

CORCEPT THERAPEUTICS INC

Form 10-K

March 31, 2008

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SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 10-K

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

For the fiscal year ended December 31, 2007

or

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

For the transition period from to

Commission File Number: 000-50679

CORCEPT THERAPEUTICS INCORPORATED

(Exact Name of Corporation as Specified in Its Charter)

Delaware

77-0487658

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(State or other jurisdiction of incorporation or organization)

(I.R.S. Employer Identification No.)

149 Commonwealth Drive

Menlo Park, CA 94025

(Address of principal executive offices, including zip code)

(650) 327-3270

(Registrant's telephone number, including area code)

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

Title of Each Class:
Common Stock, \$0.001 par value

Name of Each Exchange on which Registered:
The NASDAQ Stock Market LLC

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

None

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

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

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

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

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

Large Accelerated Filer ☐

Accelerated Filer ☐

Non-accelerated filer ☐ (Do not check if a smaller reporting company)

Smaller reporting company ☒

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

The aggregate market value of voting and non-voting common equity held by non-affiliates of the Registrant was approximately \$31,000,000 as of June 30, 2007 based upon the closing price on the Nasdaq Stock Market reported for such date. This calculation does not reflect a determination that certain persons are affiliates of the Registrant for any other purpose.

On March 31, 2008 there were 48,473,164 shares of common stock outstanding at a par value \$.001 per share.

DOCUMENTS INCORPORATED BY REFERENCE

None.

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PART I

This Annual Report on Form 10-K, or Form 10-K, contains forward-looking statements within the meaning of Section 21E of the Securities Exchange Act of 1934, as amended, and Section 27A of the Securities Act of 1933, as amended. All statements contained in this Form 10-K, other than statements of historical fact, are forward-looking statements. When used in this report or elsewhere by management from time to time, the words believe, anticipate, intend, plan, estimate, expect, and similar expressions are forward-looking statements. Such forward-looking statements are based on current expectations, but the absence of these words does not necessarily mean that a statement is not forward-looking. Forward-looking statements made in this Form 10-K include, but are not limited to, statements about:

the progress of our research, development and clinical programs and timing of the introduction of CORLUX® and future product candidates;

estimates of the dates by which we expect to report results of our clinical trials;

our ability to market, commercialize and achieve market acceptance for CORLUX or other future product candidates;

uncertainties associated with obtaining and enforcing patents;

our estimates for future performance; and

our estimates regarding our capital requirements and our needs for additional financing.

Forward-looking statements are not guarantees of future performance and involve risks and uncertainties. Actual events or results may differ materially from those discussed in the forward-looking statements as a result of various factors. For a more detailed discussion of such forward-looking statements and the potential risks and uncertainties that may impact upon their accuracy, see the Risk Factors section of this Form 10-K and the Overview and Liquidity and Capital Resources sections of the Management's Discussion and Analysis of Financial Condition and Results of Operations section of this Form 10-K. These forward-looking statements reflect our view only as of the date of this report. Except as required by law, we undertake no obligations to update any forward looking statements. Accordingly, you should also carefully consider the factors set forth in other reports or documents that we file from time to time with the Securities and Exchange Commission.

ITEM 1. BUSINESS

Overview

Corcept Therapeutics Incorporated is a pharmaceutical company headquartered in Menlo Park, California engaged in the development of drugs for the treatment of severe psychiatric and metabolic diseases. Our current focus is on the development of drugs for disorders that are associated with a steroid hormone called cortisol. Elevated levels and abnormal release patterns of cortisol have been implicated in a broad range of human disorders. Our scientific founders are responsible for many of the critical discoveries illustrating the link between psychiatric and metabolic disorders and aberrant cortisol. Since our inception in May 1998, we have been developing our lead product, CORLUX®, a glucocorticoid receptor II, or GR-II, antagonist. CORLUX modulates the effect of cortisol by selectively blocking the binding of cortisol to one of its two known receptors, the GR-II receptor, also known as the Type II or GR receptor.

Psychotic depression. We have an exclusive patent license from Stanford University for the use of GR-II antagonists to treat the psychotic features of psychotic major depression, hereinafter referred to as psychotic depression. The United States Food and Drug Administration (FDA) has granted fast track status to our program to evaluate the safety and efficacy of CORLUX for the treatment

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of the psychotic features of psychotic depression. Psychotic depression affects approximately three million people annually in the US. There is no FDA-approved treatment for psychotic depression. Psychiatrists currently use two approaches: electroconvulsive therapy (ECT), which involves passing an electrical

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current through the brain until the patient has a seizure, and combination drug therapy (simultaneous use of antidepressant and antipsychotic medications). Both ECT and combination drug therapy almost always have slow onsets of action and debilitating side effects. By modifying the level and release pattern of cortisol within the human body, we believe that CORLUX may be able to treat the psychotic features of psychotic depression more quickly and effectively and with fewer side effects than is possible with currently available treatments.

Three Phase 3 clinical trials have been completed. While the response rate to medicine exceeded the response rate to placebo in each of these studies for the primary endpoint, a 50% reduction in the Brief Psychiatric Rating Scale Positive Symptom Subscale (BPRS PSS) at day 7 sustained to day 56, in none of these studies was the difference in response rate statistically significant. However a robust relationship was demonstrated between higher dosing plasma level and a higher response rate. This relationship was tested prospectively in the last of our Phase 3 trials using a predetermined plasma concentration that correlates with response and was met with statistical significance. We believe that the confirmation of a drug concentration/response correlation threshold for efficacy provides a strong basis for our next Phase 3 study.

If we obtain FDA approval, we initially intend to market and sell CORLUX for psychotic depression in the United States directly to hospitals with in-patient psychiatric units, first focusing on those that use ECT. We then intend to expand our sales efforts to address the larger group of psychotic depression patients currently undergoing combination drug therapy. Given the concentrated nature of the initial target audience, we believe that we will be able to generate significant revenue with a relatively small, highly-focused medical education and commercialization team.

