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EQUITY RESEARCH
Company Report

December 9, 2015

Cerecor Inc. (CERC - $3.21)

Healthcare / Biotechnology

Initiation of Coverage - Changing the Depression Paradigm

Ticker:	**CERC**
Rating:	**Buy**
Price Target:	**$10.00**

We are initiating coverage of Cerecor Inc. (CERC) with a Buy rating and a $10 price target. CERC has two late stage pipeline compounds to treat depression and comorbidities associated with depression. The lead compound CERC-301 is a rapid onset of effect NMDA antagonist for patients refractory to current standard of care that is currently in Phase 2 development with top line results expected in 2H16. The National Institute of Mental Health estimates that approximately 50% of depression patients fail to respond to the standard of care depression treatments, and with over 16 million adults in the US alone (and over 150 million worldwide) the opportunity for a second line treatment is enormous. The second compound is CERC-501, a Kappa opioid receptor antagonist which is expected to start Phase 1b/2a trials for smoking cessation in 1H16 and could target the ~55 million people in the US who are cigarette smokers. By comparison Pfizer's Chantix for smoking cessation which sold ~$650 million in each of the past two years. CERC might also develop CERC-501 for alcohol and drug cessation as well. The third compound in development is CERC-406, a COMT inhibitor that is in preclinical development for cognitive impairment. With multiple catalysts in 2016 targeting some of the biggest pharmaceutical opportunities in the world, we believe CERC is an exciting undiscovered opportunity in central nervous system disorders.

- **Huge unmet need for refractory MDD patients.** Potentially up to 8 million people in the US alone are refractory to standard of care antidepressants. CERC-301 doesn't need much of that group to generate significant value.
- **Co-occurring addiction disorders a significant opportunity.** Smoking cessation, drug or alcohol addiction all represent almost 100 million people in the US overall. PFE's Chantix for smoking cessation alone has sold ~$6.5B since its launch in 2006.
- **Cognitive impairment treatment in early stages.** An earlier stage program but targeting another unmet medical need in loss of executive function in MDD patients. Another potential ~8 million patient opportunity.
- **Initiate with a Buy rating, $10 PT.** Our price target is based on a sum-of-the-parts analysis with CERC-301 valued at $6/share, CERC-501 at $2.75/share, and a net cash (end 2016) and technology at $1.25/share.

Trading Data:

Last Price (12/08/2015)	$3.21
52–Week High (10/15/2015)	$6.65
52–Week Low (12/02/2015)	$2.75
Market Cap. (MM)	$27.7
Shares Out. (MM)	8.63

Earnings Estimates: (per share)

	1Q	2Q	3Q	4Q	FY	P/E
FY17E	(0.28)	(0.30)	(0.30)	(0.33)	(1.20)	NA
FY16E	(0.27)	(0.32)	(0.42)	(0.34)	(1.35)	NA
FY15E	(0.15)	(0.78)	(0.47)	(0.28)	(1.17)	NA
FY14	(0.20)	(6.88)	(9.57)	(1.16)	(5.23)	NA

Source: Laidlaw & Company estimates

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Summary and Investment Thesis

We are initiating coverage of Cerecor Inc. (CERC) with a Buy rating and a $10 price target. CERC has two late stage pipeline compounds to treat depression and comorbidities associated with depression. The lead compound CERC-301 is a rapid onset of effect NMDA antagonist for patients refractory to current standard of care that is currently in Phase 2 development with top line results expected in 2H16. The National Institute of Mental Health estimates that approximately 50% of depression patients fail to respond to the standard of care depression treatments, and with over 16 million adults in the US alone (and over 150 million worldwide) the opportunity for a second line treatment is enormous. The second compound is CERC-501, a Kappa opioid receptor antagonist which is expected to start Phase 1b/2a trials for smoking cessation in 1H16 and could target the ~55 million people in the US who are cigarette smokers. By comparison Pfizer's Chantix for smoking cessation sold ~$650 million in each of the past two years. CERC might also develop CERC-501 for alcohol and drug cessation as well. The third compound in development is CERC-406, a COMT inhibitor that is in preclinical development for cognitive impairment. With multiple catalysts in 2016 targeting some of the biggest pharmaceutical opportunities in the world, we believe CERC is an exciting undiscovered opportunity in CNS disorders. Our $10 price target is based on a sum-of-the-parts analysis with CERC-301 valued at $6/share, CERC-501 at $2.75/share, and net cash (estimated at year end 2016) and technology at $1.25/share.

- **Huge unmet need for refractory MDD patients with CERC-301.** Potentially up to 8 million people in the US alone are refractory to standard of care antidepressants. CERC-301 is an NMDA antagonist that selectively blocks the NMDA receptor subunit 2B (NR2B) and is an oral adjunctive medication for patients with MDD who are refractory to their current antidepressant treatment and remain severely depressed.
- **CERC-501 for co-occurring addiction disorders targets another significant opportunity.** Smoking cessation, drug or alcohol addiction all represent a potential base of almost 100 million people in the US overall. PFE's Chantix for smoking cessation alone has sold ~$6.5B since its launch in 2006. CERC-501 is a selective orally bioavailable kappa opioid receptor (KOR) antagonist. If ultimately approved for adjunctive treatment of MDD and for substance use disorders separately; CERC plans to further develop CERC-501 for the concurrent treatment of MDD and substance use disorders (co-occurring disorders).

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- **CERC-406 for cognitive impairment treatment interesting, but in early stage development.** CERC-406 is in pre-IND stages for the unmet medical need of loss of executive function in MDD patients. CERC-406 is a non-nitro-catechol, brain penetrant, membrane-bound COMT inhibitor without the potential toxicity of earlier generation COMT inhibitors. By working on memory and executive function impairment has the potential to target an underserved patient sub segments and could be applicable across multiple diseases such as depression, ADHD, and schizophrenia.

