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Transforming the Possible in Neuroscience

2Q 2021 Financial Results & Business Update

August 2021

Forward-Looking Statements

This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on information currently available to management. In some cases, you can identify forward-looking statements by the following words: "may," "will," "could," "would," "should," "expect," "intend," "plan," "anticipate," "believe," "estimate," "predict," "project," "potential," "continue," "ongoing" or the negative of these terms or other comparable terminology, although not all forward-looking statements contain these words. These statements involve risks, uncertainties and other factors that may cause actual results, levels of activity, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. Although we believe that we have a reasonable basis for each forward-looking statement contained in this presentation, we caution you that these statements are based on a combination of facts and factors currently known by us and our projections of the future, about which we cannot be certain.

Forward-looking statements in this presentation include, but are not limited to: statements about the potential attributes and benefits of our product candidates, including with respect to receptor subtype selectivity, activity, side effect and tolerability profile and relevant indications; the format and timing of our product development activities and clinical trials, including the advancement of CVL-231 into a comprehensive Phase 2 program in schizophrenia, plans to evaluate the potential of this mechanism in other populations, including dementia-related psychosis, the timing and status of our Phase 1 trial of darigabat in acute anxiety and other statements regarding the design of clinical trials and the timing of initiation, completion and data readouts for clinical trials; the timing and outcome of IND submissions and other regulatory interactions; the ability to compete with other companies currently marketing or engaged in the development of treatments for relevant indications; the size and growth potential of the markets for product candidates and ability to serve those markets; the rate and degree of market acceptance of product candidates, if approved; the potential effects of the business combination; the amount and timing of payments we may receive pursuant to the tavapadon financing transaction; the sufficiency of our financial resources, including to fund the tavapadon Phase 3 development program through NDA submission; receipt of proceeds from our warrant redemption; and our cash runway.

We cannot assure you that the forward-looking statements in this presentation will prove to be accurate. Furthermore, if the forward-looking statements prove to be inaccurate, the inaccuracy may be material. Actual performance and results may differ materially from those projected or suggested in the forward-looking statements due to various risks and uncertainties, including, among others: that we may not realize the expected benefits of the financing transaction; that clinical trial results may not be favorable; uncertainties inherent in the product development process (including with respect to the timing of results and whether such results will be predictive of future results); the impact of COVID-19 on the timing, progress and results of ongoing or planned clinical trials; other impacts of COVID-19, including operational disruptions or delays or to our ability to raise additional capital; whether and when, if at all, our product candidates will receive approval from the FDA or other regulatory authorities, and for which, if any, indications; competition from other biotechnology companies; uncertainties regarding intellectual property protection; and other risks identified in our SEC filings, including those under the heading "Risk Factors" in our Quarterly Report on Form 10-Q filed with the SEC on May 17, 2021 and our subsequent SEC filings.

In light of the significant uncertainties in these forward-looking statements, you should not regard these statements as a representation or warranty by us or any other person that we will achieve our objectives and plans in any specified time frame, or at all. The forward-looking statements in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements at some point in the future, we have no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation.

Agenda

Introduction		**Matthew Calistri** *Vice President, Corporate Strategy and Investor Relations*
Overview		**Tony Coles, M.D.** *Chairperson & Chief Executive Officer*
Lead Program Updates		**Raymond Sanchez, M.D.** *Chief Medical Officer*
Early Pipeline Updates		**John Renger, Ph.D.** *Chief Scientific Officer*
Q2 Financial Performance		**Kathy Yi** *Chief Financial Officer*
Q&A	*All*	

Business & Pipeline Updates

Pipeline

- Announced **clinically meaningful and statistically significant** topline results from Phase 1b trial for **CVL-231** in schizophrenia
- **Initiated screening** in Phase 2a trial in dementia-related apathy for **CVL-871**
- Submitted IND for **CVL-354 (KORA)**, in development for MDD and substance use disorder; expect to **initiate Phase 1 in 3Q 2021**
- **Darigabat** Phase 1 acute anxiety data now expected **1H 2022**
- Upcoming R&D Event scheduled for **October 7, 2021**

Capital

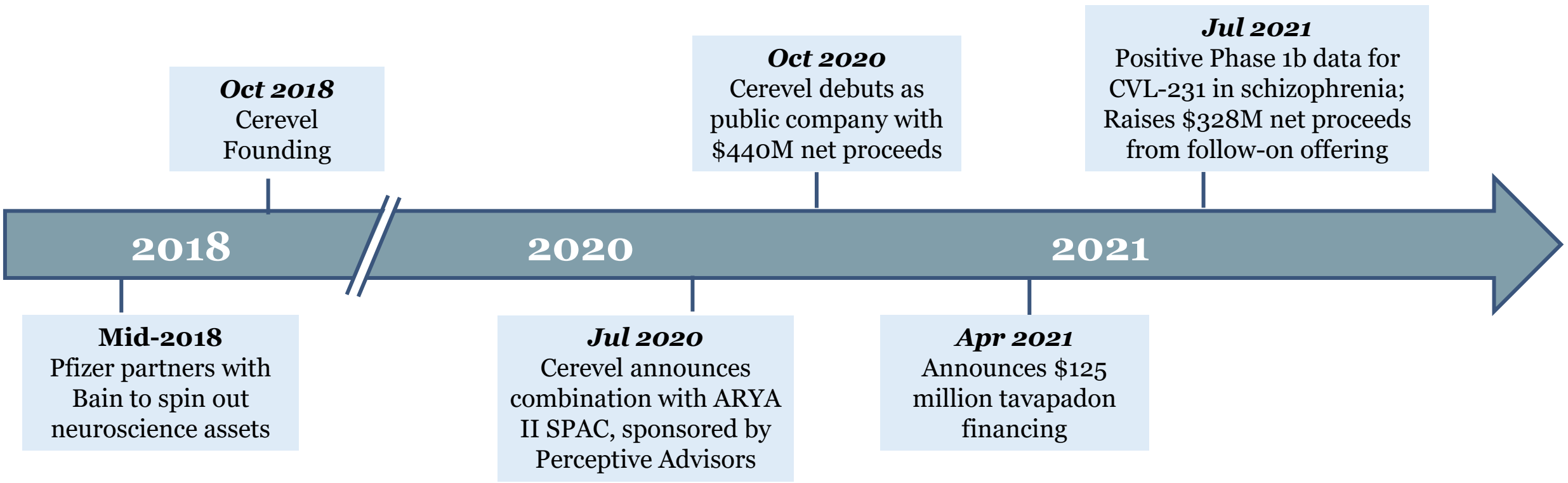
- Raised **$328 million net proceeds** from follow-on offering in July 2021
- Announced redemption of outstanding public warrants
- Cash balance of **$327 million*** as of 6/30/2021
- Cash runway projected into **2024**

*Does not include $328 million net proceeds from follow-on offering or any expected proceeds from exercise of public warrants

Company Highlights

- Pursuing a targeted approach to neuroscience that combines a differentiated understanding of neurocircuitry with advanced chemistry to develop novel therapies for CNS diseases
- Broad portfolio of 11 assets targeting large markets with significant unmet need, including schizophrenia, epilepsy and Parkinson's Disease
- Progressing towards multiple near and medium-term catalysts, with at least 7 data readouts and multiple INDs expected by the end of 2023
- Leveraging a seasoned management team with extensive expertise in neuroscience and a strong track record of over 20 prior drug approvals and commercialization

History of Innovative Dealmaking



Led by a Seasoned Life Sciences Management Team

Tony Coles, M.D.
Chairperson & Chief Executive Officer

Abraham Ceesay
President

Kathy Yi
Chief Financial Officer

Raymond Sanchez, M.D.
Chief Medical Officer

John Renger, Ph.D.
Chief Scientific Officer

Kenneth DiPietro
Chief Human Resources Officer

Kathleen Tregoning
Chief Corporate Affairs Officer

Scott Akamine
Chief Legal Officer

Yumanity Therapeutics · NPS Pharma · Vertex · Bristol-Myers Squibb · Novartis · Otsuka · Avanir pharmaceuticals

Onyx Pharmaceuticals · Biogen · Merck · Pfizer · Imbrium Therapeutics · Purdue · Sanofi

Strong Track Record of Approvals

- Abilify® (aripiprazole)
- Abilify MyCite® (aripiprazole tablets with sensor) 2, 5, 10, 15, 20, 30 mg
- Abilify Maintena® (aripiprazole) for extended release injectable suspension
- Belsomra® (suvorexant) CIV 5, 10, 15, 20 mg tablets
- JYNARQUE® (tolvaptan) tablets
- Nexavar® (sorafenib) tablets
- Samsca® (tolvaptan)
- Kyprolis® (carfilzomib) for Injection
- REXULTI® brexpiprazole
- ADHANSIA XR™ (methylphenidate HCl) CII extended-release capsules
- BLINCYTO® (blinatumomab) for injection

Cerevel's Targeted Approach to CNS Disease

Leveraging Expertise in Neurocircuitry

Differentiated Understanding of Neurocircuitry

→

Receptor Binding/Modulation

→

Highly Selective Small Molecules in Clinical Studies
Created using Pfizer world-class chemistry

Pipeline Uniquely Based on

- Targeted Receptor Subtype Selectivity
- Optimized Receptor Pharmacology
- Robust Data Packages

Deep Pipeline: Multiple Value Inflections Near & Long-Term

11 Assets

5 Clinical Assets

- Tavapadon
- Darigabat
- CVL-231
- CVL-871
- CVL-936

4 Pre-Clinical Assets

- CVL-354 (KORA)
- CVL-047 (PDE4)
- M4 Agonist*
- LRRK2*

2 Undisclosed Assets

Patient Populations

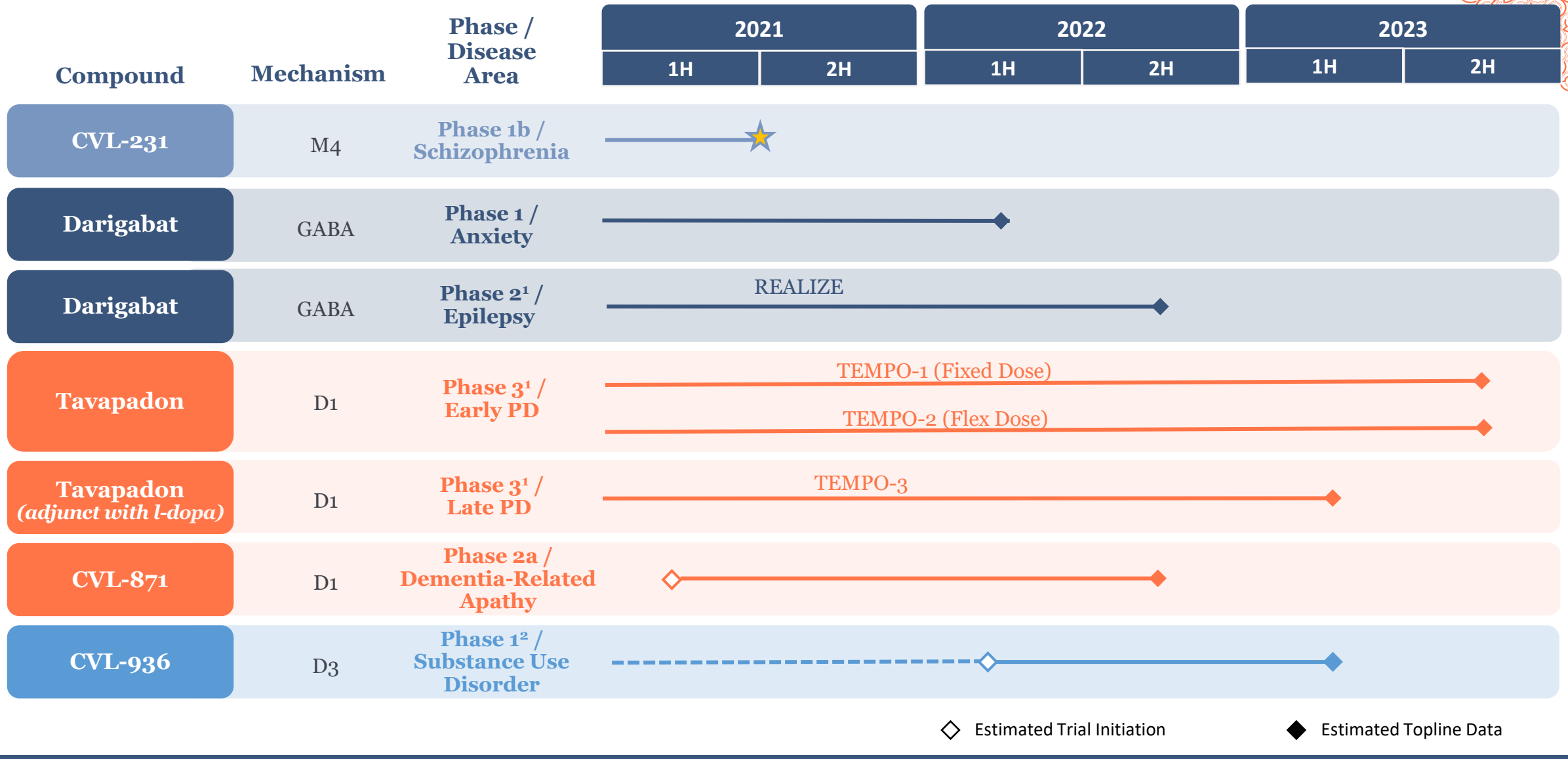
- Early Parkinson's
- Late Parkinson's
- Epilepsy
- Anxiety
- Schizophrenia
- Dementia-Related Apathy
- Substance Use Disorder

8 Trials

3	Phase 3 Trials
2	Phase 2 Trials
1	Phase 1b Trial Completed
2	Phase 1 Trials
+2	Open Label Extensions

* - Lead Optimization

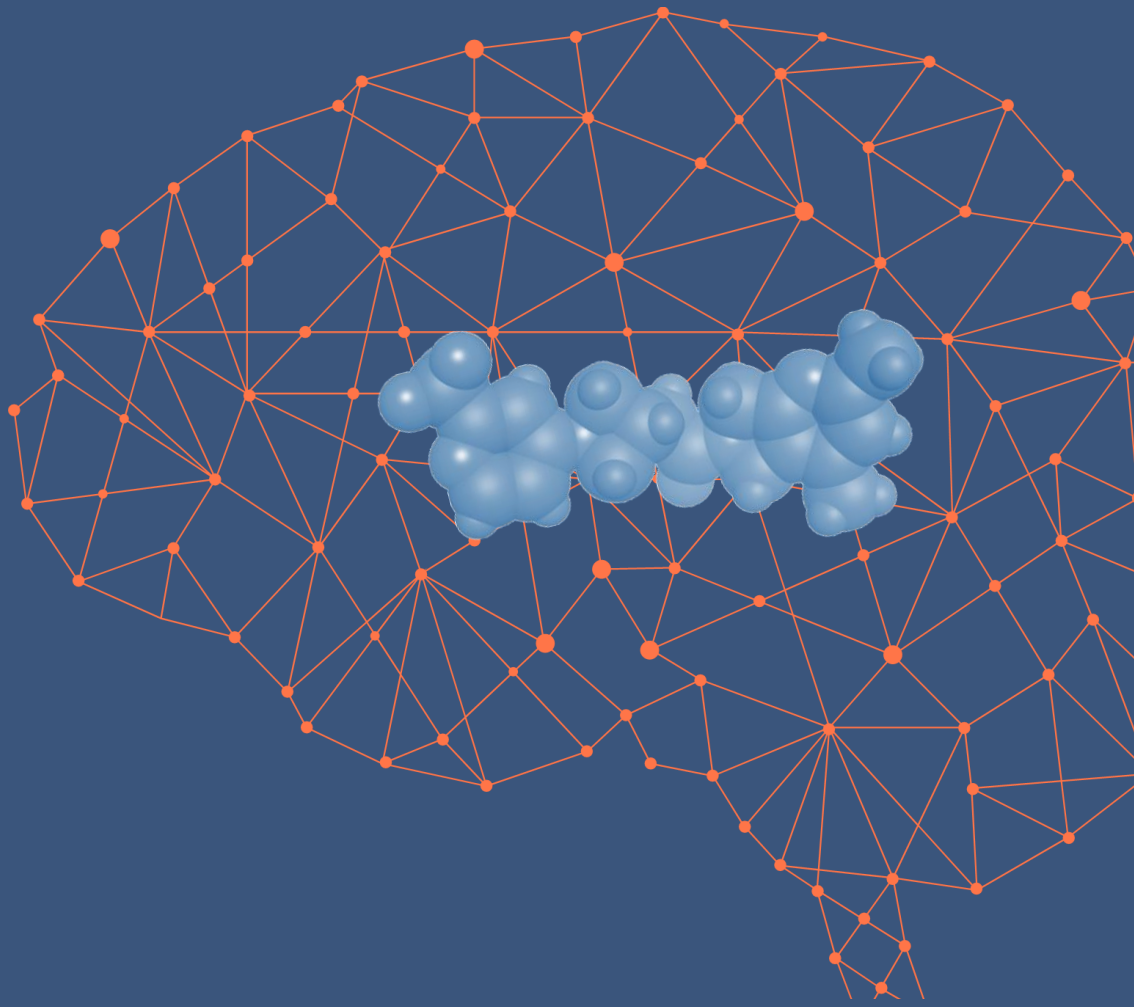
Multiple Milestones Expected Over Next Three Years



1. In addition, there are two open-label extension trials ongoing (REALIZE OLE for darigabat in epilepsy and TEMPO-4 for tavapadon)
2. We initiated a Phase 1 SAD trial for CVL-936 in January 2020. We concluded dosing of Cohort 1 of the Phase 1 SAD trial. We intend to conduct a non-clinical safety pharmacology study prior to resuming Phase 1 SAD and MAD evaluations.

M4 PAM (CVL-231) in Schizophrenia

Selectively targeting the M4 muscarinic receptor with the goal of treating psychosis-related symptoms with improved side effect profile



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Cerevel's M4 PAM is a Potential Next Generation Antipsychotic

M4 PAM (CVL-231)
Potential New Standard of Care

First-in-Class Therapy with Novel MOA

M4 Selective

Targeted Muscarinic Activity

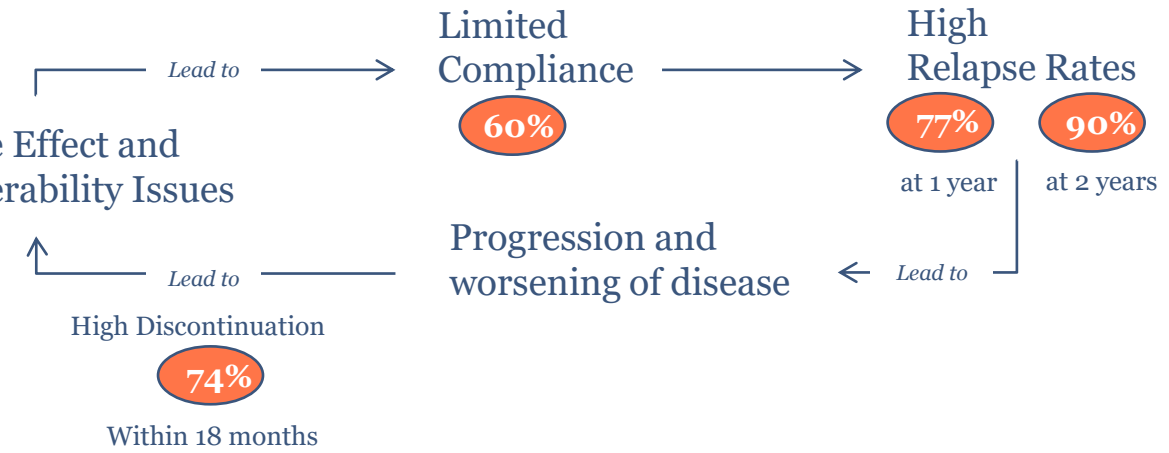
Improved Tolerability

Opportunity for Innovation in Schizophrenia
Current Standard of Care Uses Same Mechanism of Action (MOA) as Therapies from the 1950s

Large Market

- **~20M** Patients Worldwide
- **~$7B** G7 Revenues in 2018
- **~3.5%** Growth per year

Significant Need for New Treatment Option



Debilitating side effects of atypicals often lead to discontinuation and relapse, driving a vicious cycle of disease progression

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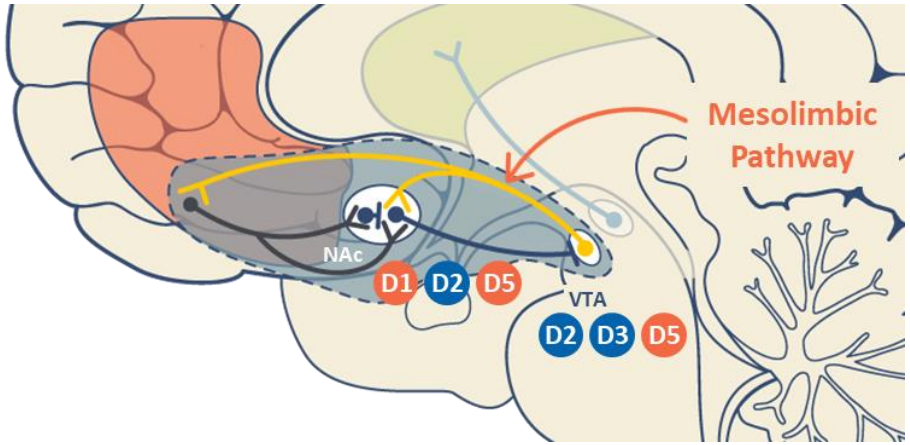
Source: World Health Organization, DRG Market Research

M4 Receptor Activation Reduces Dopamine in the Striatum

Schizophrenia symptoms driven by ***overactivity of the dopaminergic*** mesolimbic pathway



Cholinergic Neuron

M4 Auto-Receptor

CVL-231 enhances M4 auto-receptor activation in the presence of Ach

CVL-231 (PAM)

Dopaminergic tone is reduced ***without*** direct antagonistic activity on post-synaptic dopamine receptors

M4 auto-receptor activation prevents additional Ach release

Acetylcholine (Ach)

Nicotinic Receptors

Dopamine

Nucleus Accumbens

Dopamine Neuron

Originates from Ventral Tegmental Area (VTA)

In the absence of Ach, dopamine output decreases, which ***dampens hyperdopaminergic activity*** in striatum

Potential for Decreased Psychosis

Cerevel's Selective M4 Modulation: A Compelling and Novel Approach to Drive Antipsychosis

Receptor Selectivity Offers Potential Improvement over Non-Selective Compounds

Xanomeline (M1/M4 agonist) data showed targeting muscarinic receptor may improve psychosis

But development limited by GI and CV side effects

Karuna's KarXT addresses tolerability issues by adding trospium to Xanomeline to offset side effects

Combination approach with non-selective peripheral antagonist

Knock-out mouse data suggests M4 receptors drive the antipsychotic activity of Xanomeline

M1 receptors believed to contribute to GI side effects, potential cognitive benefit undetermined

CVL-231's Differentiated Approach

Target	• Highly selective for M4 receptor
Antipsychotic effect	• 19.5 pt improvement in PANSS total score at Week 6
Tolerability	• No GI-related dropouts • Not associated with EPS, akathisia, or weight gain
Dosing	• Once-daily
Titration	• None

CVL-231:
Selective Potentially Once-daily M4 PAM

>600x
more selective for M4 over M1, 3 and 5

~360x
more selective than for M2

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EPS = Extrapyramidal symptoms

Phase 1b in Schizophrenia: Topline Results

- **Clinically meaningful improvements in PANSS Total Score:**
  - 30 mg QD: -19.5 pts at week 6
  - 20 mg BID: -17.9 pts at week 6
- **Statistically significant difference in PANSS Total Score versus placebo***:
  - 30 mg QD: -12.7 pts (p=0.023) at week 6
  - 20 mg BID: -11.1 pts (p=0.047) at week 6
- **Clinically meaningful improvements in both PANSS Positive and PANSS Negative subscales**
- **Generally well-tolerated:**
  - Discontinuation rates similar between treatment and placebo (22% for each arm including placebo)
  - Not associated with extrapyramidal side effects, akathisia, or weight gain
  - Gastrointestinal (GI) side effects were infrequent and were similar between treatment and placebo
  - Serious adverse events included COVID-19, accidental overdose (cocaine), and exacerbation of schizophrenia (one instance of each); none considered related to study medication

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*Trial originally designed to be 59% powered to detect 7 point difference in PANSS total score vs. placebo
Note: Additional safety and tolerability detail can be found in the appendix.

