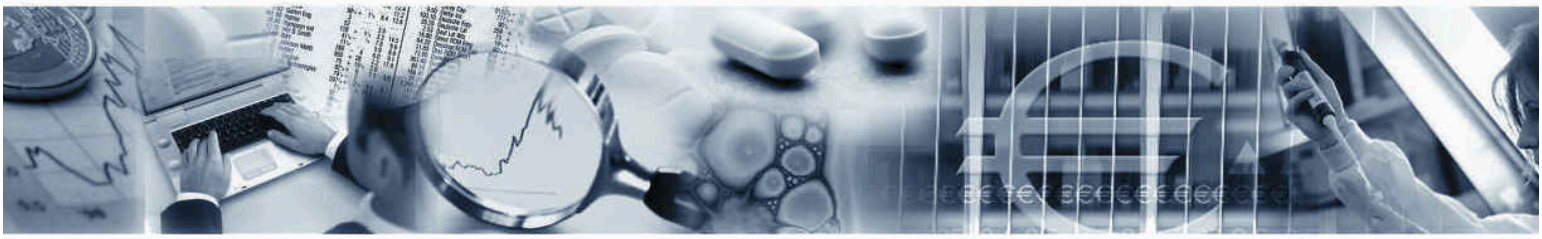


Initiating Coverage Report

Addex Therapeutics

Luctor et Emergo

Chief Research Analyst

Marcel Wijma MSc

+1 (917) 460 6185 (US)

+31 (6) 8489 2954 (NL)

m.wijma@leeuwenhoeck.com

http://www.leeuwenhoeck.com

Date: 18 October 2017

Name:	**Addex Therapeutics**
Country:	**Switzerland**
Price:	**CHF 2.10**
ISIN Code:	**CH0029850754**
Reuters Code:	**ADXN.SW**
Market Cap (CHF m):	**32.6**
EV (CHF m):	**28.7**
Cash & cash eq. (CHF m):	**3.9**
Shares outstanding (m):	**15.5**
Volume:	**24,685**
Free float:	**89.6%**
52-week Range:	**1.70-2.53**

	2014A	2015A	2016A
Total Revenues	1.03	0.79	0.40
Net (Loss)/Profit	(1.77)	(4.20)	(3.10)
Net loss per share (cents)	(0.18)	(0.39)	(0.28)
R&D costs	0.9	1.78	2.40
Cash increase/(decrease)	(0.95)	0.70	(1.23)
Cash and marketable sec.	1.98	2.63	1.40

Contents

Executive Summary 4

Company Profile 6

Technology: Allosteric Modulation 8

Parkinson's Disease and Levodopa Induced Dyskinesia 11

Pipeline: Focus on CNS related indications 16

SWOT Analysis 27

Patent Position 28

Financials 30

Management Capabilities 32

Valuation 35

Near Term Milestones 40

Competitive Landscape 41

Executive Summary

- Addex Therapeutics is a Swiss based biopharmaceutical company that is developing innovative oral therapies with a focus on neurological disorders. Addex' lead program is in preparation to start a phase III study for levodopa-induced dyskinesia associated with Parkinson's disease (PD-LID). Addex has a proprietary small molecule allosteric modulator discovery platform. The company currently has 5 drug programs in or close to the start of clinical development and another 6 in preclinical development.

- The company's proprietary allosteric modulation technology offers significant advantages over classical drugs by demonstrating better pharmacological selectivity and consequently may offer better safety and tolerability at a similar level of efficacy. Also, allosteric modulators with lower potency can be effective in situations where a similar potency orthosteric modulator fails. Moreover, as Addex focuses specifically on those therapeutic targets that are clinically validated and whose existing products have well-established markets, the risk profile of its drug pipeline is relatively low.

- Addex' lead product is dipraglurant that successfully completed a phase IIa POC trial in Parkinson's disease levodopa induced dyskinesia (PD-LID). The drug is currently being prepared to enter a Phase III trial for PD-LID with the support from the Michael J Fox Foundation for Parkinson's Research. In addition to funding more than USD 600 million in research to date, the Foundation has fundamentally altered the trajectory of progress toward a cure.

- Another promising program is ADX71441 in addiction. ADX71441 is a novel, first-in-class, oral, small molecules that has demonstrated excellent preclinical efficacy and tolerability in several rodent models of pain, anxiety, addiction and OAB and have also proven efficacy in a genetic model of Charcot-Marie-Tooth Type 1A disease (CMT1A)

- The Company's current cash position is CHF 3.6 million and we expect the company to raise additional funds in the near future or sign a lucrative partnering deal. This should be sufficient to carry out the further development of its pipeline and the important commencement of the Phase IIb trial with dipraglurant in PD-LID. Since its inception in 2002, the company raised CHF 280 million with the help of top tier investors from the US and Europe. In addition the company has received more than CHF50 million in cash inflows from partnering activities.

- **Based on NPV based valuation, we believe that Addex Therapeutics is substantially undervalued at the current share price of CHF 2.14. Using our valuation model and taking into account the future revenues from its late stage clinical pipeline as well as potential partnerships, the company's current total value should be CHF 179 million, or CHF 11.50 per share. This represents a substantial upside from the current share price.**

Company Profile

Addex Therapeutics is a Swiss based biopharmaceutical company that is developing an emerging class of oral small molecule drugs known as allosteric modulators. Allosteric modulators target a specific receptor or protein and alter the effect of the body's own signaling molecules on that target through a novel mechanism of action. The company enjoyed first-mover advantage in the process of discovering and developing allosteric modulators. Addex has developed an allostery-biased library of more than 70,000 compounds and biological assays which enable detection, optimization and confirmation of the mechanism of action of allosteric compounds. Currently, Addex has a diverse pipeline of proprietary compounds that cater to a number of major diseases. The platform is broadly applicable and has generated several molecules for indications with significant commercial potential with a focus on central nervous system (CNS) disorders with orphan drug potential. Its lead product is dipraglurant that successfully completed a phase IIa POC trial in Parkinson's disease levodopa induced dyskinesia (PD-LID). The drug is currently being prepared to enter a Phase III trial for PD-LID with the support from the Michael J Fox Foundation for Parkinson's Research. In addition to funding more than USD 600 million in research to date, the Foundation has fundamentally altered the trajectory of progress toward a cure.

Business Strategy & Partnerships

Addex' current business strategy includes the possibility of entering into collaborative arrangements with third parties to complete the development and commercialization of its proprietary drug candidates, such as the current partnership with Janssen Pharmaceuticals, Inc. ("Janssen"), a subsidiary of Johnson & Johnson, for ADX71149 in the treatment of epilepsy and other undisclosed CNS disorders. Addex also received three grants from The Michael J. Fox Foundation for Parkinson's Research, two (totaling USD 1.9 million) for the development of dipraglurant (ADX48621) in the treatment of PD-LID and one (USD 835K) for the discovery of TrkB small molecule allosteric modulators, as well as grants from the Swiss Commission for

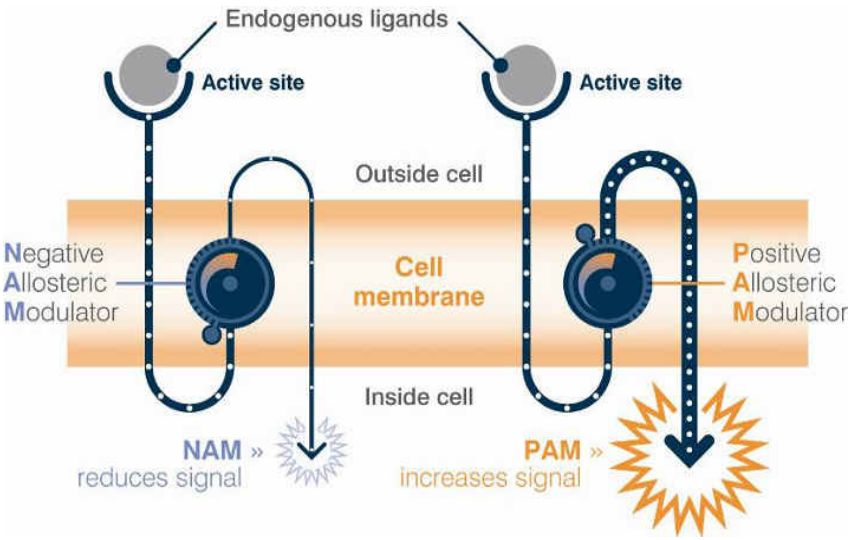
Technology and Innovation (CTI) to advance the preclinical profiling of allosteric modulator therapeutics for neurodegenerative and psychiatric diseases.

Technology: Allosteric Modulation

Allosteric modulation is a mechanism to control the function of proteins. This is done by binding an effector molecule, called an allosteric modulator, at the protein's allosteric site i.e., at the site other than the protein's active site. Natural ligands, which are produced by the body, bind to a receptor's active site. Most of the available drugs compete with these natural ligands to bind to the active site. However, since allosteric modulators bind to the protein at a site other than the active site, they do not compete with the body's natural ligands. They can bind with the receptor even in the presence of natural ligand-receptor binding. As a result, allosteric modulators act as a dimmer switch controlling the level of activation or inhibition, rather than switching the receptor on or off, allowing the body to retain its natural control over receptor activation. The allosteric modulator affects the conformation of the receptor and hence changes its shape, and by doing so it affects the signals sent by the receptor intracellularly, resulting in the alteration of the protein's activities.

