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Innovative Treatments for Central Nervous System Disorders

January 2021

Allosteric modulators for human health

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Addex Overview

Dipraglurant for dyskinesia in Parkinson's disease	➢ Pivotal registration program ongoing ➢ Study 301 start pending Covid-19 situation – expect H1 2021* ➢ US PD-LID market estimated at more than $4B ➢ FDA Orphan Drug Designation granted in PD-LID
Leading proprietary technology platform	➢ "Allosteric modulators" are a validated & differentiated pharmacological approach to address drug targets ➢ Proprietary biological screening assays and chemical library
Pipeline of in house discovered programs	➢ Blepharospasm & epilepsy Phase 2 studies starting in H1 2021 ➢ Multiple novel drug candidates entering clinical candidate selection ➢ Creating future partnership opportunities ➢ Driving long term growth
Validating partnerships with industry	➢ J&J – €109M in milestones & low double-digit royalties ➢ Indivior - $330M in milestones, tiered royalties up to double digit & funded research program
Strong balance sheet	➢ Traded on the SIX Swiss Stock Exchange under ticker ADXN ➢ ADS representing 6 shares traded on Nasdaq under ticker ADXN ➢ Pro forma cash of CHF 26 M at January 11, 2021 - Runway through 2022

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* pending removal of governmental and institutional restrictions and lessening of the impact of the global coronavirus pandemic on the U.S. healthcare system, which delayed our previously anticipated initiation in the first quarter of 2020

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 Psychosis
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

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

Venture advisor and former Partner at New Enterprise Associates.

Isaac Manke
Board member

General Partner at Acorn Bioventures.
Formerly Partner at New Leaf Venture Partners

Scientific Advisory Board

Darryle Schoepp
Chairman of SAB

Former leader of Neuroscience research department at Eli Lilly, and at Merck was Neuroscience research therapeutic area leader

Mark Bear
Picower Professor of Neuroscience at MIT

Formerly on faculty of Brown University School of Medicine and an Investigator of the Howard Hughes Medical Institute

Peter Bernstein
Principal, PhaRmaB LLC

Formerly with ICI Astra Zeneca Awarded numerous accolades including Fellow of the American Chemical Society.

Benny Bettler
Biomedicine Professor at Basel University

Formerly at Novartis and discovered allosteric modulators at $GABA_B$ receptor and recipient of the Peter Speiser Award

- World-leading neuroscientists with significant expertise in drug discovery & development
- Guiding our scientific and therapeutic area strategy
- Future exploitation of our proprietary allosteric modulation platform

In House Discovered Pipeline

Molecule / MoA	Indication	Partner	Pre-clinical	Phase 1	Phase 2	Phase 3	Milestone
Dipraglurant-IR (mGlu5 NAM)	PD-LID						Expect topline results in Q4 2022
Dipraglurant-ER (mGlu5 NAM)	Dystonia (Blepharospasm)						Expect topline results in Q4 2021
ADX71149 (mGlu2 PAM)	Epilepsy	janssen					Expect topline results in H1 2022
$GABA_B$ PAM	Addiction – alcohol use disorder	INDIVIOR					
	CMT1A						
mGlu7 NAM	Post-traumatic stress disorder	eurostars™					
mGlu2 NAM	Mild neurocognitive disorders						
mGlu4 PAM	Parkinson's disease						
mGlu3 PAM	Neurodegenerative disorders						

Lead Program Entering US Pivotal Study and Multiple Orphan Drug Opportunities

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NAM = Negative Allosteric Modulator
PAM = Positive Allosteric Modulator

IR = Immediate Release
ER = Extended Release

Dipraglurant for Parkinson's Disease

The Dipraglurant Opportunity in Dyskinesia associated with PD

Clear development & regulatory path	➢ Pivotal registration program ongoing ➢ Study 301 start pending Covid-19 situation – expect H1 2021* ➢ 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 more than $1.0B ➢ Pricing of PD therapeutics – Nuplazid ($30K p.a.) and Gocovri ($28.5K p.a.)
Dipraglurant: unique mechanism of action	➢ In house discovered, selective, orally available small molecule mGlu5 NAM ➢ PK profile mirrors that of L-dopa, ideal to treat LID ➢ Normalizes hyperglutamatergic state during L-dopa dosing
Strong data supporting dipraglurant in PD-LID	➢ Mavoglurant (Novartis mGlu5 NAM) LID data supports dipraglurant MoA & PK profile ➢ Amantadine (Gocovri) active on same biological pathway through NMDA ➢ Robust dipraglurant pre-clinical & clinical PD-LID data
Strong IP position	➢ Composition of matter through June 2025 & strong polymorph patent through 2034 (without extensions) ➢ US FDA orphan drug designation in PD-LID

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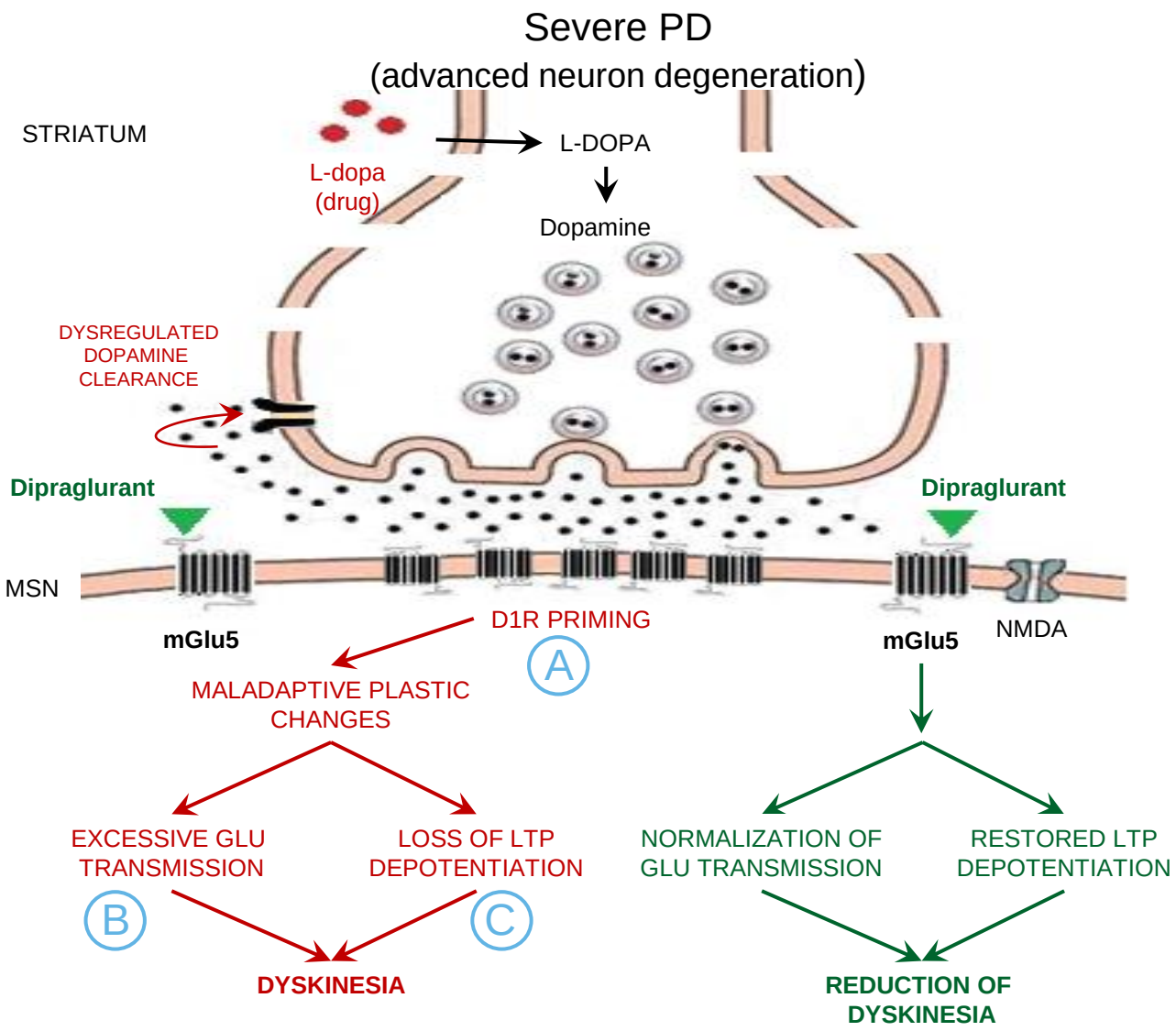
Levodopa-Induced Dyskinesia in Parkinson's Disease (PD-LID)