Antipsychotic-induced Weight Gain Mitigation. In June 2007, we announced preliminary top-line results of our proof-of-concept study evaluating the ability of CORLUX to mitigate weight gain associated with the administration of olanzapine. The top line results indicated a statistically significant reduction in weight gain in those subjects who took olanzapine plus CORLUX compared to those who took olanzapine alone. The purpose of this study was to explore the hypothesis that GR-II antagonists would mitigate weight gain associated with atypical antipsychotic medications, such as olanzapine, risperidone, clozapine and quetiapine, which are widely used to treat schizophrenia and bipolar disorder. All medications in this group are associated with treatment emergent weight gain of varying degrees and carry a warning label relating to treatment emergent hyperglycemia and diabetes mellitus. Eli Lilly provided olanzapine and financial support for this study.

Cushing's Syndrome. The FDA has granted Orphan Drug Designation for CORLUX for the treatment of endogenous Cushing's Syndrome, hereinafter referred to as Cushing's Syndrome, a disorder caused by prolonged exposure of the body's tissues to high levels of the hormone cortisol. Orphan drugs obtain seven years of marketing exclusivity from the date of approval, as well as tax credits for clinical trial costs, marketing application filing fee waivers and assistance from the FDA in the drug development process.

The Investigational New Drug application (IND) for the evaluation of CORLUX for the treatment of Cushing's Syndrome was opened in September 2007. The FDA has indicated that a single study may provide a reasonable basis for the submission of a New Drug Application (NDA) for this indication. This trial was opened for enrollment late in December 2007.

In addition to the above, we also own or have exclusively licensed issued patents and patent applications relating to the treatment of several disorders that we believe also result from, or are negatively affected by, prolonged exposure to elevated cortisol including increasing the therapeutic response to electroconvulsive therapy (ECT), mild cognitive impairment, stress disorders and the treatment of delirium. We also have filed patent applications for additional diseases that may benefit from treatment with a drug that blocks the GR-II receptor.

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We were incorporated in the State of Delaware on May 13, 1998. Our registered trademarks include Corcept® and CORLUX®. Other service marks, trademarks and tradenames referred to in this document are the property of their respective owners.

The Role of Cortisol in Disease

Cortisol is a steroid hormone that plays a significant role in the way the body reacts to stressful conditions and is essential for survival. Cortisol significantly influences metabolism, exerts a clinically useful anti-inflammatory effect and contributes to emotional stability. Insufficient levels of cortisol may lead to dehydration, hypotension, shock, fatigue, low resistance to infection, trauma, stress and hypoglycemia. Excessive levels of cortisol may lead to edema, hypertension, fatigue and impaired glucose tolerance.

Elevated levels and abnormal release patterns of cortisol have also been linked to a broad range of psychiatric and metabolic conditions, such as mood changes, psychosis and cognitive impairment. Cognition, including attention, concentration and memory, is impaired by elevated levels and abnormal release patterns of cortisol. Prolonged elevated levels of cortisol are neurotoxic and may accelerate the dementia process in patients with cognitive disorders such as Alzheimer's disease.

Many studies have shown that patients with psychotic depression have elevated levels and abnormal release patterns of cortisol. This abnormal cortisol pattern is not usually present in patients with nonpsychotic depression. More than 15 years ago, one of our scientific co-founders postulated that elevated levels of cortisol in patients with psychotic depression lead to elevated levels of dopamine, an important chemical substance found in the brain. Elevated levels of dopamine have been implicated in both delusional thinking and hallucinations. This was a clinically relevant hypothesis because it led to the concept that antipsychotic medications, which act by blocking dopamine, in combination with antidepressant medications, could be useful in treating psychotic depression. The hypothesis also led to the concept that by regulating the level and release patterns of cortisol, one could normalize dopamine levels in the brain, which may, in turn, ameliorate the symptoms of psychotic depression. In addition to cortisol's effect on dopamine levels, research has shown that prolonged elevated cortisol may also play a direct role in causing the symptoms of psychotic depression.

The challenge in regulating levels of cortisol, however, is that it is needed for natural processes in the human body. Destroying the ability of the body to make cortisol or to drastically reduce its presence would result in serious detrimental effects. To have a viable therapeutic effect, a compound must be able to selectively modulate cortisol effects.

Glucocorticoid Receptor Antagonists

Cortisol is produced by the adrenal glands and is carried via the bloodstream to the brain, where it directly influences neuronal function. In the brain, cortisol binds to two receptors, Glucocorticoid Receptor I and Glucocorticoid Receptor II, also known as GR-I and GR-II. GR-I is a high-affinity receptor that is involved in the routine functions of cortisol. It has approximately ten times the affinity of GR-II for cortisol and its binding sites are filled with cortisol nearly all the time. In general, GR-II binding sites do not fill until levels of cortisol become elevated. Short-term activation of GR-II has benefits, which include helping the individual to be more alert and better able to function under stressful conditions. Long-term activation of GR-II, however, has been shown to have significant toxicity and appears to be linked to multiple psychiatric and metabolic disease states, particularly psychotic depression. The action of cortisol can be moderated by the use of blockers, or antagonists, that prevent the binding of the hormone to its receptors. These antagonists, referred to as glucocorticoid receptor antagonists, may prevent the undesirable effects of elevated levels and abnormal release patterns of cortisol.

The discovery that the brain has high affinity and low affinity receptors for cortisol was critical to our scientific approach in treating the psychosis manifested by patients with psychotic depression because it allowed for a specific target for a potential medication. CORLUX, also known as mifepristone or RU-486, works by

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selectively blocking the binding of cortisol to GR-II while not affecting GR-I. Because of its selective affinity, we believe that CORLUX can have a therapeutic benefit by modulating the effects of abnormal levels and release patterns of cortisol without compromising the necessary normal functions of cortisol.

Overview of Psychotic Depression

Psychotic depression is a serious psychiatric disease in which a patient suffers from severe depression accompanied by delusions, hallucinations or both. These psychotic features typically develop after the onset of a depressed mood, but may develop concurrently as well. Once psychotic symptoms occur, they usually reappear with each subsequent depressive episode. Of particular importance, when the patient's mood returns to normal the psychosis also resolves.

Psychotic depression is not a simple combination of psychosis and depression, but rather a complex interaction between a predisposition to become psychotic and a predisposition to become severely depressed. In addition to psychosis, clinical features and outcomes that distinguish psychotic from nonpsychotic depression include elevated levels and abnormal release patterns of cortisol, motor abnormalities, a substantially higher suicide rate, more prominent sleep abnormalities and more potential for brain injury.