CVL-231 Phase 1b Trial Design

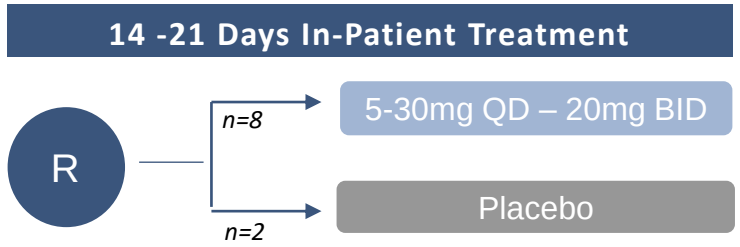
Part A: Safety Assessment

Multiple Ascending Dose

Up to 5 Cohorts (n=10 each)

Primary Objective
- Safety & tolerability

Secondary Objective
- PK

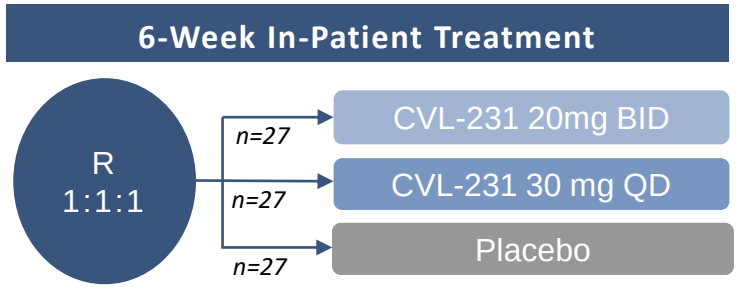


Target Patient Population

- Male and female subjects, ages 18 to 50 years
- CGI-S ≤ 4 (normal to moderately ill) at screening and Day-1
- PANSS total score of ≤ 80 at the time of screening and Day-1

Part B: Pharmacodynamics

Exploratory PD Assessment
- Positive and Negative Syndrome Scale (PANSS)*
  - PANSS Positive Score
  - PANSS Negative Score
- Clinical Global Impression – Severity Scale (CGI-S)*
- Brief Assessment of Cognition in Schizophrenia (BACS) symbol coding test*



Target Patient Population

- Male and female subjects, ages 18 to 55 years
- PANSS total score of ≥80 at screening and Day -1
- CGI-S ≥4 (moderately to severely ill) at screening and Day -1
- History of relapse and/or exacerbation of symptoms when not receiving antipsychotic treatment, excluding the current episode
- Experiencing an acute exacerbation or relapse of symptoms, with onset less than 2 months prior to screening
- Population was enriched for key positive symptoms

* Trial not designed to demonstrate statistical significance

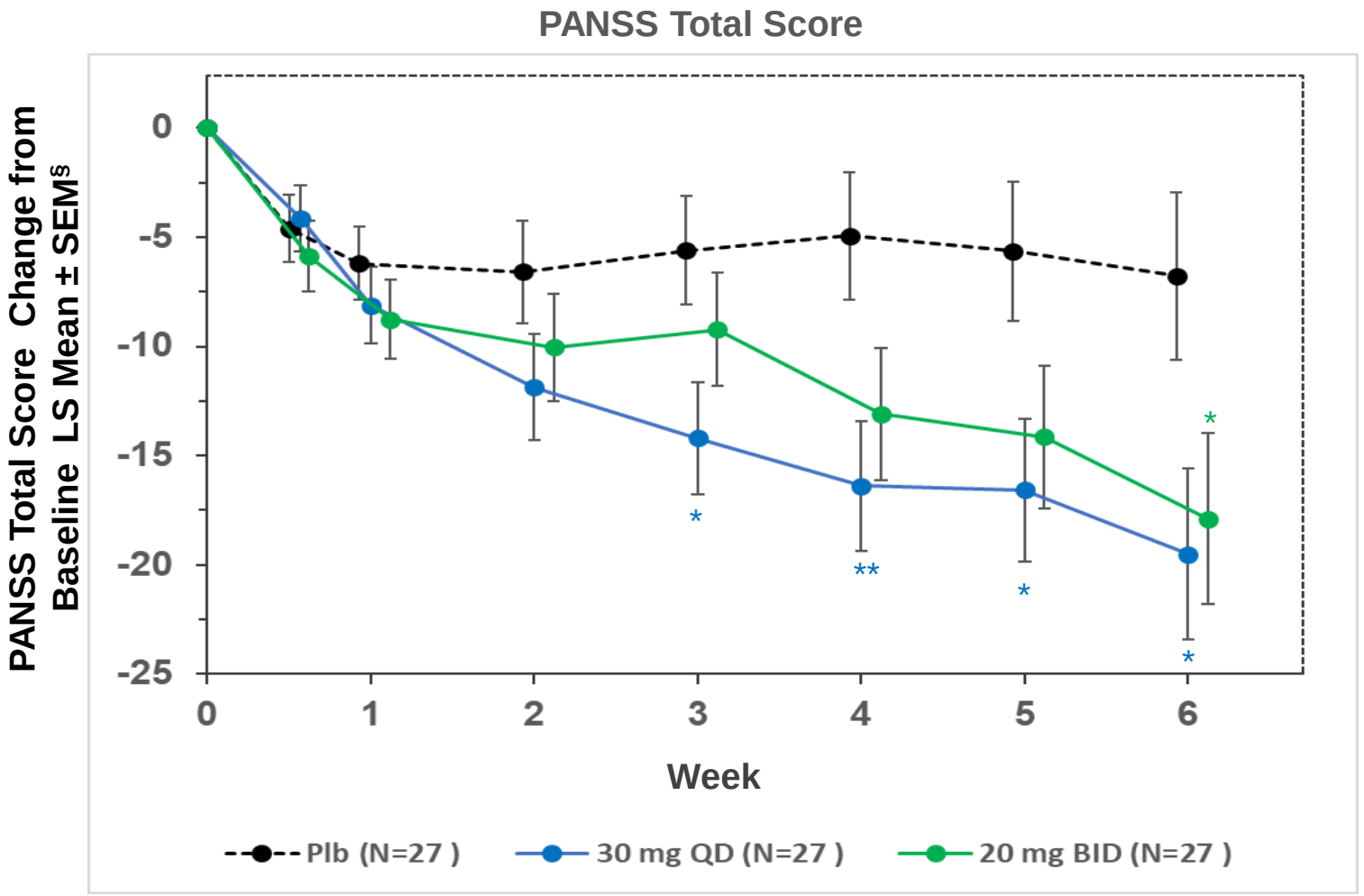
Phase 1b in Schizophrenia: Pharmacodynamic Results*

Week 6 (Day 42)	Placebo N=27	CVL-231 30 mg QD N= 27	CVL-231 20 mg BID N= 27	Combined CVL-231 N=54
PANSS Total Score				
LS Mean Change from Baseline	-6.8	-19.5	-17.9	-18.7
Difference vs Placebo (p-value)†		-12.7†	-11.1†	-11.9†
		p = 0.023	p = 0.047	p = 0.014
PANSS Positive Score				
LS Mean Change from Baseline	-2.5	-6.8	-4.9	-5.8
Difference vs Placebo (p-value)		-4.3	-2.4	-3.3
		p = 0.016	p = 0.166	p = 0.028
PANSS Negative Score at Baseline				
LS Mean Change from Baseline	0.1	-3.0	-3.6	-3.3
Difference vs Placebo (p-value)		-3.1	-3.7	-3.4
		p = 0.009	p = 0.002	p = 0.001

*Trial originally designed to be 59% powered to detect 7 point difference in PANSS total score vs. placebo

†Corresponds to Cohen's D effect sizes at Week 6 of -0.68 for CVL-231 30 mg QD, -0.59 for CVL-231 20 mg BID, and -0.64 for the two doses combined

Phase 1b: Key Pharmacodynamic Endpoint – PANSS Total Score



- **30 mg QD:** 12.7 Point improvement versus placebo at Week 6 (19.5 of 30 mg QD vs 6.8 placebo) with P=0.023
- **20 mg BID:** 11.1 Point improvement versus placebo at Week 6 (17.9 of 20 mg BID vs 6.8 placebo) with P=0.047
- **Combined CVL 231:** 11.9 Point improvement versus placebo at Week 6 (18.7 of CVL231 vs 6.8 placebo) with P=0.014

* P<0.05 vs Placebo ** P<0.01 vs Placebo

§ Derived from a mixed model for repeated measures (MMRM) with treatment group, visit, treatment group-by-visit interaction as fixed effect, baseline value as a covariate and the measurements within subject as repeated measures. An unstructured covariance matrix was used.

Dementia-Related Psychosis (DRP): Potential Opportunity for CVL-231 Beyond Schizophrenia

DRP Overview and Unmet Needs[1-7]

- Psychosis incidence ranges from 10-75% of Alzheimer's patients and varies by stage of disease
  - Upwards of 1M moderate to severe Alzheimer's patients in the G7 experience symptoms of psychosis
- Co-morbidities including agitation, aggression and depression
- Often leads to long-term care / nursing home admissions

Standard of Care

- None established
- Off-label use of atypical antipsychotics: tolerability issues heightened in this population; contribute to cognitive decline

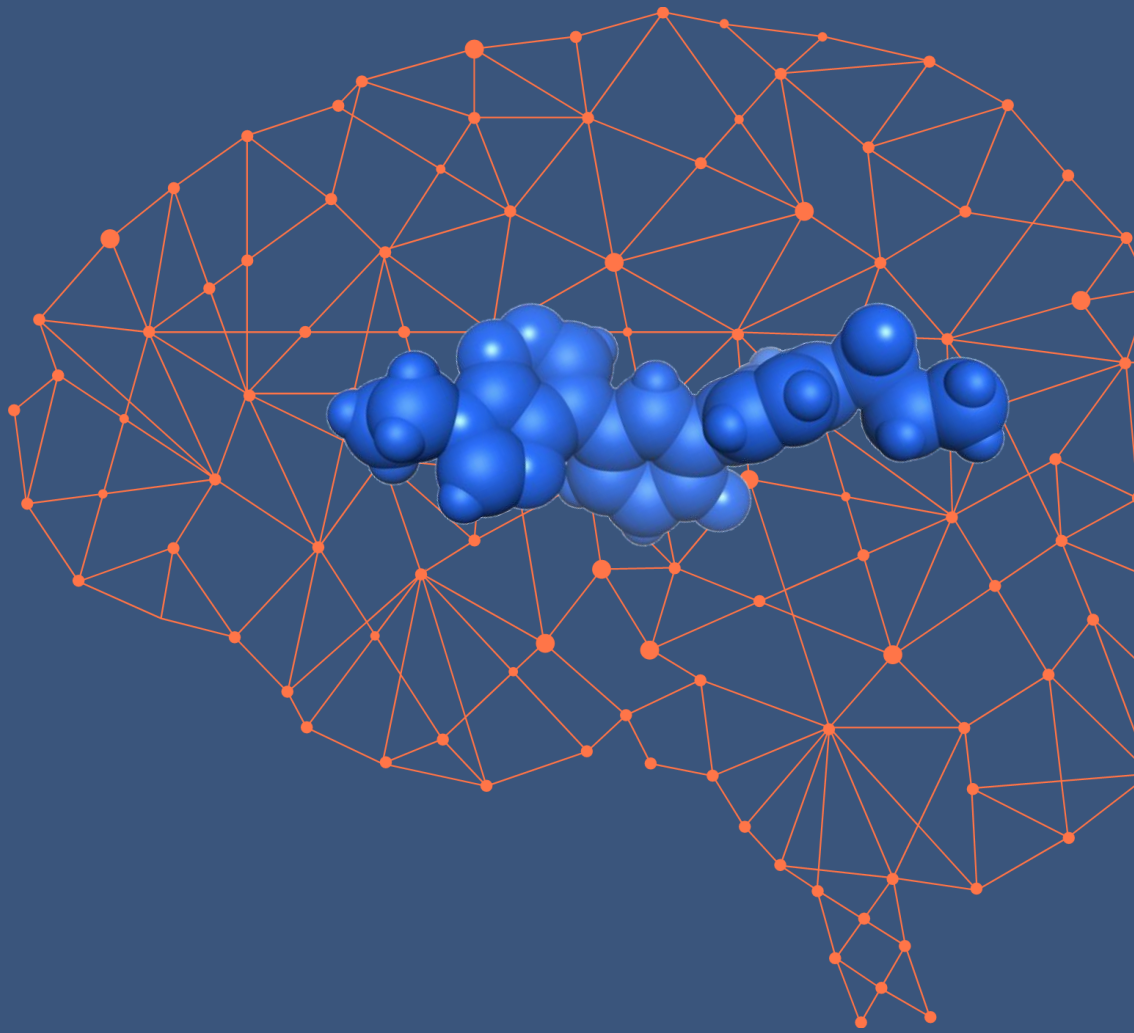
Next Steps for CVL-231

- CVL-231 side effects / tolerability observed to date are appropriate for further clinical evaluation in elderly patients
- Upcoming clinical pharmacology study in the elderly

1) Biol Psychiatry. 2014 April 1; 75(7): 542–552. 2) J Prev Alz Dis 2018: Pimavanserin in AD psychosis: Efficacy in patients with more pronounced psychotic symptoms. 3) Ballard, C., Gauthier, S., Cummings, J. et al.. Nat Rev Neurol 5, 245–255 (2009) 4) Sultzer et al., (2004) 5) Flint et al., (1991) 6) Sultzer et al., (1992) 7) Paulsen, et al. Incidence of and risk factors for hallucinations and delusions in patients with probable Alzheimer's disease. Neurology. 2000; 54:1965–1971

Darigabat ($GABA_A$ PAM)

Selectively targeting the α-2/3/5 subunits of the $GABA_A$ receptor with the goal of enhancing anti-convulsant and anxiolytic effects without dose-limiting sedation



Darigabat has Potential for Benzo-like Activity, Improved Side Effects and Chronic Dosing

Darigabat

Potential to become first-line and adjunct therapy

Targeted GABA α 2/3/5 Receptor Selectivity

Benzo-like Activity

Improved Tolerability

Potential for Reduced Abuse Liability

Opportunity for New Treatment Option in Epilepsy

HCPs and patients are dissatisfied due to insufficient activity, side effects and poor tolerability

Large Market

- ~65M Patients Worldwide
- >$6B G7 Revenues in 2018
- ~6% per year Branded AED[1] Market Growth through 2025

Benzos are highly efficacious, but…

- Poor Tolerability
- Desensitization & Loss of Efficacy
- Potential for Abuse
- Withdrawal

Potential as chronic therapy with improved side effect profile and tolerability may expand use vs. traditional benzodiazepines

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Source: World Health Organization, DRG Market Research
1. AED: Anti-Epileptic Drug

Selective $GABA_A$ Receptor PAM: Potential for Lower Seizure Rates, Reduced Side Effects

GABA α-2/3/5 Can Differentially Address Symptoms

Darigabat

GABA subtype predicted effects:	α1	α2	α3	α5
Anti-convulsant	✓✓	✓✓		
Anxiolysis		✓✓	✓✓	
Analgesia		✓✓	✓	✓✓
Muscle Relaxation		✓✓	✓✓	
Sedation	✓✓			
Cognitive Impairment	✓✓	?	?	✓
Addiction	✓✓	✓		

Benzodiazepine side effects

Role for Targeted GABA α 2/3/5 Receptor Selectivity

- >10 benzodiazepines (BZDs - broad-spectrum GABA modulators) widely prescribed for anxiety, seizures and other indications
- Significant need for better-tolerated, less sedating and less addictive GABA modulators
- BZD Rx's increased >95% from 2003-2015

To our knowledge, darigabat is the only GABA α-2/3/5 selective PAM in clinical trials for epilepsy

Darigabat Data Showed a Favorable Side Effect Profile Relative to Benzodiazepines

Multiple doses of darigabat

Phase 1 MAD Study – only mild AEs and limited somnolence up to 42.5 mg BID

Able to achieve >80% receptor occupancy without significant somnolence observed

Benzodiazepines achieve 10% to 15% receptor occupancy with significant somnolence observed

No evidence of withdrawal effects

Phase 1 MAD Study (Protocol: B7431011)

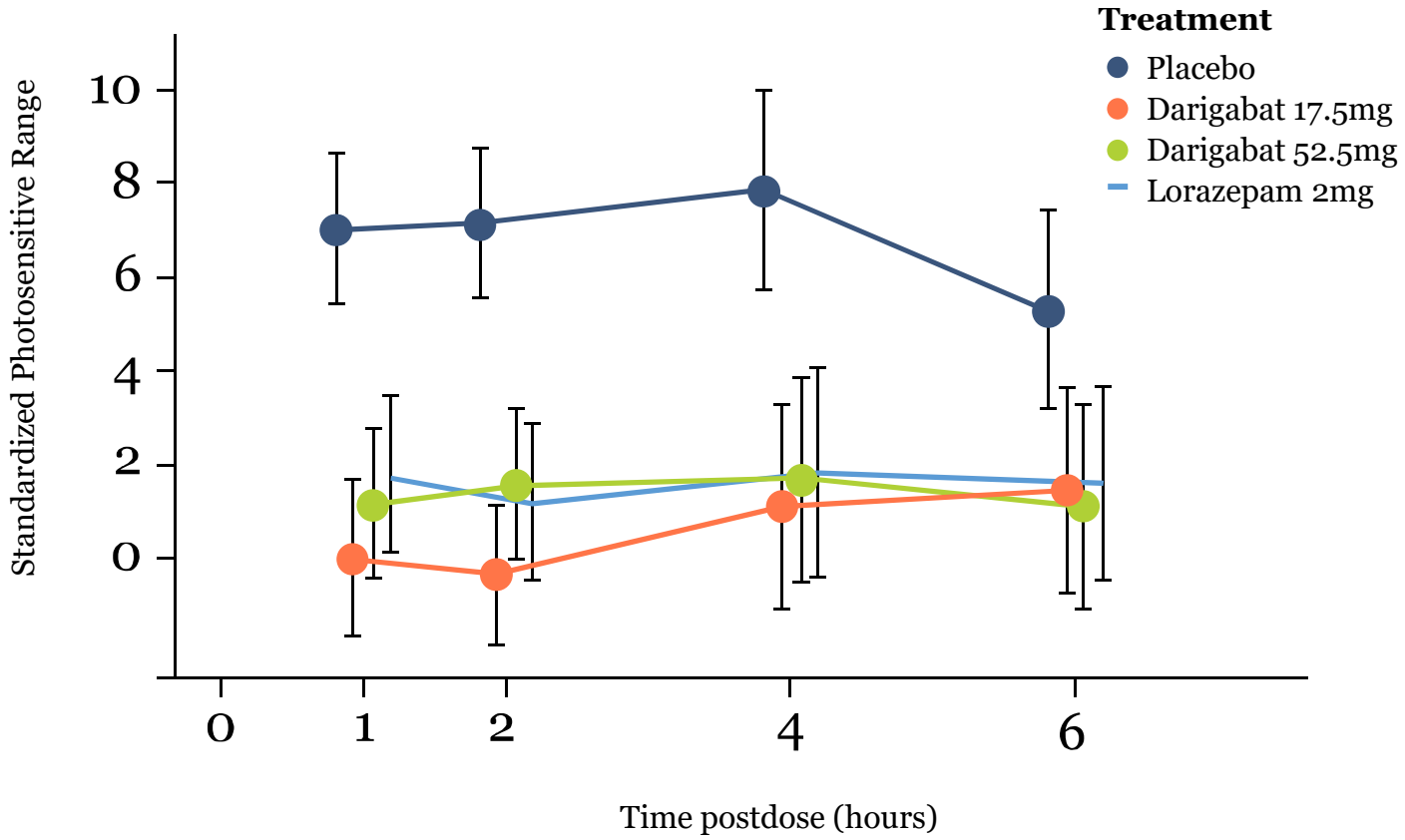
	Reaction	Week 1 (Titration)	Week 2 (Maintenance)	Week 3 (Maintenance)	Follow-up
Placebo	**No Reaction**	**4 / 4**	**4 / 4**	**3 / 4**	**4 / 4**
	Dizziness	-	-	1 / 4	-
	Somnolence	-	-	-	-
25 mg BID (~80% RO(1))	**No Reaction**	**5 / 8**	**7 / 8**	**8 / 8**	**8 / 8**
	Dizziness	2 / 8	1 / 8	-	-
	Somnolence	3 / 8	-	-	-
42.5 mg BID (>80% RO(1))	**No Reaction**	**4 / 7**	**6 / 7**	**6 / 7**	**6 / 7**
	Dizziness	3 / 7	1 / 7	1 / 7	1 / 7
	Somnolence	-	-	-	-

No somnolence observed following titration through doses of 42.5 mg BID

(1) Receptor Occupancy

Darigabat Phase 2 Data Showed Benzo-like Anticonvulsant Activity in Photosensitive Epilepsy(1)

Darigabat in Single-Dose Photosensitive Epilepsy Study



Darigabat Results

Anticonvulsant activity comparable to lorazepam

Improved sedation and AE profile compared to benzos

Complete suppression in 6 of 7 subjects

Majority of AEDs developed for epilepsy that showed positive published photoepilepsy results were approved(2)

Source: (1) IND B7431005: Phase 2 double-blind, randomized, cross-over study using lorazepam as a positive control; n=7 photoepilepsy subjects per arm; (2) Yuen and Sims. Seizure 23 (2014) 490–493

Darigabat REALIZE Trial: Data Expected 2H 2022

REALIZE Phase 2 Trial In Focal Epilepsy

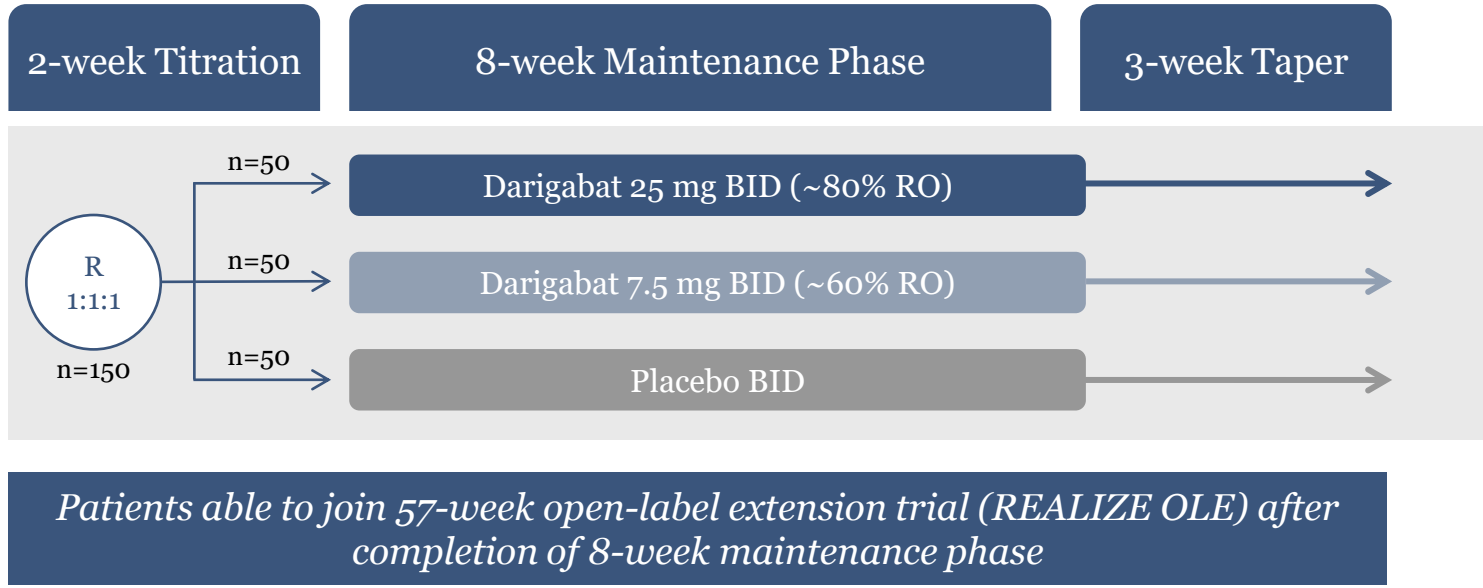
Targeting ~60 sites in 3 countries

Inclusion criteria

- Adults (18-75) with drug-resistant focal onset seizures
- History of 4+ seizures per month for at least 3 months
- 1-3 stable background AEDs allowed

Primary endpoint

- Reduction in focal onset seizure frequency



Focal epilepsy trial intended to establish proof of concept and side effect profile to support development in broader epilepsy indications

Darigabat: Phase 1 Program in Acute Anxiety

The Hypercapnia (CO2 Inhalation) Model

- CO2 inhalation challenge is translational model providing proof-of-principle for anxiolytic activity in early clinical development
- Well-established in both healthy volunteers and in patients with panic disorder
  - Hypercapnia results in increased fear and panic, as measured by Visual Analogue Scales (VAS) and the Panic Symptom List (PSL)[1]
- The proposed mechanism of the anxiety induced by hypercapnia model is decreased GABA and increased noradrenaline[2]
- The model is sensitive to drugs used to treat anxiety disorders (including benzodiazepines & SSRIs) and emerging new treatments with novel mechanisms

CO_2 inhalation induces fear and panic symptoms in healthy volunteers and panic disorder patients

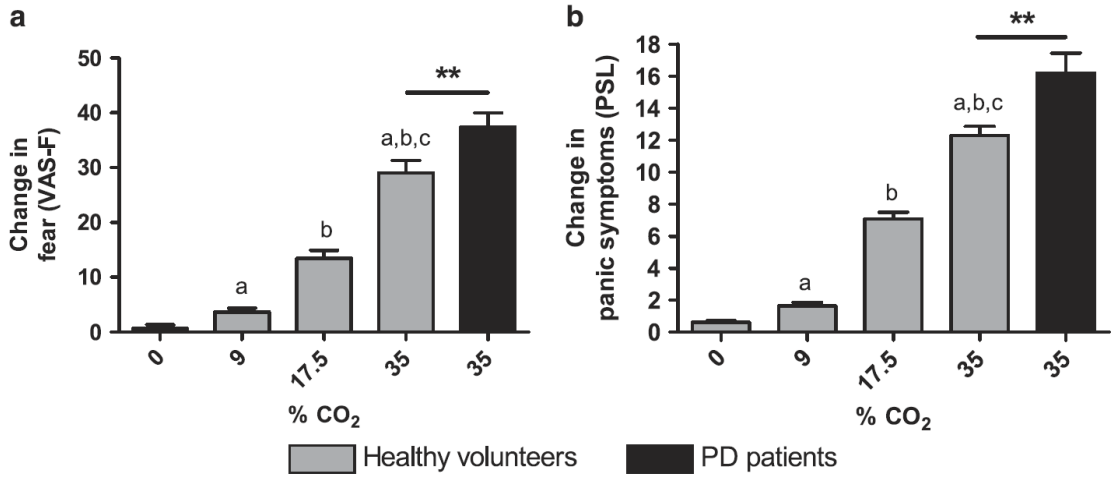
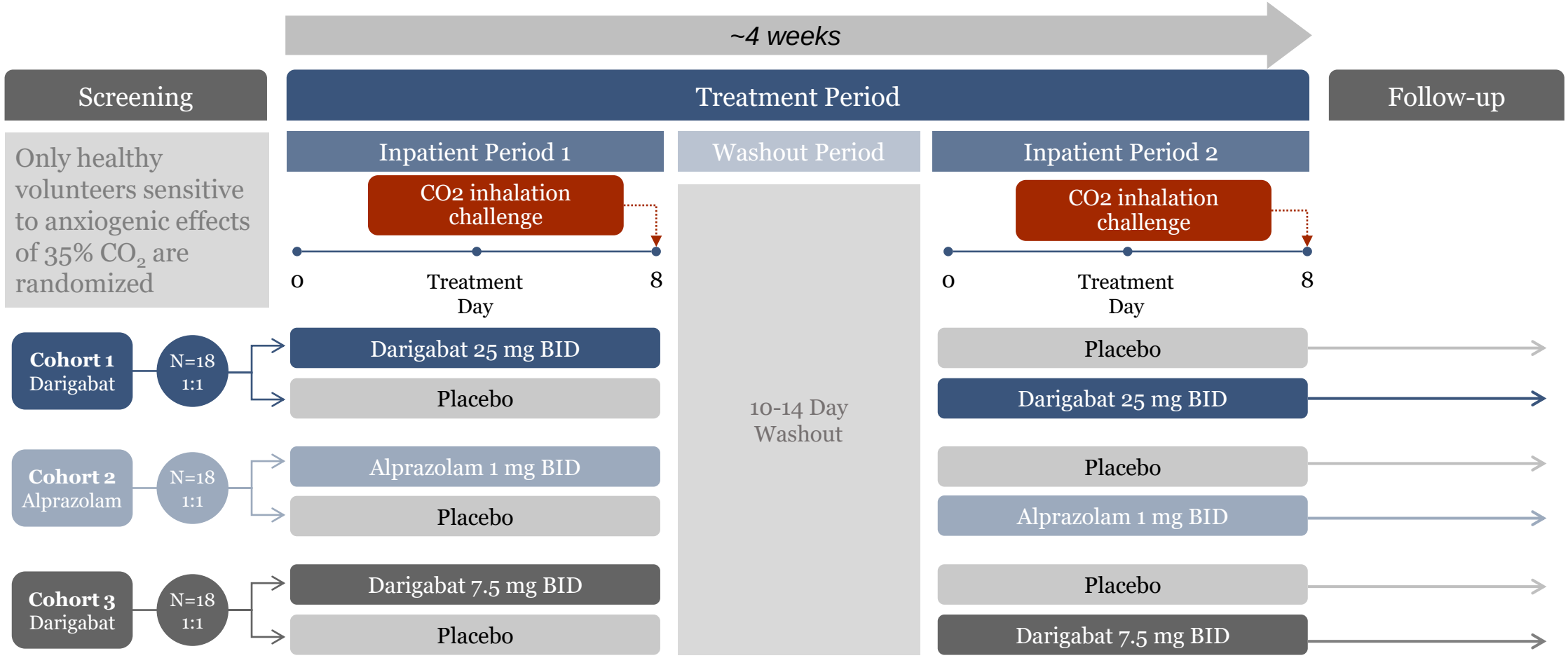


Figure 2. Effect of CO_2 on self-reported fear and panic symptoms in healthy volunteers and PD patients. In healthy volunteers (gray), both fear (**a**) and panic symptoms (**b**) increased dose-dependently. Inhaling 35% CO_2 triggered a more robust response in patients (black) when compared with healthy volunteers. Data represent mean+s.e.m. (**a**) Compared with 0% CO_2, $P < 0.001$; (**b**) compared with 9% CO_2, $P < 0.001$; (**c**) compared with 17.5% CO_2, $P < 0.001$; $^{**}P < 0.01$. PD, panic disorder; PSL, Panic Symptom List; VAS-F, Visual Analog Scale for fear.

