Innovative Treatments for Central Nervous System Disorders

November 2021
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Addex Overview

| 3 clinical programs underway | • Phase 3 Parkinson’s disease dyskinesia study – data Q4 2022  
• Phase 2 blepharospasm study – data 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 ($16.6M) at 30 September 2021 |
### 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 (mGlu5 NAM)</td>
<td>PD-LID</td>
<td></td>
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<td>Data Q4 2022</td>
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<td></td>
<td>Blepharospasm</td>
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<td>Data Q1 2022</td>
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<tr>
<td>ADX71149 (mGlu2 PAM)</td>
<td>Epilepsy</td>
<td>janssen</td>
<td></td>
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<td>Data Q3 2022</td>
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<td>GABA$_B$ PAM</td>
<td>Addiction</td>
<td>INDIVIOR</td>
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<td>CMT1A</td>
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<tr>
<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/Role</th>
<th>Experience/Contributions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tim Dyer</td>
<td>CEO / CFO</td>
<td>Co-Founder of Addex, Former with PwC, UK Chartered Accountant</td>
</tr>
<tr>
<td>Dr. Roger Mills</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>Dr. Robert Lutjens</td>
<td>Head of Discovery Biology</td>
<td>Member of Addex founding team, Formerly with Glaxo &amp; Scripps Research Institute</td>
</tr>
<tr>
<td>Dr. Jean-Philippe Rocher</td>
<td>Head of Discovery Chemistry</td>
<td>Member of Addex founding team, Formerly with Pierre Fabre, GSK and Mitsubishi</td>
</tr>
<tr>
<td>Dr. Mikhail Kalinichev</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

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

#### Scientific Advisory Board

<table>
<thead>
<tr>
<th>Name</th>
<th>Role</th>
<th>Experience/Contributions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darryle Schoepp</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>Mark Bear</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>Peter Bernstein</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>Benny Bettler</td>
<td>Biomedicine Prof. at Basel University</td>
<td>Formerly at Novartis and discovered allosteric modulators at GABA_\text{\textsubscript{B}} receptor and recipient of the Peter Speiser Award</td>
</tr>
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</table>
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:
  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
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

<table>
<thead>
<tr>
<th>Midday dose</th>
<th>Dipraglurant</th>
<th>Placebo</th>
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</thead>
<tbody>
<tr>
<td>Day 1 (50 mg)</td>
<td>19.9%</td>
<td>4.1%</td>
</tr>
<tr>
<td>Day 14 (100 mg)</td>
<td>32.3%</td>
<td>12.6%</td>
</tr>
<tr>
<td>Day 28 (100 mg)</td>
<td>31.4%</td>
<td>21.5%</td>
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</table>
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

<table>
<thead>
<tr>
<th></th>
<th>Dipraglurant</th>
<th>Placebo</th>
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</thead>
<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
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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<thead>
<tr>
<th></th>
<th>Dipraglurant</th>
<th>Placebo</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>
</tr>
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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 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 in 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>
</thead>
<tbody>
<tr>
<td>Recommended by Movement Disorder Society (MDS)</td>
<td>• FDA regulatory precedent (GOCOVRI® approval)</td>
<td>• Suboptimal for detecting treatment-related changes</td>
</tr>
<tr>
<td>Contains anchored objective clinician evaluated measures of dyskinesia</td>
<td>• Includes both patient and physician assessments of impairment</td>
<td>• Limited to patient assessments</td>
</tr>
<tr>
<td>• Less prone to placebo effect</td>
<td></td>
<td>• Prone to placebo effect</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Clinimetrics</th>
<th>UDysRS</th>
<th>mAIMS</th>
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<tbody>
<tr>
<td>• Validated</td>
<td>• Only the original version has been validated</td>
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<table>
<thead>
<tr>
<th>Development</th>
<th>UDysRS</th>
<th>mAIMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Developed in 2009 specifically for dyskinesia in PD</td>
<td>• Developed in 1970 for tardive dyskinesia in psychiatry</td>
<td></td>
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</tbody>
</table>
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 in 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:
  A. Reduction of dopamine input into striatum
  B. Increased inhibition of direct pathway from superior colliculus to trigeminal nucleus
  C. 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

Adapted from Peterson & Sjenowski, 2017

<table>
<thead>
<tr>
<th>SNC</th>
<th>Substantia Nigra pars compacta</th>
<th>SNC</th>
<th>Subst Nigra pars reticulata</th>
<th>STN</th>
<th>subthalamic nucleus</th>
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<tbody>
<tr>
<td>SC</td>
<td>Superior Colliculus</td>
<td>NRM</td>
<td>nucleus raphe magnus</td>
<td>SpV</td>
<td>spinal trigeminal nucleus</td>
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<tr>
<td>GPi/GPe</td>
<td>Globus Pallidus internal/external</td>
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</table>