Data from the National Institutes of Mental Health published in 2005 indicate that depressive disorders affect an estimated 9.5% of adults in the United States, or about 19 million people each year. Of these 19 million people, many published studies show that approximately 15-20%, or about three million people, have psychotic depression. Most patients with psychotic depression suffer their first episode of major depression between the ages of 30 and 40 and the majority will experience more than one episode in their lifetime. Psychotic depression is more prevalent than either schizophrenia or bipolar I disorder. Psychotic depression is characterized by severe depression accompanied by psychosis (delusions and/or hallucinations). People with psychotic depression are approximately 70 times more likely to commit suicide in their lifetime than the general population and often require lengthy and expensive hospital stays.

We believe that people afflicted with psychotic depression are, as a group, unrecognized and undertreated because of:

reluctance on the part of patients with psychotic depression to accurately report their psychotic symptoms;

misdiagnosis of the disease by primary care physicians;

reluctance of patients and their families to be associated with the stigma of hospitalization for psychiatric care; and

adverse side effects associated with current treatments for psychotic depression.

Current Treatments for Psychotic depression

There are two treatment approaches for psychotic depression currently used by psychiatrists: ECT and combination drug therapy. Neither of these treatments has been approved by the FDA for psychotic depression and both approaches almost always have slow onsets of action and debilitating side effects. Of the two treatments, ECT is generally considered to be more effective.

ECT involves passing an electrical current through the brain until the patient has a seizure. At least 100,000 patients receive ECT each year in the United States, with each patient requiring approximately six to twelve procedures over a period of three to five weeks. ECT is administered while the patient is under general anesthesia and the procedure requires the use of an operating room, as well as the participation of a psychiatrist, an anesthesiologist and a nurse. General anesthesia and paralytic agents are necessary to avoid fractures of the spine

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that otherwise could result from the seizures caused by ECT. Although ECT provides a reduction in depressive and psychotic symptoms, the procedure can result in cognitive impairment, including permanent memory loss, cardiovascular complications, headache, muscle ache and nausea, in addition to complications related to general anesthesia.

Combination drug therapy is an alternative treatment for psychotic depression that involves taking antipsychotic drugs such as olanzapine, haloperidol or chlorpromazine in combination with antidepressant medication. Patients on combination drug therapy often require three weeks or more to show improvement in their symptoms and treatment can take months to complete. Antipsychotic drugs can cause significant adverse side effects, including weight gain, diabetes, sedation, permanent movement disorders and sexual dysfunction.

Because a therapeutic response to ECT and combination drug therapy does not occur for several weeks, neither approach prevents lengthy and expensive hospital stays in patients who are seriously ill. Consequently, a significant need exists for a medication that provides rapid relief from the psychotic symptoms of psychotic depression, as such a medication would substantially reduce the length of suffering associated with the illness. We believe that people suffering from psychotic depression would prefer a treatment that did not involve the risks of anesthesia, adverse side effects and stigma associated with ECT or the slow onset of action associated with both ECT and combination drug therapy. If an alternative treatment was approved by the FDA and had secured third-party reimbursement, we believe that many patients with psychotic depression would choose that alternative.

CORLUX for the Psychotic Features of Psychotic Depression

CORLUX is an oral medication that we are developing to treat the psychotic features of psychotic depression. CORLUX is a GR-II antagonist that appears to mitigate the effects of the elevated and abnormal release patterns of cortisol in patients suffering from psychotic depression. We intend CORLUX to be a once-daily treatment given to patients with psychotic depression over 7 consecutive days in a controlled setting, such as a hospital or physician's office. Mifepristone, the active ingredient in CORLUX, in addition to blocking GR-II, blocks the progesterone receptor and has been approved by the FDA for termination of early pregnancy.

We believe that CORLUX may significantly reduce psychotic symptoms of psychotic depression in many patients within one week and allow patients to be more easily maintained on antidepressant therapy alone without the need for ECT or antipsychotic medication. We believe that CORLUX may be superior to currently available treatments because we believe that CORLUX will enable patients with psychotic depression to improve their quality of life more quickly and with fewer side effects than with ECT or combination drug therapy.

CORLUX for Psychotic Depression Clinical Trials

Psychiatric Rating Scales. In our clinical trials, we assess the efficacy of CORLUX utilizing psychiatric rating scales commonly used to support regulatory approval of new antipsychotic and antidepressant medications. These scales include the:

BPRS: The Brief Psychiatric Rating Scale (BPRS) is an 18-item instrument to assess psychopathology. It incorporates a range of psychiatric symptoms, including anxiety, depression, guilt, hostility and suicidality. Each of the 18 symptoms is scored on a numeric scale ranging from 1 (not present) to 7 (extremely severe).

BPRS Positive Symptom Subscale (BPRS PSS): This subscale, which is based on four items of the BPRS, assesses a patient's psychotic features by measuring the patient's conceptual disorganization, suspiciousness, hallucinatory behavior and unusual thought content.

HAM-D: The Hamilton Depression Scale (HAM-D) is a 24-item instrument designed to measure the severity of a number of depressive symptoms such as insomnia, depressed mood, concentration, ability

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to experience pleasure, and agitation. Each question has 3 to 5 possible responses, with associated scores ranging from 0 to 4. The total score is calculated from all items.

Clinical Trials. We initiated two Phase 3 trials in the United States in September 2004 (Study 07) and October 2004 (Study 06) and an additional Phase 3 trial in Eastern Europe in the second quarter of 2005 (Study 09) to evaluate the safety and efficacy of CORLUX. These three studies have been completed. The details of the results of these trials are discussed below. Prior to initiating these trials, we completed the following four clinical trials with CORLUX for the treatment of psychotic features of psychotic depression:

In 2001, we completed our first trial, an open label dose finding clinical trial with 30 patients evaluating the efficacy, tolerability and dose response of CORLUX for the treatment of the psychotic features of psychotic depression. After one week of treatment, approximately two-thirds of the patients in the two higher dosage groups experienced clinically meaningful reductions in psychosis, as measured by the BPRS PSS. A clinically meaningful reduction in psychosis represents a reduction of symptoms that are readily recognizable by patients and physicians.

