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FOCUS AREA: ALLOSTERIC MODULATORS FOR RARE NEUROLOGICAL DISORDERS

KEY DATA			SIX: ADXN
MARKET CAPITALIZATION (CHF MN)	35	**SHARE PRICE ON AUGUST 04, 2015**	3.0
ENTERPRISE VALUE (CHF MN)	31	**RISK-ADJUSTED NPV PER SHARE** (CHF)	5.0
NET CASH (END Q1 2015E) (CHF MN)	4.1	**UPSIDE/DOWNSIDE** (%)	68%
MONTHLY OPERATING EXPENSE (CHF MN)	0.2	**RISK PROFILE**	SPECULATIVE
CASH LIFE	2017	**SUCCESS PROBABILITY LEAD PROJECT**	15%
BREAK-EVEN (YEAR)	2020	**EMPLOYEES**	7
FOUNDED (YEAR)	2002	**LISTED** (YEAR)	2007

KEY PRODUCTS:	STATUS	MAJOR SHAREHOLDERS:	(%)
- DIPRAGLURANT IR (PD-LID*)	PHASE II	- HERCULIS PARTNERS	5.7
- DIPRAGLURANT ER (CERVICAL DYSTONIA)	PHASE I	- EXECUTIVE MANAGEMENT (TIM DYER)	5.6
- ADX71441 / $GABA_B$ PAM (CHARCOT-MARIE-TOOTH 1A)	PHASE I	**- FREE FLOAT**	88.7
- ADX71441 / $GABA_B$ PAM (ADDICTION)	PHASE I	- DAILY VOLUME	24,750
- ADX71149 / MGLU2 PAM (CNS** INDICATION UNDER REVIEW)	PHASE II		

UPCOMING CATALYSTS:	DATE	ANALYST(S):	BOB POOLER
- START PHASE I ADX71441 / $GABA_B$ PAM IN CMT 1A	END 2015		BP@VALUATIONLAB.COM
- ORPHAN DRUG DESIGNATION DIPRAGLURANT IR IN PD-LID	END 2015		+41 79 652 67 68
- START PHASE IIA DIPRAGLURANT ER IN DYSTONIA	H1 2016		

* PD-LID = PARKINSON'S DISEASE LEVODOPA-INDUCED DYSKINESIA; ** CNS = CENTRAL NERVOUS SYSTEM
ESTIMATES AS OF 4 AUGUST, 2015

SOURCE: VALUATIONLAB ESTIMATES, ADDEX THERAPEUTICS

Back to the future

Rare movement disorders targeted to unlock value

Addex Therapeutics' strategy is to develop compounds generated from its proprietary allosteric modulation discovery platform in rare, orphan, movement disorders caused by imbalances within the glutamatergic system. Management targets three key orphan drug opportunities: 1) dipraglurant IR with proof-of-concept in Parkinson's disease levodopa-induced dyskinesia (PD-LID); 2) dipraglurant ER in focal cervical dystonia; and 3) ADX71441 in Charcot-Marie-Tooth type 1A neuropathy. Addex also has an ongoing partnership with Janssen Pharmaceuticals Inc. (JPI) for ADX71149 in an undisclosed major central nervous system (CNS) disorder, and collaborations with the US National Institutes of Health (NIH) for ADX71441 in addiction. With cash life through 2017, Addex is actively seeking funding (grants, academic, patient group and corporate partnerships; private placements) to advance its key projects through their next value inflection point, phase II trials. We derive a risk-adjusted NPV of CHF 5.0/share (assuming a 57% dilution to raise CHF 20 mn), a WACC of 7.0% and a 15% success probability for lead project dipraglurant IR in PD-LID. Addex' risk profile is Speculative given the early stage pipeline, limited cash life, and the necessity to attract sufficient funds timely.

Key catalysts:

1) **US orphan drug designation (ODD) of dipraglurant IR in PD-LID.** A successful ODD grant should lead to lower development hurdles and costs, and faster timelines with 7 years of US market exclusivity in PD-LID.
2) **Phase IIa development of dipraglurant ER in cervical dystonia.** A proof-of-concept (POC) trial is planned to start in H1 2016 with results in 2016. Positive POC results would lead to a CHF 4.3 per share jump in our risk-adjusted NPV.
3) **New major CNS indication for ADX71149.** Partner JPI is expected to announce a new major CNS indication around year-end with POC trials to start in 2016. ADX71149 has already demonstrated POC as an add-on therapy for treating negative symptoms of schizophrenia.

Strategy & Cash Position

Considerable value locked up in existing clinical and preclinical pipeline

Through its proprietary allosteric modulator discovery platform, Addex Therapeutics has built one of the largest clinical and preclinical portfolios of allosteric modulator compounds targeting a variety of central nervous system (CNS) disorders including Parkinson's disease, dystonia, Charcot-Marie-Tooth type 1A neuropathy, addiction, depression, cognition and other neurological diseases. Addex' compounds are different from conventional small molecule drugs in that they bind to a different site (the "allosteric" site) of the receptors they target, potentially resulting in better efficacy and/or tolerability than conventional drugs. The company's clinical and preclinical projects target areas of high-unmet medical need, often with blockbuster peak sales potential. Unfortunately, after a shortfall in funding in May 2013, Addex was forced to put the majority of its projects on hold and scale down operations substantially. Consequently, there is considerable value locked up in the company's existing clinical and preclinical pipeline.

New development strategy set to unlock pipeline value

Under leadership of co-founder, major shareholder (5.6% stake), and newly appointed CEO Tim Dyer, the company completed a massive restructuring plan in 2013/2014, where the intellectual property portfolio and allosteric modulation discovery platform were secured, and the cash burn rate was significantly reduced as well as the headcount (5 FTE's). In 2015, the company completed a review of the existing pipeline portfolio, identified key areas and projects to pursue, and strengthened its cash position and board. Addex is now pursuing a new development strategy to bring the company "back to the future" of developing innovative allosteric modulation drugs, with the aim to unlock considerable value from the existing pipeline.

Key components of the new development strategy, include:

A. Focus on rare (orphan) neurological disorders
B. Enhance clinical expertise/development through collaborations
C. Secure and aim resources at projects with substantial value inflection points

A) Focus on rare neurological disorders

Addex will now focus its own clinical development efforts on "straightforward" neurological indications such as movement disorders or peripheral neuropathy caused by imbalances within the glutamate and GABA pathways, where the company has gathered a lot of expertise.

The company has also narrowed its focus on orphan (rare) disease indications. Drug development in rare diseases typically provides close and valuable interaction with regulators and key opinion leaders (KOL's), lower development hurdles and costs, faster development timelines, and incentives such as extended market exclusivity from launch.

Addex will no longer fund projects in the existing pipeline that do not fit these criteria. Attractive allosteric modulator projects outside the targeted orphan disease areas shall only be pursued after sufficient funding has been secured from a clinical development partner with the relevant expertise.

Addex' targeted pipeline projects, include:

1) **Dipraglurant IR** for Parkinson's disease levodopa-induced dyskinesia (PD-LID)
2) **Dipraglurant ER** for non-Parkinsonian dystonia
3) **ADX71441 ($GABA_B$ PAM)** for Charcot-Marie-Tooth 1A disorder (CMT 1A)
4) **ADX71441 ($GABA_B$ PAM)** for addiction
5) **ADX71149 (mGluR2)** collaboration with JPI. for an undisclosed major CNS indication

The first three projects (PD-LID, dystonia, CMT 1A) target rare neurological movement disorders caused by the glutamate or GABA pathways backed by robust scientific rational. These projects have Addex' highest priority, and with relatively little funding, could trigger substantial upside. The latter two projects (addiction, undisclosed major CNS indication) target large indications where an external partner is required. ADX71149 is partnered with JPI and targets major CNS disorders. Proof-of-concept was established as an add-on therapy for treating negative symptoms in schizophrenia. A new major CNS indication should be announced around year-end with substantial upside potential at zero cost to Addex. Full development of ADX71441 ($GABA_B$ PAM) for addiction will rely on sufficient external funding and finding the right development and commercialization partner.

B) Enhance clinical expertise/development through collaborations

Addex has rebuilt a small team of employees and expert consultants to advance its clinical and preclinical programs, which it complements with multiple academic institution, governmental organization and patient group collaborations. Implementation of such a collaborative model as opposed to building out an own clinical development organization, Addex has been able to efficiently access expertise to advance research and development of its targeted projects at low cost. Revised development plans for the targeted pipeline projects have been drafted with key opinion leaders (KOL's). Therapeutic area focused advisory panels with KOLs are currently finalizing plans to advance the pipeline as effectively and cost efficiently as possible.

Recent examples include the research collaborations for ADX71441 ($GABA_B$ PAM) with the US Charcot-Marie-Tooth Association (CMTA), the US National Institute for Drug Abuse (NIDA) for nicotine and cocaine addiction and the US National Institute on Alcohol Abuse and Alcoholism (NIAAA) for alcohol use disorder. In January 2015 Addex announced a partnership with the Dystonia Medical Research Foundation (DMRF) to explore the therapeutic use of dipraglurant in the treatment of dystonia. And in May Addex established a partnership with the University of Rome to explore the use of dipraglurant in rare genetic forms of dystonia. The company has an ongoing collaboration with the Michael J. Fox Foundation (MJFF), where it has been awarded grants of almost USD 2 mn to advance dipraglurant in PD-LID.

In December 2014 Addex announced it had revised its mGluR2 PAM collaboration (ADX71149) with Janssen Pharmaceuticals Inc. (JPI). The company received USD 757,000, while future contributions to patent costs have been reduced significantly. The amendment has no impact on Addex' right to receive royalties and milestones for up to a total of EUR 112 mn under the initial agreement, of which EUR 109 mn are remaining.

C) Secure and aim resources at projects with substantial value inflection points
The core of the new strategy is to secure the resources (clinical and financial) needed to advance the targeted orphan disease projects that create substantial shareholder value in the short and medium term. Development of a targeted project to a next inflection point, such as proof-of-concept (POC), increases the value of such a project considerably, extends out-licensing opportunities, and raises the probability of successful development and commercialization.

Importantly, a substantial amount of non-dilutive (clinical) resources will come from the multiple collaborations with academic institutions, governmental organizations and patient groups to advance the targeted projects.

As part of its strategy to secure the necessary resources to advance its pipeline for the benefit of patients, Addex continues to pursue discussions with potential industry partners. However, Addex does not want to entirely focus on the success of corporate partnerships to advance the clinical pipeline projects as in the past. Addex has identified the projects that it wishes to pursue and will execute development with or without industry partners. Consequently, additional funds will be raised through capital increases and/or private placements at the cost of dilution. However, dilution should be more than offset by reaching major clinical milestones, which increase value substantially. Ultimately, the company could pursue an M&A strategy on successful development of its targeted projects.

More than CHF 325 mn raised since inception
Over the past 13 years Addex has raised just over CHF 325 mn. CHF 137 mn was raised with the successful IPO in 2007. Prior to the IPO, management raised CHF 106 mn in three separate private funding rounds with prime healthcare investors, including Index Ventures, Sofinnova Partners (round one); TVM Capital (round two); and Roche Ventures and SR One, the venture arm of GSK (round three).

MONEY RAISED	CHF MN
PRE-IPO	106.0
IPO (INITIAL PUBLIC OFFERING)	137.0
GRANTS	2.5
UPFRONT & MILESTONE PAYMENTS	43.6
PRIVATE PLACEMENTS	36.4
TOTAL RAISED	**325.5**

ESTIMATES AS OF 4 AUGUST, 2015 SOURCE: VALUATIONLAB, ADDEX THERAPEUTICS

Addex has also been successful in raising substantial funds through private placements with high caliber institutional investors such as Biotechnology Value Fund, Visium Asset Management, Armistice Capital, and most recently with Herculis Partners, amounting to a total of CHF 36.4 mn. Herculis Partners is now a major shareholder owning 5.7% of the share capital.

In 2010 the company received a USD 900,000 grant from the Michael J. Fox Foundation for the funding of the phase IIa proof-of-concept trial of dipraglurant in Parkinson's disease, followed by a USD 1 mn grant in 2013 for the funding of certain phase IIb preparation studies and activities. Of this grant USD 800K remains available for draw down.

Addex has a history of high value partnerships with major industry players

Over time the company received substantial partnering related revenues amounting to CHF 43.6 mn. CHF 29.1 mn came from the mGluR4 (Parkinson's disease) and ADX63365 (schizophrenia) collaborations with Merck & Co. In 2011 both were unfortunately discontinued, due to an internal portfolio re-prioritization at Merck & Co. following its merger with Schering Plough.

The remaining CHF 14.4 mn came from the mGluR2 allosteric modulators (ADX71149) collaboration with Janssen Pharmaceuticals Inc. (a Johnson & Johnson company) for treating major CNS disorders, which started in 2005. This collaboration was the first partner validation of Addex' allosteric modulation discovery platform. Addex is eligible for up to EUR 109 mn of additional milestones upon development and regulatory achievements. Addex will receive low double-digit royalties (we conservatively assume 10%) on sales. Janssen Pharmaceuticals Inc. successfully completed a phase IIa proof-of-concept of ADX71149 as an add-on to current antipsychotics in schizophrenia patients in 2012. Around year-end the company expects to announce a new major CNS indication, with phase IIa proof-of-concept trials to start in 2016.

Bulk of cash used to build world's most extensive allosteric modulator pipeline

Addex has been a pioneer in building the infrastructure and developing the expertise for discovering and developing highly selective, oral, small molecule, allosteric modulator drugs. The bulk of the CHF 325 mn raised was spent on building this proprietary discovery platform. In 2010 the company encountered a major clinical development setback when ADX10059, in phase II development for treating gastroesophageal reflux disease (GERD/heartburn), migraine and anxiety, had to be terminated due to liver enzyme issues. On the positive side, in 2012 Addex successfully concluded phase IIa proof-of-concept of dipraglurant IR for treating PD-LID and its partner, JPI successfully completed a phase 2 POC in schizophrenia with ADX71149. The company now has an extensive and valuable pipeline of clinical and preclinical allosteric modulator compounds and a unique chemical library of approximately 85,000 compounds with allosteric characteristics, waiting to be unlocked.

Cash reach extended to 2017 thanks to a private placement of CHF 2.8 mn

At the FY 2014 results announcement in April, management guided for a "cash runway through 2017" with approximately CHF 4.1 mn cash in the bank and no debt, and a cash utilization of CHF 2 mn in 2015. In March 2015 Addex successfully raised CHF 2.8 mn through a private placement, while in December 2014 the company received USD 757,000 cash from the amended mGluR2 collaboration with Janssen Pharmaceutical Inc. - with a lower patent cash burn. This, combined with rigorous cash control, leads us to believe that the company should have sufficient cash through 2017 to successfully execute the first major steps of their new development strategy.

The current cash position allows Addex to conduct:

- An open label PET imaging study receptor occupancy trial with dipraglurant in 15 subjects
- Prepare dipraglurant to start a Phase IIb/III (pivotal) study in PD-LID
- Complete phase 1 development of ADX71441 ($GABA_B$ PAM)
- Prepare ADX71441 ($GABA_B$ PAM) for a phase IIA POC trial in CMT 1A
- Complete a phase IIa proof-of-concept trial with dipraglurant in focal cervical (neck muscle) dystonia

Addex will design and prepare the development of dipraglurant ER in dystonia in collaboration with the Dystonia Medical Research Foundation (DMRF). Furthermore, the company will design and prepare the development of dipraglurant IR in PD-LID in collaboration with the Michael J. Fox Foundation. Additional funds will be needed to conduct these trials.

Additional funds needed to execute the new development strategy successfully

In the table below we provide an overview of additional funds we believe are needed to advance the targeted pipeline projects. Addex will need approximately CHF 25 mn to finance: 1) a phase IIb/III (pivotal) trial of dipraglurant IR in PD-LID, 2) a phase IIa POC and phase IIb/III (pivotal) trial in cervical dystonia, 3) a phase IIa POC ADX71441 ($GABA_B$ PAM) in CMT 1A. At the current cash position of CHF 4.1 mn and available MJFF funding of CHF 0.8 mn, an additional CHF 20 mn will be needed to finance these development plans. At the current valuation this would lead to a dilution of around 57% (conservatively assumed in our risk-adjusted NPV calculation). Note: Addex could raise the required CHF 20 mn in several tranches: e.g. CHF 10 mn around year-end, and CHF 10 mn on positive POC results in dystonia in late 2016. POC in dystonia should lead to a considerable rise in the valuation and therefore lead to significantly less dilution than we currently assume.

