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# Innovative Treatments for Central Nervous System Disorders

**July 2024**

*Allosteric modulators for human health*

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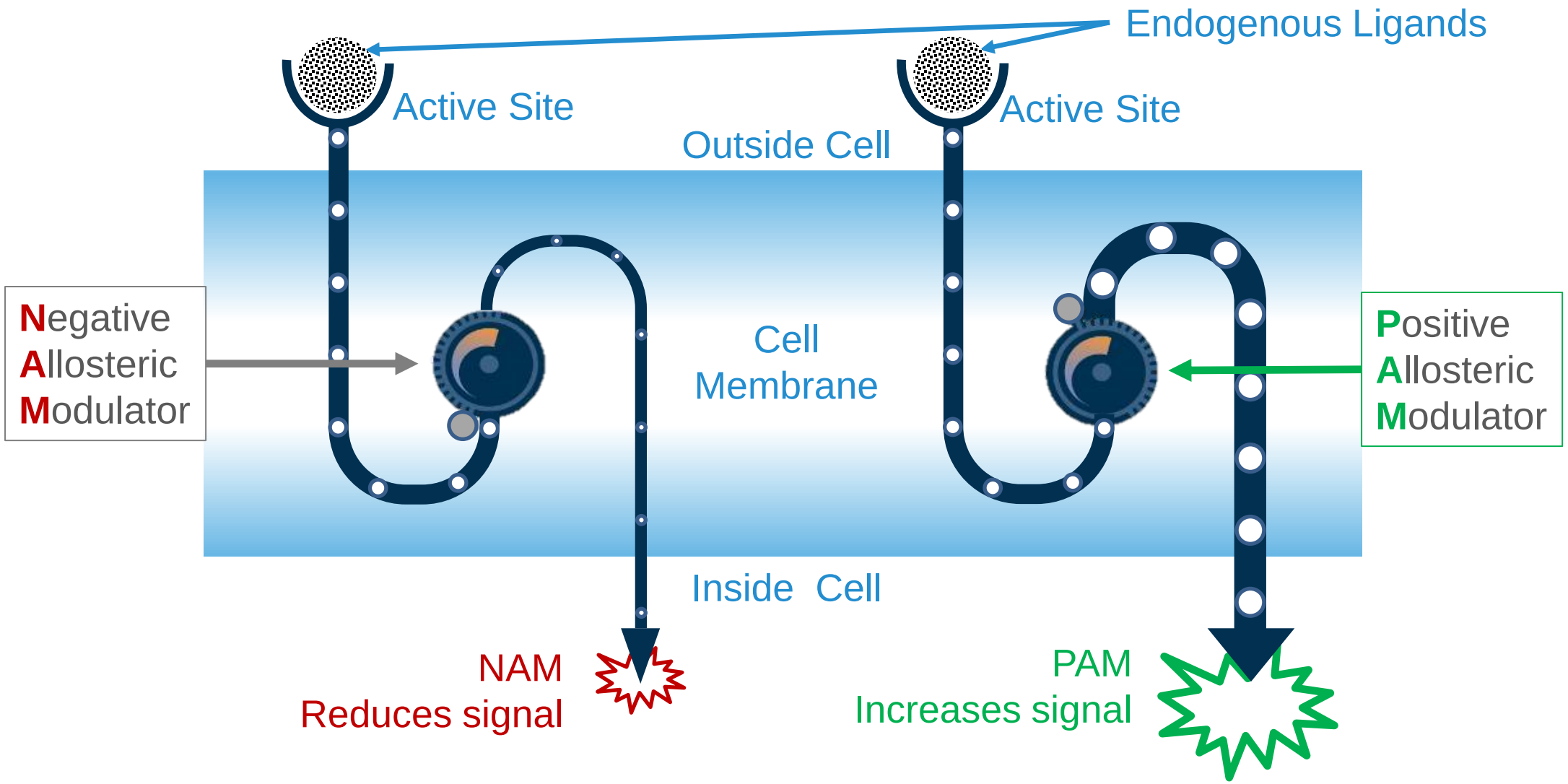
# Addex Overview

| | |
|---|---|
| High value programs reaching significant milestones | ➢ ADX71149 Phase 2 epilepsy study (J&J) – full data set being analyzed<br>➢ GABAB PAM for cough (Addex) & SUD[1] (Indivior) in CCS[2]<br>➢ Dipraglurant for PD-LID & post-stroke/TBI[3] recovery – Phase 2 ready<br>➢ Neurosterix portfolio advancing towards IND enabling studies |
| 20% equity interest in spin-out company, Neurosterix | ➢ Leading allosteric modulator drug discovery platform<br>– Validated & differentiated pharmacological approach<br>➢ Preclinical portfolio of high value programs<br>– Lead program: M4 PAM for schizophrenia<br>➢ $63M series A financing in April 2024 led by Perceptive Advisors |
| High value industry partnerships driving future value | ➢ J&J - €109M in milestones & double-digit royalties<br>➢ Indivior - $330M in milestones, royalties up to double digit & funded research program |
| Strong balance sheet & top tier US investors | ➢ Dual listed on SIX Swiss Exchange & US Nasdaq Capital Market<br>➢ CHF 1.6M ($1.8M) cash at March 31, 2024<br>– April 2024 Neurosterix spin-out: CHF 5M cash received in April 2024 & reduced future cash burn<br>➢ Cash runway extended beyond 2026 |

[1]SUD = Substance use disorder
[2]CCS = Clinical candidate selection phase
[3]TBI = Traumatic Brain Injury

# What are Allosteric Modulators?




No activation in the absence of the endogenous ligand

# Advantages of Allosteric Modulation Vs Orthosteric Drug Discovery

| | Conventional small molecules | Biologics /peptides | Nucleic acid-based therapies | Gene therapies | Allosteric modulators |
|---|---|---|---|---|---|
| Selectivity | + | ++ | +++ | +++ | ✓ |
| Differentiated pharmacology | - | - | +++ | +++ | ✓ |
| Better potential safety/tolerability | ++ | + | ++ | - | ✓ |
| Non-competitive mechanism | - | - | n/a | n/a | ✓ |
| Respect physio-logical rhythm | - | - | - | - | ✓ |
| Oral bioavailability | +++ | + | - | - | ✓ |
| Crossing BBB | +++ | - | - | - | ✓ |
| No immunogenicity | +++ | - | + | + | ✓ |
| Low cost of goods | +++ | - | - | - | ✓ |

Allosteric modulators

- Address:
  - "Undruggable" targets, such as GPCRs, RTKs, cytokine receptors and enzymes
  - mAb and peptide drug targets with oral small molecules
- Offer exquisite selectivity and superior safety profile
- Are suitable for chronic treatment as potency maintained over prolonged periods
- Require a distinct HTS and characterization approach to discovering small molecule allosteric modulators
- Proven clinical approach (diazepam, cinacalcet, etc)

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# Pipeline of In House Discovered Programs

| Molecule / MoA | Partner | Stage | | | | Milestone |
|---|---|---|---|---|---|---|
| | | Discovery | IND Studies | Phase 1 | Phase 2a | |
| ADX71149 (mGlu2 PAM) | janssen | Epilepsy | | | | Full data set for H2 2024 |
| Dipraglurant (mGlu5 NAM) | | PD-LID | | | | Ready to start Phase 2b/3 study* |
| Dipraglurant (mGlu5 NAM) | | Post-stroke/TBI recovery | | | | Ready to start Phase 2a study* |
| $GABA_B$ PAM | INDIVIOR | Substance use disorders | | | | IND enabling studies expected to start H2 2024 |
| $GABA_B$ PAM | | Chronic cough | | | | IND enabling studies ready to start H2 2024 |

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* Timings subject to financing/partnering
TBI = Traumatic brain injury
NAM = Negative Allosteric Modulator
PAM = Positive Allosteric Modulator

# ADX71149 (JNJ-40411813) for Epilepsy

*Partnered with Janssen Pharmaceuticals, Inc*

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# ADX71149 - Opportunity in Epilepsy

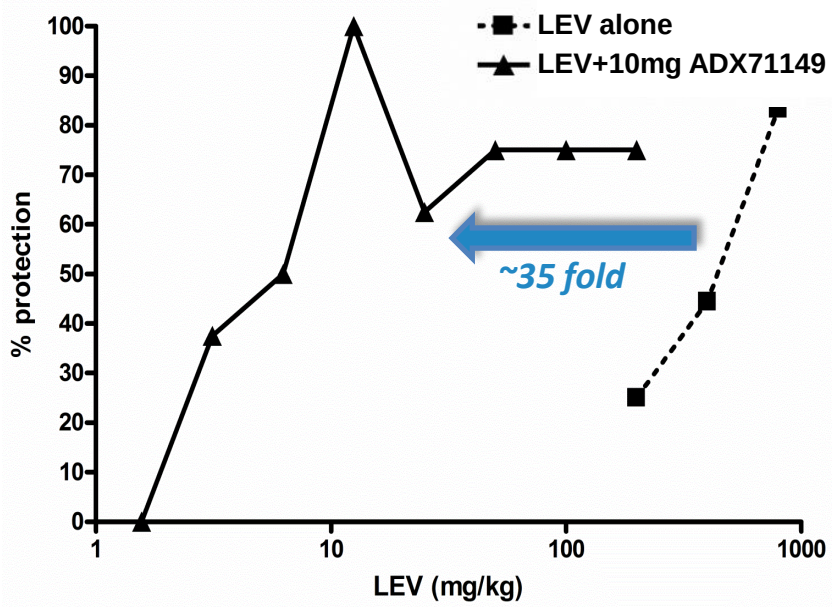
| | |
|---|---|
| Large market & unmet medical need | ➢ Market projected to reach $20 billion by 2026[1]<br>– Keppra & Briviact net sales in 2022 of €1.2 billion treating 2M patients[2]<br>➢ High proportion of refractory patients (¼ of new patients3) - combination treatments have limited therapeutic benefit<br>➢ Large underserved patient population needing improved treatment options |
| Strong MoA & synergistic effect | ➢ Selective oral mGlu2 PAM demonstrated clear MoA in epilepsy<br>➢ Showed 35-fold increase in Keppra (SV2A antagonist) efficacy<br>➢ Potential to reduce SV2A antagonist dosing – improve efficacy & reduce side effects |
| Status of development | ➢ Phase 2 study completed<br>– Cohort 1 (60 patients) completed<br>– Cohort 2 (50 patients) completed<br>– Top-line data did not show statistical significance – reported April 29, 2024<br>– Full data set being analysed |
| Strategic Partner Janssen Pharmaceuticals, Inc. | ➢ Eligible to receive €109 million in pre-launch milestones and double-digit royalties |