Figure 1. Upcoming Potential Catalysts

Event	Expected Timing
CERC-501 phase 1b/2a initaition smoking cessation	1Q16
CERC-406 preclinical IND-enabling studies	1H16
CERC-301 phase 2 top line data in MDD	2H16
CERC-501 phase 1b/2a top line data smoking cessation	2H16

Source: Company reports; Laidlaw and Company estimates

Valuation

We value CERC at $10/share based on a sum-of-the-parts valuation. CERC-301 is valued at $6/share based on a 7x multiple of 2026 WW sales of $600 million, discounted back 10 years at 15%. CERC-501 is valued at $2.75/share based on a 7x multiple of 2026 WW sales of $400 million, discounted back 10 years at 20%. We value net cash (end 2016) and the technology value at $1.25/share

Figure 2: Sum-of-the-Parts Analysis

Sum-of-the-parts valuation		
Segment	Valuation (000's)	Per share value
CERC-301	$1,038,176	$6.00
CERC-501	$452,216	$2.75
Technology value	$200,000	$1.00
Cash (end of '16E)	$40,484	$0.25
Total	$1,730,875	**$10.00**
2016 fully diluted shares out (000)		172,393

Source: Company reports; Laidlaw and Company estimates

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Company Description

Cerecor (CERC) is a clinical-stage biopharmaceutical company that is developing drugs to treat neurological and psychiatric disorders including major depressive disorder (MDD) and adjunctive treatment for substance use disorders associated with MDD. CERC key development product candidates are CERC-301, CERC-501 and CERC-406. CERC-301 is currently in Phase 2 development as an oral, adjunctive treatment of patients with MDD who are failing to achieve an adequate response to their current antidepressant treatment and are severely depressed. The drug has a fast track designation and is an N-methyl-D-aspartate (NMDA) receptor antagonist (inhibitor). The NMDA receptor is responsible for controlling neurological adaptation and CERC-301 has the potential to be a first-in-class medication that could cause a significant reduction in depression symptoms for refractory patients. CERC-301 is faster acting than the current standard of care, becoming active in a matter of days vs weeks-to-months with conventional MDD therapies. CERC-301 achieves a rapid onset of action by selectively blocking the NMDA receptor subunit 2B (NR2B), which appears to provide a rapid and significant antidepressant activity without the adverse side effect profile of non-selective NMDA receptor antagonists.

CERC is also developing CERC-501 which is expected to start Phase 1b/2a development in 1Q16 for smoking cessation. CERC-501 was acquired in February 2015, and is a potent and selective kappa opioid receptor (KOR), antagonist. KORs are believed to play key roles in modulating stress, mood and addictive behaviors, which form the basis of co-occurring disorders. CERC-501 is being developed for adjunctive treatment of MDD and for substance use disorders (smoking, alcohol, and/or cocaine). If CERC-501 ultimately receives approval for adjunctive treatment of MDD and for substance use disorders, CERC plans to further develop CERC-501 for the concurrent treatment of MDD and substance use disorders, or co-occurring disorders. CERC may develop CERC-501 in a Phase 2 clinical study in inadequately treated subjects with MDD currently on antidepressants, with a potential start date in 2H16.

CERC's third compound, currently in pre-clinical development, is CERC-406 which inhibits catechol-O-methyltransferase (COMT) within the brain. CERC-401 is expected to be developed for the treatment of residual cognitive impairment symptoms in patients with MDD.

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CERC-301 for Major Depressive Disorder (MDD)

Recently a new class of antidepressant has emerged known as antagonists of the N-methyl-D-aspartate (NMDA) receptor, a receptor subtype of the glutamate neurotransmitter system that is responsible for controlling neurological adaptation. Research on ketamine, has provided evidence that NMDA antagonists can provide significant antidepressant mood effects within 24 hours of administration, acting as rapid acting antidepressants in MDD and bipolar depression (Arch Gen Psychiatry, 2006). Moreover, research has also demonstrated that ketamine causes a rapid reduction in suicidal ideation, in contrast to conventional antidepressants that may actually worsen suicidal ideation in children, adolescents and young adults (J Clin Psychiatry, 2010). However, non-selective NMDA antagonists such as ketamine have significant limitations. Ketamine is an anesthetic, is not approved for use as an antidepressant, and causes increases in heart rate and blood pressure, hallucinations and other psychological manifestations.

CERC-301 is an NMDA antagonist that selectively blocks the NMDA receptor subunit 2B (NR2B) and is an oral adjunctive medication for patients with MDD who are refractory to their current antidepressant treatment and are severely depressed. Intravenous NMDA inhibitors such as ketamine, Rapastinel (GLYX 13) and NRX-1074 were shown in trials to reduce depression scores within 24 hours. In a pilot study by Pfizer, Traxoprodil (CP-101,606), which is also NR2B specific, did show efficacy before being halted due to QT prolongation side effects.

CERC-301 could have a rapid onset of effect within a matter of days (vs. weeks for standard of care), and may be well tolerated with fewer side effects than the leading adjunctive treatments currently available, such as atypical antipsychotics, whose treatment efficacy is hindered by side effects such as weight gain and increased risk of diabetes. We believe an antidepressant with rapid onset of effect can possibly provide its greatest benefit by quickly relieving suicidality, a risk factor for suicide.

In its completed adjunctive therapy in TRD Phase 2 study with 8mg daily dosing, CERC-301 did not meet its primary endpoint but proved to be safe and tolerable at daily doses up to 20mg. As a result, CERC is currently planning a Phase 2 efficacy study with a higher dose and revised dosing regimen of CERC-301 and expects topline results 1H16.

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Figure 3: CERC-301 8mg failed Phase 2 study design

Phase 2: CERC-301 for adjunctive major depressive disorder (MDD)	
Aim	Safety & efficacy of CERC-301 in treatment refractory MDD
Design	Rondom, 2x blind, placebo controlled, sequential parallel study
Dosing	8mg or placebo for 28 days
Endpoints	1': Hamilton Depression Ratings Scale (HAMD)-17 at 7 days 2': HAMD-17 avg. between 7-28 days after treatment; HDRS-17 after 28 days
Patients	N = 1357
Safety	Safe and well tolerated, no blood pressure or heart rate differences between
Results - 10/2014	No statistically significant difference from placebo on primary outcome

Source: Company reports

CERC-301 is currently in Phase 2 development as an oral, adjunctive treatment of patients with MDD, who are failing to achieve an adequate response to their current antidepressant treatment, and are severely depressed. CERC-301 received Fast Track Designation by the FDA in November 2013 for the treatment of MDD. Over time, the understanding of psychiatric and neurological disorders, as well as their biological underpinnings, has evolved based on a combination of clinical and pre-clinical research. Over the past 50 years, many depression therapies and hypotheses primarily have been based on changing the levels of monoamine neurotransmitters, such as serotine, norepinephrine and dopamine, in the brain. Manipulating these neurotransmitters impacts mood, but monoamine antidepressants are slow in onset, requiring multiple weeks for patients to obtain a response and patients may suffer from sexual dysfunction and other side effects from such treatment. Also, numerous studies have shown that many patients do not respond to their initial antidepressant therapy. For example, studies have showed that ~50% of patients fail to respond (achieving a 50% reduction in symptoms), and ~60% of patients fail to remit with currently available first-line antidepressant treatments (STAR-D Study, 2006).