There are two types of effectors:

- Allosteric activators: Effectors that increase the protein's activity.
- Allosteric inhibitors: Effectors that decrease the protein's activity.



Source: Addex Therapeutics

Advantages of allosteric modulation

Allosteric modulators with lower potency can be effective in situations where a similar potency orthosteric modulator fails. This is because allosteric modulators do not compete with the natural ligands, in contrast to orthosteric drugs, as they bind with the receptor on the allosteric site. Allosteric modulators are highly selective. Receptors comprise multiple functional domains and transmit several message signals to the cell. While an orthosteric drug competes with the natural ligand for it's highly conserved binding site, allosteric modulators use a different binding site, the allosteric site, which has not been subjected to conservatory evolutionary pressure. Therefore, allosteric modulators are found to be highly selective for one receptor of interest, and thus have the potential to have an increased safety profile compared to other drugs.

Since allosteric modulators bind to the receptor at a site other than the active site, the natural biological rhythm is preserved, which is lost/altered in the case of orthosteric modulators.

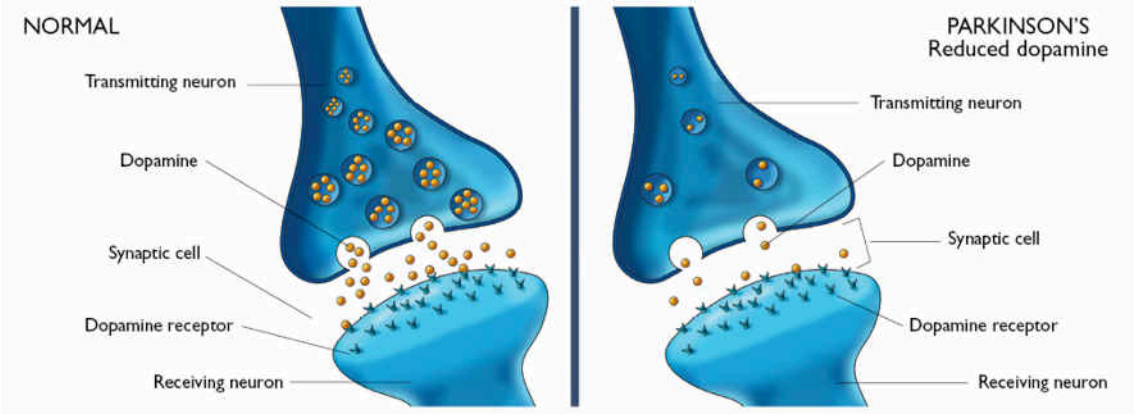
Addex has developed proprietary screening systems to identify allosteric modulators that modify the molecular response of the therapeutic target. These systems screen G-Protein Coupled Receptors (GPCRs) and non-GPCR drug targets, and provide the pharmacological data that allow lead optimization through medicinal chemistry. The company has patented several aspects of these technologies while keeping certain others as trade secrets to maintain its competitive advantage.

GPCRs are the largest protein family known, members of which are involved in all types of stimulus-response pathways, from intercellular communication to physiological senses. The diversity of functions is matched by the wide range of ligands recognized by members of the family, from photons (rhodopsin, the archetypal GPCR) to small molecules (in the case of the histamine receptors) to proteins (for example, chemokine receptors). Drugs targeting members of this integral membrane protein superfamily, which transmit chemical signals into a wide array of different cell types, represent the core of modern medicine. They account for the majority of bestselling drugs and about 40% of all prescription pharmaceuticals on the market.

Glutamate, like dopamine and serotonin, is a key signaling molecule (neurotransmitter) in the human brain involved in the control of multiple brain functions including mood, memory, perception and motor function. Too much glutamate can lead to seizures and the death of brain cells. Too little glutamate can cause psychosis, coma and death. Glutamate exerts these effects by interacting with many receptors in the brain, especially NMDA, AMPA and kainate receptors. In addition to these primary receptors, glutamate triggers other receptors, termed metabotropic because they adjust the amount of glutamate that cells release rather than simply turning glutamate transmission on or off. Eight types of metabotropic glutamate receptors (mGluR), each with different functions, have been identified. These mGluRs are attractive targets for drug treatment because of their ability to fine-tune glutamate signaling. Research shows that mGluR drugs have potential for the treatment of schizophrenia, anxiety, Parkinson's disease, fragile X syndrome, Alzheimer's disease, depression and post-traumatic stress disorder. Addex has discovered selective orally available small molecule allosteric modulators for each of the eight subtypes of mGluR, as well as for the metabotropic GABAb receptor. GABA (gamma-aminobutyric acid) is the main inhibitory neurotransmitter in the central nervous system (CNS). GABA-ergic inhibition is seen at all levels of the CNS, including the hypothalamus, hippocampus, cerebral cortex and cerebellar cortex. GABA pathways are abundant in the brain, with 50% of the inhibitory synapses in the brain being GABA mediated.

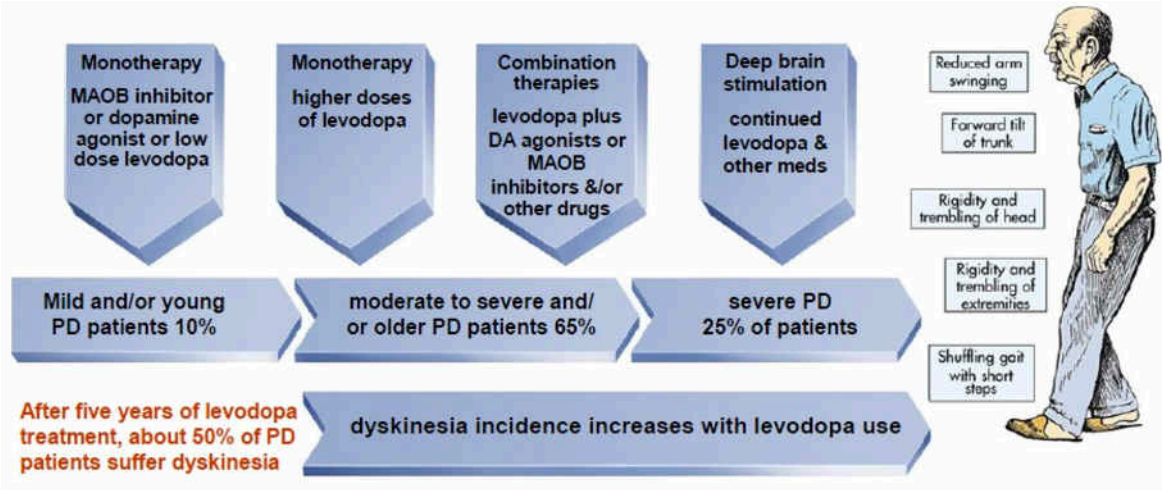
Parkinson's Disease and Levodopa Induced Dyskinesia

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.



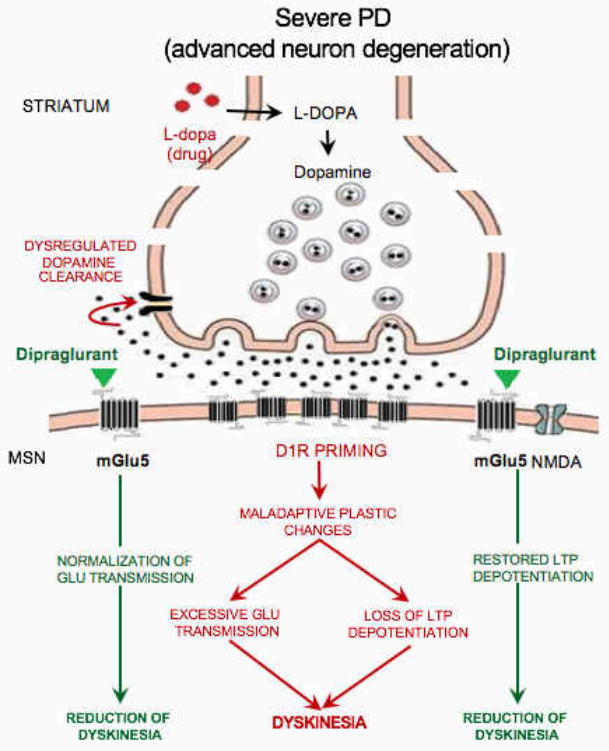
There are approximately 1,000,000 patients with Parkinson disease in the US, with 50,000 to 60,000 more diagnosed each year. Worldwide, there are approximately 4 million individuals afflicted (2.7 million in the US, Japan, and the 5 major European markets). Since the incidence of PD increases with age (the average age of onset is 60), the number of patients is likely to climb as the population of older patients grows.