[1] Fortune Business Insights April 8, 2020
[2] UCB FY 2022
[3] Xue-Ping et al, Medicine July 2019

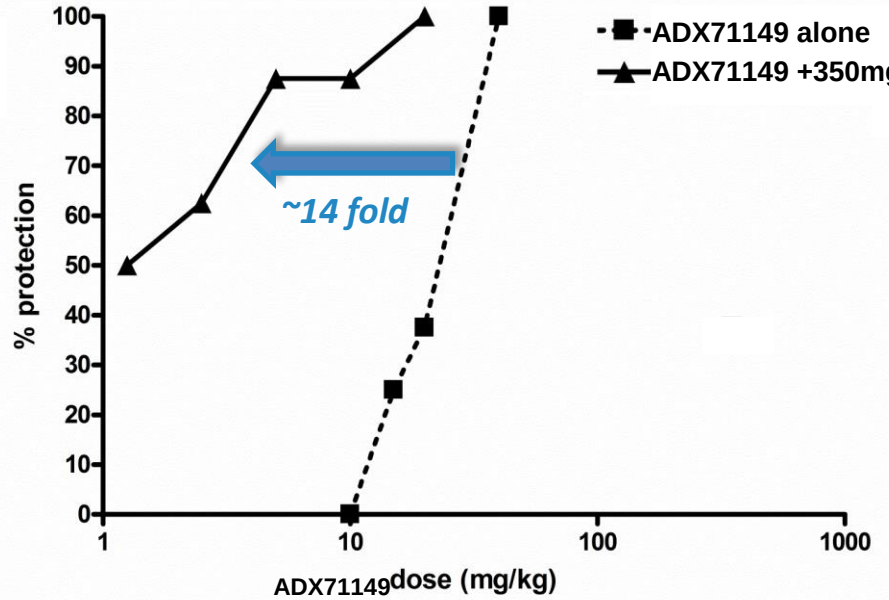
# ADX71149 Preclinical Efficacy in Epilepsy - 6Hz Model

## Validation in pharmaco-resistant mouse epilepsy model with high translational value:

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




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




- Keppra efficacy increased 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 specific only to SV2A antagonists

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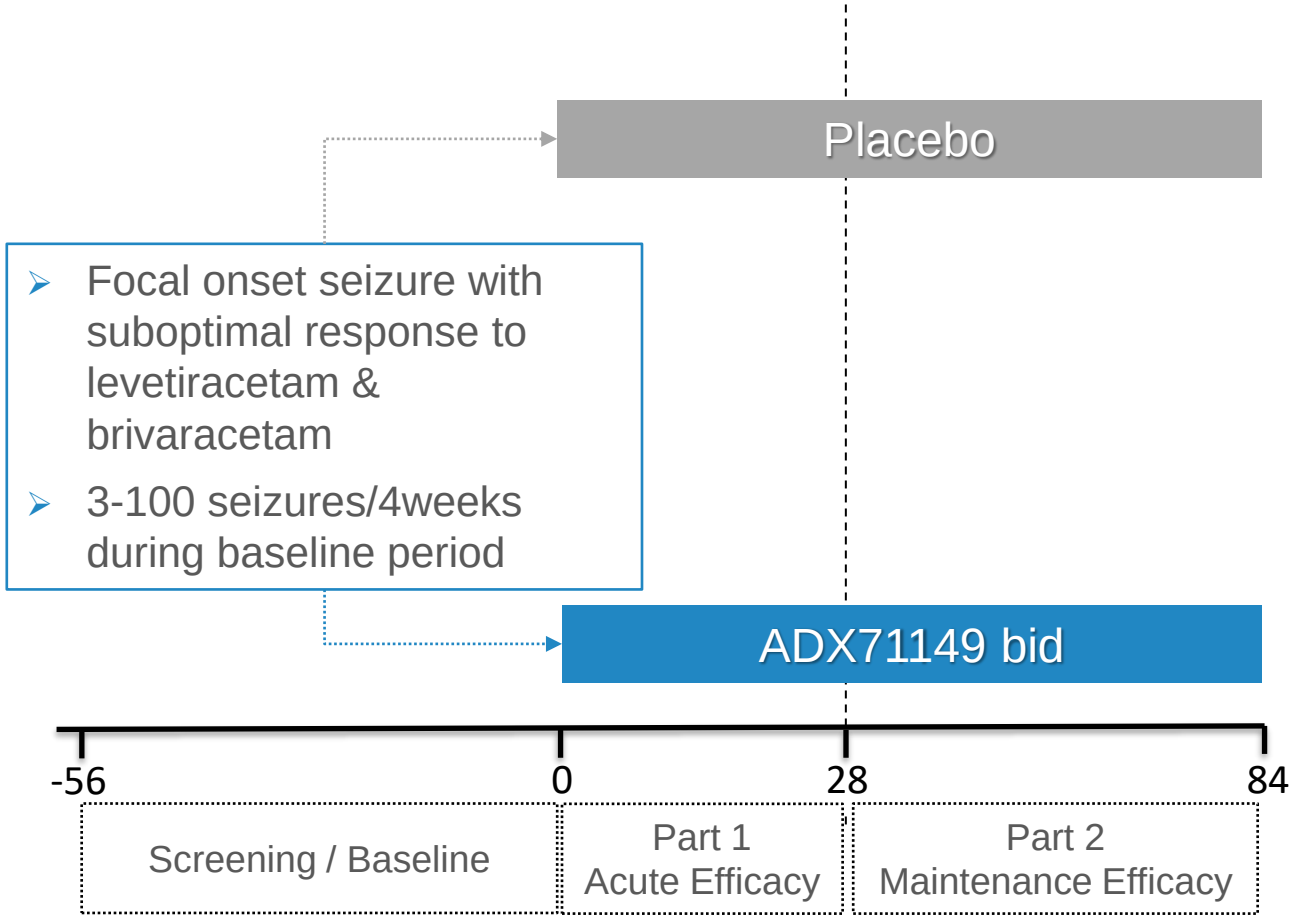
# ADX71149 Phase 2a Epilepsy Study




- Double blind placebo controlled
- Establish 28-day seizure count (over 56-day baseline period)
- Primary endpoint: time to monthly baseline seizure count
- Period 1: 4-week acute efficacy phase
- Period 2: 8-week maintenance efficacy phase
  - Subjects who do not reach or exceed their monthly baseline seizure count in Part 1 continue double-blind treatment during Part 2
- Evaluating 2 doses in 110 patients
- Cohort 1 & Cohort 2 completed study
- Top-line data did not show statistical significance – reported April 29, 2024
- Full data set being analyzed

**Full data set being analysed – expected in H2 2024**

*IRC = Independent interim review committee

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

## First-in-class program ready to start Phase 2b/3

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# Compelling Rationale to Develop Dipraglurant for PD-LID

- Large underserved patient population in need of improved treatment options
- Significant commercial opportunity with limited competition
  - 1M Parkinson's disease patients in US of which >170,000 have dyskinesia
  - Orphan drug designation granted for dipraglurant in US
  - GOCOVRI® price: $34K p.a., Nuplazid® price: $45K p.a.
  - US LID market estimated at $4B
- Strong mechanistic rationale for blocking mGlu5 to inhibit glutamate signalling
- Supportive pre-clinical data and Phase 2 clinical data
- PK profile ideally suited for treatment of LID
- Dipraglurant is active on same biological pathway as amantadine (inc. GOCOVRI®)
  - Decreases glutamatergic tone
  - Unlike amantadine, dipraglurant:
    - Restores synaptic plasticity to prune aberrant signalling
    - Highly selective with limited off target activity
- Novartis mGlu5 NAM (AFQ056) data supportive of mGlu5 target & rationale for dipraglurant PK profile

