Innovative Treatments for Central Nervous System Disorders

January 2022
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## Addex Overview

| 3 clinical programs underway | • Phase 3 Parkinson’s disease dyskinesia study – data end of Q4 2022  
|                            | • Phase 2 blepharospasm study – data end of Q1 2022  
|                            | • Phase 2 epilepsy study (J&J) – data Q3 2022 |
| Leading allosteric modulator technology platform | • Validated & differentiated pharmacological approach  
|                                              | • Proprietary biological screening assays and chemical library  
|                                              | • Track record of delivering novel drug candidates |
| In house discovered pipeline | • Significant intellectual property portfolio  
|                                | • Multiple novel drug candidates entering clinical candidate selection  
|                                | • Driving long term growth & future partnership opportunities |
| Partnerships with industry | • J&J - €109M in milestones & double digit royalties  
|                          | • Indivior - $330M in milestones, royalties up to double digit & funded research program |
| Top tier US investors | • Dual listed on SIX Swiss Exchange & US Nasdaq Capital Market  
|                        | • Cash of CHF15.5M (USD16.6M) at 30 September 2021  
|                        | • Sale of treasury shares in December 2021 raised USD10M |
# Addex Pipeline - 3 Clinical Programs Underway

<table>
<thead>
<tr>
<th>Molecule / MoA</th>
<th>Indication</th>
<th>Partner</th>
<th>Pre-clinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Milestone</th>
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<tbody>
<tr>
<td>Dipraglurant</td>
<td>PD-LID</td>
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<td>Data end of Q4 2022</td>
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<tr>
<td>(mGlu5 NAM)</td>
<td>Blepharospasm</td>
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<td>Data end of Q1 2022</td>
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<tr>
<td>ADX71149</td>
<td>Epilepsy</td>
<td>janssen</td>
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<td>Data in Q3 2022</td>
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<tr>
<td>(mGlu2 PAM)</td>
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<tr>
<td>GABA&lt;sub&gt;B&lt;/sub&gt; PAM</td>
<td>Substance use disorder</td>
<td>INDIVIOR</td>
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<td>CMT1A</td>
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<td>mGlu7 NAM</td>
<td>PTSD</td>
<td>eurostars</td>
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<tr>
<td>mGlu2 NAM</td>
<td>Mild neurocognitive disorders</td>
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<tr>
<td>mGlu4 PAM</td>
<td>Parkinson’s disease</td>
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<tr>
<td>mGlu3 PAM</td>
<td>Neurodegenerative disorders</td>
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**Lead Program Started US Pivotal Study**

NAM = Negative Allosteric Modulator
PAM = Positive Allosteric Modulator
## Experienced Team

### Leadership Team

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Experience</th>
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</thead>
<tbody>
<tr>
<td><strong>Tim Dyer</strong></td>
<td>CEO / CFO</td>
<td>Co-Founder of Addex, Formerly with PwC, UK Chartered Accountant</td>
</tr>
<tr>
<td><strong>Dr Roger Mills</strong></td>
<td>Chief Medical Officer</td>
<td>Developed Nuplazid for PD Psychosis, &gt;30 years Pharma industry incl. Pfizer, Gilead and Acadia</td>
</tr>
<tr>
<td><strong>Dr Robert Lutjens</strong></td>
<td>Head of Discovery Biology</td>
<td>Member of Addex founding team, Formerly with Glaxo &amp; Scripps Research Institute</td>
</tr>
<tr>
<td><strong>Dr Jean-Philippe Rocher</strong></td>
<td>Head of Discovery Chemistry</td>
<td>Member of Addex founding team, Formerly with Pierre Fabre, GSK and Mitsubishi</td>
</tr>
<tr>
<td><strong>Dr Mikhail Kalinichev</strong></td>
<td>Head of Translational Science</td>
<td>Neuropharmacologist with &gt;20 years experience, Formerly with Ipsen, Lundbeck and GlaxoSmithKline</td>
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### Non-executive Directors

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<thead>
<tr>
<th>Name</th>
<th>Position</th>
<th>Experience</th>
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<tbody>
<tr>
<td><strong>Vincent Lawton</strong></td>
<td>Chairman</td>
<td>Former European Head of Merck &amp; Co, Former MHRA Board member</td>
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<tr>
<td><strong>Ray Hill</strong></td>
<td>Board member</td>
<td>Former Executive Director Merck &amp; Co.</td>
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<tr>
<td><strong>Jake Nunn</strong></td>
<td>Board member</td>
<td>Venture advisor and former Partner at New Enterprise Associates</td>
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<tr>
<td><strong>Isaac Manke</strong></td>
<td>Board member</td>
<td>General Partner at Acorn Bioventures, Formerly Partner at New Leaf Venture Partners</td>
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</table>