R&D EXPENDITURE & FUNDING - OVERVIEW

ASSUMED R&D EXPENDITURE

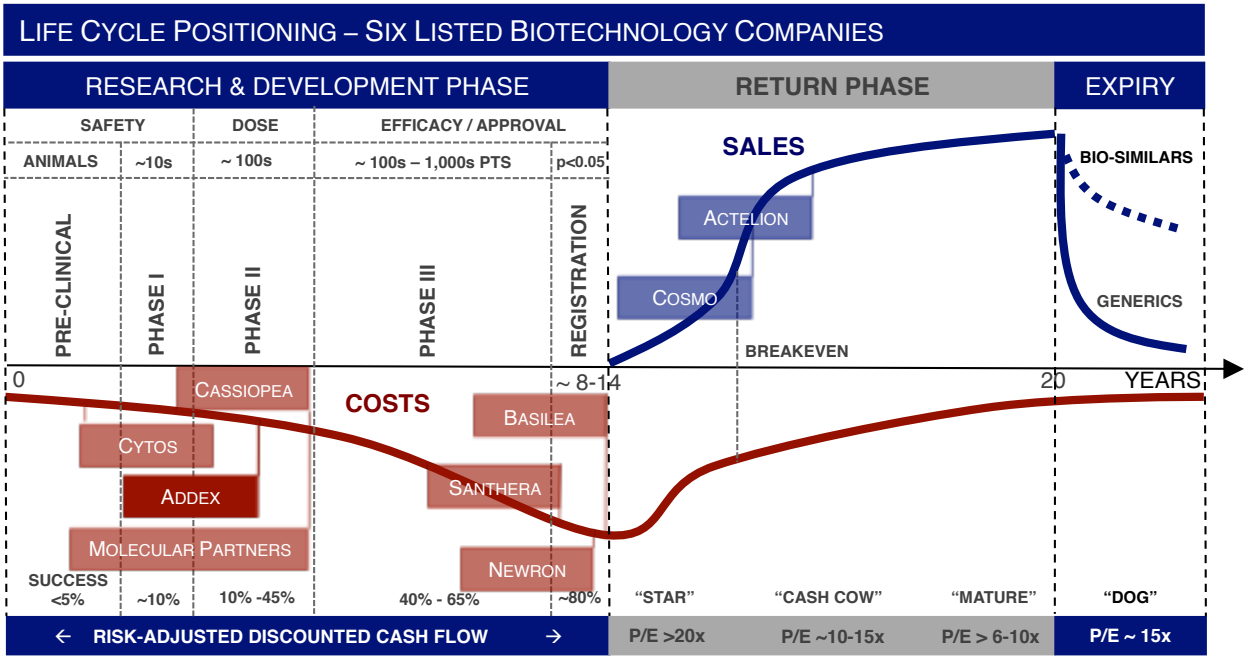
COMPOUND	INDICATION	PHASE	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	TOTAL
DIPRAGLURANT IR	**PD-LID**	PRE PHASE IIB	1								1
		TOX & CMC		3							3
		PHASE IIB (PIVOTAL)		3	3	1					7
		EXTENSION STUDY				2	2				4
		PHASE III (EU)				3	8	8	1		20
		FILING						1			1
TOTAL (CHF MN)			1	6	3	6	10	9	1	0	**36**
DIPRAGLURANT ER	**DYSTONIA**	PHASE IIA	0.5								0.5
		PHASE IIB (PIVOTAL)		2.5	2.5						5
		EXTENSION STUDY			1						1
		ER FORMULATION		1							1
		FILING				1					1
TOTAL (CHF MN)			0.5	3.5	3.5	1	0	0	0	0	**8.5**
ADX71441 (GABA B PAM)	**CMT 1A**	PRECLINICAL	0	1.5	2	0.5					4
		PHASE I	0.5	0.5							1
		PHASE IIA			3						3
		PHASE IIB (PIVOTAL)				10	5				15
		EXTENSION STUDY						2			2
		FILING							1		1
TOTAL (CHF MN)			1	2	5	11	5	2	1	0	**26.0**
TOTAL R&D FUNDS REQUIRED			**2**	**11.5**	**11.5**	**17.5**	**15**	**11**	**2**	**0**	**70.5**

SOURCE: VALUATIONLAB ESTIMATES

Positive results of the pivotal trials for dipraglurant in PD-LID and dystonia should lead to a jump in valuation and attract a lucrative development and commercialization partnership. In our forecasts we assume a partnership could occur in late 2017. The expected upfront, development and sales milestones from this partnership should be sufficient to finance the development of ADX71441 ($GABA_B$ PAM) in CMT 1A up to commercialization. Additional funding is required for addiction, which could be provided by the NIH and/or a corporate partner. Moreover, development milestones from Janssen Pharmaceuticals Inc. (up to EUR 109 mn) could provide additional funding.

Life Cycle Positioning - High Risk

We consider an investment in Addex as Speculative with currently a limited cash runway through 2017. The company has no products on the market that can provide sustainable revenue streams, while the targeted projects are still in an early development stage with a relatively low success probability. Development of these projects is largely dependent on Addex successfully and timely raising sufficient funds through various partnerships and private placements.



SOURCE: VALUATIONLAB

Valuation Overview

Risk-adjusted sum-of-parts NPV points to a fair value of CHF 5.0 per share

We derive a risk-adjusted NPV of CHF 5.0 per share (conservatively assuming a 57% dilution to raise CHF 20 mn) with cash of CHF 0.2 per share (end Q1 2015E) and overhead expenses of CHF 0.5 per share. We assume a WACC of 7.0% (which reflects the low Swiss interest environment following the decoupling of the Swiss Franc/Euro peg).

SUM OF PARTS

PRODUCT	INDICATION	PEAK SALES (CHF MN)	LAUNCH YEAR (EST)	UNADJUSTED NPV/SHARE (CHF)	SUCCESS PROBABILITY	RISK-ADJUSTED NPV/SHARE (CHF)	PERCENTAGE OF TOTAL
DIPRAGLURANT-IR (MGLU5 NAM)	PARKINSON'S DISEASE	898	2020	27.3	15%	**4.1**	74%
ADX71149 (MGLU2 PAM)	NEW MAJOR CNS INDICATION	961	2021	12.1	10%	**1.2**	22%
DIPRAGLURANT-ER (MGLU5 NAM)	DYSTONIA (NON-PD)	546	2020	25.1			
ADX71441 (GABA-B PAM)	CHARCOT-MARIE-TOOTH	549	2023	12.8			
ADX71441 (GABA-B PAM)	ADDICTION (ALCOHOL/NICOTINE)	551	2024	11.4			
NET CASH POSITION (END Q1 2015E)		4		0.2		**0.2**	4%
TOTAL ASSETS				89.1		**5.5**	**100%**
OVERHEAD EXPENSES				-0.5		**-0.5**	
NPV/SHARE (CHF)				88.6		**5.0**	
SHARE PRICE ON AUGUST 04, 2015						3.0	
PERCENTAGE UPSIDE / (DOWNSIDE)						68%	

ESTIMATES AS OF 4 AUGUST, 2015 SOURCE: VALUATIONLAB ESTIMATES

Addex' valuation currently based on two key value drivers:

1) Dipraglurant IR (PD-LID) - Fair value of CHF 4.1 per share

In our forecasts for dipraglurant IR, the instant release formulation, we only include peak sales of CHF 900 mn for treating Parkinson's disease levodopa-induced dyskinesia (PD-LID), with a 15% success rate based on positive phase IIa proof-of-concept data reported in March 2012. We exclude any value from dipraglurant ER, the extended release formulation, in non-Parkinsonian dystonia due to lack of proof-of-concept in humans. We assume Addex will raise sufficient cash to fully develop dipraglurant IR in PD-LID up to phase IIb/III, leading to significant value creation, and then sign on a development and commercialization partner in return for attractive upfront, development and sales milestones (up to CHF 160 mn) and royalty payments (25% on net sales). We calculate a risk-adjusted NPV of CHF 75 mn for dipraglurant IR in PD-LID using a WACC of 7.0% (for details see our "Forecasts and Sensitivity Analysis").

2) ADX71149 (undisclosed major CNS indication) - Fair value of CHF 1.2 per share

In November 2012, partner Janssen Pharmaceuticals Inc. announced positive results in a 92 patient phase IIa proof-of-concept study with ADX71149 as an add-on therapy in schizophrenia patients who do not fully respond to antipsychotics. No specific details of the results were disclosed, in line with the company's policy of drugs in early stage development. Addex' partner is currently reviewing a new major CNS indication to pursue with ADX71149, which is expected to be announced around year-end.

Given the positive proof-of-concept in schizophrenia, we forecast blockbuster peak sales for ADX71149 in this new major CNS indication, assuming first market launches in 2021, patent protection until at least 2028 (extension possible). Our risk-adjusted NPV amounts to CHF 22.3 mn, assuming Addex receives up to CHF 54.5 mn milestone payments from partner Janssen Pharmaceuticals Inc., 10% royalties on sales, with a success probability of 10%, and a WACC of 7.0%.

No value contributed to early stage pipeline projects, yet

We have not accounted for Addex' early stage product pipeline stemming from its proprietary allosteric modulation technology platform due to the current lack of proof-of-concept (POC). These projects could provide substantial upside once they demonstrate POC or are out-licensed to partners providing revenue streams through upfront and clinical milestone payments and royalties on sales. Addex has secured initial funding through partnerships with academic institutions, governmental organizations and patient group organizations to advance preclinical proof of concept. Positive proof-of-concept in each indication leads to considerable value creation and could attract potential out-licensing partners.

1) Dipraglurant ER (non-Parkinsonian dystonia)

The extended release formulation of dipraglurant targets non-Parkinsonian dystonia, a neurological movement disorder that leads to muscle contractions and awkward postures. Addex has a partnership with the Dystonia Medical Research Foundation (DMRF) to explore its use in dystonia. A proof-of-concept trial in focal cervical dystonia is planned to start in H1 2016 with results in 2016. Peak sales could reach CHF 500+ mn.

2) ADX71441 ($GABA_B$ PAM) (Charcot-Marie-Tooth 1A disorder)

The first indication for this compound is Charcot-Marie-Tooth 1A disorder, a rare neurological disorder that leads to disability at early age. The Charcot-Marie-Tooth Association supports preclinical evaluation. A phase I safety trial is planned to start in 2015 with results in early 2016. A phase IIa POC trial is planned to start in mid 2016. Peak sales could reach CHF 500+ mn.

3) ADX71441 ($GABA_B$ PAM) (addiction – alcohol / nicotine / cocaine)

This compound is also being explored for treating nicotine addiction and/or alcohol abuse. A partnership with the US NIAAA will evaluate ADX71441 in a battery of preclinical models to study its potential to treat alcohol use disorder, while a partnership with the NIDA will explore its potential in nicotine and cocaine addiction in a battery of preclinical models. Addex will determine which indication to pursue by the end of this year. In 2016 the company could start a phase IIa POC trial in the selected indication, depending on sufficient funding and partnering. Results would be due in 2017. Peak sales in each of these indications could easily reach CHF 500+ mn.

Sensitivities that can influence our valuation

Ability to fund key development projects: The key risk of Addex' investment case relates to the successful completion of the targeted clinical studies to create shareholder value within the projected timelines. With a cash runway through 2017, Addex does not have sufficient funds to develop all its targeted development projects up to partnering and commercialization. Established government, academic and patient group collaborations have already advanced research at a low cost. Substantial additional funds will still be needed to successfully execute the new development strategy. Addex will still need to attract development and commercialization partners for these projects and/or seek funding through capital increases, which leads to dilution, or pursue an M&A strategy.

Speed of funding: Next to attracting sufficient funds to execute the new development strategy, the time needed to attract these funds will determine the speed and amount of value creation. Slower than expected funding pushes back development plans, thereby reducing the effective patent life of each project that is delayed, impacting the total value.

Development and approval risk: Most projects are still in the early stages of development and therefore bear a high risk of failure. The valuation of Addex is currently based on two projects, dipraglurant IR (PD-LID) and ADX71149 (major new undisclosed CNS indication) with relatively low success probabilities of 15% and 10%, respectively.

Orphan drug designation (ODD) for dipraglurant IR in PD-LID: An FDA grant should lower development hurdles, costs and timelines substantially, and provide 7 years US market exclusivity. In April 2015, Adamas' long-acting amantadine (ADS-5102) was the first drug to receive US ODD in PD-LID.

Pricing and reimbursement: Following an FDA or EMA approval, dipraglurant must be priced and reimbursed by local health care providers. In the US pricing and reimbursement is typically quite straightforward. In the EU pricing and reimbursement occurs on a country-by-country basis, which can lead to different pricing, reimbursement, and potential market launch delays. However, with no treatments available for PD-LID, we assume dipraglurant IR pricing at a substantial premium to in-market branded Parkinson's drugs.

Partnering and commercialization: With no own sales force, Addex will need commercialization partners or will have to raise additional funds to build an own sales infrastructure. Upfront and sales milestones and royalties on sales from these partners could be lower than our estimates. Furthermore, the launched drugs must be successfully positioned and marketed against existing and upcoming treatments.

Patent and market exclusivity: Dipraglurant's composition of matter patent expires in 2025. Protection beyond this period will rely heavily on potential extensions, formulation patents and market exclusivities. We conservatively assume patent protection for dipraglurant IR in PD-LID until 2025 (excluding any extension) and dipraglurant ER for dystonia until 2032 (thanks to the extended release formulation patent).

External sourcing: Addex does not have its own manufacturing facilities and is dependent on external sourcing to manufacture dipraglurant according to strict regulatory specifications.

Catalysts

Addex had an excellent start of the year announcing several partnerships with the US National Institutes of Health, patient organizations and academia to advance its pipeline projects. Importantly, the company secured sufficient funding through 2017 through a CHF 2.8 mn private placement, with Herculis Partners becoming a major shareholder.

Obtaining of US orphan drug designation (ODD) should substantially reduce clinical development costs, timelines, and significantly enhance the value of dipraglurant IR. On securing sufficient funding Addex plans to start a phase IIb/III pivotal trial in Q3 2016 with headline results expected in 2017. This would trigger substantial equity upside with our risk-adjusted NPV jumping by CHF 9.5/share

CATALYST TIMELINES

TIME LINE	PRODUCT	INDICATION	MILESTONE / EVENT	COMMENT	IMPACT
2015					
7 JAN	ADX71441 ($GABA_B$ PAM)	ADDICTION (ALCOHOL)	NIAAA COLLABORATION	COLLABORATION WITH US NIAAA [1] TO EVALUATE ADX71441 IN PRECLINICAL MODELS OF ALCOHOL ABUSE	
19 JAN	DIPRAGLURANT ER	DYSTONIA	DMRF COLLABORATION	COLLABORATION WITH DMRF [2] TO DESIGN DEVELOPMENT PLAN, REGULATORY PATHWAY, KOL'S & PATIENTS FOR A PHASE II TRIAL	
19 FEB	MGLUR2 NAM	ISCHEMIC DAMAGE	PRECLINICAL	PRECLINICAL CHARACTERIZATION IN COLLABORATION WITH NEUROMED, ITALY	
9 MAR			PRIVATE PLACEMENT	ADDEX RAISES CHF 2.8 MN IN PRIVATE PLACEMENT WITH HERCULIS PARTNERS EXTENDING CASH RUNWAY THROUGH 2017	
30 APR			FY 2014 RESULTS	CASH UTILIZATION GUIDANCE OF CHF 2 MN IN 2015; CASH RUNWAY THROUGH 2017	
7 MAY	DIPRAGLURANT ER	DYSTONIA	PISANI COLLABORATION	COLLABORATION WITH PROF. PISANI TO EXPLORE DIPRAGLURANT IN RARE INHERITED FORMS OF DYSTONIA	
12 JUN			AGM	RAYMOND HILL AND TIM DYER APPOINTED TO BOARD; AUTHORIZED AND CONDITIONAL CAPITAL INCREASED	
22 JUL	DIPRAGLURANT IR	PD-LID	PET IMAGING STUDY	OPEN LABEL PET IMAGING RECEPTOR OCCUPANCY TRIAL IN 15 SUBJECTS (FINANCED)	
Q3	DIPRAGLURANT IR	ANTI-DEPRESSANT-LIKE EFFECTS	PRECLINICAL CHARACTERIZATION	IN COLLABORATION WITH POLISH ACADEMY OF SCIENCES	
Q4	ADX71441 ($GABA_B$ PAM)	ADDICTION (COCAINE)	PRECLINICAL	PRECLINICAL CHARACTERIZATION IN COLLABORATION WITH NIDA [3]	
Q4	ADX71441 ($GABA_B$ PAM)	CHARCOT-MARIE-TOOTH	PRECLINICAL	PRECLINICAL CHARACTERIZATION IN COLLABORATION WITH CMTA [4]	
Q4	MGLUR4 PAM / MGLUR7 NAM	NEURODEGENERATIVE & PSYCHIATRIC DISORDERS	PRECLINICAL	PRECLINICAL CHARACTERIZATION IN COLLABORATION WITH EPFL [5] AND UNIVERSITÉ DE LAUSANNE	
END	ADX71441 ($GABA_B$ PAM)	CHARCOT-MARIE-TOOTH	START PHASE I	START OF PHASE I SAFETY TRIALS IN HEALTHY VOLUNTEERS	
END	DIPRAGLURANT IR	PD-LID	US ODD GRANT	EXPECTED US ORPHAN DRUG DESIGNATION (ODD) GRANT BY FDA	
END	ADX71149 (MGLUR2PAM)	UNDER REVIEW	NEW CNS INDICATION	JANSSEN PHARMACEUTICALS INC. EXPECTED TO DISCLOSE NEW MAJOR CNS INDICATION AROUND YEAR-END (LOW D.D. ROYALTIES)	
END	DIPRAGLURANT IR	PD-LID	PET IMAGING STUDY	TRIAL COMPLETION	
2016					
MID	ADX71441 ($GABA_B$ PAM)	CHARCOT-MARIE-TOOTH	PHASE I RESULTS	COMPLETION OF PHASE I SAFETY TESTING	
H1	DIPRAGLURANT-ER	DYSTONIA	START PHASE II POC* TRIALS (CERVICAL/LOCAL FOCAL)	START OF PHASE IIA POC TRIAL IN COMBINATION WITH BOTILINUM TOXIN IN PATIENTS WITH CERVICAL OR LOCAL FOCAL DYSTONIA WITH PARTIAL RESPONSE (FINANCED)	
H2	ADX71149 (MGLUR2PAM) PARTNERED WITH JANSSEN	UNDER REVIEW	START PHASE II POC TRIAL	JANSSEN EXPECTED TO START OF PHASE IIA POC TRIAL IN NEW MAJOR CNS INDICATION	
H2	ADX71441 ($GABA_B$ PAM)	ADDICTION (ALCOHOL, NICOTINE)	START PHASE II POC TRIAL	START OF PHASE IIA POC TRIAL IN ADDICTION DEPENDENT ON FUNDING	
H2	ADX71441 ($GABA_B$ PAM)	CHARCOT-MARIE-TOOTH	START PHASE II POC	START PHASE IIA POC IN CMT 1A DEPENDENT ON FUNDING	
H2	DIPRAGLURANT-IR	PD-LID	START PHASE IIB/III	START OF PHASE IIB PIVOTAL TRIAL TO GAIN US APPROVAL AS AN ORPHAN DRUG; SUPPORTED BY MJFF GRANT (FURTHER FUNDING REQUIRED) - SUCCESS RATE JUMPS TO 50% FROM 15%	+ CHF 9.5
	DIPRAGLURANT-ER	DYSTONIA	RESULTS PHASE IIA TRIAL (CERVICAL/LOCAL FOCAL)	RESULTS PHASE IIA POC TRIALS IN CERVICAL OR LOCAL FOCAL DYSTONIA	+ CHF 4.3

[1] NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM; [2] DYSTONIA MEDICAL RESEARCH FOUNDATION; [3] NATIONAL INSTITUTE ON DRUG ABUSE; [4] CHARCOT MARIE TOOTH ASSOCIATION
[5] ÉCOLE POLYTECHNIQUE FÉDÉRALE DE LAUSANNE; [6] NATIONAL INSTITUTES OF HEALTH; [7] MICHAEL J. FOX FOUNDATION; * POC = PROOF-OF-CONCEPT

SOURCE: ADDEX THERAPEUTICS, VALUATIONLAB ESTIMATES

Around year-end partner Janssen Pharmaceuticals Inc. is expected to announce a new major CNS indication for ADX71149 and start proof-of-concept (POC) trials in 2016.

