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Innovative Treatments for Central Nervous System Disorders

June 2019

Allosteric modulators for human health

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Addex Overview

Dipraglurant for dyskinesia in Parkinson's disease	➢ Pivotal registration program on track to start in Q4 2019 ➢ US PD-LID market estimated at $4.2B ➢ FDA Orphan Drug Designation granted in PD-LID
Validating partnerships with industry	➢ Indivior partnership - $330m in milestones, tiered royalties up to double digit & funded research program ➢ J&J deal - €109m in milestones & low double digit royalties
World leading technology platform	➢ "Allosteric modulators" are a validated & differentiated pharmacological approach to address drug targets ➢ Proprietary biological screening assays and chemical library
Deep pipeline of first / best in class programs	➢ Creating future partnership opportunities ➢ Driving long term growth
Strong balance sheet	➢ 32.8M shares traded on the SIX Swiss Stock Exchange – ADXN ➢ Cash of CHF41.7m at 31 December 2018 ➢ Runway through 2021

Experienced Leadership Team

Tim Dyer
CEO / CFO

Co-Founder of Addex
Formerly with PwC
UK Chartered Accountant

Dr Roger Mills
Chief Medical Officer

Developed Nuplazid in PD Pyschosis
30 years in Pharma industry including Pfizer, Gilead and Acadia Pharmaceuticals

Dr Robert Lutjens
Head of Discovery Biology

Member of Addex founding team
Formerly with Glaxo & Scripps Research Institute

Dr Jean-Philippe Rocher
Head of Discovery Chemistry

Member of Addex founding team
Formerly with Pierre Fabre, GSK and Mitsubishi

Experienced Board of Directors

Vincent Lawton
Chairman

Former European Head of Merck & Co.
Former MHRA Board member

Ray Hill
Board member

Former Executive Director Merck & Co.

Jake Nunn
Board member

Partner New Enterprise Associates

Isaac Manke
Board member

Partner New Leaf Venture Partners

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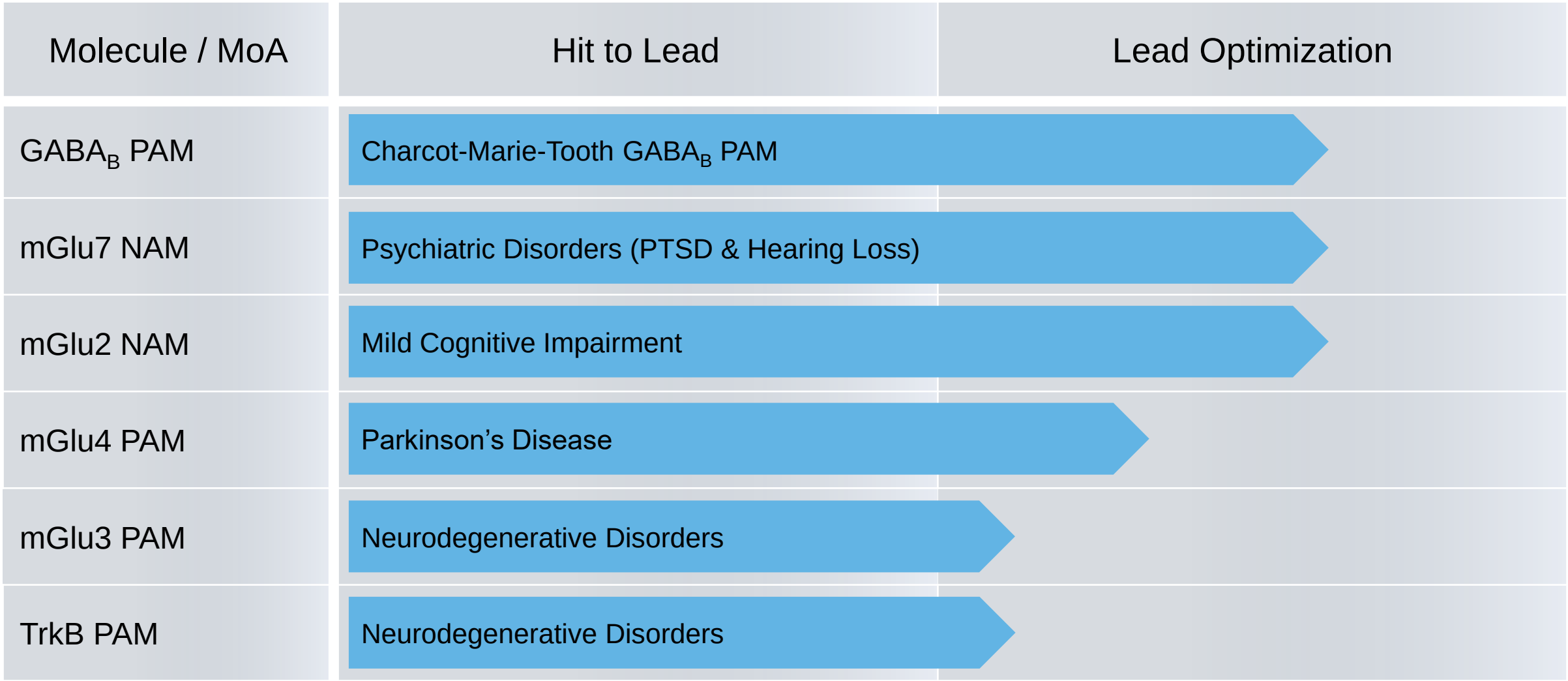
Pipeline with Lead Program Entering US Pivotal Study

Molecule / MoA	Preclinical	Phase 1	Phase 2	Phase 3
Dipraglurant-IR *(mGlu5 NAM)*	Parkinson's disease levodopa-induced dyskinesia			
Dipraglurant-ER *(mGlu5 NAM)*	Focal cervical dystonia			
ADX71149 *(mGlu2 PAM)*	Epilepsy		janssen	
$GABA_B$ PAM	Addiction	INDIVIOR		

NAM = Negative Allosteric Modulator
PAM = Positive Allosteric Modulator

Multiple orphan drug opportunities

Extensive Discovery Stage Pipeline Driving Long-Term Growth



NAM = Negative Allosteric Modulator
PAM = Positive Allosteric Modulator

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Dipraglurant in Parkinson's Disease

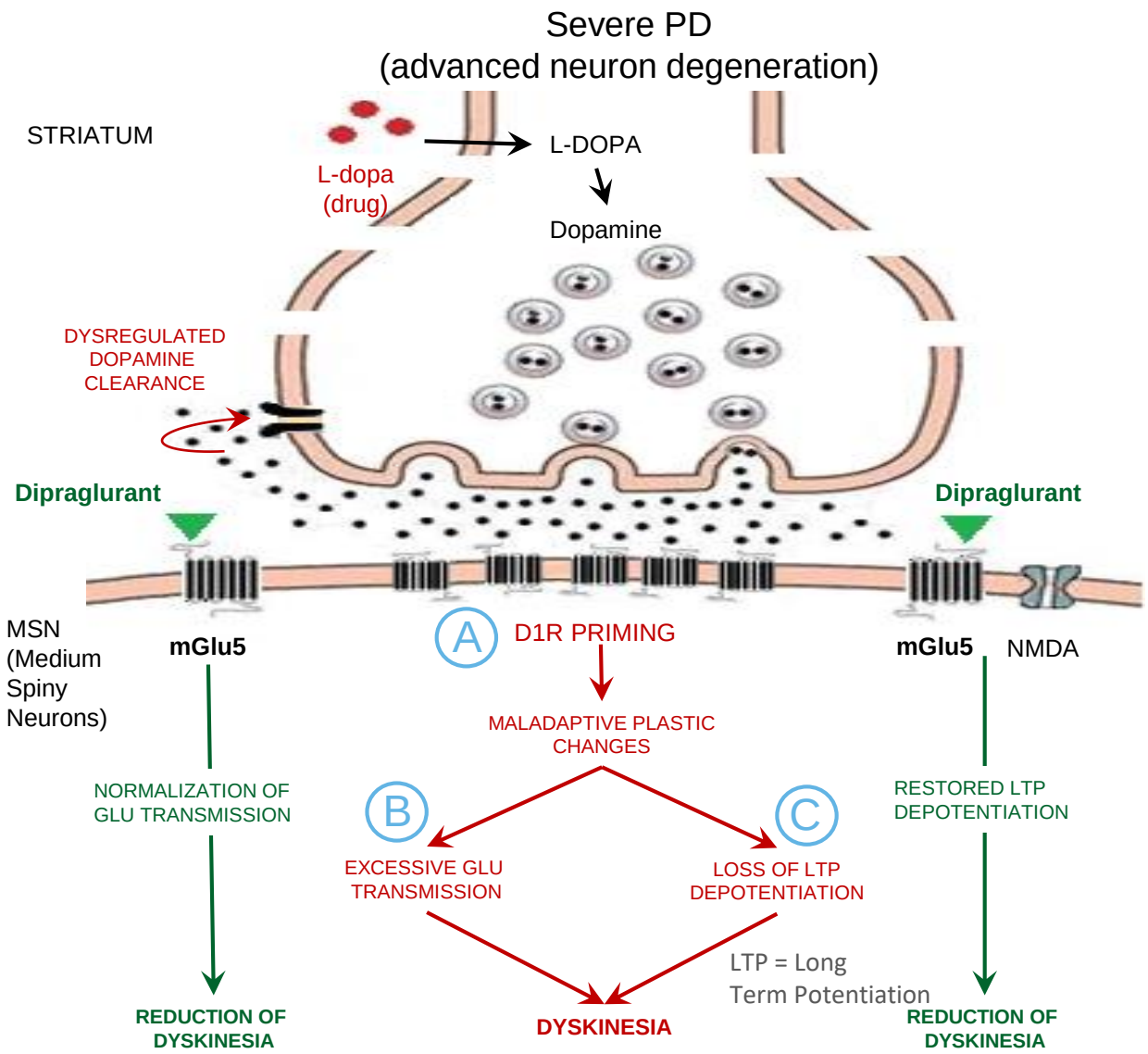
The Dipraglurant Opportunity in Dyskinesia Associated with PD