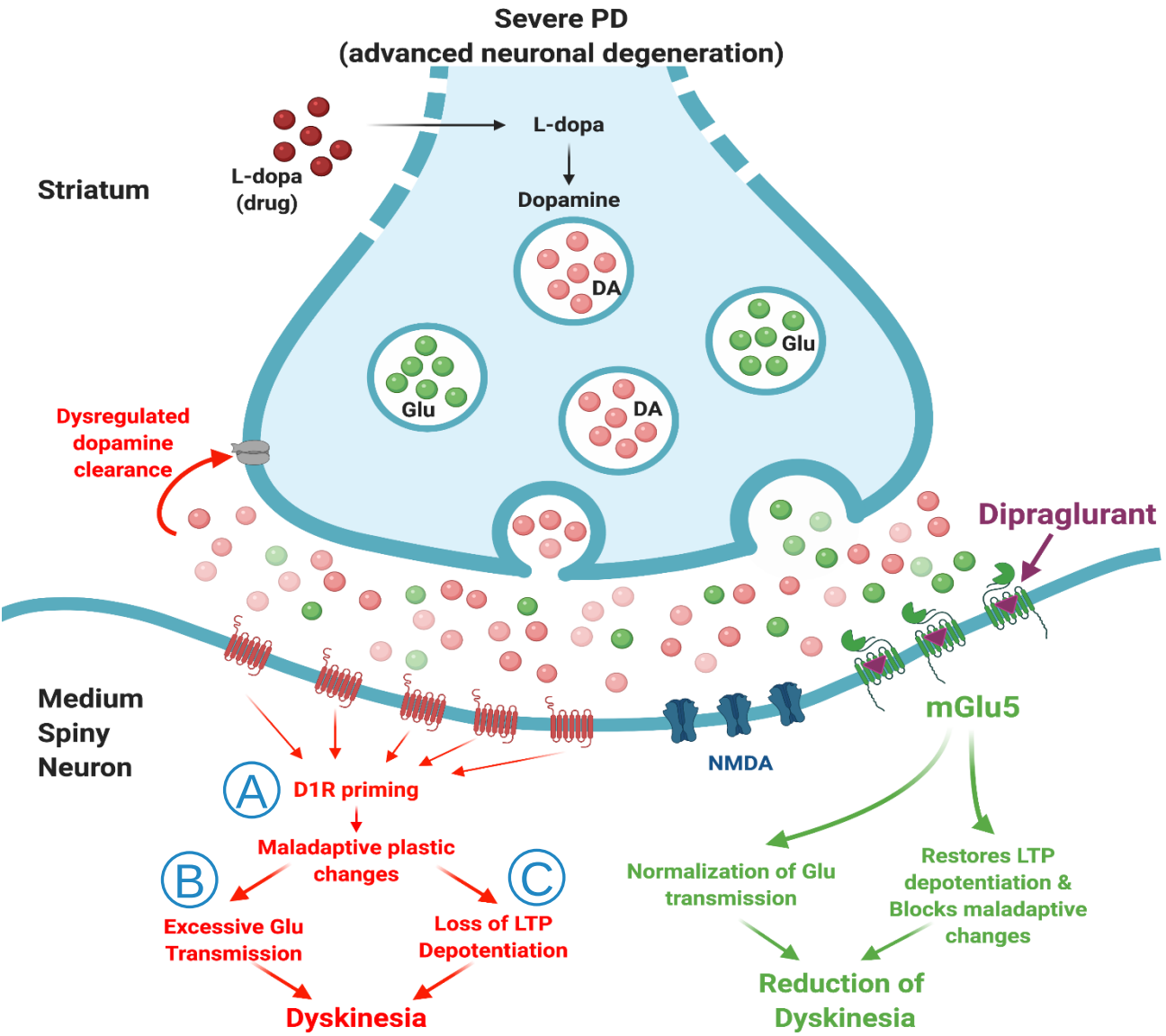


* www.drug.com

# Disability and Impact of PD-LID

| | |
|---|---|
| Invariably associated with long-term L-dopa use | ➢ Dyskinesias caused by neurodegeneration<br>➢ Dopamine replacement lowers the triggering threshold for symptoms<br>➢ LID can become as disabling as the PD symptoms themselves |
| Symptoms include dystonia, chorea, and choreoathetosis | ➢ Uncontrollable muscle contractions, twisting and writhing<br>➢ Painful and severely disabling<br>➢ Causes fatigue/exhaustion and increased risk for falls and injuries<br>➢ Social withdrawal, reduced quality of life and increased burden on caregiver |
| Prevalence related to disease duration | ➢ >40% of patients experience LID within 4-6 years of L-dopa treatment<br>➢ Increases to 90% after 9 -15 years<br>➢ Patients treated with next-generation L-dopa will still experience LID |
| PD drug efficacy wanes over time - exacerbated by emergence of LID | **Treatment becomes a balancing act requiring constant adjustments to ensure symptom control & minimize intolerable side effects** |

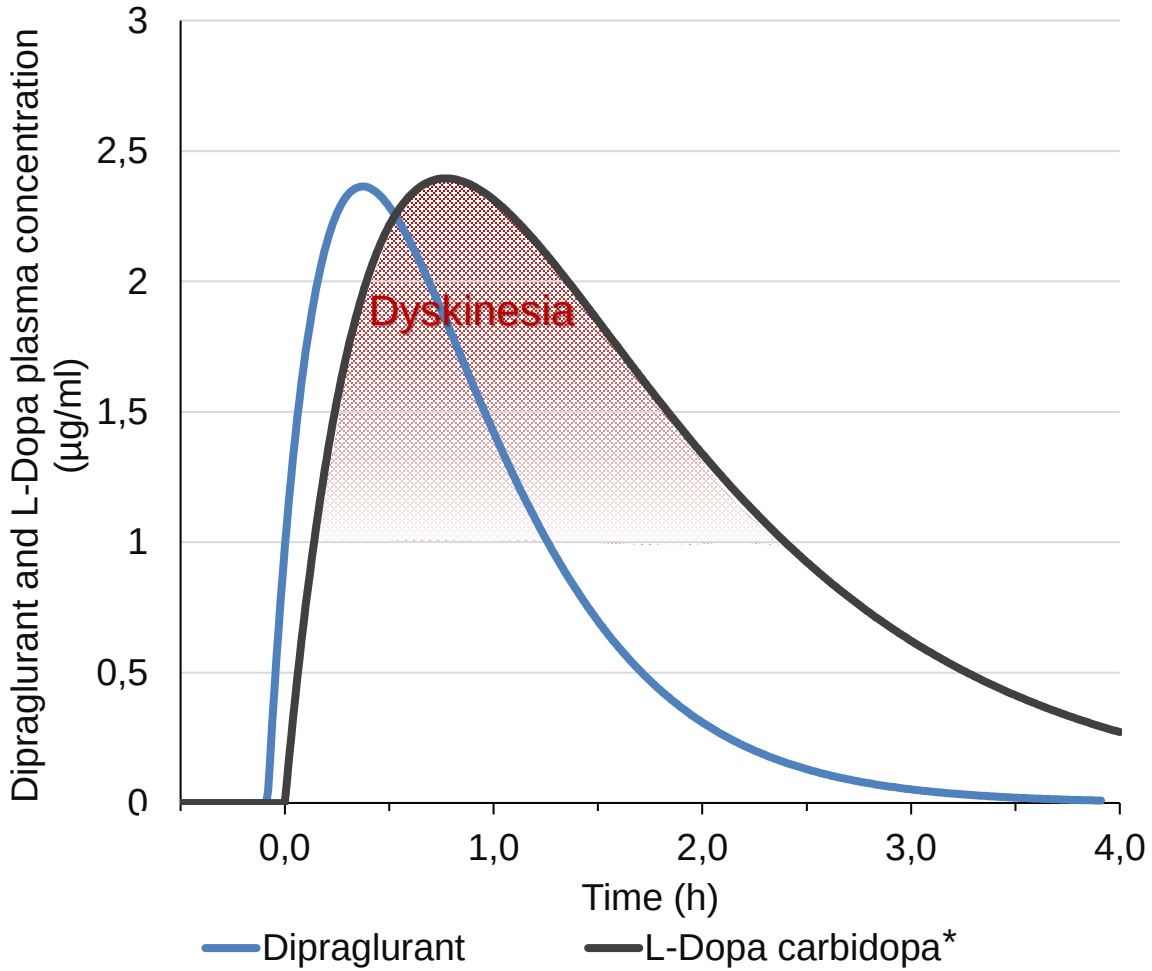
# MoA Rationale for Targeting mGlu5 Inhibition in PD-LID




- 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*
- mGlu5 receptor is an attractive target due to its modulatory action - normalizing glutamatergic activity and restoring LTP depotentiation
- Inhibiting mGlu5 decreases excess glutamatergic tone thereby controlling dyskinesia
- Dipraglurant is an oral, highly selective NAM of the mGlu5 receptor

LTP = Long Term Potentiation
D1R = D1 dopamine receptor
Glu = glutamate DA = dopamine