In H1 2016 Addex plans to start POC trials for dipraglurant ER in focal cervical dystonia with results in 2016. This would lead to a CHF 4.3/share increase in our risk-adjusted NPV.

In the next 12 months the company also plans to start POC trials of ADX71441 ($GABA_B$ PAM) in Charcot-Marie-Tooth type 1A neuropathy and addiction (on securing sufficient funding).

Technology & Pipeline

Addex is uniquely positioned with largest pipeline of allosteric modulator drugs

Addex has established a unique chemical library of approximately 85,000 compounds with allosteric characteristics. The library has been assembled from commercial and other non-pharmaceutical sources, with some parts of the library acquired under exclusive agreements. In addition, the company has developed highly specialized biological systems to identify and screen for highly potent, orally active small molecule allosteric modulators. These high-throughput detection systems have enabled Addex to build what we believe to be the largest clinical and preclinical portfolio of allosteric modulator compounds targeting a wide variety of diseases, such as Parkinson's disease, dystonia, Charcot-Marie-Tooth type 1A neuropathy, addiction, depression, cognition and other neurological diseases.

The company's proprietary allosteric modulator drug discovery platform is based on three key areas:

1. **Allosteric modulation** (better modulatory control of disease mediating receptors)
2. **GPCRs** (G-protein coupled receptors - molecular switches that control the signaling cycle)
3. **Glutamate and GABA pathways** (powerful neurotransmitters in the brain and nervous system that control normal functioning)

1) Allosteric modulation: conventional "on/off" approach vs. novel "dimmer" switch

Allosteric modulators are an emerging class of orally available small molecule drugs that may offer a competitive advantage over conventional or so-called "orthosteric" drugs. Conventional drugs work by interacting with receptors on the surface of cells or enzymes, which regulate the rate of chemical reactions within cells that can be imbalanced in disease. By binding to the target receptor site, the primary or "active" orthosteric site, they can either block (turn "off") the physiological function of the protein, or stimulate its effect (turn "on"). The conventional drug approach is similar to an electrical "on/off" switch. Conventional drugs must be able to out-compete naturally occurring substances in order to bind sufficiently to the active site. High receptor affinity is key. However, the continuous stimulation of potentially all receptor sites or a prolonged blockade of receptor functions may lead to unwanted side effects, which is a key problem with conventional orthosteric drugs. In addition, addressing the allosteric site, which unlike the orthosteric site, has not been subject to evolutionary pressure to remain unchanged, offers the unique possibility to identify and develop highly selective compounds.

Allosteric modulators bind to regulatory sites separate from the active or orthosteric site of the protein, the so-called "allosteric" site. Allosteric drugs rather modulate or control the amount of stimulation of the receptor, similar to an electrical light "dimmer" switch. Naturally occurring substances or conventional drugs can still bind to the active site preserving normal cell function. Because allosteric modulators bind to a different site, they do not have to out-compete naturally occurring substances to bind to the active site, such as conventional drugs do. As a result, allosteric modulators do not need to bind as strongly, or with as much affinity, as conventional orthosteric drugs to be effective, which facilitates discovery and development. Allosteric modulators can decrease the intensity of the signal to the cell interior (negative allosteric modulator or NAM) or increase the intensity of the signal to the cell interior (positive allosteric modulator or PAM).
Examples of approved drugs in the market based on allosteric modulation include

diazepam (anxiety, insomnia), Amgen's Sensipar (secondary hyperparathyroidism), and Pfizer's Selzentry (HIV).

2) GPCRs are and remain an important target for drug development

G-protein-coupled receptors (GPCRs) are involved in many important physiological and pathophysiological processes and are considered as one of the most successful therapeutic targets for a broad spectrum of diseases, including cancer, central nervous system disorders, diabetes, inflammation and pain. GPCRs, also known as 7 transmembrane receptors, are the largest family of cell surface receptors and account for approximately 4% of the protein-coding human genome. G-proteins are molecular switches that control the signaling cycle. They are activated by a wide variety of stimulants, including peptide and non-peptide neurotransmitters, hormones, growth factors and lipids. The G-protein system plays a central role in many signaling tasks, in about every organ system, making it an important target for drug development. These receptors are the target of more than 50% of the current therapeutic agents on the market, including more than a quarter of the 100 top-selling drugs.

3) Targeting glutamate and GABA pathways has potential for many CNS disorders

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.

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.

Key advantages of Addex' allosteric modulation drug discovery platform include:

- Capitalizes on know how of existing "conventional" GPCR targets
- "Undruggable" conventional GPCR targets can now be addressed
- Higher control over the intensity of activation or inhibition
- Greater selectivity and safety than conventional drugs
- Combination use with conventional drugs (different binding site)
- "First-in-class" compounds with strong patent protection

New development strategy focused on rare neurological movement disorders

Due to limited resources currently available, the company will now focus its own clinical development efforts on "straightforward" neurological indications such as movement disorders and peripheral neuropathy caused by the glutamatergic system, where it has gathered a lot of expertise, as opposed to complex high risk CNS/psychiatry indications.

Addex has also narrowed its focus on rare, so-called orphan disease indications. These are diseases that affect fewer than 200,000 people in the US or less than 1 in 2,000 people in the EU. Drug development in rare diseases typically provides close and valuable interaction with regulators and key opinion leaders (KOL's), lower development hurdles and costs, faster development timelines. Importantly, orphan drug designation (ODD) provides 7 and 10 years market exclusivity from launch in the US and EU, respectively.

PRODUCT PIPELINE

PRODUCT	DRUG CLASS	INDICATION	STATUS	LAUNCH YEAR (EXPECTED)	PARTNER	PEAK SALES
DIPRAGLURANT-IR	MGLUR5 NAM *	PD-LID (PARKINSON'S DISEASE LEVADOPA-INDUCED DYSKINESIA)	PHASE IIB	2020	MICHAEL J. FOX FOUNDATION	CHF 900 MN
DIPRAGLURANT-ER	MGLUR5 NAM	DYSTONIA (NON-PARKINSON)	PHASE IIA	2020	DMRF [1)]	CHF 500+ MN
ADX71149	MGLUR2 PAM **	MAJOR CNS INDICATION (UNDER REVIEW)	PHASE IIA (UNDER REVIEW)	2021E	JANSSEN PHARMA. INC.	CHF 1 BN
ADX71441	GABA-BR PAM	CHARCOT-MARIE-TOOTH A1	PHASE I	2022	CMTA [2)]	CHF 500+ MN
ADX71441	GABA-BR PAM	ADDICTION (ALCOHOL/NICOTINE)	PHASE I	2023	NIDA [3)], NIAAA [4)]	CHF 500+ MN
MGLUR2 NAM	MGLUR2 NAM	TREATMENT RESISTANT DEPRESSION, COGNITIVE DEFICITS	PRECLINICAL	TBD	NEUROMED, ITALY	TBD
MGLUR7 NAM	MGLUR7 NAM	NEUROGENERATIVE AND PSYCHIATRIC DISORDERS	PRECLINICAL	TBD	EPFL & UNIVERSITY OF LAUSANNE	TBD
MGLUR4 PAM	MGLUR4 PAM	ALS (LOU GEHRIG'S) / ORAL MULTIPLE SCLEROSIS	PRECLINICAL	TBD	MONETIZE	TBD

* NEGATIVE ALLOSTERIC MODULATOR; ** POSITIVE ALLOSTERIC MODULATOR; [1)] DYSTONIA MEDICAL RESEARCH FOUNDATION
[2)] CHARCOT MARIE TOOTH ASSOCIATION; [3)] NATIONAL INSTITUTE ON DRUG ABUSE; [4)] NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM

ESTIMATES AS OF 3 AUGUST, 2015 SOURCE: ADDEX THERAPEUTICS, VALUATIONLAB ESTIMATES

Addex' targeted pipeline projects include:

1) **Dipraglurant IR for Parkinson's disease levodopa-induced dyskinesia (PD-LID)** (first-in-class, robust POC, ODD potential)
2) **Dipraglurant ER for non-Parkinsonian dystonia** (first-in-class, strong scientific rational, ODD potential)
3) **ADX71441 ($GABA_B$ PAM) for Charcot-Marie-Tooth type IA disorder** (first-in-class, strong scientific rational, POC from baclofen, ODD potential)
4) **ADX71441 ($GABA_B$ PAM) for addiction** (first-in-class, strong scientific rational, POC from baclofen, to be partnered)
5) **ADX71149 collaboration with Janssen Pharmaceuticals Inc.** (new undisclosed major CNS indication, substantial upside potential, zero cost)

In the following section we will provide an in-depth analysis and forecasts for Addex's key driver dipraglurant in Parkinson's disease levodopa-induced dyskinesia (PD-LID).

Forecasts & Sensitivity Analysis

Dipraglurant IR (Parkinson's Disease LID)

Product Analysis

PD-LID peak sales of CHF 900 mn - Risk-adjusted NPV of CHF 4.1 per share

We forecast peak sales of CHF 900 mn for dipraglurant IR in PD-LID, assuming first market launches in 2020, a daily treatment cost between CHF 8 (EU/ROW) and USD 20 (US), and a market penetration peaking at around 15-25% in the target population. Our risk-adjusted NPV amounts to CHF 75 mn, or CHF 4.1 per share. We conservatively assume a 57% dilution needed to raise CHF 20 mn to fund trials of targeted projects including the phase IIb/III pivotal trial in PD-LID. On successful phase IIb/III results we assume Addex to sign on a development and commercialization partner. In return we expect the company to receive a CHF 30 mn upfront milestone, with development and sales milestone payments up to CHF 160 mn, 25% royalties on sales, assuming a success probability of 15%, and a WACC of 7.0% (reflecting the low Swiss interest environment after the decoupling of the Swiss Franc/Euro peg in January 2015).

Funding and partnering key to unlock value in PD-LID

Dipraglurant is a highly selective, oral, brain penetrating, small molecule, metabotropic glutamate receptor-5 negative allosteric modulator (mGluR5 NAM) discovered at Addex. Blockade of mGluR5 has been shown to have anti-Parkinson's disease and anti-dyskinetic effects in a variety of animal models as well as early trials in patients. The drug has potential in multiple indications, including: Parkinson's disease levodopa-induced dyskinesia (PD-LID), non-Parkinsonian dystonia, general anxiety disorder (GAD), Fragile X syndrome (autism), gastro-esophageal reflux disease (GERD/heartburn), migraine pain, depression and addiction.

Dipraglurant has two separate formulations for two distinct indications:

1) **Instant Release (IR)** for Parkinson's disease levodopa-induced dyskinesia (PD-LID)
2) **Extended Release (ER)** for non-Parkinsonian dystonia.

Addex decided to develop dipraglurant IR, in the instant release formulation, first in PD-LID given the significant unmet medical need and early (pre)clinical validation seen in targeting mGluR5 in this indication. The company successfully completed a phase IIa proof-of-concept (POC) trial in PD-LID in 2012 and is now planning to start a phase IIb/III pivotal trial in Q3 2016. Securing sufficient funding to start this pivotal trial is key for Addex to unlock considerable value of dipraglurant IR in PD-LID and potentially sign on a lucrative development and commercialization partner after successful completion. Dipraglurant IR would become a first-in-class treatment for PD-LID. At the end of 2013 development of Novartis' mGluR5 NAM mavoglurant (AFQ056) was discontinued in this indication due to lack of efficacy, which seems to be related to the poor profile of the compound rather than the target, as key opinion leaders remain convinced of the validity of the approach.

Additionally, Addex plans to explore the use of dipraglurant, in the extended release formulation, in non-Parkinsonian dystonia, a group of neurological movement disorders. This is based on scientific literature, own preclinical data and the observations made in the PD-LID POC trial, where four patients with dystonia responded positively to dipraglurant compared to three on placebo, despite the trial not powered to show this. In January 2015, Addex established a partnership with the Dystonia Medical Research Foundation (DMRF) to explore the therapeutic use of dipraglurant in the treatment of dystonia and in May 2015 signed the continuation of the collaboration with Dr. Pisani to further explore the potential of dipraglurant in rare genetic forms of dystonia.

PD-LID a major side effect of chronic levodopa use with a high unmet medical need

As noted, the first indication Addex is pursuing for dipraglurant IR is the acute treatment of levodopa-induced dyskinesia in patients with Parkinson's disease. Recently PD-LID has been recognized as an orphan indication in the US. Parkinson's disease is a slowly progressive degenerative disorder of the central nervous system that initially affects movement (tremor, rigidity, slowness of movement, and difficulty with walking and gait), and later cognition and behavior. Levodopa has been the most widely used and most effective drug to treat Parkinson's disease for over 30 years with most patients noticing an immediate improvement of early motor symptoms.

Dyskinesia (uncontrolled spasmodic or repetitive movements) is a major complication of the chronic treatment of Parkinson's disease. More than half of all persons treated with levodopa or other dopaminergic agonists subsequently develop dyskinesia after only several years on levodopa treatment. Levodopa-induced dyskinesia is a major complication of chronic levodopa use with a negative impact on the long-term treatment of this disease. According to a 2011 Datamonitor survey conducted among key opinion leaders in Parkinson's disease, levodopa-induced dyskinesia is the most important unmet medical need after a disease-modifying agent for Parkinson's disease.

Levodopa-induced dyskinesia is characterized by several distinct forms, including:

- **Chorea** (involuntary, uncontrolled, rapid jerky movements of the arms, legs and face)
- **Dystonia** (prolonged, repetitive muscle contractions causing twisting or jerky movements and unnatural postures, often painful)
- **Athetosis** (uncontrolled, slow, rhythmic twisting movements of hands and feet or other body parts)

Young age of onset, disease severity, duration of therapy, and total dose of levodopa are strongly correlated with the development of dyskinesia. The first manifestations of dyskinesia are usually dystonic and involve the foot on the same side of the body that is most affected by Parkinson's disease.

With time, dyskinesia may be classified in three main categories:

1. **Peak-dose dyskinesia** (when the level of levodopa reaches its peak in the blood stream)
2. **Diphasic dyskinesia** (also called "onset and end-of-dose dyskinesia" – when the levels of levodopa in the blood stream rise or fall)
3. **Off-period dyskinesia** (during so-called "off-periods" typically in the early morning)

Peak-dose dyskinesia occurs most frequently in roughly 80% of patients, followed by diphasic dyskinesia in 10-20% of patients. Although this classification is very useful in clinical practice, the different types of dyskinesia frequently overlap in a single patient when the disease progresses.

mGluR5 modulation provides a specific target to treat PD-LID

Support for a role of group I mGlu (mGlu1 and mGlu5) receptors in the pathogenesis of PD stems from early studies showing that mGluR5 antagonists ameliorate the motor alterations in animal models of parkinsonism and are neuroprotective against MPTP neurotoxicity in animals. However, the major breakthrough in the field of mGlu receptors research in Parkinson's disease was found in the management of levodopa-induced dyskinesia. Increased postsynaptic mGlu5 receptor density and specific striatal binding with selective mGlu5-receptor ligands are observed in MPTP-lesioned macaques with dyskinesia and in postmortem brains of Parkinsonian patients with dyskinesia. Further evidence for anti-dyskinetic efficacy of mGluR5 antagonists in levodopa-induced dyskinesia comes from preclinical studies in 6-OHDA-lesioned rats and MPTP monkeys. The use of allosteric modulation at this receptor has provided the specificity that is required to limit side effects as well as reduce dyskinesias in animal models and patients.

Dipraglurant IR formulation mimics levodopa uptake to offset peak dose dyskinesia

Addex has specifically developed an instant 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.