Long-term L-dopa use is invariably associated with the development of dyskinesias	➢ Dyskinesias result from the neurodegenerative process that underlies PD ➢ Dopamine replacement does not lead to dyskinesia per se, but lowers the triggering threshold for symptoms ➢ 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	➢ These can be painful, lead to weight loss, fatigue and exhaustion, with increased risk for falls and injuries ➢ Lead 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 treatment regimens must be continually adjusted to ensure adequate symptom control while minimizing intolerable side effects**

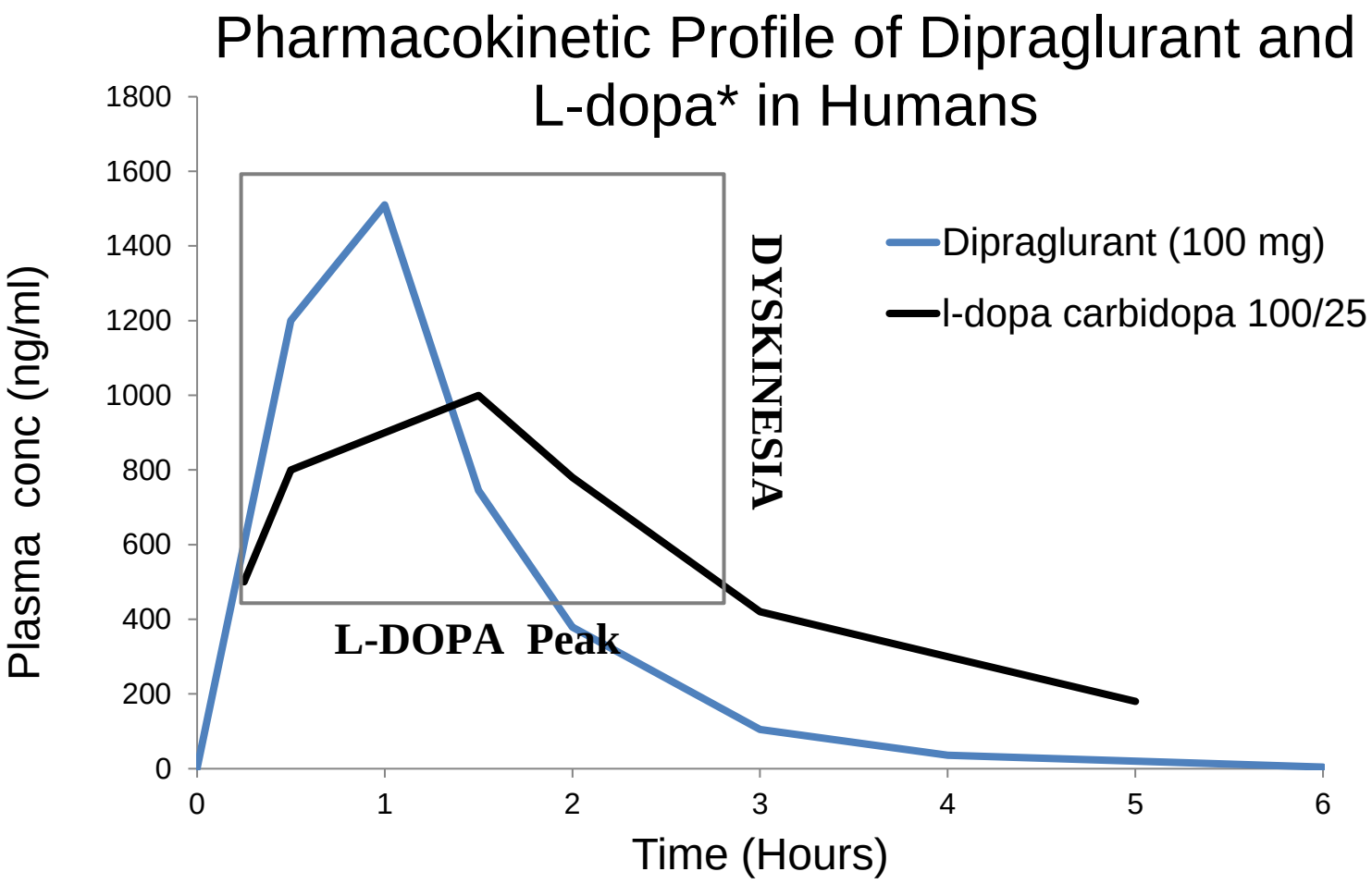
Dipraglurant - Overview and Mechanism of Action



- Loss of substantia nigra neurons combined with the non-physiological, pulsatile stimulation of dopamine receptors with L-Dopa are at the basis of LID development.
- In the striatum, **LID is the result of**:
  - A *D1 receptor priming*
  - B *Excess 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.
- Inhibition of mGlu5 decreases the excess glutamatergic tone thereby controlling dyskinesia.
- Dipraglurant is an oral, highly selective negative allosteric modulator of the mGlu5 receptor.

LTP = Long Term Potentiation
MSN = Medium Spiny Neurons

Dipraglurant PK is a Key Advantage for Treating LID

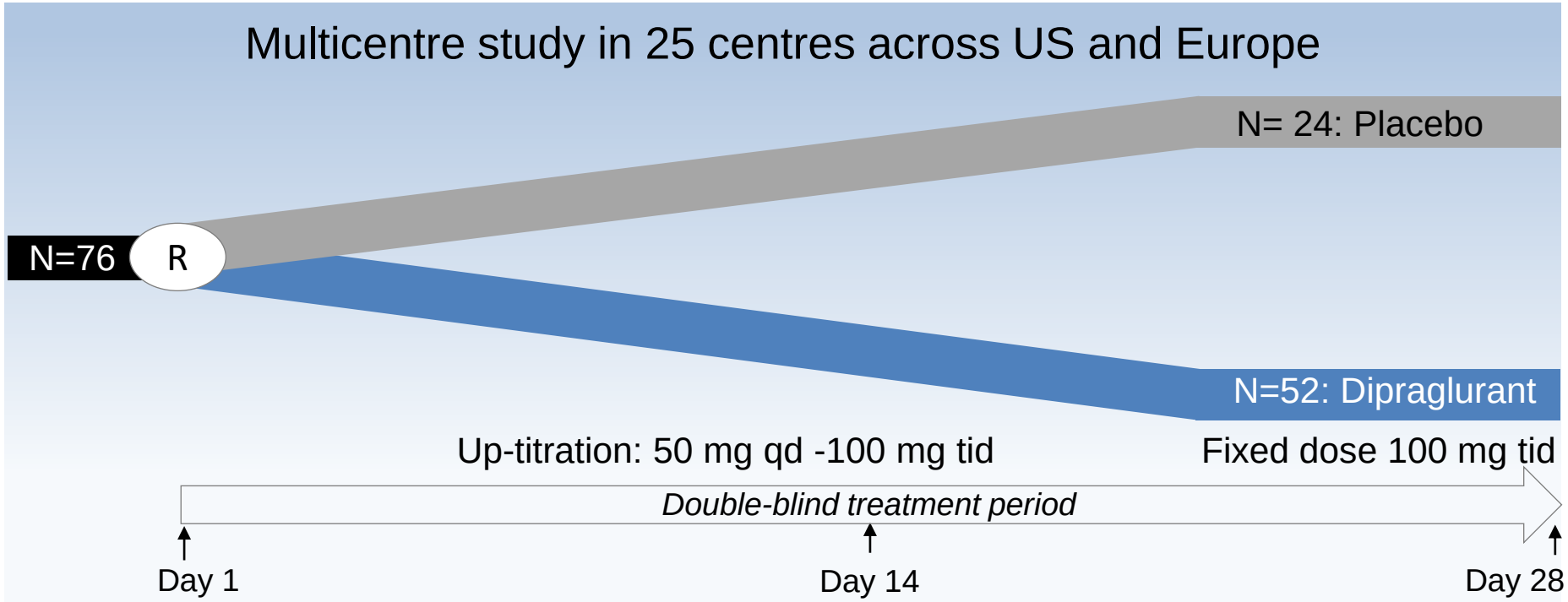


- Dyskinesia symptoms are correlated to peak levels of L-dopa
- PK profile of dipraglurant mirrors that of L-dopa
- **Dipraglurant normalizes abnormal glutamate stimulation during peak levodopa dose**