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 which 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



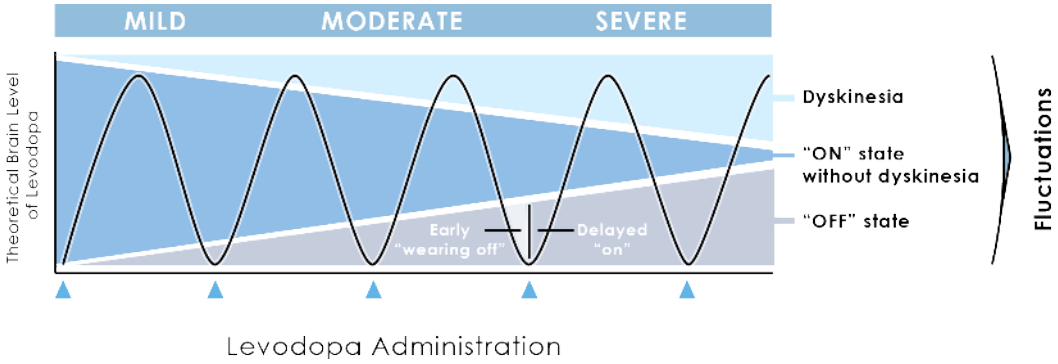
The most commonly used DRT therapy is Levodopa. It has been available for over 30 years. Levodopa (L-DOPA) is converted into dopamine in the dopaminergic neurons by dopa decarboxylase. The administration of levodopa temporarily diminishes the motor symptoms associated with the lack dopamine in the substantia 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 dosed with Levodopa to prevent Levodopa 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. After a decade on therapy, almost all PD patients require high doses of Levodopa, 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.



Long-term Levodopa use is invariably associated with the development of dyskinesias that become as disabling as the PD symptoms themselves. Dyskinesias result from the neurodegenerative process that underlies PD. The dopamine replacement does not lead to dyskinesia per se, but is thought to lower the triggering threshold for symptoms, as the neurodegeneration progresses. 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

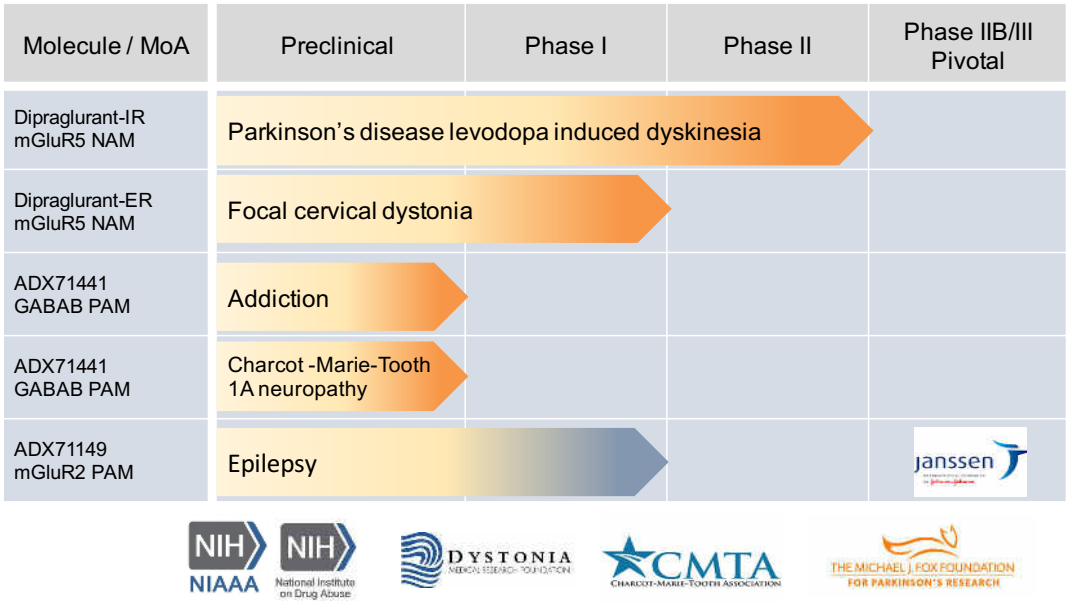
Levodopa plasma concentrations (peak-dose dyskinesia), although it may also occur when plasma concentrations of Levodopa rise and fall (diphasic dyskinesia) or during off-time (off-period dystonia).



Recently, the first treatment for PD-LID was approved by the FDA (GOCOVRI from Adamas Pharmaceuticals). Approximately 50% of PD patients will experience LID after 3 years on L-DOPA therapy. The number rises to 90% after 9 to 15 years on Levodopa 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). Currently, the most common treatment for LID is to reduce the dose of Levodopa. However, reducing the dose of Levodopa causes increased parkinsonism and worsening motor performance. Therefore, once established, LID becomes difficult to treat.

Pipeline: Focus on CNS related indications

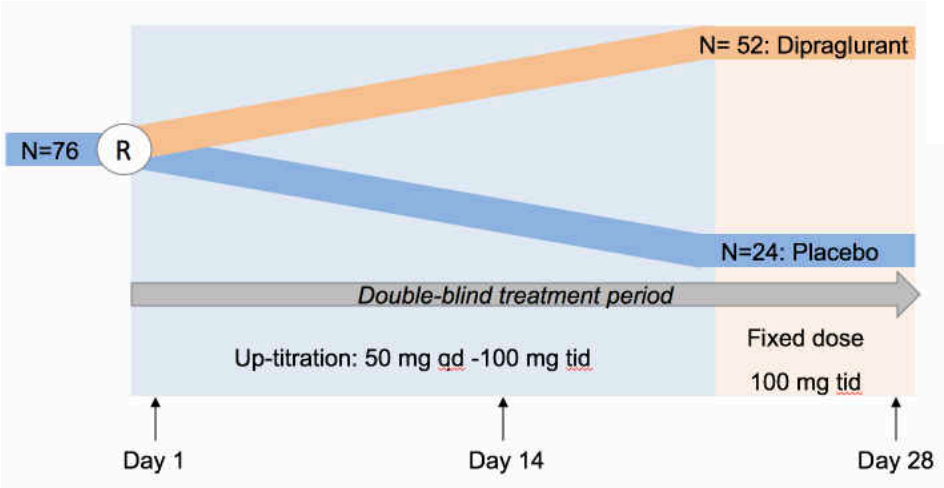
Using its allosteric modulator discovery capabilities, Addex has developed a pipeline of proprietary clinical and preclinical stage drug candidates. In 2012, the company revised its business strategy to focus on the advancement of allosteric modulators of four receptors, namely the metabotropic glutamate receptor 5 (mGlu5), the metabotropic glutamate receptor 2 (mGlu2), and the gamma-aminobutyric acid subtype B receptor (GABAb).



Dipraglurant IR in PD-LID

Addex' lead product in development is Dipraglurant IR (immediate release). Dipraglurant is a highly selective oral small molecule, which inhibits the metabotropic glutamate receptor 5 (mGluR5), and has potential to be used in combination with levodopa or dopamine agonists for treatment of Parkinson's disease (PD). The company is primarily testing dipraglurant for the treatment of PD levodopa-induced dyskinesia (PD-LID). In a double-blind, placebo-controlled, US and European Phase II study in PD-LID, data showed that dipraglurant met the primary objective of the study by exhibiting a good safety and tolerability profile. Dipraglurant also demonstrated a statistically significant reduction in LID severity with both 50 and 100 mg doses. Dipraglurant

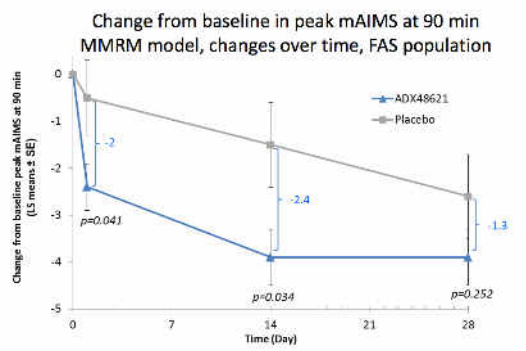
reduced dystonia severity in addition to chorea, the two major LID components. The trial was supported by a grant from The Michael J. Fox Foundation for Parkinson's Research.



N= number of patients; R= randomization
Coordinating Investigator: Prof Olivier Rascol at University Hospital, Toulouse, France

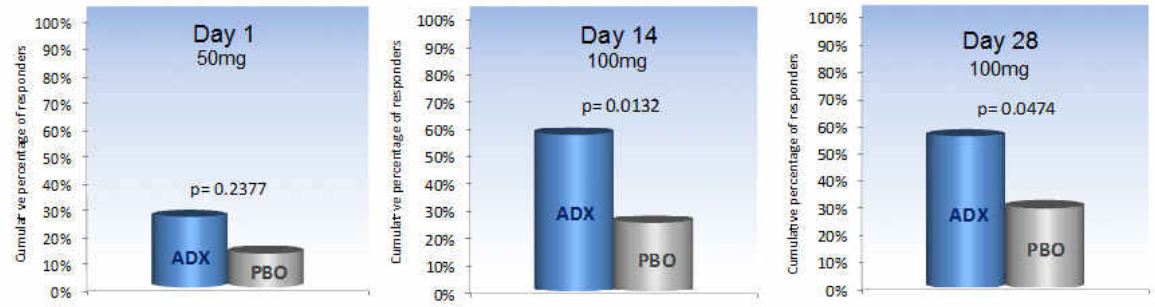
The primary endpoint was to evaluate safety and tolerability of dipraglurant IR in PD-LID patients after 4 weeks treatment. Secondary or exploratory endpoints were to evaluate effects on dyskinesia and motor symptoms, and also to identify an effective dose of dipraglurant IR. The randomized (2:1 dipraglurant IR: placebo), double blind, placebo-controlled trial was conducted at 25 sites in the US, Germany, France and Austria. A total of 76 patients were enrolled in the trial. Patients stayed on a constant dose of levodopa (300 – 1,500 mg/day) and were given dipraglurant IR or placebo together with levodopa therapy for a duration of 4 weeks. The patients followed a dose titration regimen. In the first two weeks patients received 50 mg dipraglurant IR up to three times daily until day 14. From day 14 to day 28 the dose was gradually increased to 100 mg three times daily. LID severity was measured on Day 0 (pre randomization, baseline), and on treatment Days 1 (50 mg, one dose), 14 (100 mg, 3x daily) and 28 (100 mg, 3x daily) by mAIMS (modified Abnormal Involuntary Movement Scale) performed every 30 minutes for 3 hours following a single usual levodopa dose taken around midday. Seven

body areas were scored from 0 (no LID) to 4 (severe LID) for a total 28 point score every 30 minutes. Additionally, in the home setting, patients collected diary data of "on", "off" and sleep time for 48 hours each week during Week -1 (baseline) and all 4 treatment weeks. Levodopa efficacy was evaluated during AIMS testing on Days 0, 1, 14, and 28 using UPDRS (Unified Parkinson's Disease Rating Scale) Part III (clinician scored motor evaluation). Overall UPDRS scoring was performed at screening and Day 28. On Day 28, Patient and Clinical Global Impression of Change (PGIC and CGIC) in dyskinesia and Parkinson's disease were collected. After 4 weeks, 47 out of 52 (90%) of the patients on dipraglurant IR completed the trial.