Later in 2001, we initiated two clinical trials designed to evaluate the safety and efficacy of CORLUX for the treatment of the psychotic features of psychotic depression. The two trials, which we call Study 02 and Study 03, were double-blind, placebo-controlled safety and efficacy studies.

Study 02, in which 208 patients were enrolled, showed that CORLUX was well tolerated and that there were no discernable problems with drug interactions between CORLUX and commonly prescribed antipsychotic and antidepressant medications.

Study 03, in which 221 patients were enrolled, demonstrated with statistical significance that patients in the CORLUX group were more likely to achieve a rapid and sustained reduction in psychotic symptoms than patients in the control group, as measured by a 30% reduction in the BPRS at 7 days sustained to 28 days (p value = 0.01) and a 50% reduction in the BPRS PSS at 7 days sustained to 28 days (p value = 0.01). The term *p value* is a statistical term that indicates the probability that an observed result is random. A p value of 0.05 or less is considered statistically significant. All p values for Study 03 are based on an observed cases, per protocol analysis, which takes into account only those patients who received at least 6 doses of study medication, had the BPRS assessed at day 0 and day 7 and had no major violations of the inclusion/exclusion criteria or other protocol specified criteria.

In our fourth trial, we evaluated the safety of retreatment in patients with a favorable response to treatment in Study 02 and Study 03, and our analysis indicates that patients tolerated their retreatment well.

Dose Finding Study. In January 2001, we concluded our first study, which was an open-label study designed to measure clinically meaningful reductions in the psychiatric rating scales. The 33 patients with psychotic depression enrolled in the study were randomly assigned to receive daily doses of 50 mg, 600 mg, or 1200 mg of CORLUX orally for 7 days. There was no placebo control group. After 7 days of treatment, clinically meaningful reductions in the psychiatric rating scales were observed for patients in the 600 mg and 1200 mg treatment groups, as summarized below.

	50 mg Dose Group	600 mg Dose Group	1200 mg Dose Group	600 mg and 1200 mg Dose Groups Combined
30% or greater reduction in BPRS	4/11 (36)%	7/10 (70)%	6/9 (67)%	13/19 (68)%
50% or greater reduction in positive symptom subscale of BPRS	3/11 (27)%	6/10 (60)%	6/9 (67)%	12/19 (63)%
50% or greater reduction in Ham-D scale	2/11 (18)%	5/10 (50)%	3/9 (33)%	8/19 (42)%

Results were similar in the 600 mg and 1200 mg dose groups, but there was an apparent dose-response relationship when the results of the 50 mg group were compared to the two higher dose groups. Sixty-eight

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percent of patients in the higher dose groups (600 mg and 1200 mg combined) had a clinically meaningful 30% or greater reduction in the BPRS, compared to 36% in the 50 mg group. The items in the BPRS that are most specific to psychotic depression are contained in the BPRS PSS. Every patient with psychotic depression experiences one or more of these subscale symptoms. More than 60% of patients in the higher dosage groups had a 50% or greater reduction in the BPRS PSS within one week of treatment. Each of the reductions in the psychiatric rating scales that the study measured is a clinically meaningful reduction in symptoms that would be readily recognized by patients, family members, physicians and hospital staff. None of the patients in the trial experienced clinically consequential side effects and none dropped out of the trial due to side effects.

Double-blind Clinical Trials. In June and July 2001, we initiated two double-blind, randomized clinical trials, Study 02 and Study 03, each of which was designed to enroll 200 patients and to evaluate the safety and efficacy of CORLUX in patients with psychotic depression. In each study, patients received either CORLUX or placebo. Both studies were designed and powered to test the hypothesis that the group of patients treated with CORLUX would be superior to the control group in achieving a rapid (within 7 days) and sustained (to 28 days) reduction in their BPRS score of at least 30%.

The two studies were identical in design except for one of the key entry criteria. Patients enrolled in Study 02 were allowed to receive any antipsychotic or antidepressant medications deemed appropriate by their treating physicians prior to entry into the study and throughout the week of administration of the study drugs, CORLUX or placebo. Therefore, in Study 02, patients received their usual treatment plus CORLUX or placebo. In Study 03, patients were not allowed to receive any antipsychotic or antidepressant medication for at least 7 days prior to administration of the study drug or during the week of study drug administration. All patients enrolled in the studies were treated in the hospital. After day 7, while the studies remained blinded, each treating physician was allowed to add any additional treatment, including ECT or antipsychotic, antidepressant or other psychotropic medications.

Study 02

The results of Study 02 indicated that CORLUX was well tolerated and that there were no discernable problems with clinical drug interactions when CORLUX was taken in combination with other antipsychotic or antidepressant medications. The median number of psychotropic medications that patients in Study 02 were receiving in addition to CORLUX was four. Although patients in the usual treatment plus CORLUX group more frequently achieved the study's primary endpoint, a rapid and sustained reduction in psychotic symptoms as measured by a 30% decline in the BPRS at Day 7 sustained to Day 28, than did patients in the usual treatment plus placebo group, the difference between the groups was not statistically significant. The study did demonstrate with statistical significance (p value = 0.02) that the usual treatment plus placebo group required ECT or more antipsychotic medication between Day 7 and Day 28 and was less likely to be discharged from the hospital during the week of dosing (p value = 0.05) relative to the usual treatment plus CORLUX group. Post-hoc analysis of Study 02 data further revealed that patients in the usual treatment plus CORLUX group were more likely than patients in the usual treatment plus placebo group to achieve a rapid and sustained asymptomatic condition, as measured by a BPRS score of 25 or less. Although the number of patients achieving this result was small, the difference between the usual treatment plus CORLUX group and the usual treatment plus placebo group was statistically significant (p value = 0.01). All p values for Study 02 are based on an intent-to-treat analysis, which takes into account patients in the trial who received at least one dose of study medication.