### Scientific Advisory Board

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<thead>
<tr>
<th>Name</th>
<th>Position</th>
<th>Experience</th>
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<tbody>
<tr>
<td><strong>Darryle Schoepp</strong></td>
<td>Chairman of SAB</td>
<td>Former leader of Neuroscience research department at Eli Lilly, and at Merck was Neuroscience research therapeutic area leader</td>
</tr>
<tr>
<td><strong>Mark Bear</strong></td>
<td>Picower Prof. of Neuroscience at MIT</td>
<td>Formerly on faculty of Brown University School of Medicine and an Investigator of the Howard Hughes Medical Institute</td>
</tr>
<tr>
<td><strong>Peter Bernstein</strong></td>
<td>Principal, PhaRmaB LLC</td>
<td>Formerly with ICI Astra Zeneca Awarded numerous accolades including Fellow of the American Chemical Society</td>
</tr>
<tr>
<td><strong>Benny Bettler</strong></td>
<td>Biomedicine Prof. at Basel University</td>
<td>Formerly at Novartis and discovered allosteric modulators at GABA&lt;sub&gt;g&lt;/sub&gt; receptor and recipient of the Peter Speiser Award</td>
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Dipraglurant for Levodopa-Induced Dyskinesia in Parkinson’s Disease (PD-LID)
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
  - 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 (mavoglurant) 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  
• Dopamine replacement lowers the triggering threshold for symptoms  
• LID can become as disabling as the PD symptoms themselves |
|-----------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Symptoms include dystonia, chorea, and choreoathetosis | • Uncontrollable muscle contractions, twisting and writhing  
• Painful and severely disabling  
• Causes fatigue/exhaustion and increased risk for falls and injuries  
• 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  
• Increases to 90% after 9-15 years  
• 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 |
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:

- D1 receptor priming
- Excess glutamate transmission
- 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.
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
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

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<tr>
<th>Mean % change of peak mAIMS from baseline</th>
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<tr>
<td>Midday dose</td>
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<tr>
<td>Day 1 (50 mg)</td>
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<tr>
<td>Day 14 (100 mg)</td>
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<tr>
<td>Day 28 (100 mg)</td>
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Responder Analysis Demonstrates Dipraglurant Significant Benefit

Percent of patients with ≥ 30% improvement on mAIMS

Reinforces robustness of dipraglurant anti-dyskinetic effect
Significant Improvement on CGI-C

- 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

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<thead>
<tr>
<th></th>
<th>Dipraglurant</th>
<th>Placebo</th>
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<tbody>
<tr>
<td>Improved (p&lt;0.05)</td>
<td>71.2%</td>
<td>49.9%</td>
</tr>
<tr>
<td>No change</td>
<td>17.3%</td>
<td>45.8%</td>
</tr>
</tbody>
</table>

% of Patients

Dipraglurant | Placebo

Very Much Worse | Much Worse | Minimally Worse | No Change | Minimally Improved | Much Improved | Very Much Improved

0% | 5% | 10% | 15% | 20% | 25% | 30% | 35% | 40% | 45% | 50%
Dipraglurant Demonstrated Good Safety and Tolerability in PD Patients

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

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<tr>
<th></th>
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<tbody>
<tr>
<td>Worsening Dyskinesia</td>
<td>21% (15.3%*)</td>
<td>12.5%</td>
</tr>
<tr>
<td>Dizziness</td>
<td>19%</td>
<td>12.5%</td>
</tr>
<tr>
<td>Nausea</td>
<td>19%</td>
<td>0%</td>
</tr>
<tr>
<td>Fatigue</td>
<td>15%</td>
<td>4%</td>
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</table>

* 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 Registration Program

• Pivotal registration program ongoing

• Study 301 started in June 2021
  – Data read-out expected end of Q4 2022
  – Primary endpoint: UDysRS
  – Placebo mitigation is a priority

• 12-month Open Label Study (302) on going
  – 6- and 12-month safety data

• Second pivotal registration study (303) to follow study 301 completion
Dipraglurant Pivotal PD-LID Study (301)

