms usually stop by age five, but may be replaced by other seizure types.

In complex partial seizures, consciousness is altered. Patients may exhibit automatisms (automatic repetitive behavior) such as walking in a circle, sitting and standing, or smacking their lips together. Often accompanying these symptoms are the presence of unusual thoughts, such as the feeling of déjà vu, uncontrollable laughing, fear, visual hallucinations, and experiencing unusual unpleasant odors. These symptoms are thought to be caused by abnormal discharges in the temporal lobe.

According to the Epilepsy Foundation, there are about 3 million epilepsy patients in the United States, with approximately 200,000 new cases diagnosed in the U.S. each year. Worldwide, 50 million people are estimated to have epilepsy. The incidence of epilepsy appears to depend somewhat on the age of the individual. The risk of epilepsy from birth through age 20 is approximately 1%. Within this group, incidence is highest during the first year of life and increases somewhat at the onset of puberty. From age 20 to 55 it decreases again, but increases after age 55.

Anti-epileptic drugs work through a variety of mechanisms, including inhibition of sodium ion channels and the enhancement of GABA mechanisms. Although the different types of epilepsy vary greatly, in general, available medications can only control seizures in about two-thirds of patients. CPP-115, like vigabatrin (CPP-109), is a GABA-AT inhibitor, and we are developing it initially for infantile spasms and complex partial seizures. Based on the historic use of vigabatrin in treating epilepsy, we believe that CPP-115 may ultimately work best as an adjunct therapy to existing drugs.

Vigabatrin is used in over 30 countries for the treatment of infantile spasms and for the treatment of adult complex partial seizures in patients who have failed several treatments. On August 21, 2009, Sabril® was approved for these indications in the United States.

Our Clinical Trials

CPP-109

In 2007, we initiated a randomized, double-blind, placebo-controlled U.S. Phase II(a) clinical trial evaluating the use of CPP-109 in treating subjects addicted to cocaine. The trial enrolled 186 cocaine addicted patients at 11 addiction treatment research centers and clinical research centers throughout the United States. Patients were treated for a period of 12 weeks, with an additional 12 weeks of follow-up. On May 29, 2009, we announced that the top-line data from this trial showed that CPP-109 did not demonstrate statistical significance in the primary endpoint that a significantly larger proportion of CPP-109 treated subjects than placebo-treated subjects were cocaine free during the last two weeks of the treatment period (weeks 11 and 12).

On September 30, 2009, we announced additional results from our U.S. Phase II(a) cocaine clinical trial. Based on post hoc analyses for vigabatrin levels in urine samples collected during the trial, we have concluded that less than 40% of the trial subjects were medication compliant. As a result, we now believe that the trial was inadequately powered to properly test the efficacy of CPP-109 for the treatment of patients with cocaine addiction. On the basis of a comprehensive review of the trial data, however, we concluded that: (i) CPP-109 was safe and well tolerated; and (ii) while there were no statistically significant differences between active and placebo groups for the protocol-specified primary and secondary efficacy endpoints, cocaine use as measured by benzoylecgonine (the major metabolite of cocaine) levels in urine collected from subjects were consistently lower in the CPP-109 treatment group during the 12 week treatment period, generally indicating a reduction of cocaine use; and (iii) in those subjects who were compliant with study medication, the differences between CPP-109 and placebo were amplified, which suggests that CPP-109 may facilitate abstinence, reduce overall cocaine use as measured by urine benzoylecgonine levels (an objective measure of daily cocaine usage), and reduce cocaine usage days (an objective measure of dependence severity).

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Consistent with previously published addiction trials conducted by other parties, the protocol of our cocaine trial assessed subjects medication compliance based on self-reporting and on counting the unused medication returned by subjects. The subjects self-reported a compliance level of greater than 85%, which was inconsistent with our urine data. This low medication compliance effectively reduced the power of the study, because not all subjects in the treatment group were actually treated. However, analyses of subject responses, corrected for poor medication compliance, makes the response ratios observed in our trial more consistent with the results reported by Dr. Jonathan Brodie et al. in a double-blind, placebo-controlled, 103-patient Phase II trial evaluating vigabatrin for the treatment of cocaine addiction that was completed in Mexico in 2007 (the results of which trial were published in *The American Journal of Psychiatry* in November 2009). See Clinical and Pre-Clinical Studies of Our Product Candidates Undertaken by Others below.