Clear Development & Regulatory Path	➢ Pivotal studies on track to start in Q4 2019 ➢ Manufacturing and planning ongoing ➢ Precedented FDA regulatory path
Unmet Need and Significant Commercial Opportunity in PD-LID	➢ >1M Parkinson's disease patients in US of which >170,000 have dyskinesia ➢ US LID market estimated at $4.2B ➢ Dipraglurant US peak sales estimated at $1.4B ➢ Pricing of PD therapeutics – Nuplazid at $30K p.a. and Gocovri at $28.5K p.a.
Dipraglurant: Unique Mechanism of Action	➢ First-in-class, selective, oral small molecule mGluR5 NAM ➢ PK profile mirrors that of L-dopa, making it ideal to treat LID ➢ Normalizes abnormal glutamate stimulation during L-dopa dosing
Strong IP Position	➢ Composition of matter through June 2025 & strong polymorph patent through 2034 without extensions ➢ US FDA orphan drug designation in PD-LID

Levodopa-Induced Dyskinesia in Parkinson's Disease (PD-LID)

Long-term L-dopa use is invariably associated with the development of dyskinesias	➢ Dopamine replacement does not lead to dyskinesia per se, but lowers the triggering threshold for symptoms ➢ Dyskinesias result from the neurodegenerative process that underlies PD ➢ LID can become as disabling as the PD symptoms themselves
LID symptoms: irregular migrating uncontrollable contractions or twisting and writhing due to dystonia, chorea, and choreoathetosis	➢ This can be painful, lead to weight loss, fatigue and exhaustion, with increased risk for falls and injuries ➢ Leading to withdrawal from social interaction, isolation, frustration, depression, reduced quality of life and increased burden on caregiver
Prevalence of LID is related to disease duration	➢ Within 4-6 years of L-dopa treatment, LID is experienced by >40% of patients ➢ By 9 -15 years of L-dopa treatment, LID affects 90% of PD patients ➢ Next-generation L-dopa will not negate LID
Over time PD drugs become less effective, exacerbated by the emergence of LID	**The doctor is faced with a balancing act where drug and dosing regimens must be continually optimized in order to ensure adequate symptom control while minimizing intolerable side effects**

Dipraglurant - Overview & Mechanism of Action



- Loss of substantia nigra neurons combined with the non-physiological, pulsatile stimulation of dopamine receptors are at the basis of LID development
- In the striatum, **LID is the result of**:
  - A *D1 receptor priming*
  - B *Abnormal glutamate transmission*
  - C *Loss of LTP depotentiation*
- Metabotropic glutamate receptors are attractive drug targets due to their **modulatory** action to normalize glutamatergic activity and restoration of LTP depotentiation
- **mGlu5 receptors are implicated in the control of glutamate transmission**
- Preclinical and clinical data show that mGluR5 blockade controls dyskinesia
- **Dipraglurant is an oral small molecule active as a highly selective negative allosteric modulator at the mGlu5 receptor with the potential to treat LID**

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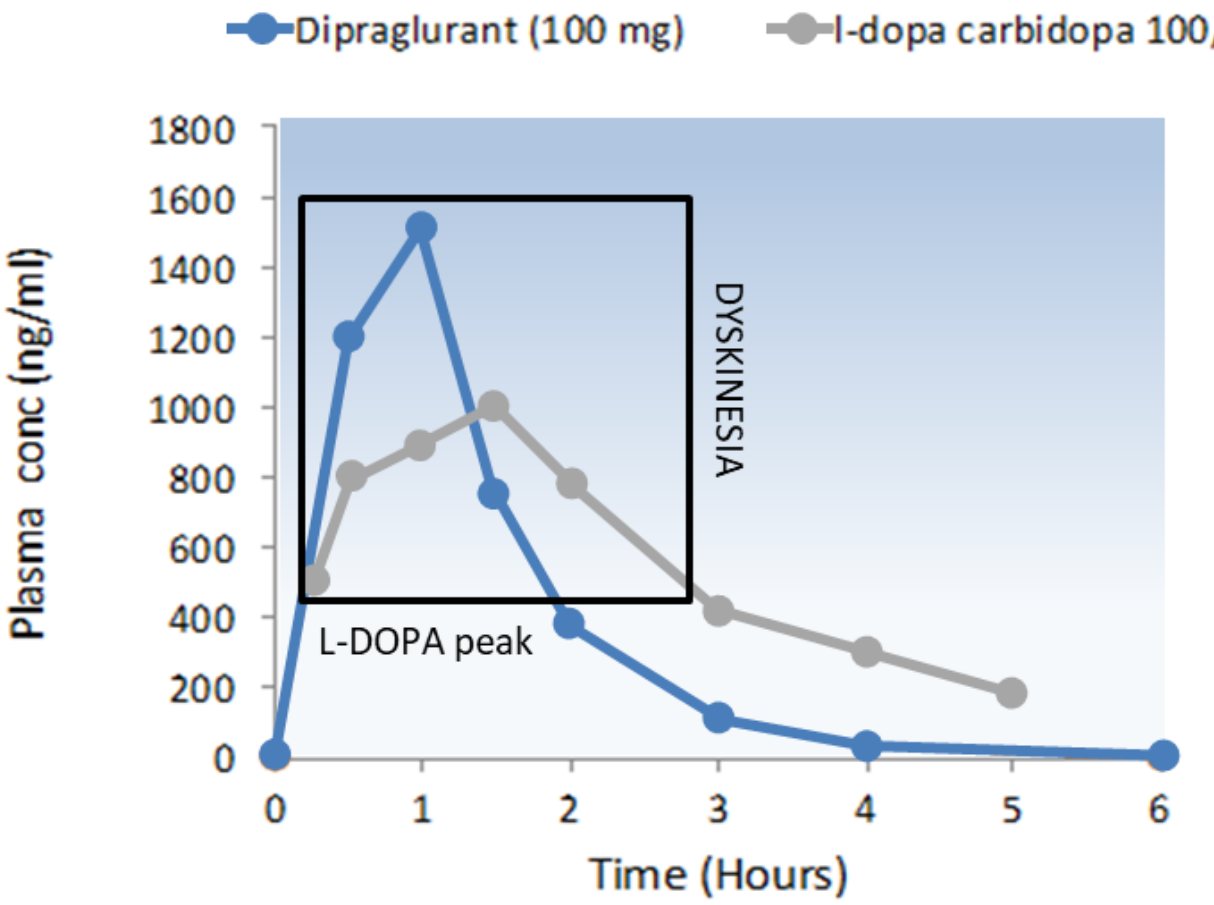
Dipraglurant PK is a Key Advantage for Treating LID



- Dyskinesia symptoms are correlated to peak levels of levodopa therapy
- PK profile of dipraglurant mirrors that of levodopa
- **Dipraglurant inhibits abnormal glutamate stimulation during peak levodopa dose but releases the receptor during normal glutamate activity**