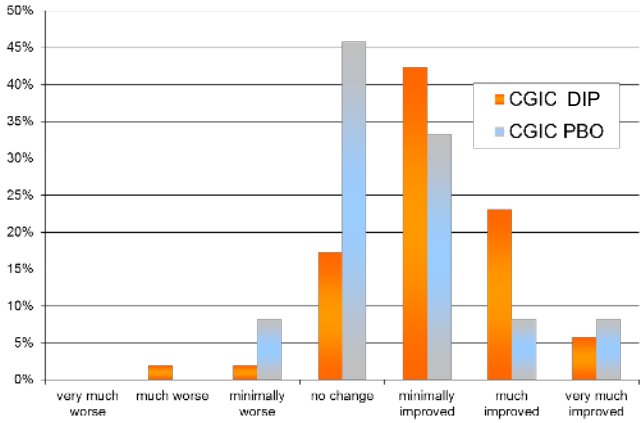
Mean % change of peak AIMS from baseline		
Midday dose	Dipraglurant	Placebo
Day 1 (50 mg)	19.9%	4.1%
Day 14 (100 mg)	32.3%	12.6%
Day 28 (100 mg):	31.4%	21.5%

Responder analysis (≥30% change of peak AIMS from baseline)			
Midday dose	Dipraglurant	Placebo	p-value
Day 1 (50 mg)	26.0%	12.5%	0.2377
Day 14 (100 mg)	56.9%	25.0%	0.0132
Day 28 (100 mg):	55.3%	29.2%	0.0474



This study found that dipraglurant therapy resulted in substantial improvements on multiple efficacy endpoints. Patients taking dipraglurant had significant reductions in modified Abnormal Involuntary Movement Scale (mAIMS) during peak levodopa concentrations and this response was maintained during the 3-hour post-dosing period. The complete data on this endpoint is shown in the graphs below. Participants receiving 50 mg dipraglurant on day 1 had a 19.9% reduction in mAIMS as compared to 4.1% for placebo (p = 0.042). After being titrated up to a 100 mg dose of dipraglurant, participants experienced a 32.3% reduction in mAIMS as compared to 12.6% for placebo (p = 0.034) on day 14.

When looking at Clinical Global Impression of Change (CGIC), there was even a greater improvement in dyskinesia with dipraglurant according to clinicians (p<0.05). CGIC is a relatively simple scale that reflects the everyday clinical practice. The assessment is done by the treating physician which makes it a more objective assessment than the more subjective mAIMS.



	Dipraglurant	Placebo
Improved (p<0.05)	71.2%	49.9%
No change	17.3%	45.8%

Source: Addex Therapeutics

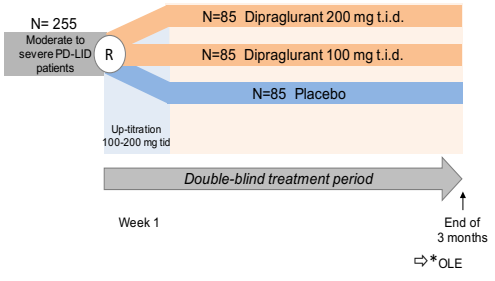
Amongst the most widely used of extant brief assessment tools in psychiatry, the CGI is a 3-item observer-rated scale that measures illness severity (CGIS), global improvement of change (CGIC)

and therapeutic response. The illness severity and improvement sections of the instrument are used more frequently than the therapeutic response section in both clinical and research settings.

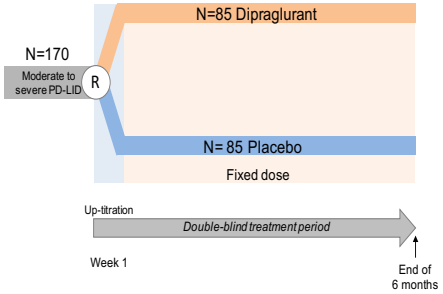
The Early Clinical Drug Evaluation Program (ECDEU) version of the CGI is the most widely used format, and asks that the clinician rate the patient relative to their past experience with other patients with the same diagnosis, with or without collateral information. The CGI has proved to be a robust measure of efficacy in many clinical drug trials, and is easy and quick to administer, provided that the clinician knows the patient well.

The company plans to launch a pivotal development program with the first clinical trial starting in 2018H2 to support regulatory filings for dipraglurant. Addex has received USD 1 million in grants from the Michael J. Fox Foundation to support ongoing preparations for this pivotal trial. This randomized, double blind, placebo-controlled Phase IIB study will assess the safety and efficacy of dipraglurant for the treatment of 255 patients with moderate to severe levodopa-induced dyskinesia. Patients will be randomized 2:1 to receive dipraglurant or placebo three times daily, with levodopa treatment regimens remaining consistent. The optimal dose will be determined via titration over a two-week period, followed by 11 weeks of a maintenance dose. The primary endpoint is change in Unified Dyskinesia Rating Scale (UDysRS) Part IV. The secondary endpoints include change in clinician-scored Unified Parkinson's disease rating scale (UPDRS) Part III, patient diaries for on and off time.

Dipraglurant 1st Pivotal LID Study (203)



Dipraglurant 2nd Pivotal LID Study (301)

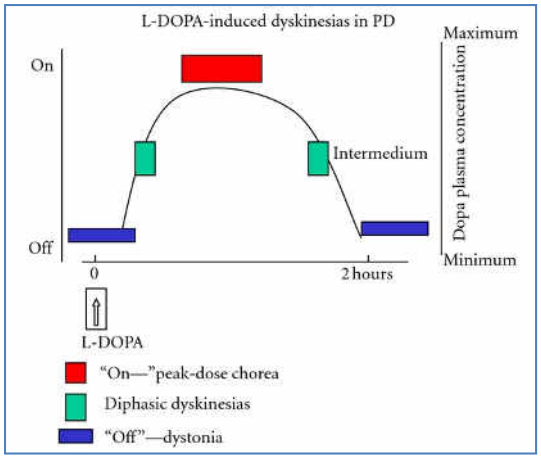


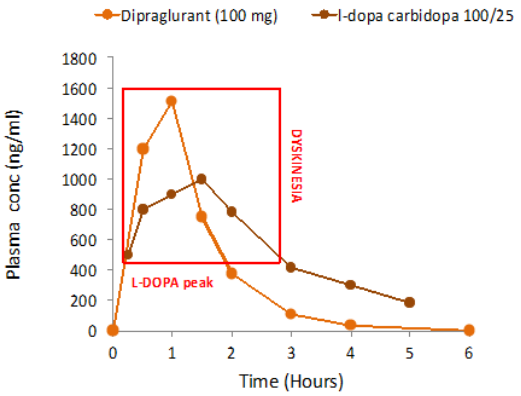
N= number of patients; R= randomisation; LID= L-Dopa induced dyskinesia; OLE = open label extension

Addex has specifically developed an immediate release (IR) formulation of dipraglurant that mimics the uptake of levodopa in patients, since peak-dose dyskinesia is the most frequent levodopa-induced dyskinesia. Levodopa has to be given 3-4 times a day due to its relatively short half-life. Peak plasma concentrations are reached 60 to 90 minutes after dosing, when peak-dose dyskinesia occurs. Dipraglurant IR, which is taken together with levodopa, has a rapid onset of action similar to levodopa, and rapid clearance that reduces unnecessary drug exposure and unwanted side effects. This profile is ideal to offset unwanted peak-dose dyskinesia.