Figure 4: CERC-301 12mg & 20mg Phase 2 study design

Phase 2: CERC-301 for adjunctive major depressive disorder (MDD)	
Aim	Safety & efficacy of CERC-301 in treatment refractory MDD
Design	10 US site, random, 2x blind, placebo controlled, 3-arm trial. 3 week wash-out, 2 week follow-up; ~80% power
Dosing	12mg, 20mg, or placebo dosed day 0 and day 7 in a fasted state
Endpoints	1': change from baseline in Bech-6 averaged between 2 & 4 days post-treatment; 2': Santen-7, HDRS-17, GAD-7-SR, SHAPS-SR, QIDS-SR, CGI-S, and CGI-I
Patients	N = 104
Results	Expected 2H16

Source: Company reports

Many modifications will be brought to the new CERC-301 trial. Since drug exposure was found to be too low, which was evident due to an absence of blood pressure and brain derived neurotrophic factor (BDNF) changes, higher doses of 12mg and 20mg will be administered weekly. In contrast to the previous study in which dosing was administered daily, dosing will now be done by intermittent administration consistent with all other NMDA depression studies.

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Figure 5: NMDA antagonist competition

	AZD8108	NRX-1074	Rapastinel	Esketamine	CERC-301
Mechanism of Action	NMDA antagonist	NMDA modulator	NMDA modulator	NMDA antagonist	**NR2B antagonist**
Phase of Development	Ph 1	Ph 2	Ph 2	Ph 3	**Ph 2**
Portential Indication	Suicidal ideation	MDD adjunctive	MDD adjunctive	MDD combination	**MDD adjunctive**
Route of Administration	Oral	IV/Oral	IV	Nasal Spray	**Oral**
Administration Schedule	tbd	tbd	1 or 2 per week	2 per week	**1 or 2 per week**
Onset of Action	Hours/days	Hours/days	Hours/days	Hours/days	**Days**

Source: Company presentation

In terms of patient qualifications to be involved in the trial, while the previous trial included high rates of personality disorders vs. mood disorders, the new trial will eliminate suicidality requirement and will employ 3rd party arbiter of screening and inclusion from MGH Psychiatry. Another difference between the new and old CERC-301 study pertains to the assessment scales. In fact, while HDRS-17 includes items which don't change over 1-2 days in the old study, the newer one will employ the Bech-6 (6 item subset of HDRS-17).

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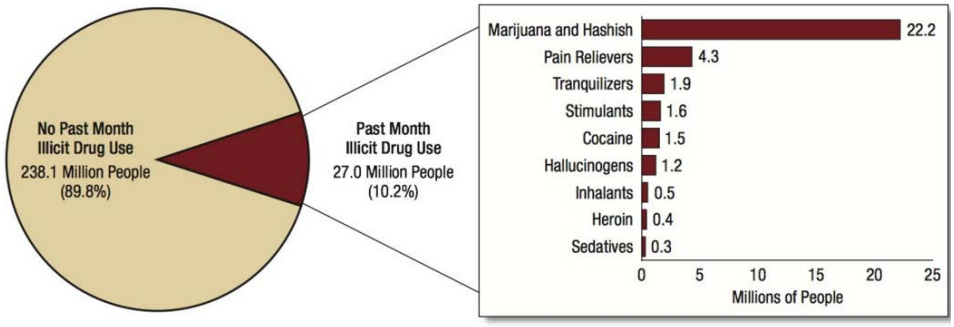
CERC-501 for Substance Use Disorders

CERC-501 is a selective orally bioavailable kappa opioid receptor (KOR) antagonist being developed for adjunctive treatment of MDD and for substance use disorders (smoking, alcohol, and/or cocaine). If ultimately approved for adjunctive treatment of MDD and for substance use disorders separately, CERC plans to further develop CERC-501 for the concurrent treatment of MDD and substance use disorders (co-occurring disorders).

In the US, ~55 million people are cigarette smokers, of which ~16 million are considered heavy drinkers, and ~25 million are illicit drug users. It was also found that ~40% of patients in a depressive episode smoke and that 68% of smokers attempt to quit each year, while less than 7% actually succeed. Unfortunately, there are currently no single therapies approved and marketed for co-occurring disorders. Kappa opioid receptors, or KORs, and their native ligand dynorphin are localized in areas of the brain which affect reward and stress and are believed to impact mood, stress and addictive disorders. CERC-501 has demonstrated target engagement in the brain using positron emission tomography imaging (PET). An estimated 16% of depressed patients also have a diagnosable addiction disorder and patients diagnosed with depression and anxiety disorders are twice as likely to abuse or be dependent on a drug or substance and vice versa. Both KORs and dynorphin, together comprising the kappa opioid system, are upregulated by stress and chronic exposure to drugs of abuse, are thought to mediate the negative emotional states seen in drug withdrawal and contribute to stress-induced reinstatement of drug seeking behavior (Pharmacology & Therapeutics, 2007).