Dipraglurant PK/PD profile is ideal for treating LID

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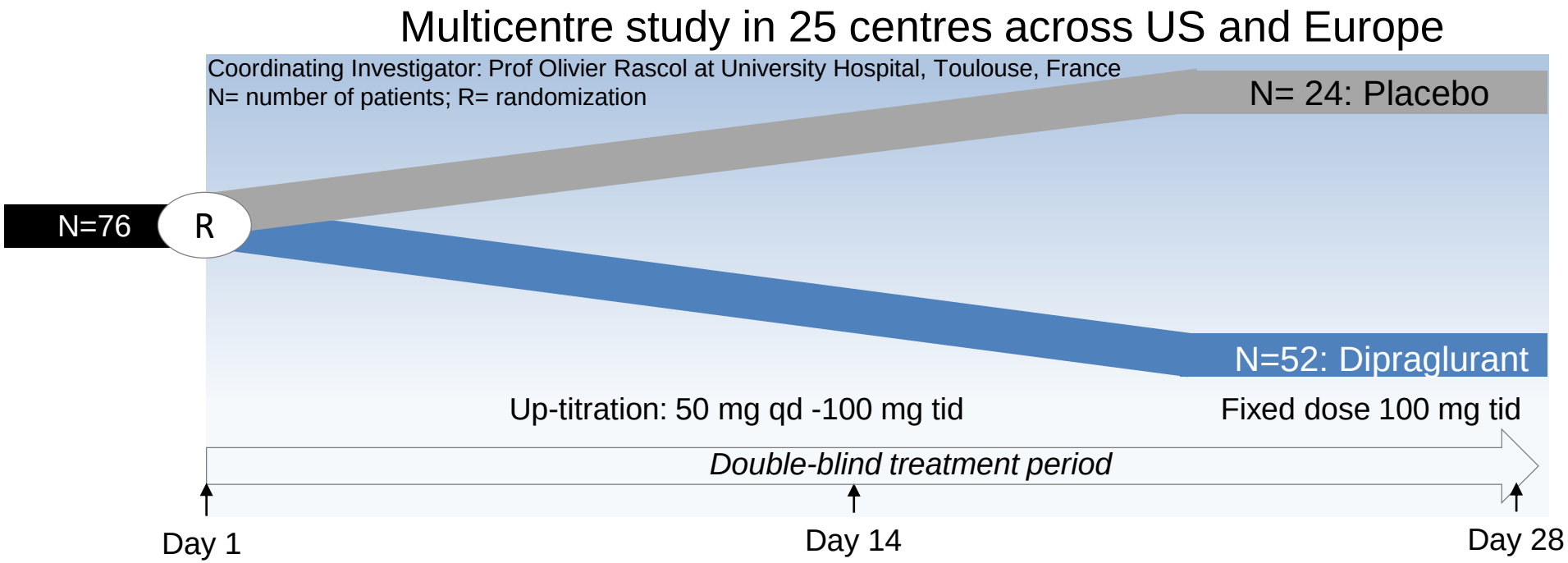
Dipraglurant EU and US Phase 2a Study in LID



Days		1-3	4-7	8-13	14-16	17-21	22-28
Dose/mg	AM			50	50	50	100
	Noon	50	50	50	100	100	100
	PM		50	50	50	100	100
	Daily	50	100	150	200	250	300

- **Primary objective**: safety & tolerability
- **Secondary objective**: exploratory efficacy:
  - Modified Abnormal Involuntary Movement Scale (mAIMS) on Day 1, 14 and 28
  - Unified Parkinson's Disease Rating Scale (UPDRS)
  - Clinician and Patient Global Impression of Change (CGIC & PGIC)
  - Pharmacokinetics (PK)
  - Patient diaries of ON & OFF time

Measuring acute effect of mid-day dose on days 1, 14 and 28

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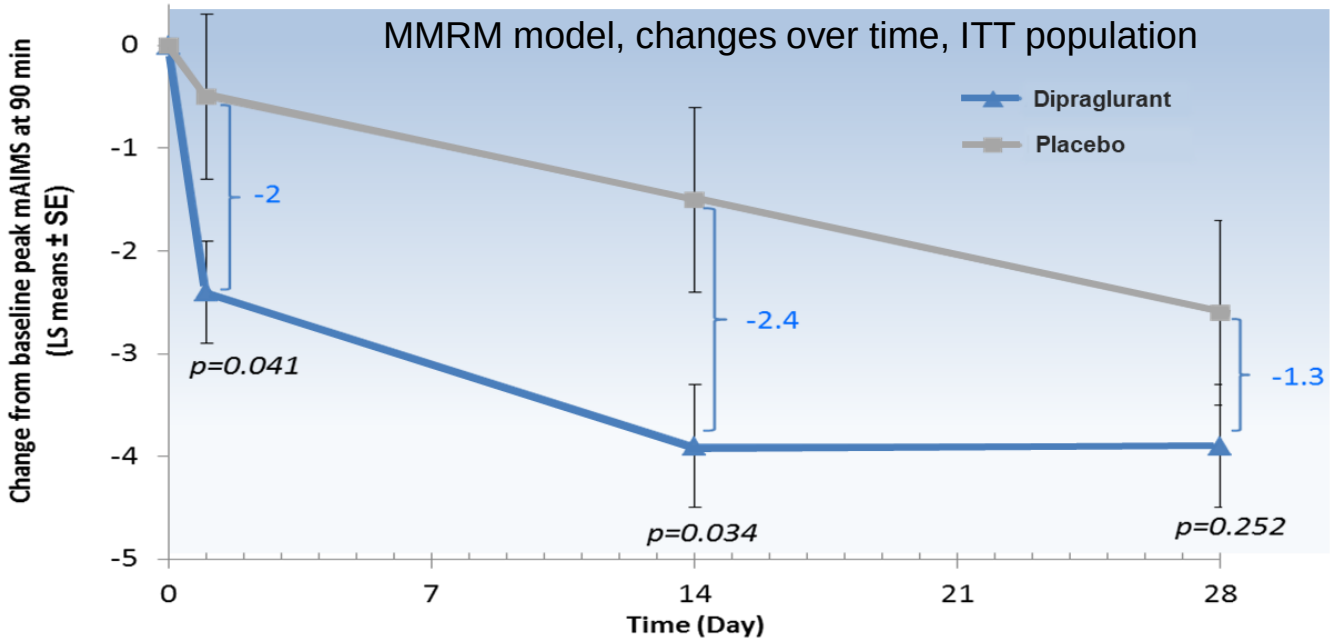
Dipraglurant Reduces LID Severity by 30%

Change from baseline in peak mAIMS at 90 min



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

- Dipraglurant had a statistically significant effect on the first day
- Dipraglurant reduced dyskinesia compared to placebo at all visits over the 28 days
- Placebo response confounded significance at day 28
- Dose titration contributed to placebo response (patients only on full dosage for last 7 days)
- No placebo-mitigating techniques deployed in study:
  - No centralized raters
  - No independent raters
  - Rater not blinded to visit number
  - Patients were more moderate than severe

Clear dose response but need to manage placebo

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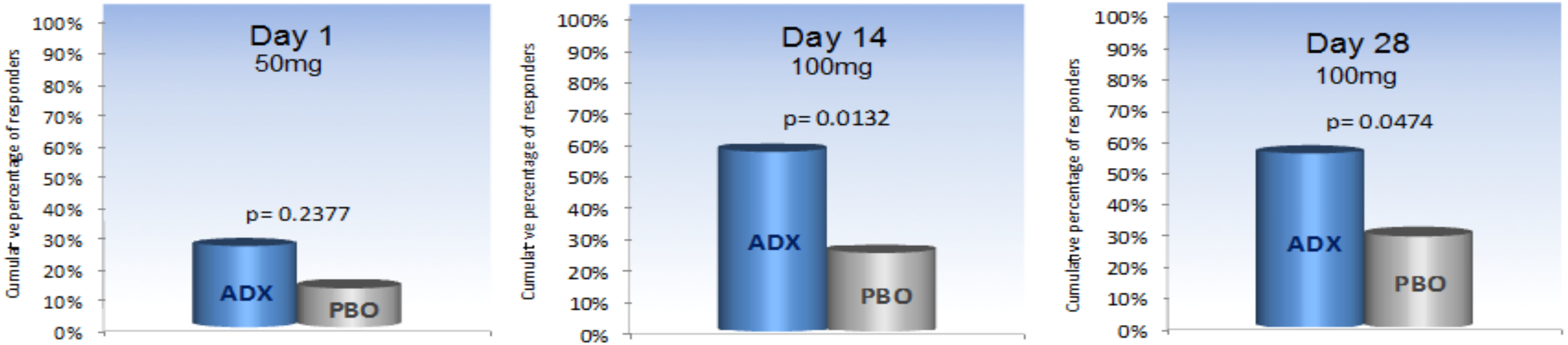
Responder Analysis Demonstrates Dipraglurant Significant Benefit

Cumulative % of Patients Showing ≥ 30% Change of Peak mAIMS from Baseline



Responder analysis (≥30% change of peak mAIMS from baseline)					
Midday dose	Dipraglurant		Placebo		p-value
Day 1 (50 mg)	n=13	26.0%	n=3	12.5%	0.2377
Day 14 (100 mg)	n=29	56.9%	n=6	25.0%	0.0132
Day 28 (100 mg)	n=27	55.3%	n=7	29.2%	0.0474

- A 30% reduction in mAIMS is clinically meaningful
  - One patient was able to hold & read a newspaper for the first time in years
  - Another patient had improved speech and became more easily intelligible