PK profile differentiates dipraglurant from other treatments

* Clinical Neuropharmacology Vol 22 No.1 pp.24-29

Dipraglurant 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
  - Change over time in UPDRS Part III
  - Clinician Global Impression of Change (CGIC)
  - 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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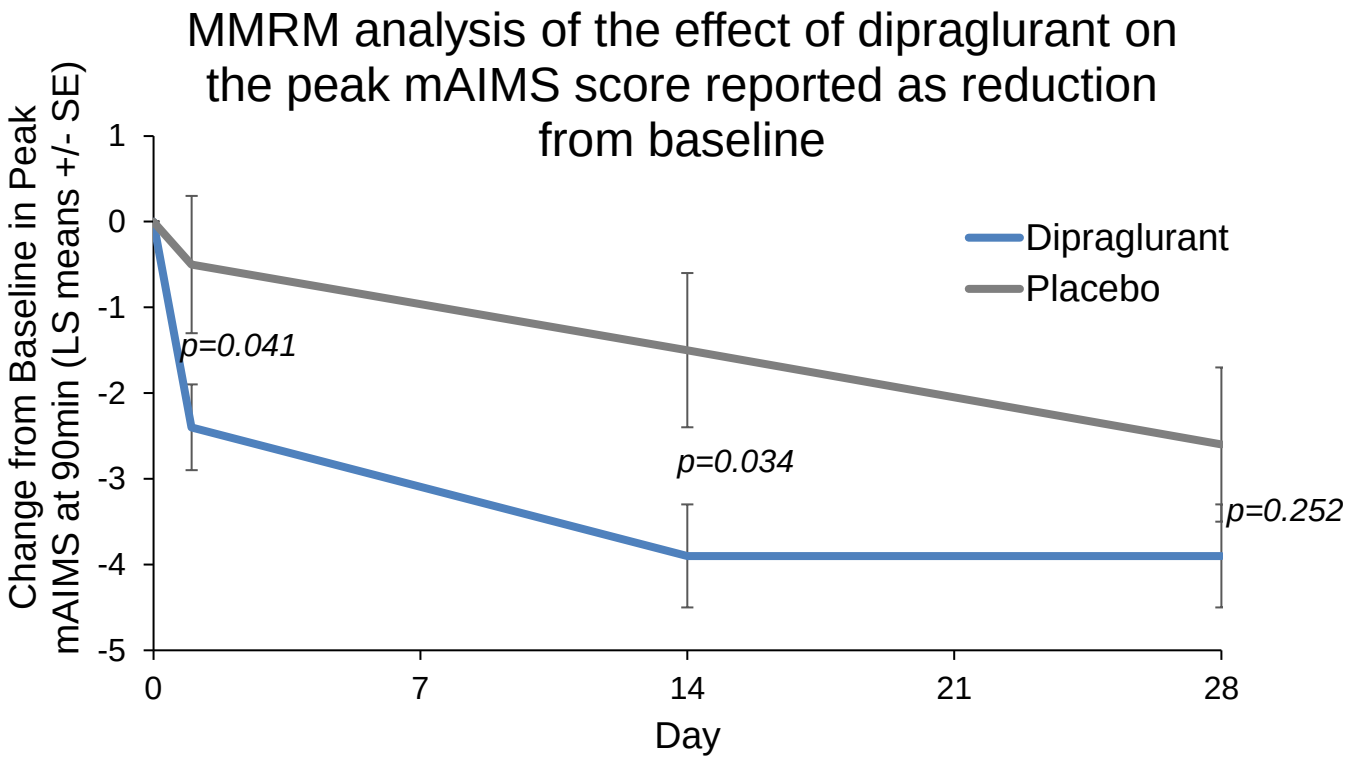
Dipraglurant Reduces LID Severity by 30%

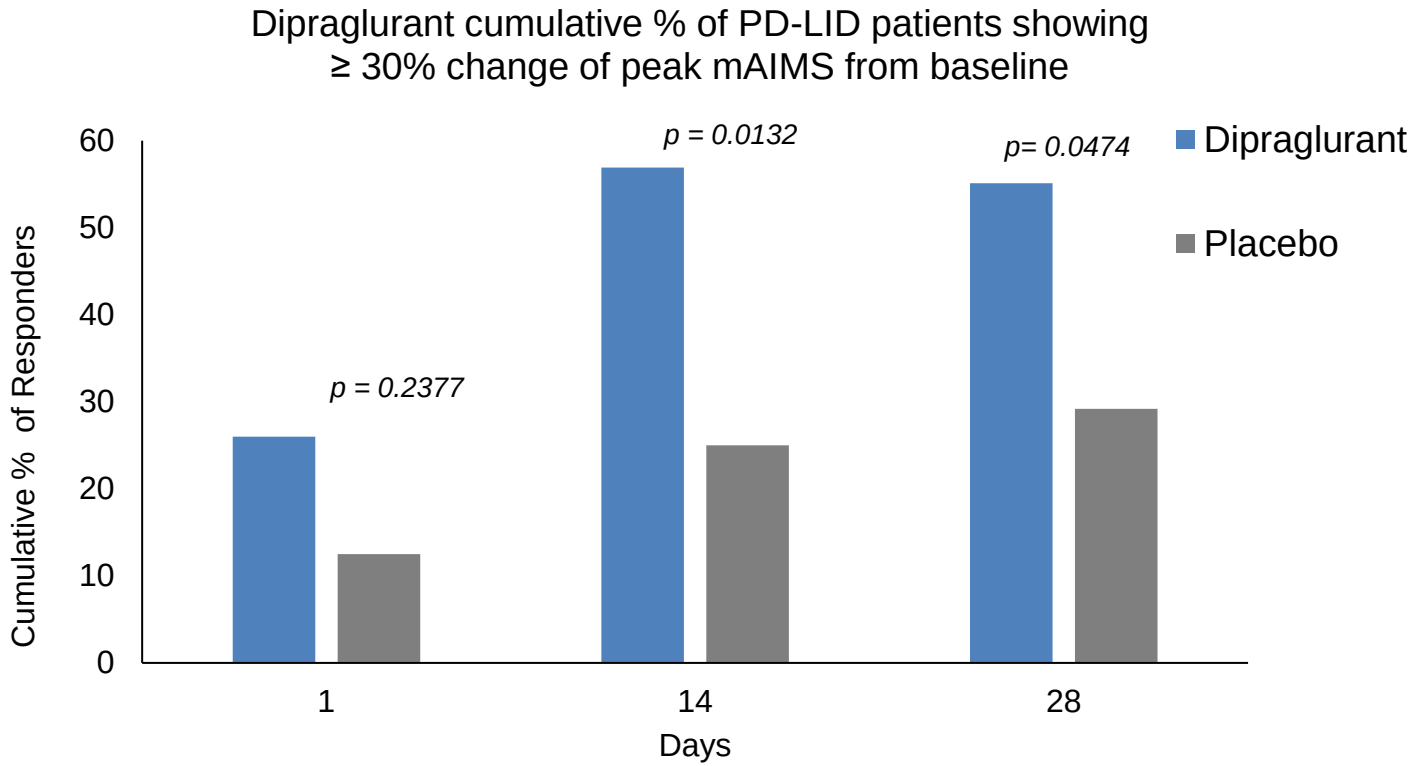


Mean % change of peak mAIMS from baseline		
Midday dose	Dipraglurant	Placebo
Day 1 (50 mg)	19.9%	4.1%
Day 14 (100 mg)	32.3%	12.6%
Day 28 (100 mg)	31.4%	21.5%

- 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 resulted in significance being lost 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