# Dipraglurant PK is a Key Advantage for Treating LID



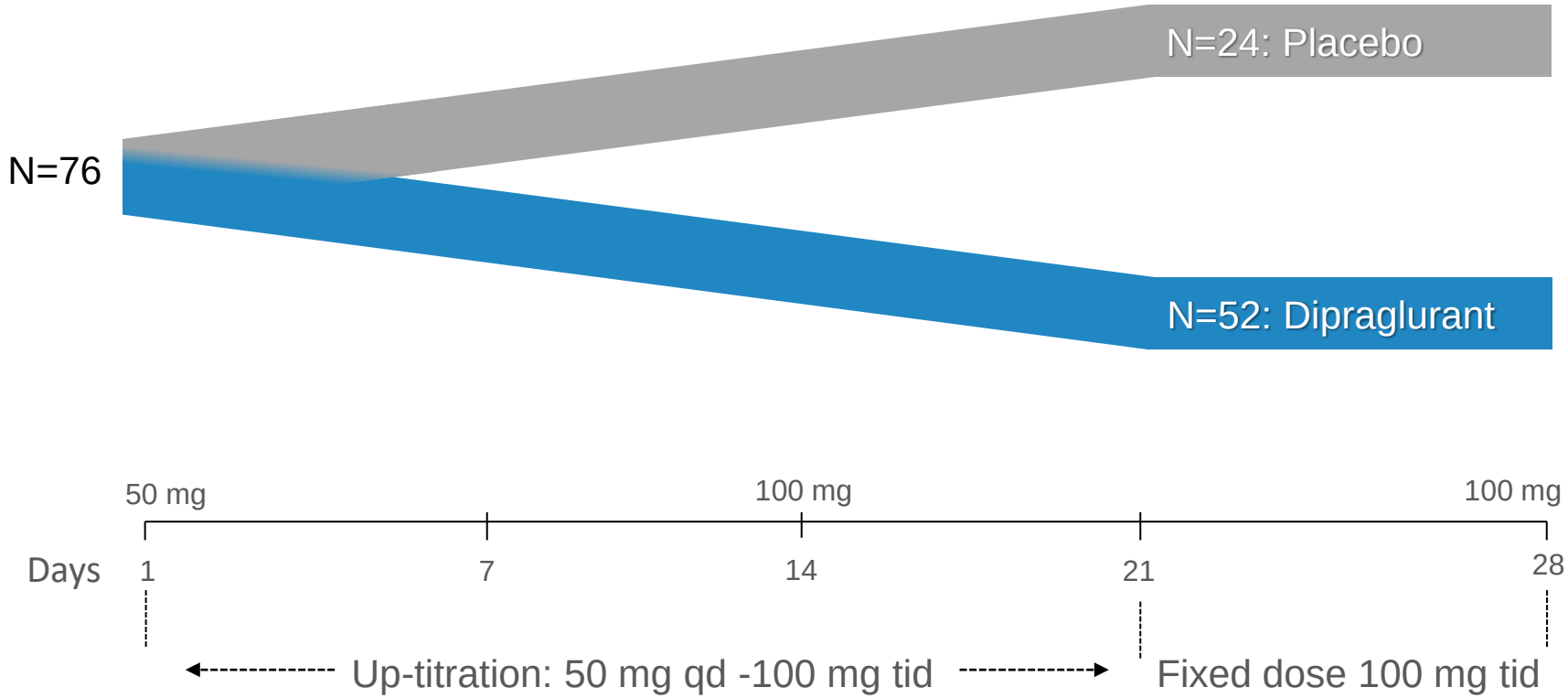

- Dyskinesia symptoms are correlated to peak levels of L-dopa
- PK of dipraglurant allows control of glutamatergic tone ahead of L-dopa Cmax

**Dipraglurant normalizes abnormal glutamate stimulation during peak levodopa dose**

Dipraglurant peaks ahead of L-dopa for optimal LID control

* Pharm Sci (www.cspsCanada.org) 20, 226 - 238, 2017

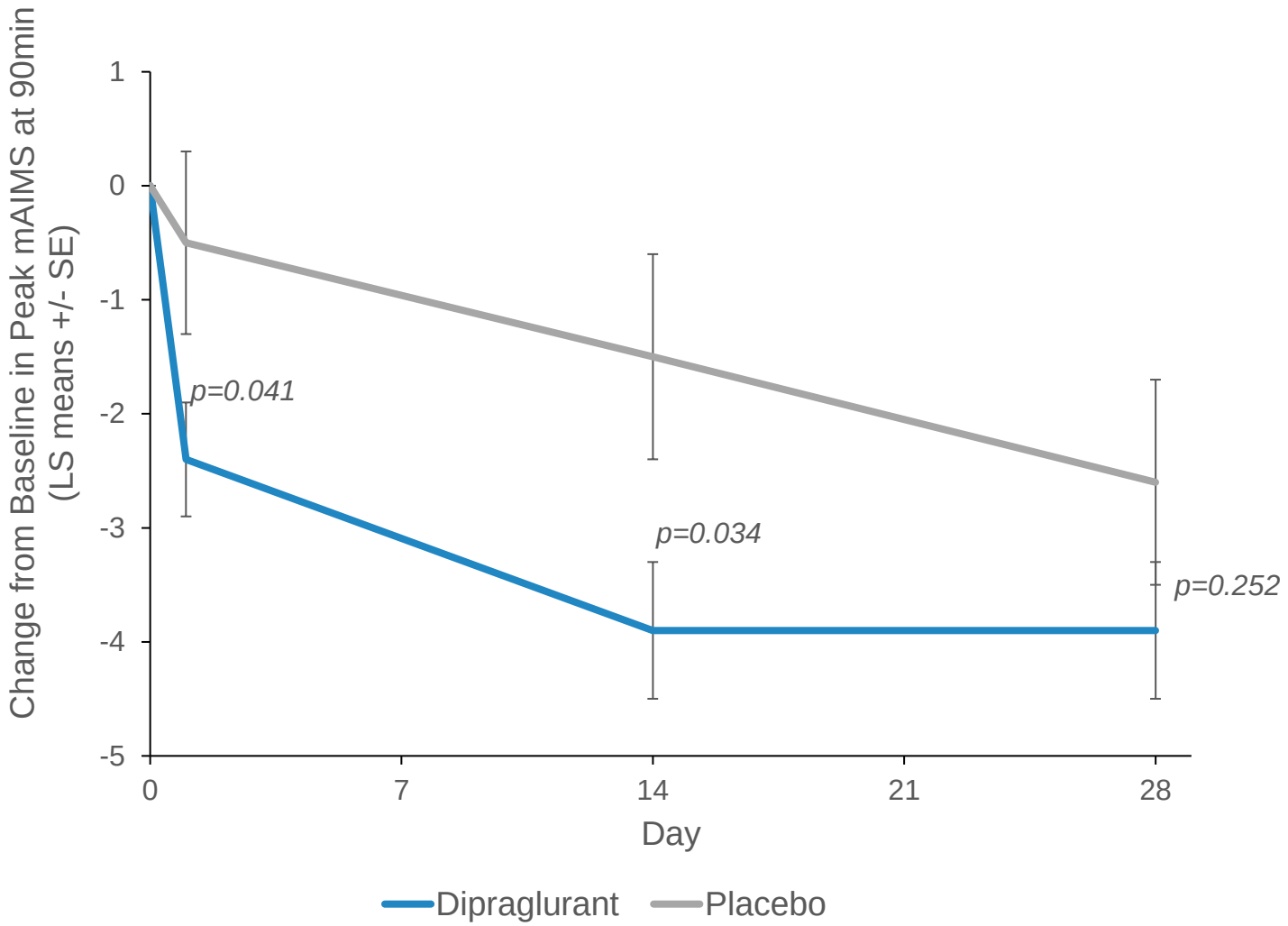
# Dipraglurant Phase 2a Study in LID (in US and Europe)




- **Primary objective:** safety & tolerability
- **Secondary objective exploratory efficacy:**
  - Modified Abnormal Involuntary Movement Scale (mAIMS) on days 1, 14 and 28
  - Clinician Global Impression of Change (CGIC)
  - Patient diaries of “On” & “Off” time