Figure 6: 2014 National Survey on Drug Use and Health



Source: NIH

In animal models it has been observed that stress produces a depressive state that is believed to be associated with the activation of KOR and subsequent downstream signaling events. Administration of agents that stimulate the KOR system, or KOR agonists that act like dynorphin, decrease dopamine levels in areas of the brain involved with executive function, produce anxiety-like and depression-like behaviors in animals, exacerbate behaviors associated with drug withdrawal and increase the reinforcing effects of substances of abuse. The

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therapeutic potential of KOR antagonism has been demonstrated in animal models of anhedonia, depression, and anxiety, and KOR antagonists reduce the signs of nicotine, heroin and alcohol withdrawal in rodent models of dependence. In terms of human proof of concept studies, Alkermes' ALKS-5461 (functional kappa antagonist) has shown Phase 3 robust results in MDD with rapid onset of effect.

Without considering nicotine dependence, there are more than 5 million adults in the United States alone who suffer from co-occurring depression and substance use disorders. Such comorbidities put patients at greater risk. For instance, depending on when MDD onset occurs, MDD has been found to be related to the course of substance dependence, impacting areas such as remission of substance dependence and relapse into substance dependence after stable remission. Recent research suggests that a history of MDD is associated with a decreased ability to quit smoking and MDD over the last year is associated with an increased likelihood of smoking relapse.

Figure 7: CERC-501 Phase 2 trial design

Phase 2: CERC-501 for substance use disorders	
Aim	Efficacy & safety of CERC-501 in smoking cessation
Design	3 US sites, random, 2x blind, placebo control, crossover study design; ~80% power to detect mean latency to smoke & # of cigs smoked)
Dosing	5mg, 10mg, crossed over to placebo
Endpoints	1': latency (minutes) to start tobacco use after 18hrs inpatient withdrawl; # of cigarettes smoled during self-administratio period; 2': craving, mood, withdrawl, anxiety, anhedonia scores, subjective effects of
Patients	N = 50
Results	Initiating 1H16, results expected 2H16

Source: Company reports

One common link between the co-occurrence of depression and substance use disorders may be stress. Sustained stressful experiences can induce despair and increase the risk of clinical depression and substance use. Stress and mood are significant components of addiction relapse. Substance use often provides relief from stress, such that the substance of abuse often becomes a potent behavioral reinforce (Journal of Nicotine & Tobacco Research, 2000). Present pharmacologic treatments for co-occurring disorders consist either of treatment for the psychiatric disorder or the treatment for the addiction, but not the treatment of the underlying connection between the two.

For example, the nonselective opioid antagonist naltrexone, an FDA-approved medication for alcohol dependence in patients who are able to abstain from alcohol in an outpatient setting prior to treatment initiation, is not FDA approved as an antidepressant or an antianxiety agent. The smoking cessation aid varenicline, a mixed nicotinic agent, is associated with depression as a serious side effect. Similarly, antidepressant medication exerts a modest beneficial effect for patients with combined depressive and substance-use disorders. It is not a stand-alone treatment, and concurrent therapy directly targeting the addiction is also indicated (Journal of the American Medical Association, 2004). Therefore, we believe a tremendous need exists for pharmacotherapies effective in the treatment of co-occurring disorders. CERC-501 was licensed by Lilly Feb 2015 and has had 3 ongoing funded studies; an NIMH Ph2 Rapid Antidepressant Response in TRD, an NIMH Ph2 FAST-MAS (Anhedonia in Depression and Anxiety), and a Cocaine Dependence POC study. CERC plans to conduct a randomized, placebo-controlled double blind cross-over human laboratory Phase 2 study to evaluate the effects of 5 mg and 10 mg of CERC-501 on tobacco withdrawal and reinstatement and assess craving,

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mood and anxiety during 18 hours of abstinence in approximately 86 cigarette smokers who currently smoke at least 15 cigarettes per day and topline results are expected 1H16.

Figure 8: Competition

	ALKS-5461 (Alkermes)	CERC-501
Mechanism of Action	"Functional" Kappa antagonist	**Selective Kappa antagonist**
Description	Fixed combination of buprenorphine and samidorphan	**Single molecule**
Potential Indication	MDD with inadequate response to standard therapies	**Co-occuring disorders**
Phase of Development	Ph3	**Ph2**
Route and Frequency of Administration	Oral, QD	**Oral, QD**

Source: Company presentation

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CERC-406 for Cognitive Impairment Symptoms

CERC-406 is CERC's preclinical lead candidate from their catechol-O-methyl transferase inhibitor (COMTi) platform and there are no FDA approved treatments for residual cognitive impairment symptoms of MDD. CERC-406 is a non-nitro-catechol, brain penetrant, membrane-bound COMT inhibitor without the potential toxicity of earlier generation COMT inhibitors. By working on memory and executive function impairment has the potential to target an underserved patient sub segments and could be applicable across multiple diseases such as depression, ADHD, and schizophrenia. To date, mechanistic data with tolcapone as the probe for addiction, depression and schizophrenia has shown improvement in executive function for various indications and in healthy volunteers.

CERC plans to develop CERC-406 as an oral adjunctive medication for patients with residual cognitive impairment symptoms suffering from MDD. Depression is one of the most common serious medical and psychiatric disorders, with greater than 150 million adults worldwide suffering from MDD at any given time (WHO, 2003). As stated previously, more than 16 million adults in the United States, which represents approximately 6.7% of its entire adult population, will suffer from MDD in a 12-month period (U.S. National Comorbidity Survey Replication, 2007). It was predicted that by 2020 MDD would be the second leading cause of disability worldwide (WHO, 2012). It is also indicated that cognitive dysfunction is an important mediator of disability in MDD (Canadian Journal of Psychiatry, 2014).