The immediate release (IR) formulation is ideally suited for acute treatment of PD-LID because:

- Its pharmacokinetic profile is similar to levodopa so drug is delivered precisely when needed.
- Its rapid onset of action is ideal for dyskinesia which can occur within 30 minutes of dosing.
- The rapid clearance reduces unnecessary drug exposure, between levodopa doses and should reduce side effects as a result.
- The PK characteristics of dipraglurant IR have potential to give flexibility of use, which is common practice and desirable in PD treatment

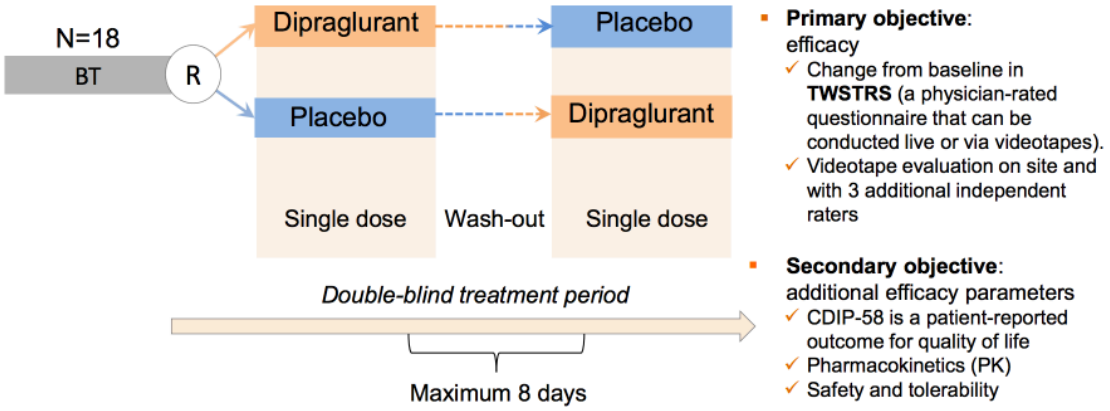




Dipraglurant has received FDA orphan drug designation for levodopa-induced dyskinesia associated with Parkinson's disease.

In October 2016, Addex announced that it will conduct a Phase IIa POC Study of dipraglurant in focal cervical dystonia (CD). Addex intends to initiate this study in 2018Q1, and anticipates the availability of data in 2018Q4.

The study was developed with support from the Dystonia Medical Research Foundation and in collaboration with investigators from the Dystonia Coalition, an international network of experts devoted to advancing research in dystonia. Addex's Phase IIa Proof of Concept study will include 18 focal CD patients who are currently sub-optimally treated with botulinum toxin (BoNT). The single center study will be double-blinded and placebo-controlled. A single dose of dipraglurant will be administered in a crossover design.



N= number of patients; R= randomisation; BT= patients treated with botulinum toxin.
Study duration of 5 weeks with screening period of 4 weeks.
Coordinating Investigator: Prof Jinnah at Emory University School of Medicine, Atlanta.

CD is the most prevalent form of dystonia; recent international prevalence estimates place the number of CD patient in the US between 50,000 and 100,000 - a range which is much higher than previously reported and considers the large portion of undiagnosed population. CD has been demonstrated to have a significant impact on quality of life. Current treatment options for focal CD include botulinum toxin BoNT injections, which generally reduce muscle spasms

temporarily for a few months. However, the interval between BoNT injections is usually longer than the duration of action, leaving patients with sub-optimal symptom relief towards the end of the treatment for weeks. In addition, most patients rarely experience any symptom free days

The combined peak US annual sales potential (excluding disease modification) for the PD indications alone is estimated to be in the range of USD 1.8 billion to USD 2.7 billion, (source: Michael J Fox Foundation). Datamonitor market research with PD specialists in the US, EU, China and India show that dipraglurant has an attractive product profile and can capture over USD 1 billion in annual revenues if fully exploited in PD indications.

ADX71441: Addiction and Charcot-Marie-Tooth Type 1A

ADX71441 (GABAB PAM) is the third program drug in Addex' proprietary allosteric modulation technology platform and is targeted for the treatment of Charcot-Marie-Tooth disorder and addiction (alcohol use disorder, cocaine and nicotine addiction). Researchers have shown that GABAB receptor agonists such as baclofen are effective in reducing drug self-administration, cravings, and anxiety, and thus promote abstinence. Baclofen, also known as chlorophenibut, is a conventional (orthosteric) stimulator (agonist) of the GABAB receptor, and is primarily used to treat spasticity and is in early development for treating alcoholism.

Charcot-Marie-Tooth (CMT) disease encompasses a heterogeneous group of inherited, progressive, chronic peripheral neuropathies. CMT type 1A (CMT1A), the most common type of CMT, is an orphan disease affecting at least 125,000 people in Europe and the U.S. The genetic mutation responsible for CMT1A is a duplication of the PMP22 gene coding for a peripheral myelin protein. Overexpression of this gene causes degradation of the neuronal sheath (myelin) responsible for nerve dysfunction, followed by loss of nerve conduction. As a result of peripheral nerve degradation, patients suffer from progressive muscle atrophy of legs and arms causing walking, running, balance problems and abnormal hand functioning. CMT1A patients end up in wheelchairs in at least 5% of cases. They might also suffer from mild to moderate sensitive

disorders. First symptoms usually appear during adolescence and will progressively evolve through patients' life.

In July 2016, Addex published that ADX71441 demonstrated positive results in a highly translational preclinical model of spasticity. ADX71441 was evaluated in a leading model of muscle spasticity, the rat transection spinal cord injury (SCI) model. Muscle hyperactivity was measured by a translational electrophysiological marker, the rate- dependent depression of the Hoffmann's reflex (H-reflex), a measurement that is also used to evaluate spasticity in patients. The SCI procedure significantly induced spasticity in rats within 5 weeks ($P < 0.001$; Mann-Whitney test), after which a single intravenous administration of ADX71441 (1, 3 or 10 mg/kg) or vehicle was administered and the degree of spasticity response was measured.

In preclinical studies, Addex has demonstrated the efficacy of ADX71441 in animal models of alcohol use disorder and nicotine withdrawal. In particular, the Company has conducted three preclinical alcohol use studies in collaboration with the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The Company also recently announced positive results from a study evaluating ADX71441 in a primate model of cocaine addiction, which was conducted in collaboration with the National Institute of Drug Abuse (NIDA). In rhesus monkeys, pre-treatment with ADX71441 dose-dependently reduced cocaine self-administration to roughly 10% of control values. This effect was observed without any concomitant effect on food intake, suggesting that ADX71441 is not broadly affecting the reward circuitry in the brain. Addex plans to launch a Phase I study comparing the safety and efficacy of ADX71441 to baclofen. Baclofen is approved for the treatment of spasticity in the US, but not alcohol use disorder (AUD), despite receiving a temporary registration in France for AUD.

In October the US National Institute on Drug Abuse (NIDA, a division of National Institutes of Health (NIH)) has awarded a USD 5.3 million grant to support human studies of ADX71441 for the treatment of cocaine use disorder. The grant was issued as part of the Grand Opportunity in

Medications Development for Substance-Use Disorders (U01), a cooperative agreement providing for both financial assistance and significant scientific support from the NIH to selected clinical programs. The human studies of ADX71441 will be conducted in coordination with the Friends Research Institute (FRI) and principal investigator, Dr. Frank J. Vocci. The studies are expected to begin in 2018H1.

ADX71149: Epilepsy (partnership with Janssen Pharmaceuticals)

ADX71149 is a novel, first-in-class potent, oral, small molecule positive allosteric modulator (PAM) of metabotropic glutamate receptor 2 (mGluR2), a Family C class of G Protein Coupled Receptor (GPCR). The development of ADX71149 is part of a worldwide research collaboration

and license agreement between Addex and Janssen Pharmaceuticals, to discover, develop and commercialize a novel mGluR2 PAM medication for the treatment of anxiety, schizophrenia and other undisclosed indications. Under the terms of the agreement, Addex is eligible for up to a total of EUR 112 million in milestone payments based on potential development and regulatory achievements. In addition, Addex is eligible for low double-digit royalties on sales of any mGluR2 PAM medication developed under the agreement.

Epilepsy is one of the most common serious neurological disorders, affecting about 65 million people globally (Thurman et al. 2011). It affects 1% of the population by age 20 and 3% of the population by age 75. Epilepsy describes a condition in which a person has recurrent seizures due to a chronic, underlying process. It also refers to a clinical phenomenon rather than a single disease entity, since there are many forms and causes of epilepsy. Epilepsy is a disease of the brain defined by any of the following conditions:

- at least two unprovoked (or reflex) seizures occurring more than 24 hours apart;
- one unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years;
- diagnosis of an epilepsy syndrome.

The synaptic vesicle protein 2A (SV2A) has been identified as a broad spectrum anticonvulsant target in models of partial and generalized epilepsy, and studies in animal models and human tissue suggest that changes in the expression of SV2A are implicated in epilepsy (Mendoza-Torreblanca et al. 2013; Kaminski et al. 2012). SV2A ligands include levetiracetam (Lynch et al. 2004), which is an antiepileptic drug commercialized under trademark Keppra®, approved in Europe and USA as a monotherapy or add-on therapy in patients diagnosed with epilepsy.

In the 6Hz psychomotor seizure test, a preclinical model of epilepsy considered to be the most relevant model of pharmacoresistant limbic seizures, ADX71149 demonstrated efficacy both stand alone and in combination with SV2a ligands including levetiracetam

(Metcalf et al. 2017). In particular, the data show that while seizures are reduced when mGluR2-acting compounds are administered alone, their combination with levetiracetam result in a potent reduction of doses required to produce full efficacy, which is important because higher doses of levetiracetam are associated with dose-limiting side effects, such as aggression, nervousness/anxiety, somnolence and fatigue. In this study, a fixed dose of ADX71149 was seen to increase the potency of levetiracetam, leading to an approximate 35-fold increase in its potency. Conversely, using a fixed dose of levetiracetam with varying doses of ADX71149 resulted in an approximate 14-fold increase in ADX71149 potency.

If this effect can be translated in the clinic, it will strongly support a rational polypharmacy concept in the treatment of epilepsy patients.