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

# Dipraglurant Improves LID by 30%

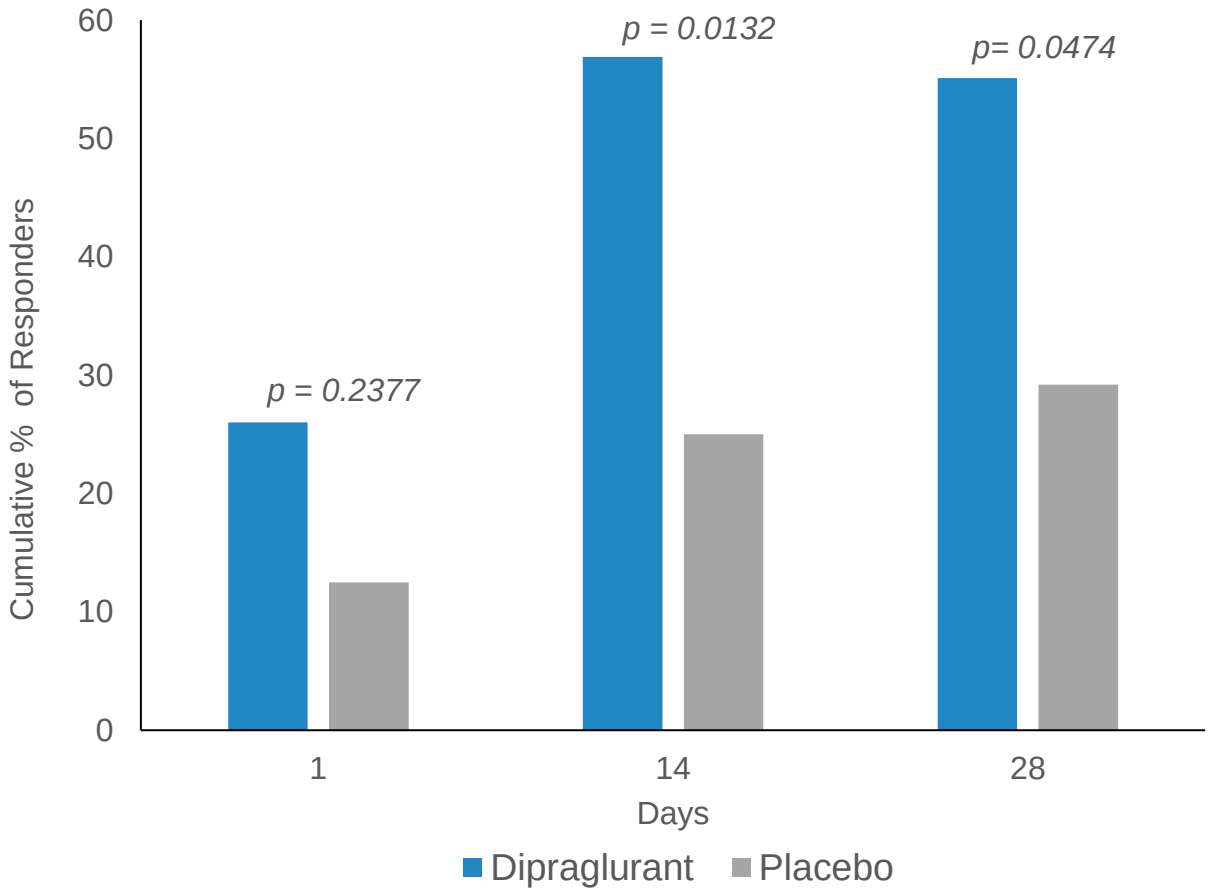



- Statistically significant effects: Day 1 (50mg) and Day 14 (100mg)
- Improvement maintained through Day 28
- Increasing placebo response caused significance to be lost at Day 28
- No placebo mitigation in study

| 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% |

# Responder Analysis Demonstrates Dipraglurant Significant Benefit

## Percent of patients with ≥ 30% improvement on mAIMS

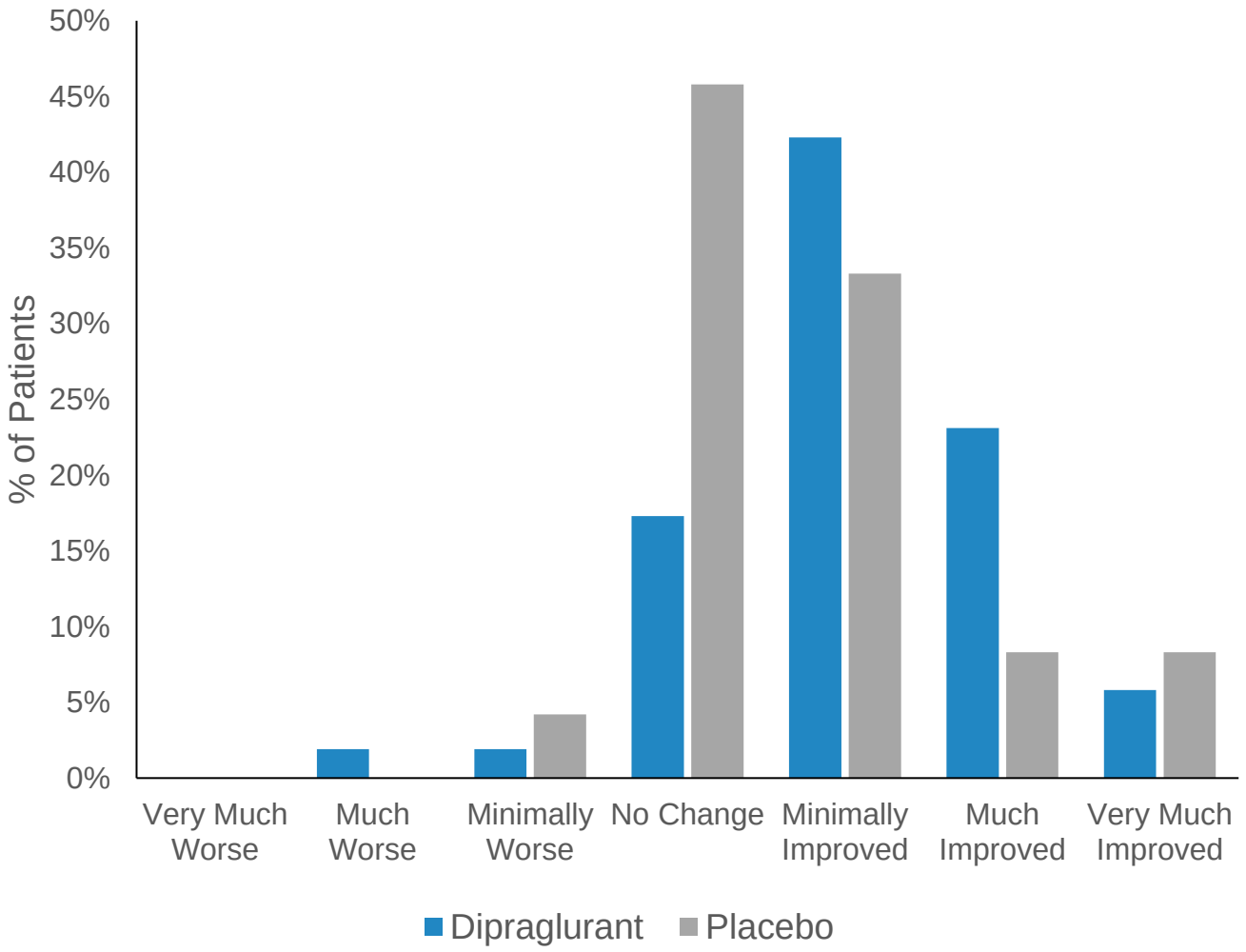



| Responder analysis (≥30% change of mAIMS from baseline) | | | | |
|---|---|---|---|---|
| Midday dose | Dipraglurant | | Placebo | |
| Day 1 (50 mg) | n=13 | 26.0% | n=3 | 12.5% |
| Day 14 (100 mg) | n=29 | 56.9%* | n=6 | 25.0% |
| Day 28 (100 mg) | n=27 | 55.1%* | n=7 | 29.2% |

*statistically significant

Reinforces robustness of dipraglurant anti-dyskinetic effect

# Significant Improvement on CGI-C




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

- Simple scale reflecting clinical assessment by treating physician
- More objective than mAIMS
- Assessed at end of study compared to baseline
- Supports use of UDysRS in pivotal program

# Dipraglurant Demonstrated Good Safety and Tolerability in PD Patients

- Adverse events common in both treatment groups (dipraglurant 88.5%, placebo 75%)
- Most common AEs:

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

* 3 of 11 AEs of “worsening dyskinesia” occurred in the follow up period (i.e., after drug discontinuation). On treatment incidence = 15.3% dipraglurant, 12.5% placebo

- AEs led to discontinuation in 2 patients (dipraglurant 100 mg)
- Fewer AEs at 50 mg (Weeks 1 and 2)  53% vs 58% placebo compared to 100 mg (Weeks 3 and 4) 73% vs 63% placebo
- No treatment effects on ECG, HR, BP, haematology and biochemistry

Safety profile supports continued development in PD-LID (KOLs and DSMB)

# Dipraglurant PD-LID - Development Status

- Fast onset of action and short half-life
  - Ideally suited normalizes abnormal glutamate stimulation during peak levodopa dose
- Extensively profiled Phase 1 studies
  - 5 studies with >100 patients, including receptor occupancy (PET ligand study)
- Phase 2 studies conducted
  - Safe and well tolerated in PD-LID patients with 7 patients exposed >6 months
  - Significant improvement in peak mAIMS from baseline (at days 1 &14)
  - Significant improvement seen in responder analysis
  - Significant Improvement on CGI-C
  - Pivotal registration study started in June 2021 and stopped in June 2022 due to poor enrolment related to COVID-19
- CMC Status
  - >30kg API in stock & 7.5kg drug product available in 50mg and 100mg tablets with placebo
- IP
  - Patent through 2034 (without extensions)
  - Use patent of mGlu5 NAMs in treatment of brain damage until 2035 – option to exclusive license

