Innovative Treatments for Central Nervous System Disorders

February 2021
Disclaimer

These materials do not constitute or form part, or all, of any offer or invitation to sell or issue, neither in the United States of America nor elsewhere, or any solicitation of any offer to purchase or subscribe for, any securities, nor shall part, or all, of these materials or their distribution form the basis of, or be relied on in connection with, any contract or investment decision in relation to any securities.

These materials contain forward-looking statements based on the currently held beliefs and assumptions of the management of Addex Therapeutics, which are expressed in good faith and, in their opinion, reasonable. Forward-looking statements involve known and unknown risks, uncertainties and other factors, which may cause the actual results, financial condition, performance, or achievements of Addex Therapeutics Ltd, or industry results, to differ materially from the results, financial condition, performance or achievements expressed or implied by such forward-looking statements.

Given these risks, uncertainties and other factors, recipients of this document are cautioned not to place undue reliance on these forward-looking statements. Addex Therapeutics Ltd disclaims any obligation to update these forward-looking statements to reflect future events or developments.
### Addex Overview

| 3 clinical programs | • Dipraglurant PD-LID study 301 starting H1 2021*  
|                      | • Dipraglurant blepharospasm Phase 2 study starting H1 2021  
|                      | • ADX71449 (J&J) epilepsy Phase 2 study starting Q2 2021 |
| Leading allosteric modulator technology platform | • Allosteric modulation is a validated & differentiated pharmacological approach to address drug targets  
|                                                      | • Proprietary biological screening assays and chemical library |
| In house discovered pipeline | • Multiple novel drug candidates entering clinical candidate selection  
|                                                      | • Driving long term growth & future partnership opportunities |
| Partnerships | • 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 CHF18.7M at 31 Dec 2021  
|                                                      | • Completed $11.5M capital increase 11 Jan 2021 |

* 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
<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>
</tr>
</thead>
<tbody>
<tr>
<td>Dipraglurant (mGlu5 NAM)</td>
<td>PD-LID</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Data Q4 2022</td>
</tr>
<tr>
<td></td>
<td>Blepharospasm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Data Q4 2021</td>
</tr>
<tr>
<td>ADX71149 (mGlu2 PAM)</td>
<td>Epilepsy</td>
<td>[Janssen]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Data H1 2022</td>
</tr>
<tr>
<td>GABA&lt;sub&gt;8&lt;/sub&gt; PAM</td>
<td>Addiction</td>
<td>[INDIVIOR]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CMT1A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mGlu7 NAM</td>
<td>PTSD</td>
<td>[Pfizer]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mGlu2 NAM</td>
<td>Mild neurocognitive disorders</td>
<td>[Eurostars]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mGlu4 PAM</td>
<td>Parkinson’s disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mGlu3 PAM</td>
<td>Neurodegenerative disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Lead Program Entering 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>
</tr>
</thead>
<tbody>
<tr>
<td>Tim Dyer</td>
<td>CEO / CFO</td>
<td>Co-Founder of Addex, Formerly with PwC, UK Chartered Accountant</td>
</tr>
<tr>
<td>Dr. Roger Mills</td>
<td>Chief Medical Officer</td>
<td>Developed Nuplazid in PD Psychosis, 30 years in Pharma industry including 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. 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>
</tbody>
</table>

## Non-executive Directors

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Experience</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>Title</th>
<th>Experience</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&lt;sub&gt;B&lt;/sub&gt; receptor and recipient of the Peter Speiser Award</td>
</tr>
</tbody>
</table>

(addex therapeutics logo)
## Milestones

<table>
<thead>
<tr>
<th>Milestone</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dipraglurant for PDLID</td>
<td></td>
</tr>
<tr>
<td>Phase 2b/3 - start study</td>
<td>H1 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 - start study</td>
<td>H1 2021</td>
</tr>
<tr>
<td>Phase 2a - topline results</td>
<td>Q4 2021</td>
</tr>
<tr>
<td>ADX71149 for Epilepsy</td>
<td></td>
</tr>
<tr>
<td>Phase 2a - start study</td>
<td>Q2 2021</td>
</tr>
<tr>
<td>Phase 2a - topline results</td>
<td>H1 2022</td>
</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>Q1 2022</td>
</tr>
</tbody>
</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