During June 2008, we initiated a randomized, double-blind, placebo-controlled U.S. Phase II clinical trial evaluating the use of CPP-109 in treating patients with methamphetamine addiction. We had planned to enroll 180 methamphetamine addicted patients at 15 addiction treatment clinical centers in the United States. However, in March 2009, in order to conserve cash, we converted our methamphetamine trial into a proof-of-concept study evaluating the results obtained from the 57 patients who had already been randomized into the trial. The patients we enrolled were treated for a period of 12 weeks and we evaluated data related to endpoints based on abstinence, reductions in methamphetamine use and craving for evidence of potential efficacy.

On September 30, 2009, we announced the top-line results of our proof-of-concept study. The results showed that there was a 2.5 times higher rate of abstinence in the last two weeks of the study in the vigabatrin group versus the placebo group. While we consider this to be an encouraging trend, the results were not statistically significant due to the small sample size. We also believe that medication compliance, similar to our previously discussed cocaine trial, was below expectations.

Based on the results from our Phase II cocaine trial and our methamphetamine proof-of-concept study, we expect that the data from those studies will be treated as supportive of any NDA application that we file.

On April 13, 2010, we signed a Clinical Trial Agreement (CTA) with NIDA to jointly conduct a U.S. Phase II(b) clinical trial evaluating CPP-109 for the treatment of cocaine addiction (the Phase II(b) Trial). As part of the CTA, NIDA, under their agreement with the Veterans Administration Cooperative Studies Program, agreed to provide substantial resources towards the completion of the Phase II(b) Trial. This approximately 200 subject double-blind, placebo-controlled trial is being conducted at twelve leading addiction research facilities across the United States. The Phase II(b) Trial, which is being overseen by us, NIDA and the VA, was initiated in November 2010 and began enrolling patients during the first quarter of 2011. Based on currently available information, we expect to have top-line results from this trial early in the first quarter of 2013. The Phase II(b) Trial is designed to confirm the safety and efficacy of CPP-109 for the treatment of cocaine addiction and if successful, we believe it will qualify as one of the adequate and well controlled trials required to support approval of an NDA for CPP-109.

Pursuant to the CTA, we have provided the study drug (and matching placebo) to the VA Clinical Pharmacy to be packaged suitably for use in the Phase II(b) Trial. In conjunction with NIDA and the VA, we have developed the Phase II(b) Trial protocol and informed consent and have submitted such documents to the FDA for review. We are also responsible for, among other duties, funding patient recruitment activities and advertising for the Phase II(b) Trial, establishing and funding a contract with a vendor capable of decrypting and converting the visual field data obtained from study subjects into a format analyzable by the VA statisticians who will interpret the study data. We have also agreed to fund the treatment costs for up to 25 study subjects. Further, pursuant to the CTA, NIDA has provided input on the protocol and informed consent and, under their agreement with the VA, is funding qualified study sites and investigators. NIDA has also presently contracted to treat more than 200 study subjects. Finally, NIDA, through its agreement with the VA, is providing clinical monitoring of all sites, pursuant to the CTA.

The CTA terminates on April 13, 2015 or upon the completion of the Phase II(b) Trial, whichever comes first, except that the CTA may be extended for two further periods of two years each by agreement of the parties if it is necessary to complete the Phase II(b) Trial. Either party may terminate the CTA upon 60 days notice without cause, or upon 30 days written notice for cause. Both NIDA and we have continuing rights under the CTA if the CTA is terminated. Among other obligations, this includes an obligation of each party to continue their respective obligations under the CTA until all study subjects enrolled in the trial at the time of such termination have completed the trial and continuing duties of confidentiality.

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The protocol for the Phase II(b) Trial has been designed to attempt to mitigate compliance issues that were observed in our previous U.S. Phase II(a) cocaine clinical trial and our methamphetamine proof-of-concept study. In the Phase II(b) Trial, subjects are being observed taking their medication on the days that they are at the trial sites for tests and therapy. Urine samples collected from subjects are also being monitored to determine whether trial subjects are taking their medication (CPP-109 or placebo). Further, the subjects are also undergoing therapy once per week and will receive substantially lower compensation for participation than in our previous trials. Finally, the trial is being conducted at 12 addiction treatment oriented centers, and the patient recruitment firm that is working with us on this trial has been directed to target trial subjects more likely to be genuinely interested in seeking treatment to overcome their addiction to cocaine. Although there can be no assurance, we believe that with these modifications we should avoid the medication compliance issues observed in our prior clinical studies.