- Primary objective: Efficacy in reducing LID
  - UDysRS change from baseline at 3 months
- Secondary objectives
  - CGI-S
  - MDS-UPDRS Part III change from baseline
  - Patient diaries, on & off time
  - Safety and tolerability

Data expected end of Q4 2022

OLS = Open label study
# UDysRS: An Improved and Validated Dyskinesia Rating Scale

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>UDysRS</th>
<th>mAIMS</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>• Recommended by Movement Disorder Society (MDS)</td>
<td>• Suboptimal for detecting treatment-related changes</td>
</tr>
<tr>
<td></td>
<td>• FDA regulatory precedent (GOCOVRI® approval)</td>
<td>• Limited to patient assessments</td>
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<tr>
<td></td>
<td>• Contains anchored objective clinician evaluated measures of dyskinesia</td>
<td>• Prone to placebo effect</td>
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<td></td>
<td>• Includes both patient and physician assessments of impairment</td>
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<td></td>
<td>• Less prone to placebo effect</td>
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<tr>
<td>Clinimetrics</td>
<td>• Validated</td>
<td>• Only the original version has been validated</td>
</tr>
<tr>
<td>Development</td>
<td>• Developed in 2009 specifically for dyskinesia in PD</td>
<td>• Developed in 1970 for tardive dyskinesia in psychiatry</td>
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Dipraglurant PD-LID Studies – Management of Placebo Response

• Use of UDysRS
  – More sensitive to changes in LID
  – Less prone to placebo response

• Raters will be qualified by the MDS
  – Expert rater review to further ensure quality

• Requirement for moderate to severe symptom scores at screening and baseline

• BPST-Dys (non-pharmacologic intervention) to be used during screening

• Longer 12-week treatment period expected to mitigate placebo response
Dipraglurant for Dystonia – Blepharospasm
Blepharospasm (BSP)

- Type of dystonia affecting eyelid muscles
  - Results in sustained eyelid closure causing substantial visual disturbance or functional blindness
  - >50% of BSP patients symptoms spread to other cranio-facial muscles

- At least 50,000 BSP patients in US, ~2000 new patients diagnosed annually

- Botulinum toxin (BoNT) injections are the only approved treatment

- Surgical approaches including myectomy are invasive and frequently not of benefit

- Phase 2 feasibility study in BSP with dipraglurant IR started in September 2021 with data expected end of Q1 2022

- Dipraglurant extended release (ER) formulation being developed

- Phase 2a proof of concept with dipraglurant ER planned for 2022

- Potential to expand to other dystonias
Dystonias are *neuro-functional* rather than *neuro-degenerative*

Common features include alterations in neuronal connectivity/function and synaptic communication

BSP pathophysiology is linked to:

- Reduction of dopamine input into striatum
- Increased inhibition of direct pathway from superior colliculus to trigeminal nucleus
- Overexcitation of the signal leading to blink reflex

Pathogenesis involves aberrant or maladaptive brain plasticity linked to excessive sensory stimulation and/or repetitive motor tasks

Dipraglurant shows robust preclinical validation:

- 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)

Dipraglurant has shown anti-dystonic effect in PD patients

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Rationale for Targeting mGlu5 Inhibition in Dystonia & BSP

Adapted from Peterson & Sjenowski, 2017

SNc = Substantia Nigra pars compacta
SNr = Subst Nigra pars reticulata
STN = subthalamic nucleus
SC = Superior Colliculus
NRM = nucleus raphe magnus
SpV = spinal trigeminal nucleus
GPI/GPe = Globus Pallidus internal/external

SNc = Substantia Nigra pars compacta
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SpV = spinal trigeminal nucleus
GPI/GPe = Globus Pallidus internal/external
Blepharospasm Phase 2 Feasibility Study

- Patients with benign essential BSP, who experience moderate/severe symptoms prior to their regular dose of BoNT
- Single center, randomized, double-blind, placebo controlled
- Approx. 15 patients
- Dipraglurant IR - 50mg, 100 mg and placebo
- Efficacy endpoints include:
  - Computational Motor Objective Rater (CMOR)
  - Clinician rating scales
  - Patient reported outcomes

First patient enrolled in Sept 2021 - data expected end of Q1 2022
ADX71149 (JNJ-40411813) for Epilepsy
Partnered with Janssen Pharmaceuticals, Inc.
### ADX71149 Opportunity in Epilepsy