Responder analysis reinforces robustness of dipraglurant anti dyskinetic effect

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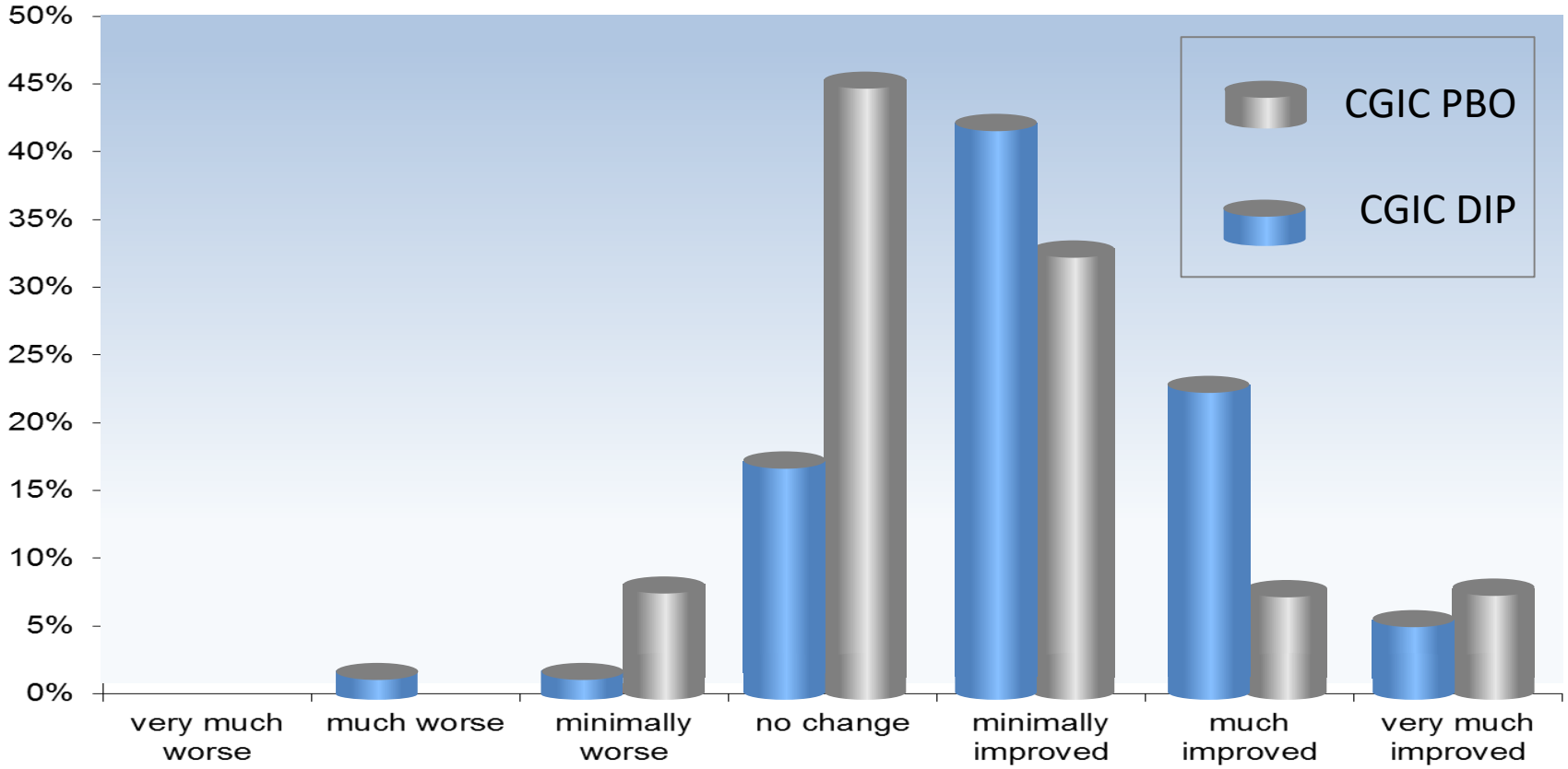
Clinician Rated Global Impression of Change - Dyskinesia



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

- Relatively simple scale that reflects everyday clinical practice
- Assessment by treating physician and thus is a more objective assessment than the more subjective mAIMS
- Assessment performed at end of study compared to baseline
- **Greater improvement in dyskinesia with dipraglurant according to clinicians (p<0.05)**

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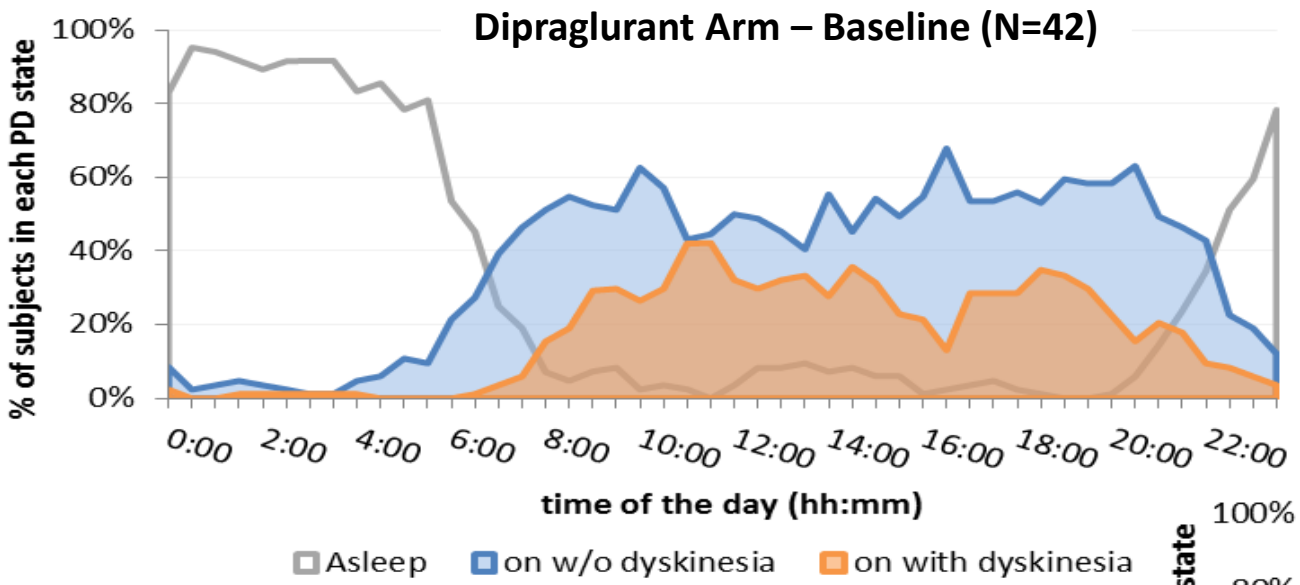
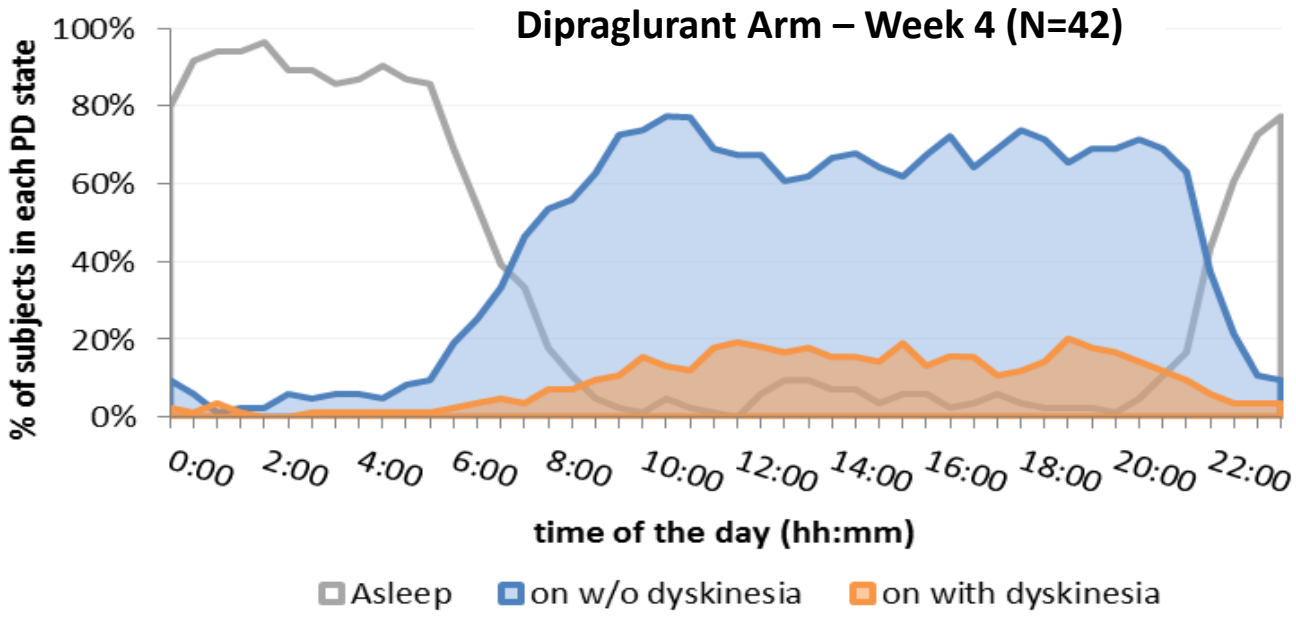
Patient Diaries – Improvement Throughout the Waking Day





