Zacks Small-Cap Research

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Addex Therapeutics Ltd. (ADDXF-OTC)

ADXN: Initiating Coverage of Addex Therapeutics with Buy Rating & $21 Target.

Current Recommendation	**Buy**
Prior Recommendation	N/A
Date of Last Change	06/20/2012
Current Price (06/20/12)	$9.78
Target Price	**$21.00**

INITIATION

We are initiating coverage of Addex Therapeutics (Swiss-SIX:ADXN.SW, OTC:ADDXF) with a 'Buy' rating and $21.00 price target. We are optimistic on the two leading pipeline candidates, dipraglurant for the treatment of Parkinson's disease levodopa-induced dyskinesia (PD-LID) and ADX71449 for the treatment of schizophrenia and major depressive disorder and anxiety. In March 2012, the company released positive data from a phase 2a trial with dipraglurant demonstrating safety, tolerability and encouraging signs of efficacy in PDLID. We expect a partnership for phase 2/3 in the coming year. Data from a phase 2a program with ADX71149, being conducted by Johnson & Johnson, is expected before the end of the year. Our DCF model shows the shares are meaningfully undervalued today.

SUMMARY DATA

52-Week High	**$9.78**
52-Week Low	**$9.78**
One-Year Return (%)	**N/A**
Beta	**N/A**
Average Daily Volume (sh)	**0**
Shares Outstanding (mil)	**8**
Market Capitalization ($mil)	**$77**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**N/A**
Insider Ownership (%)	**N/A**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2012 Estimate	**N/A**
P/E using 2013 Estimate	**N/A**

Risk Level	**Above Average**
Type of Stock	**Small-Growth**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue
(In millions of CHF)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2011		3.2 A		0.6 A	3.7 A
2012		1.4 E		5.4 E	6.8 E
2013					5.9 E
2014					10.9 E

Earnings per Share
(Reported EPS in CHF)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2011		-2.07 A		-2.12 A	-4.19 A
2012		-1.61 E		-0.68 E	-2.27 E
2013					-2.39 E
2014					-1.77 E

WHAT'S NEW

Initiating Coverage

We are initiating coverage of Addex Therapeutics (Swiss-SIX:ADXN, OTC:ADDXF) with a 'Buy' rating and $21.00 price target. Despite limited volume on U.S. OTC tracking stock ADDXF and the inability of some U.S. investors to purchase shares on the SIX, we are initiating coverage of Addex Therapeutics because we believe the company's discovery platform, focused on positive and negative allosteric modulators, offers potential significant advantages in small molecule drug development. We believe the two leading pipeline candidates, dipraglurant and ADX71149, offer blockbuster potential for their respective indications.

Addex Therapeutics is currently seeking a development and commercialization partner for dipraglurant. The drug is an oral negative allosteric modulator (NAM) of the metabotropic glutamate receptor 5 (mGluR5), for the treatment of Parkinson's disease levodopa-induced dyskinesia (PD-LID). LID is a major side-effect of Levodopa use, and a significant unmet medical need. Phase 2a clinical data on dipraglurant, reported in March 2012, demonstrated encouraging signs of efficacy, safety, and tolerability. Results showed that patients taking dipraglurant had a meaningful and statistically significant reduction in abnormal involuntary movement as soon as one day after starting dipraglurant therapy. The data was also suggestive of an increased in "on" time – the time in which a Parkinson's patient is treated with Levodopa without experiencing dyskinesia – and a reduction in "off" time – the time after which L-DOPA has left the body prior to the next Levodopa dose. We expect a partnership for the next stage in clinical development sometime over the next year.

In January 2005, Addex Therapeutics formed a research collaboration agreement with Johnson & Johnson to discover, develop, and commercialize novel allosteric modulator compounds for the treatment of anxiety, depression, schizophrenia, and Alzheimer's disease. The first of these compounds, ADX71149, has entered clinical testing and is current in a phase 2a for the treatment of schizophrenia. To date, Addex has received a total of €10.2 million in upfront and milestone payments on ADX71149, with the potential to receive an additional €109 million in future pre-launch milestones and potential low double-digit royalties on worldwide sales once commercialized.

The potential therapeutic profile of ADX71149 is intriguing. Despite significant generic competition from atypical antipsychotics, we believe an enormous market opportunity exists for ADX71149. Atypical antipsychotics carry significant side effects, including weight gain, hyperprolactinemia, increases in blood glucose, increases in serum cholesterol and triglycerides, and increased risk of cataracts, which all lead to high discontinuation rates. Additionally, atypical antipsychotics have limited efficacy in addressing the negative symptoms of schizophrenia, including social withdrawal, apathy, alogia, anhedonia, and avolition.

Addex has designed ADX71149 to address both of these limitations found with atypical antipsychotics. With over 5 million addressable schizophrenics between North America and Europe, we see ADX71149 as a potential blockbuster drug if the theoretical profile holds. For the purpose of our financial model, we have chosen a more conservative approach, modeling ADX71149 as having peak sales around $500 million. Data from the ongoing phase 2a program is expected in the next few months. J&J has also progressed ADX71149 into a phase 2a clinical trial looking to study the drug in patients with anxiety in major depressive disorder. Conventional selective serotonin reuptake inhibitors (SSRIs) for the treatment of depression carry significant side effect, one of which is anxiety. We believe there is meaningful clinical need for a drug that can treat both major depression and anxiety without exhibiting some of the debilitating side effects seen with SSRI and SNRI drugs.

DCF Target

We have built a discounted cash flow (DCF) model to value the shares of Addex Therapeutics. Our model is built in Swiss francs and assumes a launch of dipraglurant in 2017 by a yet unfound partner and ADX71149 by J&J also in 2017. We expect several additional candidates to enter the clinic over the next several years. Our model calculates fair value at CHF 217 million, which equates to a U.S. OTC price of $21.00 per share. We have posted our model in the back of the report.

INVESTMENT OVERVIEW

Addex Therapeutics, founded in 2002, is located in Geneva, Switzerland and trades on the SIX Swiss Stock Exchange under the ticker symbol ADXN (ADXN.SW). There is a U.S. tracking vehicle, ADDXF, that trades on the OTC Markets. Despite limited volume on ADDXF and the inability of some U.S. investors to purchase shares on the SIX, we are initiating coverage of Addex Therapeutics because we believe the company's discovery platform offers potential significant advantages in small molecule drug development, and the two leading pipeline candidates, dipraglurant and ADX71149 offer blockbuster potential for their respective indications.

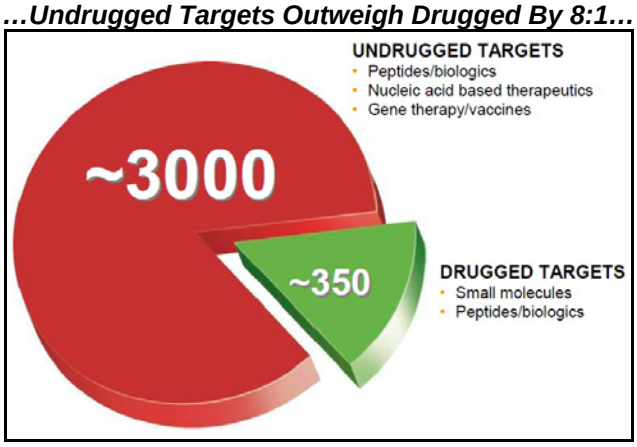
Addex Therapeutics is currently seeking a development and commercialization partner for dipraglurant. Our report is designed to familiarize investors with dipraglurant in anticipation of a deal later in 2012, as we expect substantial interest from U.S.-based pharmaceutical and biotechnology companies for the drug. We also expect a significant participation from U.S. clinical sites in the company's pivotal registration program for dipraglurant for the treatment of Parkinson's Disease – Levodopa-Induced Dyskinesia (PD-LID).

ADX71149 is already partnered with Janssen Pharmaceuticals, a division of Johnson & Johnson, Inc. Addex has also had a previous license and collaboration agreement with Merck Sharp & Dohme Research Ltd. And, according to the most recent annual report, 39.25% of current Addex Therapeutics shareholders are located in the U.S. (Switzerland is second with 23.33%).

Addex Technology

Addex Therapeutics is engaged in the discovery and development of an emerging class of oral small molecule drug candidates, called allosteric modulators. Allosteric modulators are highly selective for their intended target and confer significant therapeutic advantages over conventional orthosteric small molecule or biological drugs. The company uses its proprietary discovery platform to address receptors and protein targets that have been previously "undruggable" by conventional drug discovery methods, including G-Protein Coupled Receptors (GPCRs), receptor tyrosine kinases (RTKs), and cytokine receptors such as the TNF receptor. Management believes these targets are attractive for modulation of important diseases with unmet medical needs, but have remained inaccessible to small molecule drug discovery, creating a significant opportunity for Addex Therapeutics.

…Undrugged Targets Outweigh Drugged By 8:1…



We believe Allosteric modulators offer potential to target previously untargeted biologic pathways due to inefficiency in selectivity, safety or tolerability, immunogenicity, or bioavailability. For example, conventional small molecules may offer modest selectivity and adequate safety / tolerability, but lack meaningful efficacy and a differentiated pharmacology. Biologics and peptides, through enhanced selectivity may offer improved efficacy, but could lack the necessary safety or bioavailability for an effective drug. Gene therapies and nucleic acid-based therapies offer some of the best selectivity and pharmacology, but may have poor bioavailability and safety. Biologics and gene therapy drugs are also far more expensive to manufacture than small molecules and allosteric modulators, and thus may be relegated to 2nd or 3rd-line treatment due to their high cost and lower-tier insurance coverage. The ideal drug candidate is one that provides high selectivity for the target, with a differentiated pharmacologic and efficacy profile – perhaps a new mechanism of action – while also offering superior safety, tolerability, and bioavailability.

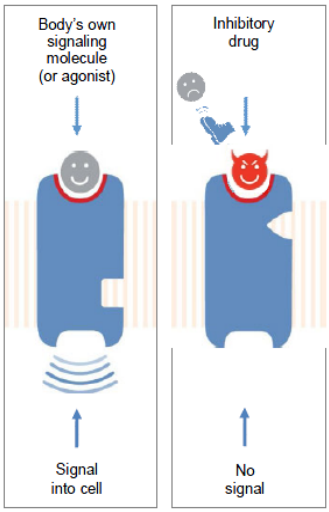
…Allosteric Modulators – The Total Package…

	Small Molecules	Biologics / Peptides	Gene & NA Therapies	Allosteric Modulators
Selectivity	✓	✓	✓✓	✓✓
Differentiated Pharmacology	X	-	✓✓	✓✓
Safety / Tolerability	✓	✓	✓	✓
Oral Bioavailability	✓✓	X	X	✓✓
Crosses Blood Brain Barrier	✓✓	X	X	✓✓
No Immunogenicity	✓✓	X	-	✓✓
Low Cost of Goods	✓✓	X	-	✓

…Explaining Allosteric Modulators…

In a conventional biologic pathway, the body's own signaling molecule may turn on (agonist) or turn off (antagonist) a signal transduction by binding to a specific receptor protein on the surface of the cell membrane. These signaling molecules may be neurotransmitters, chemokines, hormones, growth factors, cytokines, etc... In the simplest terms, the binding of these signaling factors to the receptor site elicits a physiological response. This physiological response could be a gene activation, cell proliferation or apoptosis, metabolic alteration, or the triggering another cellular signal transduction cascade.

Pharmaceutical and biotechnology companies may seek to develop drugs – small molecules or biologics / peptides – that bind to the targeted receptor to inhibit or enhance this signal transduction. The more specific the molecule is for the binding site, the lower the potential for off-site binding and unwanted side-effects. Drug candidates can also look to bind to the extracellular signal itself, altering its binding potential on the cell membrane receptor site.



An inhibitory drug competes with the body's own signal to mute the secondary response. Another drug may look to mimic the body's own signaling molecule to enhance the secondary response. Generally speaking, conventional small molecule drugs, when binding to the receptor site, work like a light switch with respect to the secondary signal transduction – they either turn it "on" or turn if "off".

Dose-ranging studies are conducted to find the proper concentration of the drug necessary to turn on or turn off the proper number of cells in an effort to find the right balance between efficacy and unwanted side-effects. Primarily, these drugs work in competition with the body's own cellular cascade to affect a therapeutic benefit.

Allosteric modulators operate under an entirely different model. Instead of working in competition with the body's own signaling molecules, they work in concert (symmetry model). And instead of acting like a light switch with respect to turning on or turning off the signal, they act more like a dimmer switch – raising or lowering the signal. Allosteric modulators do not bind to the receptor site. They are an effector molecule that binds to an allosteric (other non-active) site on the cell membrane. The body's own signal ligand is free to bind without competition to the target site. This creates a number of benefits with respect to drug development:

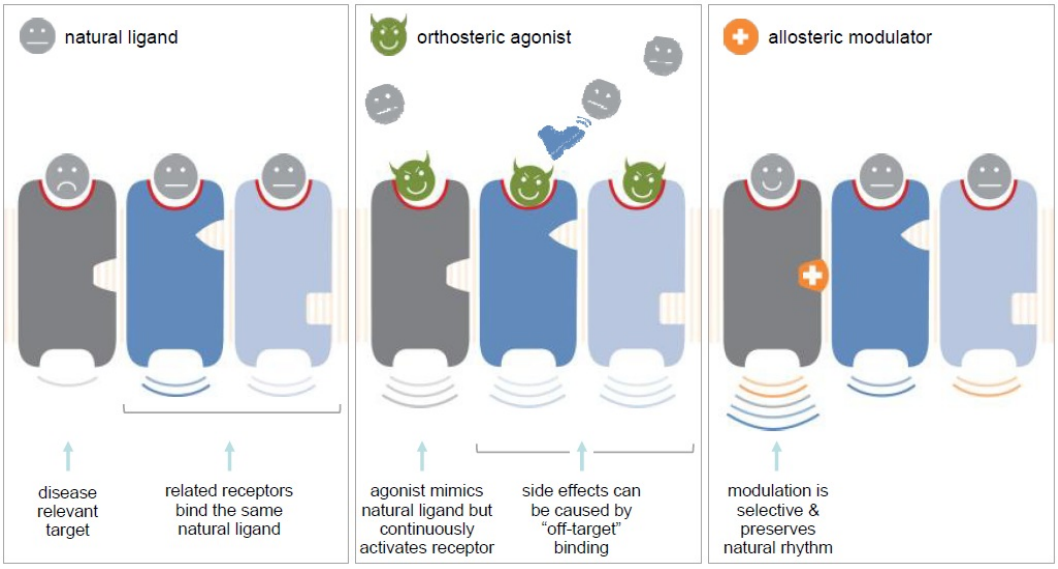
- ✓ It allows for concomitant dosing of allosteric molecules with existing small molecules to target one pathway (e.g. lowering the dose and enhancing the signal of an existing small molecule). An existing drug therapy may have a small therapeutic window based on poor tolerability at an effective dose. Using an allosteric modulator may allow for the same level of efficacy with a lower dose of the small molecule, improving tolerability.