PD-LID complications limit the use of levodopa at the cost of increased rigidity

In many cases levodopa-induced dyskinesia limits the amount of drug that can be given, curbing symptom relief. Clinical presentations of dyskinesia vary significantly. When severe or painful, they limit therapy but when mild they usually can be tolerated by patients. However, even when mild, it is widely believed that the appearance of dyskinesia foreshadows the development of other, more disabling motor complications. Therefore even mild dyskinesia may lead the physician to reduce levodopa therapy at the cost of increased rigidity for the patient. As a result, most physicians and patients are hesitant to use levodopa early in the disease. Dopamine agonists and MAO-B inhibitors are typically used in the early stages of disease.

More than half of patients affected by PD-LID severely impacting quality of life…

PD-LID affects roughly half of Parkinson's patients after only 5-10 years of levodopa treatment with the percentage of affected patients increasing over time, up to 90% in patients treated after 10-15 years on levodopa therapy. Half of these patients consider PD-LID as disabling that severely impacts their quality of life. Moreover, the severity of dyskinesia is associated with increasing depression and increased falls.

… and increasing total treatment costs

Total medical expenses for treating Parkinson's disease in the US are estimated to amount to USD 23 bn annually or around USD 19,000 per patient per year. After patients

develop PD-LID, treatment costs for these patients increase substantially to approximately USD 26,000 per year.

Statistically significant dipraglurant IR POC results in PD-LID

In 2012 Addex successfully concluded a phase IIa proof-of-concept study in PD-LID, supported by a USD 900,000 grant from the Michael J. Fox Foundation. Top line data showed a statistically significant reduction in dyskinesia, a clinical relevant (>30% reduction in mAIMS (modified Abnormal Involuntary Movement Scale), a 50 minute reduction in "off-time", and 2.3 hours more "on-time" without dyskinesia, all in week 4. Below we present a comprehensive overview of the clinical data Addex reported for dipraglurant IR in its lead indication PD-LID.

Extensive pre-clinical and early clinical work to establish safety and early POC

Addex performed extensive pre-clinical work with dipraglurant in animal models that demonstrated early proof-of-concept in Parkinson's disease. The company also conducted three phase I clinical safety trials with a total of 114 healthy persons taking dipraglurant. The drug demonstrated a favorable safety, tolerability and pharmacokinetic profile in "Study 101" (single ascending dose and food effect trial of dipraglurant), "Study 102" (single and multiple ascending dose trial of dipraglurant IR), and "Study 103" (food effect and gender trial with dipraglurant IR).

Phase IIa POC trial design in patients with moderate-to-severe PD-LID

In March 2011 Addex moved dipraglurant IR into a phase IIa POC trial in patients with moderate or severe PD-LID. The trial was supported by a USD 900,000 grant from the Michael J. Fox Foundation. 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, where a total of 83 patients were screened. A total of 76 patients were enrolled in the trial. There was an imbalance in the percentage of patients who had deep brain stimulation with a higher number in the dipraglurant IR group. Covariance analyses did not show an impact of deep brain stimulation on the overall results.

STUDY POPULATION CHARACTERISTICS

DEMOGRAPHIC AND BACKGROUND CHARACTERISTICS	DIPRAGLURANT IR N = 52	PLACEBO N = 24
AGE IN YEARS (SD)	64.2 (7.6)	62.8 (8.3)
SEX (%)	MALE: 26 (50%) FEMALE: 26 (50%)	MALE: 12 (50%) FEMALE: 12 (50%)
ETHNIC ORIGIN - WHITE (%)	45 (86.5%)	22 (91.7%)
DURATION OF PD IN YEARS (SD)	11.62 (4.96)	11.06 (3.53)
TIME OF MOST TROUBLESOME DYSKINESIA (%)	MORNING: 8 (15.4%) MIDDAY: 36 (69.2%) EVENING: 6 (11.5%) NIGHT: 2 (3.8%)	MORNING: 5 (20.8%) MIDDAY: 13 (54.2%) EVENING: 5 (20.8%) NIGHT: 1 (4.2%)
PATIENTS WITH DEEP BRAIN STIMULATION (%)	11 (20.2%)	2 (8.3%)
DURATION OF LEVODOPA TREATMENT IN YEARS (SD)	9.85 (5.28)	8.21 (3.8)
TOTAL DAILY DOSE OF LEVODOPA IN MG (MEAN SD)	371 (288)	476 (389)
DURATION OF DYSKINESIA HISTORY PRIOR TO SCREENING IN YEARS (SD)	4.52 (4.0)	4.19 (2.28)

SOURCE: ADDEX THERAPEUTICS

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. Two patients withdrew, while three patients were removed due to protocol violations.

Primary Endpoint: Safety and Tolerability

Addex announced the top-line results of the POC trial in March 2012, followed by the presentation of the full data set at the MDS (Movement Disorder Society) annual conference in June 2012.

SUMMARY OF COMMON ADVERSE EVENTS

TOTAL EVALUATED ADVERSE EVENTS	DIPRAGLURANT IR N = 52	PLACEBO N = 24
ALL ADVERSE EVENTS	46 (88.5%)	18 (75%)
DYSKINESIA (%)	11 (21.2%)	3 (12.5%)
DIZZINESS (%)	10 (19.2%)	3 (12.5%)
NAUSEA (%)	10 (19.2%)	0 (0%)
FATIGUE (%)	8 (15.4%)	1 (4.2%)
ON AND OFF PHENOMENON (%)	6 (11.5%)	2 (8.3%)
HEADACHE (%)	6 (11.5%)	3 (12.5%)
HYPERTENSION (%)	5 (9.6%)	0 (0%)
ASTHENIA (%)	4 (7.7%)	0 (0%)
VISUAL IMPAIRMENT (%)	4 (7.7%)	0 (0%)
VERTIGO (%)	4 (7.7%)	0 (0%)
FEELING DRUNK (%)	3 (5.8%)	0 (0%)
SOMNOLENCE (%)	3 (5.8%)	3 (12.5%)
HALLUCINATIONS, VISUAL (%)	3 (5.8%)	0 (0%)
FALL (%)	3 (5.8%)	0 (0%)

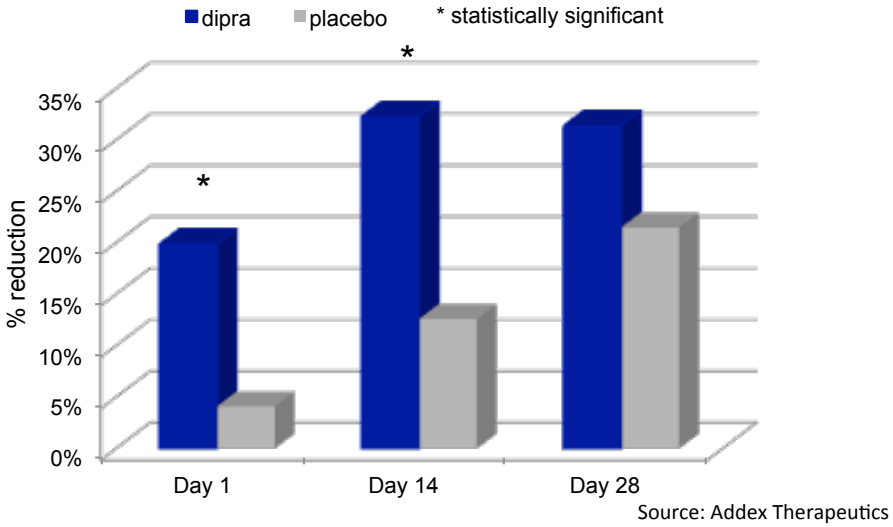
SOURCE: ADDEX THERAPEUTICS

No treatment effects were seen on any of the safety monitoring variables (e.g. ECG, heart rate, blood pressure, hematology, biochemistry). There were slightly more adverse events in the dipraglurant IR treatment group (88.5%) compared to placebo (75%). Most were mild (>80%) and not dose limiting, with the majority of patients completing the dose escalation regimen. Most common adverse events (>10%) in the dipraglurant IR group and (placebo) included: dyskinesia, dizziness, nausea, fatigue, on-off phenomenon and headache. Adverse events were marginally higher for the 100 mg dose (e.g. nervous system 40% versus placebo 29%).

Secondary/Exploratory Endpoints: Efficacy

Exploratory efficacy data showed the antidyskinetic effect of dipraglurant IR both on observer evaluated mAIMS and a trend for anti-Parkinsonian effect in the reported data.

1) Provoked mAIMS at 90 minutes (impact on peak-dose dyskinesia)

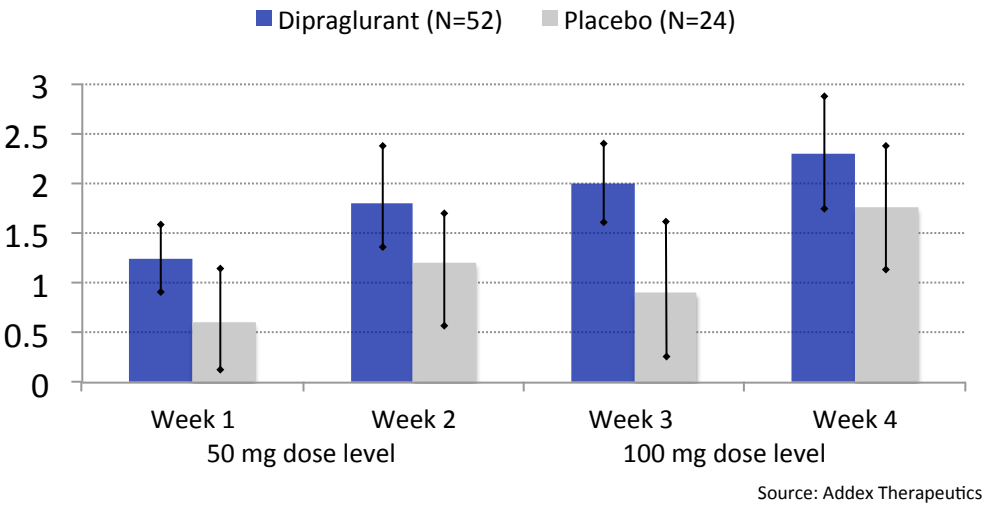


Source: Addex Therapeutics

Both the 50 and 100 mg doses of dipraglurant IR showed antidyskinetic effect. However in week 4 (day 28) an increased placebo response was seen resulting in a positive, but non-significant impact on peak-dose dyskinesia. The placebo response was thought to be caused by several factors in the trial design including: 1) visits were not blinded giving rise in clinician expectation as patient visits progressed, 2) different scorers at different sites that leads to more variable data, 3) the increase in number of pills taken during the titration, 4) not enough time at constant dose at the end of the titration period; and 5) patients were not video-recorded (no post-analysis possible).

2) Weekly diary data (48 hours) (effect on motor fluctuation from patient diaries)

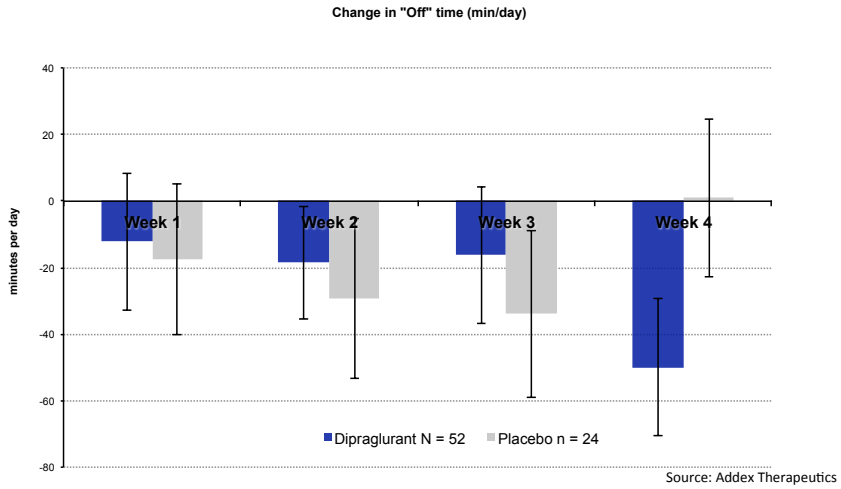
a) Impact of dipraglurant IR on daily "On" time with no dyskinesia



Source: Addex Therapeutics

In weeks 1, 2, 3 and 4, dipraglurant IR increased daily "On" time with no dyskinesia by +1,25, + 1.8, +2.0, and + 2.3 hours, respectively.

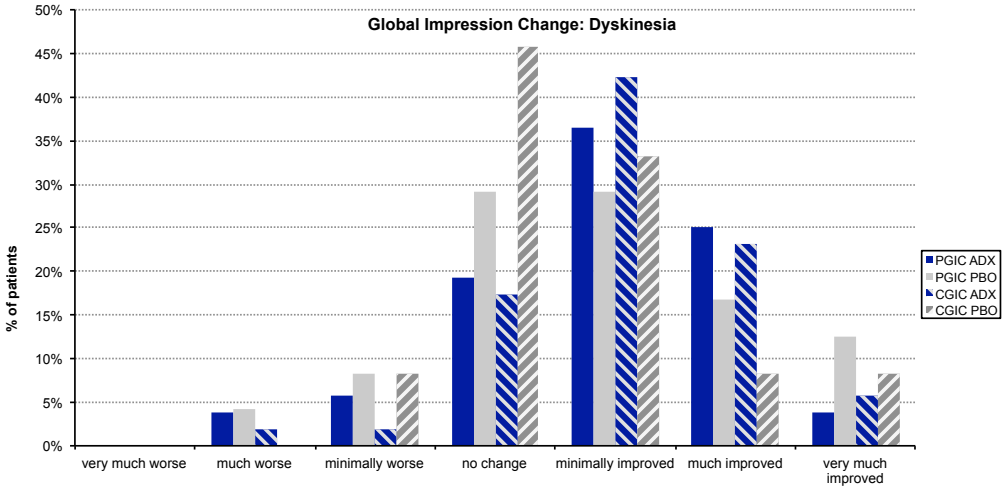
b) Impact of dipraglurant IR on daily "Off" time



Source: Addex Therapeutics

Additionally dipraglurant IR reduced daily "Off" time by 50 minutes at week 4. Dipraglurant IR did not affect levodopa efficacy in UPDRS Part III (clinician scored motor evaluation). Both parameters, daily "on" and "off" time, suggest a beneficial effect on motor fluctuation from the weekly patient diaries.

3) Patient and Clinician Global Impression of Change



Source: Addex Therapeutics

Patients PGIC: dipraglurant IR 65.3% vs. placebo 58.4%; p-value = not significant
Clinicians CGIC: dipraglurant IR 71.2% vs. placebo 49.6%; p-value = < 0.05

Clinicians rated more improvement for dipraglurant IR treatment for dyskinesia, which reached statistical significance, than patients, where a non-significant trend in favor of dipraglurant IR was seen.

Conclusion – Phase IIa POC primary endpoint met, further investigation warranted
The investigators concluded that the study met its primary endpoint in demonstrating good safety and tolerability in patients with PD-LID. Exploratory efficacy data showed antidyskinetic effect as measured by observer evaluated mAIMS and in patient-reported diary data. No negative effect of dipraglurant IR was seen on Parkinson's symptoms, with the suggestion of a beneficial effect on motor fluctuation from patient diaries, which warrants further investigation.

Future development plans of dipraglurant IR in PD-LID

To further enhance the value of dipraglurant IR, Addex has now set up a comprehensive development plan, in close consultation with key opinion leaders in the field, to explore the use of dipraglurant IR in PD-LID and potentially other indications. The current development plan includes:

- **An open label PET imaging study** receptor occupancy trial in 15 subjects (funded)
- **A phase IIb/III pivotal trial in 80-120 patients (under discussion with KOL's and regulatory experts)** The study will be multi-center (US/EU), randomized, double-blind, placebo-controlled, adaptive / seamless phase IIb/III pivotal trial with the primary objective of evaluating the efficacy of dipraglurant IR co-administered with levodopa in subjects with Parkinson's disease experiencing levodopa-induced dyskinesia (PD-LID). The study will combine "treatment selection" and "confirmation" in one trial and will be adequately powered and designed to be used as a pivotal trial. Dipraglurant IR will be administered 3 times daily, together with levodopa, for a duration of 12 weeks of treatment (on the stable dose). A validated rating scale (UDysRS - Unified Dyskinesia Rating Scale) will be used to assess efficacy. The study will also include quality of life endpoint measurements. This trial is planned to start in Q3 2016 with results in 2017. The preparation of the trial is supported by a USD 1 mn grant from the MJFF. NOTE: The start of the trial depends on Addex securing sufficient funding of approximately CHF 7 mn.
- **A phase III confirmatory trial in 120 patients**, in 3 treatment arms, with two doses given 3 times daily in combination with levodopa for 25 weeks. The trial will include an extension, open label trial with 200 patients for 52 weeks. This is an expensive trial, which we believe would cost approximately CHF 20 mn. In our forecasts, we assume a development and commercialization partner that signs on after successful phase IIb/III results will cover these costs. Alternatively, Addex could raise cash, however, at the cost of dilution. This trial, likely required by the EMA, could tentatively start in 2018 with results in 2020. EU filing could occur a year later with first launch starting in 2022. NOTE: if orphan drug designation (ODD) is obtained in the EU, the trial design could be simplified.