After 4-week treatment with dipraglurant:

- ON time with dyskinesia **reduced** during the day
- ON time without dyskinesia **increased** and maintained during the day

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Dipraglurant 50 and 100 mg Doses Demonstrated Safety and Satisfactory Tolerability in PD Patients

- Adverse events were common in both treatment groups (dipraglurant 88.5%, pbo 75%)
- The majority of patients completed the dose escalation regimen
- Most common AEs:

	Dipraglurant	Placebo
Worsening Dyskinesia	21% (**15.3%***)	12.5%
Dizziness	19%	12.5%
Nausea	19%	0%
Fatigue	15%	4%

* 3 of the 11 patients who reported “worsening dyskinesia” did so only in the follow up period (i.e. when not taking the drug). Thus the dyskinesia recurred only after therapy had stopped. Therefore the adjusted AE% is 15.3% for dipraglurant arm vs.12.5% for placebo arm.

- AEs caused discontinuation in 2 patients taking dipraglurant 100 mg
- AEs at the 50 mg dose level (wk 1 and 2) were less frequent – 53% vs 58% pbo than at the 100 mg dose level (wk 3 and 4) – 73% vs 63% pbo
- No treatment effects on any safety monitoring variables (ECG, HR, BP, haematology and biochemistry)

Safety profile suitable for continued development in PD (KOLs and DSMB)

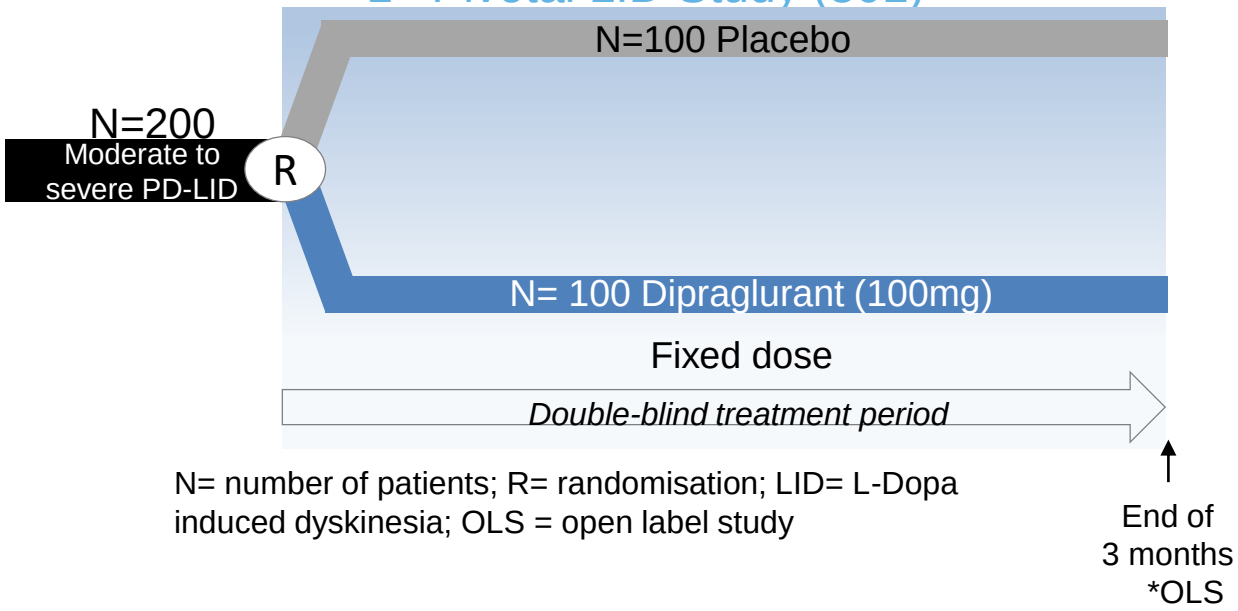
Phase 3 Trial on track to start Q4 2019

- The Phase 3 trial is fully funded and expected to report out in Q3 2021
- Considering Fast-Track and/or Breakthrough Therapy applications after first pivotal study readout
- Primary endpoint: UDysRS – more sensitive to treatment effect than mAIMS and less prone to placebo response (Goetz 2013)
- Implementing measures to manage placebo response is a priority:

Objective	Strategy
Minimize rater variability (across and within sites)	➢ Use independent (centralized) raters
Reduce expectancy bias	➢ Raters blinded to visit and do not rate the same patient at baseline and study endpoint
Exclude patients with minimal symptoms (as more likely to respond to placebo)	➢ Ensure that symptom score reflects moderate to severe symptoms that warrant therapy ➢ Ensure occur frequently enough for scale sensitivity
Exclude potential investigator rating inflation	➢ Independent oversight of screening and use of centralized rater baseline visit score as study entry gate
Draw placebo response ahead of randomization	➢ Consider non-pharmacologic intervention during screening period
Ensure no geographic bias	➢ Only include countries / sites where centralized rating is feasible

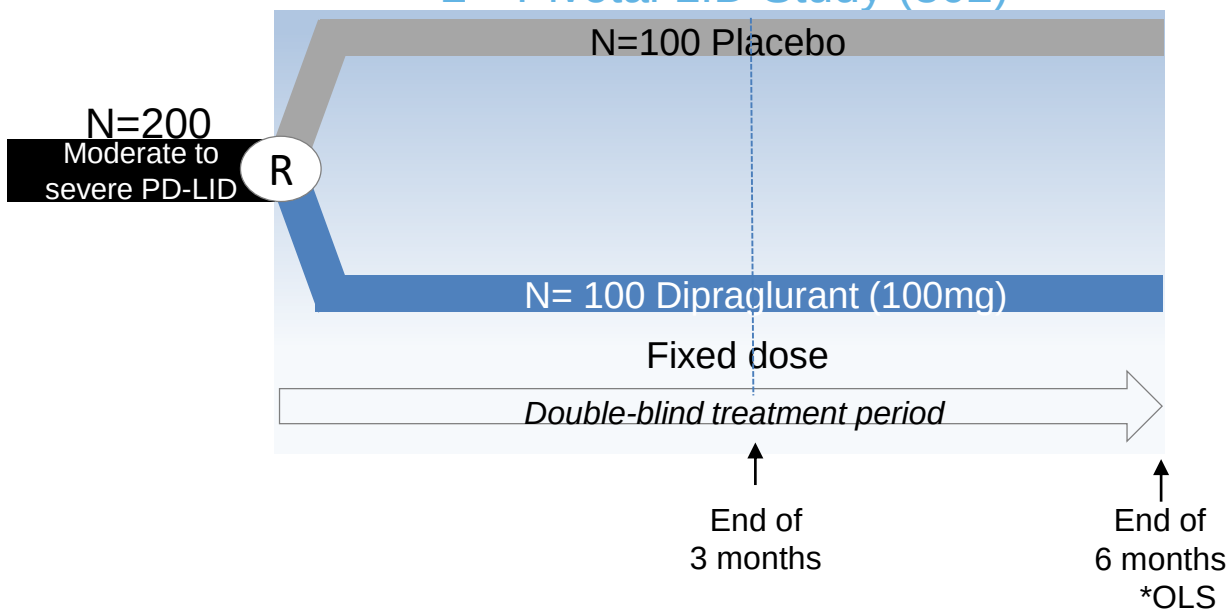
Dipraglurant pivotal registration studies

1st Pivotal LID Study (301)



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

2nd Pivotal LID Study (302)



The Primary and Secondary endpoints for both trials are the same:

- **Primary objective**
  efficacy in reducing LID
  ✓Change over time in UDysRS (wk13 from baseline)

- **Secondary objectives**
  (Safety & tolerability, additional efficacy parameters, PK)
  ✓Change over time in MDS-UPDRS Part III
  ✓Patient diaries, on & off time
  ✓CGI-S
  ✓Pharmacokinetics (PK)
  ✓Safety and tolerability

Dipraglurant LID Opportunity

LID has a large unmet need and market opportunity	➢ > 170K LID patients in US ➢ ~$1.4bn US market opportunity for dipraglurant

Limited competition – only one FDA approved medicine	➢ Gocovri (reformulation of generic amantadine): Approved on 24th August 17 – safety profile similar to generic
	➢ Dipraglurant 1st in class highly selective oral monotherapy –
	  ➢ Improved safety profile
	  ➢ Ideal PK profile mirrors levodopa – recognized by KOLs as key advantage