- ✓ Similarly, when large ligand binding sites (peptide receptors) are needed to elicit a therapeutic response, small molecules are ineffective. In a disease state, the natural peptide may be under-produced and thus the signal muted. Allosteric modulators can be used to amplify the body's muted response due to the underlying disease.

- ✓ By keeping the body's own natural ligand in place as the signaling molecule, it reduces the effects of "off-target" orthosteric agonists (or antagonists) binding by a conventional small molecule drug, potentially reducing side-effects in improving tolerability.

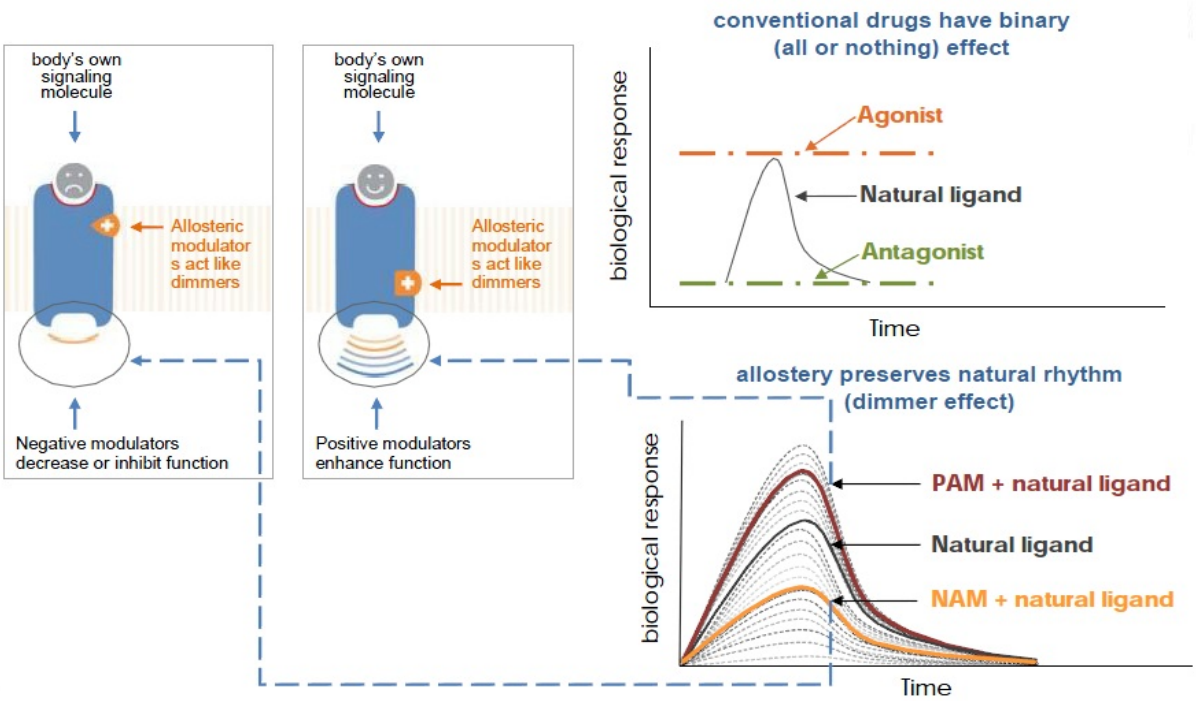
✓ While one extracellular signaling molecule (neurotransmitter, hormones, cytokines, etc…) may bind to multiple target sites eliciting different physiological responses, allosteric modulators are highly specific for the allosteric site on the cell membrane. Highly specific binding of the allosteric modulator allows for greater sensitivity on the target response, while limiting impact on other pathways in a related family.

…Allosteric Modulators Reduce "Off-Target" Binding…



There are two types of allosteric modulators: Negative Allosteric Modulators (NAMs) work to dim the signal to the cell by binding to an effector site on the cell membrane, and Positive Allosteric Modulators (PAMs) work to enhance the signal to the cell by binding to an effector site (often a different site than the NAM) on the cell membrane.

…Allosteric Modulators – A Better Light Switch…



Addex's two lead products candidates are allosteric modulators. Dipraglurant (ADX48621) is a mGluR5 negative allosteric modulator (NAM) being developed to treat Parkinson's disease levodopa-induced dyskinesia (PD-LID). ADX71149 is a mGluR2 positive allosteric modulator (PAM) being developed to treat schizophrenia and anxiety by Addex's partner, Janssen Pharmaceuticals Inc., a division of Johnson & Johnson company.

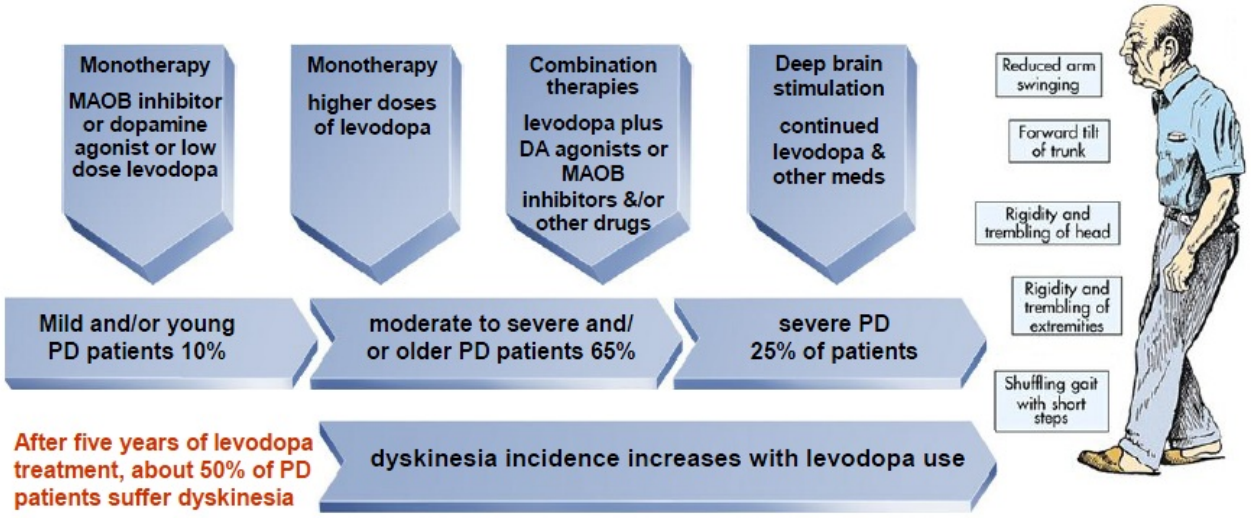
Parkinson's Disease

Parkinson's disease (PD) is a neurodegenerative brain disorder that results from the death of dopamine-generating cells in the substantia nigra region of the midbrain. PD is also characterized by the accumulation of a protein called alpha-synuclein into inclusions called Lewy bodies in neurons. The cause of PD is generally idiopathic, although some atypical cases have a genetic origin. The disease is named after the English doctor James Parkinson, who published the first detailed description in *An Essay on the Shaking Palsy* in 1817.

PD patients often exhibit marked reduction in motor control and an increase in parkinsonism (tremors, hypokinesia, rigidity, bradykinesia, and postural instability). However, as the disease progresses, patients often exhibit non-motor symptoms that include autonomic dysfunction, neuropsychiatric problems (mood, cognition, behavior or thought alterations, psychosis), and sensory and sleep difficulties. Parkinson's disease psychosis (PDP) is common in nearly 50% of PD patients a decade after initial diagnosis. Anxiety and depression are common co-morbidities. Initial signs of PD include shaking, loss of smell, difficulty writing, trouble sleeping, constipation, and poor posture. Diagnosis of a typical case is mainly based on symptoms, with tests such as neuroimaging used for confirmation.

…Treatment Options…

There is no cure for PD. Instead, physicians attempt to manage the symptoms of the disease through a multidisciplinary approach that may include pharmacological, social, and surgical options. The most common pharmaceutical treatment options are those with look to increase the level of dopamine in the brain. These include dopamine replacement therapies (DRT) combined with dopa decarboxylase inhibitors, dopamine agonists, and MAO-B inhibitors. The treatment option is often tailored specifically for the patient based on the stage and severity of the disease and the balance between good symptom control and side-effects resulting from enhancement of dopaminergic function.



The most commonly used DRT therapy is Levodopa. It has been available for over 30 years. Levodopa (L-DOPA) is converted into dopamine in the dopaminergic neurons by dopa decarboxylase. The administration of levodopa temporarily diminishes the motor symptoms associated with the lack dopamine in the substantia nigra. Unfortunately, only about 5-10% of L-DOPA crosses the blood-brain barrier. The remainder is often metabolized to dopamine elsewhere, causing a variety of side effects including nausea, dyskinesias and joint stiffness. Carbidopa, a dopa decarboxylase inhibitor, is commonly doses with Levodopa to prevent L-DOPA metabolism before it reaches the blood-brain barrier. In fact, co-formulations of Levodopa/Carbidopa (Sinemet) are available.

Despite these co-formulations, Levodopa carries significant risk of side-effects, including dyskinesia. As a result, despite its effectiveness in reducing motor symptoms associated with Parkinson's disease, physicians often attempt to delay Levodopa therapy until the disease progresses to a more moderate-to-severe stage. Most early-stage PD patients start out on MAO-B inhibitors and / or dopamine agonists, or low-dose Levodopa. However, PD is a progressive and degenerative disease, and patients typically progress to the point where starting Levodopa or increasing the Levodopa dose is necessary in five years after initial diagnosis. A decade later, almost all PD patients require high doses of Levodopa therapy, as well as surgical options including deep brain stimulation (DBS). As the dose and use of Levodopa increases, the incidence of dyskinesia also increases.

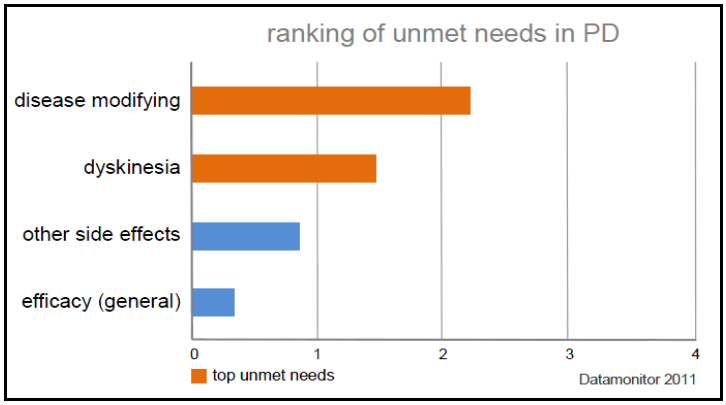
Levodopa also has a relatively short half-life, requiring dosing averaging three to four times a day. Peak plasma concentrations of Levodopa occur 60 to 90 minutes after dosing. Unfortunately, this is also when peak side effects such as dyskinesia occur. The hefty dosing requirement of Levodopa creates compliance issues, especially at night when patients may sleep through their dose schedule – dosing every six hours. The peaks and troughs associated with Levodopa create significant "on" and "off" treatment times for PD patients.

On times are when the drug is in their system and they may be experiencing dyskinesia, and off times are when the Levodopa has left their system and the patient may awake in a frozen or rigid state. Slow or controlled release intravenous formulations exists in an attempt to smooth the peaks and troughs associated with frequent Levodopa/Carbidopa dosing, but these formulations have not proven to be more effective in relieving parkinsonism, or reducing dyskinesia. In fact, a continuous infusion of Levodopa may bring upon symptoms of dyskinesia at a greater rate than quick-release / immediate release oral formulations.

Levodopa-Induced Dyskinesia

Levodopa-Induced Dyskinesia (LID) is a major side-effect of Levodopa use. LID is characterized by hyperkinetic movements, including chorea (abnormal involuntary movement), dystonia (sustained muscle contraction, abnormal posture), and athetosis (involuntary convoluted movements). It is most common at times of peak L-DOPA plasma concentrations (peak-dose dyskinesia), although it may also occur when plasma concentrations of L-DOPA rise and fall (diphasic dyskinesia) or during off-time (off-period dystonia).

In the U.S., there are an estimated 500,000 to 1 million patients suffering from Parkinson's disease. There are no approved treatment options for PD-LID. Approximately 50% of PD patients will experience LID after 4 to 6 years on L-DOPA therapy. The number rises to 90% after 10 to 15 years on L-DOPA therapy.



It is a significant problem for patients and physicians seeking treatment for PD. In fact, a survey of key opinion leaders (KOLs) in the Parkinson's treatment space showed that dyskinesia is the most important unmet medical need in the treatment of PD after a disease modifying agent (Datamonitor 2011).

The most common treatment for LID is to reduce dose on L-DOPA. However, reducing dose on L-DOPA causes increased parkinsonism and worsening motor performance. Therefore, once established, LID is difficult to treat.

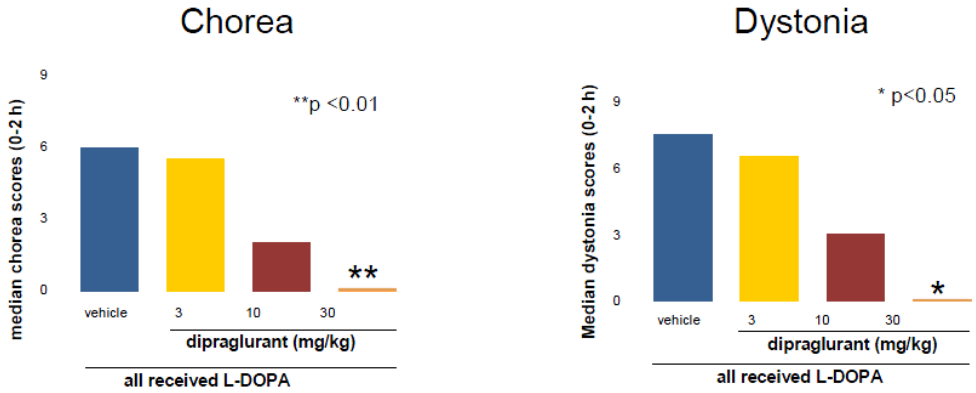
Dipraglurant

Addex Therapeutics is developing dipraglurant, an oral negative allosteric modulator (NAM) of the metabotropic glutamate receptor 5 (mGluR5), for the treatment of Parkinson's disease levodopa-induced dyskinesia (PD-LID). During the neurodegenerative process of PD, loss of striatal dopaminergic neurons results in an increase in gluatamatergic output from the substantia nigra. It has been shown that mGluR5 are abundant in the striatum and implicated in the excess glutamate activity observed in patients with PD. In fact, mGluR5 are the only mGlu receptor type involved in substantia nigra neuronal depolarization. Addex Therapeutics has demonstrated that blockade of mGluR5 has anti-PD and anti-dyskinetic effects in a variety of animal models as well as early trials in patients.