mGlu5 receptor
Excitatory (glutamate)
Inhibitory (GABA)
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 in Q1 2022
ADX71149 (JNJ-40411813) for Epilepsy
Partnered with Janssen Pharmaceuticals, Inc.
<table>
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<tr>
<th>Large market &amp; unmet medical need</th>
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<tbody>
<tr>
<td>• Market projected to reach $20 billion by 2026*</td>
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<tr>
<td>– Keppra market leader with &gt; 2M patients &amp; €800M p.a.**</td>
</tr>
<tr>
<td>• High proportion of refractory patients (¼ of new patients*** ) - combination treatments have limited therapeutic benefit</td>
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<tr>
<td>• Large underserved patient population in need of improved treatment options</td>
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<thead>
<tr>
<th>ADX71149: true synergistic MoA</th>
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<tbody>
<tr>
<td>• Selective oral mGlu2 PAM with clear MoA in epilepsy</td>
</tr>
<tr>
<td>• Showed 35-fold increase in Keppra efficacy in preclinical model</td>
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<tr>
<td>• Potential first rational polypharmacy in epilepsy</td>
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<table>
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<tr>
<th>Development path</th>
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<tbody>
<tr>
<td>• Extensive preclinical and clinical data</td>
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<tr>
<td>– 8 Phase 1 and 2 Phase 2 studies</td>
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<tr>
<td>• Janssen Pharmaceuticals, Inc. started POC study in June 2021</td>
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<tr>
<td>– Top line data expected in Q3 2022</td>
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<table>
<thead>
<tr>
<th>Strategic Partner Janssen Pharmaceuticals, Inc.</th>
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<tr>
<td>• Eligible to receive €109 million in pre-launch milestones and double digit royalties</td>
</tr>
</tbody>
</table>

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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:

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

  ![Graph 1](graph1.png)
  ![Graph 2](graph2.png)

- 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

**Timepoints**

- Screening / Baseline
- Period 1 Acute Efficacy
- Period 2 Maintenance Efficacy

**Data expected in Q3 2022**
Financials
Financials and Stock

• Cash runway through 2022
  – Cash at 30 September 2021: CHF15.5 million

• No debt

• Traded on SIX Swiss Exchange: ADXN (ISIN:CH0029850754)

• ADS representing 6 shares traded on Nasdaq: ADXN (ISIN: US00654J107; CUSIP: 00654J107)

• 34.1M outstanding shares
• 49.3M issued shares incl. treasury shares (62.3M fully diluted)
  – New Enterprise Associated - 14.21%
  – New Leaf Venture Partners - 4.86%
  – CAM Capital – 3.24%
  – Credit Suisse Asset Management – 2.46%
  – Management & board holds -12.05% (fully diluted basis)

• Analyst coverage:
  – HC Wainwright – Raghuram Selvaraju
  – Van Leeuwenhoek - Marcel Wijma
  – valuationLab - Bob Pooler
  – ZKB - Dr. Michael Nawrath
  – Baader Helvea AG - Bruno Bulic
## Milestones

<table>
<thead>
<tr>
<th>Milestone</th>
<th>Timing</th>
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<tbody>
<tr>
<td>Dipraglurant for PDLID</td>
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<tr>
<td>Phase 2b/3 – study started</td>
<td>June 2021</td>
</tr>
<tr>
<td>Phase 2b/3 - topline results</td>
<td>Q4 2022</td>
</tr>
<tr>
<td>Dipraglurant for Blepharospasm</td>
<td></td>
</tr>
<tr>
<td>Phase 2a – study started</td>
<td>Sept 2021</td>
</tr>
<tr>
<td>Phase 2a - topline results</td>
<td>Q1 2022</td>
</tr>
<tr>
<td>ADX71149 for Epilepsy</td>
<td></td>
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</tr>
<tr>
<td>GABA&lt;sub&gt;B&lt;/sub&gt; PAM for Addiction and CMT1a</td>
<td></td>
</tr>
<tr>
<td>Complete clinical candidate selection</td>
<td>Q4 2021</td>
</tr>
<tr>
<td>Start IND enabling studies</td>
<td>Q2 2022</td>
</tr>
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</table>
Summary

| 3 clinical programs – data reading out from Q1 2022 | • Phase 3 Parkinson’s disease dyskinesia study – data Q4 2022  
• Phase 2 blepharospasm study – data Q1 2022  
• Phase 2 epilepsy study (J&J) – data Q3 2022 |
|---|---|
| Technology and capabilities to deliver | • Experienced team of drug developers  
• Pioneering allosteric modulation drug development  
  – Proprietary screening assays and unique chemical library  
• All programs developed in-house, protected with >200 patents |
| Solid foundation | • Partnerships with industry leaders  
• Top tier US investors - NEA, NLV and CAM Capital Program  
• Dual listed SIX Swiss exchange & US Nasdaq |
| Promising outlook | • Rich news flow in 2022 and beyond  
  – Clinical data reading out Q1 2022, Q3 2022 and Q4 2022  
  – Multiple drug candidates in CCS |
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