1. Leibold et al., Trans Psychiatry 2016 2. Bailey et al., J Psychopharm 2011
*PD patients = Panic Disorder patients

Darigabat Phase 1 in Acute Anxiety: Data Expected 1H 2022

Randomized, double-blind, placebo- and active-controlled crossover design with multiple doses over 8 days. Primary endpoint: Panic symptoms list[1]. Doses selected to achieve ~60 and 80% receptor occupancy

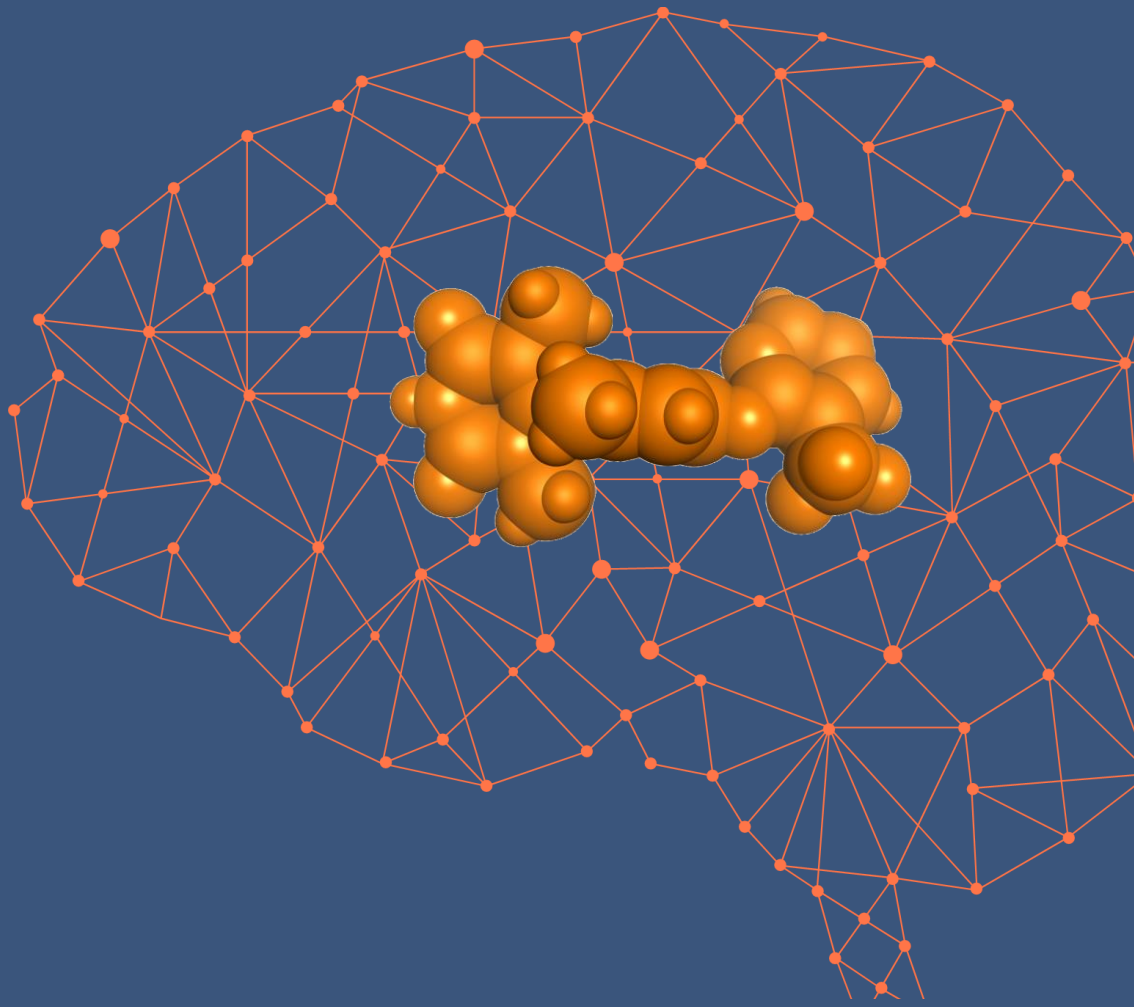


1. The Panic Symptom List (PSL) includes 13 symptoms scored across a range of 0 (absent) to 4 (very intense) that is used to assess panic anxiety. Liebold et al. Trans Psychiatry. 2016.; Bailey et al. J Psychopharm. 2011.; Malizia et al. Arch Gen Psychiatry. 1998.; Salvatore et al. Translational Psychiatry 2020.

RO = Receptor Occupancy

Tavapadon in Parkinson's Disease

Partial agonist selectively targeting the dopamine D1 receptor with the goal of enhancing motor control while minimizing side effects



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Tavapadon has Potential to be a Differentiated Treatment for Parkinson's

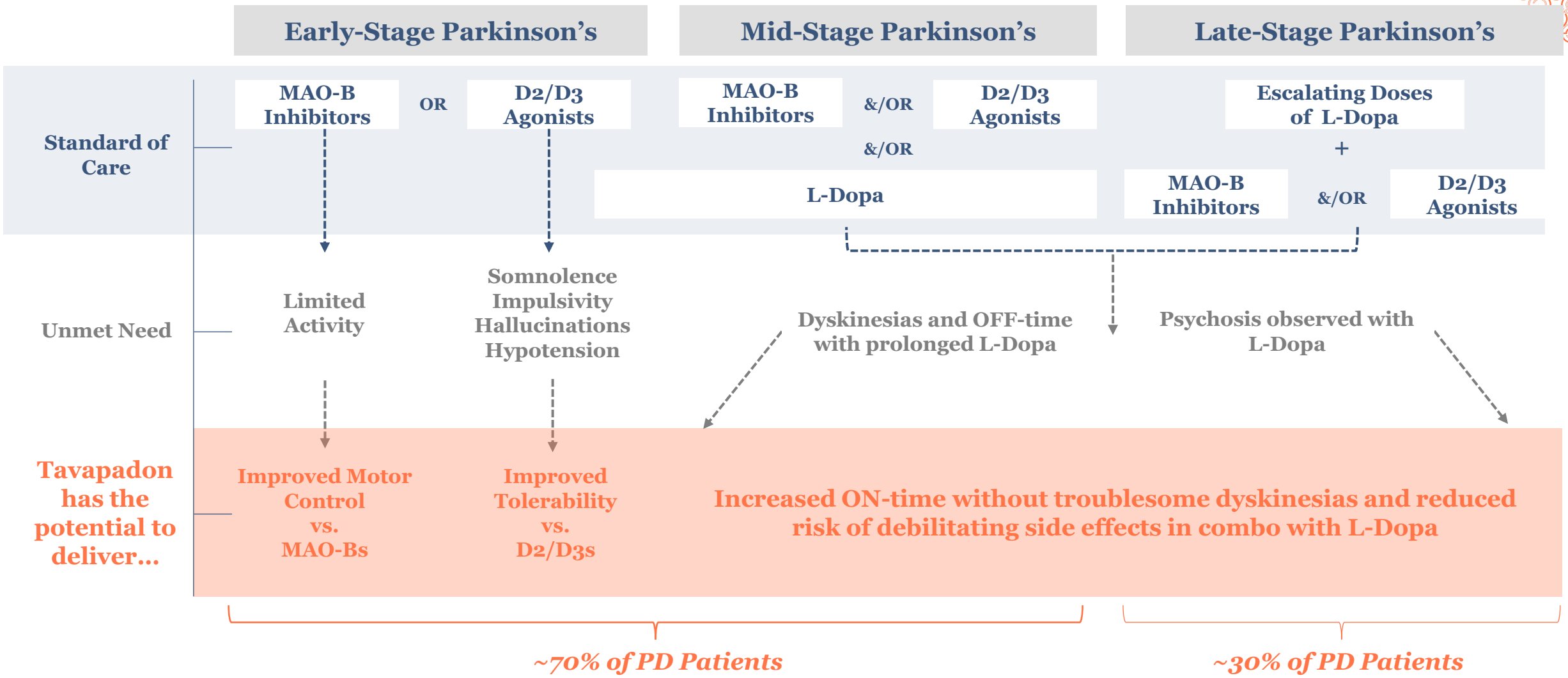
Designed to be a novel backbone therapy for patients from diagnosis to the end of treatment:

Only* D1/D5 selective molecule	**Avoid D2/D3 Side Effects:** *Sudden daytime somnolence, hallucinations, acute orthostasis and impulse control disorders*
First* partial agonist for Parkinson's	**Avoid Dyskinesias:** *Driven by receptor overexcitation*
Predictable 24-hour activity	**Sustained Effect:** *Once daily, oral dosing*
Selective direct motor pathway activation	**Superior motor control over D2/D3s full agonists**

- Feedback received from FDA on our Phase 3 program (2019)
- To our knowledge, nothing else in the symptomatic pipeline positioned to provide broad therapeutic benefit and differentiation

First-in-class potential designed to offer stable motor control and favorable side effect profile with broad monotherapy and adjunct therapy benefit

Tavapadon Designed to Address Unmet Needs Across All Stages of Parkinson's: Early and Late

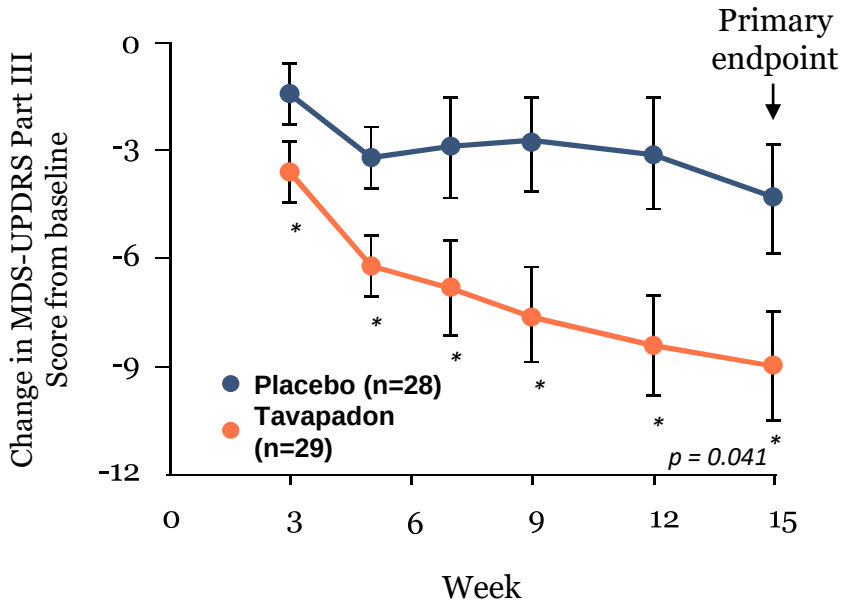


Sources: PD MED Collaborative Group. Lancet 2014; 384: 1196–205. Ahlskog JE, Mov Disord 16:448–458

Selective Direct Motor Pathway Activation Designed to Provide Differentiated Treatment Option in Early Parkinson's

Potential for motor control as good or better than D2/D3s with once-daily dosing and improved side effect profile

Phase 2 Data: Tavapadon in Early PD[1]
(Primary Endpoint: MDS-UPDRS III Motor Score)



In Phase 2, tavapadon demonstrated 4.8 point MDS-UPDRS III difference vs. placebo at week 15 (p=0.04, MMRM)

Note: Average dose of tavapadon = 9 mg; 11 patients on 15 mg top dose at week 15. Baseline Part III scores of 24.3 (tavapadon) and 25.8 (placebo).

Additional Tavapadon Phase 2 Data[1]

- When adjusted to exclude patients with baseline MDS-UPDRS II of 0 or 1, **showed improvement of ~2 points over placebo on MDS-UPDRS Part II**[2]
- Most common AEs included headache and nausea (can be mitigated with titration)
- Tavapadon's incidence of known D2/D3 side effects:
  - Somnolence: 14%
  - Nausea: 31%
  - Hallucinations: 0%[3]
  - Hypotension-Related Events: 7%
  - Dizziness: 7%

Tavapadon demonstrated 5.8 point improvement over placebo at week 15 on MDS-UPDRS Part II + III (p = 0.02, MMRM)

1. Study B7601011: (n=57) 15-week, Phase 2, double-blind, randomized, placebo-controlled flexible dose study to investigate the efficacy, safety, and tolerability of Tavapadon in subjects with early stage Parkinson's Disease Primary endpoint: Change from baseline in the MDS-UPDRS Part III total score at week 15. Allowed concomitant MAO-B inhibitors 2. Excluding 8 participants (6 treatment, 2 placebo) with baseline MDS-UPDRS Part II scores of 0 or 1 resulted in an improvement on MDS-UPDRS II at week 15 of -2.4 points for the tavapadon arm (n=19) vs -0.6 points for the placebo arm (n=20), resulting in a placebo-adjusted difference of 1.8 points (raw data, completers at week 15). Raw data placebo-adjusted difference is 1.3 points (including 8 participants). 3. Also observed 0% hallucinations in late-stage PD Phase 2 study B7601003 as adjunct to l-dopa

Tavapadon TEMPO-1 & -2 in Early PD: Data Expected 2H 2023

Registration-directed program includes three global Phase 3 trials optimally designed to maximize treatment effect

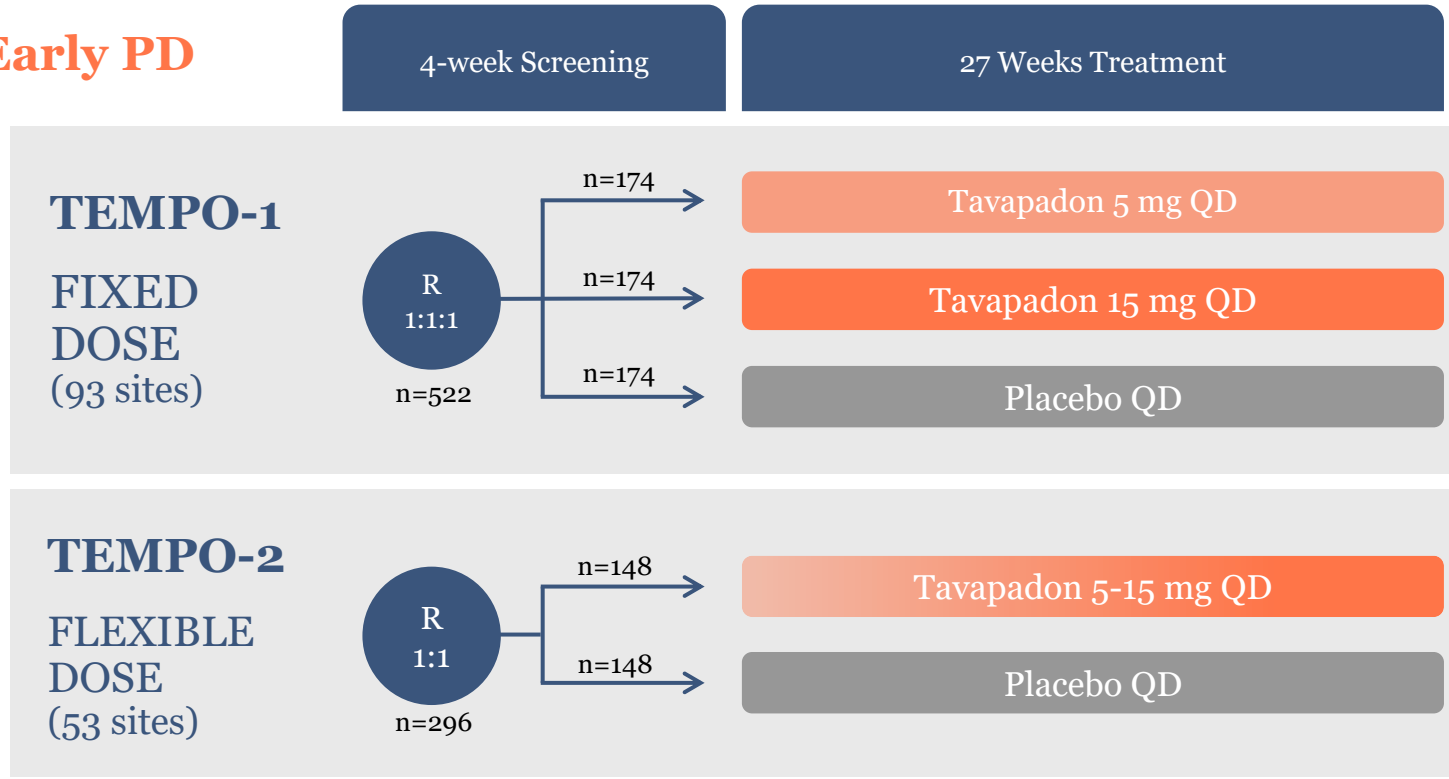
TEMPO-1 & TEMPO-2: Phase 3 in Early PD

Key inclusion criteria

- Adults 40-80 years old
- Baseline MDS-UPDRS(1) Part III Score ≥10 and Part II Score ≥2
- Modified Hoehn & Yahr(2) stage 1 to 2
- No concomitant meds except MAO-B inhibitors

Primary endpoint

- Change in MDS-UPDRS Parts II+III

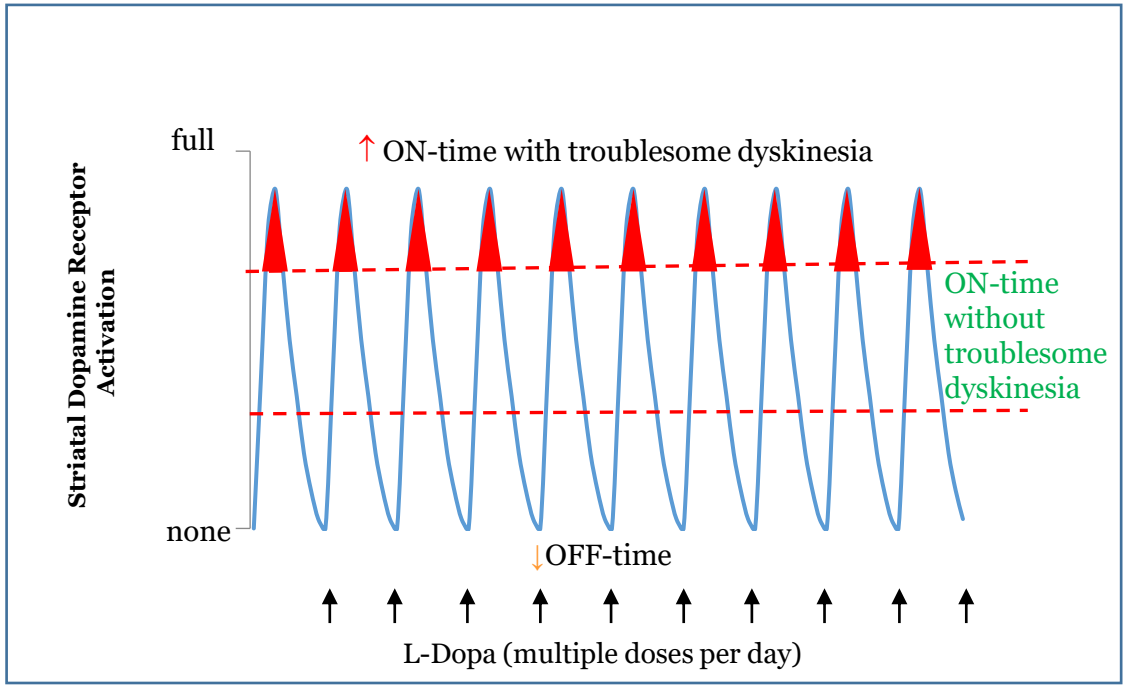


(1) MDS-UPDRS – Movement Disorder Society Unified Parkinson's Disease Rating Scale
(2) Hoehn & Yahr – staging system for characterizing the progression of symptoms for Parkinson's Disease
Note: All studies will include an open-label extension, which will further support the safety database

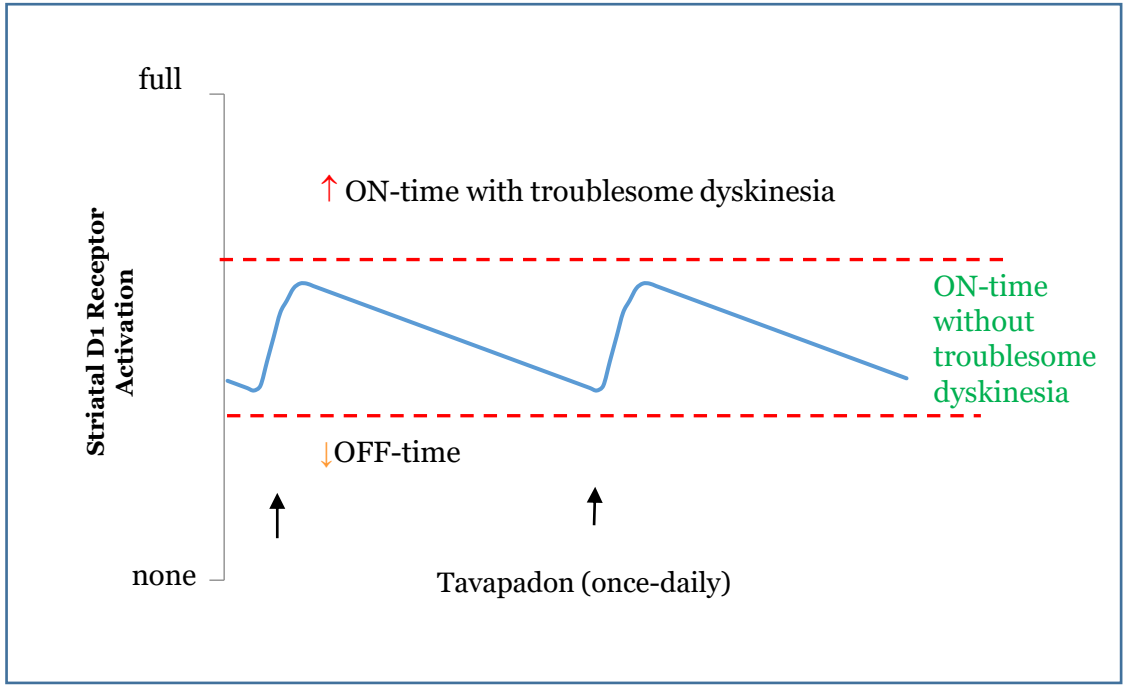
In Late PD, Goal is to Control Motor Fluctuations and Dyskinesias Driven by L-Dopa

L-Dopa vs. Tavapadon in Late-Stage PD[1]

L-Dopa is a **FULL** agonist with **SHORT** half-life



Tavapadon is a **PARTIAL** agonist with **LONG** half-life

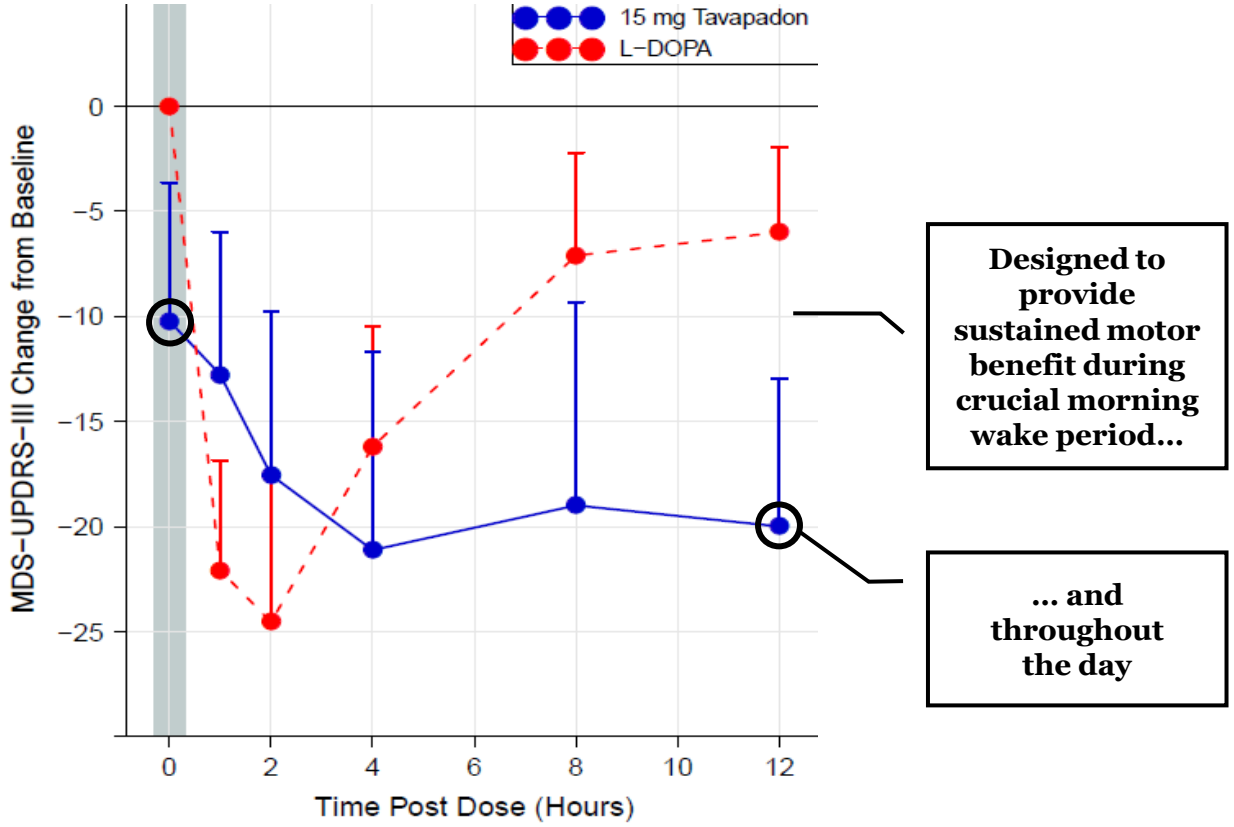


30-40% of patients experience dyskinesias within 5 years on L-Dopa[2]
40% experience OFF-time within 3-5 years on L-Dopa[2]

1) Bastide ME, et al. Prog Neurobiol. 2015;132:96-168
2) Ahlskog JE, Mov Disord 16:448–458

Predictable 24-hour Activity ➔ Sustained Effect: Once Daily, Oral Dosing

Study 1005: Tavapadon in Late-Stage PD[1]



Designed to provide sustained motor benefit during crucial morning wake period…

… and throughout the day

In an open-label Phase 1b trial, Tavapadon demonstrated motor improvement on par with L-Dopa, sustained due to its 24-hour half-life

1) Study B7601005: (n: l-dopa arm= 50, 15 mg = 11). One-sided 90% CI. Phase 1b, two-period open label dose escalation study in patients with Parkinson's disease and motor fluctuations; In period 1 of the study, l-dopa responsiveness was assessed. In period 2, levodopa was washed out and tavapadon was dosed QD over 21 days

Tavapadon TEMPO-3 in Late PD: Data Expected 1H 2023

Registration-directed program includes three global Phase 3 trials optimally designed to maximize treatment effect

TEMPO-3: Phase 3 in Late PD

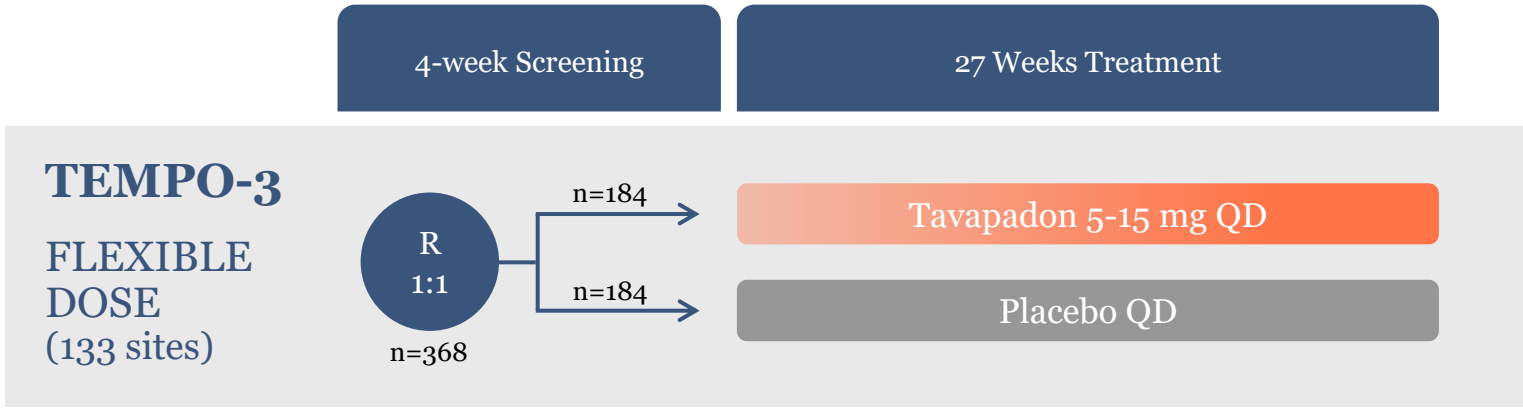
Adjunct to levodopa

Key inclusion criteria

- Adults 40-80 years old
- At least 2.5 hours OFF-time on 2 consecutive days at baseline
- Modified Hoehn & Yahr(1) stage 2 to 3, with response to L-Dopa