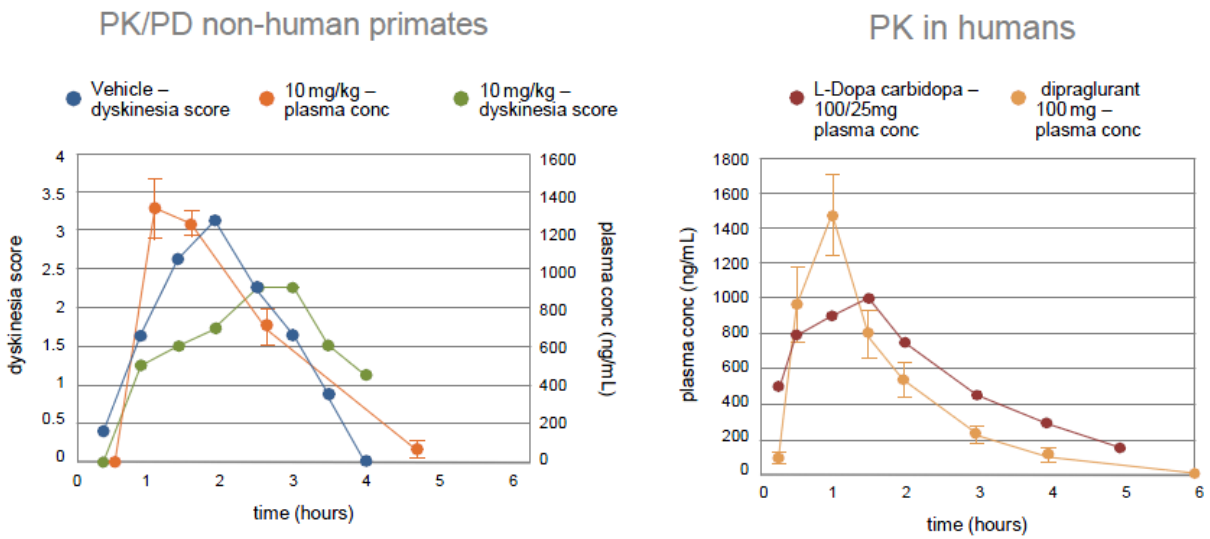
…Preclinical Data…

Addex, through a partnership with Motac Neurosciences, has conducted significant proof-of-concept work with dipraglurant (and other mGluR5 NAMs) in animal models. Motac is a private, UK-based company that provides highly-specialized services for the pharmaceutical and biotechnology industry to support the discovery and development of therapeutics for neurological and psychiatric disorders. Motac personnel have extensive expertise in neurodegenerative conditions, particularly Parkinson's disease and other movement disorders. Motac also has an intellectual property portfolio protecting a number of novel therapeutic targets for movement disorders, which it is developing through strategic alliances and partnerships.

In a preclinical model of Parkinson's disease levodopa-induced dyskinesia (PD-LID), designed to be predictive of efficacy in humans, dipraglurant (immediate release 3, 10, and 30 mg/kg) or placebo was administered 30 minutes prior to Levodopa in a 4-way crossover protocol. Behavior was recorded upon Levodopa administration and video was reviewed by trained observers. Dyskinesia (chorea and dystonia) as well as Parkinson's disability were scored during the first 10 minutes of every half-hour period over two hours. Results (presented below) show a marked and statistically significant reduction in dyskinesia symptoms on the 10 and 30 mg/kg dose.



Pharmacokinetic (PK) and Pharmacodynamic (PD) data demonstrated predictive efficacy in humans with LID at an effective concentrations of approximately 1000 ng/ml. Since LID occurs most commonly at peak L-DOPA plasma concentration (peak-dose dyskinesia), Addex has designed an optimal formulation of dipraglurant that mirrors the clinical PK/PD of Levodopa (CMax of approximately 1500 ng/ml). Accordingly, dipraglurant offers rapid onset of action and rapid clearance to reduce unnecessary drug exposure and unwanted side-effects. We believe the immediate release (IR) formulation of dipraglurant offers the kind of dosing flexibility that physicians treating patients with PD seek given the significant variability in Levodopa dosing per patient.



…Phase I Clinical Data…

Addex Therapeutics has conducted three phase 1 clinical studies in a total of 132 healthy male and female subjects demonstrating a desired safety, tolerability, dose titration, and PK profile.

- Study 101: Single ascending dose (Part-1, n=48) and food effect study (Part-2, n=16) dosing 20, 50, 100, 250, 400, and 500 mg of dipraglurant.
- Study 102: Single (100mg) and multiple (50, 100, 200 mg) ascending dose study with immediate release (IR) formulation over 7 days.
- Study 102: Gender and food effect study of dipraglurant-IR in health male (n=15) and female (n=15) subjects aged 50 to 70 years of age.

…Phase 2a Data…

Based on the encouraging safety, tolerability and PK data from the three phase 1 programs, Addex Therapeutics moved into a phase 2a, randomized, double-blind, placebo-controlled study of dipraglurant-IR in March 2011. The trial screened 83 patients with moderate-to-severe LID at 25 clinical sites split evenly between the U.S., France, Germany, and Austria. Patients were maintained on a constant dose of Levodopa (300 – 1500 mg/day), and dosed dipraglurant-IR (or placebo) at the time of their Levodopa dosing over 4 weeks. Work from this phase 2a trial was supported by a $0.900 million grant from The Michael J. Fox Foundation (MJFF).

Data from the phase 2a study was released in March 2012. The primary endpoint of the study was safety and tolerability. A total of 76 patients were randomized between dipraglurant (n=52) and the placebo (n=24). After four weeks, 90% of the patients on dipraglurant (47 out of 52) completed the study. Two patients withdrew and three patients were removed for protocol violations.

- *Safety Tolerability Results*

The study treatment duration was 4 weeks and patients followed a dose titration regimen, receiving 50 mg doses up to three times daily in the first 2 weeks of the study until day 14; and then from day 14 to day 28, they escalated to 100 mg three times daily. Results show that both dose levels, 50 and 100 mg, were well tolerated and there were no safety concerns arising from any of the safety monitoring parameters. These include things like heart rate, blood pressure, ECG, and blood tests looking for impact on liver function. Adverse events in the study were common in both the dipraglurant and placebo arms, coming in at 88.5% and 75%, respectively. The data did show typical mGluR-type adverse events, such as vertigo, blurred vision, and a drunk feeling, but management noted that none of these were severe and did not compromise the use of the drug.

Tolerability	**Dipraglurant (n=52)**	**Placebo (n=24)**
Completers	90%	100%
Adverse Events	89%	75%
Blood Tests	“normal”	“normal”

- *Efficacy Results*

Secondary endpoints in the study were exploratory, but centered on validated clinical measures for Parkinson’s disease assessment, including the modified Abnormal Involuntary Movement Scale (modified AIMS), patient and clinician global impression of change (PGIC & CGIC), Unified Parkinson’s Disease Rating Scale (UPDRS), and the Hospital Anxiety & Depression Scale (HADS).

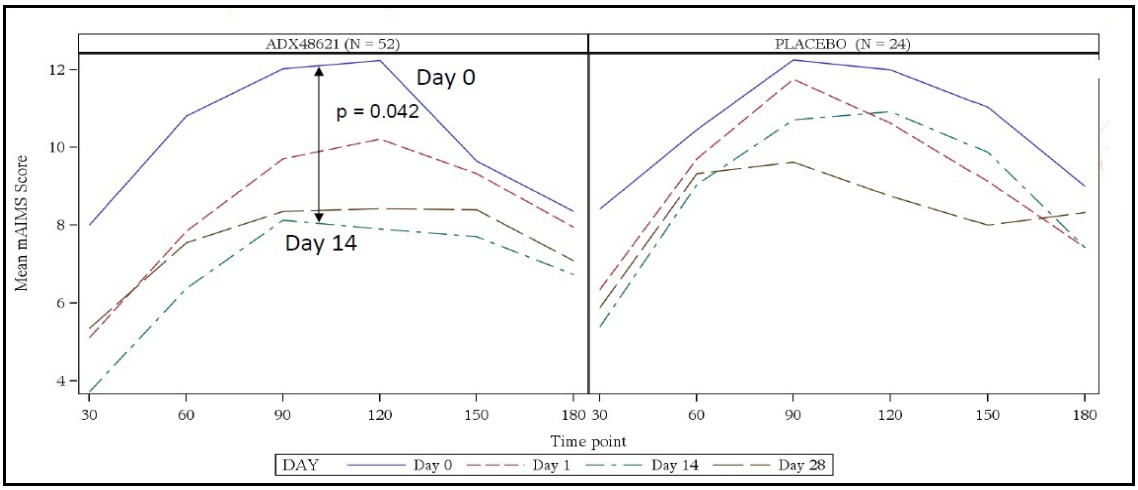
Results on the modified AIMS scale showed statistically significant improvement on days 1 and 14, with impressive and clinically relevant reductions in the dipraglurant group on all three periods tested (days 1, 14, and 28). Management will have to control the escalating placebo response in the next clinical study, as the Day 28 data just missed statistical significance.

Reduction in modified AIMS	**Dipraglurant (n=52)**	**Placebo (n=24)**	
Day 1	19.9%	4.1%	*p=0.042*
Day 14	32.3%	12.6%	*p=0.034*
Day 28	31.4%	21.5%	*n/s*

Interestingly, management did not notice a difference in the AIMS reduction trends for patient with an implanted electronic DBS (deep brain stimulation) device. DBS is a sort of pacemaker for the brain for patients with severe PD. This is encouraging and opens the door for potential dipraglurant use in this population.

Targeted reduction in Levodopa-induced dyskinesia severity over the entire 3 hour post Levodopa dose period (area under the curve) demonstrated solid results for dipraglurant at Day 14 (32.7%) and Day 28 (27.5%). The Day 14 AIMS AUC_{0-3} data was statistically significant (p=0.042) at Day 14, but missed at Day 28 due to lack of statistical powering and a meaningful reduction in the Day 28 AIMS AUC_{0-3} data for the placebo. This is something management will have to address in the next clinical trial, and remains our single biggest concern going forward.

…Modified AIMS AUC_{0-3} Data…

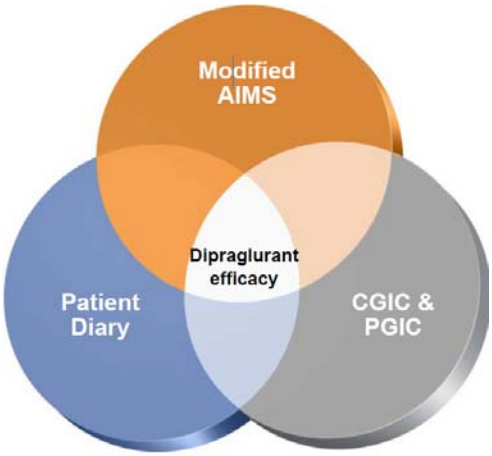


We note for the above data, patients received 50 mg TID dipraglurant on Day 1, 100 mg TID on Day 14, and 100 mg TID on Day 28. We expect that for the phase 2b program, patients will be on 100 mg TID for a much longer period of time. Phase 1 data suggests up to 500 mg per day was well-tolerated.

The phase 2a trial also included 7 patients with dystonia. The data were limited, so no statistical analyses was performed, but management noted a similar response with respect to the magnitude of improvement for the 4 patients receiving dipraglurant to the response in chorea. This opens the doors to potential further studies in patients with peak dose dystonia in the future.

Patient and Clinician Global Impression of Change (PGIC & CGIC) and patient diary data also yielded encouraging results. The data showed no increase in "off" time seen with dipraglurant use – meaning that dipraglurant had no detrimental effect on the underlying Levodopa efficacy. In fact, by week 4 of the study, the mean "off" time for the dipraglurant group actually decreased by 50 minutes compared to no change for the placebo. This is suggestive of a beneficial (symbiotic) effect of dipraglurant on parkinsonian symptoms. UPDRS (motor function scoring) remained unchanged at all treatment visits during the 4 week program, again suggestive of no detrimental effect to the drug.

…Clear Proof-of-Concept…



Similarly, an increase in "on" time effect without dyskinesia was observed for the dipraglurant group compared to the placebo in all 4 weeks of treatment. By week 4 of the study, patients in the dipraglurant group had an extra 2.3 hours per day of "on" time without dyskinesia. We find this to be highly clinically relevant, and suggestive for a potential blockbuster opportunity.

…What's Next…

The next steps for management with dipraglurant-IR for PD-LID is to partner for the registration program. Addex Therapeutics is currently seeking a partner with the expertise and capability to fully exploit dipraglurant's attractive commercial potential. This will most likely include follow-on, extended release formulations of dipraglurant for non-Parkinson's dystonia. The above phase 2a data not only clearly demonstrates proof-of-concept for dipraglurant in PD-LID, but also validates the company's research on the mGluR5 target. Management's goal is to secure a partnership within the next year.

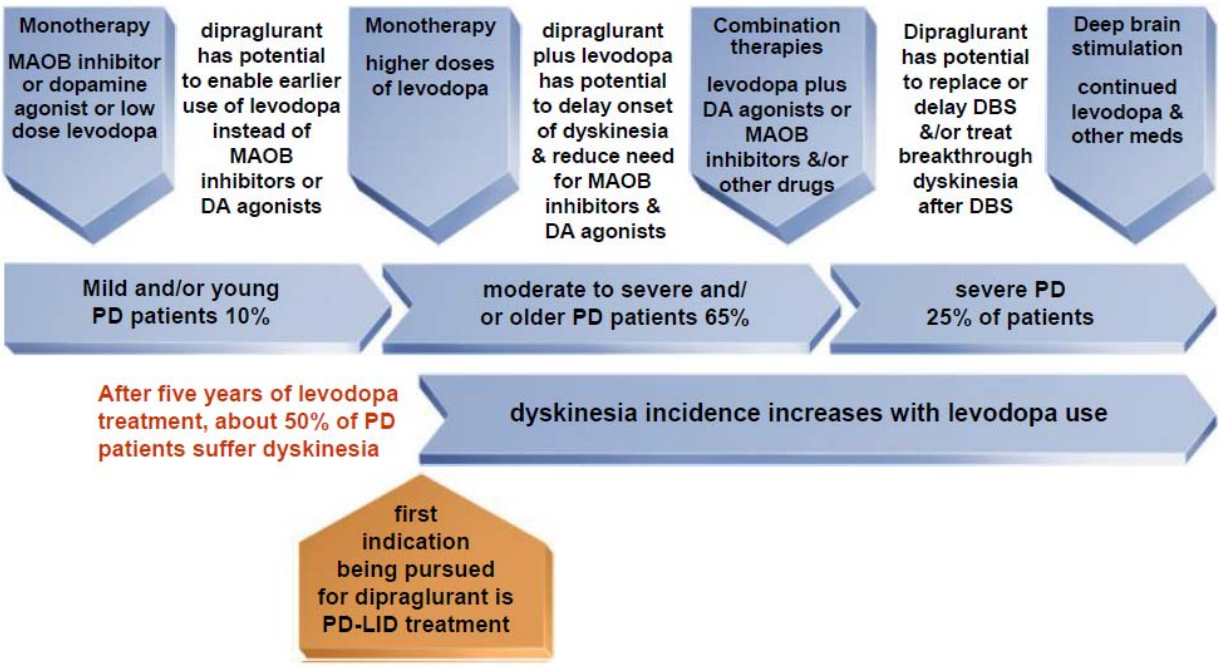
From a regulatory standpoint, Addex and its partner will need to submit two statistically significant pivotal trials to the U.S. FDA and European authority when seeking approval for dipraglurant. Additional analysis of the phase 2a program has recently completed, with data expected later this week at the Movement Disorder Society meeting. Once the final analysis has been presented, management will step up the partnering efforts. Additional funding with the MJFF is a potential as well. Our guess is that one pivotal phase 2b will commence in 2013, followed by one confirmatory phase 3 in 2015.