SWOT Analysis

Strengths	Weaknesses
Strong management with extensive relevant technical, commercial and financial expertise	Operating losses cumulating year-on-year
Vast expertise in CNS related diseases	Delay pipeline development
Relevant partnerships with credible partners	Low share price makes sizable raise challenging
Limited Cash Burn	Limited Cash

Opportunities	Threats
Profitable partnerships and license agreements with large pharmaceuticals	Uncertainty about the outcome of clinical trials
High unmet medical need	Higher level of expenditure than budgeted
Large potential markets	

Patent Position

Patent Overview: Strategy

Addex owns more than six US and 114 foreign patents and a number of pending patent applications that cover various aspects of its allosteric modulator technologies and discovery platform, including several classes of compounds which are potentially useful as modulators of mGluR5, mGluR2 and mGluR4. More specifically, the patents and patent applications cover compounds, pharmaceutical compositions, uses of compounds for medical treatments. The company's patent strategy is to file patent applications on innovations and improvements to cover a significant majority of the major pharmaceutical markets in the world. Addex typically files priority applications at the United Kingdom Patent Office to establish a priority date for the generic subject matter and examples which are available at the filing date of each invention. Subsequently, the company files international applications under the Patent Cooperation Treaty (PCT) with extra examples to support the scope of the claims. After the International Phase, Addex file patent applications in selected countries representing potential major markets for its drug candidates (National/Regional Phase).

Generally, patents have a term of twenty years from the earliest priority date, assuming all maintenance fees are paid. In some instances, patent terms can be increased or decreased, depending on the laws and regulations of the country or jurisdiction that issued the patent. Wherever appropriate and legally possible, the company aims at obtaining patent protection for novel molecules, composition of matter and uses for drugs and inventions originating from its research and development efforts, as well as new manufacturing and other processes and formulations. Addex aims to position the claims of its applications to exploit gaps in prior art.

Jointly with Janssen, Addex has 6 patent families covering compounds which are useful as mGluR2 PAMs. From these patent families, only one has not been published and all the other patent families have entered the National/Regional phase (30 months from the priority date). The

company has 2 patent families covering compounds which are useful as mGluR5 NAMs of which 56 patents have been granted. Dipraglurant is explicitly exemplified and claimed as a compound and as a pharmaceutical composition in one of the granted patent family. ADX71149 is explicitly exemplified and claimed as a compound and as a pharmaceutical composition in one of its National/Regional phase patent families.

The patent positions of pharmaceutical and biotechnology companies are uncertain and involve complex legal and factual issues. There can be no assurance that patents that have issued will be held valid and enforceable in a court of law. Even for patents that are held valid and enforceable, the legal process associated with obtaining such a judgment is time consuming and costly. Additionally, issued patents can be subject to opposition or other proceedings that can result in the revocation of the patent or maintenance of the patent in amended form, and potentially in a form that renders the patent without commercially relevant or broad coverage. Besides, Addex' competitors may be able to circumvent and otherwise design around its patents. Even if a patent is issued and enforceable, because development and commercialization of pharmaceutical products can be subject to substantial delays, patents may expire early and provide only a short period of protection, if any, following the commercialization of a product covered by any of the company's patents.

Financials

For the half year ended 30 June 2017, Addex reported a net loss of CHF 1.8 million compared to a net loss of CHF 1.5 million in the same period last year. During this period, gross R&D expenditures totaled CHF 1.1 million (2016H1: CHF 1.1 million). G&A expenses increased from CHF 0.7 million to CHF 0.8 million, mainly due increased business development and general corporate activities

Total cash at the end of June 2017 amounted to CHF 3.6 million, compared to CHF 2.3 million at the end of 2016H1. Addex received proceeds from the sale of treasury shares and to a lesser degree it received some grant awards. The company manages to keep the monthly cash burn limited to CHF 0.1-0.2 million.

In the past months, Addex has achieved some important milestones with its pipeline. Most importantly, the company made meaningful progress in preparations for the Phase IIb trial for its lead program, dipraglurant for the treatment of levodopa-induced dyskinesia associated with Parkinson's disease, which we expect to initiate in 2018H1. The company announced it will initiate a Phase IIa POC study of dipraglurant in Focal Cervical Dystonia. The study was developed with support from the Dystonia Medical Research Foundation and in collaboration with investigators from the Dystonia Coalition, an international network of experts devoted to advancing research in dystonia. Cervical dystonia is a rare disorder that is not easy for most doctors to treat. BoNT (botulinum toxin injections) provides partial relief for many patients, but it has its limitations. The study is expected to commence in 2017Q3. In 2016H2, Addex also announced the initiation of a new study with ADX88178 in a non-human primate model of cocaine addiction. This study is conducted in collaboration with the US National Institute of Drug Abuse.

Profit & Loss Statement

CHF million	2014A	2015A	2016A	2017H1
Revenues	1.0	0.8	0.411	0.227
R&D Costs	(0.9)	(1.8)	(2.461)	(1.148)
SG&A	(1.9)	(1.7)	(1.080)	(0.831)
Tax escrow account write-off	(-)	(1.2)	(-)	(-)
Operating Profit/(Loss)	(1.8)	(3.9)	(3.130)	(1.752)
Finance result	-	(0.3)	0.019	(0.035)
Net Profit/(Loss)	(1.8)	(4.2)	(3.149)	(1.787)

Consolidated statement of cash flows

CHF million	2014A	2015A	2016A
Cashflow from operating activities	(1.800)	(2.628)	(2.694)
Cash flow from investing activities	0.373	0.400	(0.01)
Cash flow from financing activities	0.472	2.930	1.492
Cash and cash equivalents at beginning of the period	2.913	1.980	2.633
Net change in cash and cash equivalents	(0.953)	0.701	(1.204)
Cash and cash equivalents at the end of the year	1.980	2.633	1.416

Management Capabilities

Addex Therapeutics is being built by seasoned biotechnology innovators. The company is led by an experienced Board and management team, which has been responsible for the development of the business and has a long term track record of developing, protecting and commercializing innovative scientific products and processes. In the past several years, Addex has been investing in developing a team of experts that have a focus on patient outcomes and can deliver results. Its board and senior management team are highly experienced in the early and late stage development and commercialization of therapeutics in CNS.

Management Team

Tim Dyer, Chief Executive Officer

Since co-founding Addex in 2002, Mr Dyer has played a pivotal role in building the Addex Group, raising CHF280 million of capital, including Addex IPO and negotiating licensing agreements with pharmaceutical industry partners that generated more than CHF50 million in cash inflows. Prior to founding 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 two years performing inward investment due diligence on local financial institutions in the Ex-Soviet Union. Mr Dyer has extensive experience in finance, corporate development, business operations and the building of start-up companies and served as a member of the Swiss government innovation promotion agency coaching team from 2011 to 2016. Mr Dyer also serves on the advisory board of the École polytechnique fédérale de Lausanne Management of Technology MBA program. He serves on the boards of Abionic SA, a private medical device start-up company focused point of care in vitro diagnostics and Qwane Biosciences SA, a private drug development tool company focused

on commercializing microelectrode array technologies. Mr Dyer is also founder and managing partner of TMD Advisory, a CFO services company. He is a UK Chartered Accountant and holds a BSc (Hons) in Biochemistry and Pharmacology from the University of Southampton, UK.

Dr. Roger Mills, Chief Medical Officer

Dr. Mills, who joined Addex in 2016, brings more than 25 years of biopharmaceutical industry experience at both large global pharmaceutical companies and smaller biotechnology companies, including Acadia Pharmaceuticals, Pfizer, Gilead Sciences, Abbott Laboratories and Wellcome, across a spectrum of disease areas. His extensive track record includes managing drug development programs from Investigational New Drug Application preparation through to post-marketing and OTC products, including NUPLAZID™ for the treatment of Parkinson's Disease Psychosis, as well as regulatory affairs and business development activities. Most recently, Dr. Mills was with Acadia Pharmaceuticals for nine years, serving as Executive Vice President, Development and Chief Medical Officer. In this role, he oversaw the largest ever international Phase III program in Parkinson's Disease Psychosis, and led the Company's New Drug Application submission to the US Food and Drug Administration (FDA) for NUPLAZID, which was subsequently approved and remains the first and only medication approved by the FDA in this indication. Dr. Mills currently serves as a Visiting Professor at the Centre for Age Related Diseases, Institute of Psychiatry, Psychology and Neuroscience, King's College London. He received his medical degree from Imperial College, Charing Cross Hospital Medical School, London, United Kingdom. Dr. Mills is coauthor of more than 50 research publications and patents.

Dr. Robert Lütjens, Head of Discovery

Dr. Lütjens, who joined Addex at its founding in 2002, is an accomplished preclinical drug developer and expert in allosteric modulation with more than 20 years experience. At Addex, Dr Lütjens was responsible for establishing the Addex small molecule allosteric modulator biology

platform. He led the high through put screening campaigns which successfully discovered the Addex clinical and preclinical pipeline of first in class small molecule allosteric modulators. He has played a pivotal role in the development of Addex portfolio which delivered several molecules into the clinic, including dipraglurant for PD-LID. In addition, Dr. Lütjens managed numerous research collaborations both with academic and industrial partners, and in particular 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 over 25 peer-reviewed publications and co-inventor on patents covering screening methods or chemical compounds.