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

<table>
<thead>
<tr>
<th></th>
<th>Dipraglurant</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worsening Dyskinesia</td>
<td>21%</td>
<td>12.5%</td>
</tr>
<tr>
<td>(15.3%*)</td>
<td></td>
<td></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>
</tbody>
</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)
# U DysRS: An Improved and Validated Dyskinesia Rating Scale

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>UDysRS</th>
<th>mAIMS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recommended by Movement Disorder Society (MDS)</strong></td>
<td>• Contains anchored objective clinician evaluated measures of dyskinesia</td>
<td>• Suboptimal for detecting treatment-related changes</td>
</tr>
<tr>
<td><strong>FDA regulatory precedent (GOCOVRI® approval)</strong></td>
<td>• Includes both patient and physician assessments of impairment</td>
<td>• Limited to patient assessments</td>
</tr>
<tr>
<td><strong>Less prone to placebo effect</strong></td>
<td></td>
<td>• Prone to placebo effect</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinimetrics</th>
<th>UDysRS</th>
<th>mAIMS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Validated</strong></td>
<td></td>
<td>• Only the original version has been validated</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Development</th>
<th>UDysRS</th>
<th>mAIMS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Developed in 2009 specifically for dyskinesia in PD</strong></td>
<td></td>
<td>• Developed in 1970 for tardive dyskinesia in psychiatry</td>
</tr>
</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 PD-LID Registration Program Started

• Pivotal registration program ongoing

• Study 301 expected to start H1 2021*
  – Data read-out expected Q4 2022
  – Primary endpoint: UDysRS
  – Placebo mitigation is a priority

• 12-month Open Label Study (302) starting in parallel to study 301
  – 6- and 12-month safety data

• Second pivotal registration study (303) to follow study 301 completion

* 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
Dipraglurant First 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

**OLS = Open label study**

- **N = 140** Moderate to severe PD-LID
- **N = 70** Placebo
- **N = 70** Dipraglurant (100mg)

3 month double-blind treatment period

Primary End Point
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 expected to start in H1 2021 and read out data by the end of 2021

• 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

---

**Rationale for Targeting mGlu5 Inhibition in Dystonia & BSP**

Adapted from Peterson & Sjenowski, 2017

---

**Legend**

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

**Diagram Notes**

- **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

---

**References**

Peterson & Sjenowski, 2017
Blepharospasm Phase 2 Feasibility Study Design

- 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
ADX71149 (JNJ-40411813) for Epilepsy
Partnered with Janssen (JNJ)
## ADX71149 Opportunity in Epilepsy

### Large market & unmet medical need
- Market projected to reach $20 billion by 2026*
  - Keppra market leader with approx. 2.2M 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 expect to start POC study in Q2 2021
  - Top line data expected in H1 2022

### Partnership with Janssen
- Eligible to receive €109 million in pre-launch milestones and double digit royalties

---

* Fortune Business Insights; April 08, 2020 12:20 ET
** UCB H1 2020 – sales end H1 €440M
*** Xue-Ping et al, Medicine - July 2019
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**

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
- Partial onset seizure with suboptimal response to levetiracetam
- ≥ 3 seizures during baseline period

• Double blind placebo controlled
• Establish 28-day seizure count (over 56-day baseline period)
• Primary endpoint: time to 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
Financials
Financials and Stock

- **Cash runway through 2022**
  - Cash at 31 Dec 2020: CHF18.7 million
  - Completed $11.5M capital increase 11 Jan 2021

- **No debt**

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

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

- **39.7M 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 - 3.01%
  - 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
## Summary

### 3 clinical programs starting in H1 2021
- Dipraglurant PD-LID registration study
- Dipraglurant blepharospasm Phase 2
- ADX71449 (J&J) epilepsy Phase 2

### 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 2021 and beyond
  - Clinical programs
  - Multiple drug candidates in CCS
ALLOSTERIC MODULATORS FOR HUMAN HEALTH

www.addextherapeutics.com