Academic collaborations are ongoing in other disease areas for dipraglurant, including:

- Treatment of migraine
- Treatment resistant depression

Potential to become a cornerstone treatment in PD-LID

With no cure for Parkinson's disease, the primary aim of therapy is to relieve patients from Parkinson symptoms, keep the patient functional as long as possible, and avoid treatment related side effects as much as possible. These side effects typically develop after 4–10 years of levodopa therapy, and affect approximately 50-75% of all patients. The "wearing-off" effect is the most common type, and "delayed-on," "no-on," and "on-off" fluctuations, as well as dyskinesia (in roughly 40% of patients) and cognitive worsening, may also develop as the disease progresses. Collectively, motor fluctuations represent a significant source of disability in advanced Parkinson's patients, and reducing these is a major goal of patient management. Adjunctive medications, including dopamine agonists, anticholinergics, MAO-B inhibitors, and COMT inhibitors, each may reduce the frequency or duration of "off"

periods, but none does so completely, and each contributes its own side effects which may limit optimal dosing.

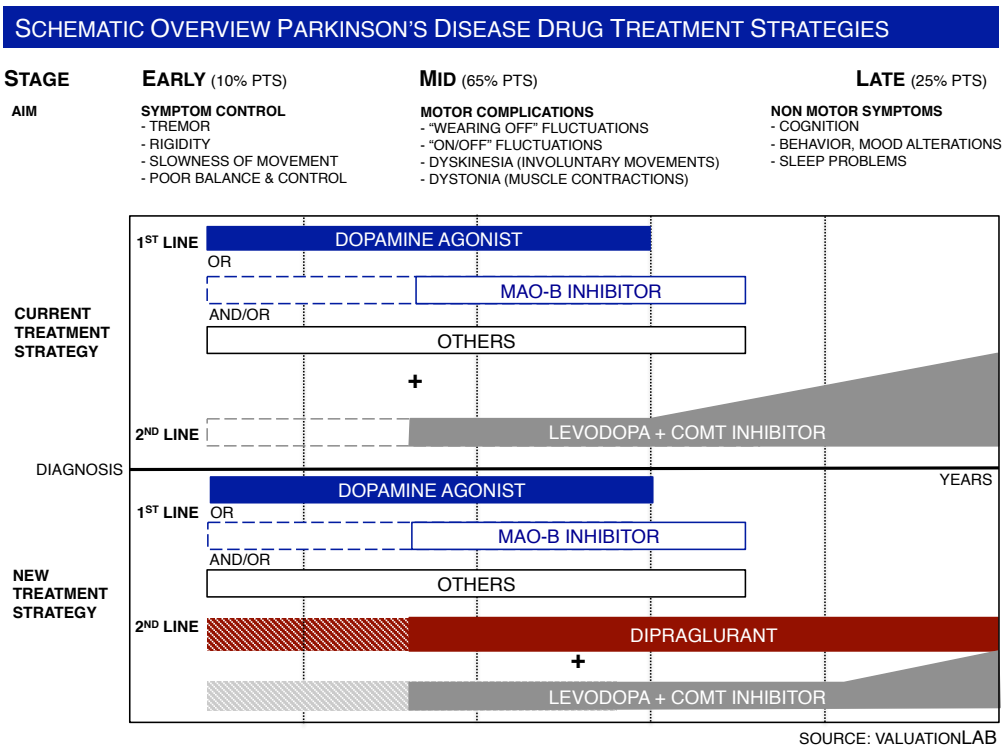
Therapies aim to delay and limit levodopa use to avoid appearance of dyskinesia

These problems have led to the development of strategies, which aim to limit or delay the onset of levodopa-related complications and have become the key drivers for the Parkinson's disease market. Dopamine agonists and MAO-B inhibitors are primarily given in the early stages of disease to delay the use of levodopa. As the disease progresses, patients are then given levodopa, at the lowest effective dose. With further disease progression the dose of levodopa usually has to be increased to maintain control of Parkinson symptoms, however, often at the cost of levodopa-induced side effects such as dyskinesia.

Potential to use levodopa earlier in treatment in combination with dipraglurant IR

We believe dipraglurant IR has such a promising profile in that it has the potential to reduce levodopa-induced dyskinesia without worsening Parkinson symptoms. Moreover, the drug may have a favorable effect on dystonia, as seen in seven patients in the POC trial. As a result, dipraglurant IR could be given to patients early in disease on top of mainstay levodopa to potentially delay the occurrence and reduce the onset of PD-LID after prolonged levodopa use. Consequently, physicians would be able to use levodopa, the most effective Parkinson's treatment to control symptoms and keep patients as functional as long as possible, early in the disease, without the risk of patients developing PD-LID after only several years into treatment.

SCHEMATIC OVERVIEW PARKINSON'S DISEASE DRUG TREATMENT STRATEGIES



SOURCE: VALUATIONLAB

Challenge is to find drugs that treat PD-LID but do not worsen Parkinson symptoms

Current treatments leave significant room for improvement both in terms of sustained efficacy and safety/tolerability. The challenge is to find drugs that treat PD-LID but do not worsen Parkinson symptoms and demonstrate maintenance of effect for a sufficient duration of time. Novartis departure from the field with its mGluR5 inhibitor, mavoglurant

(AFQ056), at the end of 2013 due to lack of efficacy leaves Addex' dipraglurant IR as the first in class compound for treating PD-LID.

Other compounds in development for treating PD-LID include:

- **ADS-5102** (amantadine controlled-release) from Adamas Pharmaceuticals is now the most advanced PD-LID treatment in development and is designed to address the limitations of immediate-release amantadine by increasing its plasma concentration. Amantadine is considered a weak therapy for Parkinson's disease. Based on positive phase II/III "EASED" trial results in PD-LID, the compound is now in phase III development. On positive results, Adamas plans to file an NDA in 2016, with ADS-5102 potentially becoming the first approved treatment for PD-LID. In April 2015 the FDA granted ADS-5102 orphan drug designation for treating PD-LID, thereby recognizing PD-LID as an orphan indication for the first time.
- **Eltoprazine** (5HT1a/1b partial agonist) from Amarantus BioScience is a small molecule drug, which reported positive results in a phase IIa POC in 22 patients with PD-LID in 2012. Management targets USD 750 mn peak sales for PD-LID in the US alone (we are more conservative with our dipraglurant IR PD-LID forecast of USD 460 mn in the US). Eltoprazine is also being evaluated in a number of other neurology-focused indications including ADHD (attention deficit hyperactivity disorder) and cognition.
- **AVP-923** (dextromethorphan/quinidine) from Avanir Pharmaceuticals is a fixed dose combination therapy that started enrolling 16 patients with PD-LID in a phase IIa POC trial in 2013. AVP-923's lead indication is agitation in patients with Alzheimer's disease. AVP-923 use may be limited due to certain cardiovascular risks and drug interactions. In 2014, Otsuka Pharmaceutical acquired Avanir for USD 3.5 bn.
- **AQW051** (alpha7 nicotinic receptor) from Novartis has shown promise in pre-clinical animal models of PD-LID in MPTP monkeys. This compound may be the follow-up of Novartis' mavoglurant (AFQ056), also an mGluR5 NAM like dipraglurant that was discontinued in PD-LID in 2014 due to the lack of efficacy. No safety concerns were reported.

Blockbuster peak sales achievable for dipraglurant IR in PD-LID

Based on the promising profile of dipraglurant IR reported in the positive POC trial and limited compounds in development for PD-LID, we believe dipraglurant IR could generate blockbuster sales potential when developed successfully. Of the roughly 800,000 diagnosed Parkinson's patients in the US today, roughly 65% of these have mid-stage disease with around 40% experiencing PD-LID. Assuming a pricing of CHF 20 per patient per day (reflecting a relatively low price for an orphan disease) and a peak penetration of 25%, this would amount to peak sales of CHF 380 mn today and peak sales of CHF 480 mn in the US in 2025 (assuming a 2% annual patient growth) one year before loss of orphan drug exclusivity. Assuming orphan drug designation granted by the FDA in the fall, US launch could occur as soon as 2020.

A similar calculation can be made for Europe and the rest of the world, where a larger patient base leads to peak sales of almost CHF 470 mn in 2024. However, we assume a significantly lower daily treatment price of CHF 8 per patient (where we do not assume orphan drug designation), peak penetration of 15% and first launch to occur in 2022 (for details see "Forecast and Sensitivity Analysis" on the following page).

Forecasts & Sensitivity Analysis
Dipraglurant IR in PD-LID

DIPRAGLURANT-IR (MGLUR5 NAM) - FINANCIAL FORECASTS FOR PARKINSON'S DISEASE

INDICATION	PARKINSON'S DISEASE: ACUTE TREATMENT OF MODERATE-TO-SEVERE LEVODOPA INDUCED DYSKINESIA
DOSAGE	3-4X DAILY 50-100 MG
PRICE	US: USD 20 PER DAY / EU: CHF 8 PER DAY
STANDARD OF CARE	NO TREATMENT APPROVED TO TREAT LEVODOPA-INDUCED DYSKINESIA - DELAY USE OF LEVODOPA WITH DOPAMINE AGONISTS AND MAO-B INHIBITORS
UNIQUE SELLING POINT	POTENTIALLY FIRST DRUG ON THE MARKET TO TREAT DYSKINESIA CAUSED BY CHRONIC LEVODOPA TREATMENT

7Ps ANALYSIS

PATENT	AT LEAST 2025 (COMPOSITION OF MATTER) EP1765795 GRANTED 2013; UP TO 2035 (POLYMORPH PATENTS) FILED 2015; POTENTIAL 7 YEARS US ORPHAN DRUG EXCLUSIVITY
PHASE	PHASE IIA COMPLETED, PHASE IIB/III PLANNED FOR Q3 2016; DEVELOPMENT DEPENDS ON ORPHAN DRUG DESIGNATION AND SUFFICIENT FUNDING (OR PARTNERING)
PATHWAY	STRAIGHTFORWARD REGULATORY PATHWAY: REDUCING DYSKINESIA & "OFF-TIME", WHILE IMPROVING "ON-TIME"; POTENTIAL ORPHAN DRUG DESIGNATION IN US
PATIENT	REAL IMPROVEMENT IN QUALITY OF LIFE OF PATIENT WITH LESS DEBILITATING DYSKINESIA LOWERING THE NEED FOR OUTSIDE HELP
PHYSICIAN	PATIENTS CAN BE GIVEN MAINSTAY LEVODOPA EARLIER AND MORE AGGRESSIVELY EXTENDING THE TIME THIS EFFECTIVE TREATMENT CAN BE GIVEN
PAYER	REDUCTION OF DYSKINESIA DELAYS AND SIGNIFICANTLY LOWERS COSTS OF OUTPATIENT CARE, NURSING HOMES OR HOSPITALIZATION
PARTNER	MJFF * FUNDED PHASE IIA AND PARTIALLY PHASE IIB/III - PARTNERING OPPORTUNISTIC; WE ASSUME ADDEX TO RAISE FUNDS FOR PHASE IIB/III AND THEN LICENSE OUT

REVENUE MODEL

EUROPE / REST OF WORLD	**2014E**	**2015E**	**2016E**	**2017E**	**2018E**	**2019E**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**
NUMBER OF PARKINSON'S PATIENTS (MN)	3.4	3.4	3.5	3.6	3.6	3.7	3.8	3.9	3.9	4.0	4.1
GROWTH (%)	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
PATIENTS DIAGNOSED (%)	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%	70%
PATIENTS DIAGNOSED (MN)	2.3	2.4	2.4	2.5	2.5	2.6	2.6	2.7	2.8	2.8	2.9
MID-STAGE PATIENTS (%)	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%
NUMBER OF MID-STAGE PATIENTS (MN)	2.2	2.2	2.3	2.3	2.4	2.4	2.5	2.5	2.6	2.6	2.7
MID-STAGE PATIENTS WITH PD-LID (%)	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
NUMBER OF PATIENTS WITH PD-LID (MN)	0.9	0.9	0.9	0.9	0.9	1.0	1.0	1.0	1.0	1.0	1.1
PENETRATION (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	4.0%	10.0%	15.0%
NUMBER OF PATIENTS (MN)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2
COST OF THERAPY PER YEAR (CHF)	2'920	2'920	2'920	2'920	2'920	2'920	2'920	2'920	2'920	2'920	2'920
SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**119**	**304**	**466**
CHANGE (%)										155%	53%
ROYALTY (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	25.0%	25.0%	25.0%
ROYALTIES (CHF MN)	0	0	0	0	0	0	0	0	30	76	116
UPFRONT & MILESTONE PAYMENTS (CHF MN)		0	0	30	0	0	0	20	30	0	50
R&D COSTS (CHF MN)	0	-1	-6	-3	-6	-10	-9	-1	0	0	0
PROFIT BEFORE TAX (CHF MN)	**0**	**-1**	**-6**	**27**	**-6**	**-10**	**-9**	**19**	**60**	**76**	**166**
TAX RATE (%)	0%	0%	0%	0%	0%	0%	0%	0%	20%	20%	20%
TAXES (CHF MN)	0	0	0	0	0	0	0	0	-12	-15	-33
PROFIT (CHF MN)	**0**	**-1**	**-6**	**27**	**-6**	**-10**	**-9**	**19**	**48**	**61**	**133**

UNITED STATES	**2014E**	**2015E**	**2016E**	**2017E**	**2018E**	**2019E**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**
NUMBER OF PATIENTS (MN)	1.1	1.1	1.1	1.1	1.2	1.2	1.2	1.2	1.3	1.3	1.3
GROWTH (%)	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
PATIENTS DIAGNOSED (%)	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%
NUMBER OF PATIENTS DIAGNOSED (MN)	0.9	0.9	0.9	0.9	0.9	1.0	1.0	1.0	1.0	1.0	1.1
MID STAGE PATIENTS (%)	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%
NUMBER OF MID STAGE PATIENTS (MN)	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.7	0.7	0.7
MID STAGE PATIENTS WITH PD-LID (%)	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%	40%
NUMBER OF PATIENTS WITH PD-LID (MN)	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.3	0.3	0.3	0.3
PENETRATION (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	5.0%	11.0%	16.0%	20.0%	23.0%
NUMBER OF PATIENTS (MN)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1
COST OF THERAPY PER YEAR (CHF)	7'090	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844
SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**87**	**195**	**289**	**368**	**432**
CHANGE (%)								124%	48%	28%	17%
ROYALTY (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	25.0%	25.0%	25.0%	25.0%	25.0%
ROYALTIES (CHF MN)	0	0	0	0	0	0	22	49	72	92	108
UPFRONT & MILESTONE PAYMENTS (CHF MN)	0	0	0	0	0	0	0	0	0	30	0
R&D COSTS (CHF MN)	0	0	0	0	0	0	0	0	0	0	0
PROFIT BEFORE TAX (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**22**	**49**	**72**	**122**	**108**
TAX RATE (%)	0%	0%	0%	0%	0%	0%	5%	15%	20%	20%	20%
TAXES (CHF MN)	0	0	0	0	0	0	-1	-7	-14	-24	-22
PROFIT (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**21**	**41**	**58**	**98**	**86**

	2014E	**2015E**	**2016E**	**2017E**	**2018E**	**2019E**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**
GLOBAL SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**87**	**195**	**408**	**673**	**898**
CHANGE (%)								124%	110%	65%	33%
GLOBAL PROFIT (CHF MN)	**0**	**-1**	**-6**	**27**	**-6**	**-10**	**12**	**60**	**106**	**159**	**220**
CHANGE (%)						67%	-216%	420%	75%	50%	38%

WACC (%)	7.0%
NPV TOTAL PROFIT (CHF MN)	**502**
NUMBER OF SHARES (MN)	18.4
NPV PER SHARE (CHF)	**27.3**
SUCCESS PROBABILITY	15%
RISK ADJUSTED NPV PER SHARE (CHF)	**4.1**

SENSITIVITY ANALYSIS

		WACC (%)							
	CHF/SHARE	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0
	50%	15.5	14.9	14.2	13.6	13.1	12.5	12.0	11.5
	45%	14.0	13.4	12.8	12.3	11.8	11.3	10.8	10.4
	40%	12.4	11.9	11.4	10.9	10.4	10.0	9.6	9.2
	35%	10.9	10.4	10.0	9.5	9.1	8.8	8.4	8.1
SUCCESS PROBABILITY	30%	9.3	8.9	8.5	8.2	7.8	7.5	7.2	6.9
	25%	7.8	7.4	7.1	6.8	6.5	6.3	6.0	5.8
	20%	6.2	5.9	5.7	5.5	5.2	5.0	4.8	4.6
	15%	4.7	4.5	4.3	**4.1**	3.9	3.8	3.6	3.5
	10%	3.1	3.0	2.8	2.7	2.6	2.5	2.4	2.3

* MJFF = MICHAEL J. FOX FOUNDATION

ESTIMATES AS OF 4 AUGUST, 2015

SOURCE: VALUATIONLAB ESTIMATES

Unique Selling Point

With no effective treatment for Parkinson's disease levodopa-induced dyskinesia (PD-LID), dipraglurant IR has the potential to become first in class and one of the first drugs for this indication. Successful treatment of PD-LID could change the way Parkinson's disease is treated, enabling physicians to use levodopa, earlier and more aggressively.

7P's Analysis

Patent: A solid composition-of-matter patent protects dipraglurant IR until 2025. Assuming first launches in 2020, the drug has an effective patent life of at least 5 years. Additional patents covering polymorphs and formulations of dipraglurant could extend patent protection to 2032. Orphan drug market exclusivities could provide longer protection as well (7 years and 10 years from launch in the US and EU, respectively).

Phase: Addex has completed a phase IIa proof-of-concept study in levodopa-induced dyskinesia in Parkinson's disease. The company is preparing, in consultation with key opinion leaders, for a phase IIb/III study in PD-LID, scheduled to start in Q3 2016. The initiation of the study depends on securing sufficient funds through investors or a partner. The estimated probability of success at this stage of development is 15%.