…Market Opportunity & Sales Forecast…

Parkinson's disease is the second most common neurodegenerative disorder after Alzheimer's disease. According to the National Institute of Neurological Disorder and Stroke, there are an estimated 500,000 people in the U.S. living with Parkinson's disease. The National Parkinson's Foundation estimates 50,000 to 60,000 new cases are diagnosed each year. The European Parkinson's Disease Association (EPDA) estimates another 500,000+ Parkinson's patients in Europe. According to an article published in The Lancet in June 2006 (5(6):525-35), *Epidemiology of Parkinson's Disease,* prevalence of Parkinson's is about 0.3% of the whole population in industrialized countries. PD is more common in the elderly and prevalence rises from 1% in those over 60 years of age to 4% of the population over 80.

Between North America and Europe, we estimate there are over 1 million addressable patients living with Parkinson's disease. Roughly 90% of these patients are considered moderate to severe in their parkinsonian symptoms, as many mild cases of Parkinson's go undiagnosed. The majority of these 900,000 moderate to severe Parkinson's patients will be on Levodopa therapy, either monotherapy or in combination with MAOB inhibitors.

Parkinson's patients and physicians feel strongly PD-LID is a high unmet medical need. After five years of Levodopa treatment, 50% of PD patients will suffer from dyskinesia. The number jumps to 90% of PD patients following 10 years for Levodopa therapy. With dipraglurant, Addex Therapeutics and its commercialization partner have an opportunity to target the vast majority of these 900,000 patients, as early as their second or third year of Levodopa therapy.



We estimate dipraglurant could cost approximately $3,000 per year in the U.S. We believe that 30% peak penetration is reasonable considering the significant nature of the problem and lack of competition from existing therapies. Peak sales for dipraglurant-IR in PD-LID are $650 million in our view.

Between North American and Europe, there are another 700,000 patients living with non-Parkinson's dystonia. The development of dipraglurant-ER opens the door to potential indications in dystonia, anxiety/depression, compulsive disorders, and other motor symptoms and movement disorders. We estimate another 1 million patient opportunity for dipraglurant-ER. This sort of follow-on / life-cycle management product could deliver peak sales between $500 million and $1 billion.

As noted above, we expect Addex Therapeutics to partner the dipraglurant molecule with a larger pharmaceutical company with the expertise and capability to fully exploit this sort of broad commercial potential. A partnership before phase 2b could bring an upfront payment in the area of $25 to $50 million (USD) and mid-double digit royalties on worldwide sales.

Schizophrenia

Schizophrenia is a complex mental disorder characterized by a breakdown in the thought process and poor emotional response and control. The most common symptoms of schizophrenia are auditory hallucinations, paranoia, delusions, or disorganized speech and thinking, and deterioration in cognition. Patients suffering from schizophrenia experience significant social dysfunction, cannot tell what is real and what is not real, and often are unable to maintain a job or hold relationships. Schizophrenia symptoms usually develop slowly over months or years, and patients may go through periods of time where the disease is well-control or uncontrollable.

Schizophrenia affects both men and women equally. It usually begins in the teen years or young adulthood, but it may also begin later in life. Late adult-onset schizophrenia is more common in women. Childhood-onset schizophrenia begins after age five. Childhood schizophrenia is rare and can be hard to tell apart from other developmental problems in childhood, such as autism. The cause of Schizophrenia is still unknown, but there is a genetic component and risk of developing schizophrenia increases dramatically (13-fold increase in risk) if a first-degree relative has or had the disease.

People with schizophrenia are likely to have additional (comorbid) conditions, including major depression and anxiety disorders. Substance abuse is common in nearly 50% of schizophrenia patients. Social problems, such as long-term unemployment, poverty and homelessness, are also common. The average life expectancy of people with the disorder is 10 to 15 years less than those without, the result of increased physical health problems and a higher suicide rate (about 5%).

Schizophrenia is often described in terms of positive and negative symptoms. Positive symptoms are those that most individuals do not normally experience but are present in people with schizophrenia. Positive symptoms generally respond well to medication. Negative symptoms are deficits of normal emotional responses or of other thought processes, and respond less well to medication. Research suggests that negative symptoms contribute more to poor quality of life, functional disability, and the burden on others than do positive symptoms. People with prominent negative symptoms often have a history of poor adjustment before the onset of illness, and response to medication is often limited.

- Positive Symptoms: Hallucinations, delusions, bizarre behavior, disordered and illogical thoughts and speech. Hallucinations and delusions may be tactile, auditory, visual, olfactory and/or gustatory in nature.

- Negative Symptoms: Social withdrawal, apathy, inability to experience pleasure, attention defect, and blunted or muted affect and emotion. These may manifest in poverty of speech (alogia), inability to experience pleasure (anhedonia), lack of desire to form relationships (asociality), and lack of motivation (avolition).

…Treatment Options…

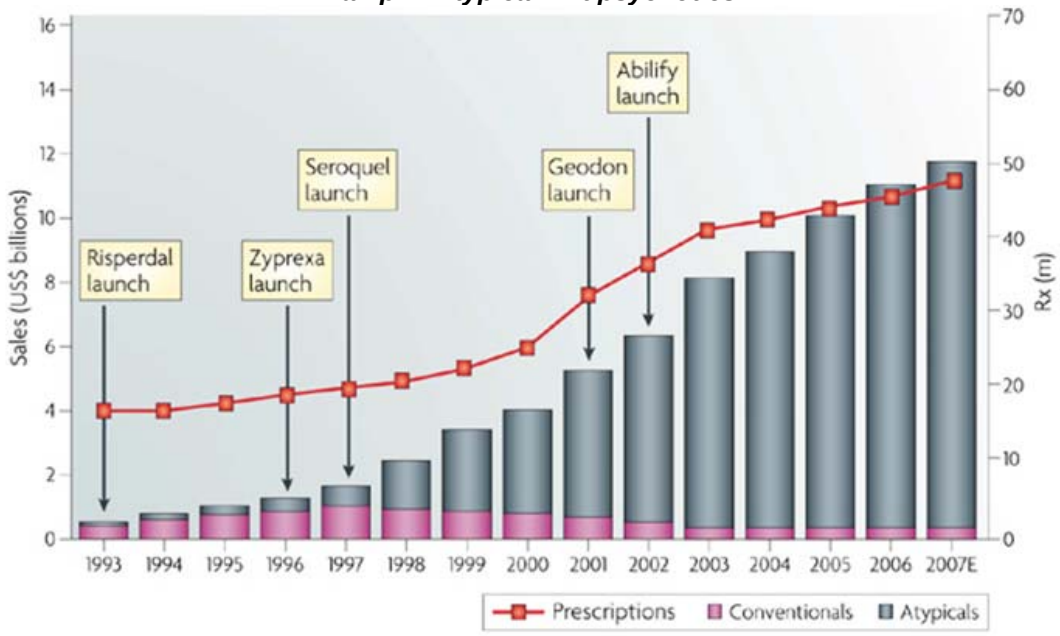
There is no cure for schizophrenia, and managing symptoms remains a challenge. No single approach is considered effective for all patients. Antipsychotics have been a mainstay of the treatment paradigm since the approval of chlorpromazine (Thorazine) in the mid-1950s. Chlorpromazine, along with other typical (first-generation / conventional) antipsychotics such as fluphenazine and haloperidol, where used commonly to treat the symptoms of schizophrenia until the 1990's when atypical (second-generation) antipsychotics were commercialized.

Typical antipsychotics like haloperidol offer significant efficacy in addressing the positive symptoms of the disease, but carry meaningful side-effects, including dry mouth, muscle stiffness, cramping, tremors, and weight gain. Serious side-effects include tardive dyskinesia, akathisia (restlessness), parkinsonism, and dystonia, as well as neuroleptic malignant syndrome (NMS). Chlorpromazine is not used due to the risk of an acute condition involving a severe and dangerous leukopenia, known as agranulocytosis.

The first atypical antipsychotic developed was clozapine in 1971. It offered significant efficacy advantages with respect to managing the positive symptoms of schizophrenia, but was withdrawn from the market in 1975 due to the serious risk of agranulocytosis. In 1989, based on the powerful efficacy previously seen in treating treatment-resistant schizophrenia, the U.S. FDA allowed clozapine to return to the market with a black box warning. Safer second-generate atypical antipsychotics such as olanzapine (Lilly's Zyprexa in 1996), risperidone (J&J's Risperdal in 1994), and quetiapine (AstraZeneca's Seroquel in 1997) were developed in 1990's. Drugs such as ziprasidone (Pfizer's Geodon in 2000), aripiprazole (Bristol's Abilify in 2001), and paliperidone (J&J's Invega in 2007) followed in the 2000's. These atypical antipsychotics offered improved tolerability. Nevertheless, each of the aforementioned drugs, except the recently launched Invega, has achieved annual sales over $1 billion.

In fact, Eli Lilly's Zyprexa (olanzapine) peaked at nearly $4.8 billion in sales in 2007. J&J's Risperdal (risperidone) peaked at $4.4 billion in 2007. In total, the atypical antipsychotic class peaked at $16 billion in annual sales before patents began expiring in 2007.

…Ramp In Atypical Antipsychotics…



Source: Nature Reviews (June 2008)

…CATIE Sheds Some Light…

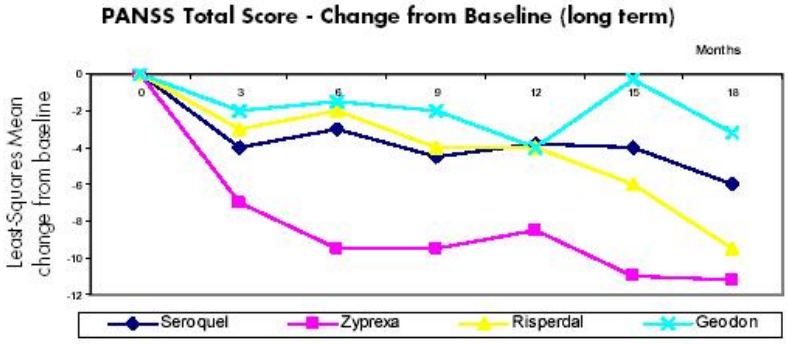
In December 2000, the U.S. National Institute of Mental Health funded and coordinated the Clinical Antipsychotic Trials in Intervention Effectiveness (CATIE) study. The goal of CATIE was to determine which medications are most effective in helping to improve the quality of life in people with schizophrenia. Essentially, CATIE looked at the balance between efficacy and tolerability for atypical antipsychotics. The trial lasted for 18 months and enrolled approximately 1,500 patients at over 50 clinical sites in the U.S. CATIE tested Zyprexa (n=330), Risperdal (n=333), Seroquel (n=329), Geodon (n=183), and clozapine, among others, head-to-head.

Data from the CATIE study shed some important light on how schizophrenia patients are treated. The data show:

- ✓ Only about 25% of patients were able to stay on their starting medication for the entire 18 months. The other 75% discontinued due to side-effects or bounced between medications over the 18 month period. Adverse events were common in all treatment arms.

CATIE Results	Zyprexa (olanzapine)	Risperdal (risperidone)	Seroquel (quetiapine)	Geodon (ziprasidone)
Patients Enrolled at Day-1	330	333	329	183
Discontinued Treatment				
…Due to Efficacy	15%	27%	28%	24%
…Due to Tolerability	19%	10%	15%	15%
…No Reason / Other	30%	36%	39%	40%
Total Discontinued	**64%**	**74%**	**82%**	**79%**
Adverse Events				
…Serious Adverse Events	10%	10%	9%	10%
…Moderate Averse Events	70%	83%	65%	64%
…Insomnia	16%	28%	18%	24%
…GI AEs	27%	27%	20%	19%
…Weight Gain > 7% Baseline	30%	14%	16%	7%
Median Change in HbA1c	+7.0 mg/dl	+5.5 mg/dl	+4.3 mg/dl	+2.5 mg/dl
Median Change in Cholesterol	+8.5 mg/dl	-3.0 mg/dl	+3.5 mg/dl	-1.0 mg/dl
Median Change in Triglycerides	+33.5 mg/dl	+3.0 mg/dl	+17.5 mg/dl	-7.0 mg/dl
New cataract Formations	1%	~1%	1%	~1%
Mean Change in QT Interval	1.2±1.8 sec	0.2±1.8 sec	5.9±1.9 sec	1.3±2.2 sec
Median Change in Prolactin	-0.9 ng/ml	+9.2 ng/ml	-2.7 ng/ml	-4.5 ng/ml

- ✓ Clozapine was shown to be the most effective drug, but its use was sparing given the risk for agranulocytosis and the weekly blood monitoring tests required for prescription. With respect to the big four drugs listed above, Zyprexa and Risperdal faired the best on the primary efficacy endpoint of reduction in Positive and Negative Symptom Scale (PANSS).