**Large market & unmet medical need**
- Market projected to reach $20 billion by 2026*
  - Keppra market leader with > 2M patients & €800M p.a.**
- High proportion of refractory patients (¼ of new patients***): combination treatments have limited therapeutic benefit
- Large underserved patient population in need of improved treatment options

**ADX71149: true synergistic MoA**
- Selective oral mGlu2 PAM with clear MoA in epilepsy
- Showed 35-fold increase in Keppra efficacy in preclinical model
- Potential first rational polypharmacy in epilepsy

**Development path**
- Extensive preclinical and clinical data
  - 8 Phase 1 and 2 Phase 2 studies
- Janssen Pharmaceuticals, Inc. started POC study in June 2021
  - Top line data expected in Q3 2022

**Strategic Partner Janssen Pharmaceuticals, Inc.**
- Eligible to receive €109 million in pre-launch milestones and double digit royalties

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* Fortune Business Insights April 8, 2020  
** UCB FY 2020  
*** Xue-Ping et al, Medicine July 2019
ADX71149 Preclinical Efficacy in Epilepsy - 6Hz Model

- Preclinical validation in pharmaco-resistant mouse epilepsy model:

  - 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
ADX 71149 Phase 2a Epilepsy Study

- Partial onset seizure with suboptimal response to levetiracetam
- 3-100 seizures/4weeks during baseline period

- Placebo n=20
- ADX71149 50mg bid n=40

- 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 Period 1 continue double-blind treatment during Period 2

Data expected in Q3 2022
Financials and Stock

- **Cash runway through H1 2023**
  - Cash at 30 September 2021: CHF15.5 million (USD16.6 million)
  - Sale of treasury shares in December 2021 raised USD10 million

- **No debt**

- **Traded on SIX Swiss Exchange: ADXN (ISIN: CH0029850754)**


- **37.9M outstanding shares**
- **49.3M issued shares incl. treasury shares (77.9M fully diluted)**
  - New Enterprise Associated - 12.62%
  - Armistice Capital LLC - 7.62%
  - New Leaf Venture Partners - 4.32%
  - CAM Capital - 2.87%
  - Credit Suisse Asset Management - 2.18%
  - Management & board holds - 10.71% (fully diluted basis)

- **Analyst coverage:**
  - HC Wainwright - Raghuram Selvaraju
  - Van Leeuwenhoek - Marcel Wijma
  - valuationLab - Bob Pooler
  - Baader Helvea AG – Leonildo Delgado
  - ZKB
### Milestones

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<thead>
<tr>
<th>Milestone</th>
<th>Timing</th>
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<tr>
<td>Dipraglurant for PDLID</td>
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<tr>
<td>Phase 2b/3 – study started</td>
<td>June 2021</td>
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<tr>
<td>Phase 2b/3 - topline results</td>
<td>End of Q4 2022</td>
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<tr>
<td>Dipraglurant for Blepharospasm</td>
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<tr>
<td>Phase 2a – study started</td>
<td>Sept 2021</td>
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<td>Phase 2a - topline results</td>
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<td>ADX71149 for Epilepsy</td>
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<tr>
<td>GABA&lt;sub&gt;B&lt;/sub&gt; PAM for Addiction and CMT1a</td>
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<tr>
<td>Start IND enabling studies</td>
<td>2022</td>
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<tr>
<td>mGlu7 NAM for PTSD</td>
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<tr>
<td>Start IND enabling studies</td>
<td>2022</td>
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<tr>
<td>Summary</td>
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<td><strong>3 clinical programs – data reading out from end Q1 2022</strong></td>
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<tr>
<td><strong>Technology and capabilities to deliver</strong></td>
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<tr>
<td>• Experienced team of drug developers</td>
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<td>• Pioneering allosteric modulation drug development</td>
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<td>‒ Proprietary screening assays and unique chemical library</td>
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<tr>
<td>• All programs developed in-house, protected with &gt;200 patents</td>
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<td><strong>Solid foundation</strong></td>
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<tr>
<td>• Partnerships with industry leaders</td>
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<tr>
<td>• Top tier US investors - NEA, NLV and CAM Capital Program</td>
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<tr>
<td>• Dual listed SIX Swiss exchange &amp; US Nasdaq</td>
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<td><strong>Promising outlook</strong></td>
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<tr>
<td>• Rich news flow in 2022 and beyond</td>
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<tr>
<td>‒ Clinical data reading out end of Q1 2022, Q3 2022 and end of Q4 2022</td>
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<tr>
<td>‒ Multiple drug candidates in CCS</td>
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</tr>
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