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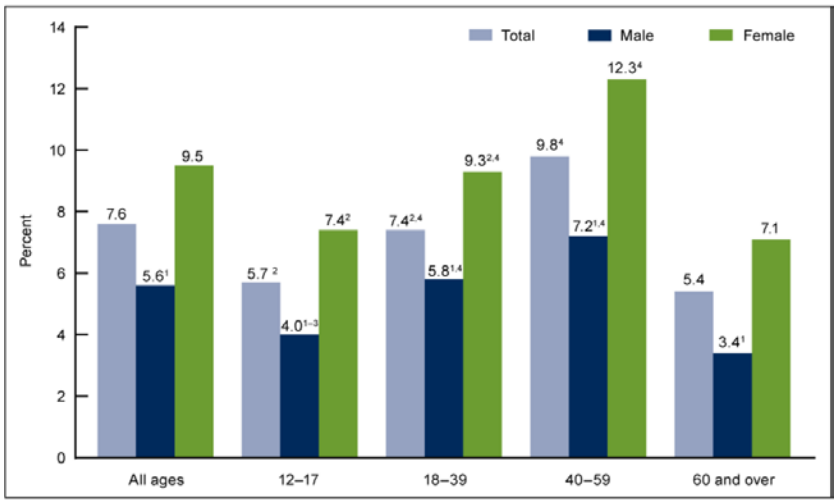
Figure 9: Percentage of persons 12 or older with depression in the US 2009-2012



[1]Males have significantly lower rates than females overall and in every age group.
[2]Significantly different from 40–59. [3]Significantly different from 18–39. [4]Significantly different from 60 and over.
NOTES: Depression is defined as having moderate to severe depressive symptoms. Access data table for Figure 1 at: http://www.cdc.gov/nchs/data/databriefs/db172_table.pdf#1.

Source:CDC/NCHS. National Health and Nutrition Examination Survey 2009-2012

Self-perceived cognitive impairment has always been recognized as a clinical manifestation of MDD. Cognitive domains that are measurably impaired in MDD include attention, memory, processing speed and executive function. Up to 50% of patients with MDD exhibit measureable cognitive deficits (Canadian Journal of Psychiatry, 2014). Deficits in attention and executive function may persist even after remission. Cognitive dysfunction and functional impairments are two of the most common residual complaints among patients with MDD who achieve symptomatic remission. Cognitive dysfunction may represent a dimension of MDD that is independent of mood symptoms. Although standard antidepressants may improve cognitive deficits in MDD, these effects may be limited in magnitude. Also, there is preliminary evidence indicating that cognitive deficits in patients with MDD may predict the failure to respond to antidepressants (Psychiatric Times, 2009). CERC believes that there might then be a subgroup of patients who require additional treatment alternatives.

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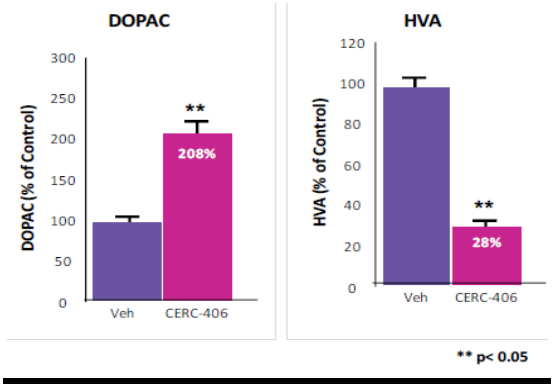
COMT Inhibitor Platform for Executive Function

Their catechol-O-methyl transferase inhibitor (COMTi) platform is comprised of a new generation of compounds with selectivity for membrane-bound COMT, the dominant form of COMT found within the central nervous system. CERC's COMT inhibitors could selectively increase dopamine levels in the prefrontal cortex, thereby improving executive function. CERC's development efforts are focused on a new generation of potent inhibitors that could avoid off-target toxicity and side effects seen with the previous generation of inhibitors, such as liver toxicity observed in tolcapone and diarrhea observed with entacapone and tolcapone. In cerebrospinal fluid (CSF), the inhibition of COMT leads to an increase in the amounts of dihydroxyphenylacetic (DOPAC), and a decrease in the amounts of homovanillic acid (HVA). CERC plans to use this biomarker strategy and combining it with a pharmacogenomic approach to develop their COMT inhibitor as an hypothesis-driven, biology-based, genotype-specific, and targeted treatments of the impairment of executive function.

Figure 10: Preclinical CSF dopamine biomarkers after oral dosing in rate



Source: Center for Disease Control

CERC's compounds are intended to have higher levels of penetration and selectivity for brain COMT, which may lead to higher efficacy with lower administered doses. CERC's COMTi platform includes compounds with varying degrees of selectivity of peripheral versus brain COMT inhibition, including some that work on both peripheral and brain COMT, and some that work primarily on brain COMT. This could provide options for developing different compounds for different disease states. For example a COMTi for Parkinson's disease may need to provide both central and peripheral inhibition, in order to benefit both the movement impairments of Parkinson's disease and the cognitive symptoms of the disease.

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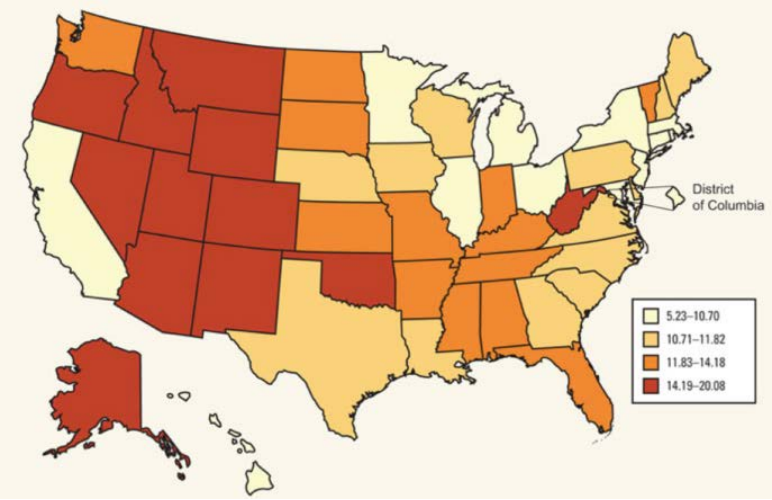
Background on Depression and Suicide

Depression is one of the most common serious medical and psychiatric disorders, with greater than 150 million adults worldwide suffering from major depressive disorder (MDD) at any given time (WHO, 2003). More than 16 million adults in the United States, which represents approximately 6.7% of its entire adult population, will suffer from MDD in a 12-month period (U.S. National Comorbidity Survey Replication, 2007). Over time, the understanding of psychiatric and neurological disorders, as well as their biological underpinnings, has evolved based on a combination of clinical and pre-clinical research.