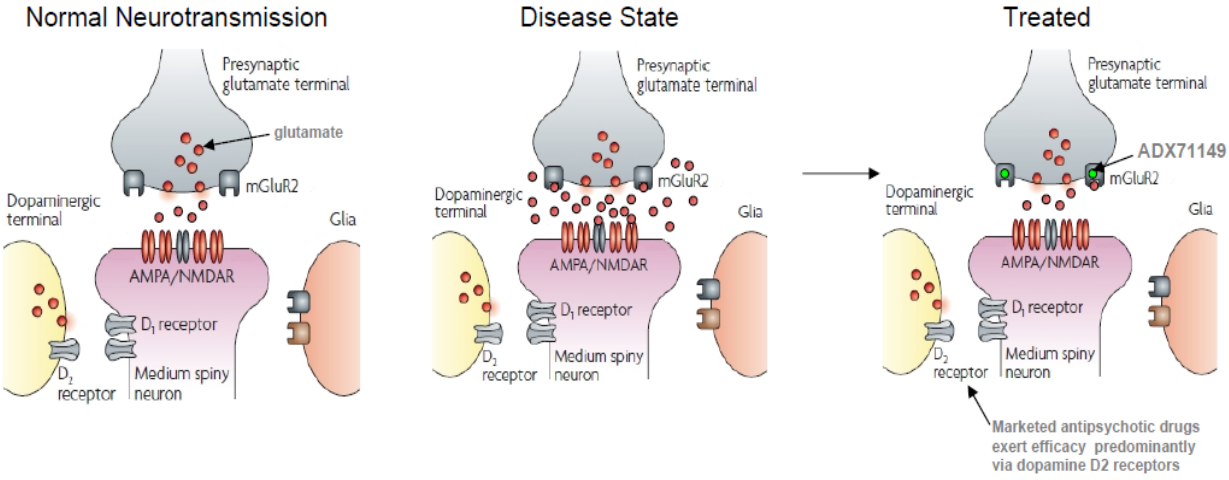
Results from CATIE show that schizophrenia patients, even on mega-blockbuster drugs like Lilly's Zyprexa and J&J's Risperdal, are poorly treated. Zyprexa offered up the best results with respect to efficacy (reduction in PANSS) and discontinuation, but still has significant detrimental side-effects including weight gain and an increase in cholesterol and triglycerides. Pfizer's Geodon, perhaps the most tolerability from a side-effect profile, offered up the least efficacious results. We conclude, from CATIE, that despite significant generic competition from both typical and atypical antipsychotics, the market remains attractive and in dire need of new medications. A highly efficacious and tolerable drug for schizophrenia, with a new mechanism of action, is a potential mega-blockbuster.

ADX71149

Addex Therapeutics is developing ADX71149, a positive allosteric modulator (PAM) of metabotropic glutamate receptor 2 (mGluR2). ADX71149 has the potential to be the first oral, non-dopaminergic drug that may address both the positive and negative symptoms of schizophrenia.

Management believes that ADX71149 is differentiated from the marketed antipsychotics noted above in that it may show efficacy on negative symptoms and avoid compliance-limiting side effects like weight gain, hyper-prolactinemia, and extrapyramidal symptoms such as akathisia, parkinsonism, and dystonia, which are associated with the use of dopamine antagonists.

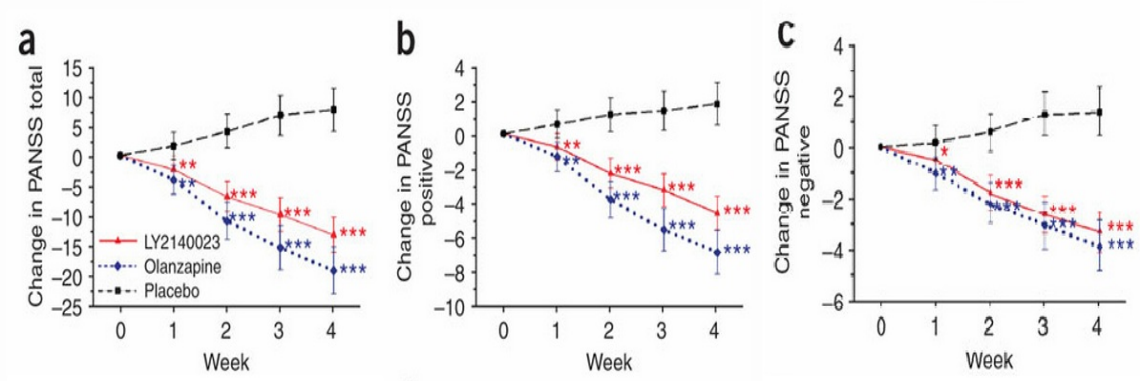
The mGluR2-PAM mechanism of action may also present the opportunity for synergistic co-therapy with approved atypical antipsychotics. In normal neurotransmission, there is a balance or glutamate at the dopaminergic terminal. Glutamate is a powerful transmitter in the brain and integral to the normal functioning of memory, learning and perception. Too much glutamate can lead to seizures and the death of brain cells. In the schizophrenia disease state, the dopaminergic terminal may be flooded with excess glutamate, causes both positive and negative symptoms of the disease. ADX71149 is designed to bring the balance back to a more normalized state.



Source: Nature Reviews (June 2008)

Proof-of-concept of this mechanism has been demonstrated by Eli Lilly in a phase 2 clinical trial with LY2140023, a mGluR2/3 agonist, in 118 patients with schizophrenia. The data show that LY2140023 demonstrated significantly greater response rates compared to placebo in reducing PANSS (figure a), as well as the individual components of both positive (figure b) and negative (figure c) symptoms of the disease.

…LY2140023 Proof-of-Concept…



…Partnership With J&J…

In January 2005, Addex Therapeutics and Ortho-McNeil Pharmaceutical, Inc., a division of Johnson & Johnson Company, entered into a worldwide research collaboration and license agreement to discover, develop, and commercialize novel compounds modulating allosterically G-Protein Coupled Receptors for the treatment of anxiety, depression, schizophrenia, and Alzheimer's disease. The deal came with €7.2 million for two years of research funding by J&J, which was later expanded when the duo selected ADX71149 to move into clinical studies.

In June 2009, Ortho-McNeil announced they had started a phase 1 clinical study of ADX71149. Addex received a €1 million payment from Ortho-McNeil as a result of the trial initiation. ADX71149, now in the hands of Janssen Pharmaceuticals at J&J, handles all costs associated with development of ADX71149. Addex has received a total of €10.2 million in upfront and milestone payments to date on ADX71149, with the potential to receive an additional €109 million in future milestones and potential low double-digit royalties on worldwide sales once commercialized.

We view J&J as an outstanding partner for Addex Therapeutics. J&J clearly has an interest in the schizophrenia market, having commercialized Risperdal in 1994, a long-acting release (LAR) formulation of risperidone called Risperdal-Consta in 2002, and most recently Invega (paliperidone) in 2007. The potential for future milestone totaling €109 million – all pre-commercialization, nearly three-times the current market value of the company, along with low double-digit royalties on sales a potential mega-blockbuster drug presents enormous upside for Addex shareholders. Finally, we are pleased that J&J shares Addex vision for the drug, seeking additional indications in anxiety and major depressive disorder.

…Phase 2a Ongoing In Schizophrenia…

In March 2011, Ortho-McNeil-Janssen Pharmaceuticals (OMJP) announced the start of a phase 2a clinical trial with ADX71149 for the treatment of schizophrenia. The announcement triggered a €2 million milestone payment from J&J to Addex Therapeutics. The phase 2a program is a double-blind, placebo-control, EU-centered study seeking to enroll 105 patients with schizophrenia in two parts:

- ✓ Part-A (monotherapy): 15 subjects with sub-acute positive symptoms will be treated in an open-label design with a recommended starting dose of 50 mg ADX71149 BID. Then according to tolerability, as judged by the investigator, the dose may be increased stepwise to 100 mg BID up to the recommended target dose of 150 mg BID. In principle, the open label treatment phase will last for maximally 12 weeks. Endpoints will examine tolerability, safety and efficacy.

- ✓ Part-B (add-on therapy): In 90 subjects with residual positive symptoms or predominant negative symptoms or in subjects with insufficient response to clozapine, ADX71149 will be administered in a double-blind, placebo controlled, 2:1 (ADX71149:placebo) randomized design at 2 different dose levels, 50 mg BID up to maximally 150 mg BID, and this as adjunctive therapy to their currently prescribed antipsychotic. Endpoints will examine tolerability, safety and efficacy.

We expect data from this phase 2a study by the end of the year. We will be looking for similar data to the phase 2 data released and published by Eli Lilly noted above, with clear and statistically significant reductions in PANSS, as well as particular efficacy on the negative symptom side of the scale.

- Primary outcome measures
  - Safety
  - Tolerability
- Secondary outcome measures
  - Positive and negative syndrome scale (**PANSS**)
  - Clinical Global Impression Schizophrenia (**CGI-SCH**)
  - Subjective well-being under neuroleptics scale (**SWN**)

…What's Next…

If positive, we expect that J&J will look to move into a phase 2b or phase 2/3 trial in 2013. The nature of the next trial in schizophrenia depends on how strong a dose response is seen with ADX71149, and whether or not J&J feels comfortable moving into a pivotal registration program or if another dose-ranging study is necessary to further quantify the response seen.

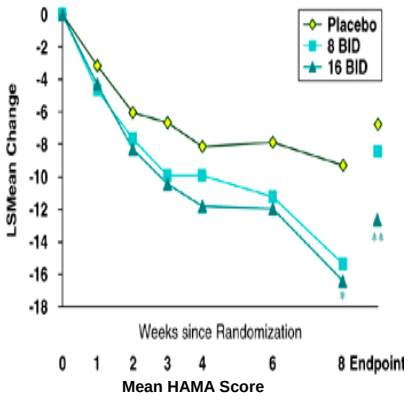
…Phase 2a Ongoing In Anxiety / Major Depressive Disorder…

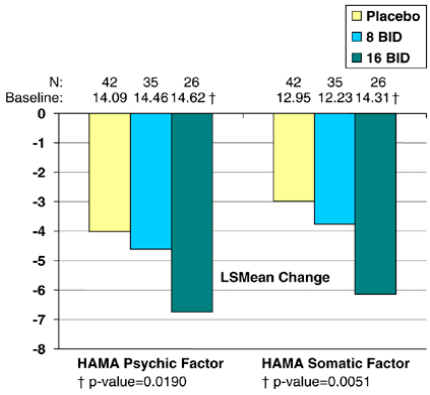
In June 2012, Addex and Janssen accounted the initiation of a multicenter, double-blind, phase 2a study of ADX71149 in adults with major depressive disorder (MDD) who are also suffering anxiety symptoms. Addex and J&J believe that the mGluR2-PAM mechanism of action, regulation of glutamate around the NMDA and AMPA receptors, may find utility in treatment patients with depression and anxiety while avoiding some of the predominant side effects associated with antidepressants, including selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors.

The multicenter, double-blind, placebo-controlled study is designed to evaluate the efficacy and overall safety and tolerability of ADX71149 as an adjunctive treatment to an antidepressant in 94 adults with major depressive disorder with anxiety symptoms. Oral ADX71149 will be administered twice-daily (BID) at doses ranging from 25 mg to 150 mg. Patients will continue to take the same daily dose of their antidepressant.

The primary endpoint of the study is the change from baseline in the Hamilton Anxiety Rating scale (HAM-A6) score. Secondary endpoints include change from baseline of several other clinician-administered rating scales designed to assess the severity of depression and anxiety symptoms.

Proof-of-concept with mGluR2/3 agonists has been demonstrated and published in the August 2007 edition of Neuropsychopharmacology, entitled, "*Efficacy and Tolerability of an mGlu2/3 Agonist in the Treatment of Generalized Anxiety Disorder*" (33,1603-1610). Results with Eli Lilly's candidate, LY544344, are presented below.





Event	Placebo *N*=44 *n* (%)	8 mg b.i.d. *N*=37 *n* (%)	16 mg b.i.d. *N*=30 *n* (%)	Total *N*=111 *n* (%)
Patients with ≥1 TEAE	25 (56.8)	20 (54.1)	20 (66.7)	65 (58.6)
Nausea	5 (11.4)	8 (21.6)	9 (30.0)	22 (19.8)
Headache	2 (4.5)	3 (8.1)	3 (10.0)	8 (7.2)
Abdominal pain (upper)	2 (4.5)	2 (5.4)	1 (3.3)	5 (4.5)
Dizziness	2 (4.5)	2 (5.4)	2 (6.7)	5 (4.5)
Fatigue	2 (4.5)	1 (2.7)	1 (3.3)	5 (4.5)
Diarrhea NOS	2 (4.5)	2 (5.4)	2 (6.7)	4 (3.6)
Dysmenorrhea	2 (4.5)	0 (0.0)	2 (6.7)	4 (3.6)
Dyspepsia	2 (4.5)	0 (0.0)	1 (3.3)	3 (2.7)
Feeling abnormal	2 (4.5)	1 (2.7)	0 (0.0)	3 (2.7)
Insomnia	3 (6.8)	0 (0.0)	0 (0.0)	3 (2.7)

Data from this phase 2a study is expected towards the end of 2013. Investor focus over the near-term is on the phase 2a schizophrenia trial noted above, but we believe that expanding the label on ADX71149 to include major depressive disorder and anxiety, a market that is vastly under-served by existing medications, could double the market opportunity for J&J.

…Market Opportunity & Sales Forecasts…

According to the U.S. National Institute of Mental Health (NIMH), approximately 1.1% of the U.S. adult population, or roughly 2.4 million people, have schizophrenia. Health Canada estimates 280,000 citizens with schizophrenia. The European Brain Council estimates 2.5 million people in industrialized Europe with schizophrenia. On a global basis, the World Health Organization (WHO) estimated 24 million people in developed nations with the disease.

There is no cure for schizophrenia. Data published in February 1994 by the Department of Psychological Medicine, Institute of Psychiatry in London, UK in the Acta Psychiatrica Scandinavica. (Feb;89(2):135-41), notes the average onset of schizophrenia for both men and women is around 25 years old. The average life expectancy for a schizophrenia patient is 65 years old. That means that the average schizophrenic patient is on therapy for 40 years.

Based on data from the CATIE study, and EPSILON (European Psychiatric Services: Inputs linked to Outcome Domains and Needs) study, a similar analysis of schizophrenia care to the CATIE study conducted in five European countries, approximately 70% of schizophrenia patients on therapy discontinue treatment during an 18 month period. Most will switch to a new therapy. In fact, it is considered normal for a schizophrenic patient to switch between as many as three or four medications over a decade period.

⇒ Therefore, despite the presence of significant generic competition from previous blockbuster drugs like Zyprexa (olanzapine) and Risperdal (risperidone), and new medications like Invega (paliperidone), we believe there is an enormous prescription opportunity for a new medication with a differentiated profile.

Schizophrenia is a significant financial burden on the U.S. healthcare system. The NIMH estimates over $65 billion is spent each year on care for schizophrenia patients, with direct costs for pharmaceutical products over $5 billion (mostly all atypical antipsychotics are generic), $10 billion in outpatient care, and $10 billion inpatient and long-term acute care. Between North America and Europe, schizophrenia, directly and indirectly, is a $100 billion burden.

⇒ Therefore, despite the presence of significant generic competition (noted above) we believe there is meaningful pricing opportunity for a new medication with a differentiated profile.

Addex and J&J are also studying ADX71149 in patients with anxiety that have major depressive disorder (MDD). Similar to the dynamics of the schizophrenia market, there is significant branded and generic treatment options available for the treatment of depression and anxiety, including SSRI's Celexa (citalopram), Lexapro (escitalopram), Paxil (paroxetine), Prozac (fluoxetine), Luvox (fluvoxamine), and Zoloft (sertraline), and SNRI's Pristiq (desvenlafaxine), Cymbalta (duloxetine), Savella (milnacipran), and Effexor (venlafaxine).