Generally, the process of seeking approval of an NDA requires multiple clinical trials, including two pivotal U.S. Phase III clinical trials. In our case, because CPP-109 is intended to treat a serious condition for which there is no approved therapy, there is a possibility that if the data from the Phase II(b) Trial is sufficiently compelling, that the FDA will allow us to file an NDA for CPP-109 on the basis of this trial, when combined with the data from the previous clinical trials and studies of vigabatrin to treat addiction. However, it is more likely that the FDA will require at least one Phase III trial supported by the safety and efficacy data obtained from our Phase II(b) clinical trial before they will allow us to file an NDA for CPP-109, even if the data from our currently ongoing Phase II(b) clinical trial are compelling. Further, even if the FDA permits us to file an NDA based on the results of our current Phase II(b) trial, it is unlikely that we will be in a position to submit an NDA for CPP-109 before sometime in the second half of 2013. Finally, if the FDA requires more than one Phase III clinical trial, our NDA submission would be delayed even further. There can be no assurance that the data from our ongoing Phase II(b) Trial will be sufficiently compelling or that even if such data are sufficiently compelling, that the FDA will allow us to file an NDA for CPP-109 based on the results of that trial.

Lundbeck s exclusivity for Sabril® tablets to treat refractory complex partial seizures in adults will expire on August 20, 2014. We currently expect to submit a 505(b)(2) application in submitting an NDA for CPP-109. A 505(b)(2) application is one that relies, at least partially upon, data that a company does not own or have right of reference to, including published literature. A 505(b)(2) application can also rely upon the FDA s previous rulings on safety and efficacy for previously approved products. Data on manufacturing, bioequivalence, and bioavailability must be submitted, along with information to support any change relative to the previously approved product, as well as information on the patent status of the product previously approved and the product for which an NDA has been submitted. See Regulatory Matters The Hatch Waxman Act below. There can be no assurance whether, or to what extent, the FDA will accept 505(b)(2) data for any NDA that we may file for CPP-109.

CPP-115

On November 1, 2010 we announced key results for our initial series of safety and efficacy evaluations in a number of animal and in-vitro laboratory studies:

In visual safety studies of rats exposed for 90 days to either CPP-115, vigabatrin or placebo, CPP-115 caused substantially less retinal damage than vigabatrin at well above the expected therapeutic doses.

The oral pharmokinetic behavior of CPP-115 in rats supports further development as an orally delivered pharmacotherapy.

CPP-115 was found to not inhibit or induce metabolic enzymes and is not itself metabolized. As a result, drug-drug interactions or other metabolism-related side effects are unlikely. Additionally, non-metabolized drugs are advantageous for treating drug addicts, a population that often has impaired liver function.

With the exception of its biochemical target, GABA-aminotransferase, CPP-115 did not show any clinically significant binding to 111 of the most prominent receptors, proteins and transporters.

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Additionally, CPP-115 showed no binding to other GABA-related targets (GABA receptors and transporters). Therefore, CPP-115 is very specific and not likely to induce drug-drug interactions or unintended side effects.

CPP-115 did not show any interference with the hERG channel and is therefore not likely to induce heart arrhythmias.

CPP-115 did not show any abnormalities in an in-vitro battery of genotoxicity studies and thus is not likely to be carcinogenic.

CPP-115 did not show any inhibition of ALT and AST at doses far above the expected therapeutic dosage. This is in contrast to vigabatrin s known inhibition at therapeutic doses of these key liver transaminase enzymes.

CPP-115, like vigabatrin, was found to significantly reduce seizures in accepted animal models of epilepsy, as evaluated by the National Institutes of Health s Anticonvulsant Screening Program (ASP) at lower doses than vigabatrin.

CPP-115 was found to eliminate cocaine-conditioned place preference and significantly reduced cocaine-induced dopamine surge, key tests needed to demonstrate a drug s effectiveness as a potential treatment for stimulant addiction. These effects were observed at doses more than 200 times lower than that needed by vigabatrin to achieve the same effect.

During the third quarter of 2011, we completed our IND-enabling studies for CPP-115 and filed an IND for CPP-115 in November 2011. Following the acceptance of our IND, we began enrollment for our Phase I(a) human clinical trial evaluating the safety of CPP-115, and expect to have results from this trial during the second quarter of 2012. Subject to the results of this trial and the availability of funding, we hope to begin other human clinical trials for CPP-115 in late 2012 or early 2013.