Study 03

The results of Study 03 indicated that CORLUX was well tolerated as demonstrated by the finding that there was no statistically significant difference in adverse events observed between the CORLUX group and the placebo group. Study 03 also demonstrated with statistical significance (p value = 0.01) that patients who received CORLUX were more likely than patients who received placebo to achieve a rapid and sustained reduction in psychosis as measured by the study's original primary endpoint, a 30% reduction in the BPRS at

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Day 7 sustained to Day 28. Study 03 also showed with statistical significance (p value = 0.01) that patients in the CORLUX group were more likely than patients in the placebo group to achieve a 50% reduction in the BPRS PSS at Day 7 sustained to Day 28. In addition, patients in the placebo group were more likely than patients in the CORLUX group to receive antipsychotic medication between Day 7 and Day 28, although this difference was not statistically significant. All p values for Study 03 are based on an observed cases, per protocol analysis, which takes into account only those patients who received at least 6 doses of study medication, had the BPRS assessed at Day 0 and Day 7 and had no major violations of the inclusion/exclusion criteria or other protocol specified criteria.

At the request of the FDA, we followed the last third of patients enrolled in this trial to Day 56. Of those patients who exhibited at least mild psychotic symptoms on Day 0 (score ≥ 12 on the BPRS PSS), Study 03 showed with statistical significance that patients receiving CORLUX were more likely than patients receiving placebo to achieve a 50% reduction in the BPRS PSS at Day 7 sustained to Day 56 (p value = 0.03).

We indicated to the FDA shortly before the study concluded that we would use as our primary endpoint for the study the number of patients who became asymptomatic at the end of one week as measured by the BPRS, a differentiating characteristic that we had noted in post-hoc Study 02 analysis. In Study 03, as in Study 02, only a small number of patients became asymptomatic at the end of one week and, in Study 03, there was no statistically significant difference between the CORLUX and placebo groups.

Of the approximately 480 patients who were enrolled in these completed Phase 2 studies, over 240 individuals were treated with CORLUX. The drug seemed to be well tolerated by these patients, with a low incidence of adverse events. In Studies 02 and 03, the most commonly reported adverse events were headache, dizziness, nausea and sedation. The incidence of these adverse events was similar in the control and CORLUX groups. In Study 02, rash was the only adverse event where there was a statistically significant difference (p value = 0.05) between groups: 4% occurrence in the CORLUX group compared to no occurrences in the control group. In Study 03, there was no statistically significant difference in the occurrence of any adverse event.

We have also conducted a small open label study to evaluate the safety of retreatment in patients who had a favorable response to treatment in Study 02 and Study 03. Twenty-eight patients completed the study. Our analysis indicates that patients tolerated their retreatment well.

Phase 3 Clinical Trials. We have completed three randomized, double-blind, placebo-controlled Phase 3 clinical trials to further assess the safety and efficacy of CORLUX for the treatment of the psychotic features of psychotic depression. Two of these trials (Study 06 and Study 07) were conducted primarily in the United States. The third trial (Study 09) was conducted in Eastern Europe. The design of all three trials was based on the design of Study 03, described above.

The primary endpoint for Study 06 and Study 07 was the proportion of patients with at least a 50% improvement in the BPRS PSS at both Day 7 and Day 56. This type of endpoint is known as a categorical endpoint. Patients must have had at least mild psychotic symptoms (BPRS PSS ≥ 12) to enter the studies and were hospitalized if clinically necessary. BPRS PSS assessments were also be made at Days 14, 28 and 42. The primary endpoint for Study 09 was the proportion of patients with at least a 50% improvement in the BPRS PSS at both Day 7 and Day 28. A secondary endpoint of Study 09 was the same as the primary endpoint for Study 06 and Study 07.

Study 07

The first of these trials, Study 07, which began in September 2004, enrolled 257 patients at 25 sites in the United States and Europe with a randomized one-to-one distribution into either a treatment or a placebo arm. Patients in the treatment arm received 600 mg of CORLUX once daily for a period of seven days. Patients did not take any antidepressant or antipsychotic medication for at least one week before beginning the seven day

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treatment period. After the seven days of CORLUX treatment, all patients received antidepressant therapy through Day 56. Treatment with antipsychotic medications or electroconvulsive therapy was not allowed at any time during the study.

In August 2006 we announced the results of Study 07. In this study 30.5% of the patients receiving CORLUX and 28.6% of the patients receiving placebo met the primary endpoint. This was not a statistically significant difference in response rate. The two key secondary endpoints of Study 07 also failed to achieve statistical significance. There was an unusually high placebo response rate in this trial. At Day 56, for example, approximately 80% of the patients in both of the arms of the study were responders as measured by a 50% improvement in BPRS PSS score.

Even though Study 07 did not meet its primary endpoint, an analysis of the data from this clinical trial revealed some items of interest that helped us to determine the direction for the continued development of CORLUX for treating psychotic depression including the fact that patients with higher plasma levels of CORLUX separated from patients who took placebo with statistical significance.

An item of interest in Study 07 was a statistically significant site by treatment effect. A site by treatment analysis is conducted for all clinical trials to know if the results seen at one site are generalizable to patients seen at another site. A statistically significant site by treatment effect indicates that the effect of treatment with a drug is not uniform at the various clinical sites participating in the clinical trial. One site may have a large difference in the response rate favoring the drug group and another site may have a large difference in the response rate favoring the comparator group. When a site by treatment interaction is statistically significant, it is not possible to know which sites represent the true activity of the drug.

Another interesting observation from Study 07 was that patient enrollment did not have an even pace. 150 patients were enrolled in the first 480 days of the study (September 2004 through December 2005) and 107 patients in the last 120 days. An analysis of the results of the first 150 patients revealed a statistically significant difference on the primary endpoint favoring patients who took CORLUX compared to those who did not. Most of the clinical sites enrolling patients during this time had participated in the conduct of Study 02 and Study 03.

The sites that had enrolled the first 150 patients continued enrolling patients until the trial was fully enrolled at the end of April 2006. By the end of the study this group of sites had enrolled a total of 215 patients, approximately the same total number of patients enrolled in Study 03. The primary endpoint was also met with statistical significance with these 215 patients. After January 1, 2006, in order to increase the speed of enrollment we added eight additional sites. These sites had not participated previously in clinical trials sponsored by Corcept. The eight sites that joined the trial in 2006 enrolled a total of 42 patients. In this group of 42 patients, those who took placebo had a substantially higher response rate on the primary endpoint than those who took CORLUX. The disparate outcome between the group of 215 patients and the group of 42 patients resulted in a statistically significant site by treatment effect.