Primary endpoint

- Change in ON-time without troublesome dyskinesia



(1) Hoehn & Yahr – staging system for characterizing the progression of symptoms for Parkinson's Disease
Note: All studies will include an open-label extension, which will further support the safety database

Tavapadon Commercial Potential in Parkinson's

Tavapadon Target Profile

- Novel D1/D5 mechanism
- Potential similar or better motor control[1]
- Potential favorable side effect profile[2]
- 1x Once-daily dosing

Pricing & Launch

Branded US price analogs $8-10K+/year

Payor research supports broad Medicare and Commercial coverage at price of $8K+ /year

Strong side effect profile and motor control differentiation would reduce reimbursement restrictions

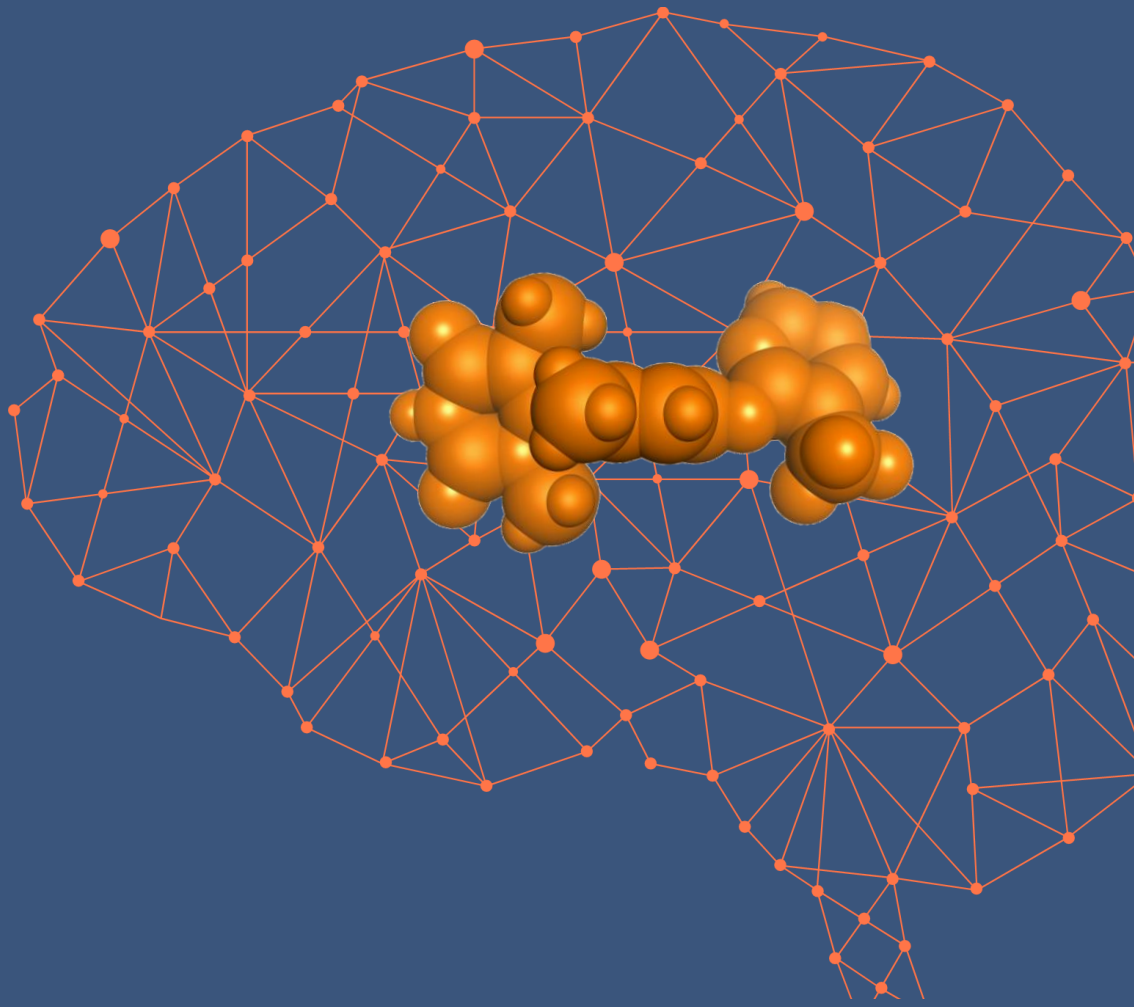
Patients and physician research supports acceptability of branded co-pays for a tavapadon-like differentiated profile

Differentiated profile supports pricing comparable to branded market leaders which have broad reimbursement

(1) As suggested by two Phase 2 studies in late (Study 1003) and early stage PD (Study 1011) and a Phase 1b in advanced PD (Study 1005);
(2) As suggested consistently across 272 subjects across nine clinical trials (four Phase 1 trials, two Phase 1b trials and three Phase 2 trials)

CVL-871 in Dementia-Related Apathy

Partial agonist selectively targeting the dopamine D1 receptor with the goal of modulating motivation and reward pathways to address apathy in patients with mild-to-moderate dementia



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High Unmet Need in Apathy, which Affects ~50% of Patients with Dementia[1]

What is Apathy?

Leading neuropsychiatric symptom in dementia

Social disengagement and loss of emotion leads to:

- Impaired decision-making
- Lack of empathy, affection, or concern
- Loss of interest in personal well-being and relationships
- Inability to initiate and maintain normal daily activities
- Interference with basic function*

CVL-871: Potential to be the First Treatment for Dementia-Related Apathy

High Unmet Need

- >50M Dementia Patients Worldwide[2]
- >3x Mortality Risk[3]
- Among strongest predictors of disease progression[4]
- Early institutionalization & ↑ caregiver burden[4]

No Approved Treatment

Off-label use of…

Acetylcholinesterase Inhibitors	No proven effect
SSRIs/SNRIs	No established benefit May worsen apathy symptoms
Methylphenidate	Schedule II stimulant with CV risk in elderly patients

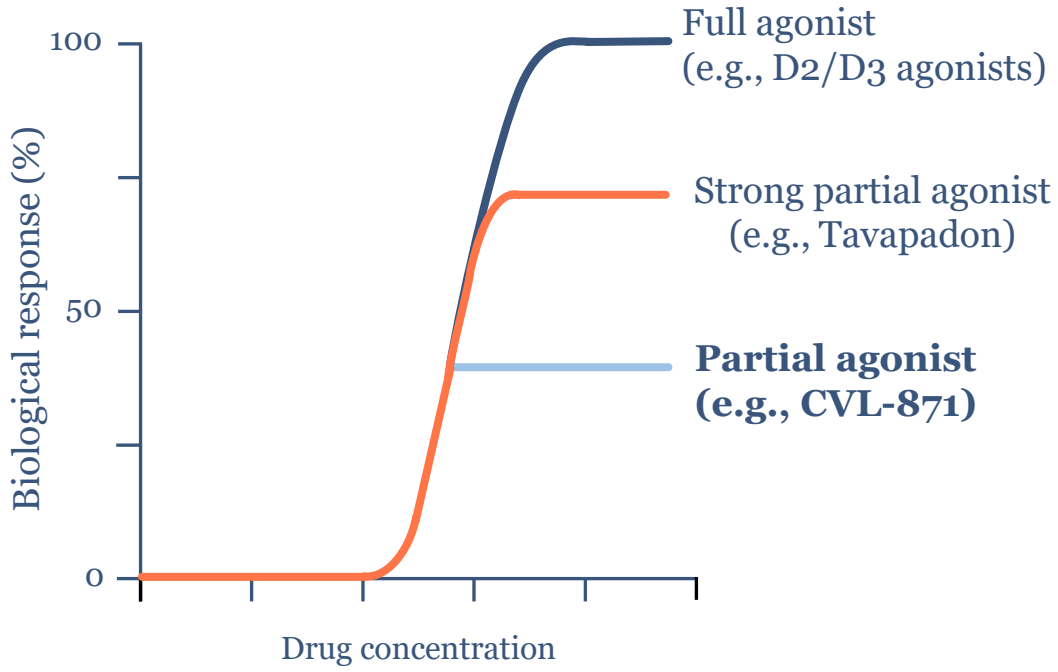
Sources: 1. Zhao, et al. J Affect Disord 2016. 2. ADI, 2015. 3. Linde, et al. Intl Journ Geriatric Psych 2016 4. Lancot et al 2017, Onyike et al 2007, Hongisto et al 2018, Lyketsos et al 2002. Bionest Research, May 2020.
*Including personal hygiene, eating, dressing, taking medication

CVL-871: D1/D5 Partial Agonism for Dementia-Related Apathy

CVL-871 Summary

- Like tavapadon, CVL-871 is a selective D1/D5 partial agonist
- While tavapadon drives up to ~70% biological response at the D1/D5 receptors, CVL-871 has ~40% partial agonism
- Potentially optimal level of agonism for modulating neuronal pathways related to motivation and reward
- Dopaminergic enhancement may improve apathy based on historical studies of methylphenidate
- Potential non-stimulant option for treatment of dementia-related apathy

Degrees of Agonism (Illustrative)



CVL-871 Phase 2a Exploratory Trial: Data Expected 2H 2022

Phase 2a Trial in Dementia-Related Apathy

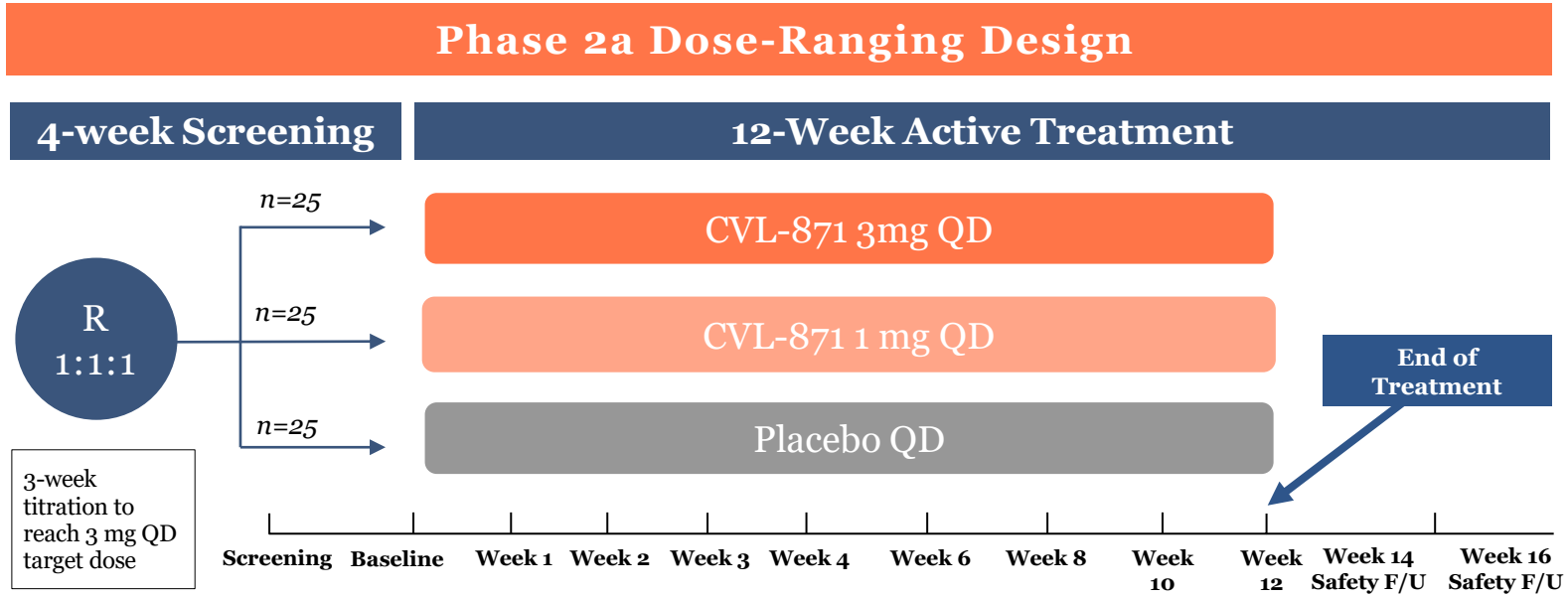
Key inclusion criteria

- Adults 50-85 years old
- NPI-Apathy domain frequency and severity scores each ≥ 2
- Mild-to-moderate dementia
- MMSE 15-26; CDR 0.5-2.0

No primary endpoint

Exploratory efficacy measures

- **Apathy/Global:** NPI/NPI-C, DAIR, AES-C, mADCS-CGIC/CGIS, Caregiver CGIC/CGIS
- **Function:** DAD, Zarit Caregiver Burden
- **Cognition:** ADAS-Cog13, Trail Making A, Digit Span, COWAT



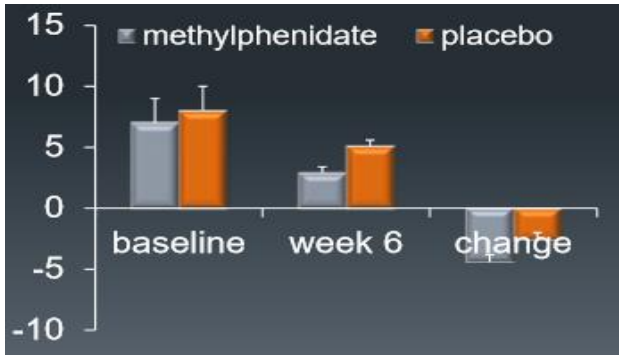
Increased D1 Receptor Activation May Improve Apathy

D1 Activation Potential in Apathy

- Dopamine acting via D1 in the striatum directly promotes motivation and goal-directed behavior
- D1 density reduces with age and reduction in dopamine signaling is associated with behavioral / psychological symptoms of dementia (BPSD)
- Methylphenidate (MPH), an NDRI*, significantly improved apathy in AD patients in 2 independent Phase 2 trials

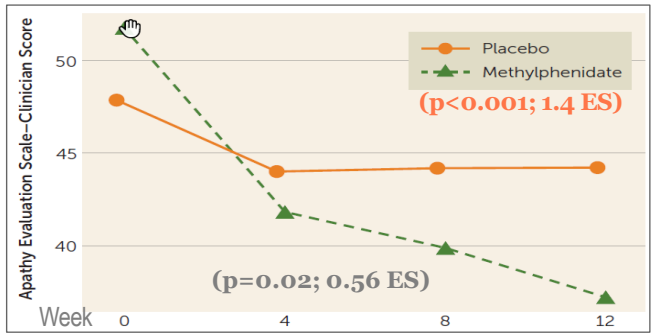
Methylphenidate Phase 2 Trials

ADMET1 Trial: showed NPI Apathy global score improvement of 1.8 points over placebo



ADMET1 Trial-Rosenberg, et al J Clin Psychiatry 2013

Veterans AD Apathy Trial: showed AES-C score improvement of 9.9 points over placebo at week 12



Veterans AD Apathy Trial, Padala et.al, Am J Psychiatry 2018

*NDRI = norepinephrine-dopamine reuptake inhibitor

Sources: Karrer etal, Neurobiol Aging 2017; Volkow etal, Am J Psychiatry, 1998; Flanigan et al, J. Neurosci., 2017

Transforming the Possible in Neuroscience

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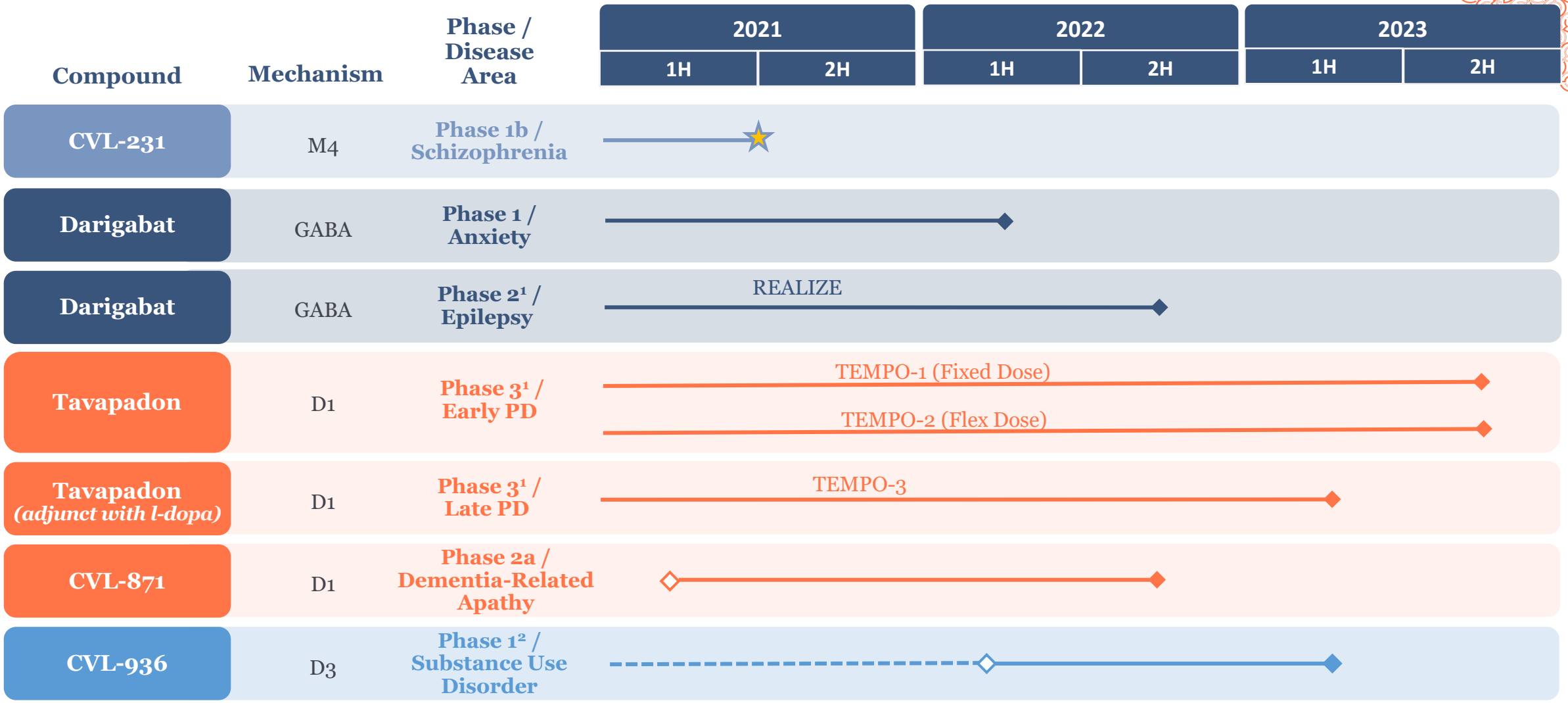
Multiple Milestones Expected Over Next Three Years



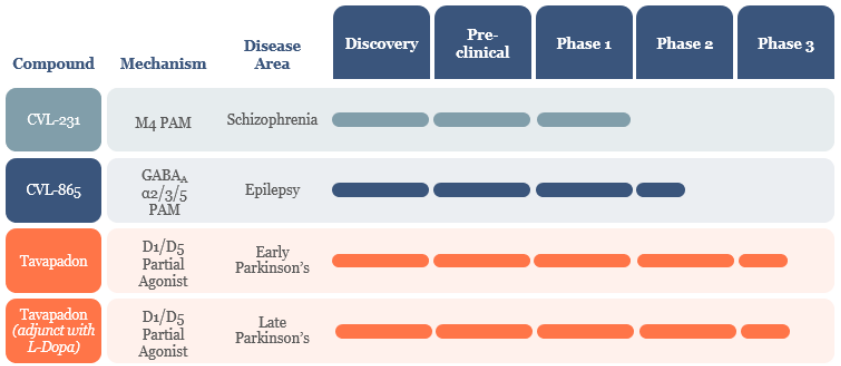
1. In addition, there are two open-label extension trials ongoing (REALIZE OLE for darigabat in epilepsy and TEMPO-4 for tavapadon)
2. We initiated a Phase 1 SAD trial for CVL-936 in January 2020. We concluded dosing of Cohort 1 of the Phase 1 SAD trial. We intend to conduct a non-clinical safety pharmacology study prior to resuming Phase 1 SAD and MAD evaluations.

Cerevel is Transforming Possibilities for Tomorrow

Multiple Programs Aimed at Providing New Options for Millions of Patients

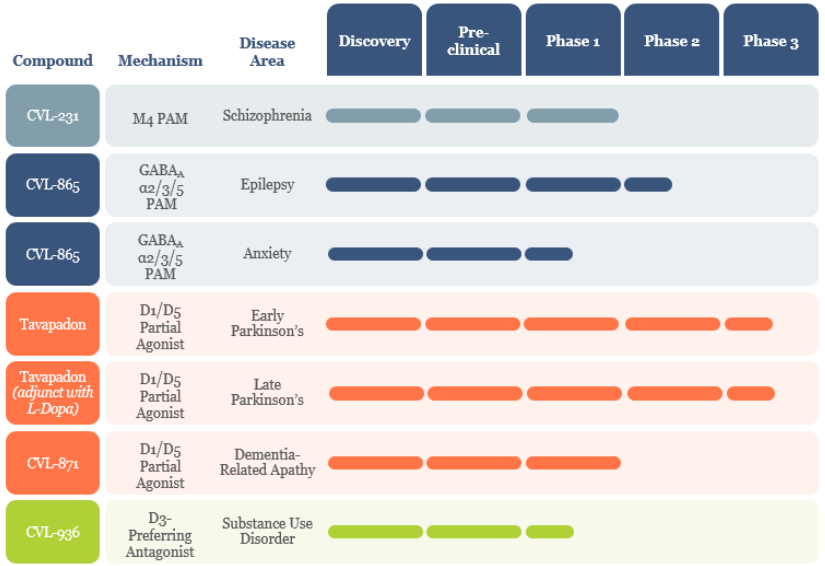
Multiple near-term milestones

- Schizophrenia
- Epilepsy
- Parkinson's



Expansion to other diseases

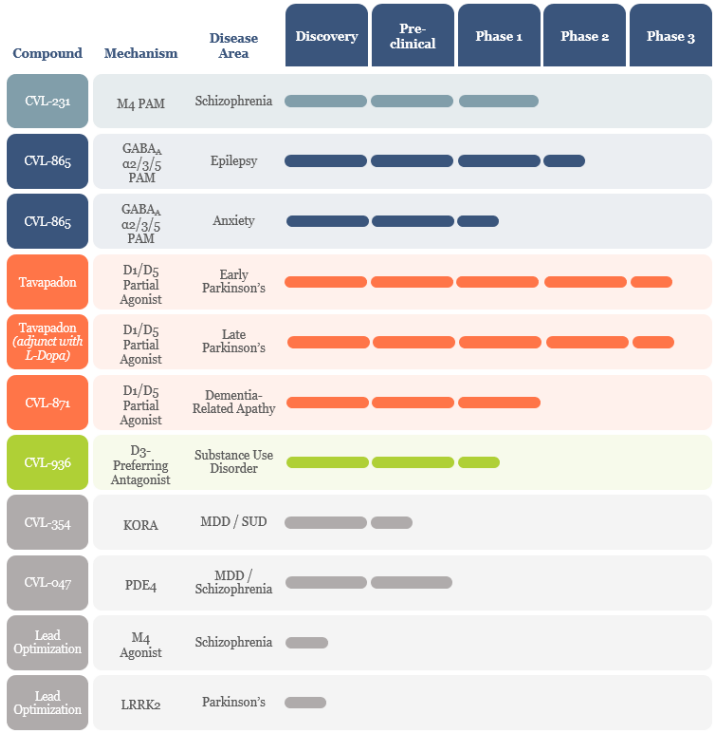
- Alzheimer's Psychosis
- Anxiety
- Apathy
- Substance Abuse Disorder



Long-term discovery efforts

Disease-modifying therapies based on human genetics and novel targets addressing:

- Neuronal loss
- Synaptic health



50M+ Patients WW ⊕ 100M+ Patients WW → Premier Neuroscience Company

Appendix

Who We Are is in Our Name

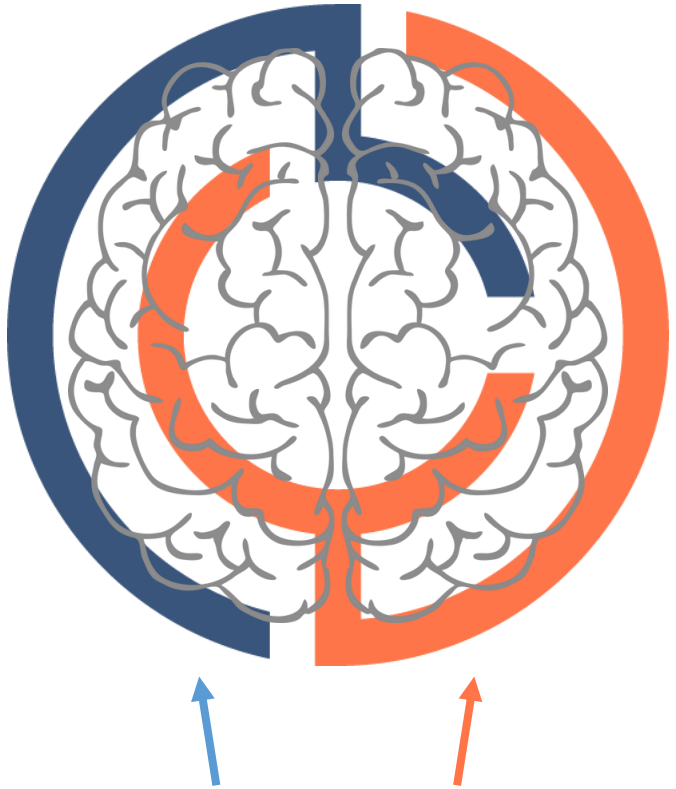
cerevel

cere = cerebrum

revel = revelation/reveal

We are bold thinkers, deep experts, resilient pathfinders, and transparent partners who push the boundaries of scientific understanding to unlock breakthrough CNS therapies that could have real impact on people's lives.

