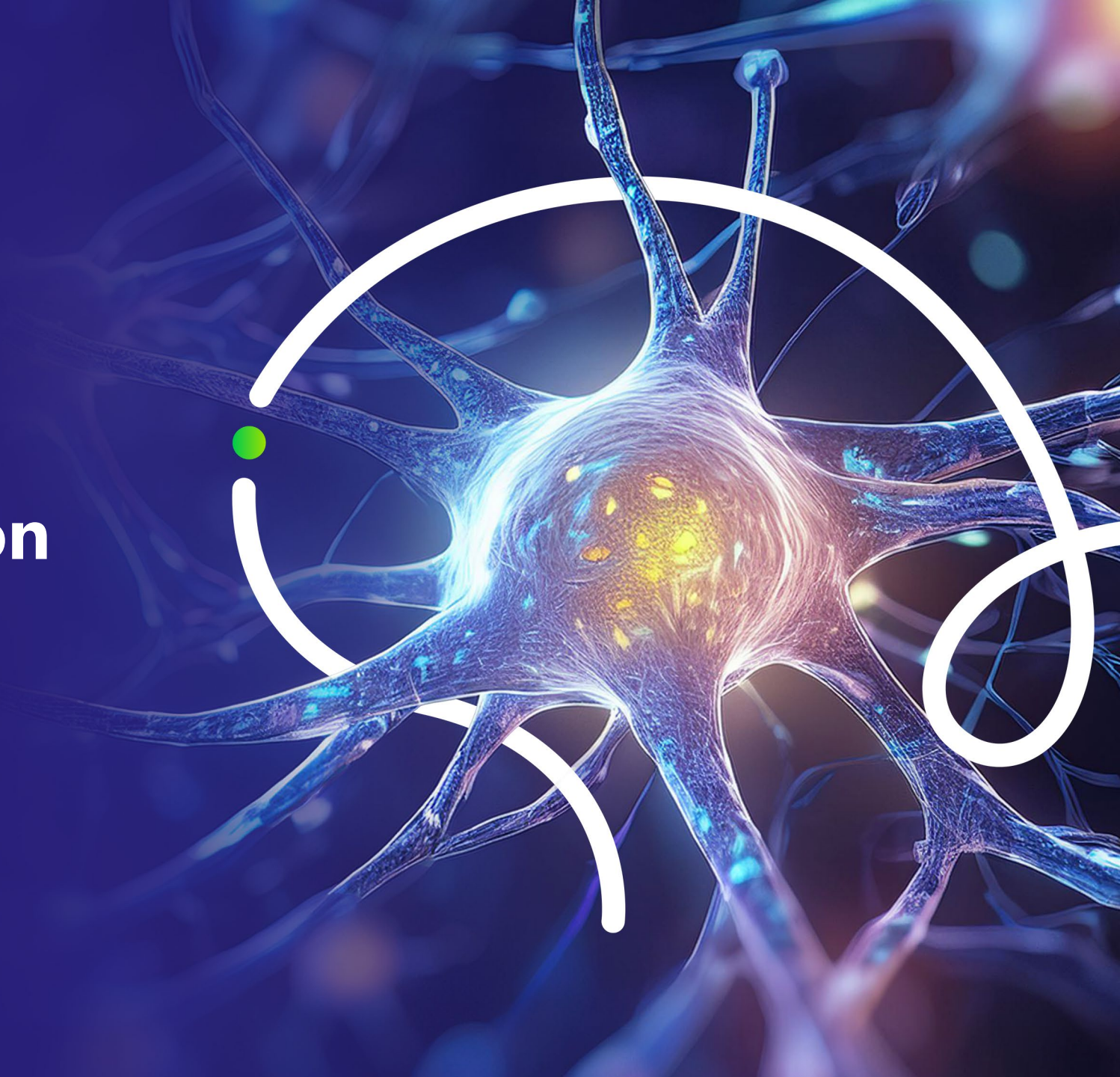
Ovid Therapeutics

Corporate Presentation

November 2025



Forward-looking statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 including, without limitation, statements regarding the potential therapeutic benefits of Ovid's current or future product candidates and pipeline programs; the reproducibility and durability of any favorable results initially seen to date in clinical trials; expectations regarding the duration of its cash runway and the expectation that it will support Ovid's operations and development programs; statements regarding the expected timing of the initiation, completion, and results and data of Ovid's clinical studies, including the proposed Phase 2a trial design for OV329; expected timing of IND submission for OV4041 oral; the potential use, development and therapeutic opportunity of OV329, OV350 and other compounds from Ovid's library of direct activators of KCC2; expectations regarding results and data, including safety and tolerability data, for OV329 support future development and therapeutic potential; the suitability of OV329 for a range of indication opportunities; the clinical and regulatory development of the KCC2 compounds in Ovid's library, including OV350 IV and OV4071 oral; the suitability of Ovid's library of novel, direct KCC2 transporter activators for a range of formulations and administrations; and expectations regarding the size of the market for Ovid's current or future product candidates and pipeline programs. You can identify forward-looking statements because they contain words such as "will," "may," "plan," "believes," "intends," "anticipates," "design," "advance," "target," "seek," "expects," "demonstrates," and "potential," and similar expressions (as well as other words or expressions referencing future events, conditions or circumstances).

Forward-looking statements are based on Ovid's current expectations and assumptions. Because forward-looking statements relate to the future, they are subject to inherent uncertainties, risks and changes in circumstances that may differ materially from those contemplated by the forward-looking statements, which are neither statements of historical factor guarantees or assurances of future performance. Important risks that could cause actual results to differ materially from those in the forward-looking statements include, without limitation: uncertainties inherent in the preclinical and clinical development and regulatory approval processes; whether interim or preliminary results from a clinical trial will be predictive of the final results of the trial or the results of future trials; the risk that Ovid may be unable to raise additional capital and could be forced to delay, further reduce or to explore other strategic options for