Figure 11: 2000-2006 US Suicide Rate per 100,000 population



Source: Center for Disease Control

Over the past 50 years, many depression therapies and hypotheses primarily have been based on changing the levels of monoamine neurotransmitters, such as serotonin, norepinephrine and dopamine, in the brain. Manipulating these neurotransmitters impacts mood, but monoamine antidepressants are slow in onset, requiring multiple weeks for patients to obtain a response and patients may suffer from sexual dysfunction and other side effects like weight gain, diabetes, EPS and somnolence from such treatment. Numerous studies have shown that many patients do not respond to their initial antidepressant therapy. For example, studies have showed that ~50% of patients fail to respond (achieving a 50% reduction in symptoms), and ~60% of patients fail to remit with currently available first-line antidepressant treatments (STAR-D Study, 2006). As such, physicians commonly will switch patients' antidepressants to manage depression, and patients may require two or three courses of treatment, before achieving satisfactory relief. The depression may persist following a course of treatment and additional medications may need to be used adjunctively. These adjunctive agents may include atypical antipsychotics, like aripiprazole and quetiapine, or other agents such as buproprion, and lithium.

Suicide is often a grave complication associated with depression. Studies have shown that approximately 60% of severely depressed patients have expressed suicidal ideation (J.Clin. Psychiatry, 2003). Worldwide, almost 1 million lives are lost yearly due to suicide, which translates to 3,000 suicides per day (WHO, 2012). In 2012, suicide represented the second leading cause of death among 15-29 year olds globally.

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Major Risks

Exogenous events could impact our outlook. We believe pharmaceutical companies have the least control over competitive, political, and regulatory risks. Although we have incorporated competitive assumptions into our forecasts, there may be other risks beyond the scope of our analysis. Changes in the drug reimbursement system, as well as any political or regulatory amendments, may significantly influence the earnings power of these companies.

Actual clinical results and the FDA's conclusions may deviate from expectations. Many of our assumptions are based on a review of incomplete clinical trial data available in the public domain. Often, our conclusions are drawn from early stage data, which may not be reflected by pivotal studies. Furthermore, the FDA's conclusions may not coincide with our own, materially changing our revenue and earnings assumptions.

Compliance issues, product recalls, and other mandates by regulatory authorities could materially change our expectations. Regulatory compliance issues, ranging from accounting irregularities to defective manufacturing practices, could materially change our assumptions and earnings outlook. Unanticipated product recalls and labeling changes could also have adverse consequences on our earnings assumptions.

Legal risks could lead to additional liabilities and revenue loss. In addition to the expenses incurred by patent challenges, product liability and other legal suits could occur and lead to additional liabilities and revenue loss, which could substantially change our financial assumption

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Management

Blake M. Paterson, M.D. Co-Founder, President and CEO. Dr. Paterson has served as CEO, President, and a member of the Board since May 2011. Prior to joining the company Dr. Paterson founded Fells Laboratories LLC, a biotechnology company, where he served as Managing Director. Dr. Paterson has served as a part-time faculty member at the Johns Hopkins School of Medicine in the Division of Neuroanesthesia and Neurological Critical Care in the Department of Anesthesia and Critical Care Medicine. Dr. Paterson previously served as Co-founder, CEO and President for Alba Therapeutics, a clinical-stage biopharmaceutical company. He also served on Alba Therapeutics' Board. Prior to founding Alba, Dr. Paterson served in various executive positions at Eli Lilly including Director of New Product Planning for the Neurosciences portfolio where he was responsible for the strategy to create a somatic pain label for Cymbalta. Dr. Paterson completed his anesthesiology residency and fellowships in critical care medicine and cardio-thoracic anesthesia at Massachusetts General Hospital, where he also was an instructor in Anesthesia, Harvard Medical School.

Ronald Marcus, M.D, CMO and Head, Regulatory Affairs. Dr. Marcus has served as the CMO and Head of Regulatory Affairs since May 2015. He has more than 20 years of experience in the development of neuroscience drugs at both Spinifex Pharmaceuticals, or Spinifex, and Bristol-Myers Squibb. Dr. Marcus was most recently the CMO at Spinifex, where he was responsible for driving clinical development and regulatory strategy of its lead compound for neuropathic pain. Prior to that, Dr. Marcus held various positions at Bristol-Myers Squibb including Group Director of Neuroscience Strategic Unit and Executive Director of Neuroscience Global Clinical Research. In addition to the approval of over 20 NDAs and supplemental NDAs globally for Abilify, Dr. Marcus also led the successful development of Serzone, an antidepressant. Further, Dr. Marcus has authored more than 80 peer-reviewed publications. Dr. Marcus received his B.A. in Psychology from the University of Virginia. He earned his M.D. from SUNY Buffalo and completed his psychiatry residency and the National Institute of Mental Health research fellowship at Cornell University, New York Hospital-Westchester Division.

Mariam E. Morris, CFO. Ms. Morris has served as the CFO since August 2015, prior to which she was the interim CFO since May 2015. Ms. Morris was the sole proprietor of Mariam Morris CPA, a full service tax, accounting and business consulting firm, which she founded in January 2009 and operated until August 2015. Prior to that, Ms. Morris was the CFO of Sucampo Pharmaceuticals from February 2004 to July 2009. From 1991 until 2001, Ms. Morris was an auditor for PricewaterhouseCoopers.