An important teaching from Study 07 derives from an analysis of the relationship between the concentration of CORLUX in patients' blood on Day 7 and the likelihood that patients meet the response criteria of the primary endpoint. Patients with CORLUX plasma levels higher than 1650 nanograms per milliliter had statistically significant greater response rates observed than did patients who received placebo.

Study 09

Study 09 was a randomized, double-blind, placebo-controlled study in which 247 patients were enrolled at 17 sites. The primary endpoint, a responder analysis, was the proportion of patients with at least a 50% improvement in the BPRS PSS score at both Day 7 and Day 28. We announced the results of this study in September 2006. The study revealed no meaningful separation in response between patients receiving CORLUX and patients receiving placebo on the primary endpoint. The two key secondary endpoints of Study 09 also failed

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to achieve statistical significance. Study 07 had an extremely high placebo response rate; the magnitude of the placebo response rate in Study 09 was unprecedented. At Day 56, for example, approximately 95% of the patients in both of the arms of the study were responders as measured by a 50 percent improvement in BPRS PSS score. Although not the primary or a key secondary endpoint, it is interesting to note that there was a statistically significant separation between the CORLUX group and the comparator group on their change from baseline to Day 56 on the BPRS PSS scale. Change from baseline to study end is an endpoint commonly used to measure the efficacy of antipsychotic and antidepressant medications. However, because of the already high degree of response in the comparator group, it is difficult to determine how much additional clinical utility is conferred by this finding. We do not expect to be able to use Study 09 as one of the two positive efficacy trials required by the FDA for a fileable NDA.

Study 06

Study 06, which began in October 2004, enrolled 443 patients at 45 sites in the United States and Europe. These patients were evenly distributed among three active dose groups (300 mg, 600 mg and 1200 mg) and a placebo group, with patients receiving once daily dosing for a period of seven days. The three dosing levels respond to the FDA's request to supplement data on a range of doses to augment the data provided by our open label dose ranging study completed in 2001. Patients in the study did not take any antidepressant and antipsychotic medication for at least one week before the seven day treatment period and received antidepressant therapy starting on Day 1 through Day 56. As with Study 07, treatment with antipsychotic medications or electroconvulsive therapy was not allowed at any time during this study.

We reported the initial results of this trial in March 2007. These results indicated that this study did not achieve statistical significance with respect to the primary endpoint. However, there was a statistically significant correlation between plasma levels and clinical outcome achieved during treatment. Patients whose plasma levels rose above a predetermined threshold statistically separated from both those patients whose plasma levels were below the threshold and those patients who received placebo. In particular, those patients in Study 06 who achieved a predetermined level of 1661 nanograms of CORLUX per milliliter of plasma separated from the placebo group with statistical significance. At substantially lower plasma levels, there was no distinguishable difference in response rates between patients who received CORLUX and those receiving placebo. This study confirms our previous similar finding in Study 07 that at higher plasma levels the drug candidate is able to demonstrate desired clinical effects. Further, the incidence of serious adverse events did not differ between placebo and any of the three CORLUX dose groups.

Fourth Phase 3 trial Study 14

We believe that the confirmation of a correlation between drug concentration and clinical response, as well as other observations from Study 06 and our two other recently completed Phase 3 clinical trials, serves as a strong basis for our next Phase 3 study which commenced in March 2008. The protocol for this trial incorporates the learnings from the three completed trials that address the established relationship between increased drug plasma levels and clinical response and attempts to decrease the random variability observed in the results of the psychometric instruments used to measure efficacy. We have met with the FDA to discuss and seek their input concerning the design of this trial. In this trial we will use a CORLUX dose of 1200 mg once per day for seven days because, as expected, at this dose more patients achieved the predetermined plasma concentration in Study 06. In Study 06, 80% of the patients who took 1200 mg of CORLUX achieved a drug plasma level sufficient to separate responders from non-responders. We believe that this change in dose as well as other modifications to the protocol should allow us to determine the efficacy of CORLUX in the treatment of the psychotic features of psychotic depression. In addition, in our initial review of a summary of the safety data, we have seen no difference between any of the dose levels used in Study 06.

Given the serious nature of psychotic depression, the lack of any approved drugs for the disorder and the data from our first clinical trial, the FDA has granted a fast track designation for CORLUX for the treatment of the psychotic features of psychotic depression. In addition, the FDA has indicated that CORLUX will receive a

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priority review if no other treatment is approved for psychotic depression at the time we submit our New Drug Application, or NDA.

Additional Non-Efficacy Trials and Pre-clinical Studies. In support of an eventual NDA submission, we plan to conduct additional clinical trials to assess the safety of retreatment of patients with CORLUX. We also plan to conduct several small trials to evaluate how the drug acts on the human body, how the human body acts on the drug and the drug's safety. In addition to our clinical trials, we have completed a standard 12-month toxicology study in the dog and a carcinogenicity study in the rat. A second carcinogenicity study in the mouse is underway. These studies are designed to meet FDA requirements and the guidelines of an international regulatory body called the International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use.

Clinical Trial Agreements. Many of our Phase 3 clinical trials are conducted through the use of clinical research organizations (CROs.) At our request, these organizations oversee clinical trials at various institutions to test the safety and efficacy of our product candidates for the targeted indications. The preparation for our fourth Phase 3 clinical trial, Study 14, evaluating CORLUX for the treatment of the psychotic features of psychotic depression is being conducted under a Letter of Intent with ICON Clinical Research, LP (ICON) and we are in negotiations with ICON regarding an agreement for the conduct of the full trial activities. In addition, we entered into an agreement with MedAvante, Inc., effective March 17, 2008, to provide centralized psychiatric rating services of patients to be screened and enrolled in Study 14. This agreement may be terminated by Corcept with 30 days notice to MedAvante.

The previous three Phase 3 trials for CORLUX for this indication were conducted under clinical development agreements with other CROs that will be responsible for the completion of final reporting under these trials.