At peak, the SSRI/SNRI market in the U.S. eclipsed $20 billion in branded sales. However, SSRI's and SNRI's carry significant side effects, including risks of constipation, diarrhea, dizziness, dry mouth, fatigue, headaches, impotence, insomnia, nausea, tremors, and amazingly, anxiety. Treating patients for MDD and anxiety with drugs that are known to have a side effect of increased anxiety simply does not make sense.

Plus, SSRI's also present increased risk of suicide in children and adolescents (by roughly 80%) and agitation and hostility (by roughly 130%), so much so that the U.S. FDA placed a black box warning on all SSRI products in 2004. In the UK, the Medicines and Health products Regulatory Agency (MHRA) bans all SSRI use in children, except Prozac and Luvox for the treatment of obsessive-compulsive disorder.

Without yet seeing the proof-of-concept data from either phase 2a program with ADX71149, we cannot predict peak sales forecasts. However, we can provide a range based on the theoretical profile of ADX71149 and the outcome of the registration programs.

Theoretical Profile of ADX71149	**Sales Range Forecast**
In Schizophrenia	
Fully differentiated profile on safety (no weight gain, no hyperprolactinemia, no change in HBA1c or cholesterol) and efficacy (statistically significant reduction positive & negative symptoms)	> $1 Billion
Improvement in safety / tolerability, but still limited efficacy on the negative symptoms of schizophrenia (or) Improvement in negative symptoms but with similar "atypical" side effects	$250 to $750 million
New mechanism of action, but no real benefit in terms of side effects / tolerability or efficacy profile with respect to negative symptoms	< $250 million
In MDD w/ Anxiety	
Meaningful improvement in symptoms of depression and anxiety, without common side effects and tolerability issues seen with SSRI / SNRI drugs	> $1 Billion
Improvement in depression symptoms, but no real meaningful benefit in anxiety or improvement in common side effects / tolerability seen with SSRI / SNRI drugs	$0 to $500 million

As noted above, Addex Therapeutics is eligible to receive an additional €109 million in future pre-commercialization milestones and low double-digit royalties on worldwide sales of ADX71149 at J&J once commercialized.

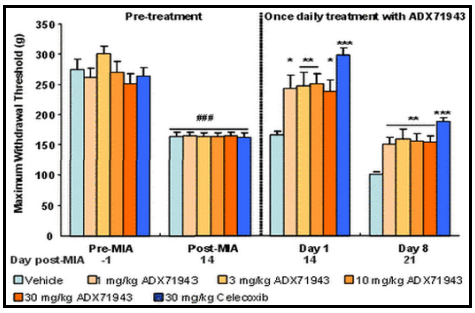
PIPELINE

Addex Therapeutics possesses a deep preclinical pipeline with a handful of GABA and mGlu candidates targeting neurological and inflammatory diseases. The company also has candidates targeting GLP-1, TNFR1, and TkkB for various CNS and metabolic disorders in earlier-stage preclinical trials. The most advanced preclinical candidate is an oral GABA-B receptor PAM in late-stage preclinical studies with an investigational new drug application (IND) planned for the fourth quarter 2012.

GABA-B PAM

Addex Therapeutics is currently in IND-enabling activities with an oral, first-in-class, $GABA_B$ (g-aminobutyric acid subtype B) positive allosteric modulator (PAM) that has demonstrated excellent preclinical efficacy and tolerability in several rodent models of osteoarthritis (OA) pain and overactive bladder (OAB).

Chronic OA pain represents a significant unmet medical need and a potential blockbuster opportunity for Addex's $GABA_B$-PAM, ADX71943. We believe the drug can be positioned in-between generally-safe and low-efficacy NSAIDs and more powerful but poorly tolerable opioid drugs. A long-acting, well tolerated, oral drug, without abuse liability, would represent a major advancement in pain management and a large market opportunity. The preclinical data generated to date with ADX71943 suggests a competitive and attractive profile – i.e. significant analgesic effect without troublesome side effects associated with opioids such as sedation, nausea, constipation, and potential addiction / abuse.



Overactive bladder (OAB), also known as urge urinary incontinence, represents another significant opportunity for Addex Therapeutics. According to the U.S. Department of Health and Human Services (HHS), there are an estimated 25 million U.S. adults suffering from OAB. OAB is currently treated with anticholinergic drugs like oxybutynin, but limited efficacy and side effects like dry mouth, constipation, blurred vision and tachycardia, significantly limit their use. In addition, there is growing awareness that anticholinergic OAB drugs can cause adverse CNS effects, ranging from cognitive impairment to episodes of psychosis. We believe a long-acting oral drug without the anti-muscarinic liabilities associated with oxybutynin would represent a major advancement in OAB treatment and a large market opportunity. Addex is developing ADX71441 in late-stage preclinical studies with an IND planned for the fourth quarter 2012.

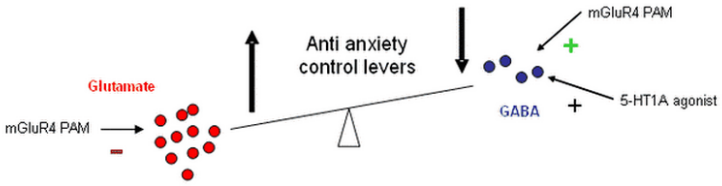
mGluR4 PAM

Addex Therapeutics is developing metabotropic glutamate receptor 4 (mGluR4) positive allosteric modulators (PAM) in various stages of preclinical studies. Recent research on this receptor shows that activation plays a key modulatory role in many CNS and non-CNS pathways. As a result, mGluR4 is emerging as a promising non-dopaminergic target for potential disease-modifying properties and the treatment of motor and non-motor symptoms in patients with Parkinson's disease. Addex was previously working with Merck on the development of mGluR4 PAMs for PD, but regained full rights to this platform in September 2011.

Currently, Addex is developing a novel, nanomolar, selective, brain penetrant and orally bioavailable mGluR4 PAM, ADX88178, that has demonstrated efficacy in several different rodent models of PD. Data shows that ADX88178 reverses haloperidol induced catalepsy (HIC) in rats after oral administration. More importantly, the combination of ADX88178 with a low dose of L-DOPA enabled a robust, dose-dependent reversal of the forelimb akinesia deficit and did not worsen dyskinesia induced by L-DOPA in rats. This is consistent with an L-DOPA sparing action that may prove to be therapeutically useful for the management of motor symptoms of PD.

Addex also believes that mGluR4 PAM may also represent a new generation of anxiolytic therapeutics agents for the treatment of anxiety disorders. Research shows that based on presynaptic localization of mGluR4 in brain areas involved in anxiety and mood disorders, mGluR4 may dampen excessive brain excitability. We see this as a meaningful potential opportunity considering that anxiety disorders are among the most prevalent psychiatric disorders and are co-morbid with Parkinson's disease.

The mechanism of action seems to involve GABA-ergic and serotonergic systems. Preclinical rodent models of anxiety displayed an anxiolytic-like activity and the ability to balance the neurotransmission between the direct / indirect pathways via D1 and D2 receptors.



TNFR1 NAM

The TNF pathway is targeted by several marketed biologic drugs generating over $16 billion in annual revenues. These include Amgen's Enbrel (etanercept), Abbott's Humira (adalimumab), and J&J's Remicade (infliximab). These drugs are injectable and have been associated with serious side effects, including immunogenicity, injection site reactions, serious infections including tuberculosis, and risk of developing lymphoma. Addex believes that developing an oral TNF receptor molecule could provide a superior safety and tolerability profile to the aforementioned injectable drugs. However, identifying orally available TNF receptor molecules through traditional high-throughput screening (HTS) methods has proven quite difficult.

Addex has developed a proprietary technology termed AddeLite that allows tagging of TNFR while preserving their signaling function. The AddeLite Tag allows real time assessment of shape changes affecting all TNFR in response to ligation with either its natural ligands or synthetic allosteric modulators in a high throughput setting. Using this technology, Addex have been able to identify and optimize drug-like small molecules targeting the extracellular domain of TNFR1. TNFR1 NAMs are now progressing through lead generation phase of preclinical testing.

TrkB PAM

TrkB belongs to the large receptor tyrosine kinase (RTK) family, and is the receptor for the BDNF neurotrophin, which has been shown to exert strong survival and neuroprotective effects on the neurons of the central nervous system. This has potential therapeutic implications in the treatment of Alzheimer's disease (AD), Huntington's disease, and Parkinson's disease (PD).

To date, identification of small molecules acting as selective TrkB agonists has been poor, probably due the small size of a typical small compound relative to the large peptide binding site which spans a receptor dimmer. Those that have been identified have offered poor clinical results due to their short half-life and poor blood brain barrier penetration. Management believes that allosteric modulation may offer a unique opportunity to discover small molecular weight, non peptidergic molecules, with better potential clinical outcomes targeting TrkB. Addex has developed several assays, called ProxyLite, allowing the real-time assessment of TrkB activation upon BDNF binding in HTS format.

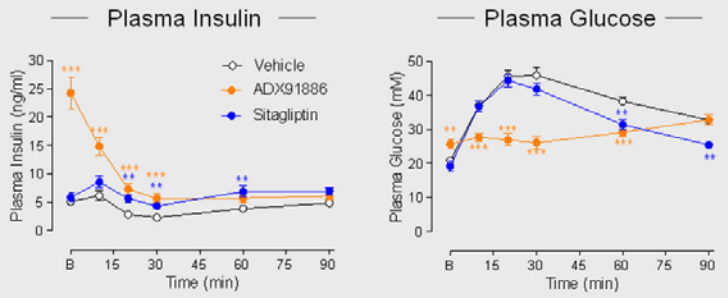
Using these different assays, Addex has successfully identified several novel drug-like chemical series having TrkB positive allosteric modulation activity. These potential first-in-class chemical series are currently progressing towards lead optimization phase of preclinical testing.

GLP1R PAM

GLP-1 is a polypeptide secreted from the L cells of the distal gut as a cleavage product of proglucagon. It is released into the circulation after a meal and increases insulin and suppresses glucagon secretion; it also delays gastric emptying, and suppresses appetite. GLP-1 peptide derivatives have emerged as an effective therapeutic class in the treatment of type 2 diabetes given their lack of a common side effect, weight gain, seen with older diabetic medications. Injectable GLP-1 peptide drugs are commercially available, including Amylin's Byetta (exenatide) and Novo Nordisk's Victoza (liraglutide), with sales approaching $1 billion worldwide. Side effects include nausea, vomiting, diarrhea, headache, and risk of hypoglycemia (low blood sugar) and pancreatitis.

DPP-4 inhibitors that look to block the production of the dipeptidyl peptidate-4 enzyme that inactivates incretin GLP-1 are also commercially available. These include Merck's Januvia (sitagliptin), Bristol's Onglyza (saxagliptin), and Boehringer's Tradjenta (linagliptin). Sales of DPP-4 molecules, which eclipse $5 billion worldwide, dwarf those of the GLP-1 agonists due to better tolerability, easier and more convenient oral dosing, and lower risk of serious side effects like hypoglycemia and pancreatitis.

Addex believes they have discovered GLP1R positive allosteric modulators (PAM) that dramatically increase insulin secretion and improve oral glucose tolerance. These molecules would be oral, providing a dosing advantage over the commercially available injectable GLP-1 molecules. Additionally, the allosteric modulation may avoid ectopic GLP1 simulation believed to be the underlying cause of the risk of pancreatitis. Preclinical db/db mice data suggests a unique profile compared to Merck's $3+ billion blockbuster Januvia (sitagliptin).



PIPELINE CHART

Molecule / Mechanism	PRECLINICAL: Assay Development & Screening	PRECLINICAL: Hit-to-Lead	PRECLINICAL: Lead Optimization	PRECLINICAL: Clinical Candidate Selection	PRECLINICAL: IND Enabling	CLINICAL: Phase I	CLINICAL: Phase II	Partner
Dipraglurant-IR (ADX48621) mGluR5 NAM – Parkinson's disease levodopa induced dyskinesia (PD-LID) #								
ADX71149 mGluR2 PAM – schizophrenia				Funded and developed by JPI*				janssen
ADX71149 mGluR2 PAM – major depressive disorder with anxiety				Funded and developed by JPI*				janssen
Dipraglurant-ER (ADX48621) mGluR5 NAM – non-Parkinsonian dystonias								
GABA-BR PAM – overactive bladder								
mGluR4 PAM – Parkinson's disease, anxiety, multiple sclerosis								
mGluR2 NAM – Alzheimer's, depression								
mGluR7 NAM – anxiety / depression, PTSD								
TrkB PAM (RTK superfamily) – neurodegenerative and other diseases								
GLP1R PAM – type II diabetes								
TNFR1 NAM (TNF receptor superfamily) – RA; psoriasis; IBD; Alzheimer's; MS								

NAM = negative allosteric modulator (inhibitor)
PAM = positive allosteric modulator (activator)
*Janssen Pharmaceuticals Inc., formerly Ortho-McNeil-Janssen Pharmaceuticals Inc.
partially funded by a grant from the Michael J. Fox Foundation for Parkinson's Research

Wholly-owned by Addex
Partnered

MANAGEMENT BIOS

Bharatt Chowrira, Ph.D. – President and Chief Executive Officer

Dr. Chowrira, who joined Addex in 2011, has a strong track record in the biopharmaceutical industry with over 17-years of experience, combining a unique blend of research, licensing, corporate development, operations and legal expertise. Most recently, Dr. Chowrira was the Senior Vice President and Chief Operating Officer of Nektar Therapeutics, a NASDAQ-traded U.S. biopharmaceutical company. At Nektar he led a team that established several revenue-generating strategic alliances. He also led efforts to streamline, realign and integrate operations across research, manufacturing, business development, marketing and multiple R&D sites. Dr. Chowrira previously served as Executive Director, Worldwide Licensing & External Research at Merck & Co., Inc. Prior to that, he was a key member of the executive management team that restructured and re-launched Sirna Therapeutics, a development-stage biopharmaceutical firm focused on the discovery and development of RNAi-based drugs, which was acquired by Merck & Co., Inc. He has a Ph.D. in Microbiology and Molecular Genetics from the University of Vermont and a law degree (J.D.) from the College of Law at the University of Denver. Dr. Chowrira is a registered U.S. patent attorney and a licensed member of the Colorado Bar Association.

Tim Dyer – Chief Financial Officer

Since co-founding Addex in 2002, Mr. Dyer has played a pivotal role in building the Addex Group, raising CHF 263 million of capital, including Addex IPO, and negotiating licensing agreements with pharmaceutical industry partners. Prior to joining Addex he spent 10 years with Price Waterhouse (PW) & PricewaterhouseCoopers (PwC) in the UK and Switzerland as part of the audit and business advisory group. At PwC in Switzerland, Mr. Dyer's responsibilities included managing the service delivery to a diverse portfolio of clients including high growth start-up companies, international financial institutions and venture capital and investment companies. At PW in the UK, Mr Dyer gained extensive experience in audit and transaction support; spending 2 years performing inward investment due diligence on local financial institutions in the Ex-Soviet Union. He serves on the boards of Abionic SA, a private medical device start-up company focused on allergy diagnostics and Qwane Biosciences SA, a private drug development tool company focused on commercializing microelectrode array technologies. He is a UK Chartered Accountant and holds a BSc (Hons) in Biochemistry and Pharmacology from the University of Southampton.