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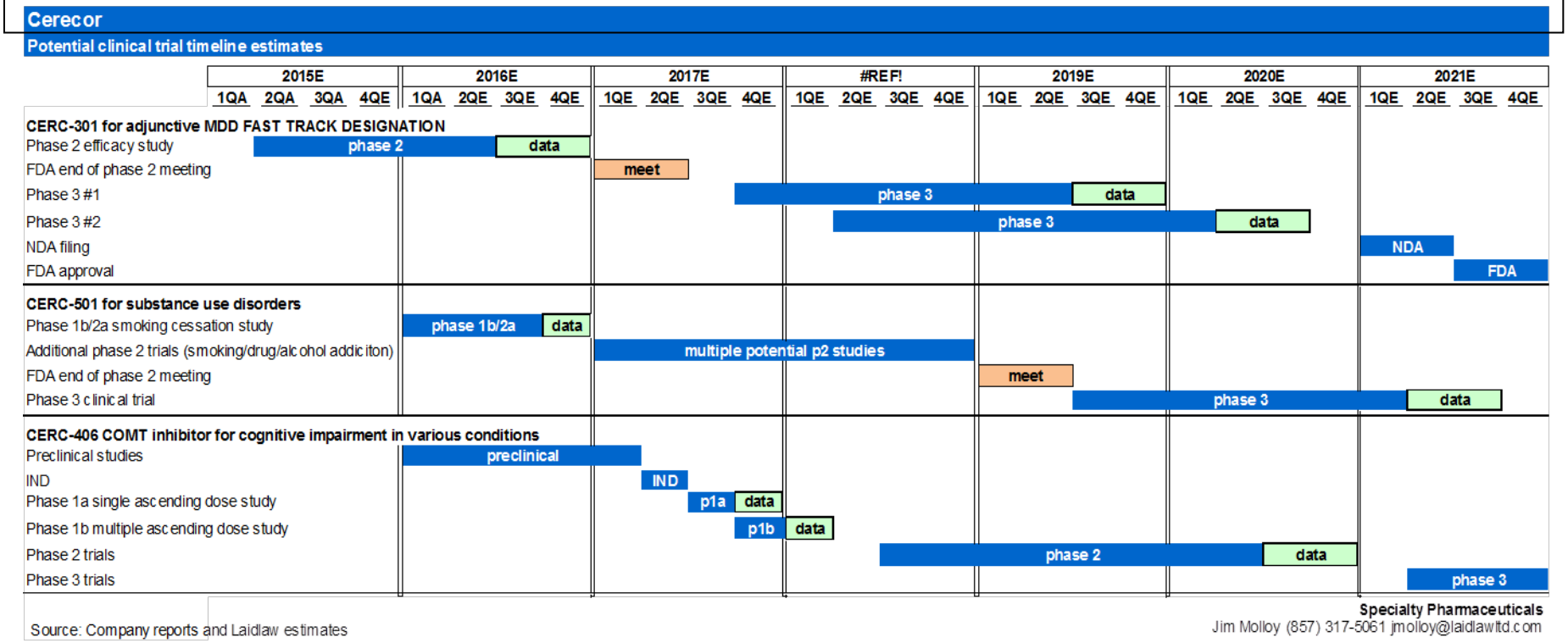
Figure 12: Potential clinical trial timelines



Source: Company reports; Laidlaw & Company estimates

Figure 13: Quarterly Income Statement

Cerecor
Quarterly income statement

($000's except per share)	2014A 1QA	2014A 2QA	2014A 3QA	2014A 4QA	2014A Year	2015E 1QA	2015E 2QA	2015E 3QA	2015E 4QE	2015E Year
R&D	2,750	2,861	4,371	2,259	12,241	1,723	1,875	1,237	1,250	6,086
SG&A	879	795	1,627	1,574	4,875	761	1,016	722	1,000	3,498
Operating income/(loss)	**(3,629)**	**(3,656)**	**(5,998)**	**(3,833)**	**(17,116)**	**(2,484)**	**(2,891)**	**(1,959)**	**(2,250)**	**(9,584)**
Interest income/(expense)	0	(795)	(190)	(222)	(1,206)	(218)	(219)	(197)	(175)	(810)
Adj-Net income/(loss)	**(3,628)**	**(4,450)**	**(6,188)**	**(4,055)**	**(18,322)**	**(2,702)**	**(3,110)**	**(2,157)**	**(2,425)**	**(10,394)**
Change in warrant value		386	348	1,532	2,266	(535)	198	1,465	1,250	2,378
GAAP net income/(loss)	**(3,628)**	**(4,064)**	**(5,840)**	**(2,523)**	**(16,056)**	**(3,238)**	**(2,913)**	**(691)**	**(1,175)**	**(8,017)**
Adj-EPS ex-non-cash	**($0.20)**	**($6.88)**	**($9.57)**	**($1.16)**	**($5.23)**	**($0.15)**	**($0.78)**	**($0.47)**	**($0.28)**	**($1.17)**
GAAP EPS as reported	**($0.20)**	**($6.28)**	**($9.03)**	**($0.72)**	**($4.58)**	**($0.18)**	**($0.73)**	**($0.15)**	**($0.14)**	**($0.90)**
Shares out (000)	17,800	647	647	3,502	3,502	18,194	4,000	4,630	8,630	8,864
Fully diluted shares (000)	85,287	60,647	42,673	46,002	157,852	204,849	189,000	158,914	169,614	180,594

Source: Company reports and Laidlaw estimates

Specialty Pharmaceuticals
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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

Figure 14: Annual Income Statement

Cerecor
Annual income statement

($000's except per share)	2014A	2015E	2016E	2017E	2018E	Comments
R&D	12,241	6,086	9,750	12,700	13,000	Trials ramp 2016-2017
SG&A	4,875	3,498	3,500	4,250	7,150	
Operating income/(loss)	**(17,116)**	**(9,584)**	**(13,250)**	**(16,950)**	**(20,150)**	
Interest income/(expense)	(1,206)	(810)	(375)	(75)	0	
Adj-Net income/(loss)	**(18,322)**	**(10,394)**	**(13,725)**	**(17,025)**	**(20,150)**	
Change in warrant value	2,266	2,378	0	0	0	
GAAP net income/(loss)	**(16,056)**	**(8,017)**	**0**	**0**	**0**	
Adj-EPS ex-non-cash	**($5.23)**	**($1.17)**	**($1.35)**	**($1.20)**	**($1.25)**	
GAAP EPS as reported	**($4.58)**	**($0.90)**				
Shares out (000)	3,502	8,864	10,143	14,168	16,130	
Fully diluted shares (000)	157,852	180,594	172,393	179,168	181,130	

Source: Company reports and Laidlaw estimates

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Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