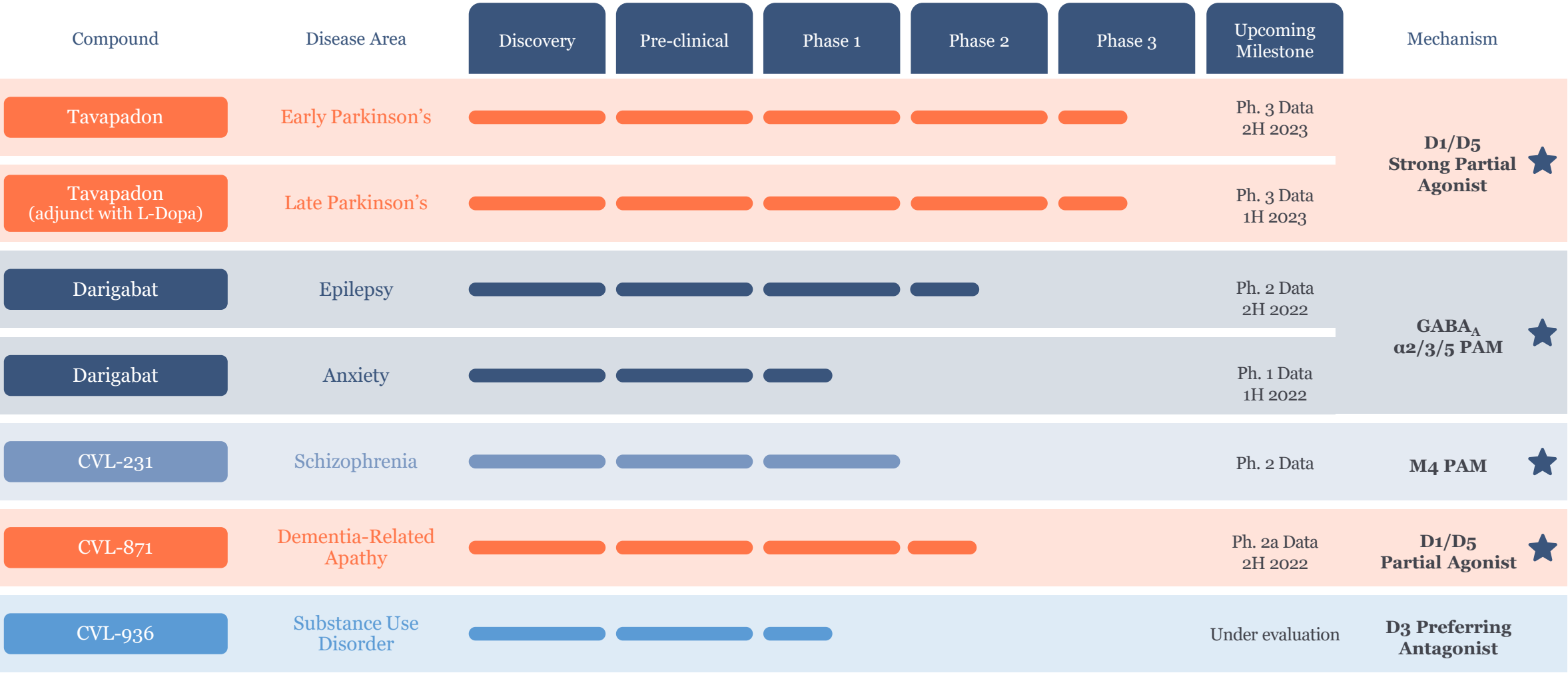
Brain hemispheres



Lock in key

Selective / Targeted Mechanisms

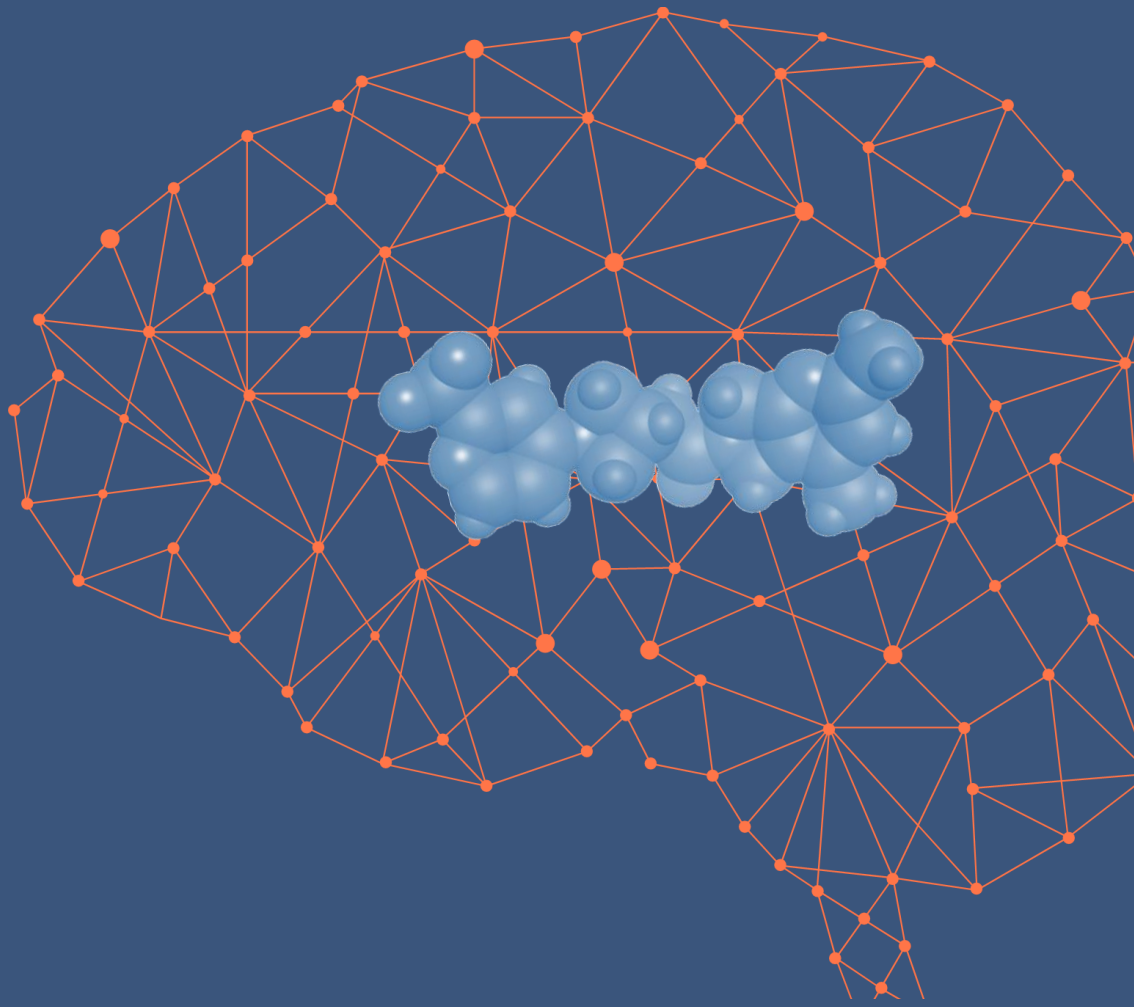
Cerevel Clinical Pipeline: Broad, Deep and Diverse



★ Asset overview included in this presentation

M4 PAM (CVL-231) in Schizophrenia

Additional Slides



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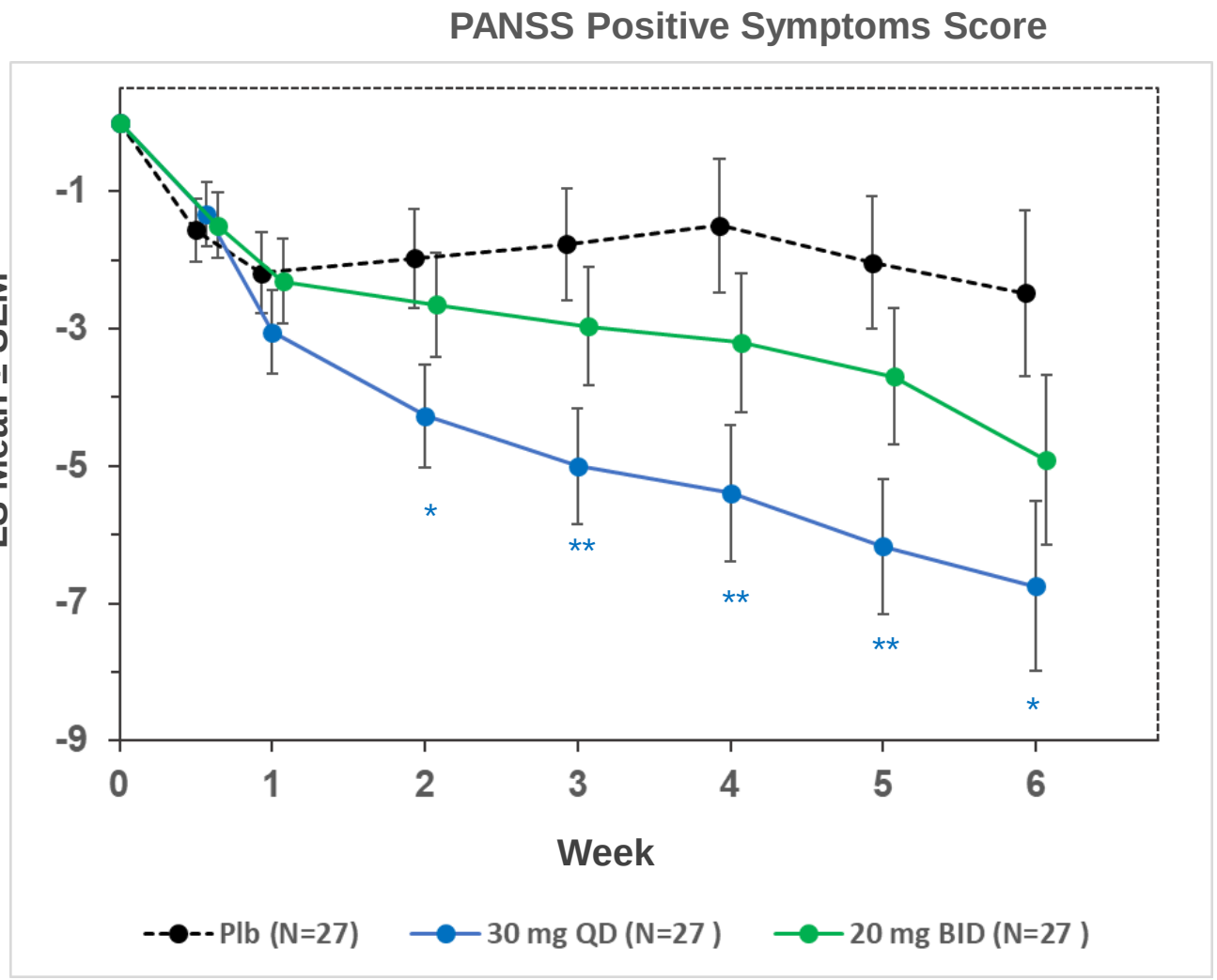
Phase 1b Part B: Demographics & Baseline Characteristics

	PBO N= 27	CVL-231 30 mg QD N= 27	CVL-231 20 mg BID N= 27	All CVL-231 N= 54	Total N= 81
Demographics					
Age (years) at Screening: Mean (SD)	41 (9.7)	41 (8.1)	38 (9.8)	40 (9.0)	40 (9.2)
% Male: N (%)	19 (70%)	23 (85%)	21 (78%)	44 (81%)	63 (78%)
Race: N (%)					
Black or African American	17 (63%)	20 (74%)	19 (70%)	39 (72%)	56 (69%)
White	9 (33%)	7 (26%)	7 (26%)	14 (26%)	23 (28%)
Other	1 (4%)	0	1 (4%)	1 (2%)	2 (2%)
Weight (kg) Prior to Dosing: Mean (SD)	90.0 (16.0)	85.4 (13.3)	85.4 (15.4)	85.4 (14.3)	86.9 (14.9)
Disease Characteristics at Baseline: Mean (SD)					
PANSS Total Score	93 (8.8)	93 (7.3)	97 (7.9)	95 (7.7)	95 (8.1)
PANSS Positive Score	24 (2.7)	25 (3.0)	26 (2.6)	26 (2.8)	25 (2.8)
PANSS Negative Score	23 (3.3)	22 (3.7)	24 (3.8)	23 (3.8)	23 (3.6)
CGI-S Score	5 (0.6)	5 (0.5)	5 (0.7)	5 (0.6)	5 (0.6)

Phase 1b: PANSS Positive Symptoms Score

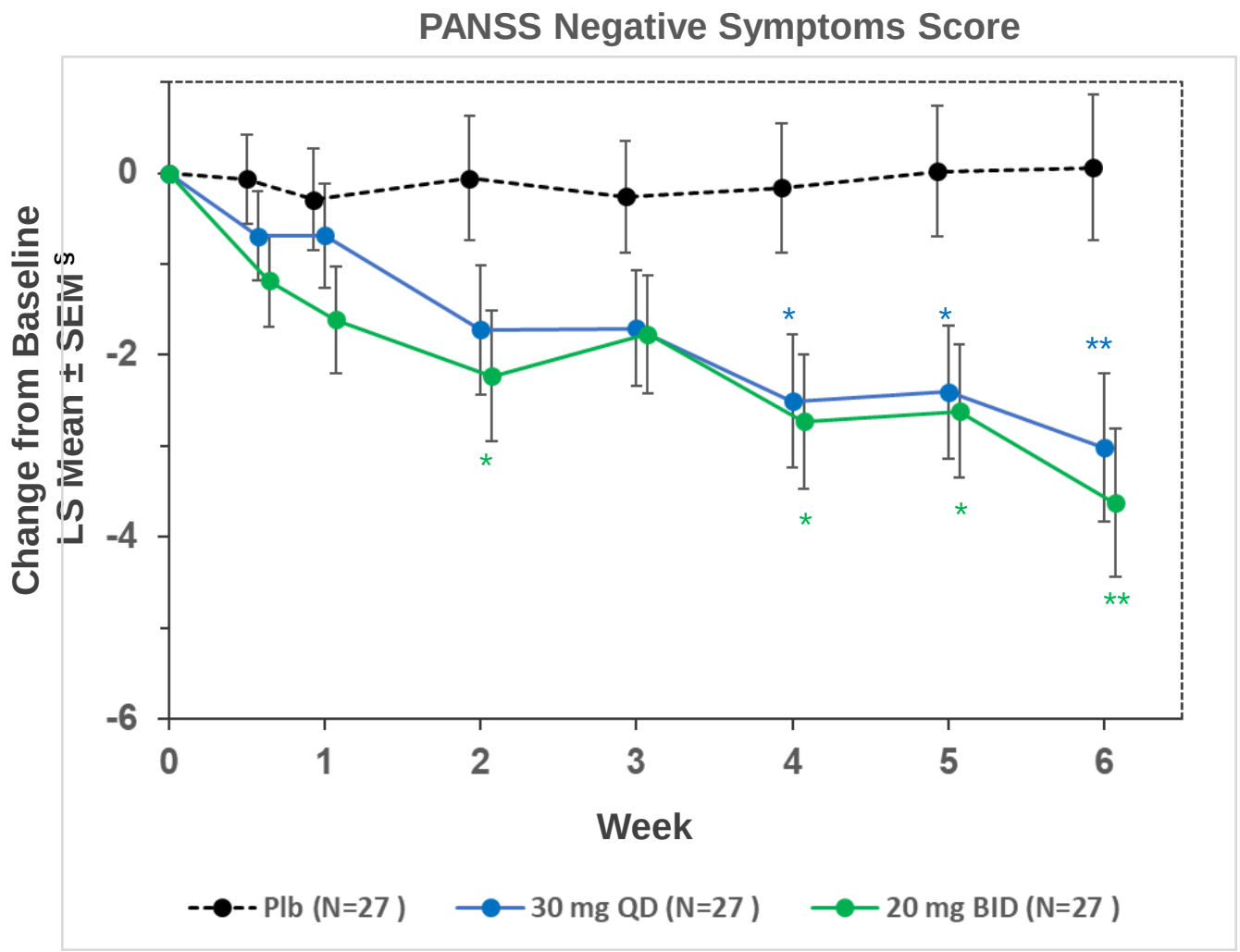


- 30 mg QD: 4.3 Point improvement versus placebo at Week 6 (6.8 of 30 mg QD vs 2.5 placebo) with P=0.016
- 20 mg BID: 2.4 Point improvement versus placebo at Week 6 (4.9 of 20 mg BID vs 2.5 placebo) with P=0.166
- Combined CVL 231: 3.3 Point improvement versus placebo at Week 6 (5.8 of CVL231 vs 2.5 placebo) with P=0.028

* P<0.05 vs Placebo ** P<0.01 vs Placebo

§ Derived from a mixed model for repeated measures (MMRM) with treatment group, visit, treatment group-by-visit interaction as fixed effect, baseline value as a covariate and the measurements within subject as repeated measures. An unstructured covariance matrix was used.

Phase 1b: PANSS Negative Symptoms Score



- 30 mg QD: 3.1 Point improvement versus placebo at Week 6 (3.0 of 30 mg QD vs -0.1 placebo) with P=0.009
- 20 mg BID: 3.7 Point improvement versus placebo at Week 6 (3.6 of 20 mg BID vs -0.1 placebo) with P=0.002
- Combined CVL 231: 3.4 Point improvement versus placebo at Week 6 (3.3 of CVL231 vs -0.1 placebo) with P=0.001

*** P<0.05 vs Placebo ** P<0.01 vs Placebo**

§ Derived from a mixed model for repeated measures (MMRM) with treatment group, visit, treatment group-by-visit interaction as fixed effect, baseline value as a covariate and the measurements within subject as repeated measures. An unstructured covariance matrix was used.

Phase 1b: Safety & Tolerability – Adverse Events

	PBO N= 27	CVL-231 30 mg QD N= 27	CVL-231 20 mg BID N= 27	All CVL-231 N= 54
Days on IMP				
Mean (SD)	37 (9.6)	36 (12.8)	35 (13.6)	36 (13.1)
Range	8, 42	4, 42	2, 42	2, 42
Number (%) Subjects with TEAE	14 (52%)	14 (52%)	15 (56%)	29 (54%)
Number (%) Subjects with TEAE Related to IMP	10 (37%)	7 (26%)	12 (44%)	19 (35%)
Number (%) Subjects with Serious TEAE	0	2 (7%)	1 (4%)	3 (6%)
Number (%) Subjects with AE of Special Interest (AESI)	3 (11%)	2 (7%)	4 (15%)	6 (11%)
Number (%) Subjects with TEAE Leading to Discontinuation of IMP	0	2 (7%)	1 (4%)	3 (6%)

Phase 1b: Safety & Tolerability - Adverse Events

Incidences of All CVL-231 ≥ 2% and > Placebo

	PBO N= 27	CVL-231 30 mg QD N= 27	CVL-231 20 mg BID N= 27	All CVL-231 N= 54
Number (%) Subjects				
Headache	7 (26%)	8 (30%)	7 (26%)	15 (28%)
Nausea	1 (4%)	2 (7%)	2 (7%)	4 (7%)
Back pain	1 (4%)	2 (7%)	1 (4%)	3 (6%)
Blood creatine phosphokinase increased	0	1 (4%)	2 (7%)	3 (6%)
Dizziness	0	1 (4%)	2 (7%)	3 (6%)
Dry mouth	0	3 (11%)	0	3 (6%)
Somnolence	0	1 (4%)	2 (7%)	3 (6%)
Pruritus	0	1 (4%)	1 (4%)	2 (4%)

Serious AEs (SAEs) and AEs of Special Interest (AESIs)

	PBO N= 27	CVL-231 30 mg QD N= 27	CVL-231 20 mg BID N= 27	All CVL-231 N= 54
Number (%) Subjects with SAE				
COVID-19	0	0	1 (4%)	1 (2%)
Accidental overdose**	0	1 (4%)	0	1 (2%)
Schizophrenia**	0	1 (4%)	0	1 (2%)
Number (%) Subjects with AESI*				
Blood pressure increased	2 (7%)	0	0	0
Heart rate increased	1 (4%)	0	1 (4%)	1 (2%)
Blood pressure diastolic increased	0	0	1 (4%)	1 (2%)
Sinus tachycardia	0	0	1 (4%)	1 (2%)
Psychotic disorder**	0	0	1 (4%)	1 (2%)
Schizophrenia**	0	1 (4%)	0	1 (2%)
Accidental overdose**	0	1 (4%)	0	1 (2%)

***AEs leading to discontinuation of treatment with IMP. No other AE leading to discontinuation of IMP*

* AESIs specified in the protocol include 1. AEs that result in the discontinuation of treatment with IMP, 2. events that satisfy the criteria for suspected Hy's Law (ALT or AST >3 × ULN, AND serum bilirubin ≥2 × ULN, AND alkaline phosphatase <2 × ULN), 3. events of heart rate >120 bpm, and confirmed by ECG, 4. events of confirmed QTcF is >500 msec or the increase from baseline is >75 msec in subjects without significant hypokalemia, 5. events of mean triplicate blood pressure reading >160 mmHg systolic or >100 mmHg diastolic with confirmation.

Safety & Tolerability

Cardiovascular AESI Summary

- Vital sign and ECG abnormalities defined in the AESI section of the protocol required immediate reporting to the sponsor for collection and risk characterization regardless of clinical significance.
  - Protocol defined CV abnormality thresholds included HR >120 bpm, systolic BP >160 mmHg, or diastolic BP >100 mmHg.
- Six (6) subjects had CV abnormality threshold AESI's reported (3 on active treatment, 3 on placebo). No subjects were symptomatic, and no events were considered clinically significant or associated with other reported AEs.

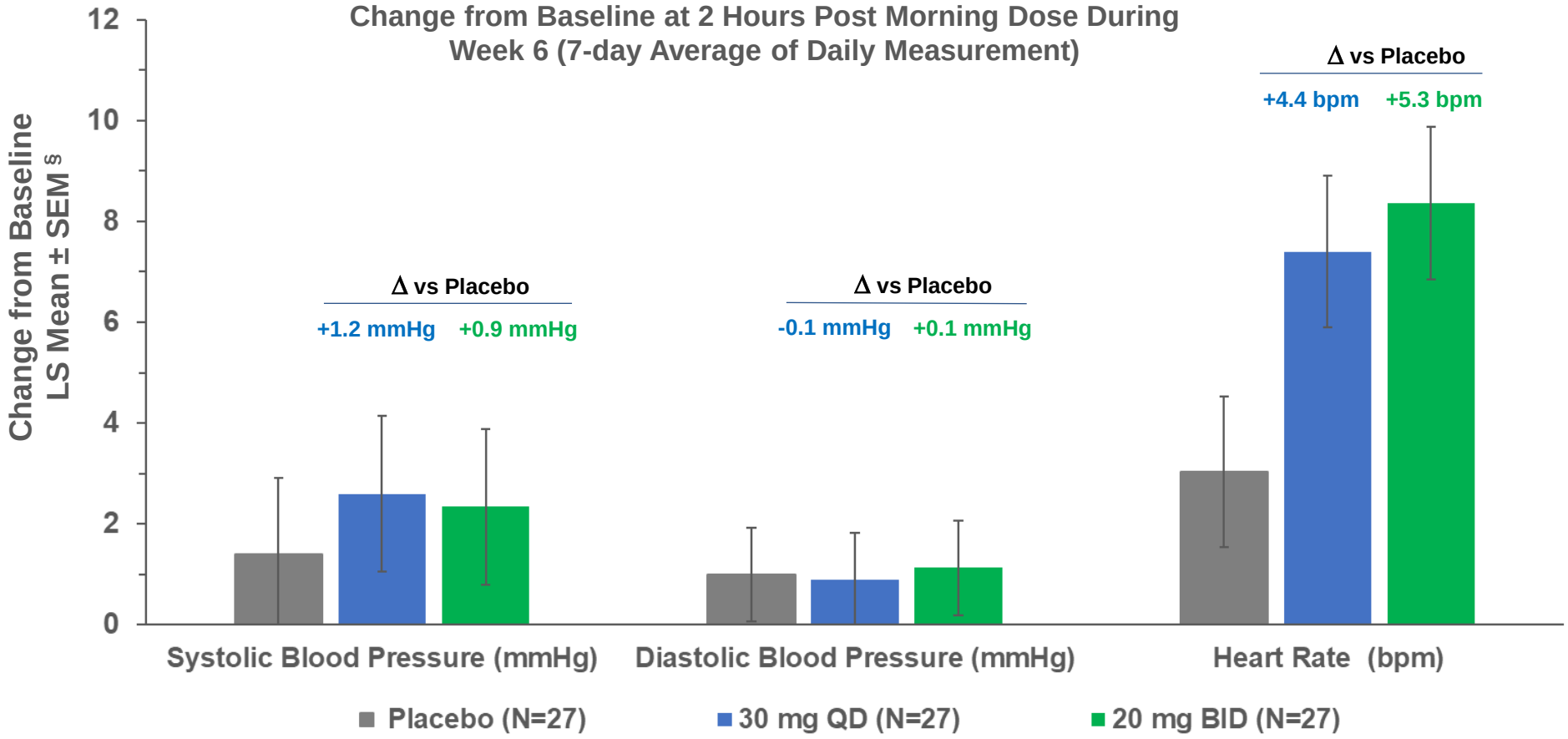
	Subject	Treatment	AESI PT (severity)	Start Date	End Date	Baseline value	Vital Sign Abnormality
Placebo	1	Placebo	Blood pressure increased (mild)	Day 23 (2 hrs post a.m. dose)	Day 23	Systolic BP: 121 mmHg	Systolic BP: 168 mmHg
	2	Placebo	Blood pressure increased (mild)	Day 10 (2 hrs post a.m. dose)	Day 10	Systolic BP: 127 mmHg	Systolic BP: 162 mmHg
	3	Placebo	Heart rate increased (mild)	Day 21 (2 hrs post p.m. dose)	Day 22	HR: 75 bpm	HR: 128 bpm
CVL-231 (20 mg BID)	4	CVL-231 20 mg BID	Heart rate increased (mild)	Day 21 (2 hrs post p.m. dose)	Day 22	HR: 78 bpm	HR: 121 bpm
	5	CVL-231 20 mg BID	Sinus tachycardia (moderate)	Day 1 (2 hrs post a.m. dose)	Day 1	HR: 83 bpm	HR: 123 bpm
	6	CVL-231 20 mg BID	Blood pressure diastolic increased (mild)	Day 25 (2 hrs post p.m. dose)	Day 26	Diastolic BP: 81 mmHg	Diastolic BP: 111 mmHg
				Day 28 (2 hrs post p.m. dose)	Day 29	Diastolic BP: 81 mmHg	Diastolic BP: 103 mmHg
				Day 39 (2 hrs post p.m. dose)	Day 40	Diastolic BP: 81 mmHg	Diastolic BP: 104 mmHg

* AESI = Adverse Events of Special Interest. AESIs specified in the protocol include 1. AEs that result in the discontinuation of treatment with IMP, 2. events that satisfy the criteria for suspected Hy's Law (ALT or AST >3 × ULN, AND serum bilirubin ≥2 × ULN, AND alkaline phosphatase <2 × ULN), 3. events of heart rate >120 bpm, and confirmed by ECG, 4. events of confirmed QTcF is >500 msec or the increase from baseline is >75 msec in subjects without significant hypokalemia, 5. events of mean triplicate blood pressure reading >160 mmHg systolic or >100 mmHg diastolic with confirmation.

Safety & Tolerability

Blood Pressure and Heart Rate Effects

- Modest elevations in SBP, DBP, and HR that were observed with CVL-231 compared to placebo that decreased over time, with the average change from baseline during Week 6 in SBP, DBP, and HR for both the 30 mg QD and 20 mg BID groups showing no clinically meaningful difference versus placebo

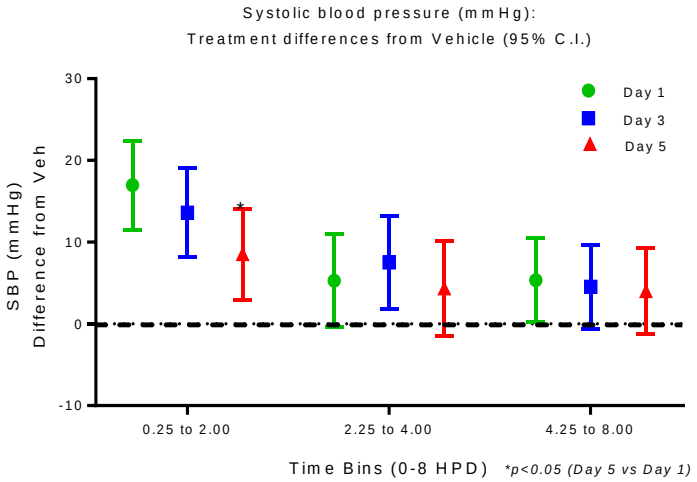


§ Derived from a mixed model for repeated measures (MMRM) for weekly average with treatment group, visit, treatment group-by-visit interaction as fixed effect, baseline value as a covariate and the measurements within subject as repeated measures. An unstructured covariance matrix was used.