Charlotte Keywood – Chief Medical Officer

Dr. Keywood has overseen Addex medical and regulatory activities since the company was founded in 2002, which includes five Phase 2a and three Phase 2b trials for products in development for smoking cessation, anxiety, migraine, gastroesophageal reflux disease and Parkinson's disease. Dr. Keywood has 20 years of experience in drug development and medical marketing across a broad range of therapeutic areas. During this time she has worked in the U.S. and Europe and has been responsible for all stages of clinical development, including pre- and post-registration and pharmacovigilance activities. Dr. Keywood, acting as a consultant, served from 2001 to 2003 as Medical Director for Axovan, a Swiss biotech company acquired by Actelion in 2003. From 1996 to 2001 she was Medical Director at CNS Vernalis, where she helped bring a new migraine drug, frovatriptan, to the market. Dr Keywood is a cardiologist who completed her postgraduate training at St Thomas' Hospital, London.

Sonia Poli – Head of Non-Clinical Development and CNS Projects

Dr. Poli, who joined Addex in 2004, is an accomplished drug R&D professional with over 16 years international experience in large and small pharmaceutical companies with extensive experience and knowledge of drug discovery and preclinical development. At Addex she has provided preclinical support for ongoing clinical development programs and has overseen the transition of four products into clinical development for indications including smoking cessation, anxiety, schizophrenia, migraine, gastroesophageal reflux disease and Parkinson's disease. She worked from 1997 to 2004 in the drug metabolism and pharmacokinetics (DMPK) area at Roche, where she was a key inventor and global head of a multidimensional optimization approach for drug discovery and development and played an important role in selecting clinical candidates in CNS indications, including Alzheimer's disease, Parkinson's disease, bi-polar disorders and anxiety. Dr. Poli obtained her degree and doctorate in Industrial Chemistry at the University of Milan in 1993 and completed a post-doctoral fellowship at the CNRS, in Paris, in the group of Prof. D. Mansuy in 1997. Dr. Poli is co-author of more than 30 research publications and patents.

Jean-Philippe Rocher – Head of Chemistry

Since joining Addex at its inception, Dr. Rocher has been responsible for establishing the company's chemistry capabilities and building Addex small molecule allosteric modulator chemistry platform. He has played a pivotal role in the success of both internal and partnered programs including Addex mGluR5 negative allosteric modulator and mGluR2 positive allosteric modulator programs which have both progressed into phase 2 clinical development. Prior to joining Addex, Dr. Rocher was director of chemistry at Devgen NV, Gent, Belgium from 2001 to 2002; senior research scientist for GlaxoSmithKline KK in Tsukuba, Japan from 1997 to 2001. During the course of his career as a medicinal chemist, he has discovered several pre-clinical and clinical candidates for CNS, inflammatory diseases and cancer. He completed a Pharm. D and obtained his PhD at the Faculty of Pharmacy of Lyon, France in 1987. Dr. Rocher started his career as a research scientist in the dermatology research centre of Galderma at Sophia-Antipolis, France. He is co-author of more than 30 research publications and patents.

Robert Lutjens – Head of Biology

Since joining Addex at its inception, Dr. Lütjens has been responsible for establishing the company's biology capabilities and building the Addex small molecule allosteric modulator biology platform. He played a pivotal role, managing Addex research collaborations with Janssen Pharmaceuticals Inc., which has led to the successful progression of the first mGluR2 positive allosteric modulator into man. Prior to joining Addex, he completed a postdoctoral fellowship in the Department of Neuropharmacology at the Scripps Research Institute, in La Jolla, CA, where he focused on understanding molecular changes involved in addiction disorders. Dr Lütjens obtained his degrees in Biology from the University of Geneva, his master's at the Swiss Institute for Experimental Cancer Research and his Ph.D. thesis at the Glaxo Institute for Molecular Biology in Geneva and the Institute for Cellular Biology and Morphology in Lausanne. Dr. Lütjens is co-author of multiple peer-reviewed publications and co-inventor on patents covering screening methods or chemical compounds.

Chris Maggos – Director of Business Development

Since joining Addex in 2007, Mr. Maggos has contributed to corporate communication, investor relations and business development efforts. He participated in the Addex IPO, clinical trial results, out-licensing activities, a PIPE financing and other events. Mr. Maggos was Senior Writer for BioCentury, a biotechnology trade publication, from 2001 to 2007. He was an Associate at a New York City hedge fund focused on biotechnology, called Casdin Capital Partners (later Cooper Hill Partners), from 1997 to 2000. Mr. Maggos worked as a technician, studying the molecular neurobiology of drug dependence at The Rockefeller University, where he coauthored 11 scientific publications, from 1993 to 1997. He received a BA in English Literature from Yale University in 1993.

Andre J. Mueller – Chairman of the Board of Directors

Mr. Mueller was born in 1944 and is a Swiss citizen. He has extensive experience in creating and running successful biopharmaceutical companies. Mr. Mueller was a member of the founding team of Actelion Ltd (SIX:ATLN), where he was CFO for 5 years and vice chairman until April 2009. He also was the first VP of Finance and Administration and later, CFO, at Biogen (now Biogen Idec), where he oversaw several financing rounds, including Biogen's IPO. Mr. Mueller started his career with CIBA Ltd and Sandoz (now Novartis) where he held a number of managerial positions in the Pharma, Plant Protection and Finance divisions both at headquarters in Basel and in the U.S. He was a Founding Partner and Director of Investments for Genevest, the first Swiss venture capital organization. He has a degree in Chemical Engineering from the University of Geneva and an MBA from INSEAD. He is a board member and Chairman of the audit Committee of Synthes Inc. (SIX:SYST).

Vincent Lawton – Vice Chairman of the Board of Directors

Professor Lawton was born in 1949 and is a U.K. citizen. He was Vice President Merck Europe and Managing Director of MSD UK until he stepped down in 2006, after 26 years' service internationally for Merck & Co Inc. He was appointed CBE (Commander of the British Empire) by the Queen of England for services to the Pharmaceutical Industry. During his tenure, MSD UK achieved sustained commercial success, launching many new medicines to the market in a wide range of therapeutic areas, becoming the fastest growing company in the market over a number of years. He worked in commercial, research and senior management roles in France, the US and Canada, Spain and throughout Europe. As President of the UK Industry Association, the ABPI, he negotiated industry pricing, worked with Government bodies to help establish the UK Globally as a leading centre of clinical research. He is the Chairman of Aqix Ltd, a private UK biotechnology company, member of the Board of the Medicines Regulator, the MHRA and is a Senior Strategy Advisor for Imperial College Department of Medicine, University of London. He also serves as a consultant to a number of leading healthcare organizations. He was educated at the University of London and holds undergraduate and PhD degrees in Psychology.

VALUATION & RECOMMENDATION

Initiating Coverage

We are initiating coverage of Addex Therapeutics (Swiss-SIX:ADXN.SW, OTC:ADDXF) with a 'Buy' rating and CHF 20 ($21.00) price target. Despite limited volume on U.S. OTC tracking stock ADDXF and the inability of some U.S. investors to purchase shares on the SIX, we are initiating coverage of Addex Therapeutics because we believe the company's discovery platform, focused on positive and negative allosteric modulators, offers potential significant advantages in small molecule drug development. We believe the two leading pipeline candidates, dipraglurant and ADX71149, offer blockbuster potential for their respective indications.

…Dipraglurant To Fill A Significant Unmet Medical Need…

Addex Therapeutics is currently seeking a development and commercialization partner for dipraglurant. The drug is an oral negative allosteric modulator (NAM) of the metabotropic glutamate receptor 5 (mGluR5), for the treatment of Parkinson's disease levodopa-induced dyskinesia (PD-LID). LID is a major side-effect of Levodopa use. Levodopa (L-DOPA) is the leading dopamine replacement therapy (DRT) for the treatment of Parkinson's disease. LID is characterized by hyperkinetic movements, including chorea (abnormal involuntary movement), dystonia (sustained muscle contraction, abnormal posture), and athetosis (involuntary convoluted movements). It is most common at times of peak L-DOPA plasma concentrations (peak-dose dyskinesia), although it may also occur when plasma concentrations of L-DOPA rise and fall (diphasic dyskinesia) or during off-time (off-period dystonia).

Phase 2a clinical data on dipraglurant, reported in March 2012, demonstrated encouraging signs of efficacy, safety, and tolerability. Results showed that patients taking dipraglurant had a meaningful and statistically significant reduction in abnormal involuntary movement as soon as one day after starting dipraglurant therapy. The data was also suggestive of an increased in "on" time – the time in which a Parkinson's patient is treated with Levodopa without experiencing dyskinesia – and a reduction in "off" time – the time after which L-DOPA has left the body prior to the next Levodopa dose.

The profile of dipraglurant is one we believe physicians and patients will look very favorably upon. LID is a significant problem that emerges during the treatment of Parkinson's patients. Levodopa is a staple of modern Parkinson's treatment and LID is essentially unavoidable a decade after Levodopa dosing begins. The only available treatment for LID is to reduce Levodopa dosing, which in turn worsens the underlying symptoms of Parkinson's disease.

There are over 1 million Parkinson's patients between North American and Europe on Levodopa therapy. Within five years of beginning treatment, approximately 50% will develop LID. We see dipraglurant as a $650 million opportunity. We expect substantial partnering interest from global pharmaceutical and biotechnology companies for the drug. Management at Addex would like to partner dipraglurant for the next stage in clinical development, which we expect to be a pivotal phase 2b program to begin in 2013, by the end of the year. Based on the market potential for dipraglurant, we can foresee an upfront payment greater than $25 million, with backend potential milestones eclipsing $200 million plus double-digit royalties on worldwide sales.

Based on these parameters, we have built a financial model that assumes a U.S. and European launch of dipraglurant in 2017. We see dipraglurant alone worth over $120 million (~ CHF 100 million) in market value. Addex is currently trading with a market value of only CHF 63.2 million. We believe the dipraglurant market opportunity is being vastly under-valued by investors in both the U.S. and Switzerland.

…ADX71149 Offers Unique Potential Profile…

In January 2005, Addex Therapeutics formed a research collaboration agreement with Johnson & Johnson to discover, develop, and commercialize novel allosteric modulator compounds for the treatment of anxiety, depression, schizophrenia, and Alzheimer's disease. The first of these compounds, ADX71149, has entered clinical testing and is current in a phase 2a for the treatment of schizophrenia. To date, Addex has received a total of €10.2 million in upfront and milestone payments on ADX71149, with the potential to receive an additional €109 million in future pre-launch milestones and potential low double-digit royalties on worldwide sales once commercialized.

The potential therapeutic profile of ADX71149 is intriguing. Despite significant generic competition from atypical antipsychotics, we believe an enormous market opportunity exists for ADX71149. Atypical antipsychotics carry significant side effects, including weight gain, hyperprolactinemia, increases in blood glucose, increases in serum cholesterol and triglycerides, and increased risk of cataracts, which all lead to high discontinuation rates. Additionally, atypical antipsychotics have limited efficacy in addressing the negative symptoms of schizophrenia, including social withdrawal, apathy, alogia, anhedonia, and avolition.

Addex has designed ADX71149 to address both of these limitations found with atypical antipsychotics. With over 5 million addressable schizophrenics between North America and Europe, we see ADX71149 as a potential blockbuster drug if the theoretical profile holds. For the purpose of our financial model, we have chosen a more conservative approach, modeling ADX71149 as having peak sales around $500 million. Data from the ongoing phase 2a program is expected around the end of the year.

J&J has also progressed ADX71149 into a phase 2a clinical trial looking to study the drug in patients with anxiety that have major depressive disorder. Conventional selective serotonin reuptake inhibitors (SSRIs) for the treatment of depression carry significant side effect, one of which is anxiety. We believe there is meaningful clinical need for a drug that can treat both major depression and anxiety without exhibiting some of the debilitating side effects seen with SSRI and SNRI drugs. Similar to our forecasts for the drug in schizophrenia, we have chosen a heavy discount rate and conservative stance on modeling sales for ADX71149 in anxiety with MDD.

Despite these conservative forecasts, we see ADX71149 as worth an estimated $75 million (~ CHF 70 million) in value. This includes a heavily discounted NPV on the potential €109 million in pre-launch future milestones and potential low double-digit royalties on worldwide sales of ADX71149 from J&J once commercialized.

…Pipeline Kicks In Upside…

Besides the two clinical stage candidates noted above, Addex Therapeutics possesses a deep preclinical pipeline, which includes a handful of GABA and mGlu candidates targeting neurological and inflammatory diseases. The company also has candidates targeting GLP-1, TNFR1, and TkkB for various CNS and metabolic disorders in earlier-stage preclinical trials. The most advanced preclinical candidate is an oral GABA-B receptor PAM in late-stage preclinical studies with an investigational new drug application (IND) planned for the fourth quarter 2012. We see Addex's allosteric modulator pipeline and chemistry platform worth an estimated $25 million (~ CHF 20 million)

DCF Target

We have built a discounted cash flow (DCF) model to value the shares of Addex Therapeutics. Our model is built in Swiss francs and assumes a launch of dipraglurant in 2017 by a yet unfound partner and ADX71149 by J&J also in 2017. We expect several additional candidates to enter the clinic over the next several years. Our model calculates fair value at CHF 217 million, or CHF 20 per share, which equates to a U.S. OTC price of $21 per share. We have posted our model in the back of the report.

RISKS

We see four major risks to our rating and price target:

1. Dipraglurant has successfully completed a phase 2a study. Addex management must now find a development and commercialization partner willing to take the drug into pivotal registration studies. Parkinson's disease programs are high risk, mainly because of the progressive nature of the disease and the difficulty to control high notoriously placebo response. Difficulty in finding a partner could push our launch forecasts back beyond 2017. Should the drug fail in the pivotal program, either due to lack of efficacy, unforeseen side effects, or the inability of Addex or its partner to mitigate the high placebo response seen in the phase 2a trial (at day 28), it would reduce our price target by over 50%.

2. ADX71149 has yet to complete its first phase 2a trial in schizophrenia. It is difficult to model sales of a drug before seeing proof-of-concept and safety data through a phase 2 trial. Instead, what we have done is model sales based on the theoretical profile of the drug and then heavily discounted these sales to account for the added risk of the early-stage nature of the program. The fact that Addex has secured a premier development and commercialization partner in J&J gives us some confidence. Our model assumes that Addex collects roughly 50% of the future €109 million in future pre-commercialization milestones eligible under the collaboration through 2022. However, if the profile of ADX71149 were to come up short of our expectations in either ongoing phase 2a trial, in schizophrenia and major depressive disorder with anxiety, we would have to reduce our price target.