Clear dose response but need to manage placebo

Responder Analysis Demonstrates Dipraglurant Significant Benefit



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	55.1%	n=7	29.2%	0.0474

- A 30% reduction in mAIMS
  - 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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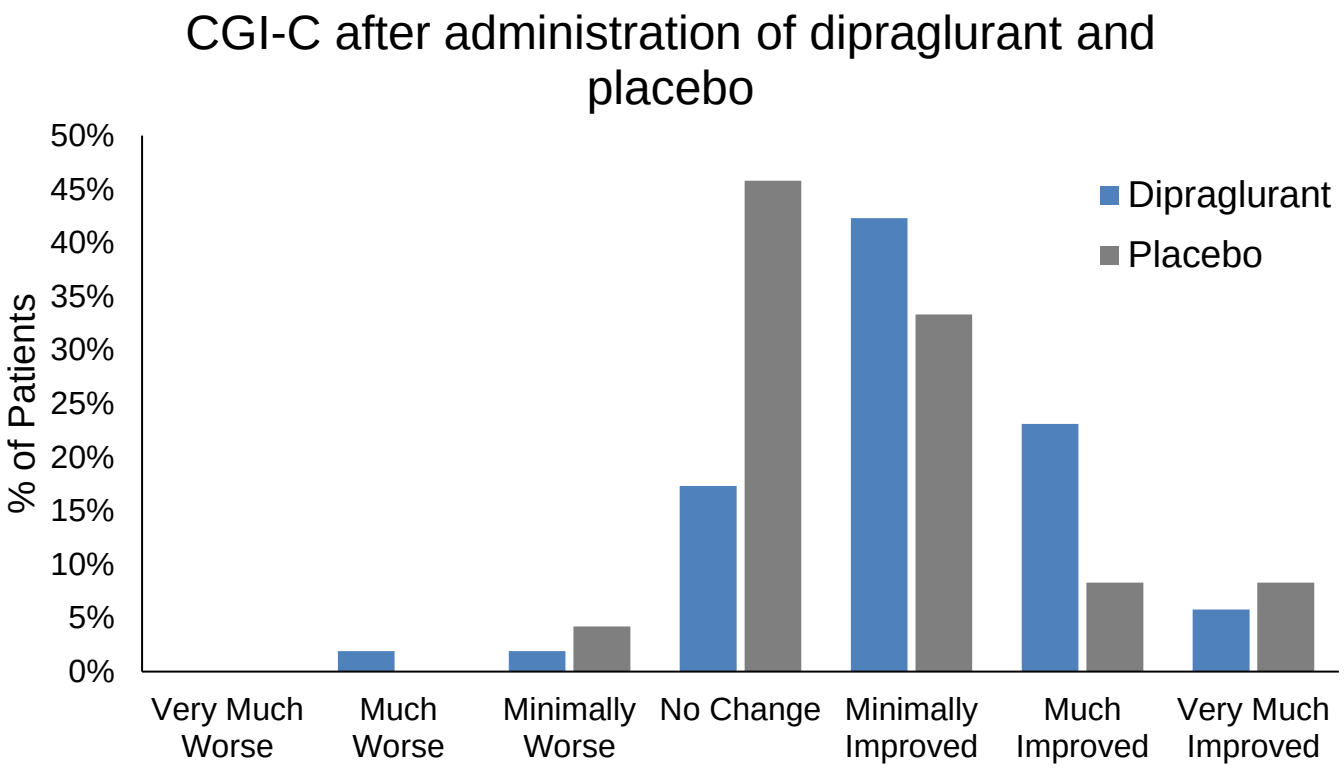
Clinician Rated Global Impression of Change - Dyskinesia



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

- Relatively simple scale reflecting everyday clinical practice
- Assessment by treating physician and thus more objective than the more subjective mAIMS
- Supports benefit of using UDysRS in pivotal program
- Assessed at end of study compared to baseline

Dyskinesia improved with dipraglurant according to clinicians (p<0.05)

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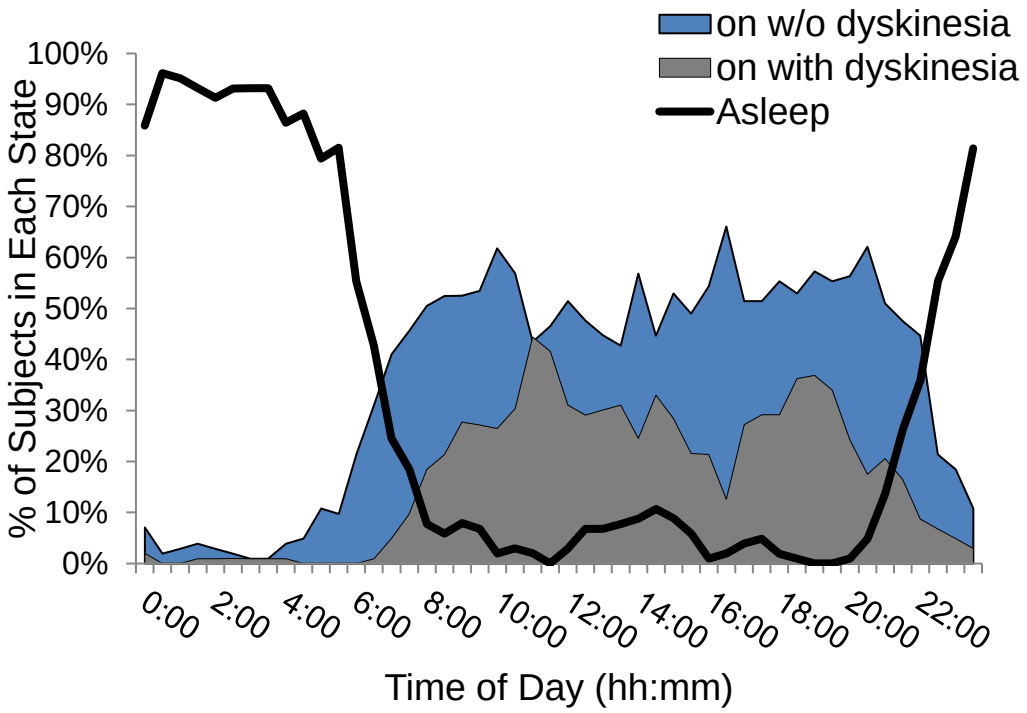
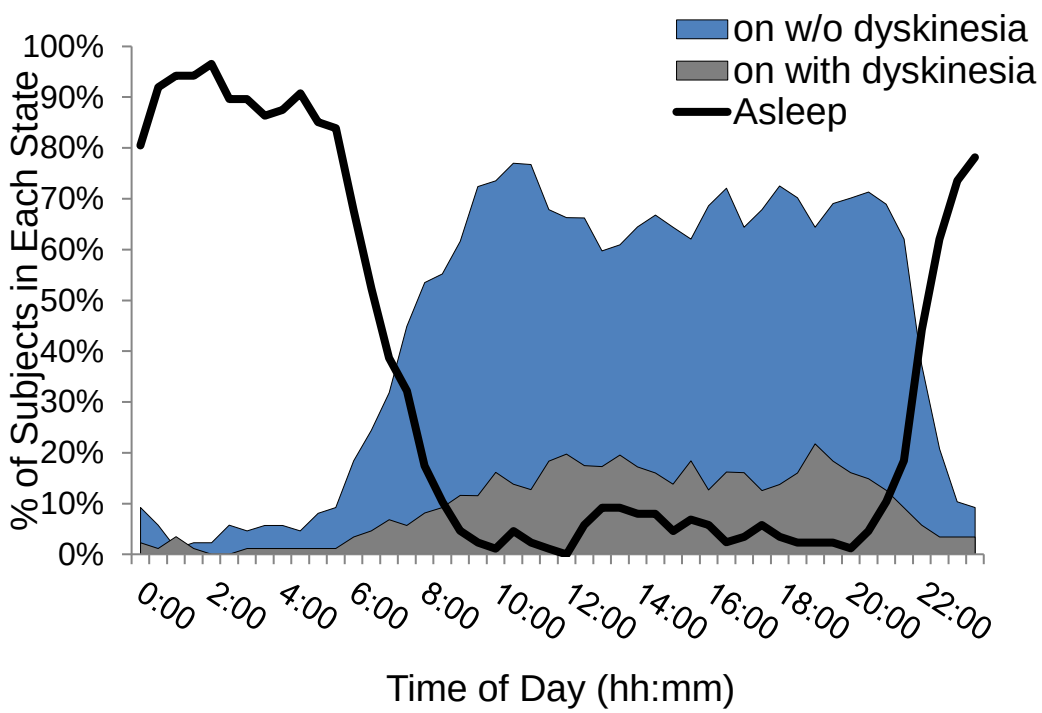
Patient Diaries – Improvement Throughout the Waking Day

Pattern of motor complications of dipraglurant patients over the course of a day, as reported in patients' diaries

Baseline (n=42)



Week 4 (n=42)



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 Demonstrated Good Safety and Tolerability in PD Patients

- Adverse events were common in both treatment groups (dipraglurant 88.5%, placebo 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 ECG, HR, BP, haematology and biochemistry