Clinical and Pre-Clinical Studies of our Product Candidates Undertaken by Others

The primary focus of our product development efforts is on our clinical trials and pre-clinical studies. However, we have in the past supported and will continue in the future to support pre-clinical studies and clinical trials by academic investigators of the use of vigabatrin for the treatment of addiction and various forms of epilepsy and other central nervous system disorders, including members of our Scientific Advisory Board and the academic institutions with which they are affiliated. In some cases, we may provide unrestricted sponsorship funds for such studies. In other cases, we may provide alternative assistance to the investigator, most typically providing CPP-109 or CPP-115 drug substance or dosage form as well as matching placebo. We expect to continue supporting investigator studies in the future to the extent that they meet criteria acceptable to us. Such criteria include research on the use of vigabatrin and/or CPP-115 to treat addiction, various forms of epilepsy and/or other central nervous system disorders, to assist investigators in designing their studies so that such studies are most appropriately conducted and, to the extent possible, to make sure that these investigator studies potentially complement, and do not adversely impact, our activities.

A study describing the positive results obtained in an investigator-initiated, Phase II, randomized double-blind, placebo-controlled trial conducted in Mexico in 2007 was published in the November 2009 issue of The American Journal of Psychiatry, a world leading peer-reviewed medical journal. The paper, entitled Randomized, Double-Blind, Placebo-Controlled Trial of Vigabatrin for the Treatment of Cocaine Dependence in Mexican Parolees, was authored by Jonathan D. Brodie, M.D., Ph.D., Brady G. Case, M.D., Emilia Figueroa, M.D., Stephen L. Dewey, Ph.D., James A. Robinson, M.Ed., Joseph A. Wanderling, M.A. and Eugene M. Laska, Ph.D. Drs. Dewey, Brodie and Laska are members of our Scientific Advisory Board. The trial provided evidence that vigabatrin may be effective in the treatment of cocaine addiction. One hundred and three (103) community-based, non-hospitalized cocaine addicted individuals participated in this trial conducted at a single site in Mexico City, Mexico. Of the 103 participants, 50 were treated with vigabatrin and 53 received placebo. A total of 53 subjects completed the 9 week treatment period. Twice-weekly urine screening tests were obtained from each subject in order to objectively evaluate each subject s cocaine use. All subjects were also offered one group

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counseling session per week. The primary outcome measure of the trial was no self-reported cocaine use or positive urine tests for cocaine use during the last three weeks of the nine-week trial.

Eighteen subjects fulfilled the criteria for the primary outcome measure. Fourteen of the 50 subjects treated with vigabatrin (28.0%) versus four of the 53 subjects treated with placebo (7.5%) met the primary outcome measure. This result was statistically significant with a p-value of 0.009 (A p-value represents the probability that, if the test is repeated, a similar observation will be made. In addition, 12 of the abstinent subjects on vigabatrin versus 2 of the abstinent placebo subjects remained abstinent for 4 additional weeks (p=0.002). Generally, a p-value of less than 0.05 indicates that the different results between treatment groups were unlikely to be random). Additional findings included increased retention and self-reported abstinence from alcohol favoring vigabatrin.

Two of our collaborators have received a $1.2 million grant from the U. S. Department of Defense to conduct an animal study of the use of vigabatrin in combination with opiates to effectively manage pain while reducing the potential for opiate addiction. This research is being conducted by a research team led by Wynne K. Schiffer, Ph.D. and Stephen L. Dewey, Ph.D. of The Feinstein Institute for Medical Research at North Shore Long Island Jewish Health System (LIJ) and by Jonathan D. Brodie, M.D., Ph.D. from the Department of Psychiatry at New York University s School of Medicine. Opioid abuse is one of the many substance addiction indications covered under our exclusive license of Brookhaven s vigabatrin use patent portfolio. We are supplying study materials (CPP-109) to facilitate this study.