Figure 15: Balance Sheet

Cerecor
Balance sheet

($000's except per share)	2014A	3Q15A	2015E	2016E	2017E	2018E
Current Assets						
Cash & equiv	$11,742	$3,308	$22,143	$40,484	$22,134	$21,259
Prepaid exp & other	360	105	110	125	125	125
Restricted cash	58					
Total Current Assets	**12,161**	**3,413**	**22,253**	**40,609**	**22,259**	**21,384**
Restricted cash, net current	117	117	117			
Deferred financing costs		1,880	1,880			
PP&E	39	41	50			
Other				2,500	3,850	4,500
Total Assets	**12,317**	**5,451**	**24,300**	**43,109**	**26,109**	**25,884**
Current Liabilites						
Current LTD	1,906	3,129	2,653			
Accounts payable	931	1,952	2,000			
Accrued & other	975	1,520	1,500			
Warrant liability	70	54	50			
Investor rights obligation	1,112					
Total Current Liabilites	**4,994**	**6,654**	**6,203**	**7,000**	**7,750**	**8,250**
LTD, net current	5,308	3,195	3,195			
Other LT liability		107	110	125	150	200
Total Liabilities	**10,302**	**9,957**	**9,508**	**7,125**	**7,900**	**8,450**
Shareholders' Equity						
Series A conv. preferred	10,463	10,463	10,463	10,463	10,463	10,463
Series A-1 conv. preferred	3,389	3,389	3,389	3,389	3,389	3,389
Series B conv. preferred	14,493	14,493	14,493	14,493	14,493	14,493
Total conv. pref. stock	28,346	28,346	28,345	28,345	28,345	28,345
Common shares	1	1	1	2	2	2
Additional paid in capital	16,742	17,063	37,536	58,727	57,977	77,352
Accumulated deficit	(43,073)	(49,915)	(51,090)	(51,090)	(68,115)	(88,265)
Total SE (deficit)	**(26,331)**	**(32,851)**	**(13,552)**	**7,639**	**(10,136)**	**(10,911)**
Total liabilities & SE	**12,317**	**5,451**	**24,300**	**43,109**	**26,109**	**25,884**

Source: Bloomberg LP; Company reports; Laidlaw & Company estimate

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Figure 16: Cash flow Statement

Cerecor
Statement of cash flows

($000's except per share)	2014A	3Q15A	2015E	2016E	2017E	2018E
Operating Cash Flow						
Net Income/Loss	(16,056)	(6,842)	(10,394)	(13,725)	(17,025)	(20,150)
Depreciation	29	17	20	25	50	75
Loss on asset sale	18					
Stock comp expense	1,087	321	350	400	425	450
Write off def offering costs	1,064					
non-cash int expense	989	205	300	300	300	300
Change in warrant liability	(2,266)	(1,128)	(2,250)			
Changes in Assets & Liabilites	(383)	811	875	404	(600)	(150)
Cash from operations	**(15,518)**	**(6,615)**	**(11,099)**	**(12,596)**	**(16,850)**	**(19,475)**
Investing Activities						
PP&E	(20)	(20)	(50)	(250)	(500)	(750)
Cash from investing	**(20)**	**(20)**	**(50)**	**(250)**	**(500)**	**(750)**
Financing Activities						
Issue conv pref stock	2,250					
Issue term loan	7,390		24,050	35,381	0	20,350
Issue Series B & common	14,584					
Payments on term debt		(1,024)	(1,500)	(3,195)		
Deferred financing costs	(365)	(775)	(1,000)	(1,000)	(1,000)	(1,000)
Cash from financing	**23,859**	**(1,799)**	**21,550**	**31,186**	**(1,000)**	**19,350**
Change in cash	**8,321**	**(8,434)**	**10,401**	**18,341**	**(18,350)**	**(875)**
Cash, start of period	3,421	11,742	11,742	22,143	40,484	22,134
Cash, end of period	**11,742**	**3,308**	**22,143**	**40,484**	**22,134**	**21,259**

Source: Bloomberg LP; Company reports; Laidlaw & Company estimates

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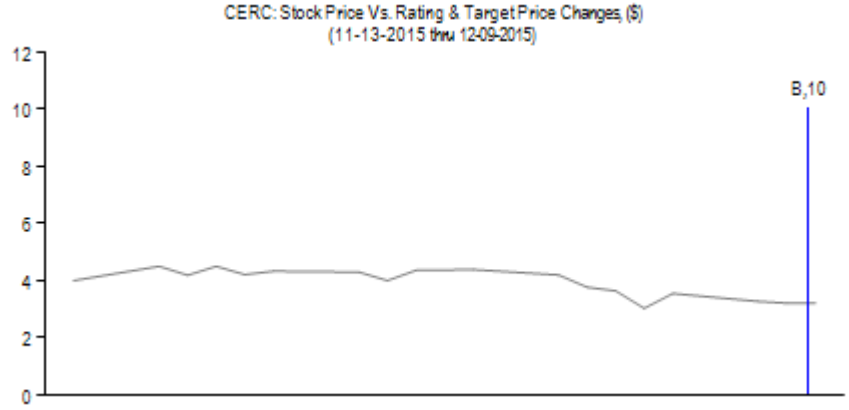
Additional information available upon request.

‡ Laidlaw & Company has received compensation from the subject company for investment banking services in the past 12 months and expects to receive or intends to seek compensation for investment banking services from the company in the next three months.

RATINGS INFORMATION

Rating and Price Target Change History



3 Year Rating Change History

Date	Rating	Closing Price ($)
12/09/2015	Buy (B)	3.21*

3 Year Price Change History

Date	Target Price ($)	Closing Price, ($)
12/09/2015	10.00	3.21*

** Previous Close12/8/2015*

Source: Laidlaw & Company **Created by: Blue-Compass.net**

Laidlaw & Company Rating System*		% of Companies Under Coverage With This Rating	% of Companies for which Laidlaw & Company has performed services for in the last 12 months	
			Investment Banking	Brokerage
Strong Buy (SB)	Expected to significantly outperform the sector over 12 months.	0.00%	0.00%	0.00%
Buy (B)	Expected to outperform the sector average over 12 months.	63.64%	27.27%	3.03%
Hold (H)	Expected returns to be in line with the sector average over 12 months.	0.00%	0.00%	0.00%
Sell (S)	Returns expected to significantly underperform the sector average over 12 months.	0.00%	0.00%	0.00%

ADDITIONAL COMPANIES MENTIONED

Pfizer (PFE – Not Rated)

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