First-in-class program for PD-LID ready to start Phase 2b/3

# Dipraglurant (mGlu5 NAM) for Post-Stroke Recovery

*Targeting neuroplasticity early in rehabilitation to promote recovery*

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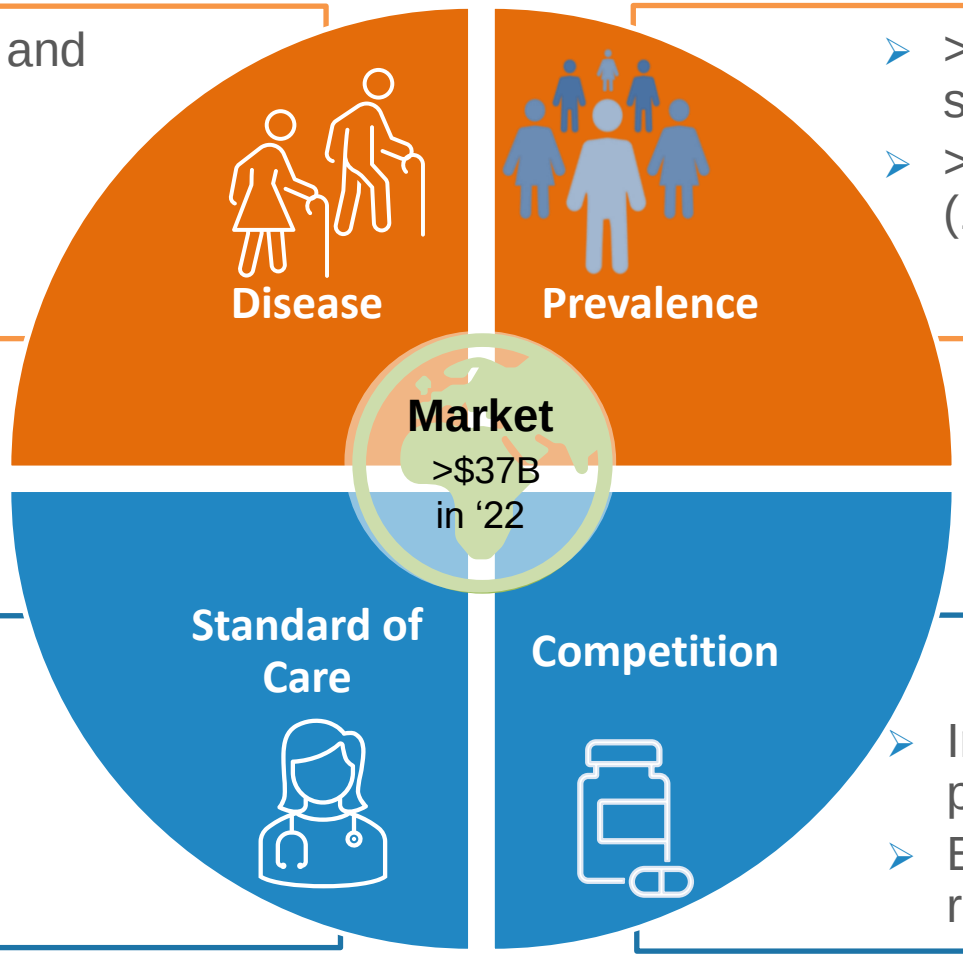
# Post Stroke Recovery - Unmet Medical Need & Commercial Opportunity

- Post stroke many suffer motor and somatosensory impairment
- Potential chronic disability & low quality of life




- >101 million ischemic stroke survivors worldwide[1]
- >12 million new strokes/yr (1 every 3 seconds)[1]

- Rehabilitation therapies: e.g. physiotherapy, speech, communication and cognitive
- Majority never fully recover

- Intense early rehabilitation promotes recovery
- But, no approved drugs to support rehabilitation therapies

Urgent medical need to promote sensorimotor recovery in post-stroke patients

[1]World Stroke Association 2022

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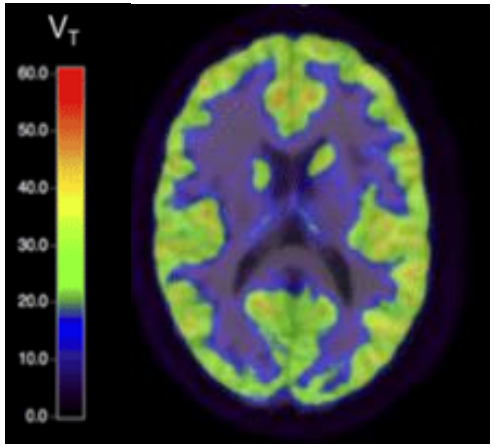
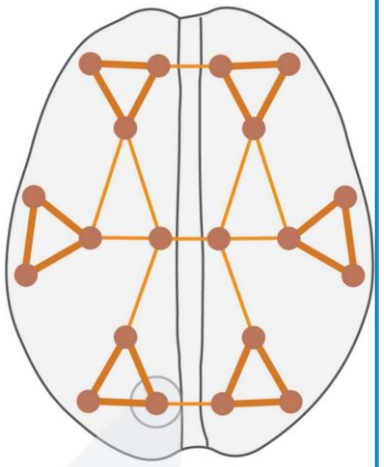
# mGlu5: An Innovative Target for Post-Stroke Recovery

## Healthy brain

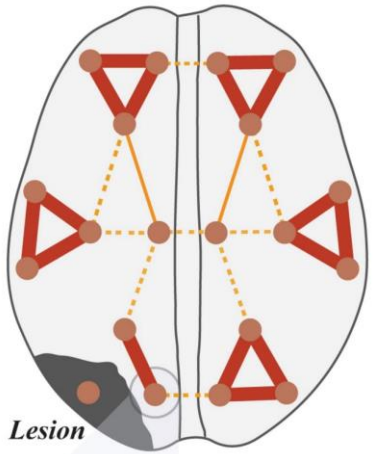



mGlu5 brain distribution



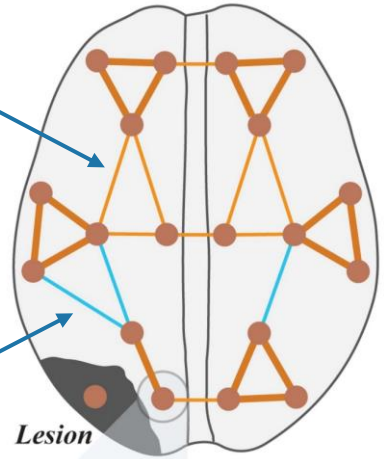
Good inter & intra-nodal connectivity

## mGlu5 NAM supports post-stroke recovery




Recovery

+ mGlu5 NAM




### mGlu5

- Densely expressed in the brain
- Involved in neural plasticity
- Modulates excitation/inhibition equilibrium

### Lesion effects:

- Confined neural tissue necrosis
- Network disruption & module segregation
- Imbalance in excitation/inhibition

### mGlu5 NAM promotes synaptic plasticity

- Cortical reorganization & new functional pathways (a)
- Connectivity changes toward pre-lesion state (b)
- Restoration of excitation/inhibition equilibrium

Adapted from DOI: 10.3389/fnsys.2021.806544

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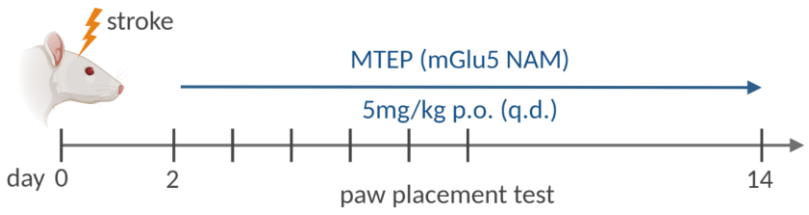
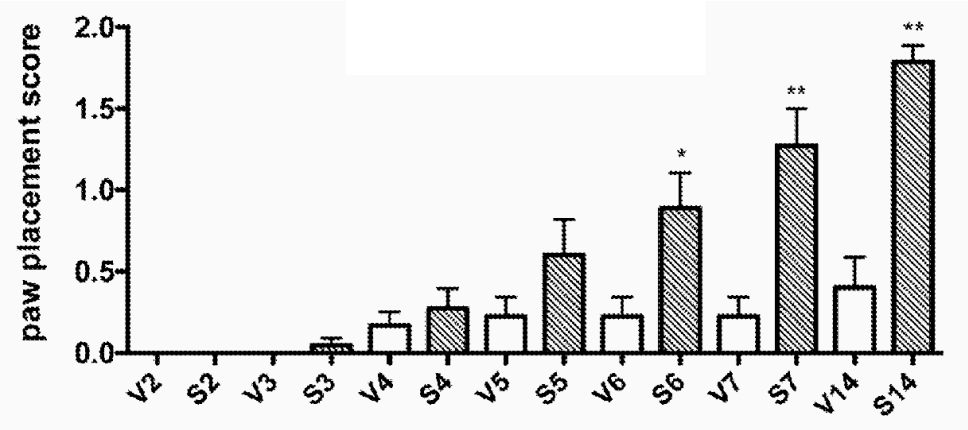
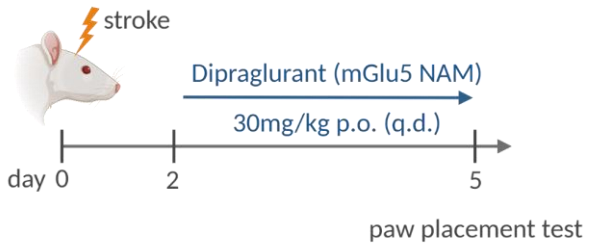
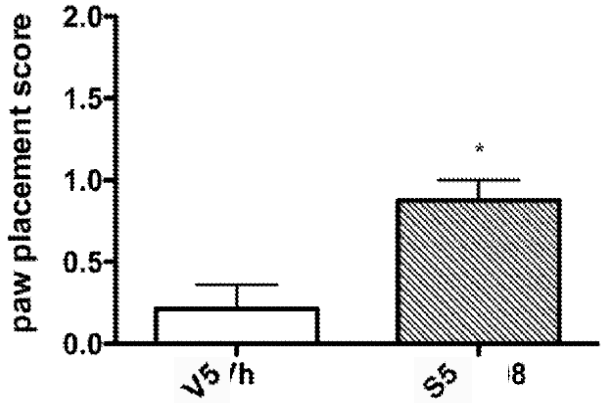
# Preclinical Data: mGlu5 Inhibition Promotes Post-Stroke Recovery