A team of researchers led by Kyle M. Kampman, M.D., Associate Professor of Psychiatry at the Veteran s Administration Medical Center Department: Psychiatry affiliated with the University of Pennsylvania School of Medicine s Treatment Research Center have initiated a randomized, double-blind placebo-controlled study in 60 cocaine and alcohol co-dependent subjects. Subjects are receiving either CPP-109 (vigabatrin) or matching placebo, in addition to weekly counseling for eight weeks. The primary outcome measures are cocaine abstinence confirmed by twice weekly urine drug screens and alcohol abstinence measured by self-report. Recruitment is targeted to be completed in 12 months. NIDA is providing the majority of funding for this study as part of a pilot trial program included in a P50 center grant. The goal of this pilot project is to rapidly screen medications for the treatment of comorbid alcohol and cocaine dependence in small clinical trials. The program also utilizes state of the art techniques to ensure excellent medication adherence and treatment retention so that reliable results can be obtained rapidly to inform future larger trials. We have provided CPP-109 and matching placebo and financial support to conduct eye-safety examinations to facilitate the study.

An animal study reporting positive pre-clinical efficacy in a rat multiple hit model in which the use of CPP-115 was evaluated for the treatment of infantile spasms was reported on at the American Epilepsy Society s 65th Annual Meeting held in December 2011. The study was authored by Stephen W. Briggs, Tomonori Ono, MD, PhD, Solomon L. Moshe, MD and Aristea S. Galanopoulou, MD, PhD of the Saul R. Korey Department of Neurology, Dominick P. Purpura Department of Neuroscience, Laboratory of Developmental Epilepsy, The Comprehensive Epilepsy Center (CEC) at Montefiore Medical Center / Albert Einstein College of Medicine of Yeshiva University, Bronx, New York. The study concluded that (i) CPP-115 suppresses spasms in the multiple-hit model of IS, with onset of effect as early as the day after the first dose; (ii) the therapeutic doses of CPP-115 were well tolerated in developing rat pups; and (iii) CPP-115 showed efficacy for a longer duration at lower doses that were better tolerated than the previously tested therapeutic vigabatrin doses.

CPP-115 is being evaluated by the Anticonvulsant Screening Program (ASP) of the National Institute of Neurological Disorders and Stroke (NINDS), one of the institutes within the National Institutes of Health (NIH). To date, CPP-115 has been tested in about 20 animals models of epilepsy, including maximal electric shock (MES) in both rats and mice, corneal kindling in mice, minimal clonic seizure (6 Hz) model in mice, and subcutaneous picrotoxin (scPIC). CPP-115 was also evaluated for potential efficacy in neuroprotection and neuropathic pain models. CPP-115 has shown significant potential in a variety of epilepsy models and NIH is continuing the evaluation of CPP-115 in other models of epilepsy. An evaluation of CPP-115 in additional models for neuropathic pain is also ongoing.

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We recently agreed to provide study materials (CPP-109) and financial support for a small proof-of-concept study to be undertaken at an academic institution in the United States to evaluate the use of CPP-109 in treating Tourette Syndrome. This proof-of-concept study is expected to take approximately one year to complete.

Competitive Landscape

Disease Background and Our Market Opportunity

We are focusing primarily on two market opportunities that can be exploited by pharmacotherapies that inhibit GABA-aminotransferase (GABA-AT); drug addiction and epilepsy.

Drug Addiction. Research has established that neurochemical signals responsible for craving and addiction can be modulated through a GABA-ergic mechanism. We have been developing CPP-109 for the treatment of drug addiction and will also be evaluating CPP-115 for potential use in the treatment of drug addiction as well. Due to the differing stages of development for these two drugs, we expect CPP-109 to be approved as the first drug to treat cocaine addiction with CPP-115 following later for both epilepsy and then cocaine, methamphetamine and/or other forms of drug addiction.

Epilepsy. Epilepsy is not a neurological disorder with a single underlying cause, but is instead a complex spectrum of neurological disorders with many neurological origins exhibiting a large variation of severities. As such, there are a large number of therapies spanning many pharmacological mechanisms of actions, several medical devices, and in extreme cases, neurosurgical procedures including up to removal of half of the brain. We will develop a new drug, CPP-115, that reduces neuronal excitability through a GABA-ergic mechanism.

CPP-109

While there are no currently approved therapies for cocaine addiction, we are aware of certain other therapies that are under development. These can be broadly classified into six groups:

Cocaine-mimetics. The mechanism of action of these drugs is similar to cocaine. None of these approaches have, to our knowledge, shown any efficacy.