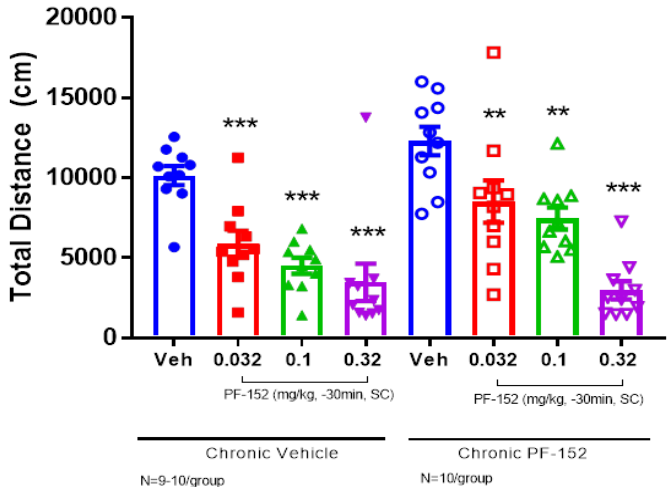
Cardiovascular Effects may be Attenuated with Titration and Repeat Dosing

Repeated dosing of M4 Agonist Tool in rodents showed attenuation of cardiovascular effects without impact on antipsychotic activity; in addition, CVL-231 showed attenuation of heart effects in a 3-month canine toxicology study

5-Day repeat dosing of M4 Agonist Tool: Attenuation of Blood Pressure Effects in Mice



14-Day repeat dosing of M4 Agonist Tool: No Attenuation of Antipsychosis in Mice



3-Month Study of CVL-231: Attenuation of Heart Rate Effects in Canines

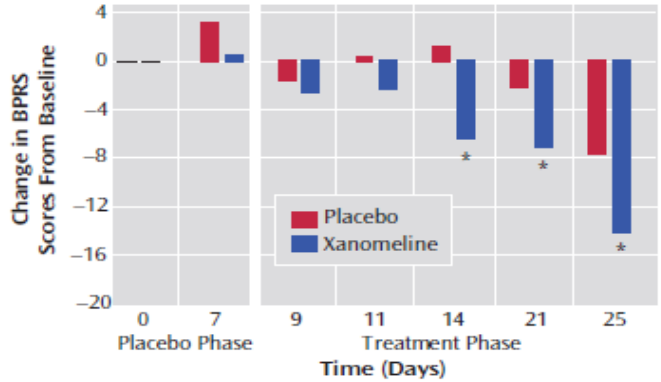
- On Day 1, observed heart rate increases were statistically significant and outside normal range
- On Days 43 and 90, heart rate increases were small and not statistically significant; all mean heart rate values were within normal range and not considered adverse

Xanomeline Clinical Data: Compelling Activity, Limited by Side Effect Profile

Xanomeline (Non-selective Agonist) Impacted Symptoms…

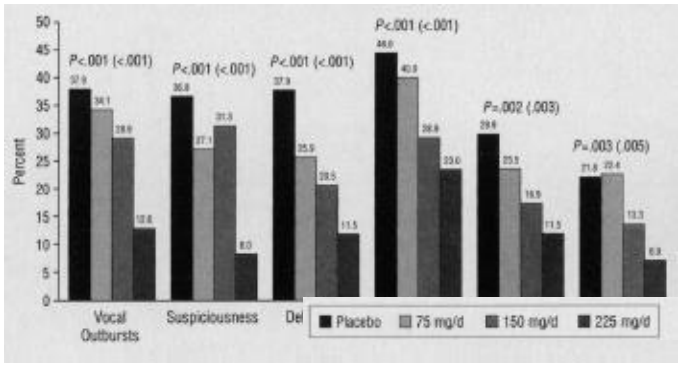
2008 Phase 2 in Schizophrenia

Statistically significant impact on total BPRS and PANSS scores in schizophrenia patients[1]



1997 Phase 2 in Alzheimer's Disease

Statistically significant impact on agitation and other psychosis-related endpoints in Alzheimer's patients[2]



…But Development Was Limited by GI Side Effects

Table 3. Adverse Events*

Event	Placebo (n=87)	Dose† Low (n=85)	Medium (n=83)	High (n=87)	Total (N=342)	P‡
Sweating	4 (4.6)	12 (14.1)	38 (45.8)	66 (75.9)	120 (35.1)	<.001
Nausea	17 (19.5)	24 (28.2)	29 (34.9)	45 (51.7)	115 (33.6)	<.001
Vomiting	8 (9.2)	11 (12.9)	33 (39.8)	37 (42.5)	89 (26.0)	<.001
Dyspepsia	7 (8.0)	20 (23.5)	23 (27.7)	21 (24.1)	71 (20.8)	.007
Chills	1 (1.1)	8 (9.4)	22 (26.5)	32 (36.8)	63 (18.4)	<.001
Chest pain	1 (1.1)	5 (5.9)	13 (15.7)	10 (11.5)	29 (8.5)	.004
Increased salivation	0 (0)	2 (2.4)	6 (7.2)	21 (24.1)	29 (8.5)	<.001
Syncope	4 (4.6)	3 (3.5)	11 (13.3)	11 (12.6)	29 (8.5)	.03
Fecal incontinence	0 (0)	4 (4.7)	1 (1.2)	6 (6.9)	11 (3.2)	.04
Nausea and vomiting	2 (2.3)	0 (0)	1 (1.2)	7 (8.0)	10 (2.9)	.009
Dysphagia	1 (1.1)	0 (0)	2 (2.4)	6 (6.9)	9 (2.6)	.03

*Only events statistically significant at P<.05 are given. Values are number (percentage) of patients unless otherwise indicated.
†Low-dose group received 25 mg of xanomeline tartrate 3 times a day; medium, 50 mg 3 times a day; high, 75 mg 3 times a day.
‡Pearson χ^2 test.

Xanomeline's high rate of nausea, vomiting and dyspepsia is likely driven by non-selective muscarinic agonism

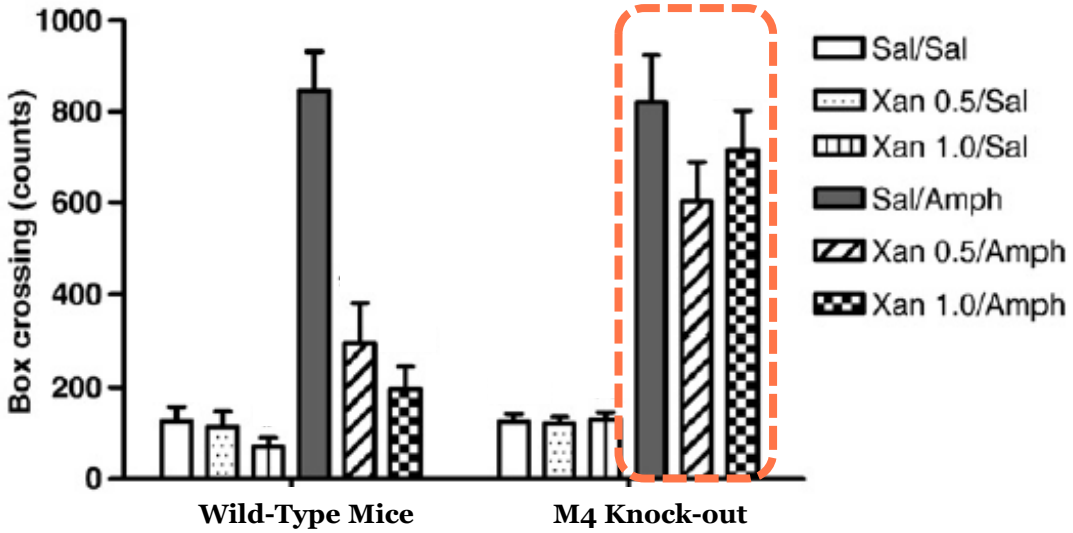
Mechanism Supported by Phase 2 Data for KarXT

- Karuna is developing a BID fixed-dose combination of xanomeline with trospium to offset side effects of GI, dry-mouth and constipation
- In a 5-week Phase 2 study, KarXT demonstrated an 11.6 point reduction in PANSS total score from baseline vs. placebo (p<0.0001)
- ~70 completers in each arm, with discontinuation rates similar between placebo and treatment group
- Represents a robust reproduction of 2008 xanomeline Phase 2 data in schizophrenia

Source: 1. Shekhar et al. Am J Psychiatry, Vol 165, Aug 2008. N=20. 2 active and 3 placebo-arm patients discontinued the study, none due to adverse events.
2. Bodick et al. Arch Neurol, Vol 54, Apr 1997. N = 343. 52% of patients in high-dose arm discontinued treatment due to adverse events.

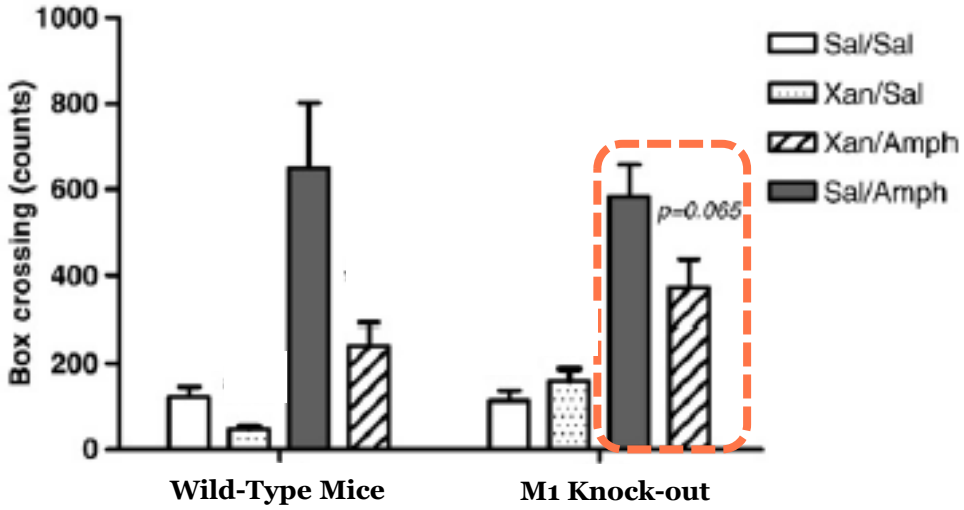
Preclinical Evidence: M4 Modulation Drives Antipsychosis

No Effect of Xanomeline in M4 Knock-out Mouse Model



Xanomeline had no effect on amphetamine-induced hyperactivity in M4 knock-out mice

Reduction in Hyperactivity in M1 Knock-out Mice



Xanomeline reduced hyperactivity in M1 knock-out mice

In mouse studies, M4 receptors drive the antipsychotic activity of xanomeline

Source: Woolley, et al. European Journal of Pharmacology 603 (2009)

Important Insights on Side Effects of M4 PAM

Results of a Phase 1 SAD trial indicated CVL-231 was generally well-tolerated with asymptomatic transient effects on heart rate and blood pressure

Phase 1 SAD Trial (N=17)

- Tested doses up to 30 mg
- Relatively well tolerated with no SAEs
- Most frequently reported treatment-emergent AEs: fatigue, dizziness, headache and dry mouth
- Moderate treatment-emergent transient increases in blood pressure and pulse rate observed
- Cardiovascular effects were asymptomatic and transient in nature

Insights

- Preclinical studies show CV effects attenuated with repeat dosing
- KarXT data also suggest that CV effects attenuate over time with repeat dosing
- Tolerability may be differentiated in schizophrenia patients and CV effects may be attenuated with repeat dosing and/or titration.

Phase 2 data for MK-7622 (M1 PAM) in Alzheimer's disease

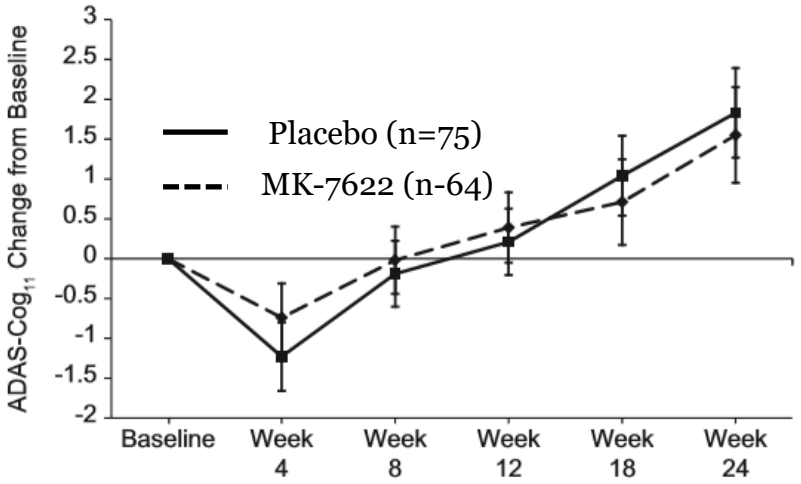
Summary

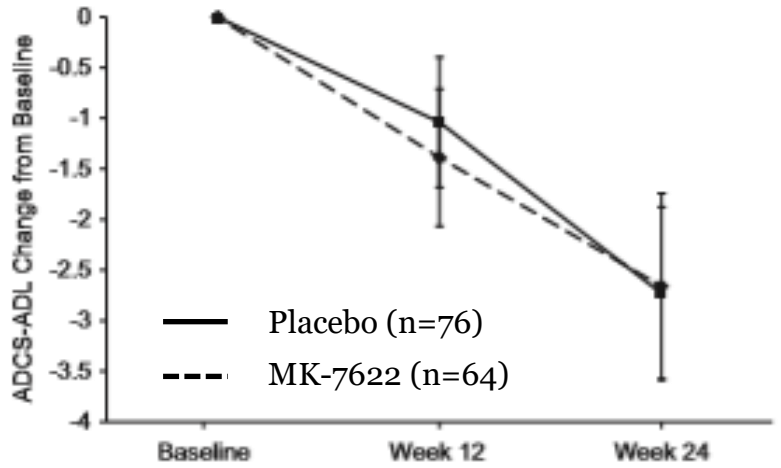
- Randomized double-blind proof-of-concept trial as adjunctive therapy in mild-to-moderate Alzheimer's disease
- Conducted by Merck; data published 2018
- Trial stopped early for futility

Results

- **No difference from placebo on either cognition or activities of daily living (ADL) scales**
- Discontinuation rate of 16% on MK-7622 vs 6% on placebo
- Cholinergically-related adverse event rate of 21% on drug vs 8% on placebo

Results in Cognition and ADL





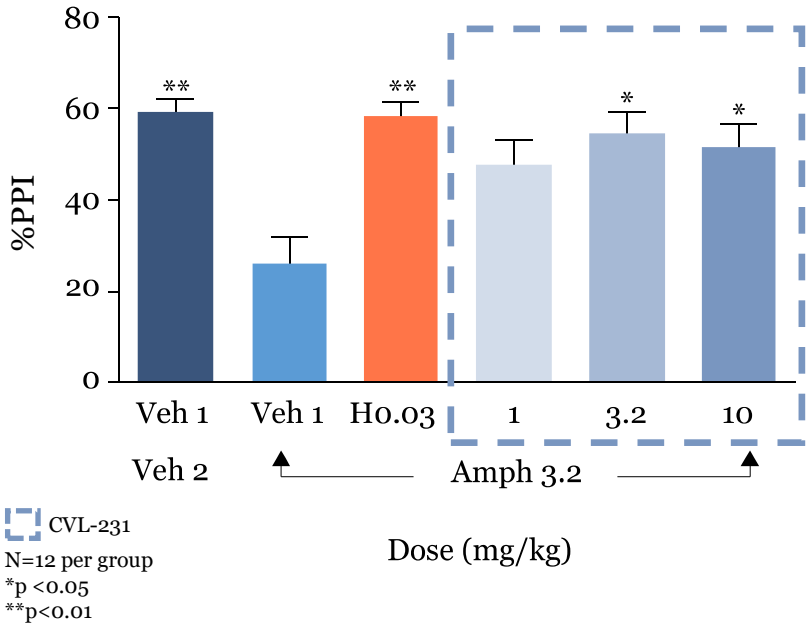
Side Effect Profile

Most Common AEs (>5%)	MK-7662 (n=119)	Placebo (n=120)
Diarrhea	18 (15.1%)	7 (5.8%)
Headache	11 (9.2%)	6 (5.0%)
Rhinorrhea	7 (5.9%)	1 (0.8%)
Urinary Incontinence	6 (5.0%)	0 (0.0%)
Weight Decrease	6 (5.0%)	2 (1.7%)
Urinary Tract Infection	6 (5.0%)	7 (5.8%)
Fall	2 (1.7%)	6 (5.0%)

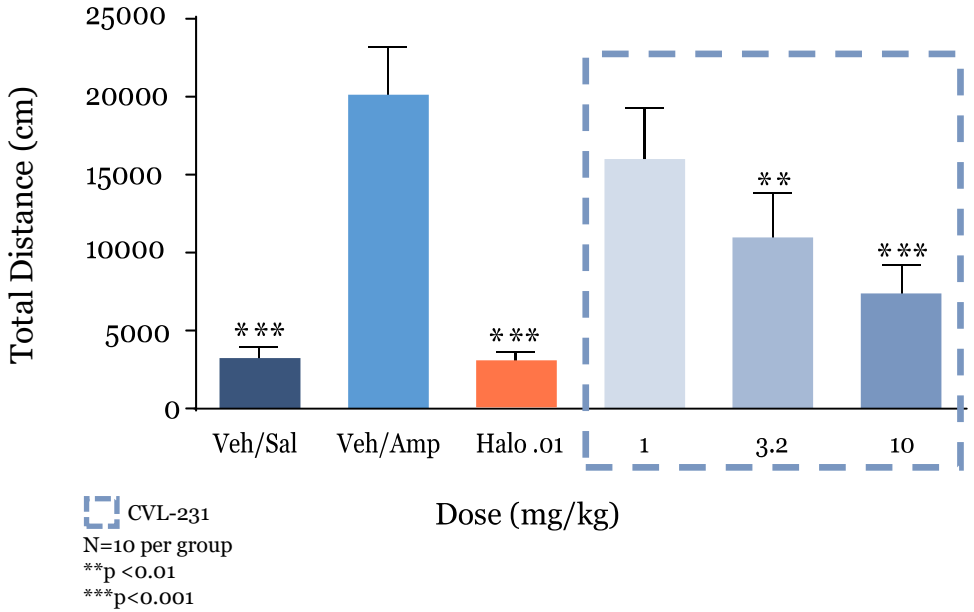
Source: T. Voss et al. / Alzheimer's & Dementia: Translational Research & Clinical Interventions 4 (2018) 173-18 1

M4 PAM Preclinical Data in Psychosis

CVL-231 showed similar effect to haloperidol in reversing amphetamine-disrupted Pre-pulse Inhibition (PPI) in rats



CVL-231 showed dose-dependent reductions on amphetamine-induced locomotion in rats



In multiple rodent models of psychosis, CVL-231 demonstrated antipsychotic activity consistent with atypical antipsychotics

Source: Cerevel studies.

Potential Indications for M4 PAM Beyond Schizophrenia

Pipeline in a Pill

Goal to be a novel MOA and next generation treatment in Schizophrenia

Aiming for a Side Effect and Tolerability Profile Appropriate for Chronic Use in Elderly Populations

Potential Large Indications Worldwide

Schizophrenia	**~21M** Patients
Alzheimer's Psychosis	~20M Patients
Cognition	>50M Patients
PD-LID	~5M Patients

Potential to expand use outside of core schizophrenia population to behavioral and psychological symptoms of dementia

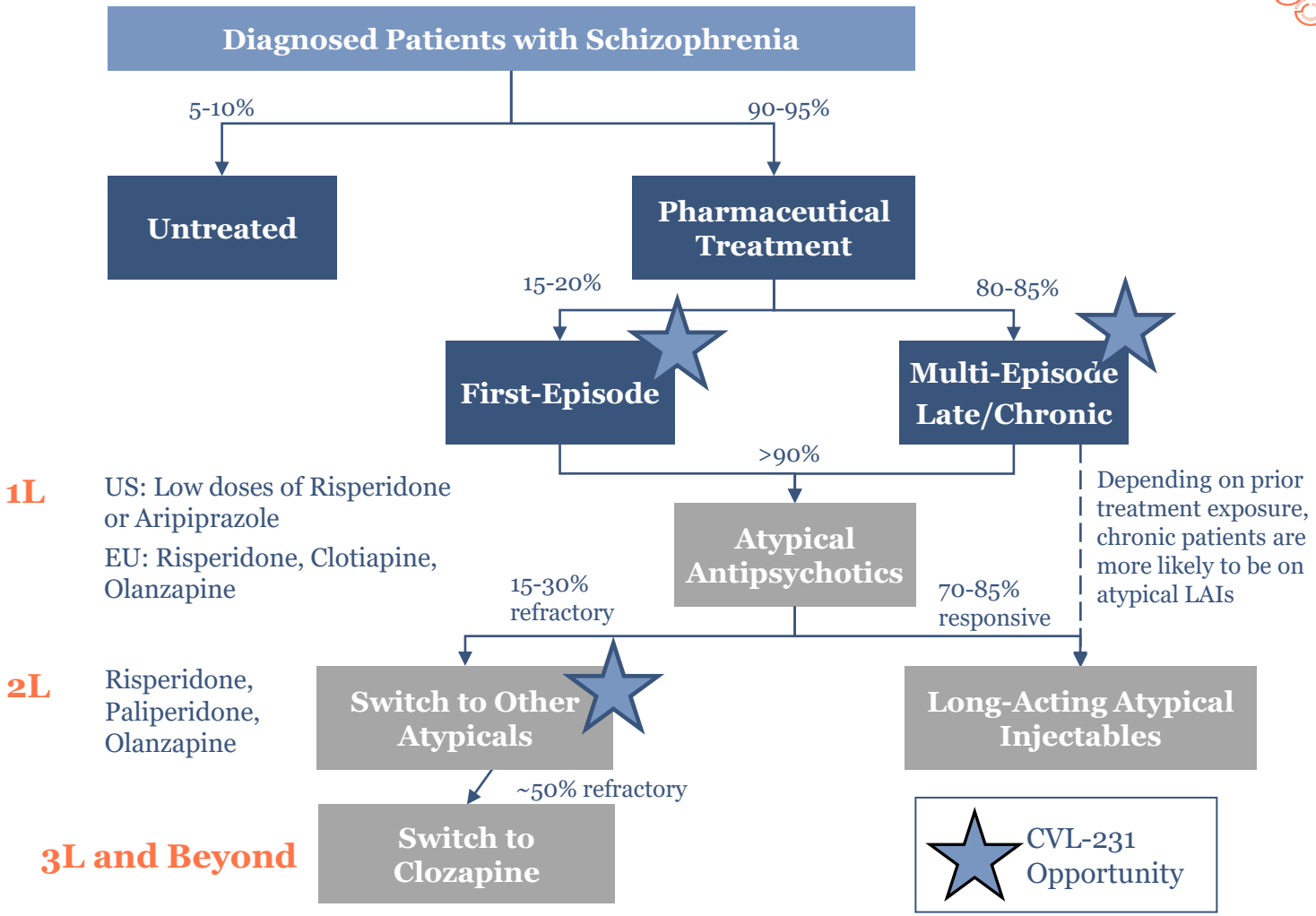
Source: DRG Market Research reports, World Health Organization

CVL-231 Commercial Potential in Schizophrenia

Potential for CVL-231 to be a New Standard of Care

- Potential for improved SE profile could position CVL-231 as first-line treatment of newly diagnosed and ongoing schizophrenia patients, including DRP
- If an improved tolerability and metabolic profile is demonstrated, CVL-231 could displace atypical antipsychotics in patients with treatment-related side effects

Multiple Potential Entry Points for CVL-231 in the Treatment Paradigm

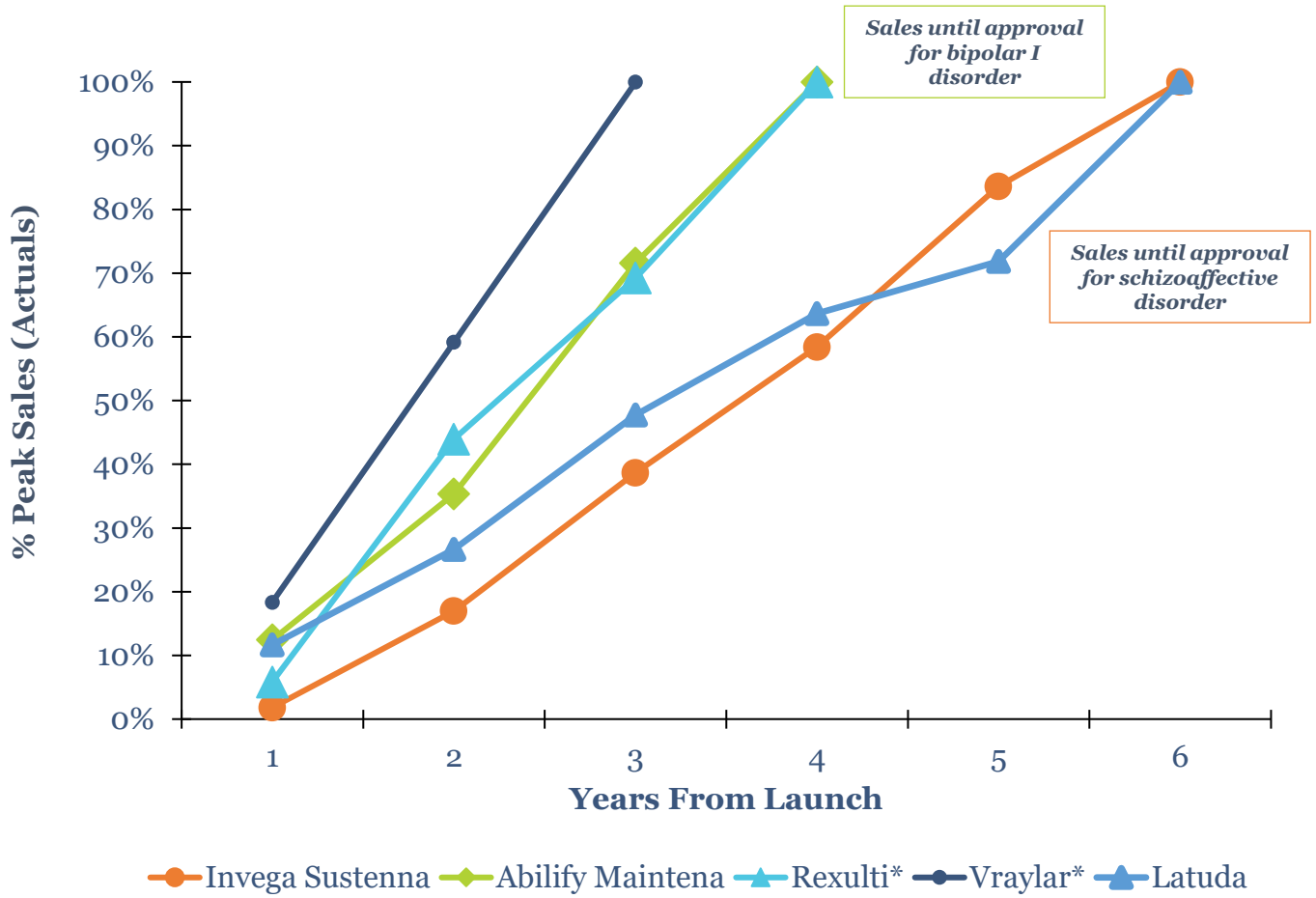


Schizophrenia Therapies: Rapid Historic Uptake Despite Limited Differentiation

Drug	US 2018 Schizophrenia Sales	2018 US Share
Latuda (lurasidone)	$973M	13.5%
Invega Sustenna (paliperidone LAI)	$981M	6.2%
Rexulti (brexpiprazole)	$449M	8.1%
Abilify Maintena (aripiprazole LAI)	$331M	2.1%
Vraylar (cariprazine)	$164M	2.6%

Schizophrenia US Sales Ramp – Actuals

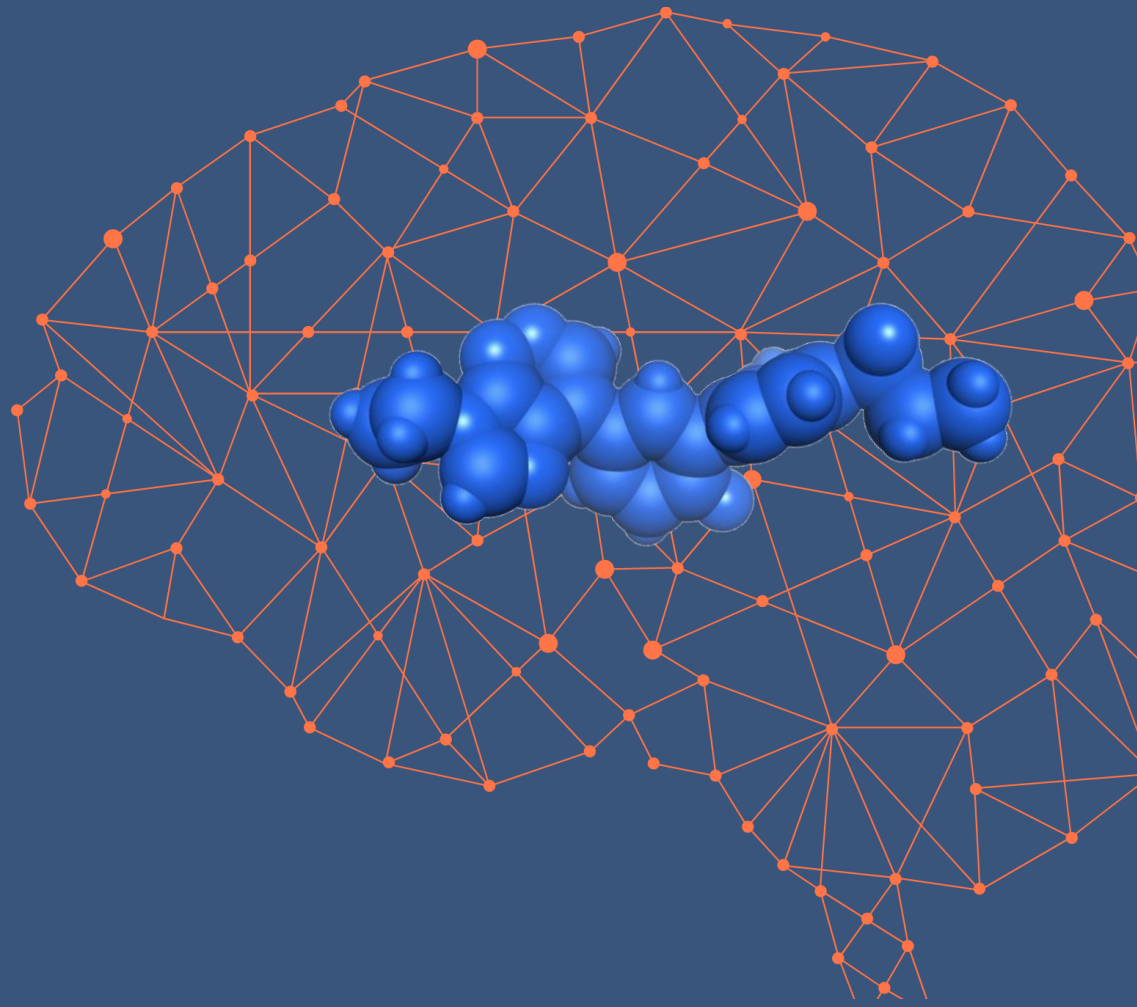
(through 2018 or until first non-schizophrenia indication launch)



Source: Huron analysis, EvaluatePharma.
*Represents sales until first non-schizophrenia indication launch.