Clear development plan with precedented regulatory path	➢ Precedented regulatory path paved by Gocovri (Adamas) ➢ Two registration trials (301 and 302) with Open Label extension ➢ UDysRS – more sensitive to treatment effect than mAIMS and less prone to placebo response (Goetz 2013) ➢ Implementing measures to manage placebo response is a priority
Strong patent and market exclusivity	➢ NCE and polymorph patent provide protection through 2034 without extensions and data exclusivity ➢ Orphan Drug Designation – 7 years of market exclusivity

Promising Preclinical Pipeline

$GABA_B$ PAM Program for Charcot-Marie-Tooth 1A Neuropathy

- Rationale :
  - $GABA_B$ is the metabotropic receptor for GABA, main inhibitory neurotransmitter
  - Activation of $GABA_B$ is validated through the use of baclofen (GABAB orthosteric agonist)
    - Clinical efficacy in Charcot-Marie-Tooth type 1A (CMT1A)
  - Positive Allosteric Modulator (PAM) is a differentiated pharmacological approach
    - Preclinical validation of efficacy of ADX71441 in rodent model of CMT1A
    - Potential safety and efficacy advantages – lack of tolerance and less side effects
- Status:
  - Lead optimization ongoing – progressing towards IND-enabling studies
  - Novel chemical series with potent, selective, orally bioavailable and brain penetrant compounds
  - Significant novel IP in the field

mGlu7 PAM Program for PTSD & Hearing Loss

- Rationale :
  - Based on mGlu7 receptor knockout mice phenotype & mGlu7 receptor pharmacological inhibition studies, negative allosteric modulators show strong potential for anxiety related disorders such as PTSD
  - In addition, other potential indications include pain, neuroprotection, schizophrenia, ADHD, age-related hearing impairment, tinnitus…
- Status:
  - Late lead optimization – progressing towards IND-enabling studies
  - Two main chemical series with potent, selective, orally bioavailable and brain penetrant compounds
  - Preclinical proof-of-concept efficacy in rodent PTSD model; inner ear studies ongoing
  - Significant novel IP in the field

mGlu2 NAM Program for Mild Cognitive Impairment

- Rationale :
  - Potential to treat medical conditions linked to lowered glutamate levels in the brain via restoration of a normalized glutamatergic tone
    - Alzheimer's Disease (AD) : cognitive deficits and possible disease-modifying effect (slowing disease progression via reduction of build-up of amyloid β induced by mGluR2 activation (Kim et al) and reducing chromogranin A-induced microglial neurotoxicity (Taylor et al)
    - Major Depressive Disorder: ketamine-like antidepressant efficacy in depressed patients whilst avoiding the NMDA associated negative effects of ketamine.
- Status:
  - Late lead optimization – 12 month program leading to IND-enabling studies
  - Several novel chemical series with potent, selective, orally bioavailable and brain penetrant compounds
  - Significant novel IP in the field

mGlu4 PAM Program for Parkinson's Disease

- Rationale :
  - Novel non-dopaminergic approach to treat Parkinson's disease with potential to treat both motor and non-motor symptoms, disease modifying neuroprotective potential:
    - Motor functions: in acute and chronic preclinical models of PD, mGluR4 PAM demonstrated improvement
    - Neuroprotection: effect in MPTP mice model demonstrated with mGluR4 PAM
    - Non-motor (anxiety): mGluR4 activators demonstrated anxiolytic-like response in preclinical rodent models
  - Potential to treat a broad range of debilitating autoimmune disorders linked to aberrant TH17 responses, including rheumatoid arthritis, psoriasis, inflammatory bowel disease and uveitis
  - Raised interest in field following recent acquisition of Prexton Therapeutics by Lundbeck
- Status:
  - Late lead optimization – progressing towards IND-enabling studies
  - Several novel chemical series with potent, selective, orally bioavailable and brain penetrant compounds with significant novel IP potential

mGlu3 PAM Program for Neurodegenerative Diseases

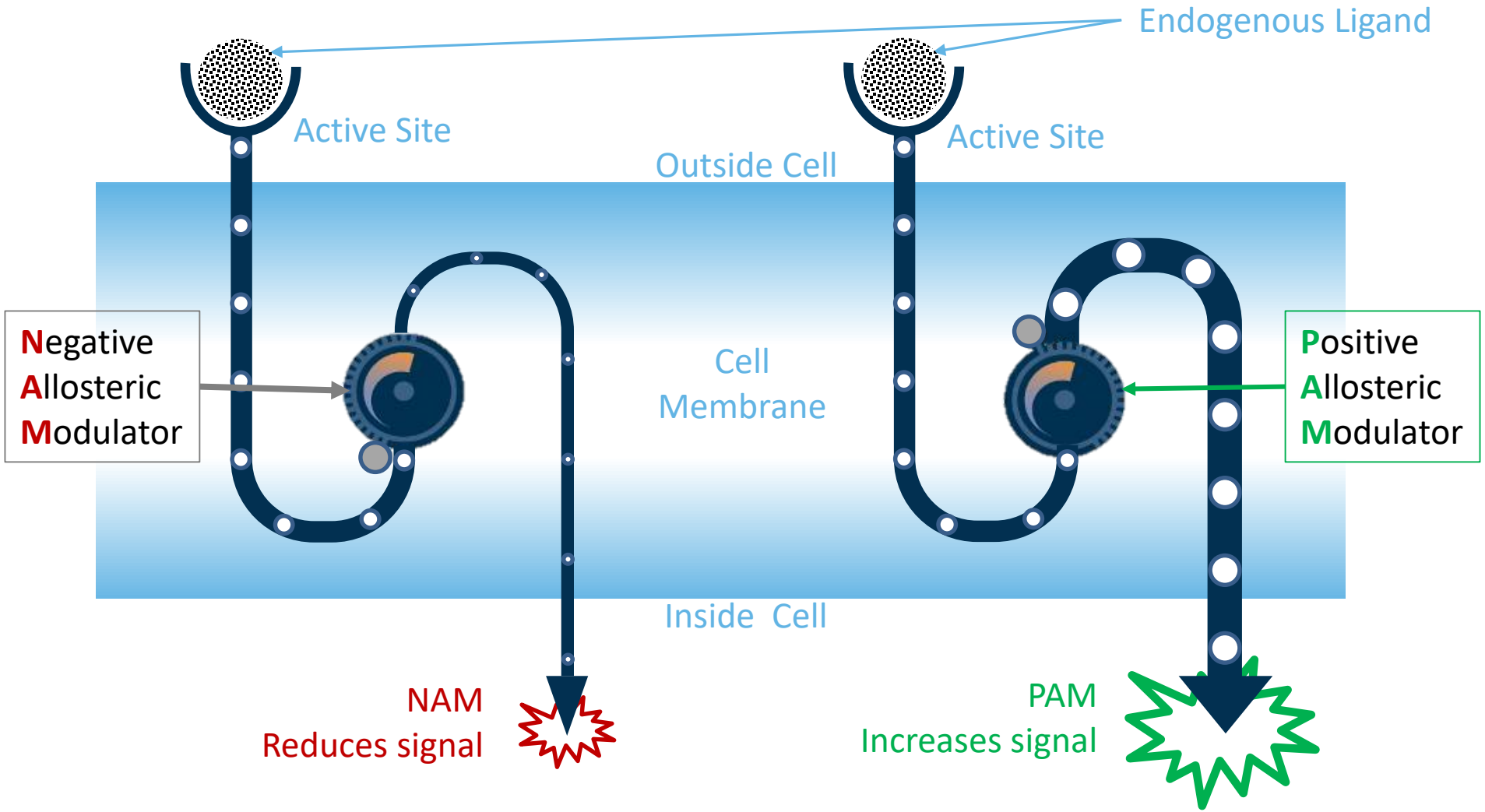
- Rationale :
  - Novel mechanism of action for treatment of neurodegenerative disorders & schizophrenia
  - Strong rational and preclinical validation through:
    - Schizophrenia: studies demonstrate GRM3 gene variants association; mGlu3 receptor KO mice exhibit a “schizophrenia-like” phenotype
    - Neurodegenerative diseases: demonstrated neuroprotective effects, with “dual” action: reduction of glutamate release, potentially lowering excitotoxic insult; and stimulation of production of growth factors such as GDNF & TGFβ1
- Status:
  - Lead generation stage
  - Several novel chemical series identified from internal screening of corporate library with proprietary screening tools – two series prioritized
  - Most potent and selective mGlu3 PAMs ever described – first in class potential
  - Significant novel IP in the field

TrkB PAM Program for Neurodegenerative Diseases

- Overview :
  - TrkB is the receptor for BDNF (brain-derived neurotrophic factor)
  - TrkB activation is preclinically validated
    - neurodegenerative diseases - Alzheimer's, Parkinson's, or Huntington's disease
    - potential for both disease-modifying and symptomatic treatment
  - PAM approach enabled identification of novel chemistry
- Status:
  - Lead generation stage
  - Supported with grants from Michael J Fox Foundation and InnoSuisse
  - Collaboration with University of Geneva access to complex *in vitro*, *ex vivo* and *in vivo* models to probe neuroprotective and neurogenesis potential of TrkB PAMs