MTEP treatment resulted in a significant recovery of functional control



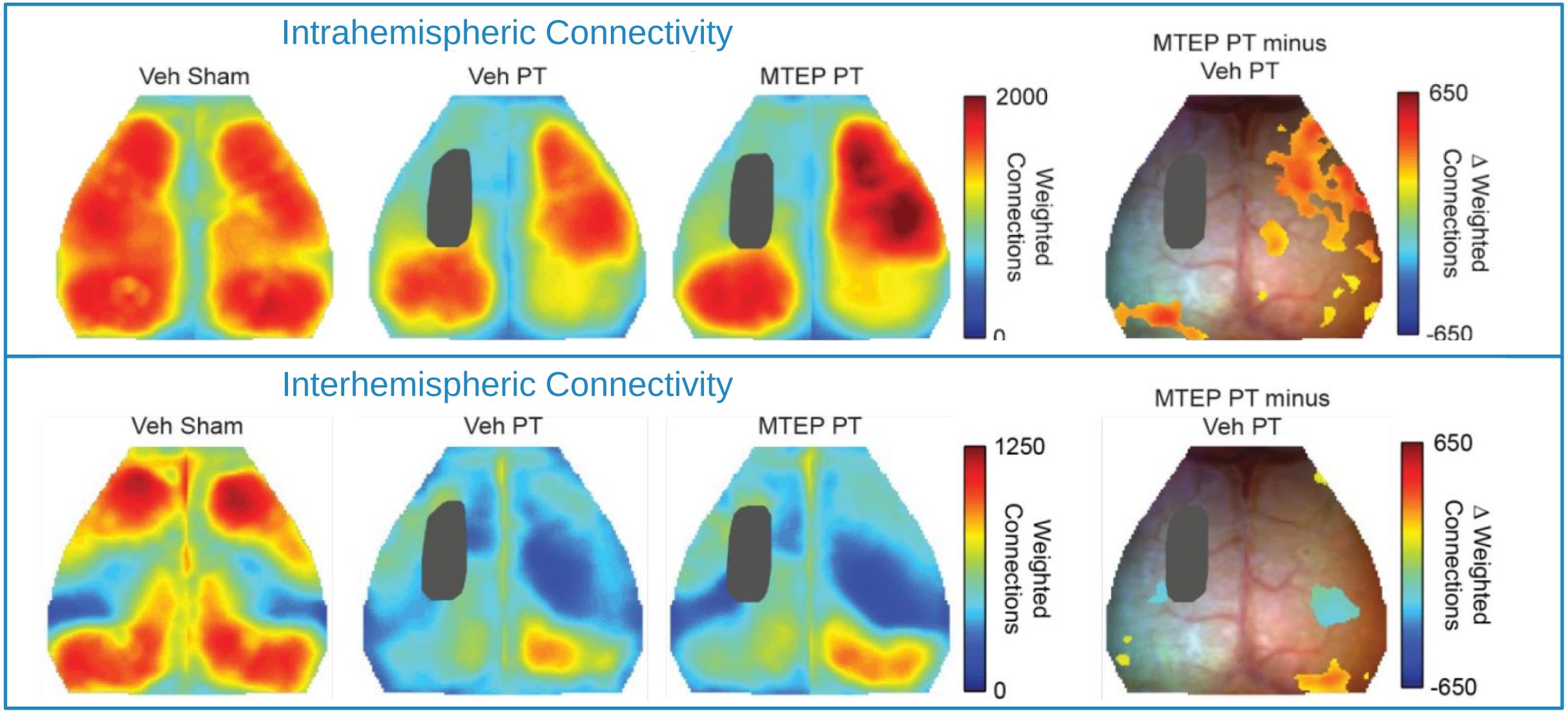




Dipraglurant significantly restored functional control in a one-week experiment

## Dipraglurant enhanced functional brain recovery in a rat model of experimental stroke

From patent application PCT WO2017/171594A1

# MRI Imaging Data: Post-Stroke Resting State Functional Connectivity




mGlu5 inhibitors promote intra- and inter-hemispheric connectivity following stroke

https://doi.org/10.1093/brain/awad293

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# Dipraglurant for Post-stroke/TBI Recovery - Development Status

- Fast onset of action and short half-life
  - Ideally suited for concurrent dosing with rehabilitation
- Extensively profiled Phase 1 studies
  - 5 studies with >100 patients
  - Including receptor occupancy (PET ligand study)
- Phase 2 studies conducted
  - Safe and well tolerated in patients suffering from neurological disease – Parkinson's disease
  - Mild to moderate CNS type AEs at doses < 200mg
  - 7 PD-LID patient exposed >6 months
- CMC Status
  - >30kg API in stock
  - 7.5kg drug product available in 50mg and 100mg tablets with placebo
- IP
  - Patent through 2034 (without extensions)
  - Use patent of mGlu5 NAMs in treatment of brain damage until 2035 – option to exclusive license

First-in-class program for post-stroke recovery ready to start Phase 2

# GABAB PAM for Substance Use Disorders (Indivior Partnership)

*Going beyond baclofen to treat substance use disorders with improved safety and tolerability*

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# GABAB PAM for Substance Use Disorder

| | |
|---|---|
| Large market & unmet medical need | ➢ High prevalence;1.8% of US population[1]<br>➢ Current treatments have undesirable side-effects and prone to relapse<br>➢ Burden to society in US is >$600B annually[2] |
| Clinically validated MoA | ➢ Baclofen (GABAB agonist) used off label for alcohol use disorder<br>➢ ADX71441 attenuates alcohol self-administration and relapse to alcohol seeking in rats[3] and alcohol consumption in mice[4]<br>➢ ADX71441 reduces cocaine self-administration in non-human primates[5] |
| Status of program and near-term milestone | ➢ Addex is executing Indivior funded GABAB PAM research program<br>➢ Multiple compounds in late clinical candidate selection phase<br>➢ Differentiated leads and backups with robust novel IP potential<br>➢ IND enabling studies expected to start in H2 2024 |
| Strategic partnership with Indivior | ➢ Eligible to receive $330 million in milestones and tiered royalties from high single digits to low double digits<br>➢ Conducting a funded research program to discover novel GABAB PAMs<br>– Right to select compounds for development in reserved indications |

[1]Merikangas et al. 2010
[2]NIDA
[3]Augier et al 2017
[4]Hwa et al 2014
[5]Addex int. report

# GABAB PAM for Chronic Cough

*Going beyond baclofen to treat cough with improved safety and tolerability*

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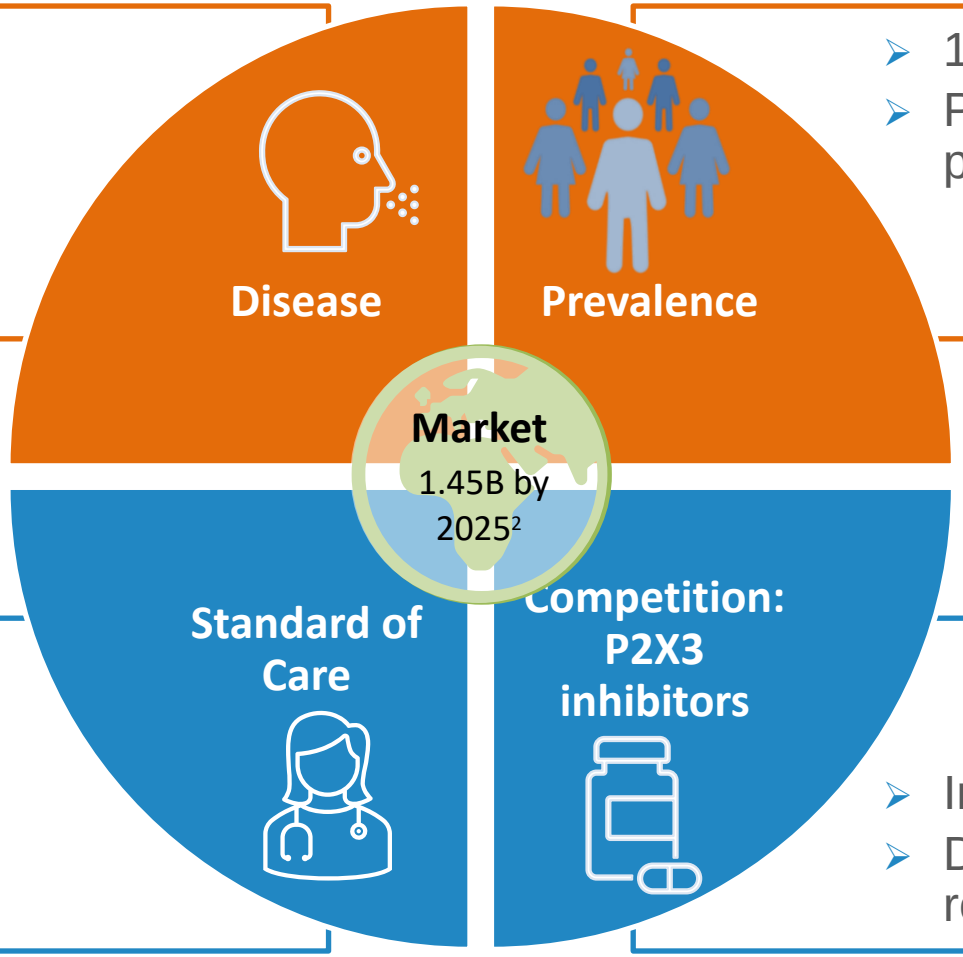
# Cough - Unmet Medical Need and Commercial Opportunity

- Chronic cough
  - Lasting > 8 weeks[1]
  - >700 coughs/day
- Multiple comorbidities




- 10% in the general population[4]
- Persists >1 year in 60% of patients

- Ineffective in 30% of patients[3]
- Hindered by side-effect risks

- Ineffective in ~30% of patients
- Discontinuation in ~20% (taste related side-effects)

High unmet medical need for an efficacious and safe treatment of cough

[1]Morice et al. *Eur Respir Rev* 2021
[2]Cough Remedies Market Share, Size and Industry Growth Analysis 2021 - 2026 (industryarc.com)
[3]Ryan *Expert Opin Pharmacother* 2018
[4] Song WJ, Chang YS, Faruqi S, et al. 2015.