Cocaine-antagonists. These compounds are intended to prevent a cocaine induced dopamine surge by limiting the release of dopamine (drugs that act on GABA receptors, for example) or drugs that block the effects of a cocaine induced dopamine surge (dopamine receptor antagonists, for example). All of the known drugs in this class, with the exception of the GABA-AT inhibitors (CPP-109 and CPP-115) are receptor active and could require increasing dosing over time. None of these compounds are presently approved for marketing to treat addiction.

Dopamine ß-hydroxylase inhibitors. These compounds block the enzyme that converts dopamine to norepinephrine, which raises dopamine levels in the central nervous system (CNS). We believe that this strategy is designed to address withdrawal, rather than craving and euphoria. This approach, to our knowledge, has yet to show any efficacy.

Analeptics. These compounds stimulate the central nervous system. None of these compounds are presently approved for marketing to treat addiction, although we believe that one such product is currently undergoing Phase II clinical trials.

Addiction Vaccines. These vaccines are designed to block cocaine or methamphetamine transport into the brain. They are not broadly immunogenic in humans to date and require several injections. They also may not address issues relating to craving or other behaviors associated with cocaine or methamphetamine addiction. We also believe that they can be overwhelmed by increasing dosages of the abuse

drug. To date, reported data from clinical trials have not shown that the vaccines are capable of facilitating the attainment and maintenance of abstinence, a key therapeutic goal.

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D3 Antagonists. These compounds block the dopamine signal at the subclass of dopamine receptor (D3) thought to be responsible for the reward signals stimulated by drugs of abuse. Glaxo Smith Kline (GSK) developed a D3 antagonist (GSK598809) through Phase I for cocaine addiction, but has halted development of all CNS drugs and announced that it is exiting the CNS drug market segment. GSK is seeking to divest this asset. Abbott Laboratories is currently in Phase II development of ABT-925, another D3 antagonist, for the treatment of schizophrenia. Independent academic investigators are evaluating ABT-925 for the treatment of cocaine addiction and smoking cessation. Other D3 antagonists may also be under development.

On August 21, 2009, the FDA approved two NDAs for Sabril® for the treatment of infantile spasms and as an adjunctive (add-on) therapy for adult patients with refractory complex partial seizures who have failed several treatments. The NDAs are for different formulations of Sabril®, and both NDAs are held by Lundbeck. Because of the risks of visual field damage associated with vigabatrin, Sabril® was approved under an FDA-mandated REMS program.

We are not aware of any on-going or planned studies by Lundbeck intended to evaluate Sabril® for any addiction indication, and we believe that any attempted commercialization by Lundbeck of Sabril® for the treatment of cocaine and/or other addictions would violate our licensed patents (and we have advised Lundbeck of our belief in that regard). We would vigorously assert our intellectual property rights if Lundbeck sought to market Sabril® for the treatment of any addictive or obsessive compulsive conditions covered by our patents. There can be no assurance we would be successful in that regard.

CPP-115 for Epilepsy

Epilepsy represents a large and growing market opportunity. Sales of drugs currently marketed for the treatment of epilepsy totaled approximately $8.9 billion in the United States during 2006, according to IMS Health. These sales included prescriptions of these drugs for both epilepsy and other indications, including neuropathic pain.

The market for epilepsy treatments is highly competitive. Large pharmaceutical companies, including Pfizer (Neurontin®, Lyrica®, Dilantin®, Zarontin®), J&J (Topamax®), UCB (Keppra®), Abbott (Depakote®), GSK (Lamictal®), Roche (Klonopin®), and Novartis (Trileptal®) sell, or are developing, epilepsy therapies. However, as stated earlier, approximately one-third of all epilepsy patients are refractory to treatment with any currently available epilepsy treatments. It is difficult to determine sales of products specifically for epilepsy as many of these products are used in other indications such as neuropathic pain, migraine, dementia, and bipolar disorders.

Intellectual Property Rights

Licensing and Patents

Protection of our intellectual property and proprietary technology is a strategic priority for our business. We rely on a combination of patent, trademark, copyright and trade secret laws along with institutional know-how and continuing technological advancement, to develop and maintain our competitive position. Our ability to protect and use our intellectual property rights in the continued development and commercialization of our technologies and products, operate without infringing the proprietary rights of others, and prevent others from infringing our proprietary rights, is crucial to our continued success. We will be able to protect our products and technologies from unauthorized use by third parties only to the extent that they are covered by valid and enforceable patents, trademarks or copyrights, or are effectively maintained as trade secrets, know-how or other proprietary information. See Item 1A., Risk Factors Risks Related to Our Intellectual Property.