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

Dipraglurant PD-LID Registration Program Started

- Pivotal registration program ongoing
- Study 301 start pending Covid-19 situation – expect H1 2021*
  - Study is expected to read out in Q4 2022
- 12 month Open Label Study (302) starting in parallel to study 301
  - 6 and 12 month safety data
- Primary endpoint: UDysRS – more sensitive to treatment effect than mAIMS and less prone to placebo response (Goetz, 2013 and 2008)
- Placebo mitigation is a priority
- Second pivotal registration study (303) to follow study 301 completion
- Considering Fast-Track and/or Breakthrough Therapy applications after first pivotal study readout

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Dyskinesia Rating Scales: UDysRS verses mAIMS

	UDysRS	mAIMS
Characteristics	• Recommended scale by Movement Disorder Society • FDA regulatory NDA precedent (Adamas - Gocovri) • Contains anchored objective clinician evaluated measures of dyskinesia • UDysRS has both patient-based perceptions of disability and physician assessments of impairment and disability embedded in the single scale • Less prone to placebo effect	• mAIMS alone was identified as suboptimal in detecting treatment-related changes • mAIMS patient driven • More prone to placebo effect
Clinimetric properties	• Validated	• Only the original version has been validated
History	• Developed in 2009 specifically for dyskinesia in PD patients	• Developed in 1970 to assess tardive dyskinesia in psychiatric patients

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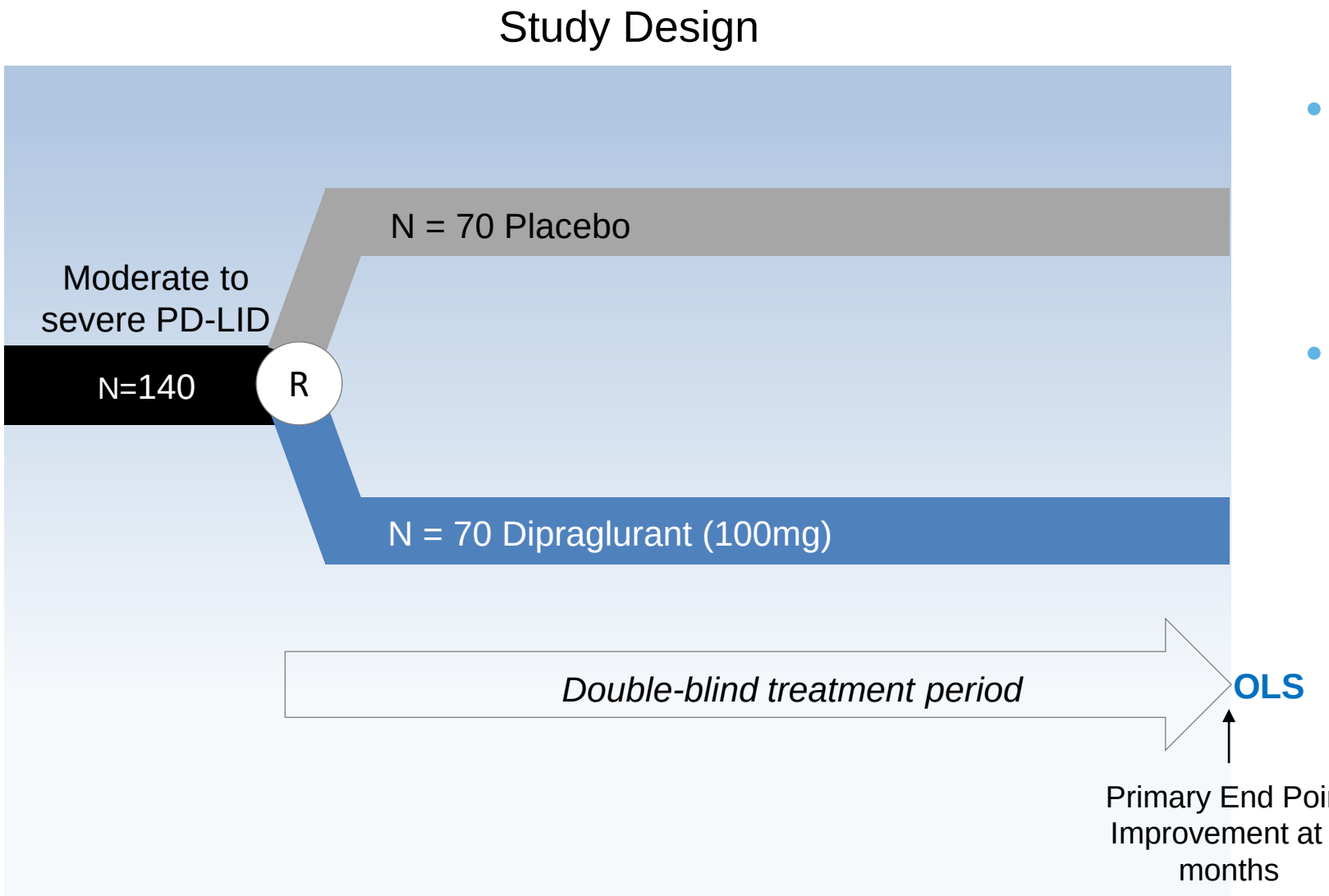
UDysRS= Unified Dyskinesia Rating Scale

Dipraglurant PD-LID Studies – Management of Placebo Response

- Use of UDysRS to measure efficacy endpoint instead of the mAIMS
  - UDysRS is more sensitive to changes in LID than mAIMS (Goetz et al., Mov Disord 2013)
  - Less prone to placebo response than mAIMS which lacks the functional/objective items of UDysRS
- Raters will be qualified by the Movement Disorder Society to assess LID using the UDysRS scores
- Expert rater training service will review UDysRS ratings during the study to further ensure quality as well as inter- and intra-rater reliability
- Randomized patients will be required to have dyskinesia scores at screening and baseline that reflect moderate to severe symptoms
- A non-pharmacologic intervention BPST-Dys will be incorporated into the screening period to help exclude placebo-responders from the randomized, controlled portion of the study
- The longer 12 week randomized treatment period should also help to mitigate placebo response, compared to the 4-week treatment duration tested in the proof-of-concept study

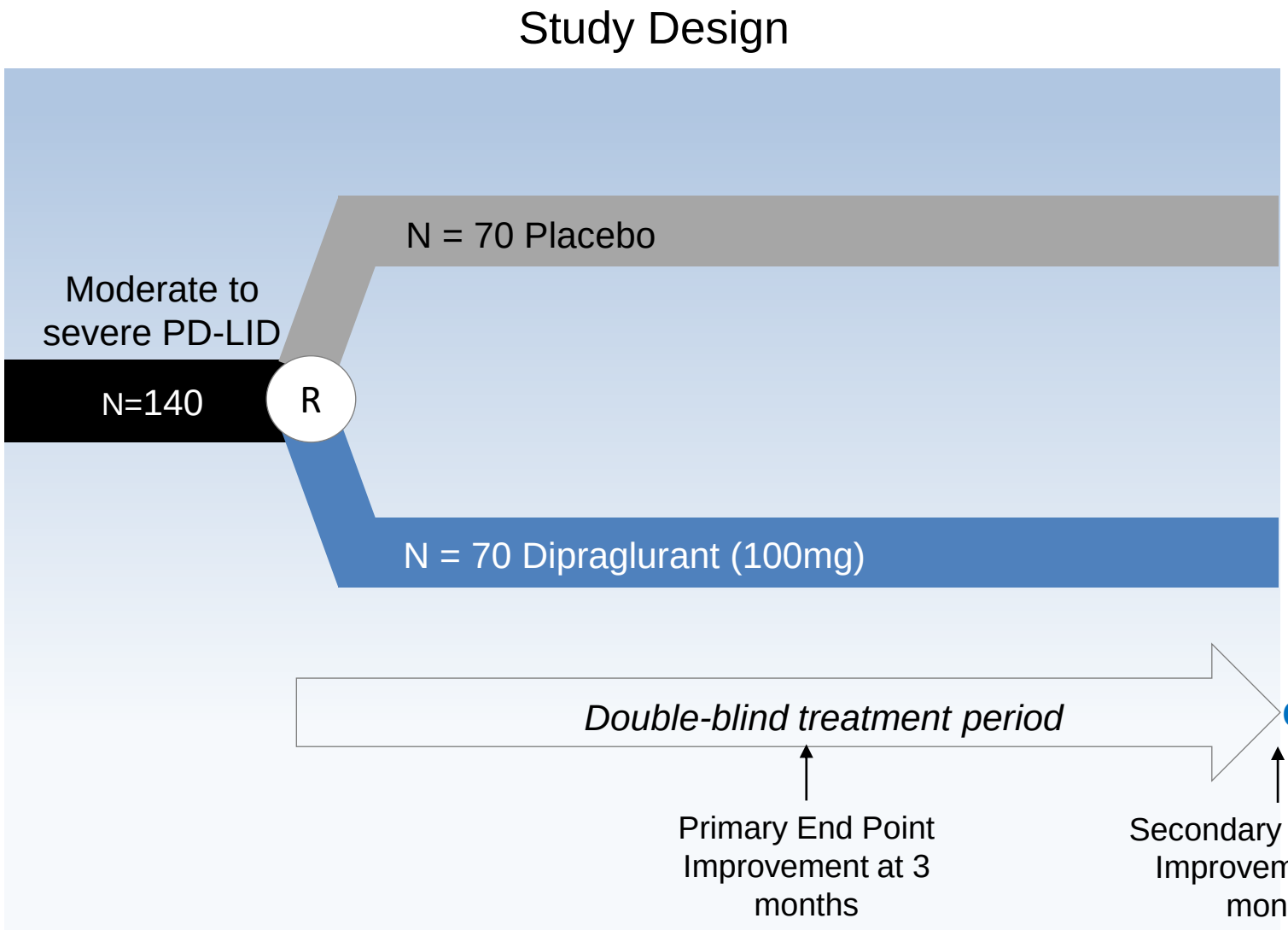
Dipraglurant First Pivotal PD-LID Study (301)