Pathway: We believe the regulatory pathway in PD-LID is quite straightforward and similar to the proof-of-concept trial demonstrating a clinical benefit in reducing dyskinesia and "off-time", while improving "on-time". Approval should be attained with at least one positive pivotal phase III demonstrating a significant benefit in reducing levodopa-induced dyskinesia using the UDysRS score. Orphan drug designation could simplify the regulatory pathway potentially with less patients, lower costs and faster timelines.

Patient: With no effective treatment for PD-LID, dipraglurant IR has the potential to have a major change on the quality of life for patients with moderate to severe Parkinson's disease, by reducing debilitating dyskinesia without worsening Parkinson’s symptoms. Moreover, patients can postpone the need for outside help.

Physician: Dipraglurant IR can be added to mainstay levodopa therapy, thereby reducing one of the main side effects of levodopa being dyskinesia. Consequently, moderate PD patients can be given levodopa treatment earlier and more aggressively, and severe PD patients can be treated longer and more aggressively with levodopa.

Payer: The largest share of direct cost in Parkinson's disease comes from inpatient care and nursing homes, while the share from medication is substantially lower. Any delay in the progression of the disease or reduced debilitating side effects, in particular dyskinesia, has a substantial impact on total treatment costs. The average total treatment cost of a Parkinson’s patient in the US rises to USD 26,000 from USD 19,000 when PD-LID occurs.

Partner: The Michael J. Fox Foundation supported the phase IIa development with a USD 900,000 grant. With the support of an additional USD 1 mn grant Addex plans to start a phase IIb/III trial in PD-LID in Q3 2016. The company plans to develop dipraglurant by itself with the help of grants and/or investments from investors. Addex continues to actively seek a development and commercialization partner to fully develop dipraglurant in its two main indications, PD-LID and non-Parkinsonian dystonia’s.

Parkinson's Disease Market

Parkinson's disease is the second most common neurodegenerative disorder after Alzheimer's disease. Nevertheless, the Parkinson's disease market is relatively small in terms of sales at around USD 4 bn, reflecting the lack of new efficacious treatment introductions, with most drugs no longer patent protected. Major players included Novartis, Bristol-Myers Squibb and GlaxoSmithKline. Several smaller players have developed new formulations (extended/controlled-release, patches, orally disintegrating tablets) extending the patent life of some existing branded drugs. The combined direct (medication, inpatient care) and indirect cost (inability to work) of Parkinson's disease is estimated to be nearly USD 23 bn per year in the US alone.

PARKINSON'S DISEASE - KEY FACTS	
MARKET SIZE	USD 4 BN
PREVALENCE	7-10 MN GLOBALLY, 1 MN IN US, > 1 MN IN EU
INCIDENCE	300,000 GLOBALLY, 100,000 IN US, >100,000 IN EU; 0.3% OF POPULATION
UNDERLYING CAUSE	- LOSS AND DEGENERATION OF DOPAMINERGIC NEURONS IN STRIATA NIGRA - LOSS OF STRATIAL NEUROTRANSMITTER DOPAMINE
SYMPTOMS	- TREMOR (SHAKING OF HANDS, ARMS, LEGS, JAW, FACE) - RIGIDITY (LIMBS, TRUNK) - BRADYKINESIA (SLOWNESS OF MOVEMENT) - POSTURAL INSTABILITY (POOR BALANCE AND COORDINATION)
DRUG CLASS (KEY BRANDS)	- LEVODOPA/CARBIDOPA (MADOPAR, SINEMET CR, PARCOPA, STALEVO, DUODOPA) - DOPAMINE AGONIST (MIRAPEX, REQUIP, APOKYN, PARLODEL, NEUPRO PATCH) - MAO-B INHIBITORS (AZILECT, ELDEPRYL, ZELAPAR ODT, XADAGO) - COMT INHIBITORS (COMTAN, TASMAR) - ANTICHOLINERGICS (COGENTIN, ARTANE) - OTHER (SYMMETREL FOR DYSKINESIA, EXELON FOR DEMENTIA)
MAJOR PLAYERS (KEY BRANDS)	- NOVARTIS (STALEVO, PARLODEL, COMTAN) - BRISTOL MYERS SQUIBB (SINEMET CR) - GLAXOSMITHKLINE (REQUIP) - TEVA (AZILECT) - UCB (NEUPRO PATCH) - BOEHRINGER INGELHEIM (MIRAPEX ER) - US WORLDMEDS (APOKYN) - VALEANT (ZELAPAR ODT, TASMAR) - ABBVIE (DUODOPA) - ENDO PHARMACEUTICALS (SYMMETREL) - ZAMBON/NEWRON (XADAGO)

SOURCE: VALUATIONLAB, NIH, WHO, PARKINSONS.ORG, PDF.ORG

Parkinson's disease affects an estimated 7-10 million people globally with about 1 million patients in the US and a similar amount in the EU, with significant prevalence growth expected due to an aging population. The disease is a slowly progressive degenerative disorder of the central nervous system that initially affects movement, and later cognition and behavior. Dementia commonly occurs in the advanced stage of disease. The mean age of onset is typically around 60 years (rare in people under the age of 40 years). In people taking medication (levodopa), the progression time of symptoms to a stage of high dependency from caregivers may range from 8 to 15 years.

Three stages of severity are usually distinguished;

1) **Early stage**, in which the patient has developed some disability and where drug treatment may be required (dopamine agonists, anticholinergics, MAO-B inhibitors)
2) **Mid stage**, where the symptoms can be rather severe and include the inability to walk straight or stand, with a noticeable slowing of movements (bradykinesia).
3) **Late or advanced stage**, in which an individual develops severe motor complications (dyskinesia) related to levodopa use. Most patients are unable to complete day-to-day tasks and usually cannot live on their own.

Early in the disease the most obvious symptoms are movement-related. These include tremor, rigidity, slowness of movement, and difficulty with walking and gait. The motor symptoms of the disease result from the death of dopamine-generating cells in the

substantia nigra, a small tract of neurons in the brain containing dopamine, which control voluntary movements. The cause of this cell death is still unknown.
The severity and progression of Parkinson's disease is measured using several rating scales such as the **Hoehn and Yahr** (focus on movement symptoms) or **UPDRS** (United Parkinson's Disease Rating Scale - more comprehensive than Hoehn and Yahr, taking into account cognitive difficulties, daily activities and treatment complications).

Current drug treatment aims to delay symptoms and use of levodopa
Because there is no cure for Parkinson's disease, the primary aim of treatment is to relieve symptoms and keep the patient functional as long as possible. Current treatments are effective at managing the early motor symptoms, mainly through the use of (generic) levodopa and dopamine agonists. Mainstay treatment is levodopa, an oral precursor of the neurotransmitter dopamine. It is well established as the most effective treatment for Parkinson's disease for over 30 years, with most patients noticing an immediate improvement. However, as the disease progresses and dopamine generating cells continue to be lost, these drugs eventually become ineffective at treating the symptoms and at the same time produce **dyskinesia**, a complication marked by involuntary jerking and twisting movements. Other treatment related complications include end-of-dose deterioration, unpredictable "on/off" motor fluctuations, hypotension, nausea, anorexia and psychiatric effects. These problems have led to the development of strategies that aim to limit or delay the onset of levodopa-related complications and have become the key drivers for the Parkinson's disease market with the introduction of dopamine agonists, MOA-B and COMT inhibitors. Dopamine agonists and MAO-B inhibitors are primarily used as mono-therapy in the early stages of the disease to delay the use of levodopa. **Dopamine agonists** work by directly stimulating the dopamine receptors to bypass degenerating brain cells. **MOA-B inhibitors** block a key enzyme that is responsible for the breakdown of dopamine. **COMT-inhibitors** block an enzyme responsible for the breakdown of levodopa in the body, thereby increasing the amount of levodopa available to reach the brain. Consequently COMT inhibitors are prescribed together with levodopa. When drug treatment is no longer sufficient to control symptoms, lesional surgery or deep brain stimulation (DBS), through implantation of a so-called brain pacemaker can be of use. In the final stages of disease, palliative care is provided to enhance quality of life.

New market entrants expected to spark growth
The introduction of new drugs, improved formulations of existing drugs (e.g. improved formulations of levodopa), and the ageing of the population (higher prevalence) should drive growth in the Parkinson’s disease market.

New molecules and novel approaches include: Newron/Zambon's **Xadago** (approved in EU, re-filed in the US) a dual mechanism of action drug that provides both MAO-B and glutamate inhibition, adenosine 2a (A2a) agonists such as Kyowa-Kirin's **istradefylline** (US: phase III SPA, Japan approved as Nouriast) and Biotie's **tozadenant** (phase III 2015E). New drugs that specifically target levodopa-induced dyskinesia include: Adamas’ **ADS-5102** (phase III), Amarantus BioScience’s **eltoprazine** (phase II), Addex’ **dipraglurant IR** (phase II), Avanir’s **AVP-923** (phase II) and Novartis’ **AQW051** (preclinical) (for details see page 25)

Pipeline – Targeted R&D projects that can unlock value

Addex has identified 3 early stage pipeline projects, which can unlock substantial value in a relatively short time with limited funds required. These include:

1) **Dipraglurant ER** for treating non-Parkinsonian dystonia
2) **ADX71441 ($GABA_B$ PAM)** for treating Charcot-Marie-Tooth
3) **ADX71441 ($GABA_B$ PAM)** for treating alcohol/nicotine addiction

The company has established government, academic and patient group collaborations to advance its pipeline and technology platform at a relatively low cost, and is actively seeking investment. Management continues to pursue partnerships for all projects, but now opportunistically. Addex will pursue ADX71441 ($GABA_B$ PAM) in addiction only when it secures sufficient funding from an external partner. Revised plans for the key pipeline projects have been drafted with key opinion leaders (KOL's) and therapeutic area focused KOL advisory panels have been put in place. Progression of each project leads to a substantial increase in value and a higher probability of approval and partnering.

NOTE: Only dipraglurant IR (PD-LID) and ADX71149 (new undisclosed major CNS indication to be announced around year-end by Janssen Pharmaceuticals Inc.) are in our current valuation, as the other targeted projects have no validated proof-of-concept, yet.

1) Dipraglurant ER - Non-Parkinsonian dystonia's

A second major indication for Addex' dipraglurant ER, in the extended release formulation, is for treating non-Parkinsonian dystonia's. Dystonia is a hyperkinetic movement disorder characterized by involuntary and sustained muscle contractions that produce repetitive abnormal, sometimes painful, movements and positions (postures). The movements may resemble a tremor. Dystonia is often initiated or worsened by voluntary movements, and symptoms may overflow into adjacent muscles. There are multiple types of dystonia and numerous diseases and conditions may cause dystonia.

Dystonia is classified by:

1) **Clinical characteristics:** such as age onset, body distribution, nature of symptoms, associated features such as neurological symptoms
2) **Cause:** primary (idiopathic or genetic/hereditary) or secondary (induced by drugs (e.g. neuroleptics), toxins (e.g. lead poisoning), infection, or metabolic disorders

Dystonia can affect a single part of the body (focal), multiple areas (segmental) or the whole body (generalized). Clinicians use these classifications to guide diagnosis and treatment. Specific information on the prevalence of dystonia has been difficult to establish due to different methodologies for case ascertainment. A meta-analysis points to a prevalence of primary dystonia of 16.43 per 100,000. An estimated 300,000 people in the United States have been diagnosed with a dystonia of some type.

Cause unknown but excess glutamate release a common underlying mechanism

The precise cause of primary dystonia is unknown. Dystonia is thought to be caused by an abnormality in or damage to the central nervous system, likely originating in those parts of the brain concerned with motor function, such as the basal ganglia, and the GABA

(gamma-aminobutyric acid) producing Purkinje neurons located in the cerebellum. There may be abnormalities in the brain's ability to process a group of chemicals called neurotransmitters that help cells in the brain communicate with each other. There also may be abnormalities in the way the brain processes information and generates commands to move. In many cases it may involve some genetic predisposition towards the disorder combined with environmental conditions. Nevertheless, all have a common underlying mechanism: an excess glutamate release in brain regions controlling movement.

Non-Parkinsonian dystonia's – no viable treatments available

Currently, there are no medications to prevent dystonia or slow its progression. There are, however, several treatment options that can ease some of the symptoms of dystonia, so physicians can select a therapeutic approach based on each individual's symptoms.

Current non-medical treatment options for dystonia include:

- **Physical intervention:** such as physical therapy and massage, including sensory biofeedback techniques (effects questionable).
- **Surgery:** such as denervation of selected muscles to provide relief (irreversible) or deep brain stimulation for severe generalized dystonia's, which reduces severity of symptoms by approximately 50%, but significantly varies between individuals with DYT1 patients benefiting most.

Several medications are used to treat dystonia in an effort to find a combination that is effective for a specific person. Not all patients respond well to the same medications. Medications that have had positive results in some dystonia patients include:

- **Anticholinergics:** which block the effects of the neurotransmitter acetylcholine may provide some relief, such as benzatropine and trihexyphenidyl; sedation and memory loss limit their usefulness at higher doses and in older patients.
- **Anticonvulsants:** such as clonazepam, diazepam or carbamazepine that regulate the neurotransmitter GABA; drowsiness is their common side effect.
- **Muscle relaxants:** Baclofen continuously delivered by a pump placed in the abdomen is used to treat patients exhibiting muscle spasticity along with dystonia.
- **Botox injections:** into dystonic muscles can reduce spasms for 1 to 4 months. However, repeated injections become less effective due to production of neutralizing antibodies. Dysport (by Ipsen) is a next-generation injection approved to treat cervical (neck muscles) dystonia.

Potential to treat dystonia backed by pre-clinical models and in PD-LID patients

Dipraglurant has the potential to reduce excess glutamate release in the brain regions controlling movement, by inhibiting mGluR5 receptor activity. Dipraglurant has shown positive anti-dystonia effect in multiple animal models as well as in Parkinson's patients. Dipraglurant has demonstrated encouraging results in animal models such as the PD-LID MPTP monkey (drug-induced dyskinesia with features of dystonia); the DYT1 mouse model (genetic generalized dystonia); and the Tottering mouse model (paroxysmal dystonia).

Importantly, in the phase IIa POC trial of dipraglurant IR in patients with PD-LID, seven patients that also had dystonia, experienced a positive effect after 4 weeks treatment. This was the first early evidence of efficacy in humans with dystonia, and supports Addex decision to explore the use of dipraglurant in dystonia.

Clinical trial program dipraglurant ER in dystonia

- **Phase II POC trial H1 2016:** Addex plans to start a phase IIa POC trial in patients with focal cervical dystonia. The study design currently defined in collaboration with Dr. Jinnah and the DMRF is expected to have an adaptive design in 10 patients with an escalating dose of dipraglurant, one-day protocol in the same patient – possibility to follow up with repeated dosing (funding of approximately CHF 0.5 mn required).

Orphan drug designation and a phase III trial design is under discussion with an advisory panel of KOL's in the field of dystonia

Peak sales of CHF 500+ mn in non-PD dystonia (NOT included in our valuation)

Peak sales in dystonia could amount to CHF 500+ mn assuming an annual treatment prices between USD 7,300 and CHF 4,380, penetration rates reaching up to 30% and patent protection until 2032 (based on ER formulation patent). We assume Addex successfully raises approximately CHF 8 mn to fund the POC and phase IIb/III pivotal trials, and then signs on a partner in return for upfront, development and sales milestones, and a 25% royalty on sales. Positive POC results would lead to a jump of CHF 4.3/share.