Allosteric Modulation Becoming an Exciting Space

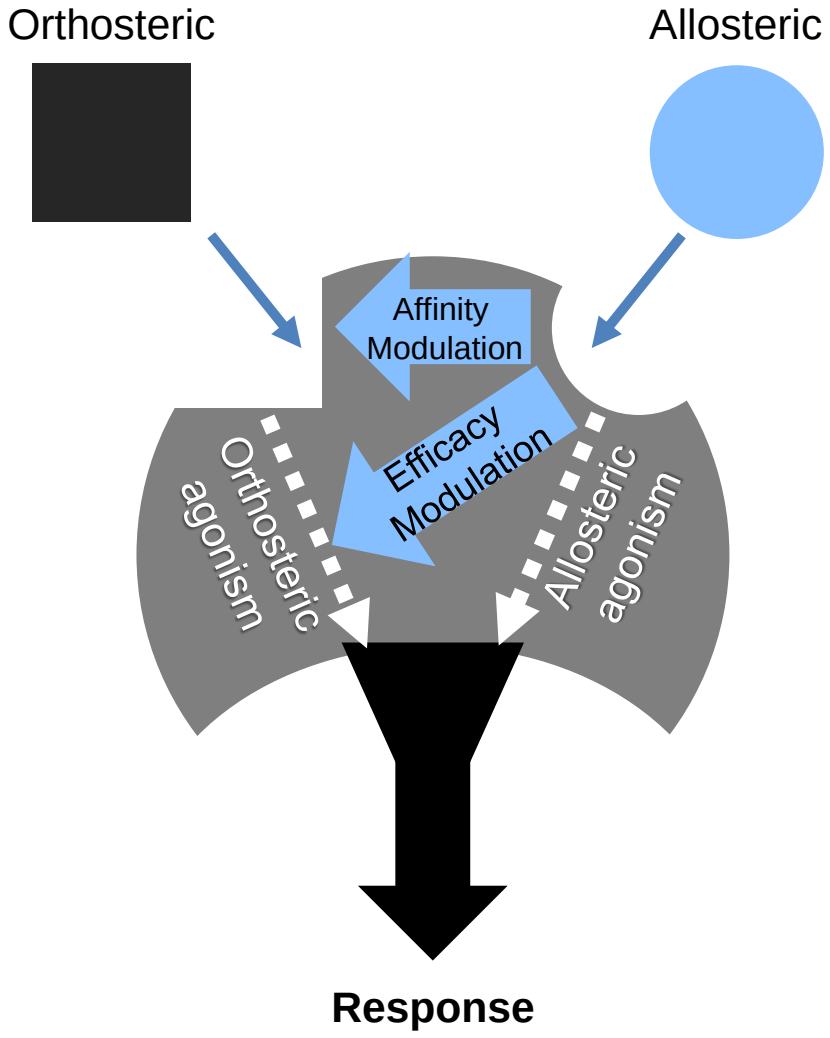
What are Allosteric Modulators?



Addex is based on a world leading technology platform

Allosteric Compounds Provide Unique Opportunity

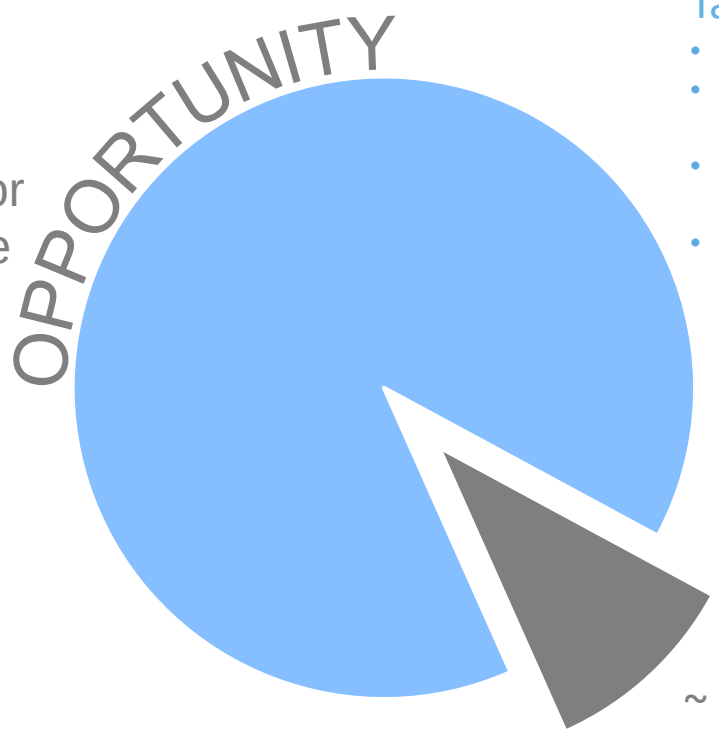
- Allosteric drugs bind to a position away from the orthosteric (primary) binding site of a target protein
- Allosteric sites can be targeted with great selectivity by allosteric modulators
- When bound by an allosteric drug, the target protein subtly changes in shape either having a positive or negative effect on the biochemical activity induced by the natural ligand
- In addition, an allosteric drug may change the affinity of the target protein for its natural ligand, either increasing or decreasing it. This induces an up/down regulation of the signalling
- This is particularly useful in neurological disorders where fully blocking the target site may be undesirable. Importantly, allosteric drugs are more modulatory, respecting the natural rhythm of receptor activation
- Allosteric modulators therefore have the potential to unlock undruggable targets



Source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2907734/

Allosteric Modulators Could Unlock Multiple “Undruggable” Targets

- Hitting previously undruggable targets is an increasingly exciting space with the vast majority of well understood drug targets currently being undruggable
- There is an opportunity for allosteric compounds to find hits for well-validated targets which have no approved drugs because traditional orthosteric approaches have failed to deliver
- Allosteric sites are largely unexplored for drug discovery although it is an increasingly hot area
- There are a number of proprietary technologies that Addex has developed to identify new allosteric approaches in addition to many years of “know-how” held by the employees of the company
- Allosteric approaches are also interesting as the IP landscape is less crowded so there may be greater freedom to operate.



~ 3000 Undrugged Targets

- Peptides/Biologics
- Nucleic acid based therapeutics
- Gene therapy/vaccines
- **Allosteric modulators**

~ 350 Drugged Targets

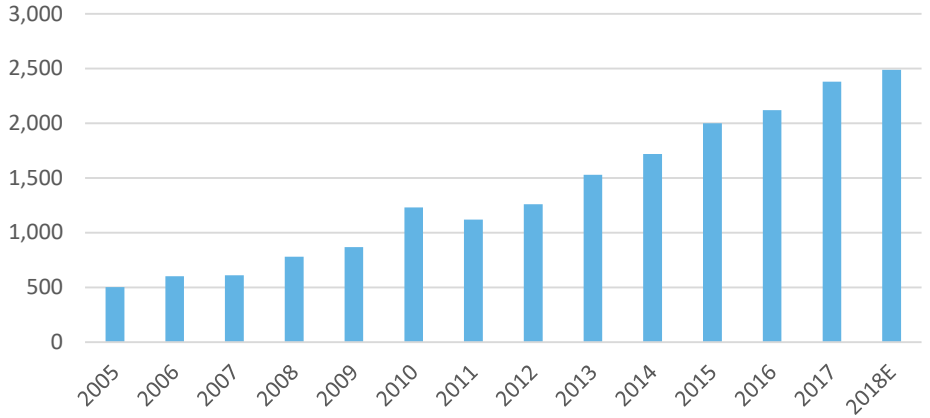
- Small molecules
- Peptides/Biologics
- **Allosteric modulators**

Allosteric Approach Gathering Momentum

- Academic research into allosteric receptor modulators is increasing – as shown by chart on right