Valuation

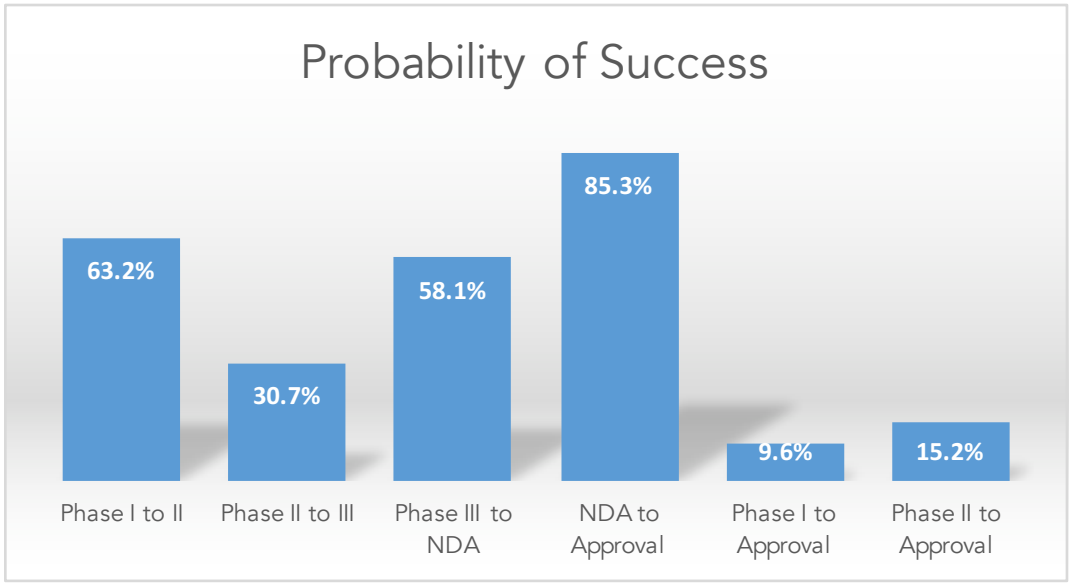
We have increased our valuation on Addex Therapeutics to CHF 179 million or CHF 11.50 per share from CHF 76 million or CHF 6.50 per share due to the fact that we have increase our LOA and market potential for Addex' lead product dipraglurant. At this moment, we do not address value to the preclinical programs in Addex' pipeline, including its ADX71441 programs. This is a potential upside for the company, since this program is expected to go into the clinic in 2018H1. That would justify a potential rerating in 2018.

Phase Success and Likelihood of Approval (LOA)

In estimating a value for the clinical programs with dipraglurant, we made use of several studies that were done on the clinical development success rates for investigational drugs to measure success rates for investigational drugs. We analyzed individual drug program phase transitions from January 1, 2006 to December 31, 2015. For the ten years studied, 9,985 transitions in the Biomedtracker database were analyzed. A phase transition is the movement out of a clinical phase – for example, advancing from Phase I to Phase II development, or being suspended after completion of Phase I development. These transitions occurred in 7,455 clinical drug development programs, across 1,103 companies (both large and small), making this the largest study of its kind. With this broad set of data, we aimed to capture the diversity in drug development across levels of novelty, molecular modalities, and disease indications. Only company-sponsored, FDA registration-enabling development programs were considered; investigator-sponsored studies were excluded from this analysis.

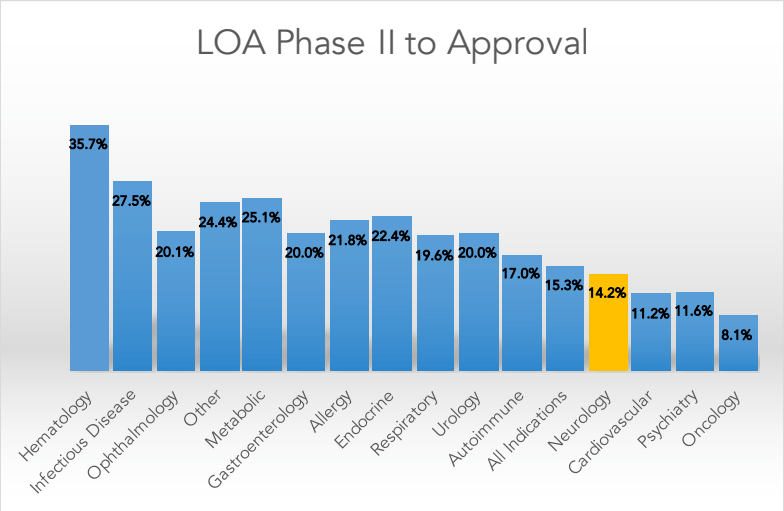
The Phase I transition success rate was 63.2% (n=3,582). As this Phase is typically conducted for safety testing and is not dependent on efficacy results for candidates to advance, it is common for this phase to have the highest success rate among the clinical phases across most categories analyzed in this report. Phase I success rates may also benefit from delayed reporting bias, as some larger companies may not deem failed Phase I programs as material and thereby not report them in the public domain. The Phase II transition success rate (30.7%, n=3,862) was substantially

lower than Phase I, and the lowest of the four phases studied. As this is generally the first stage where proof-of-concept is deliberately tested in human subjects, Phase II consistently had the lowest success rate of all phases. This is also the point in development where industry must decide whether to pursue the large, expensive Phase III studies and may decide to terminate development for multiple reasons including commercial viability. The second-lowest phase transition success rate was found in Phase III (58.1%, n=1,491). This is significant as most company-sponsored Phase III trials are the longest and most expensive trials to conduct. The probability of FDA approval after submitting a New Drug Application (NDA) or Biologic License Application (BLA), taking into account re-submissions, was 85.3% (n=1,050). Multiplying these individual phase components to obtain the compound probability of progressing from Phase I to U.S. FDA approval (LOA) reveals that only 9.6% (n=9,985) of drug development programs successfully make it to market (see graph below)



Source: BIO Industry Analysis

Major disease areas were segmented according to the convention used by Biomedtracker, and categorized 21 major diseases and 558 indications for the 2006-2015 timeframe. As can be seen in the graphs below, there is a wide range of Likelihood of Approval (LOA) from Phase I, II and III.



Valuation dipraglurant HR in PD-LID

In estimating a value for dipraglurant in PD-LID, we took into account potential markets in the US and Europe with a total number of potential patients with PD-LID of 180,000 in the US and 225,000 in Europe, with a market launch in the US in 2022 and 2023 in Europe. We calculate a Risk adjusted Discount Rate of 15%. Annual pricing is conservatively set at USD 20,000 for the US and USD 10,000 for Europe which is actually lower than pricing of competitive drugs (Pimavanserin for PDP is priced at USD 26,000 whereas Igrezza is even priced at USD 60,000) Although we believe that Addex will potentially partner its program in PD-LID with a large pharmaceutical, in our model we have calculated its value by marketing the drug independently. We estimate that a peak market share of 25-30% is possible. Compared with the report of BioMedTracker (see neurological disorders), we used a somewhat higher LOA of 20% as we believe that the vast amount of data justifies that. This leads to a total valuation of CHF 141 million or CHF 9.10 per share.

Valuation in PD-LID US Market

Year	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	
No of patients US (yoy growth 2.5% as of 2015)	186682	229619	235360	241244	247275	253457	259793	266288	272945	279769	
Penetration	0.5%	1.5%	3.0%	6.0%	10.0%	14.0%	18.0%	20.0%	22.0%	24.0%	
Total Revenues (USD m)	**23.0**	**70.6**	**144.7**	**296.7**	**506.9**	**727.4**	**958.6**	**1091.8**	**1231.0**	**1376.5**	
Margin 50%	11.5	35.3	72.4	148.4	253.5	363.7	479.3	545.9	615.5	688.2	
WACC 15%	0.43	0.38	0.33	0.28	0.25	0.21	0.19	0.16	0.14	0.12	
NPV (million)	5.0	13.3	23.7	42.2	62.7	78.2	89.6	88.7	87.0	84.6	
Total NPV (million)											**490.2**
LOA 20%											**98.0**

Valuation in PD-LID European Market

Year	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	
No of patients Europe (yoy growth 2.5% as of 2015)	224019	275543	282432	289493	296730	304148	311752	319546	327534	335723	
Penetration	0.5%	1.5%	3.0%	6.0%	10.0%	14.0%	18.0%	20.0%	22.0%	25.0%	
Total Revenues (USD m)	**14.1**	**43.4**	**89.0**	**182.5**	**311.8**	**447.4**	**589.6**	**671.4**	**757.1**	**881.8**	
Margin 50%	7,1	21,7	44,5	91,2	155,9	223,7	294,8	335,7	378,5	440,9	
WACC 15%	0.38	0.33	0.28	0.25	0.21	0.19	0.16	0.14	0.12	0.11	
NPV (million)	2,7	7,1	12,7	22,6	33,5	41,8	47,9	47,4	46,5	47,1	
Total NPV (million)											**215.6**
LOA 20%											**43.1**

Valuation dipraglurant ER in Focal Dystonia

In estimating a value for dipraglurant in focal dystonia, here we also took into account potential markets in the US and Europe with a total number of potential patients with focal dystonia of 75,000 in the US and 90,000 in Europe, with a market launch in the US and Europe in 2023. We calculate a Risk adjusted Discount Rate of 15%. Annual pricing is set at USD 40,000 for the US and USD 20,000 for Europe which is comparable with pricing of competitive drugs. Although we believe that Addex will potentially partner its program in dystonia with a large pharmaceutical, in our model we have calculated its value by marketing the drug independently. We estimate that a peak market share of 20-25% is possible. Compared with the report of BioMedTracker (see neurological disorders), we used a LOA of 8%. This leads to a total valuation of CHF 38 million or CHF 2.46 per share.