- Primary objective is efficacy in reducing LID
  - Change over time in UDysRS at 3 months
- Secondary objectives
  - Change over time in MDS-UPDRS Part III
  - Patient diaries, on & off time
  - CGI-S
  - Safety and tolerability

R= Randomisation
OLS = Open label study

Dipraglurant Confirmatory Phase 3 PD-LID Study (303)



- Primary objective efficacy in reducing LID
  - Change over time in UDysRS at 3 months
- Secondary objectives
  - Change over time in UDysRS at 6 months
  - Change over time in MDS-UPDRS Part III
  - Patient diaries, on & off time
  - CGI-S
  - Pharmacokinetics (PK)
  - Safety and tolerability

R= Randomisation
OLS = Open label study

Dipraglurant LID Opportunity

LID has a large unmet need and market opportunity	➢ > 170K LID patients in US ➢ ~ $1B US peak sales potential for dipraglurant
Limited competition – only one FDA approved medicine	➢ Gocovri (reformulation of generic amantadine) approved in August 2017, safety profile similar to generic ➢ Dipraglurant is a highly selective orally available mGlu5 NAM ➢ Improved safety profile & ideal PK profile mirrors levodopa
Clear development plan with precedented regulatory path	➢ Two registration trials (301 and 303) with Open Label Study (302) ➢ UDysRS is more sensitive to treatment effect & less prone to placebo response (Goetz 2013 & 2008) ➢ Implementing measures to manage placebo response in registration program
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

Dipraglurant for Dystonia – Blepharospasm

The Dipraglurant Opportunity in Dystonia – Blepharospasm (BSP)

Disease and prevalence	➢ Dystonia is the third most common movement disorder characterized by involuntary muscle contractions and spasms affecting up to 300,000 people in the US ➢ BSP affects eyelid muscles resulting in sustained eyelid closure causing substantial visual disturbance or functional blindness ➢ In >50% of BSP patients symptoms spread to other facial muscles ➢ At least 50,000 BSP patients in US, with up to 2000 new patients diagnosed annually
Unmet need & commercial opportunity	➢ Botulinum toxin injections are the only approved treatment ➢ Off label use of anticholinergic agents, dopamine receptor agonists and baclofen have marginal efficacy and side effects ➢ Surgical approaches including myectomy are invasive and frequently not of benefit
Robust Data with dipraglurant in dystonia	➢ Dose-dependent reduction of dystonia in MPTP-lesioned NHP model of PD-LID ➢ Effective in tottering mouse model of generalized dystonia ➢ Reverses synaptic plasticity alterations observed in two distinct genetic models of dystonia (DYT1 mice & DYT25 rats)
Status of development	➢ Phase 2 feasibility study in BSP with Dipraglurant IR expected to start in H1 2021 and read out data by the end of 2021 ➢ Explore use of Computational Motor Objective Rater (CMOR) with established BSP rating scales ➢ Dipraglurant extended release formulation being developed ➢ Phase 2a proof of concept with dipraglurant ER planned for 2022

ADX71149 (JNJ-40411813) for Epilepsy
Partnered with Janssen (JNJ)

ADX71149 Opportunity in Epilepsy

Large market & unmet medical need	➢ Keppra (levetiracetam) market leader with €770 million ➢ Significant unmet medical need remains due to dose limiting side effects ➢ No rational polypharmacy treatment in epilepsy - combination treatment in refractory patients has limited therapeutic benefit
ADX71149: true synergistic mechanism of action	➢ Selective, orally available small molecule mGlu2 PAM ➢ ADX71149 showed 35 fold increase in Keppra efficacy in preclinical 6Hz model ➢ Synergy could yield first rational polypharmacy drug in epilepsy

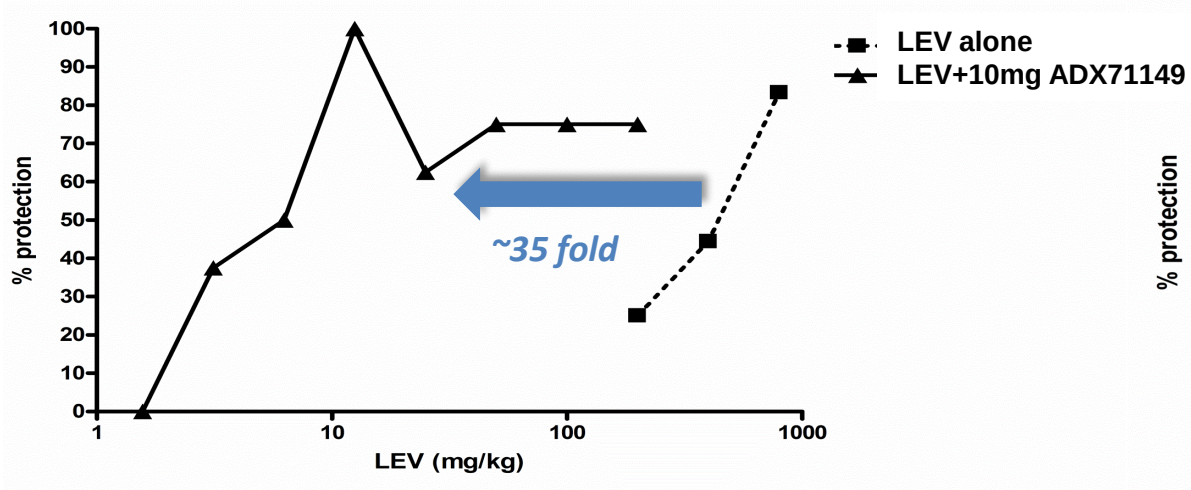
Development path	➢ Extensive preclinical and clinical data package
	  ➢ 8 Phase 1 and 2 Phase 2 studies completed
	➢ Janssen expect to start epilepsy POC study in H1 2021
	  ➢ Top line data expected in H1 2022

Partnership with Janssen	➢ To date, Addex has received €10.2 million in upfront, research funding and milestones ➢ Eligible to receive €109 million in additional pre-launch milestones and low double digit royalties

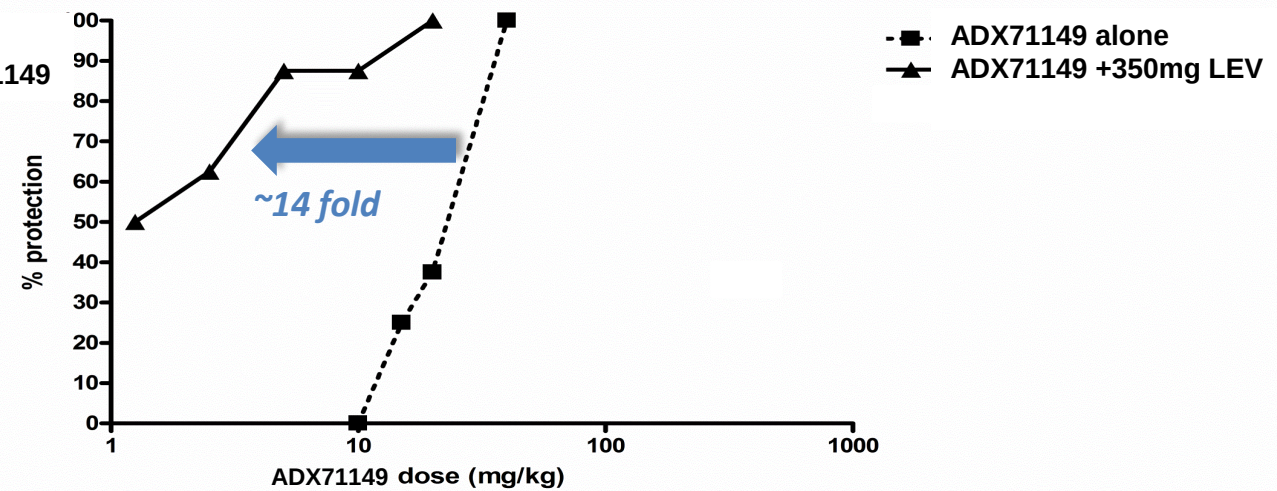
ADX71149 Preclinical Efficacy in Epilepsy – 6Hz Model