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# Standard of Care in Cough - Strengths and Weaknesses

| Use / side-effects | Dextro-metorphan | Opioids | Gabapentin & pregabalin | Amitriptyline | P2X3* | GABAB Agonist Baclofen | GABAB Addex PAM |
|---|---|---|---|---|---|---|---|
| Treatment type | Chronic | Acute | Acute | Acute | Chronic | Acute | Chronic |
| Risk of Abuse | Yes | Yes | Yes | Yes | No | No | No |
| Respiratory | No | Yes | Yes | Yes | No | Yes | No |
| Other CNS | Yes | Yes | Yes | Yes | No | Yes | No |
| Gastrointestinal | Yes | Yes | No | No | No | No | No |
| Taste-related | No | No | No | No | Yes** | No | No |

- P2X3 inhibitor Gefapixant
  - * Ineffective in 30% of patients
  - ** Taste-related side effects observed in up to 97% of patients, leading to discontinuation in up to 20% of patients[1]

A highly selective and targeted GABAB PAM has the potential to offer best-in-disease efficacy and tolerability profile suitable for chronic treatment

[1]Niimi et al. *Allergology International* 2022

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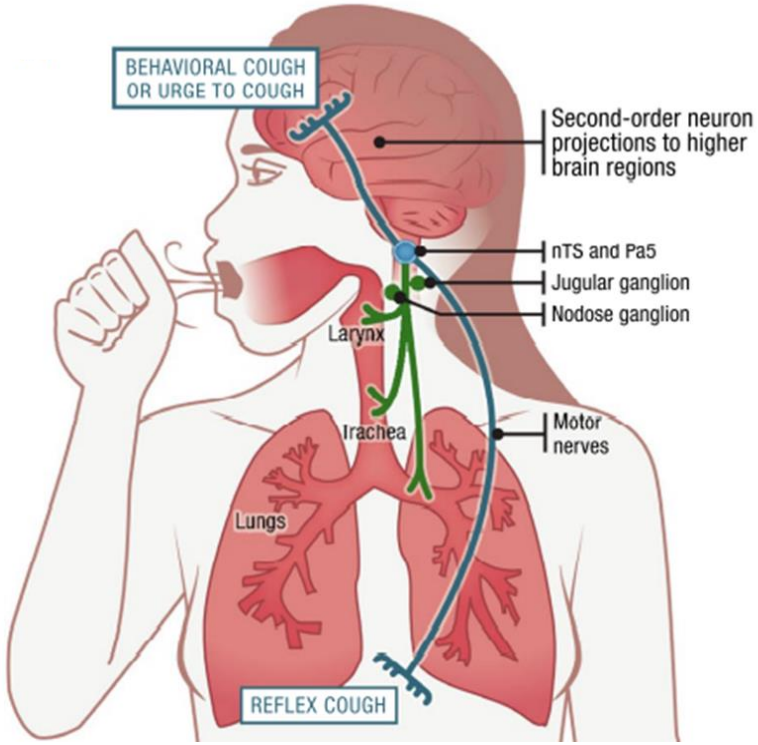
# GABAB Receptor - Validated Target in Cough

- GABAB receptor
  - Expressed throughout the cough neural circuit
  - Activation reduces neuronal excitability
  - Potential for broad application in cough patients
- Baclofen, an orthosteric agonist
  - Used off-label in patients with chronic cough
  - Clinical studies with cough patients showed efficacy
  - Efficacious in healthy volunteer and multiple preclinical models
- Selective GABAB PAM
  - Differentiated pharmacology
  - Improved efficacy and tolerability demonstrated in preclinical models
  - Absence of receptor desensitization with chronic treatment




The anatomical mediators of cough (1)

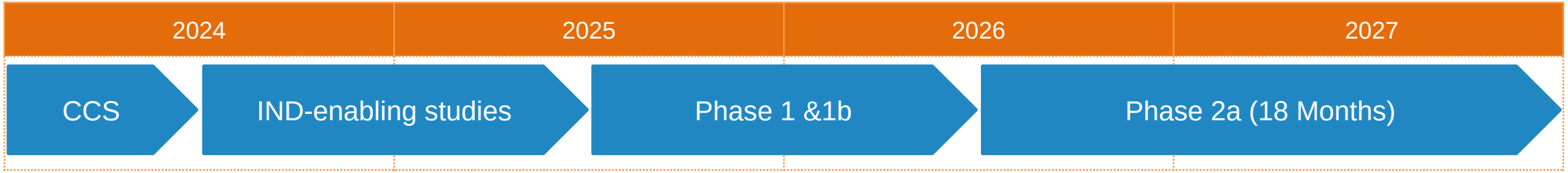
GABAB PAM offers potential for improved treatment for cough patients

[1]Mazzone and McGargey *Clin Pharm Ther* 2021

# Target Product Profile, Project Status and Development Plan

- A first-in-class GABAB PAM to treat Cough
  - Once-a-day oral dosing suitable for chronic treatment
  - Reduction in cough bouts and severity and improvement in quality of life
  - Superior tolerability with no taste related side-effects
- Status of program: clinical candidate selection
  - Potent, highly selective compounds with good developability properties identified
  - In vivo PoC with PK/PD confirmed in multiple preclinical models of cough with comparable efficacy to P2X3 inhibitors

## Development Plan




On track for first-in-human studies in 2025

Timings beyond CCS are subject to funding/partnering

# 20% Equity Interest in Neurosterix

*Well funded preclinical portfolio of high value assets*

# Neurosterix

- Addex spin-out company
  - Series A funding of $63 million in April 2024 led by Perceptive Advisors
  - Addex contributed allosteric modulator drug discovery platform and portfolio of preclinical programs
  - Addex received CHF5 million and a 20% equity interest
- High value pipeline advancing toward the clinic:
  - M4 PAM for schizophrenia
    - Clinically validated target
    - IND enabling studies expected to start in H2 2024
  - mGlu7 NAM for stress related disorders
    - First-in-class program
    - IND enabling studies expected to start in H2 2024
  - mGlu2 NAM for mild neurocognitive disorders
    - Progressing through lead optimization

Multiple high value programs funded to significant milestones

# Addex Financials and Stock

# Financials and Stock

- Cash at March 31, 2024:
  CHF 1.6M (USD 1.8M)
  - CHF 5M from sale of Neurosterix received in April 2024
- No debt
- Traded on SIX Swiss Exchange: ADXN (ISIN:CH0029850754)
- ADS representing 120 shares traded on Nasdaq: ADXN (ISIN: US00654J206; CUSIP: 00654J206)

- 128.26 M outstanding shares
  - Armistice Capital LLC – 26.13%*
  - New Enterprise Associates – 3.03%*
- 184.35M shares incl. treasury shares (254.03M fully diluted)
  - Management & board holds – 13.47%*
- Analyst coverage:
  - HC Wainwright - Raghuram Selvaraju
  - valuationLab - Bob Pooler
  - Baader Helvea AG - Leonildo Delgado
  - ZKB – Edouard Riva

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* Percentage of ownership calculated based on shares and warrants divided by fully diluted shares

# Summary

| | |
|---|---|
| Multiple high value partnerships | ➢ ADX71149 (mGlu2PAM) epilepsy Phase 2a – top-line results not statistically significant<br>➢ GABAB PAM for substance use disorder (Indivior) in clinical candidate selection<br>➢ 20% equity interest in Neurosterix (backed by Perceptive Advisors) |
| In house programs driving future value | ➢ Dipraglurant - PD-LID Phase 2b ready to start<br>➢ Dipraglurant - post-stroke recovery Phase 2a ready to start<br>➢ GABAB PAM for chronic cough in clinical candidate selection |
| Solid foundation | ➢ Partnerships with industry leaders - JnJ & Indivior<br>➢ Top tier US investors - Armistice Capital, NEA and NLV<br>➢ Dual listed SIX Swiss exchange & US Nasdaq<br>➢ Strong balance sheet and cash runway through 2026 |
| Promising outlook | ➢ GABAB PAM - start IND enabling studies in H2 2024<br>➢ Dipraglurant Phase 2 ready to start Phase 2 in PD-LID &/or post-stroke recovery<br>➢ Neurosterix lead program - M4 PAM<br>– IND enabling studies expected to start H2 2024 |

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# ALLOSTERIC MODULATORS FOR HUMAN HEALTH