GR-II Antagonist Platform

We have assembled a patent portfolio covering the treatment of psychiatric and metabolic disorders that may benefit from drugs that block the GR-II receptor. In addition to psychotic depression, we own or have exclusively licensed issued patents for the use of GR-II antagonists for the prevention and treatment of stress disorders, for increasing the therapeutic response to ECT and for the treatment of :

weight gain following treatment with antipsychotic medication,

early dementia, including early Alzheimer's disease;

mild cognitive impairment;

gastroesophageal reflux disease;

cognitive deterioration in adults with Down's Syndrome;

delirium; and

psychosis associated with cocaine addiction.

We believe that cortisol plays a role in a variety of other diseases. We have eight pending U.S. method of use patent applications covering GR-II antagonists for the treatment of various diseases.

Clinical Trials in Other Psychiatric and Metabolic Disorders.

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Cushing's Syndrome. In July 2007, the FDA granted Orphan Drug Designation for CORLUX for the treatment of Cushing's Syndrome a disorder caused by prolonged exposure of the body's tissues to high levels of the hormone cortisol. Sometimes called hypercortisolism, it is relatively rare. An estimated 10 to 15 of every one million people are affected each year. It most commonly affects adults aged 20 to 50.

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Orphan Drug Designation is a special status granted by the FDA to encourage the development of treatments for diseases or conditions that affect fewer than 200,000 patients in the United States. Orphan drugs obtain seven years of marketing exclusivity from the date of approval, as well as tax credits for clinical trial costs, marketing application filing fee waivers and assistance from the FDA in the drug development process.

In September 2007, the Investigational New Drug application (IND) for the evaluation of CORLUX for the treatment of Cushing's Syndrome was opened. The FDA has indicated that a single 50-patient open label study defined by the protocol submitted with the application for the IND may provide a reasonable basis for the submission of an NDA for this indication. This trial was opened for enrollment late in December of 2007.

Antipsychotic-induced Weight Gain Mitigation. In June 2007, we announced top-line results of our proof-of-concept study evaluating the ability of CORLUX to mitigate weight gain associated with the administration of olanzapine. The results indicated a statistically significant reduction in weight gain in those subjects who took olanzapine plus CORLUX compared to those who took olanzapine alone. The purpose of this study was to explore the hypothesis that GR-II antagonists would mitigate weight gain associated with atypical antipsychotic medications, such as olanzapine, risperidone, clozapine and quetiapine, which are widely used to treat schizophrenia and bipolar disorder. All medications in this group are associated with treatment emergent weight gain of varying degrees and carry a warning label relating to treatment emergent hyperglycemia and diabetes mellitus. Eli Lilly provided olanzapine and financial support for this study.

In this study, 57 lean, healthy men (body mass index of 25 or less) were randomized to receive either olanzapine plus placebo (n=22), olanzapine plus CORLUX (n=24) or CORLUX plus placebo (n=11). This study took place in an institutional setting where daily weights were recorded and a range of metabolic parameters were measured. In the two week study, subjects in the olanzapine alone group gained an average of 2.5 pounds more than subjects in the olanzapine plus CORLUX group and 2.2 pounds more than subjects in the CORLUX alone group, which are highly statistically significant differences (p<.001). The difference in weight gain trajectory was apparent in the first days of the study, reaching statistical significance during the first week. The increase in waist circumference in subjects who received olanzapine alone was also significantly greater than subjects who received olanzapine plus CORLUX (p<.01). The study was not designed to have statistical power to detect significant effects or metabolic measures, however, notable non-statistically significant group differences were observed. Compared to patients taking olanzapine plus CORLUX, patients taking olanzapine plus placebo experienced greater increases from baseline to end of study in both triglycerides and fasting insulin. No unexpected study drug related adverse events were observed.

The combination of olanzapine and CORLUX is not approved for any indication. The purpose of this study was to explore the hypothesis that GR-II antagonists would mitigate weight gain associated with atypical antipsychotic medications. The group of medications known as atypical antipsychotics, including olanzapine, risperidone, clozapine and quetiapine, are widely used to treat schizophrenia and bipolar disorder. All medications in this group are associated with treatment emergent weight gain of varying degrees and carry a warning label relating to treatment emergent hyperglycemia and diabetes mellitus.

In April 2005, we announced results from two preclinical studies conducted in a rat model of olanzapine-induced weight gain. These studies demonstrated that CORLUX's GR-II antagonist action has the potential to both reduce the weight gain associated with olanzapine and to prevent the weight gain associated with the initiation of treatment with olanzapine.

Discovery Research

In early 2003, we initiated a discovery research program to identify and patent more selective GR-II antagonists in order to develop a pipeline of products for proprietary use. Our discovery chemistry was conducted at a contract research organization in the United Kingdom. Through the research program, we identified and filed patent applications for three distinct series of GR-II antagonists. These compounds appear to be as potent as

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Corcept's lead product CORLUX in blocking cortisol but, unlike CORLUX, they do not appear to block the progesterone or other steroid receptors. Currently we are evaluating one compound identified under our research program, which develops particularly high plasma and brain concentrations in an animal model, in a human microdosing study using Xceleron's Accelerator Mass Spectrometry technology under an agreement signed in July 2007.

Medical Education and Commercialization

We intend to develop our own medical education and commercialization infrastructure in the United States for CORLUX because we believe that the initial market for psychotic depression in the United States is highly concentrated and accessible. We anticipate that this will include hiring a small, experienced field sales force of up to approximately 35 people. We intend to focus initially on patients who are candidates for ECT by marketing to hospitals and psychiatrists that perform ECT. We estimate that there are approximately 900 hospitals with more than 30 in-patient psychiatric beds. Of these, we estimate that approximately 300 offer ECT. We believe that approximately 1000 psychiatrists administer most ECT procedures. Subsequently, we also intend to expand our commercialization efforts to address the larger set of patients with psychotic depression currently undergoing combination drug therapy, which would require an increase in the size of our initial sales force.

We believe that a significant opportunity exists to further expand the market for the treatment of the psychotic features of psychotic depression beyond patients currently treated by ECT and combination drug therapy. A large portion of the people who suffer from psychotic depression remain unrecognized and undertreated. We intend to develop medical educational programs to alert the medical community about early diagnosis of psychotic depression and increase awareness regarding CORLUX.

We are planning for the commercialization of CORLUX. To achieve commercial success for any approved product, we must either develop a sales and marketing force or enter into arrangements with others to market and sell our products.