Darigabat in Epilepsy

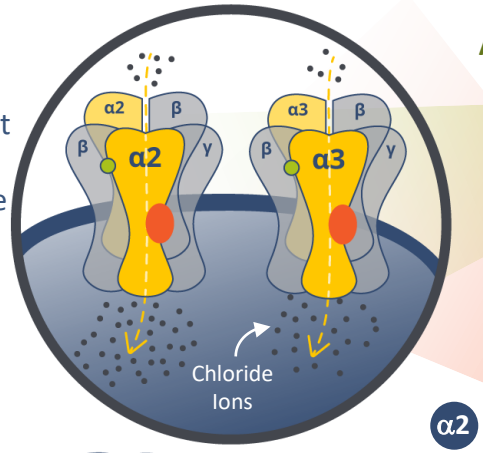
Additional Slides



cerevel

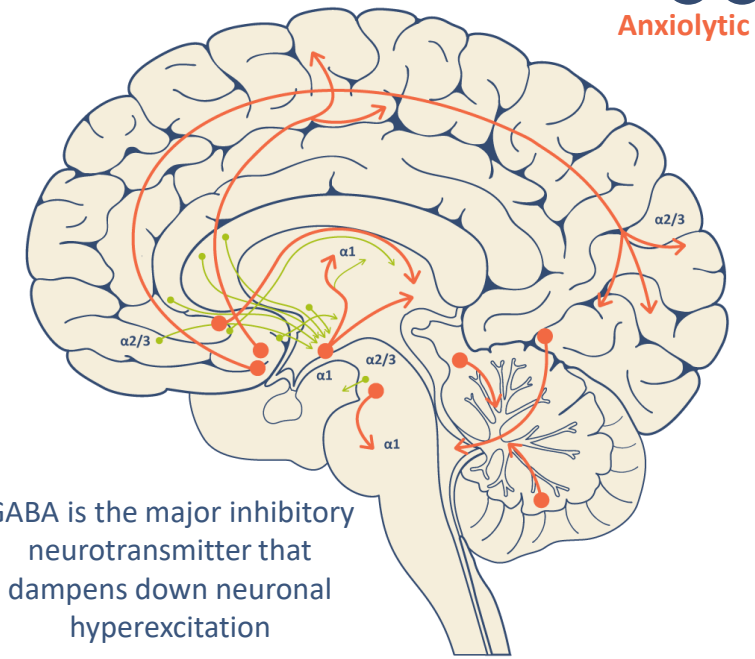
Darigabat Mechanism: Selective α2/3/5 $GABA_A$ Receptor PAM

α2 α3 α5 Darigabat is designed to selectively enhance $GABA_A$ receptor activity at α2/3/5 subunits

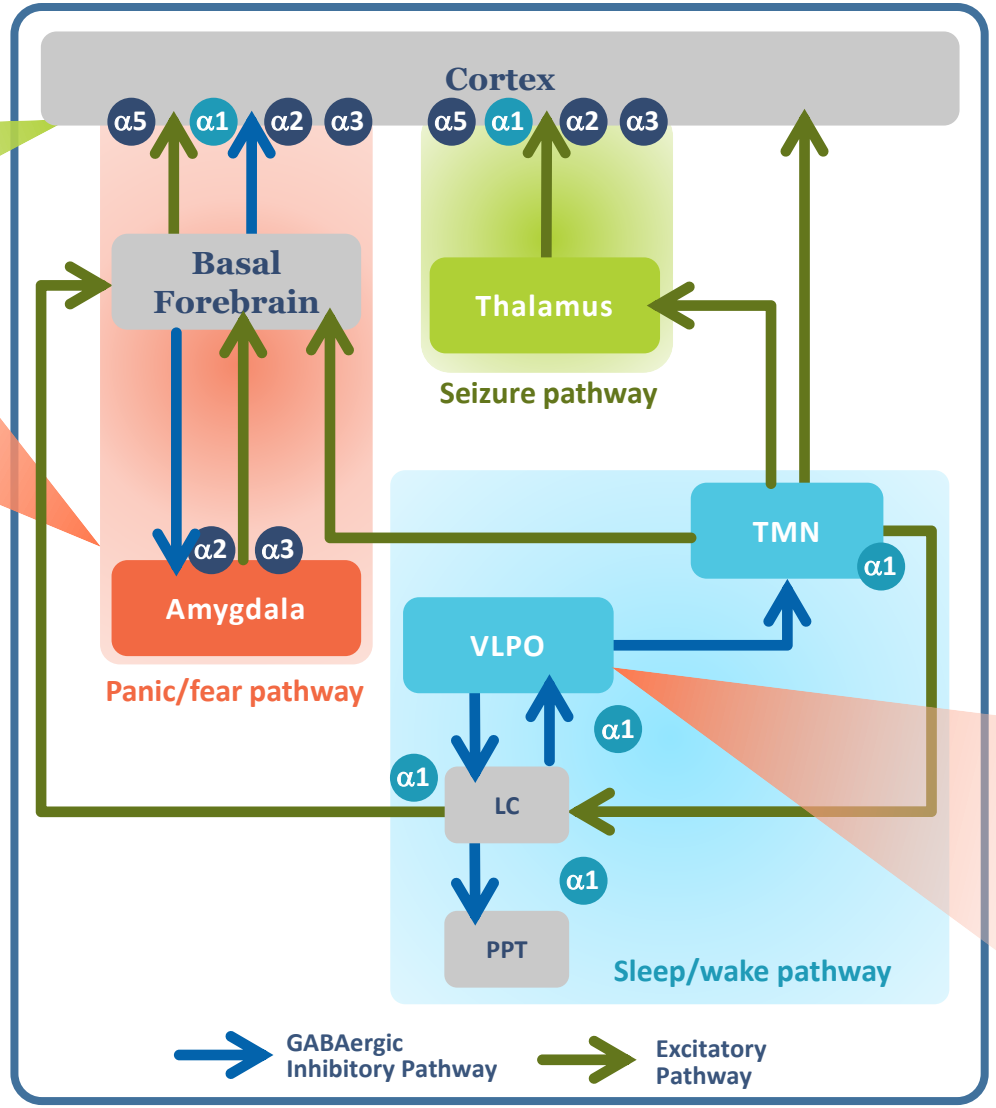


α1 α2 Anticonvulsant effect

α2 α3 Anxiolytic effect



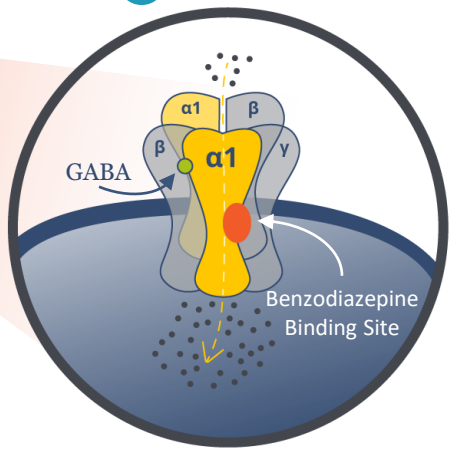
GABA is the major inhibitory neurotransmitter that dampens down neuronal hyperexcitation



α1 Benzodiazepines ***non-selectively*** enhance $GABA_A$ receptor activity, which can cause side effects primarily driven by α1 subunit activation

- Sedation
- Cognitive impairment
- Addiction

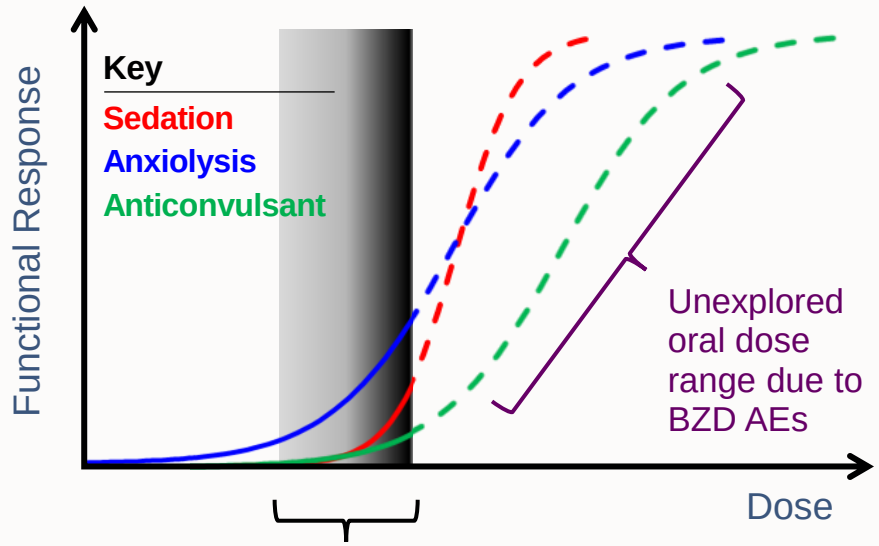
α1 Sedative effect



The Problem With Benzodiazepines (abridged…)

- BZDs are efficacious in a range of indications but use and dose is limited by adverse events, even at low receptor occupancy
  - Sedation, somnolence, cognitive impairment, falls, overuse, misuse and addiction
- In general, BZDs are used acutely in epilepsy but not indicated for chronic use due to tolerance or loss of efficacy
- Darigabat has the potential to be used chronically by minimizing adverse events, risk of tolerance and abuse

Clinical experience with BZDs



Dose range for oral classical BZDs limited by sedative AEs
(<20% receptor occupancy)

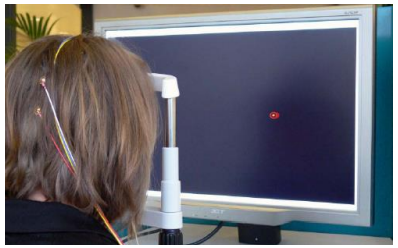
Darigabat: Favorable Pharmacology in NeuroCart, Differentiated From a BZD

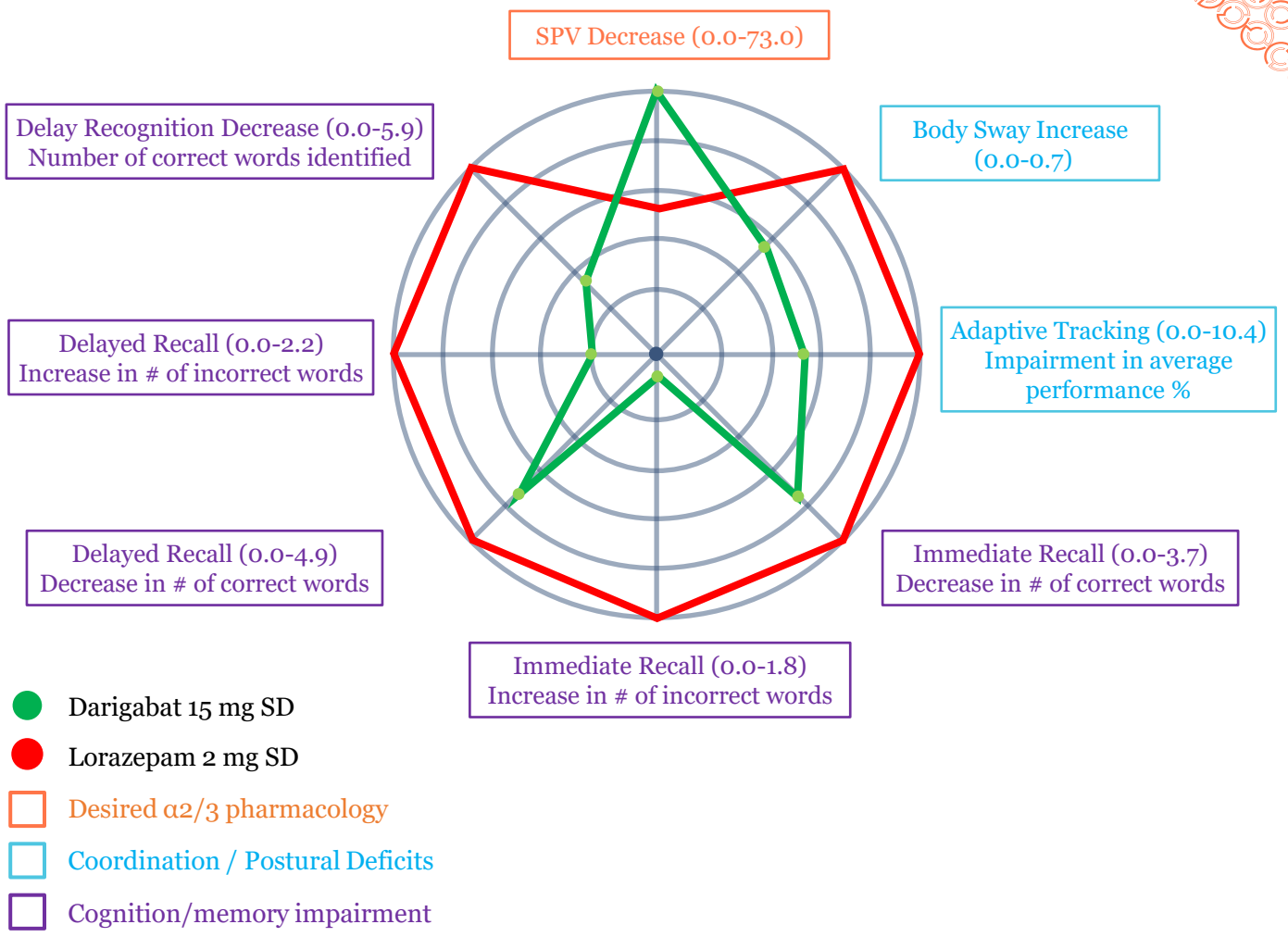
NeuroCart is a comprehensive battery of tests to evaluate CNS functional domains

Darigabat first-in-human study tested the following brain functions based on known $GABA_A$ receptor pharmacology:

- Saccadic peak velocity (SPV) - desired α2/3 pharmacology
- Body sway - undesired α1 pharmacology
- Adaptive tracking - undesired α1 pharmacology
- Visual-verbal learning test - undesired α1/5 pharmacology

- Relative to 2 mg lorazepam, darigabat demonstrated a larger decrease in SPV and smaller impairment on body sway, adaptive tracking and cognitive tests



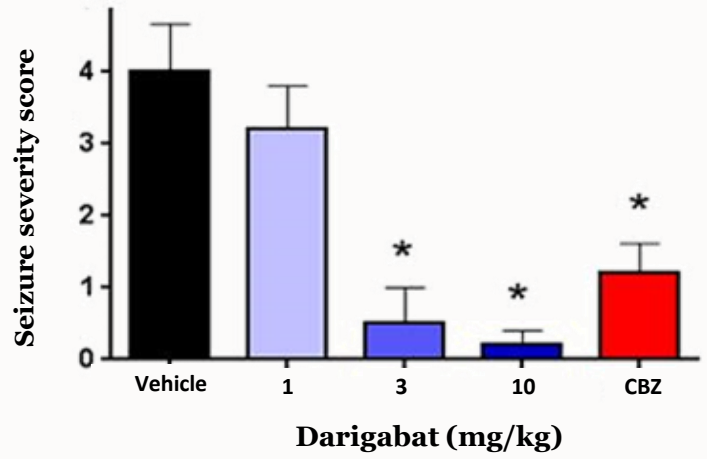


Source: IND B7431001 - Phase 1 double-blind, randomized, placebo controlled, cross-over single dose escalation study to investigate the safety, tolerability, pharmacokinetics and pharmacodynamics of PF-06372865 in healthy subjects

Darigabat is Anticonvulsant in a Range of Preclinical Models

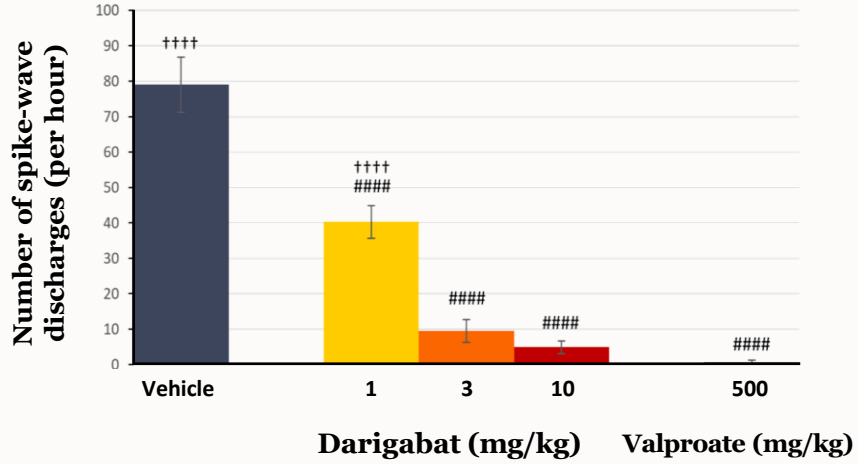
- Strong correlation of animal models of seizures translating to clinical activity across mechanism
- Darigabat demonstrated broad spectrum activity at ~>50% receptor occupancy
  - Darigabat is active in pentylenetetrazol-induced seizures
  - Amygdala kindling is a validated model for predicting activity in focal seizures
  - Genetic absence epilepsy rat model predictive of activity in absence (generalized) seizures

Amygdala Kindled Seizures in Rats: Focal Seizures



* p<0.05

Genetic Absence Epilepsy in Rats: Generalized Seizures



†††† p<0.0001 vs. valproate; #### p<0.0001 vs. vehicle

Darigabat demonstrated broad spectrum preclinical anticonvulsant activity, potentially through high receptor occupancy at α2 subunits

Duveau et al., CNS Neurosci Ther 2019; Owen et al., J Med Chem 2019

Potential Indications for Darigabat Beyond Epilepsy

Pipeline in a Pill

Potential for benzo-like activity with targeted GABA α 2/3/5 receptor selectivity

Benzos (Non-selective GABA Modulators) Widely Prescribed for Seizures, Anxiety, and Other Indications

Potential Large Indications Worldwide

Epilepsy	~65M Patients
Anxiety Disorders	~13M Patients (G7)
Agitation	15-20M Patients
Bipolar Disorder	~46M Patients

Significant need for GABA modulators that are better tolerated, less sedating, less addictive and supportive of chronic use

Source: DRG Market Research reports, World Health Organization

Darigabat TPP: Benzo-like Activity for *Chronic* Treatment

Darigabat Summary

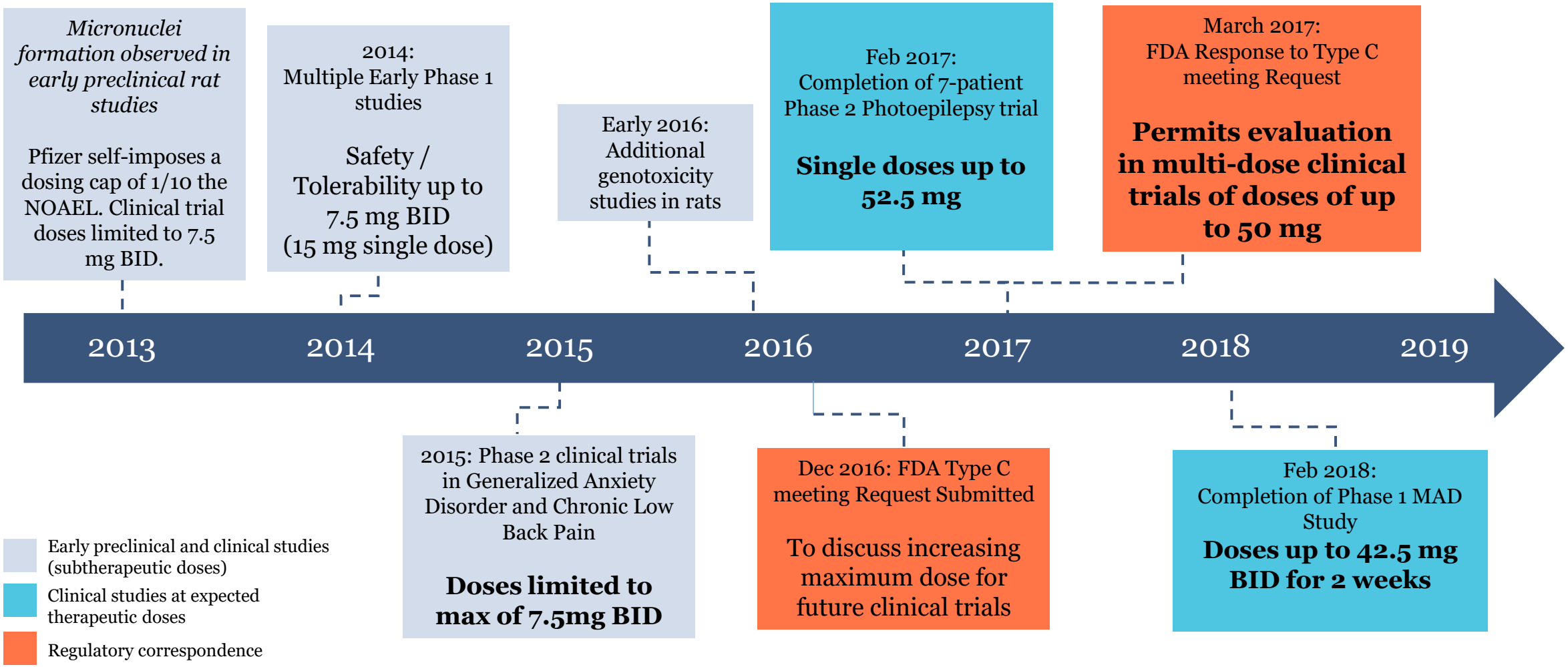
- Large markets (Focal & Generalized)
- Novel mechanism
- Potential for better activity than chronic treatment alternatives
- Potentially favorable side effect profile
- $ Attractive pricing analogs

Pricing & Launch

- High branded sales despite many generics
- Branded US price analogs >$10K/year
- Complex to change treatment in epilepsy
- 7-year+ average uptake in the category

History of Darigabat Development

- Results of early clinical trials were believed to be limited by Pfizer's self-imposed dosing cap



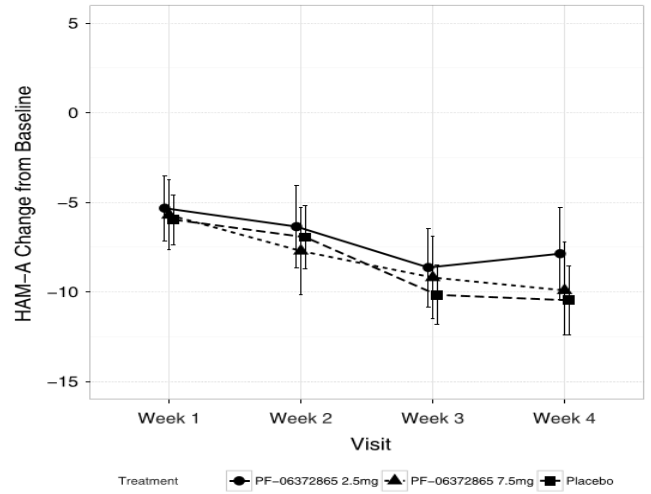
Prior Clinical Studies in Anxiety and Chronic Low Back Pain

Use of subtherapeutic doses and small sample size believed to account for lack of activity in prior trials

Phase 2: Generalized Anxiety Disorder

- Included drug-resistant patients on continued background treatment
- Sequential parallel comparison design
- Primary endpoint: HAM-A (minimum score at entry 20-22)
- 4 weeks on treatment: 2.5 mg BID darigabat, 7.5 mg BID darigabat, placebo
- Study stopped early for project prioritization - 90 enrolled of planned 384

Darigabat not differentiated from placebo on HAM-A

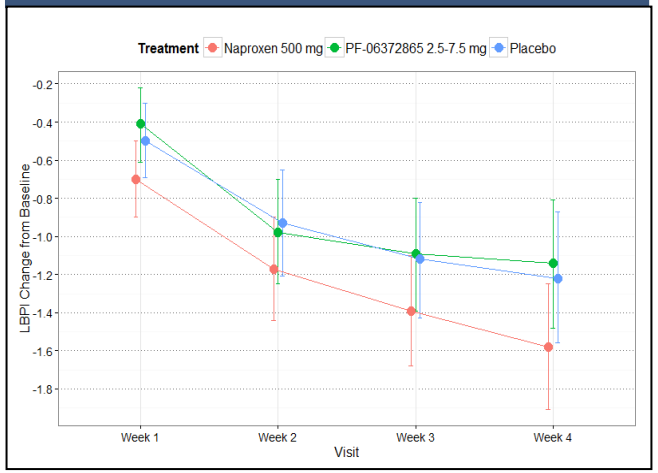


> 50% receptor occupancy remains unexplored in anxiety

Phase 2: Chronic Low Back Pain

- Included patients with > 6 months low back pain without radiculopathy
- Primary endpoint: change from baseline in low back pain score (measured on numerical rating scale)
- 4 weeks on parallel treatment: 7.5 mg BID darigabat, placebo, 500 mg naproxen BID (positive control)
- Study stopped early for futility following a planned interim analysis after 50% subjects had completed treatment

Darigabat not differentiated from placebo, naproxen



> 50% receptor occupancy remains unexplored in pain

Darigabat Favorable Side Effect & Tolerability Profile Across Trials

Darigabat has been tested in 289 subjects and was generally well-tolerated. There have been no clinically significant side effect observations from physical examination, vital sign measurements, laboratory safety assessments, or ECG parameters and no reports of sedation across single and multiple dose trials