3. Addex exited 2011 with CHF 36 million in cash and investments. We forecast operating burn in the first half of the year will be roughly CHF 11 million, meaning Addex will exit June 2012 with CHF 25 million on the books. The company will require additional funding in 2013 to start the next stage in development for dipraglurant. As noted above, Addex is seeking a development and commercialization partner for dipraglurant. The company is also speaking with the Michael J. Fox Foundation (MJFF) for additional funding. We believe management should be able to secure north of CHF 20 million upfront from the deal. This would provide funding throughout 2013 and into 2014. However, if management cannot secure a deal before early 2013, the company will need to raise cash through a secondary offering. Raising approximately CHF 15 million would be 25% dilutive at the current price, and reduce our price target accordingly. We note this could potentially come simultaneously with a U.S. listing on a major exchange.

4. The U.S. OTC listed shares under ADDXF have virtually no volume. U.S. investors have historically shown little interest in buying foreign pharmaceutical or biotech companies not listed on a U.S. exchange, even when ADRs are available. For example, Novartis and Roche, two of the world's largest pharmaceutical companies, both based in Switzerland and listed on the Swiss SIX like Addex Therapeutics, are vastly under-owned by U.S. retail and institutional investors. Novartis, ranked number one in the world by pharmaceutical product sales, despite having NYSE listed ADRs, finds less than 2% its shareholders located in the U.S. Roche, owner of the worlds three best-selling oncology products, with listed shares on the OTCQX under RHHBY, finds less than 5% of its shareholders located in the U.S. Despite solid fundamentals, two potential blockbuster drugs in mid-stage trials, a massive preclinical pipeline, a marquee partner in Johnson & Johnsons for one of those drugs, Addex may continue to struggle to attract U.S. investors. Nevertheless, we see the shares are vastly undervalued on the Swiss SIX. We believe that Addex could be gearing up for a listing on a major U.S. exchange in 2013. This would be a significant positive for the stock.

PROJECTED FINANCIALS

Addex Therapeutics Ltd.
Income Statement

	2010 E	H1 A	H2 A	2011 A	H1 E	H2 E	2012 E	2013 E	2014 E	2015 E
Royalties on Dipraglurant	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
YOY Growth	-	-	-	-	-	-	-	-	-	-
Royalties on ADX71149	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
YOY Growth	-	-	-	-	-	-	-	-	-	-
Pipeline	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
YOY Growth	-	-	-	-	-	-	-	-	-	-
Collaborations / Other	**4.000**	**3.173**	**0.570**	**3.743**	**1.400**	**5.400**	**6.800**	**5.900**	**10.950**	**20.100**
YOY Growth	-	-	-	-	-	-	-	-	-	-
Total Revenues	**4.000**	**3.173**	**0.570**	**3.743**	**1.400**	**5.400**	**6.800**	**5.900**	**10.950**	**20.100**
YOY Growth	-	-	-	-	-	-	-	-	-	-
R&D	31.165	14.558	13.428	27.986	11.000	8.500	19.500	20.000	20.000	20.000
% R&D	-	-	-	-	-	-	-	-	-	-
SG&A	6.433	3.299	3.432	6.731	2.700	2.300	5.000	5.300	5.600	6.000
% SG&A	-	-	-	-	-	-	-	-	-	-
Operating Income	(33.598)	(14.684)	(16.290)	(30.974)	(12.300)	(5.400)	(17.700)	(19.400)	(14.650)	(5.900)
Operating Margin	*0.0%*	-	-	-	-	-	-	-	-	-
Net Other Income	(0.048)	(0.143)	(0.024)	(0.167)	(0.100)	(0.100)	(0.200)	(0.200)	(0.200)	(0.200)
Pre-Tax Income	(33.645)	(14.83)	(16.31)	(31.141)	(12.400)	(5.500)	(17.900)	(19.600)	(14.850)	(6.100)
Taxes	0	0	0	0	0	0	0	0	0	0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	(33.645)	(14.83)	(16.31)	(31.141)	(12.400)	(5.500)	(17.900)	(19.600)	(14.850)	(6.100)
Net Margin	*0.0%*	-	-	-	-	-	-	-	*-135.6%*	*-30.3%*
Reported EPS	**(5.69)**	**(2.07)**	**(2.12)**	**(4.19)**	**(1.61)**	**(0.68)**	**(2.27)**	**(2.39)**	**(1.77)**	**(0.71)**
YOY Growth	-	-	-	-	-	-	-	-	-	-
Wt. Ave Shares Outstanding	5.9	7.2	7.7	7.4	7.7	8.1	7.9	8.2	8.4	8.6

Source: Zacks Investment Research, Inc. *Jason Napodano, CFA*

Addex Therapeutics Ltd.
Discounted Cash Flow Model

YEAR	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022
Dipraglurant Model (Assumes Partnership)											
Royalties On WW Sales (PD-LID)	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 4	CHF 16	CHF 40	CHF 77	CHF 97	CHF 117
YoY Growth	-	-	-	-	-	-	*326%*	*153%*	*92%*	*27%*	*20%*
Royalties On WW Sales (NPD)	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 6	CHF 14	CHF 21	CHF 29	CHF 36
YoY Growth	-	-	-	-	-	-	-	*138%*	*52%*	*38%*	*24%*
ADX71149 Model (Existing Partnership With J&J)											
Royalties On WW Sales (Schizo)	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 10	CHF 26	CHF 48	CHF 68	CHF 89
YoY Growth	-	-	-	-	-	-	-	*153%*	*85%*	*41%*	*32%*
Royalties On WW Sales (MDD/Anxeity)	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 5	CHF 10	CHF 32	CHF 44
YoY Growth	-	-	-	-	-	-	-	-	*108%*	*212%*	*39%*
Collaborative Payments / Licenses	CHF 7	CHF 6	CHF 11	CHF 21	CHF 26	CHF 51	CHF 1	CHF 11	CHF 1	CHF 11	CHF 1
YoY Growth	-	-	-	-	-	-	*-98%*	*1000%*	*-91%*	*1000%*	*-91%*
Pipeline	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0
YoY Growth	-	-	-	-	-	-	-	-	-	-	-
Total Revenues (in Millions)	**CHF 7**	**CHF 6**	**CHF 11**	**CHF 21**	**CHF 26**	**CHF 55**	**CHF 33**	**CHF 95**	**CHF 157**	**CHF 236**	**CHF 286**
R&D	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 20*	*CHF 22*	*CHF 23*
% SG&A	*278.6%*	*333.3%*	*181.8%*	*95.2%*	*76.9%*	*36.6%*	*61.0%*	*20.9%*	*12.7%*	*9.3%*	*8.0%*
SG&A	*CHF 5*	*CHF 5*	*CHF 6*	*CHF 6*	*CHF 7*	*CHF 7*	*CHF 8*	*CHF 10*	*CHF 12*	*CHF 14*	*CHF 16*
% R&D	*71.4%*	*88.3%*	*50.9%*	*28.6%*	*25.0%*	*12.8%*	*24.4%*	*10.5%*	*7.6%*	*5.9%*	*5.6%*
EBIT	CHF -18	CHF -19	CHF -15	CHF -5	CHF -1	CHF 28	CHF 5	CHF 65	CHF 125	CHF 200	CHF 247
Depreciation and amortization	*CHF 3*	*CHF 3*	*CHF 3*	*CHF 3*	*CHF 3*	*CHF 4*	*CHF 4*	*CHF 4*	*CHF 4*	*CHF 4*	*CHF 4*
EBITDA	**(CHF 21)**	**(CHF 22)**	**(CHF 18)**	**(CHF 8)**	**(CHF 4)**	**CHF 24**	**CHF 1**	**CHF 62**	**CHF 121**	**CHF 196**	**CHF 243**
EBITDA margin	-	-	*-161.8%*	*-39.5%*	*-15.0%*	*44.2%*	*3.6%*	*64.7%*	*77.2%*	*83.1%*	*85.0%*
Cash Taxes	*CHF 0*	*CHF 0*	*CHF 0*	*CHF 0*	*CHF 0*	*CHF 2*	*CHF 0*	*CHF 19*	*CHF 42*	*CHF 69*	*CHF 85*
Tax Rate	-	-	-	-	-	*10.0%*	*20.0%*	*30.0%*	*35.0%*	*35.0%*	*35.0%*
Balance Sheet											
Investing Activities	CHF 1	CHF 1	CHF 2	CHF 2	CHF 2	CHF 2	CHF 3	CHF 3	CHF 3	CHF 3	CHF 4
Working Capital	CHF -1	CHF -2	CHF -2	CHF -3	CHF -3	CHF -4	CHF -4	CHF -5	CHF -5	CHF -6	CHF -6
Finance Expense	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1	CHF 1
Capital employed	CHF 1	CHF 1	CHF 1	CHF 0	CHF 0	CHF -0	CHF -1	CHF -1	CHF -1	CHF -1	CHF -2
ROIC	*1750.0%*	*2573.3%*	*2920.0%*	*2000.0%*	*#DIV/0!*	*11081.3%*	*955.6%*	*8729.9%*	*12486.4%*	*16027.5%*	*16489.0%*
Discounted Cash Flow											
Net Income	CHF -21	CHF -22	CHF -18	CHF -8	CHF -4	CHF 22	CHF 1	CHF 43	CHF 79	CHF 128	CHF 158
Depreciation and amortization	CHF 3	CHF 3	CHF 3	CHF 3	CHF 3	CHF 4	CHF 4	CHF 4	CHF 4	CHF 4	CHF 4
Net Cash Changes	CHF 1	CHF 1	CHF 1	CHF 0	CHF 0	CHF -0	CHF -1	CHF -1	CHF -1	CHF -1	CHF -2
Post-Tax Operating Cash Flow	CHF -17	CHF -19	CHF -14	CHF -5	CHF -1	CHF 25	CHF 4	CHF 46	CHF 81	CHF 130	CHF 161
Dividends	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0	CHF 0
Free Cash Flow	**(CHF 17)**	**(CHF 19)**	**(CHF 14)**	**(CHF 5)**	**(CHF 1)**	**CHF 25**	**CHF 4**	**CHF 46**	**CHF 81**	**CHF 130**	**CHF 161**

Assumptions	
Beta:	N/A
Equity Risk Premium:	6.5%
Risk Free Rate:	1.5%
Probability Adjustment:	150%
Discount Rate:	**17.8%**

Valuation	
10-Year Model:	CHF 55
Terminal Growth:	2%
Terminal Value:	CHF 166
Cash - Debt:	CHF 34
Firm Value:	**CHF 188**

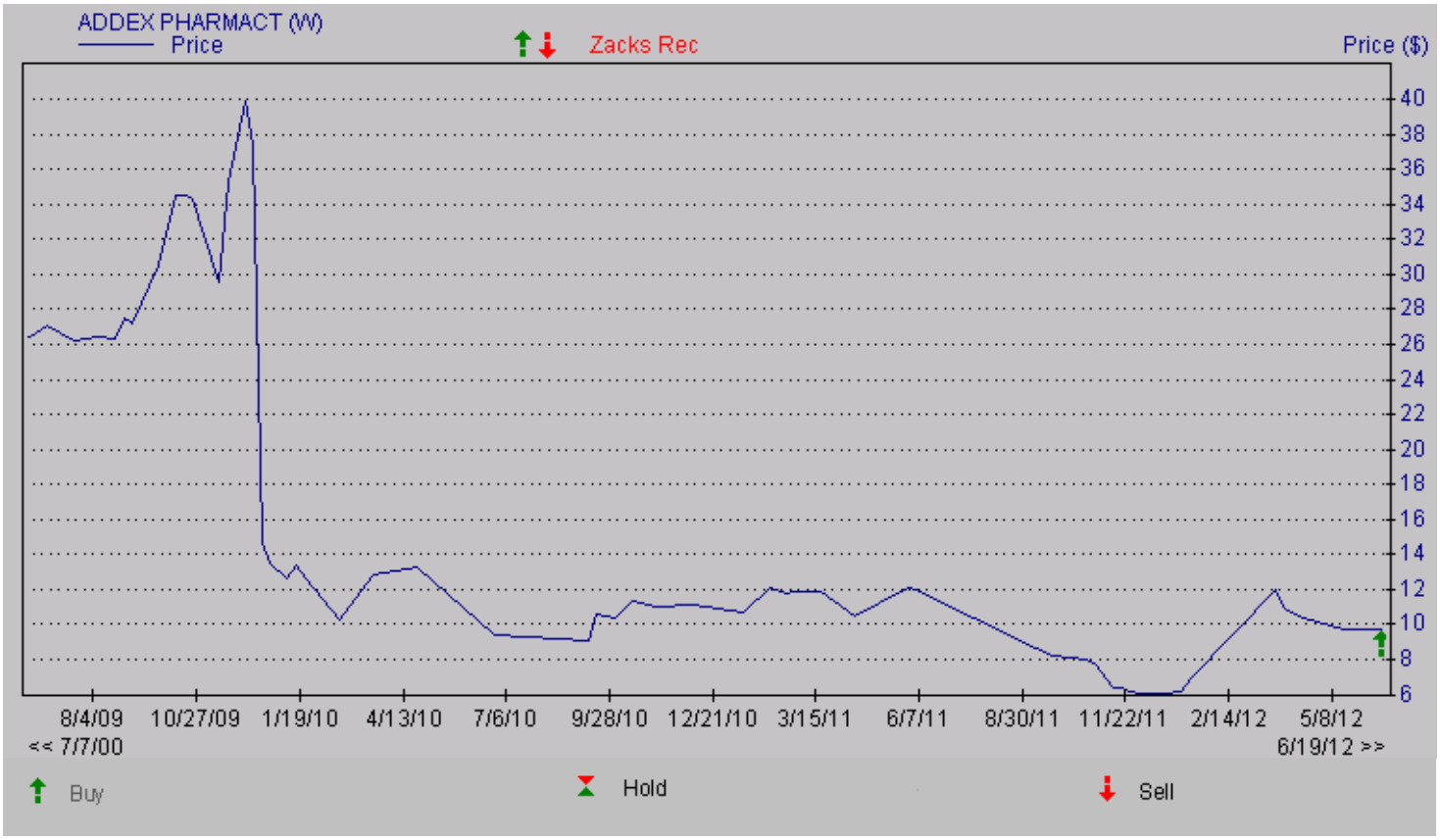
Price Target	
Basic Outstanding:	7.8
Warrants & Options:	1.4
Dilution:	0
Adjusted Shares:	9.2
Target Price:	CHF 20

CHF/CHF
0.95

ADDXF
$21.39

Source: Zacks Investment Research, Inc. *Jason Napodano, CFA*

HISTORICAL ZACKS RECOMMENDATIONS



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