Brookhaven License Agreement

We have been granted an exclusive, worldwide license from Brookhaven to nine patents relating to the use of vigabatrin for a range of indications, including the treatment of a wide variety of substance addictions, with expiration dates for the issued patents between 2018 and 2023, with the principal patents expiring in 2018. Additionally, we received approval from the European Union (EU) with respect to one of our principal patents, which has allowed us to seek registration for this patent in eighteen EU member states.

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The license agreement, which is dated as of April 30, 2006 and which supersedes a previous license agreement that was entered into in 2002, grants us an exclusive worldwide license, including the right to sublicense, to make, have made, use, and/or sell licensed products and practice the licensed process with respect to the medical application in humans of vigabatrin under certain patent rights. These rights are subject to the United States government s rights to practice the licensed process for its own use. The purpose of this agreement is to permit us to commercialize products upon the receipt of government regulatory approval for the use of vigabatrin for the treatment of human drug addiction and addiction-related behavior. In exchange for such rights, we paid Brookhaven an initial fee of $50,000 and have agreed to pay a fee of $100,000 in the year of NDA approval for CPP-109, $250,000 in each of the second and third years following approval, and $500,000 per year thereafter until the last patent expires. In addition, upon the filing of an NDA for CPP-109 and the approval of an NDA for CPP-109, we will be obligated to reimburse Brookhaven for certain expenses it incurs in connection with the filing, prosecution and maintenance of all patents and patent applications included in the patent rights we have licensed. We also have the right to enter into sub-license agreements, and if we do a royalty of 20% of any sub-license fees will be payable to Brookhaven.

We have also agreed to consult with Brookhaven on at least a quarterly basis with respect to drug development steps taken and progress made toward the objective of gaining marketing approval from the FDA for any licensed product from the beginning of our agreement through the date the FDA grants us its approval to sell any licensed product. We have also agreed to have in effect and maintain a liability insurance policy in an amount of at least $1,000,000 to cover claims arising out of the manufacture and use of licensed products and such policy shall designate Brookhaven as an additional insured. We have agreed to increase and maintain, throughout the life of the agreement and for five years after its termination, liability insurance coverage in the amount of at least $5,000,000 upon acceptance by the FDA of our application to commence Phase III clinical trials involving licensed products. Our agreement with Brookhaven expires simultaneously with the expiration of the last to expire patent it has licensed to us.

During July 2010, we announced that the European Patent Office has granted to Brookhaven a European patent for the use of vigabatrin for the prevention of addiction to opioids (e.g. oxycodone, hydrocodone) used in pain management. By dampening dopamine release and thus, the euphoria associated with opioids, the opioid/vigabatrin combination may lower or prevent addictive liability without adversely affecting pain relief. Further, we announced in December 2010 that the Canadian Intellectual Property Office has granted to Brookhaven a patent for the use of vigabatrin for the prevention of addiction in pain management. The patent is broad and includes the use of vigabatrin/CPP-109 in combination with opioids (e.g., oxycodone, hydrocodone) for pain management. We license these patents from Brookhaven.

Brookhaven has formally advised us that they believe that the amount due them for patent related expenses as of December 31, 2011 was approximately $1.3 million. We believe that we are only contingently liable to Brookhaven for approximately $166,000, and we have advised Brookhaven that we are disputing their determination of patent-related expenses due under the license agreement. There can be no assurance as to the outcome of this matter. In any event, no patent-related expenses are due to Brookhaven under the license agreement until we submit an NDA for CPP-109.

Northwestern University License Agreement

On August 27, 2009, we entered into a license agreement with Northwestern under which we acquired worldwide rights to commercialize new GABA aminotransferase inhibitors and derivatives of vigabatrin which have been discovered and patented by Northwestern. Under the terms of the license agreement, Northwestern granted us an exclusive worldwide license to certain composition of matter patents related to the new class of inhibitors and a patent application relating to derivatives of vigabatrin. We have designated the lead compound to be developed under this license as CPP-115.

We believe that the newly licensed compounds are the only known GABA aminotransferase inhibitors in existence or in development other than vigabatrin. We also believe, based on our pre-clinical testing to date of CPP-115, that the newly licensed compounds are significantly more potent than vigabatrin with less visual side effects than vigabatrin. We plan to seek to develop these compounds for the treatment of several indications, including epilepsy (specifically, complex partial seizures and infantile spasms) and drug