I. Across Phase 1 trials:

- 81 healthy subjects received single doses of darigabat (0.04 to 100 mg); 55 healthy subjects received multiple doses of darigabat (2.5 to 42.5 mg BID)
- Most common AEs: dizziness, somnolence, and fatigue. All AEs across trials have been mild or moderate in severity
- No drug-related SAEs in Phase 1 trials
- Titration in multiple dose healthy volunteer studies appeared to reduce the incidence of somnolence and dizziness

II. Across Phase 2 trials:

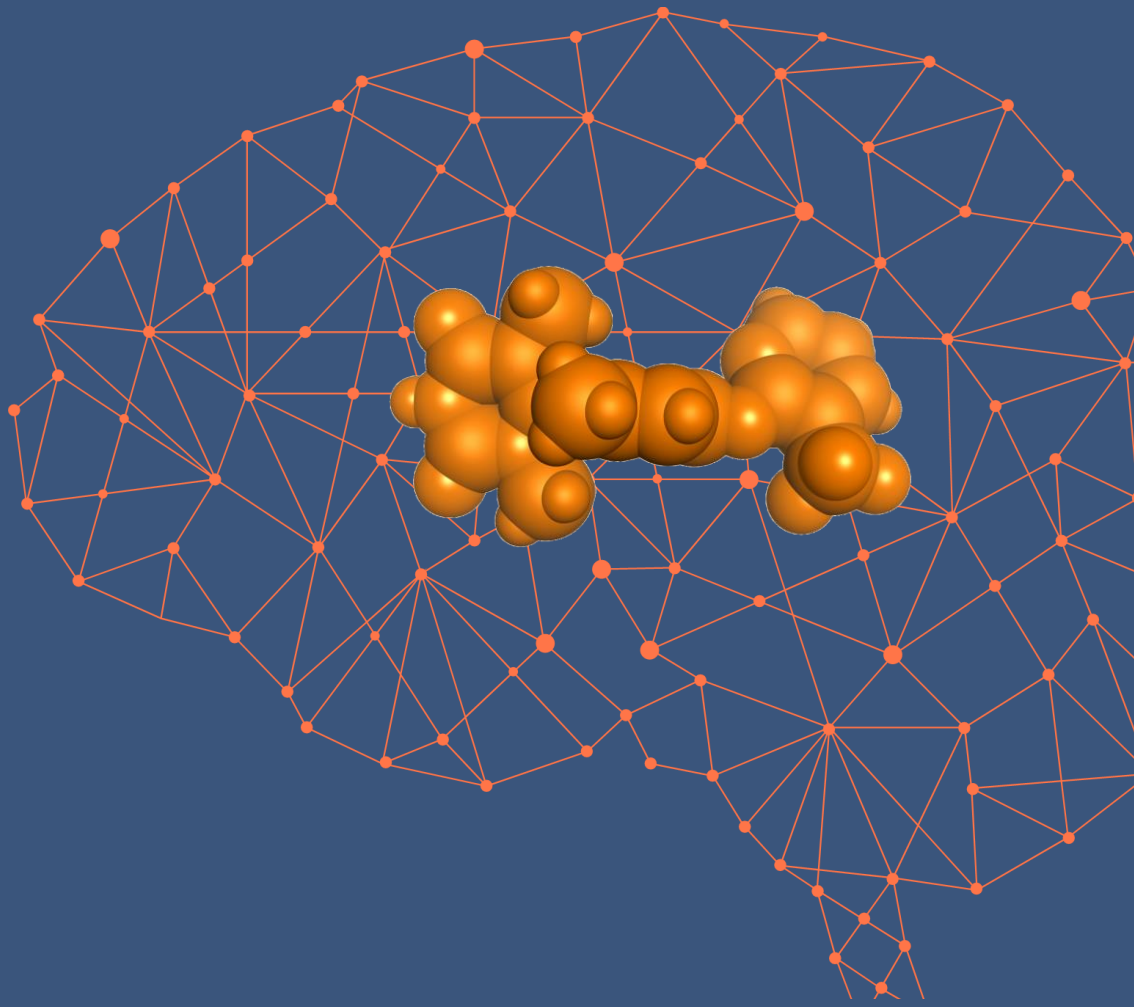
- 146 subjects received multiple doses of darigabat (2.5 to 7.5 mg BID); 7 subjects with documented photosensitive epilepsy received single doses of 17.5 mg and 52.5 mg in a crossover trial
- Most common AEs: dizziness and somnolence; the majority of AEs were mild or moderate
- In Study B7431007, there was limited increase in sleepiness as measured by the Epworth Sleepiness Score with either darigabat 7.5 mg, darigabat 2.5 mg or placebo at Week 2 and Week 4
- In Study B7431006, one patient experienced an SAE (transient ischemic attack) that was considered related to darigabat by the investigator. The patient had a history of high cholesterol levels and high blood pressure and was diagnosed with diabetes mellitus after the onset of TIA
- Use of titration in multi-dose Phase 2 trials appeared to mitigate CNS effects, including somnolence, over time

III. Other considerations:

- No evidence to date of withdrawal effects
- No evidence of the bone marrow effects seen in preclinical studies
- Reproductive effects are being addressed for all trials with requirements for contraception and standard warnings

Tavapadon in Parkinson's Disease

Additional Slides

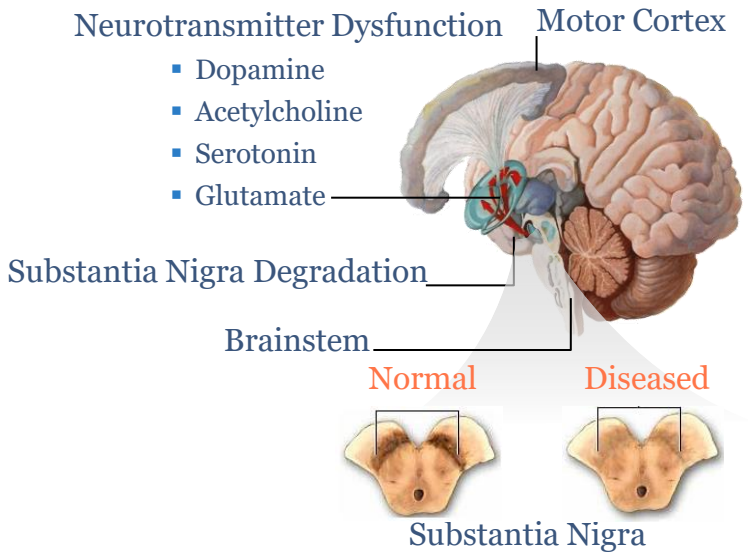


Parkinson's Disease Overview

Parkinson's disease is a progressive neurodegenerative disorder affecting regions of the brain that control balance and movement

Description

- Parkinson's disease is a degenerative neurological disorder characterized by progressive depletion of dopaminergic neurons in the substantia nigra region of the brain
- The lack of dopamine causes neurons to fire without normal control, leaving patients unable to control or direct their movement



Common Symptoms

- Symptoms of Parkinson's disease can be segmented into two categories – motor and non-motor:
  - Motor symptoms include tremor, decreased bodily movement (hypokinesia), slowness of movement (bradykinesia), stiffness and poor balance
  - Non-motor symptoms include cognitive dysfunction, psychosis, mood disorders, fatigue, *etc.*

Progression

- As symptom severity increases, patients often require increased doses of medication with decreasing efficiency, leading to "off" episodes
  - "Off" episodes are characterized by decreased motor function when patient's plasma drug levels fall below therapeutic levels
- Long-term levodopa use also leads to the development of dyskinesia or uncontrolled movement in PD patients

Genetic Indications

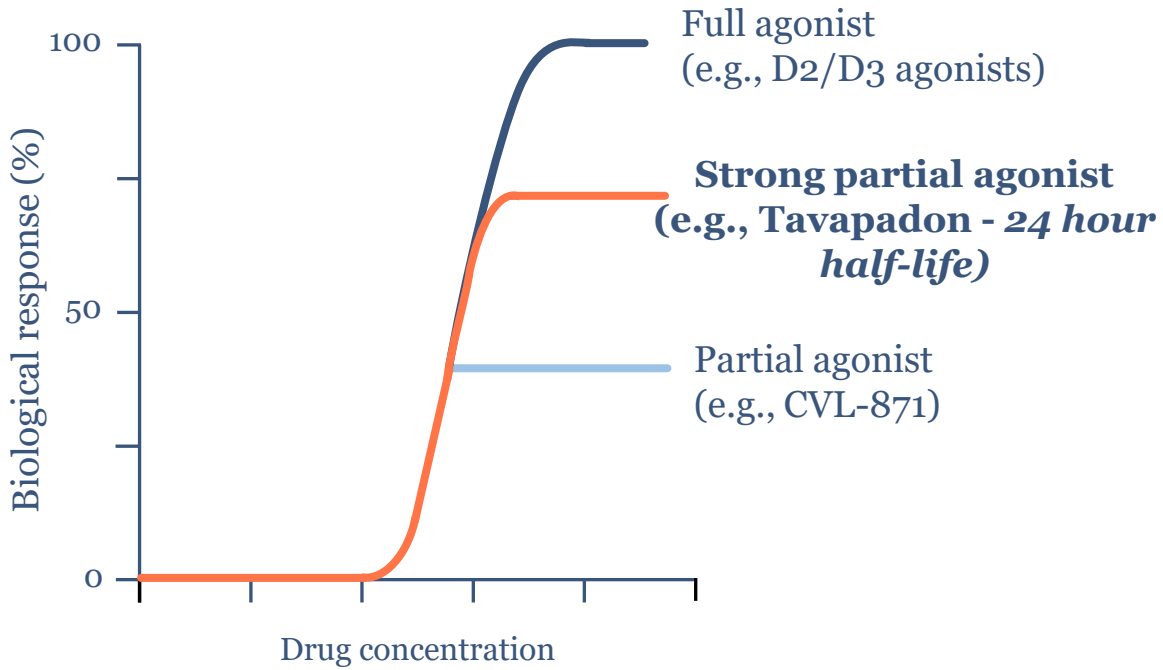
- Approximately 15% of Parkinson's patients have a family history of the disease. Such familial cases of the disease can be caused by mutations in the LRRK2, SNCA, PARK7, PINK1 or PRKN genes
- LRRK2 mutations attract greater attention from researchers since there are more known populations with this risk factor
  - G2019S is the most common LRRK2 mutation accounting for 3-6% of familial PD, and 1-2% of sporadic cases worldwide
  - This mutation is especially frequent in the Ashkenazi Jew and ArabBerber populations

Selectively Targeting Partial Agonism Designed to Improve Motor Control and Tolerability

D1/D5 Receptor Selectivity

D2/D3 Activation *(Indirect Pathway)*	Potential Effect	D1/D5 Activation *(Direct Pathway)*
+	Motor Control	++
	Cognition	++
	Motivation / Drive	++
-	Dose-Limiting Hypotension	
-	Impulse Control Disorders	
-	Sudden Daytime Sleepiness	

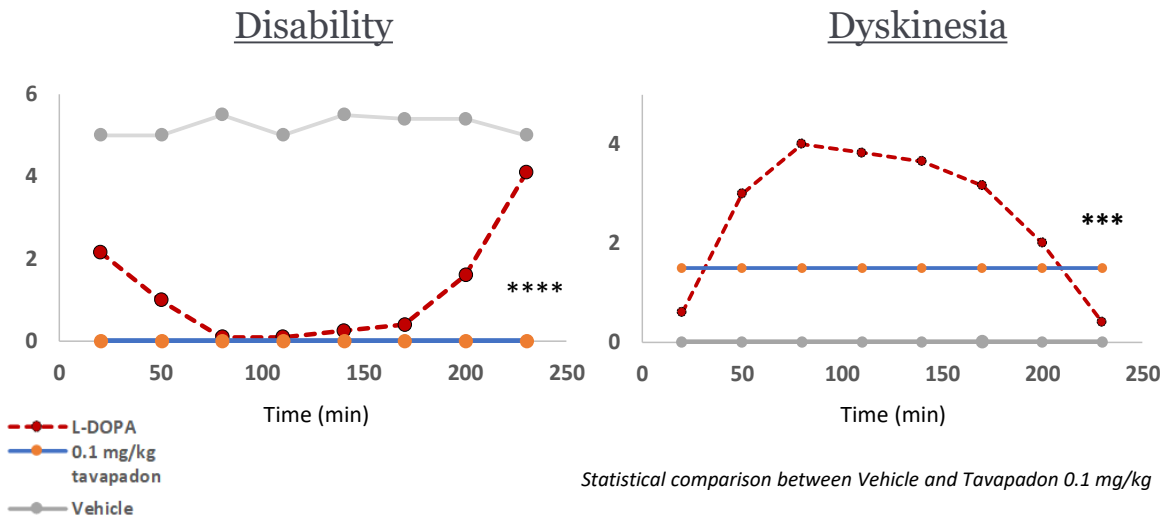
Degrees of Agonism (Illustrative)



First ***Partial Agonist*** for Parkinson's ➔ Avoids Dyskinesias

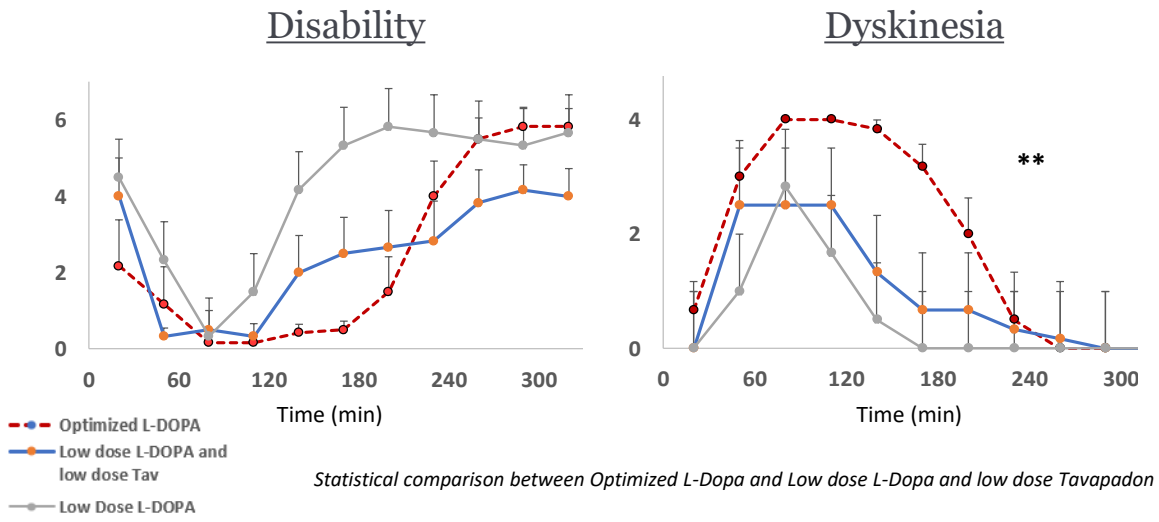
Unique and attractive combination of activity and low dyskinesias in highly translatable model of Parkinson's

Effect of Tavapadon vs. L-dopa on Disability and Dyskinesia



Statistical comparison between Vehicle and Tavapadon 0.1 mg/kg

Potential to Reduce L-dopa Dose to Achieve Motor Control with Reduced Dyskinesia



Statistical comparison between Optimized L-Dopa and Low dose L-Dopa and low dose Tavapadon

Compared to L-dopa, tavapadon robustly reduced parkinsonian symptoms in the MPTP-lesioned primate model with a more ***durable effect*** and ***lower dyskinesia*** levels

The combination of 33% L-dopa dose with 40% tavapadon dose showed ***similar activity to L-dopa alone with statistically significant reduction in dyskinesia***

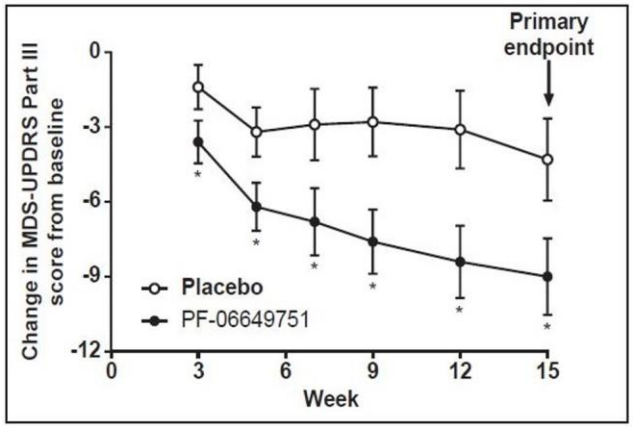
NHP = non-human primate. Mean Disability Score and Dyskinesia scores. Low-dose L-dopa at 33% of optimized L-dopa dose and low dose tavapadon at 0.04mg/kg.
Source: Young et al, ACS Chem. Neurosci., 2020.

Phase 3 Program Designed to Show Superior Treatment Profile

Phase 2 Results Inform Phase 3 Design

Phase 2 Results

MDS-UPDRS III



MDS-UPDRS II+III

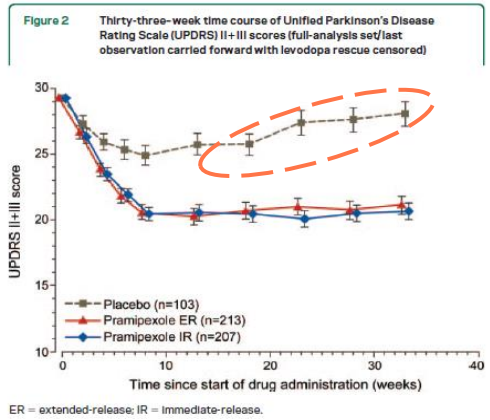
5.8 point improvement vs. placebo at week 15 on MDS-UPDRS II + III

Phase 3 Design

Baseline score of 2 or greater on MDS-UPDRS II

~2 point improvement vs. placebo on Part II, excluding participants with baseline score of 0 or 1 in Phase 2*

Placebo Attenuation at 6 Months



Further upside from forced titration to achieve maximum tolerated dose of tavapadon (less than 40% of patients were on the top dose in Phase 2)

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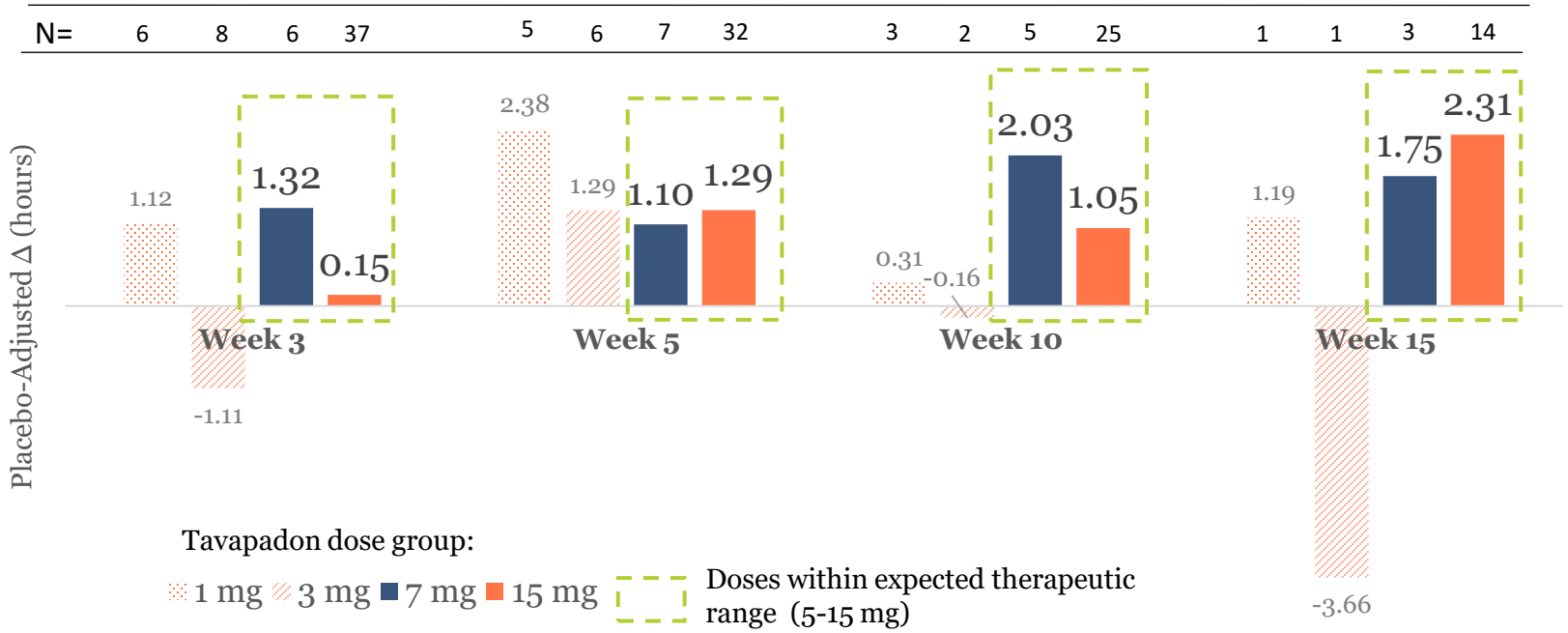
* Based on informal post hoc analysis

Tavapadon Phase 2 Results: Clinically Meaningful ON-Time without Troublesome Dyskinesias

Tavapadon generally demonstrated at least 1 hour of improvement in ON-time without troublesome dyskinesias in late-stage PD



Note: Tavapadon showed -0.6 hour reduction on the primary endpoint, change in OFF-time at week 10

Tavapadon showed clinically meaningful benefit within therapeutic dose range of 5 mg to 15 mg

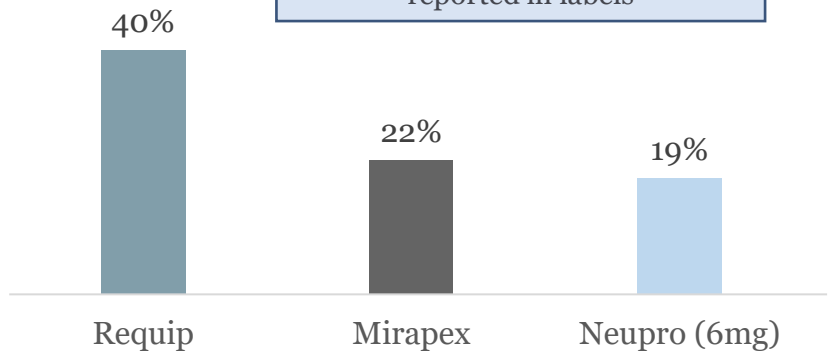
Trial was terminated early based on results of interim analysis for the primary endpoint of reduction in OFF-time compared to placebo at week 10.

Historical D2/D3 Labels Show Significant Side Effect Profile

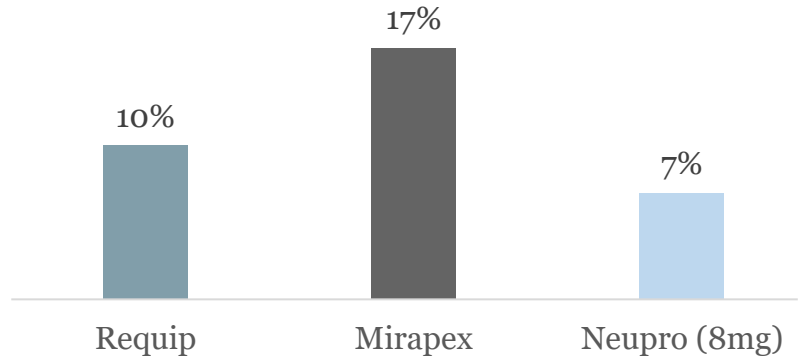
D2/D3 Side Effects: Sudden daytime somnolence, hallucinations, nausea and acute orthostasis

Somnolence
(Monotherapy)

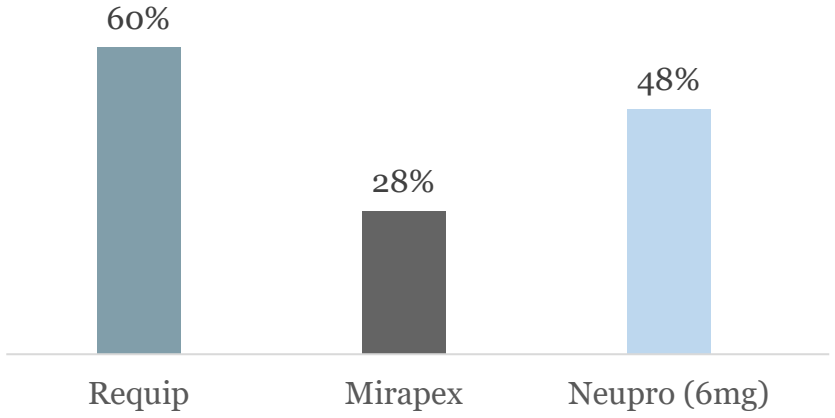
Sudden daytime somnolence is safety concern, but rates are not reported in labels



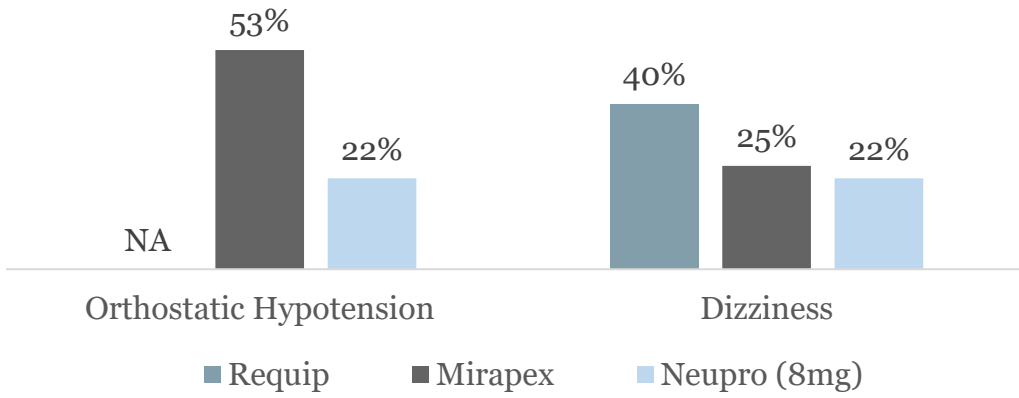
Hallucinations
(Adjunct to L-Dopa)



Nausea
(Monotherapy)



Hypotension-related Events
(Adjunct to L-Dopa)



Source: Requip, Mirapex, and Neupro labels (package inserts). Neupro 6mg dose indicated for early-stage PD; 8mg dose indicated for late-stage PD.
NA = Not reported in label (package insert).

Overview of Tavapadon Clinical Trials To Date

Protocol ID	Phase	Trial End Date	N= (active/total)	Design
B7601001	Phase 1	7 Feb 2014	18/18	Single ascending dose (0.25-2.5 mg) in healthy volunteers (HV)
B7601002	Phase 1	16 Apr 2015	61/77	Multiple ascending dose study in HV (0.5-5 mg QD)
B7601007	Phase 1	04 Dec 2014	9/9	Single ascending dose (0.25 and 0.75 mg) with an antiemetic
B7601006	Phase 1	14 Sept 2017	11/11	CYP3A Victim DDI
B7601005	Phase 1b	10 Mar 2016	45/50	Open label multiple ascending dose (5/15/25 mg) in PD patients Adjunct with lowering of levodopa dose
B7601009	Phase 1b	28 Feb 2016	18/18	Placebo controlled single ascending dose (0.75/1/3/6/9 mg) in PD patients Monotherapy
B7601003	Phase 2	10 Nov 2017	85/108	Adjunct with levodopa (1/3/7/15 mg) in advanced PD patients (w/ OFF-time ≥2.5h at baseline) Three week dose titration, 15 weeks total dosing
B7601011	Phase 2	29 Jan 2018	29/57	Monotherapy in newly diagnosed PD patients; flexible dosing Seven week dose titration, 15 weeks total dosing

Note: B7601017 OLE also enrolled 5 patients for 4 months with minimal data collected.

Thank you

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