- Preclinical validation in pharmacoresistant mouse epilepsy model:

ED_{50} shift of Keppra by adding low dose of ADX71149

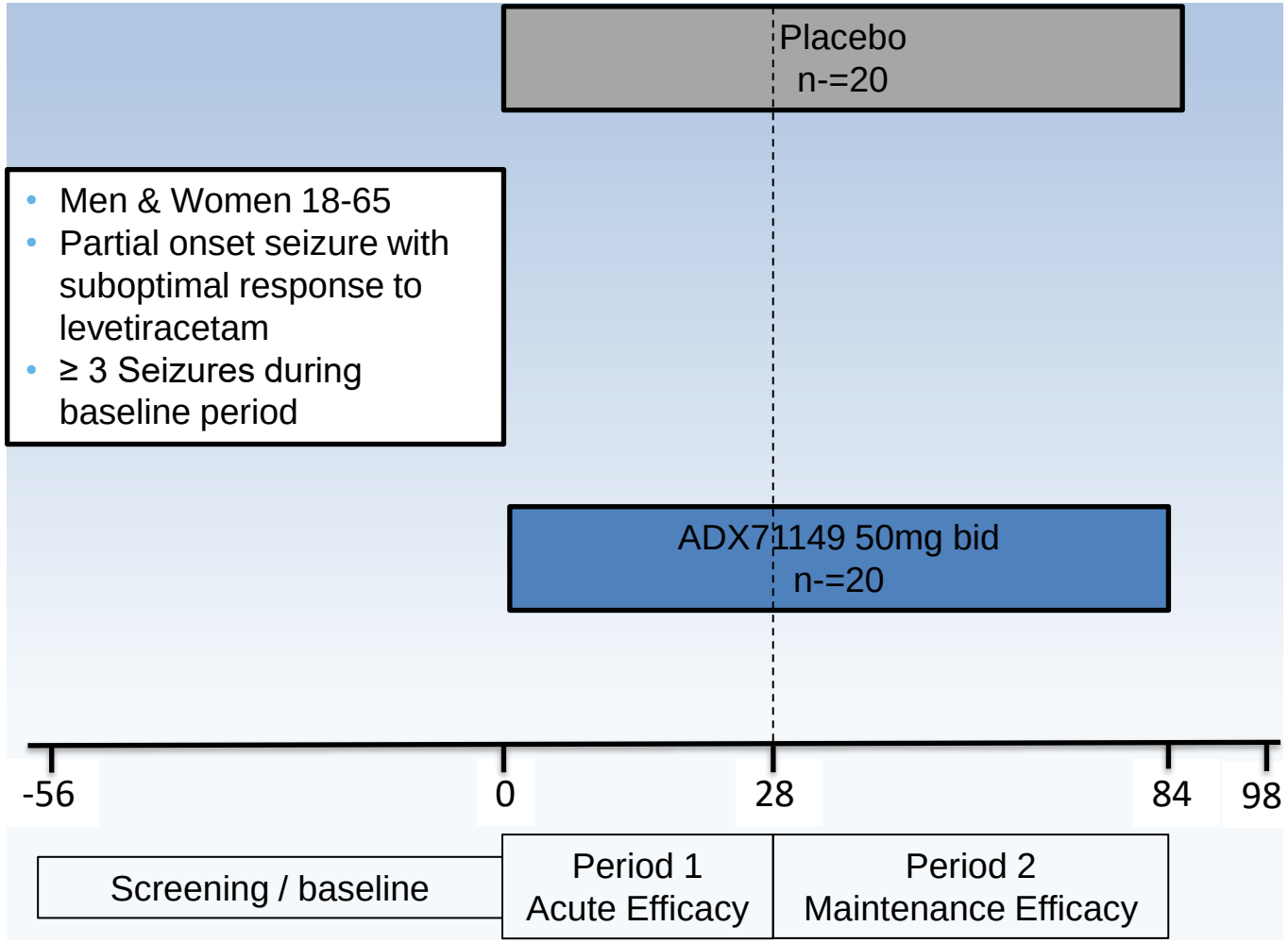


ED_{50} shift of ADX71149 by adding ED_{50} dose of LEV



- Keppra efficacy increased by 35 fold when administered with a low dose of ADX71149.
- Low dose of Keppra leads to 14 fold increase in efficacy of ADX71149
- True synergistic effect
- Similar effect observed with Briviact

ADX 71149 Phase 2a POC Epilepsy Study Design



- Double blind placebo controlled
- 56 day baseline period
  - establish 28 day seizure count
- Period 1: 4-week acute efficacy phase
  - Time to baseline seizure count
- Period 2: 8-week maintenance efficacy phase
  - Subjects who do not reach/ exceed their monthly baseline seizure count in Period 1 (Week 4) will continue their double-blind treatment during Period 2

$GABA_B$ Positive Allosteric Modulators (PAM) for Addiction & Charcot-Marie-Tooth Type 1A (CMT1A) Neuropathy

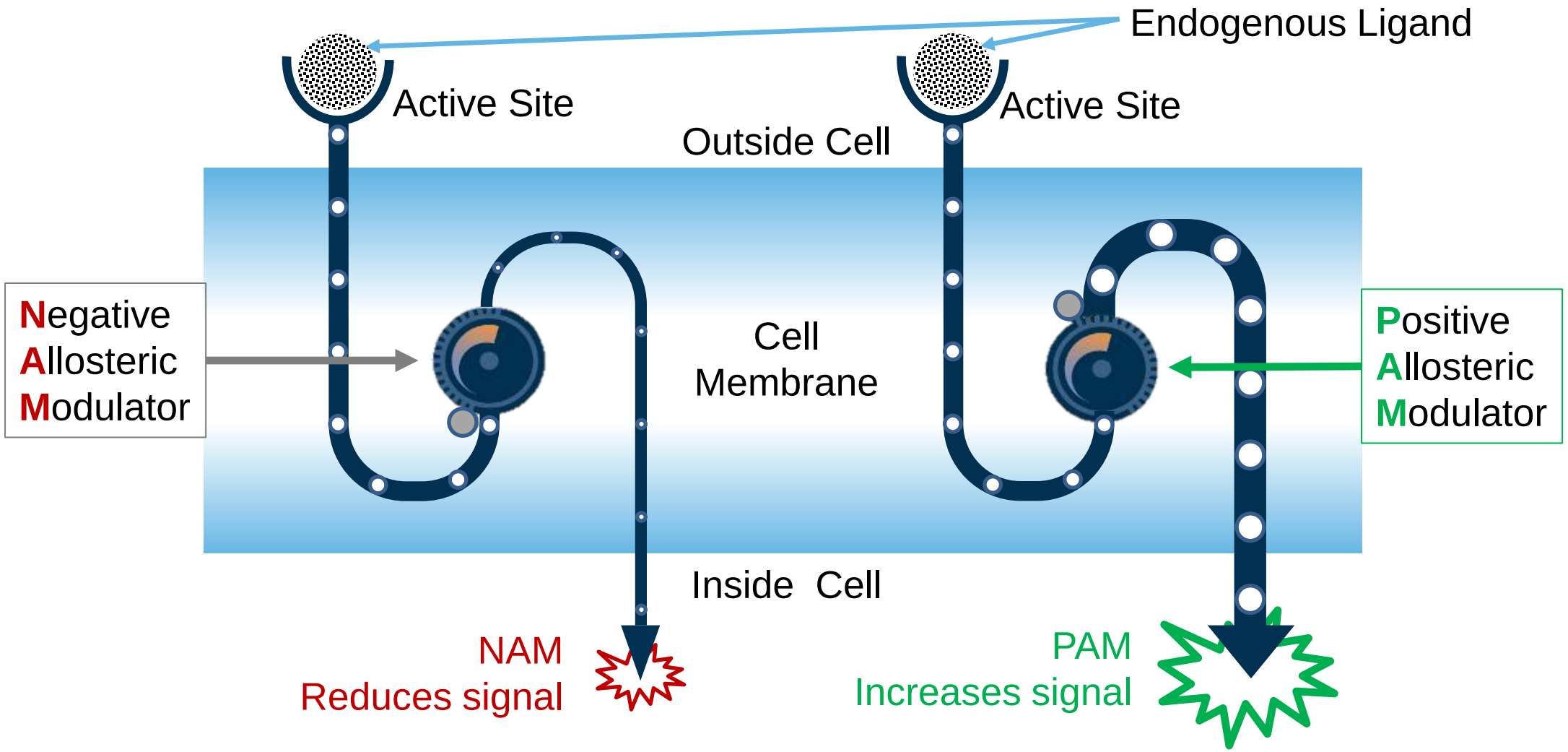
Collaboration with Indivior for Addiction