DIPRAGLURANT-ER (MGLUR5 NAM) - FINANCIAL FORECASTS FOR NON-PARKINSONIAN DYSTONIA

INDICATION	NON-PARKINSONIAN DYSTONIA
DOSAGE	ONCE OR TWICE DAILY 50-100 MG
PRICE	US: USD 20 PER DAY / EU: CHF 12 PER DAY
STANDARD OF CARE	MUSCLE RELAXANTS (BACLOFEN), ANTICONVULSANTS (CLONAZEPAM, DIAZEPAM), BOTOX INJECTIONS OR DEEP BRAIN STIMULATION
UNIQUE SELLING POINT	EFFECTIVE THERAPY THAT TARGETS UNDERLYING MECHANISM (EXCESS GLUTAMATE RELEASE IN BRAIN CONTROLLING MOVEMENT) WITH GOOD TOLERABILITY

7PS ANALYSIS

PATENT	AT LEAST 2025 (COMPOSITION OF MATTER) EP1765795 GRANTED 2013; UP TO 2033 (EXTENDED RELEASE) WO2013186311 FILED 2013; UP TO 2035 (POLYMORPH PATENTS)
PHASE	PHASE IIA PROOF-OF-CONCEPT TRIALS TO START IN H1 2016 WITH RESULTS IN 2016 - POSITIVE POC UNLOCKS SUBSTANTIAL VALUE WITH 15% SUCCESS PROBABILITY
PATHWAY	NON-PARKINSONIAN DYSTONIA COULD POTENTIALLY BE SEEN AS AN ORPHAN DRUG INDICATION LEADING TO FASTER REVIEW TIMES
PATIENT	POTENTIAL REDUCTION OF AMOUNT AND SEVERITY OF MUSCLE CONTRACTIONS/SPASMS LEADS TO A SUBSTANTIAL IMPROVEMENT IN THE QUALITY OF LIFE FOR A PATIENT
PHYSICIAN	FIRST EFFECTIVE TREATMENT TO REDUCE THE AMOUNT AND SEVERITY OF MUSCLE CONTRACTIONS/SPASMS WITHOUT TOLERABITY ISSUES
PAYER	IMPROVEMENTS IN QUALITY OF LIFE AND LEAD TO A SUBSTANTIAL REDUCTION IN OVERALL TREATMENT COSTS SUCH AS OUTPATIENT CARE OR NURSING HOMES
PARTNER	WE ASSUME ADDEX TO SEEK/RAISE FUNDS FOR PHASE IIA AND B; ON POSITIVE RESULTS WE ASSUME SAME PARTNER AS FOR PD-LID AND AT SAME TIME

REVENUE MODEL

EUROPE / REST OF WORLD	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
NUMBER OF PATIENTS (MN)	0.6	0.6	0.6	0.6	0.6	0.6	0.7	0.7	0.7	0.7	0.7
GROWTH (%)	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
PATIENTS DIAGNOSED (%)	60%	60%	60%	60%	60%	60%	60%	60%	60%	60%	60%
PATIENTS DIAGNOSED (MN)	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
PERCENTAGE TREATED (%)	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%
PATIENTS TREATED (MN)	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
PENETRATION (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	3.0%	9.0%	14.0%	18.0%	21.0%
NUMBER OF PATIENTS (MN)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
COST OF THERAPY PER YEAR (CHF)	4'380	4'380	4'380	4'380	4'380	4'380	4'380	4'380	4'380	4'380	4'380
SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**26**	**79**	**126**	**165**	**196**
CHANGE (%)								206%	59%	31%	19%
ROYALTY (%)	0.0%	0.0%	0.0%	0.0%	0.0%	25.0%	25.0%	25.0%	25.0%	25.0%	25.0%
ROYALTIES (CHF MN)	0	0	0	0	0	0	6	20	31	41	49
UPFRONT & MILESTONE PAYMENTS (CHF MN)	0	0	0	0	10	20	0	0	0	0	0
R&D COSTS (CHF MN)	0	-1	-4	-4	-1	0	0	0	0	0	0
PROFIT BEFORE TAX (CHF MN)	**0**	**-1**	**-4**	**-4**	**9**	**20**	**6**	**20**	**31**	**41**	**49**
TAX RATE (%)	0%	0%	0%	0%	0%	0%	5%	15%	20%	20%	20%
TAXES (CHF MN)	0	0	0	0	0	0	0	-3	-6	-8	-10
PROFIT (CHF MN)	**0**	**-1**	**-4**	**-4**	**9**	**20**	**6**	**17**	**25**	**33**	**39**

UNITED STATES	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
NUMBER OF PATIENTS (MN)	0.3	0.3	0.3	0.3	0.4	0.4	0.4	0.4	0.4	0.4	0.4
GROWTH (%)	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%
PATIENTS DIAGNOSED (%)	60%	60%	60%	60%	60%	60%	60%	60%	60%	60%	60%
PATIENTS DIAGNOSED (MN)	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
PERCENTAGE TREATED (%)	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%	50%
PATIENTS TREATED (MN)	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
PENETRATION (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	4.0%	10.0%	15.0%	19.0%	22.0%
NUMBER OF PATIENTS (MN)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
COST OF THERAPY PER YEAR (CHF)	7'090	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844	6'844
SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**30**	**77**	**117**	**151**	**179**
CHANGE (%)								155%	53%	29%	18%
ROYALTY (%)	0.0%	0.0%	0.0%	0.0%	0.0%	25.0%	25.0%	25.0%	25.0%	25.0%	25.0%
ROYALTIES (CHF MN)	0	0	0	0	0	0	8	19	29	38	45
PROFIT BEFORE TAX (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**8**	**19**	**29**	**38**	**45**
TAX RATE (%)	0%	0%	0%	0%	0%	0%	5%	15%	20%	20%	20%
TAXES (CHF MN)	0	0	0	0	0	0	0	-3	-6	-8	-9
PROFIT (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**7**	**16**	**23**	**30**	**36**

	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
GLOBAL SALES (CHF MN)	**0**	**0**	**0**	**0**	**0**	**0**	**56**	**156**	**243**	**316**	**375**
CHANGE (%)								179%	56%	30%	19%
GLOBAL PROFIT (CHF MN)	**0**	**-1**	**-4**	**-4**	**9**	**20**	**13**	**33**	**49**	**63**	**75**
CHANGE (%)	-100%		600%	0%	-357%	122%	-34%	149%	47%	30%	19%

ESTIMATES AS OF 4 AUGUST, 2015

SOURCE: VALUATIONLAB ESTIMATES

2) ADX71441 ($GABA_B$ PAM) - Charcot-Marie-Tooth disorder

ADX71441 ($GABA_B$ PAM) is another drug candidate stemming from Addex' proprietary allosteric modulation technology platform and is targeted for the treatment of Charcot-Marie-Tooth disorder, alcohol use disorder and nicotine addiction.

CMT one of the most common inherited neurological disorders

Charcot-Marie-Tooth (CMT) disorder is one of the most common inherited neurological disorders, affecting approximately 1 in 2,500 people with an estimated 150,000 patients in the US. The disease is named after the three physicians who first identified it in 1886: Jean-Martin Charcot, Pierre Marie and Howard Henry Tooth. The disorder is also known as hereditary motor and sensory neuropathy (HMSN) and comprises a group of disorders that affect peripheral nerves. These are nerves that lie outside the brain and spinal cord and supply the muscles and sensory organs in the limbs.

Caused by gene mutations affecting the peripheral nerve axon or myelin sheath

CMT is caused by mutations in genes that produce proteins involved in the structure and function of either the peripheral nerve axon (long thin part of a nerve cell that transmits nerve impulses) or the myelin sheath (fatty insulation layer around the axon that prevents the loss of these nerve impulses), and affect the normal function of the peripheral nerves. As a result, these nerves slowly degenerate and lose the ability to communicate with their distant targets. This leads to muscle weakness and atrophy in the extremities (arms, legs, hands, or feet). In some cases the degeneration of sensory nerves results in a reduced ability to feel heat, cold and pain. The gene mutations in CMT are usually inherited.

CMT is a disabling disease that severely affects mobility and occurs in adolescence

Onset of symptoms typically occurs in adolescence or early adulthood. The severity of symptoms varies greatly among individuals and progression of symptoms is gradual. A typical feature includes weakness of the foot and lower leg muscles, which may result in foot drop and a high-speed gait with frequent tripping and falling. Foot deformities, such as high arches and hammertoes are characteristic due to weakness of the small muscles in the feet. Pain can range from mild to severe. Some people may need to rely on foot or leg braces or other orthopedic devices to maintain mobility. In rare cases individuals may have respiratory muscle weakness. CMT is not considered a fatal disease and patients with most forms of CMT have a normal life expectancy.

No disease modifying treatment for CMT available

There is no cure for CMT, but physical therapy, occupational therapy (e.g. muscle strength training, muscle and ligament stretching, stamina training), braces and other orthopedic devices, and even orthopedic surgery can help with the disabling symptoms of the disease. Painkillers are prescribed for individuals with severe pain.

Overproduction of PMP-22 causes abnormal structure & function of myelin sheath

There are many forms of CMT disorder, including CMT 1, CMT 2, CMT 3, CMT 4, and CMT X. CMT 1A is the most common variant of the disorder that affects approximately 1 in 5,000 people. CMT 1A is caused by having an extra copy of the gene (on chromosome 17) that contains the instructions for making the protein called "peripheral myelin protein-22 (PMP-22) that leads to the production of excessive amounts of PMP-22, which is a critical component of the myelin sheath. Overexpression of this gene causes structure and

function of the myelin sheath to be abnormal. Genetic testing is available to diagnose some types of CMT. In other cases a nerve biopsy may be needed to confirm diagnosis.

$GABA_B$ involved in the regulation of the production of PMP-22

$GABA_B$ (gamma-aminobutyric acid sub-type B) has been shown to regulate the production of PMP-22 using baclofen. Baclofen, also known as chlorophenibut, is a conventional (orthosteric) stimulator (agonist) of the $GABA_B$ receptor, and is primarily used to treat spasticity and is in early research stages for treating alcoholism. A recent phase II study (double-blind, placebo-controlled, 3-arms, oral liquid formulation 2x daily for 12 months), with PXT-3003, an oral fixed low-dose formulation of baclofen, naltrexone (opioid receptor antagonist) and sorbitol (sweetener) showed positive results in 80 adult patients with CMT1A. PXT-3003 has been developed by the French private biopharmaceutical company Pharnext and is expected to enter phase III development in 2015 with a potential launch in 2017/18. In 2014 the EMA and FDA granted orphan drug designation (ODD) to PXT-3003 for the treatment of Charcot-Marie-Tooth disease type 1A (CMT 1A).

Potential to become a convenient once-daily oral therapy for Charcot-Marie-Tooth

However, baclofen has a relatively short half-life of 2-4 hours and therefore needs to be given frequently throughout the day to remain efficacious. Moreover, in some cases tolerability can occur when given over longer periods of time. Addex' ADX71441 ($GABA_B$ PAM) is a first in class, selective, oral, small molecule activator of the $GABA_B$ receptor through positive allosteric modulation (PAM), and only acts when the natural ligand (GABA) activates the receptor, therefore respecting the physiological cycle of activation. The compound has a potentially better pharmacokinetic profile than baclofen with a significantly longer half-life (predicted 24 hours), which would lead to convenient once-daily dosing. In preclinical studies, no signs of tolerance development on efficacy were seen after repeated dosing with ADX71441 ($GABA_B$ PAM), while CNS related clinical signs (e.g. dizziness) decreased and disappeared after repeated dosing.

Collaboration with CMT Association to advance ADX71441 ($GABA_B$ PAM) in CMT 1A

In October 2014 Addex entered into a collaboration with the CMTA (Charcot-Marie-Tooth Association) to profile ADX71441 ($GABA_B$ PAM) in a battery of preclinical models, including neurological and behavioral models of CMT 1A disorder and assess the potential to impede the development of motor and sensory control defects associated with normal disease progression.

Previously the compound was found to be efficacious in a genetic animal (rat) model of CMT 1A. The production of PMP-22 was down-regulated and compound muscle action potential in transgenic CMT rats was increased. Grip strength loss was prevented in CMT rats compared to wild type. Histopathology performed on CMT 1A treated rats chronically with ADX71441 ($GABA_B$ PAM) showed reduced axonal loss, demonstrating a modification of disease progression. IND (investigational new drug) enabling studies have been completed and the compound has been accepted for phase I clinical testing in the EU in 2013. Due to lack of funding, the phase I trial was not started.

Clinical trial program ADX71441 ($GABA_B$ PAM) in CMT 1A

- **Phase I trial in 2015:** Addex plans to start phase I trials in healthy volunteers: a classical single ascending dose and multiple ascending dose trial design to evaluate the safety and tolerability of ADX71441 in healthy volunteers, with some measurements of pharmacodynamic effects.

- **Phase IIa POC trial in mid 2016:** Addex plans to start a phase IIa POC trial in patients with CMT 1A. This will be a randomized, placebo-controlled, 3-arm, study in subjects with CMT 1A. Duration of treatment will depend on the ongoing discussions with KOLs. Primary endpoints are currently under discussion, but will probably include the CMTES/ONLS composite score and functional test (CMTNS) improvement.

Peak sales of CHF 500+ mn in Charcot-Marie-Tooth 1A (NOT included in valuation)
With an estimated 230,000+ CMT 1A patients in the US and Europe and an assumed annual treatment cost of CHF 7,300 per patient, and a penetration rate up to 35%, peak sales could conservatively amount to CHF 500+ mn. We currently exclude ADX71441 ($GABA_B$ PAM) forecasts for CMT 1A from our risk-adjusted NPV for Addex due to the lack of POC. Positive POC in CMT 1A disorder would lead to an increase of CHF 1.9 per share.

3) ADX71441 (GABA-B PAM) – Addiction (alcohol / nicotine)

There is increasing evidence that activating the $GABA_B$ receptor plays an important role in addiction such as cocaine, alcohol and nicotine. GABA is the major inhibitory neurotransmitter in the brain and is implicated in the modulation of central reward processes. Administration of $GABA_B$ receptor agonists or $GABA_B$ PAMs (positive allosteric modulators) decreased self-administration of various drugs of abuse.

Early evidence of conventional $GABA_B$ receptor agonists in addiction…
$GABA_B$ receptor agonists such as baclofen inhibited cue-induced reinstatement of nicotine and cocaine-seeking behavior in rodents. These animal behavioral models are used to create a disease model of drug addiction that resembles human addiction behavior. Animals are conditioned to perform one action, typically a lever press, in order to receive a drug. The effect of the treatment can be measured by the reduction of the lever press. The use of baclofen has also proven efficiency in craving, drinking and anxiety reduction, thereby promoting abstinence. Similar effects on addiction were seen in animal behavior models with other conventional $GABA_B$ receptor agonists, such as the scientific research compounds CGP44532 (reduces cocaine reinforcement, decreased nicotine self-administration in the rat), and SKF-97541 (reduces cocaine sensitization in the rat).

… as well as with $GABA_B$ PAMs
There is even growing early evidence that $GABA_B$ PAMs have an effect on addiction in animal behavior models. The scientific research compound CGP7930 demonstrated reduced self-administration of ethanol and cocaine and had anxiolytic effects in animal studies. BHF-177, a new GS39783 analogue (which is limited to research purposes only due to genotoxicity, which may cause cancer) has shown to reduce self-administration of nicotine in animal models and does not induce genotoxicity as seen with GS39783. And finally, Addex' own commercial compound ADX71441 ($GABA_B$ PAM) demonstrated early efficacy in several animal models in alcohol consumption and nicotine withdrawal.

PAMs may have an edge over conventional $GABA_B$ receptor agonists
As a result compounds that activate the GABAB receptor could provide new pharmaceutical treatment options for various addictions. Baclofen, which has been long on the market as a muscle relaxant, is now being developed for treating alcoholism using high doses. However, broad use of baclofen was limited due to its unfavorable side effect profile, which includes sedation, tolerance, cognitive disruption and withdrawal syndrome

(e.g. hallucinations, delusions, confusion, agitation and delirium), in particular in chronic use at high doses.

ADX71441 ($GABA_B$ PAM), being a positive allosteric modulator, appears to have an important edge over baclofen with a differentiated tolerability and pharmacokinetic profile. No sign of tolerance development on efficacy has been seen after repeated dosing while initial central nervous system related side effects diminish and disappear after repeated dosing. Furthermore, the long half-life of ADX71441 ($GABA_B$ PAM) bodes for convenient once daily dosing, increasing patient compliance.

Healthcare agencies worldwide actively spur research in addiction

Currently, there are no effective and well-tolerated prescription drugs for alcoholism or nicotine addiction. The World Health Organization estimates that about 140 million people throughout the world suffer from alcohol dependence. This leads to a lot of harm to the individuals, their families and society. Excessive alcohol consumption damages almost every organ in the body and the cumulative toxic effects can cause both medical (cirrhosis of the liver, pancreatitis, heart disease, peptic ulcers, sexual dysfunction) and psychiatric (epilepsy, dementia, psychosis, anxiety & depression) problems. Excessive alcohol consumption is estimated to cost USD 223 bn per year in the US alone.

Smoking is the leading preventable cause of death in many countries. It is estimated that each year one in every five deaths in the US is the result of smoking. Economically, more than USD 96 bn of total US healthcare costs each year are attributable directly to smoking. Nicotine is the primary reinforcing component of tobacco that leads to addiction. Cigarette smoking harms nearly every organ in the body, and can lead to lung, mouth, throat, esophagus, stomach, pancreas, cervix, kidney and bladder cancer, as well as acute myeloid leukemia, and chronic obstructive pulmonary disease (COPD).

An effective and well-tolerated therapy that addresses alcohol or nicotine addiction addresses a significant unmet medical need. No wonder that many healthcare agencies across the world stimulate research in this area, through partnerships, providing scientific and research resources and financial grants.

Two partnerships with leading organizations of the NIH

Addex has established important partnerships with two organizations of the US NIH (National Institutes of Health) to explore the use ADX71441 ($GABA_B$ PAM) in addiction and drug abuse.

1. **NIDA collaboration for nicotine and cocaine addiction**
   At the end 2013 Addex announced a partnership with the NIDA (National Institute on Drug Abuse) to evaluate the pharmacology of ADX71441 ($GABA_B$ PAM) and ADX88178, an mGlu4 PAM in preclinical models of drug abuse and addiction. The collaboration will evaluate Addex drug candidates, ADX71441 ($GABA_B$ PAM) and ADX88178 in a battery of preclinical models to study their potential as treatments for nicotine and cocaine addiction.

2. **NIAAA collaboration for alcohol use disorder**
   In January 2015 Addex entered a collaboration with the NIAAA (National Institute on Alcohol Abuse and Alcoholism to evaluate the pharmacology of ADX71441 ($GABA_B$ PAM), in preclinical models of alcohol use disorder. The collaboration will

evaluate ADX71441 ($GABA_B$ PAM) in a battery of preclinical models of alcohol use disorder.

Both collaborations should help Addex define the best clinical development path for ADX71441 ($GABA_B$ PAM). The next step is to complete phase I in early 2016 and then start phase IIa POC in an addiction population with the US NIH (NIDA or NIAAA) and/or pharma partner in 2016. Important to note is that Addex will not start an addiction development program without significant non-dilutive funding sources and third party expertise due to the complexity of clinical development in this area.