Manufacturing

As a drug development entity, we intend to continue to utilize our financial resources to complete the development of CORLUX and advance other product candidates rather than diverting resources to establishing our own manufacturing facilities.

We intend to continue to rely on experienced contract manufacturers to produce our product candidates. We have entered into manufacturing agreements with two contract manufacturers, Produits Chimiques Auxiliaires et de Synthèse SA (PCAS) and ScinoPharm Taiwan (ScinoPharm), to produce the active pharmaceutical ingredient, or API, for CORLUX. The agreement with PCAS is for an initial period of five years with an automatic extension for one additional year unless either party gives twelve months prior notice that it does not want the extension. There is no guaranteed minimum purchase commitment under this agreement. If PCAS is unable to manufacture the product for a consecutive six-month period, we have the right to terminate the agreement. The agreement with ScinoPharm obligates us to purchase at least \$1,000,000 of bulk mifepristone per year following the commercial launch of CORLUX. This agreement is terminable by either party at any time. We have also entered into an agreement with another contract manufacturer, PharmaForm, L.L.C., for the production of CORLUX tablets for use in clinical activities. In the event we are unable, for whatever reason, to obtain mifepristone or CORLUX from our contract manufacturers, we may not be able to identify alternate manufacturers able to meet our needs on commercially reasonable terms and in a timely manner, or at all. To date, our need for CORLUX tablets has been limited to the amounts required to support our clinical trials.

Competition

If approved for commercial use as a treatment for the psychotic features of psychotic depression, CORLUX will compete with established treatments, including ECT and combination drug therapy.

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ECT has been shown to be the most effective treatment for psychotic depression, but it carries the risks of general anesthesia, potential memory loss and other adverse effects as well as the stigma associated with the procedure. Use of CORLUX does not require anesthesia and, in our clinical trials conducted to date, patients treated with CORLUX have not exhibited the adverse effects associated with ECT.

Other competitors include companies that market antipsychotic drugs that are used off-label as part of combination drug therapy for psychotic depression. To reduce the psychotic features of psychotic depression, these drugs generally are taken in combination with antidepressant medication over a period of weeks to several months. Unlike the use of CORLUX, this extended course of treatment may put patients at risk of significant adverse side effects, including weight gain, diabetes, sedation, permanent movement disorders and sexual dysfunction. Antipsychotics include Bristol-Myers Squibb's Abilify, Novartis' Clozaril, Pfizer's Geodon and Navane, Ortho-McNeil's Haldol, Janssen Pharmaceutica's Risperdal, AstraZeneca's Seroquel, GlaxoSmithKline's Stelazine and Thorazine, Mylan's Mellaril, Schering Corporation's Trilafon and Eli Lilly's Zyprexa.

We are aware of one clinical trial that has taken place, conducted by the pharmaceutical division of Akzo Nobel, for a new chemical entity for the treatment of psychotic depression. This new medicine is a GR-II antagonist, the commercial use of which would be covered by our patent. In 2004, Akzo Nobel filed an observation in our exclusively licensed European patent application with claims directed to psychotic depression, in which Akzo Nobel challenged the claims of that patent application. In 2005, we filed a rebuttal to Akzo Nobel's observation. In February 2006, the European Patent Office, or EPO, allowed our patent application. In July 2006, the patent was issued. We are not aware of any public disclosures by any company, other than Akzo Nobel, regarding the development of new medicinal products to treat psychotic depression. However, other companies may be developing new drug products to treat psychotic depression and the other conditions we are exploring. Our present and potential competitors include major pharmaceutical companies, as well as specialized pharmaceutical firms. Most of our competitors have considerably greater financial, technical and marketing resources than we do. We expect competition to intensify as technical advances are made.

We are aware that Laboratoire HRA Pharma has received an Orphan Drug Designation in the United States and Europe for the use of mifepristone to treat a subtype of Cushing's Syndrome and has begun a clinical trial in Europe and the United States. If this product is approved for commercialization before CORLUX, our potential future revenue could be reduced if there is off-label use of mifepristone for psychotic depression or for Cushing's Syndrome that cannot be protected by our intellectual property.

Many colleges, universities and public and private research organizations are also active in the human health care field. While these entities focus on education, they may develop or acquire proprietary technology that we may require for the development of our product candidates. We may attempt to obtain licenses to this proprietary technology.

Our ability to compete successfully will be based on our ability to develop proprietary products, attract and retain scientific personnel, obtain patent or other protection for our product candidates, obtain required regulatory approvals and manufacture and successfully market our future products either alone or through outside parties.

Intellectual Property

Patents and other proprietary rights are important to our business. It is our policy to seek patent protection for our inventions, and to rely upon trade secrets, know-how, continuing technological innovations and licensing opportunities to develop and maintain our competitive position.

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Under an agreement with Stanford University, we have licensed exclusive rights to the following issued U.S. patents and any corresponding foreign patents:

U.S. Patent Number	Subject Matter	Expiration Date
U.S. Pat. No. 6,150,349	Use of GR-II antagonists in the treatment of psychotic major depression	October 5, 2018
U.S. Pat. No. 6,362,173	Use of GR-II antagonists in the treatment of cocaine-induced psychosis	October 5, 2018
U.S. Pat. No. 6,369,046	Use of GR-II antagonists in the treatment of early dementia	February 4, 2019

We are required to make milestone payments and pay royalties to Stanford University on sales of products commercialized under any of the above patents. We are currently in compliance with our obligations under the agreement. If Stanford University were to terminate any of our exclusive licenses due to breach of the license on our part, we would not be able to commercialize CORLUX for the treatment of the psychotic features of psychotic depression, cocaine-induced psychosis or early dementia.

We also own issued U.S. patents for the use of GR-II antagonists in the treatment of mild cognitive impairment, for the treatment of weight gain following treatment with antipsychotic medication, for the prevention and treatment of stress disorders, for the treatment of delirium, the treatment of gastroesophageal reflux disease, for inhibiting cognitive deterioration in adults with Down's Syndrome and for increasing the therapeutic response to ECT. In addition, we have three U.S. composition of matter patent applications covering specific GR-II antagonists and eight U.S. method of use patent applications covering certain GR-II antagonists, including the treatment of:

catatonia;