$GABA_B$ PAM for Addiction and CMT1A

- $GABA_B$ is the metabotropic receptor for GABA, main inhibitory neurotransmitter
  - Activation of $GABA_B$ is beneficial in alcoholism and CMT1A through use of baclofen
  - PAM is a differentiated approach, resulting in potential safety and efficacy advantages over baclofen
- Indivior partnership since 2018
  - Addex is leading a funded research program to deliver novel drug candidates that is in clinical candidate selection phase
  - Addex has right to select back up compounds at clinical candidate stage for development in retained indications including CMT1A neuropathy
  - Financials: upfront of USD5.0M, USD5.6M research funding received to date, USD2.8M committed and USD330M of development, regulatory and commercial milestones as well as tiered royalties up to double-digit
- CMT1a is an orphan genetic peripheral polyneuropathy involving duplication of the PMP22 gene
  - Prevalence: 3 in 10,000 - upper limit of orphan classification
  - Most common inherited neurological disease
  - Regulatory path to NDA submission paved by Pharnext PX3003 (fixed dose combination of baclofen, naltrexone and sorbitol)

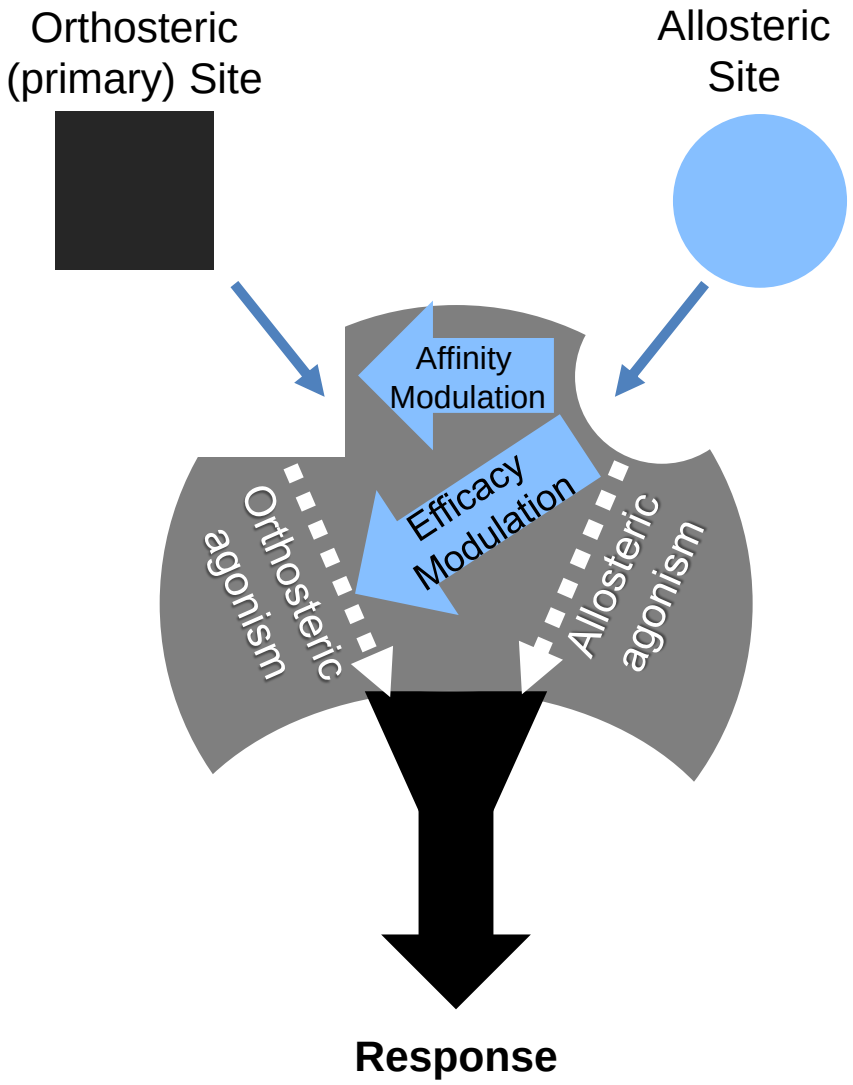
Addex Allosteric Modulation Technology Platform Becoming an Exciting Space

What are Allosteric Modulators?



Addex is based on a leading technology platform

Using Allosteric Modulation to Discover Novel CNS Drug Candidates



Potential benefits

- Novel, orally available drug class
- Superior receptor sub-type selectivity compared to orthosteric ligands
- Bind to non-competitive sites and therefore potential to address intractable targets
- Re-address well characterized and clinically validated GPCR targets
- Potential for improved safety due to selectivity and modulatory pharmacology
- Potentially superior efficacy over long term due to lack of tolerance from more modulatory pharmacology
- Clinical use in combination with competitive agonists

*Modified from : Nat Rev Drug Discov."*Allosteric modulators of GPCRs: a novel approach for the treatment of CNS disorders". Jeffrey Conn, et al

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Financials

Financials and Stock

- Cash runway through 2022
  - Cash at September 30, 2021; CHF17.8 million
  - Pro forma cash following capital increase announced on January 11, 2021: CHF26 million
- Market capitalization: approx. CHF 64.6M
- No debt
- Traded on SIX Swiss Exchange: ADXN (ISIN:CH0029850754)
- ADS representing 6 shares traded on Nasdaq: ADXN (ISIN: US00654J107; CUSIP: 00654J107)

- 39,748,635 shares outstanding 56.2M (fully diluted, including treasury shares)
  - New Enterprise Associated – 14.21%
  - New Leaf Venture Partners - 4.77%
  - CAM Capital - 4.02%
  - Credit Suisse Asset Management – 2.87%
  - Management & board holds -12.15% (fully diluted basis)
- Analyst coverage:
  - Van Leeuwenhoek - Marcel Wijma
  - valuationLab - Bob Pooler
  - ZKB - Dr. Michael Nawrath
  - Baader Helvea AG – Bruno Bulic

Milestones

Milestone	Timing
Dipraglurant – LID	
Phase 2b/3 study in LID – start dosing	H1 2021*
Phase 2b/3 study in LID – results	Q4 2022
Dipraglurant – Dystonia (Blepharospasm)	
Phase 2a in Blepharospasm – start dosing	H1 2021
Phase 2a in Blepharospasm – results	Q4 2021
ADX71149 – Epilepsy	
Phase 2a POC study in Epilepsy – start dosing	H1 2021
Phase 2a POC study in Epilepsy – results	H1 2022
$GABA_B$ PAM	
Expect IND enabling studies to be initiated	2022

* pending removal of governmental and institutional restrictions and lessening of the impact of the global coronavirus pandemic on the U.S. healthcare system, which delayed our previously anticipated initiation in the first quarter of 2020

Summary

- Leading technology platform
- Proprietary in-house discovered pipeline
- Partnerships with industry
- Lead asset, dipraglurant moving into pivotal study for large unmet need and commercial opportunity with limited competition
  - Strong mechanism of action rational
  - Experienced team of drug developers
- Top tier US investors – NEA, NLV and CAM Capital
- Dual listed on SIX Swiss Exchange & US Nasdaq
- Rich news flow from pipeline in 2021 and 2022

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