Valuation in Focal Dystonia EU Market

Year	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	
No of patients US (yoy growth 2.5% as of 2015)	94144	96498	98910	101383	103917	106515	109178	111908	114705	117573	
Penetration	1.0%	3.0%	6.0%	9.0%	12.0%	15.0%	17.0%	18.0%	19.0%	20.0%	
Total Revenues (USD m)	18,8	57,9	118,7	182,5	249,4	319,5	371,2	402,9	435,9	470,3	
Margin 50%	9,4	28,9	59,3	91,2	124,7	159,8	185,6	201,4	217,9	235,1	
WACC 15%	0,38	0,33	0,28	0,25	0,21	0,19	0,16	0,14	0,12	0,11	
NPV (million)	3,5	9,5	16,9	22,6	26,8	29,9	30,2	28,5	26,8	25,1	
Total NPV (million)											**167.7**
LOA 8%											**13.4**

Valuation in Focal Dystonia US Market

Year	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	
No of patients European (yoy growth 2.5% as of 2015)	78453	80415	82425	84486	86598	88763	90982	93256	95588	97977	
Penetration	1.0%	3.0%	6.0%	10.0%	14.0%	17.0%	19.0%	20.0%	21.0%	22.0%	
Total Revenues (USD m)	31,4	96,5	197,8	337,9	484,9	603,6	691,5	746,1	802,9	862,2	
Margin 50%	15,7	48,2	98,9	169,0	242,5	301,8	345,7	373,0	401,5	431,1	
WACC 15%	0,38	0,33	0,28	0,25	0,21	0,19	0,16	0,14	0,12	0,11	
NPV (million)	5,9	15,8	28,1	41,8	52,1	56,4	56,2	52,7	49,3	46,1	
Total NPV (million)											**309.0**
LOA 8%											**24.7**

Near Term Milestones

In the past 12 months, Addex has already reached a number of important milestones that brought the company back on track towards commercialization of its lead candidate. In the coming 12 months, we expect a number of important milestones that can drive the stock price upwards. These are:

- ADX71441 Phase I – start dosing: 2018H1
- Dipraglurant Phase IIa POC study in focal cervical dystonia – start dosing: 2018H1
- ADX71441 Phase I – results: 2018H2
- Dipraglurant first pivotal study in LID registration program – start dosing: 2018H2
- Dipraglurant Phase II POC study in focal cervical dystonia – results: 2019H1
- ADX71441 Phase IIa POC in CMT1a – start dosing: 2019H1
- Dipraglurant first pivotal study in LID registration program – results: 2020H1
- ADX71441 Phase IIa POC in CMT1a – results: 2020H1
- Dipraglurant second pivotal study in LID registration program – start dosing: 2020H1
- Dipraglurant second pivotal study in LID registration program – results: 2021H2

Competitive Landscape

During examination of comparable companies we looked at companies that have programs in development in both PD-LID (dipraglurant), in addiction (ADX71441) and CMT1A (ADX71441) since we believe these programs to be the most promising.

Peer Group in PD-LID

Adamas Pharmaceuticals (ADMS)

Adamas is a pharmaceutical company that is developing new medicines to improve the daily lives of those affected by chronic neurologic disorders, including Alzheimer's disease, Parkinson's disease, multiple sclerosis and epilepsy. Its lead program, GOCOVRI (formerly known as ADS-5102) was recently approved by the FDA for the treatment of dyskinesia in PD patients. GOCOVRI is a high dose 274 mg amantadine (equivalent to 340 mg amantadine HCl) taken once-daily at bedtime that delivers consistently high levels of amantadine from the morning and throughout the day when dyskinesia occurs. Dyskinesia is a consequence of levodopa-based Parkinson's disease treatment and is characterized by involuntary and non-rhythmic movements that are purposeless and unpredictable, which impact the activities of daily living. GOCOVRI's positive benefit/safety profile was established in two Phase III controlled clinical trials in Parkinson's disease patients with dyskinesia. In Study 1, patients treated with GOCOVRI demonstrated statistically significant and clinically relevant reductions in dyskinesia, with a 37 percent reduction in Unified Dyskinesia Rating Scale (UDysRS) total score vs. 12 percent for placebo at Week 12. These results were confirmed in Study 2 in which GOCOVRI achieved a 46 percent reduction in UDysRS vs. 16 percent for placebo. Additionally, key secondary data from Parkinson's disease patient reported diaries in Study 1 and Study 2 respectively, showed that GOCOVRI-treated patients experienced a 3.6 and 4.0 hour increase in functional time daily (defined as ON time without troublesome dyskinesia) vs. a 0.8 and 2.1 hour increase for placebo-treated patients at Week 12. The increases in functional time were achieved

by decreases in both ON time with troublesome dyskinesia and OFF time. The placebo-adjusted reduction in OFF time in both studies was approximately 1 hour per day. The most commonly observed adverse reactions (> 10 percent and greater than placebo) with GOCOVRI were hallucinations, dizziness, dry mouth, peripheral edema, constipation, fall and orthostatic hypotension. GOCOVRI is expected to be available in the fourth quarter, and formally launched with the full deployment of Adamas's sales force in January 2018.

Osmotica

Osmotica Pharmaceutical is a US based specialty pharmaceutical company engaged in developing pharmaceutical products. It is focused on drug delivery technologies with a special focus in neurology-based therapies. Osmotica Pharmaceutical uses its proprietary osmotic technology platform to develop branded and generic pharmaceutical products. Its Osmodex® is a family of proprietary technologies that combines laser drilled tablet technology with variety of single active and multiple active drug delivery devices. Last year, the FDA granted Orphan Drug Status to Osmolex ER for the treatment of PD-LID. Osmolex ER, a proprietary drug formulation of Amantadine HCl Extended Release Tablets utilizing Osmotica's patented Osmodex technology platform. Currently, the company is sponsoring two Phase III clinical trials evaluating the efficacy and safety of Osmolex ER in PD patients with LID. These two Phase III clinical studies were commenced in early 2014 for Osmolex™ ER in the United States, Canada, France, Germany and Spain.

- ALLAY-LID I: a 16 week study with 162 patients; and
- ALLAY-LID II: a 26 week study with 162 patients.

Osmotica Pharmaceutical anticipates that it will file a new drug application via a 505(b) submission for Osmolex™ ER in 2017.

Peer Group in ADX71441

Indivior

Indivior PLC is a specialty pharmaceutical company focused on addiction treatment. The company was incorporated in September 2014 as a result of demerger of Reckitt Benckiser Pharmaceuticals Inc from RB Group. Indivior is focused on advancing its therapeutic pipeline to address the growing health epidemic of addiction and related mental health disorders. Indivior's product pipeline includes: RBP-6000, a buprenorphine 1 month depot in Atrigel® indicated for the treatment of opioid dependence; RBP-6300, a buprenorphine hemiadipate oral swallowable tablet with abuse-deterrent properties indicated for the treatment of opioid dependence; RBP-8000, a cocaine esterase indicated for the treatment of cocaine intoxication. This product has concluded a Phase II trial. No results were made public.

Pharnext

Pharnext is a clinical stage biopharmaceutical company developing new therapeutics that simultaneously target multiple key disease pathways for severe orphan and common neurological diseases. The proprietary R&D platform of Pharnext is based on network pharmacology. It allows the development of synergistic combinations of repositioned drugs – pleodrugs – which benefit from an outstanding safety profile and IP with strong enforceability. The company's two lead pleodrugs are PXT3003 for the treatment of Charcot-Marie-Tooth disease type IA (Phase III ongoing) and PXT864 for Alzheimer's disease (Phase IIa completed) and other neurologic indications (Parkinson's disease, amyotrophic lateral sclerosis). PXT3003, Pharnext's lead pleodrug, has shown positive results both in preclinical and Phase 2 studies published in the Orphanet Journal of Rare Diseases (OJRD) in December 2014. In preclinical studies in two different rodent models, PXT3003 inhibited the overexpression of the PMP22 gene, improved myelination of peripheral nerves and clinical / sensory impairments. In a Phase II clinical trial in 80 adult patients with CMT1A, PXT3003, beyond stabilization, improved multiple efficacy endpoints, particularly the ONLS score (Overall Neuropathy Limitation Scale) which measures patient

disability. The FDA suggested the use of ONLS as a primary efficacy endpoint in clinical trials in CMT. In addition, PXT3003 was safe and well tolerated. A pivotal Phase III clinical trial is ongoing in 323 patients with CMT1A. Results are expected in 2018H2. Pharnext is also considering a study in pediatric patients.

Analyst: Marcel Wijma MSc
Marcel Wijma, Chief Research Officer and managing partner, has a longstanding history in financial biotech research. After selling Van Leeuwenhoeck Research (VLR) to SNS Securities in 2006, he established an award winning analyst team in biotech/life sciences at SNS Securities. In 2009, Marcel was awarded by Financial Times/Starmine as being one of the Top-3 biotech analysts in Europe. Later that year, Marcel purchased VLR from SNS Securities after which the company was reconstituted. At VLR, he leads the professional VLR research organisation, which is augmented by selected external financial researchers with a specialisation in Life Sciences. Mr. Wijma has a Masters degree in Financial Economics from Erasmus University in Rotterdam.