Peak sales of CHF 500+ mn in addiction (NOT included in valuation)
Needless to say, an effective and well-tolerated drug for alcohol abuse or nicotine addiction addresses a blockbuster market potential. Pfizer's smoking cessation drug Chantix (varenicline) generated USD 846 mn in 2008, only two years after launch. A black box warning for the risk of suicide in 2009 led to a decline in sales. Nevertheless, the drug continues to generate sales of more than USD 500 mn per year. There is an even larger need for effective prescription drugs for treating alcoholism. Sales of leading drugs such as Merck KGaA's Campral (acamprosate) disappointed, not topping annual sales of USD 100 mn. In practice patient response rates are relatively modest and not always consistent, while tolerability issues have hampered broad uptake. Therefore, we believe peak sales for ADX71441 ($GABA_B$ PAM) could easily amount to CHF 500+ mn, if developed successfully in just one of the targeted indications. Given the early stage of development, the lack of proof-of-concept in humans, and uncertainty which indication Addex will pursue, we have excluded any sales forecasts for ADX71441 ($GABA_B$ PAM) in our valuation.

Income Statement

ADDEX THERAPEUTICS

SHARE PRICE (CHF) 3.00

INCOME STATEMENT (CHF MN)	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
PRODUCT SALES (INCL. PARTNER SALES)	**0**	**0**	**0**	**0**	**0**	**0**	**143**	**487**	**1'103**	**1'792**	**2'646**
CHANGE (%)								241%	126%	62%	48%
ROYALTIES	**0**	**0**	**0**	**0**	**0**	**0**	**36**	**101**	**208**	**351**	**562**
CHANGE (%)								184%	105%	69%	60%
UPFRONT & MILESTONE PAYMENTS	**0**	**0**	**0**	**30**	**15**	**20**	**0**	**50**	**90**	**110**	**70**
CHANGE (%)					-50%	33%	-100%		80%	22%	-36%
OTHER REVENUES	**0.73**	**0.50**	**0.10**	**0.10**	**0.10**	**0.10**	**0.10**	**0.10**	**0.10**	**0.10**	**0.10**
CHANGE (%)	411%	-31%	-80%	0%	0%	0%	0%	0%	0%	0%	0%
REVENUES (EXCL. PARTNER SALES)	**0.73**	**0.50**	**0.10**	**30.10**	**15.10**	**20.10**	**35.78**	**151.40**	**298.10**	**460.78**	**631.88**
CHANGE (%)	411%	-31%	-80%	30000%	-50%	33%	78%	323%	97%	55%	37%
COGS	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
CHANGE (%)											
GROSS PROFIT	**0.73**	**0.50**	**0.10**	**30.10**	**15.10**	**20.10**	**35.78**	**151.40**	**298.10**	**460.78**	**631.88**
CHANGE (%)	411%	-31%	-80%	30000%	-50%	33%	78%	323%	97%	55%	37%
MARGIN (%)	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
R&D	**-0.93**	**-2.00**	**-11.50**	**-11.50**	**-17.50**	**-15.00**	**-11.50**	**-2.50**	**-0.50**	**-0.50**	**-0.50**
CHANGE (%)	-90%	115%	475%	0%	52%	-14%	-23%	-78%	-80%	0%	0%
S,G&A	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**	**-1.59**
CHANGE (%)	-70%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
OTHER OPERATING INCOME/(EXPENSE)	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
OPERATING COSTS	**-2.52**	**-3.59**	**-13.09**	**-13.09**	**-19.09**	**-16.59**	**-13.09**	**-4.09**	**-2.09**	**-2.09**	**-2.09**
CHANGE (%)	-83%	42%	264%	0%	46%	-13%	-21%	-69%	-49%	0%	0%
EBIT (BEFORE EXCEPTIONALS)	**-1.80**	**-3.09**	**-12.99**	**17.01**	**-3.99**	**3.51**	**22.68**	**147.31**	**296.01**	**458.68**	**629.78**
CHANGE (%)	-88%	72%	320%	-231%	-123%	-188%	547%	549%	101%	55%	37%
MARGIN (%)	-247%	-619%	-12994%	56%	-26%	17%	63%	97%	99%	100%	100%
EBIT	**-1.80**	**-3.09**	**-12.99**	**17.01**	**-3.99**	**3.51**	**22.68**	**147.31**	**296.01**	**458.68**	**629.78**
CHANGE (%)	-88%	72%	320%	-231%	-123%	-188%	547%	549%	101%	55%	37%
MARGIN (%)											
EBITDA	**-1.69**	**-2.98**	**-12.87**	**17.16**	**-3.80**	**3.78**	**23.07**	**147.85**	**296.77**	**459.75**	**631.28**
CHANGE (%)	-88%	77%	332%	-233%	-122%	-200%	510%	541%	101%	55%	37%
MARGIN (%)	-232%	-596%	-12867%	57%	-25%	19%	64%	98%	100%	100%	100%
D&A	**0.11**	**0.12**	**0.13**	**0.15**	**0.20**	**0.28**	**0.39**	**0.54**	**0.76**	**1.07**	**1.49**
NET FINANCIAL INCOME/(EXPENSES)	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**	**0.02**
PROFIT/LOSS BEFORE TAXES	**-1.77**	**-3.07**	**-12.97**	**17.03**	**-3.97**	**3.53**	**22.71**	**147.33**	**296.03**	**458.70**	**629.81**
CHANGE (%)	-88%	73%	322%	-231%	-123%	-189%	544%	549%	101%	55%	37%
MARGIN (%)	-244%	-614%	-12972%	57%	-26%	18%	63%	97%	99%	100%	100%
TAXES	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**-1.78**	**-19.70**	**-59.60**	**-95.25**	**-131.10**
TAX RATE (%)	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	7.9%	13.4%	20.1%	20.8%	20.8%
NET PROFIT/LOSS	**-1.77**	**-3.07**	**-12.97**	**17.03**	**-3.97**	**3.53**	**20.92**	**127.63**	**236.43**	**363.45**	**498.70**
CHANGE (%)	-88%	73%	322%	-231%	-123%	-189%	493%	510%	85%	54%	37%
MARGIN (%)	NM	-614%	-12972%	57%	-26%	18%	58%	84%	79%	79%	79%
NET PROFIT/LOSS (EXCLUDING MILESTONES)	**-1.77**	**-3.07**	**-12.97**	**-12.97**	**-18.97**	**-16.47**	**20.92**	**77.63**	**146.43**	**253.45**	**428.70**
MARGIN (%)					-126%	-82%	58%	51%	49%	55%	68%
EPS (CHF)	**-0.18**	**-0.26**	**-0.82**	**1.08**	**-0.25**	**0.22**	**1.32**	**8.08**	**14.97**	**23.01**	**31.57**
CHANGE (%)	-89%	48%	213%	-231%	-123%	-189%	493%	510%	85%	54%	37%

ESTIMATES AS OF 4 AUGUST, 2015

SOURCE: VALUATIONLAB ESTIMATES

NOTE: At the end of FY 2014 Addex Therapeutics had a total of CHF 190 mn unrecorded tax loss carried forward. Due to the uncertainties as to whether Addex Therapeutics can use these, we have excluded them from our forecasts.

Ratios & Balance Sheet

ADDEX THERAPEUTICS

SHARE PRICE (CHF) 3.00

RATIOS	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
P/E		-11.4x	-3.7x	2.8x	-11.9x	13.4x	2.3x	0.4x	0.2x	0.1x	0.1x
P/S		70.2x	473.8x	1.6x	3.1x	2.4x	1.3x	0.3x	0.2x	0.1x	0.1x
P/NAV		13.1x	4.8x	1.8x	2.0x	1.8x	1.0x	0.3x	0.1x	0.1x	0.0x
EV/EBITDA		-10.4x	-2.4x	1.8x	-8.2x	8.2x	1.3x	0.2x	0.1x	0.1x	0.0x

PER SHARE DATA (CHF)	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
EARNINGS	**-0.18**	**-0.26**	**-0.82**	**1.08**	**-0.25**	**0.22**	**1.32**	**8.08**	**14.97**	**23.01**	**31.57**
CHANGE (%)	-89%	48%	213%	-231%	-123%	-189%	493%	510%	85%	54%	37%
CASH	**0.20**	**0.20**	**0.60**	**1.69**	**1.45**	**1.69**	**3.04**	**11.15**	**26.17**	**49.25**	**80.92**
CHANGE (%)	-38%	0%	202%	181%	-14%	17%	80%	267%	135%	88%	64%
DIVIDENDS	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
PAYOUT RATIO (%)	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
NET ASSET VALUE	**0.23**	**0.23**	**0.62**	**1.71**	**1.47**	**1.71**	**3.06**	**11.18**	**26.19**	**49.27**	**80.94**
CHANGE (%)	-30%	-2%	171%	175%	-14%	16%	79%	265%	134%	88%	64%

BALANCE SHEET (CHF MN)	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
NET LIQUID FUNDS	**2.0**	**2.3**	**9.5**	**26.7**	**22.9**	**26.7**	**48.0**	**176.2**	**413.4**	**777.9**	**1'278.1**
TOTAL ASSETS	**4.0**	**4.3**	**11.5**	**28.7**	**24.9**	**28.7**	**50.0**	**178.2**	**415.4**	**779.9**	**1'280.1**
TOTAL SHAREHOLDERS' EQUITY	**2.3**	**2.7**	**9.8**	**27.0**	**23.3**	**27.1**	**48.4**	**176.5**	**413.7**	**778.3**	**1'278.5**
CHANGE (%)	-22%	15%	266%	175%	-14%	16%	79%	265%	134%	88%	64%
RETURN ON EQUITY (%)	-76%	-114%	-132%	63%	-17%	13%	43%	72%	57%	47%	39%
TOTAL EQUITY	**2.3**	**2.7**	**9.8**	**27.0**	**23.3**	**27.1**	**48.4**	**176.5**	**413.7**	**778.3**	**1'278.5**
FINANCIAL DEBT	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
EMPLOYEES	7	7	7	7	7	7	7	7	7	7	7
CHANGE (%)	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%

CASH FLOW STATEMENT (CHF MN)	2014	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E
NET PROFIT / (LOSS)	-1.8	-3.1	-13.0	17.0	-4.0	3.5	20.9	127.6	236.4	363.5	498.7
DEPRECIATION & AMORTIZATION	0.1	0.1	0.1	0.2	0.2	0.3	0.4	0.5	0.8	1.1	1.5
OTHER NON-CASH ITEMS	-0.1	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
NET CASH USED IN OPERATING ACTIVITIES	**-1.8**	**-2.5**	**-12.8**	**17.2**	**-3.8**	**3.8**	**21.3**	**128.2**	**237.2**	**364.5**	**500.2**
CASH FLOW FROM INVESTING ACTIVITIES	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
FREE CASH FLOW	**-1.4**	**-2.5**	**-12.8**	**17.2**	**-3.8**	**3.8**	**21.3**	**128.2**	**237.2**	**364.5**	**500.2**
CASH FLOW FROM FINANCING ACTIVITIES	0.5	2.8	20.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
CHANGE IN LIQUID FUNDS	**-1.0**	**0.3**	**7.2**	**17.2**	**-3.8**	**3.8**	**21.3**	**128.2**	**237.2**	**364.5**	**500.2**

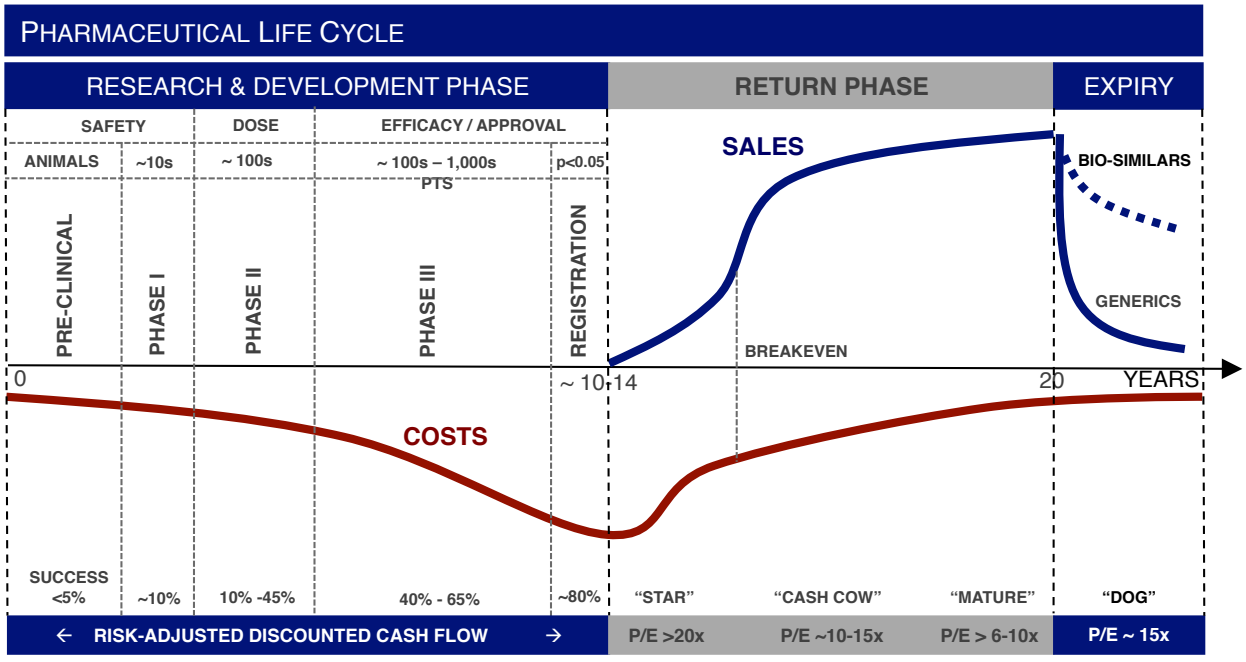
ESTIMATES AS OF 4 AUGUST, 2015

SOURCE: VALUATIONLAB ESTIMATES

APPENDIX

Pharmaceutical life cycle

To determine the value of a prescription (bio)pharmaceutical compound, it is critical to understand its life cycle. Fortunately, all compounds follow the same life cycle. The clock starts ticking after the compound is patented, providing 20 years of protection from generic competition. Market exclusivities can extend this protection period. The average Research & Development Phase takes 10-14 years, leading to an effective Return Phase of 6-10 years. The Development Phase has 3 distinct Phases, focused on safety (Phase I), dose (Phase II) and efficacy/clinical benefit (Phase III). The compound is filed for registration/approval at the FDA (US) or EMA (EU). The Return Phase is characterized by a star, cash cow, and mature phase. After patent expiry (or loss of market exclusivity) generic manufacturers may copycat the branded prescription drug, at significantly lower costs, leading to a sales and earnings implosion of the branded drug.



SOURCE: VALUATIONLAB

Success probabilities & Royalties

In our risk-adjusted NPV calculations, we use standardized success probabilities based on historical clinical success rates. The success rate increases as the project progresses through development. Sales and earnings forecasts are based on the clinical and competitive profile of the compound. The more advanced the compound is, the more accurate the forecasts become as the target market can be defined. We conservatively exclude projects that lack Phase IIa proof-of-concept data in our valuations.

SUCCESS PROBABILITIES & ROYALTIES

DEVELOPMENT STAGE	AIM	WHAT / WHO	SUCCESS PROBABILITY (%)	COSTS (USD MN)	ROYALTIES (%)
PRE-CLINICAL	SAFETY & PHARMACOLOGY DATA	LAB TESTS / ANIMALS - NO HUMANS!	< 5	3	
PHASE I	SCREENING FOR SAFETY	HEALTHY VOLUNTEERS (10'S)	5-10	3	< 5
PHASE IIA	PROOF-OF-CONCEPT	PATIENTS WITH DISEASE (10'S)	10-15		
PHASE II	ESTABLISH THE TESTING PROTOCOL	PATIENTS WITH DISEASE (100'S)	10-35	5	5-15
PHASE IIB	OPTIMAL DOSAGE	PATIENTS WITH DISEASE (100'S)	20-45	5-10	
PHASE III	EVALUATE OVERALL BENEFIT/RISK	PATIENTS WITH DISEASE (1,000'S)	40-65	> 20-1,000	10-25
REGULATORY FILING	DETERMINE PHYSICIAN LABELING	CLINICAL BENEFIT ASSESSMENT	80-90		
APPROVAL	MARKETING AUTHORIZATION	PHYSICIANS FREE TO PRESCRIBE	100		15-30

SOURCE: VALUATIONLAB, TUFTS, FDA, EMA, CLINICALTRIALS.GOV

Important Research Disclosures

valuationLAB AG is an independent life science research boutique with no securities or banking services. The company does not hold any positions in the securities mentioned in this report.

Our financial analyses are based on the "Directives on the Independence of Financial Research" issued by the Swiss Bankers Association in January 2008.

Purpose of the Research

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Risk Qualification

Speculative	less than 1 year cash and breakeven beyond 1 year
High Risk	less than 2 years cash and sales from 2 or less marketed products
Medium Risk	more than 3 years cash and sales from at least 2 marketed products (patent expiry > 5 years)
Low Risk	self-sustaining cash flows, sales from > 3 marketed products (patent expiry > 5 years)

Analyst Certification

The research analyst(s) identified on the first page of this research report hereby attest that all of the views expressed in this report accurately reflect their personal views about any and all of the subject securities or issuers. In order to ensure the independence of our research analysts, and their immediate household, are expressly prohibited from owning any securities in the valuationLAB AG research universe, which belong to their sector(s). Neither the research analyst nor his/her immediate household serves as an Officer, Director or Advisory Board Member of **Addex Therapeutics Ltd.**

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