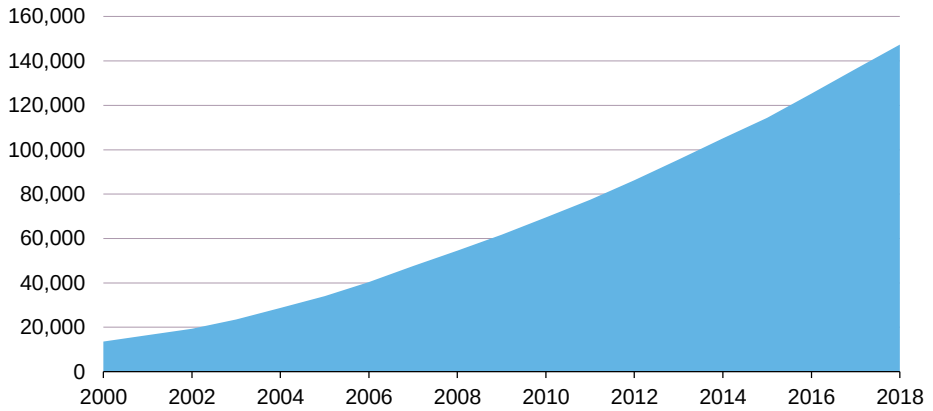
- Ongoing research into identifying structural information about alternative binding sites in proteins are a boon for Addex
  - Addex is well positioned to benefit from the explosion in knowledge in this field and in biology more broadly. As we understand more about underlying cellular biology, the number of potential allosteric targets should increase rapidly
  - Some scientists are taking a lead from nature – which commonly uses allosteric sites for enzyme regulation – as one can be sure such sites are important (e.g. Hotspot therapeutics)
  - Relay therapeutics has developed a system to model protein movement over time to uncover potential new binding sites that would not be visible in static X-ray crystallography

Number of papers published that reference allosteric modulators, by year



Source: Google Scholar search for "allosteric modulator"

Number of Protein Structures Deposited in Protein Data Bank



Source: Protein Data Bank

Allosteric Approach Gathering Momentum

Multiple examples of allosteric modulators opening up new treatment possibilities

- **Novartis' ABL001** is an allosteric modulator, and demonstrated strong anti-tumour responses as monotherapy in heavily pre-treated patients with Chronic Myeloid Leukemia (including those resistant to Glivec/Tasigna)

- **Gilead's** allosteric candidate **GS-0976** (acquired as lead asset from its acquisition of Nimbus Apollo for $400m upfront and $800m of milestones) recorded a **positive P2 readout in NASH** in October 2017

- **KRAS** - a molecular on/off switch implicated in up to 30% of cancers, but there is no obvious blocking site: this **"undruggable" target naturally lends itself towards an allosteric approach**. In March 2018, a joint research team from Wellspring and J&J reported the first small molecule KRas inhibitor that worked in animal models. It targeted a mutated cysteine in an allosteric pocket of KRas.

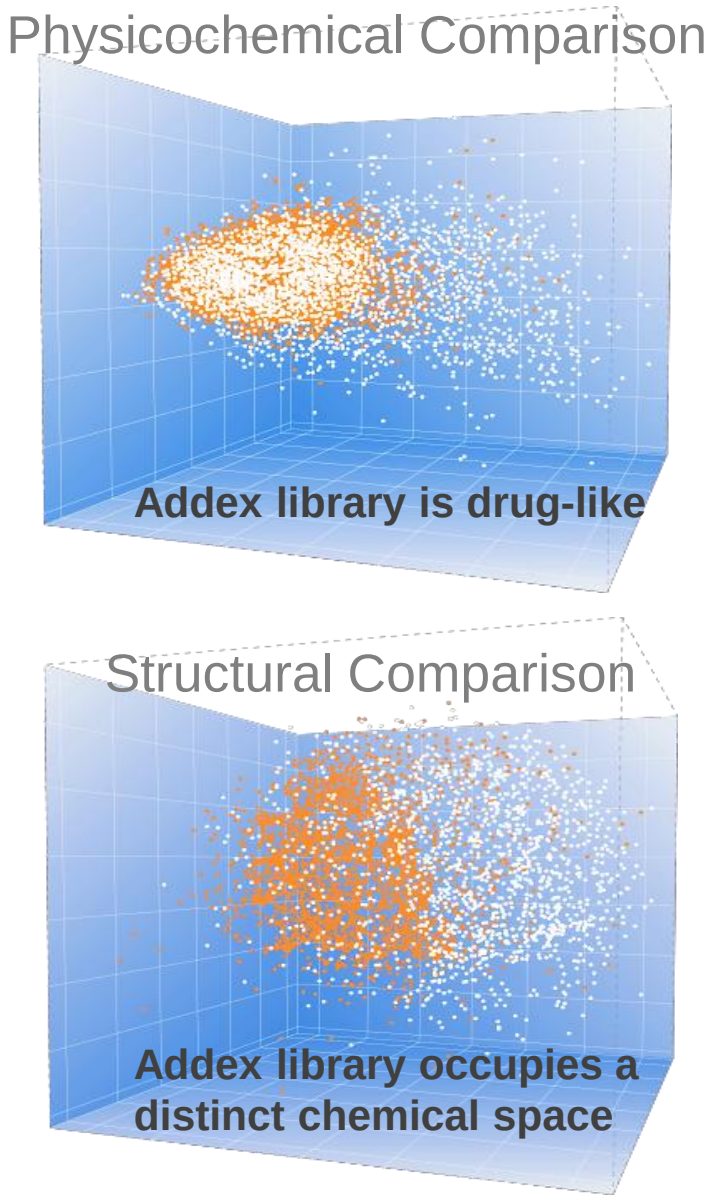
Allosteric Space Heating Up

- **HotSpot Therapeutics** was co-founded by Nimbus employees who developed allosteric modulator GS-0976, which was acquired by Gilead for $1.2bn as part of Nimbus Apollo
  - HotSpot looks for natural regulatory sites on proteins, on the basis that evolution has perfected these sites as regulatory switches over millions of years – they claim to have identified over 100 sites to date
  - They completed a $45m series A financing in July 2017, but have yet to IPO
- **Revolution Medicines** try to discover new oncology drugs targeting currently undrugged proteins
  - They specialise looking at atypical binding sites, including (but not exclusively) allosteric sites; they are yet to IPO
- **Cadent Therapeutics** recently raised $40m & signed a deal with Novartis for their NMDA NAM in P2
- In addition, several Big Pharma companies have developed or in-licensed allosteric drugs:

Company	Allosteric Drug	Target	Dev Phase	Primary Indication
Roche	RG7816	$GABA_A$ alpha5	Ph1-2	Autism Spectrum Disorder
Sanofi	RMC-4630	SHP2	Ph1-2	Solid tumours
Astellas	ASP-4345	D1R	Ph2	Cognitive Impairment in SCZ
Eli Lilly	LY3154207	D1R	Ph2	Dementia in PD
AstraZeneca	Selumetinib	MEK1/2	Ph2	Liver cancer

What Differentiates Addex's Allosteric Platform?

- Addex library of ~80k compounds is drug-like, but occupies distinct chemical space (see charts right)
- Fast-hit library is used to validate the initial hit – increasing quality of candidates and saving on cost
- Addex hit confirmation rate of 70-95% vs industry average of 10-30%
- Over 150 proprietary biological assays
- Non-calcium proximal assays are more sensitive and have greater fidelity – leading to fewer false positives and negatives
- Proven track record, with a Phase 3 candidate and collaborations with major Pharma companies



Addex Platform Already Validated – Indivior Partnership Case Study

- **The alliance with Indivior** focusing on discovery of $GABA_B$ Positive Allosteric Modulators (PAMs) for addiction
- $GABA_B$ is the metabotropic receptor for GABA, main inhibitory neurotransmitter
- Activation of $GABA_B$ is validated through the use of baclofen ($GABA_B$ orthosteric agonist)
  - Approved for the treatment of spasticity
  - Clinical efficacy in alcoholism and Charcot-Marie-Tooth type 1A (CMT1A)
- Positive Allosteric Modulator (PAM) is a differentiated pharmacological approach
  - Potential safety and efficacy advantages – lack of tolerance and less side effects
- Worldwide license and collaboration on $GABA_B$ PAM
- Addex leading funded research effort to deliver drug candidates for preclinical / clinical development
  - Addex has right to select back up compounds at clinical candidate stage for development in retained indications including CMT1A neuropathy
- Financial terms:
  - Upfront of USD 5 million & USD 4 million research funding over 2 years
  - USD 330 million of development, regulatory and commercial milestones
  - Tiered royalties up to double-digit royalties

Financials

Financials and Stock

- Cash runway through 2021
  - Cash of CHF41.7M at 31 December 2018
  - Fully funded through dipraglurant study 301 readout
- Traded on SIX Swiss Exchange: ADXN (ISIN:CH0029850754)
- 32,848,635 shares outstanding 44.6M (fully diluted)
  - New Enterprise Associated – 13.91%
  - New Leaf Venture Partners – 4.86%
  - CAM Capital – 4.86%
  - Credit Suisse Asset Management – 4.87%
  - Management & board holds - 16% (fully diluted basis)
- Analyst coverage:
  - Van Leeuwenhoek – Marcel Wijma
  - ValuationLab – Bob Pooler
- Market capitalization: approx. CHF50M
- No debt

Upcoming Major Development Milestones

Milestone	Timing
Dipraglurant – LID Phase 3 Registration Program	
Complete manufacturing of Drug Product	Q3 2019
Study 301 – start dosing	Q4 2019
Study 301 – Top line data	Q3 2021

addex Therapeutics

ALLOSTERIC MODULATORS FOR HUMAN HEALTH

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