certain of its development programs; the risk that Ovid may not be able to realize the intended benefits of its business strategy; Ovid's ability to identify business development targets or strategic partners, to enter into strategic transactions on favorable terms, or to consummate and realize the benefits of any business development transactions; Ovid's or any of its partners' abilities to meet anticipated deadlines and milestones; and/or unanticipated impacts or delays due to macroeconomic and geopolitical conditions. Additional risks that could cause actual results to differ materially from those in the forward-looking statements are set forth under the caption "Risk Factors" in Ovid's quarterly report on form 10-Q filed with the Securities and Exchange Commission (SEC) on August 13, 2025, and in future filings Ovid makes with the SEC. Any forward-looking statements contained in this presentation speak only as of the date hereof, and Ovid assumes no obligation to update any forward-looking statements contained herein, whether because of any new information, future events, changed circumstances or otherwise, except as otherwise required by law.

Medicines to quell conditions caused by neural hyperexcitability

Foundational biological targets

implicated in conditions driven by neural hyperexcitability and which have broad potential therapeutic utility

Pipeline of highly specific small molecules

intended to culminate in a fully integrated neurotherapeutics company with multiple clinical-stage programs and commercial medicines

Differentiated mechanisms of action

that stand out in a growing field of me-too medicines

Anticipated cash runway into 2H 2028[1]

expected to fund multiple value-driving catalysts

1. As of September 30, 2025, as adjusted for initial proceeds from private placement announced in October 2025

Focused high-value pipeline with differentiated mechanisms of action

Programs	Indication opportunities	Preclinical	Phase 1	Phase 2	Anticipated milestones
GABA-aminotransferase (GABA-AT) inhibitor					
OV329	Drug-resistant adult focal onset seizures (FOS)				Findings for 7 mg dose safety, tolerability, PK & exposure in healthy volunteers (Q1 2026) Phase 2a initiation (Q2 2026); Phase 2a topline results (mid-2027)
Potassium-chloride cotransporter 2 (KCC2) direct activator portfolio					
OV350 IV	First-in-human direct activation of KCC2				Phase 1 topline findings (Q4 2025) to include safety, tolerability and PK results
OV4071 oral	Psychosis assoc. with Parkinson's disease and Lewy body dementia and schizophrenia				Regulatory submission & clearance (Q1 2026) Phase 1 initiation (Q2 2026) Proof-of-mechanism ketamine study (mid-2026) Patient Phase 1b initiations (Q3 2026) Topline PoC results in PDP/LBD and SCZ (Q1 2027)
OV4041 oral	Generalized anxiety disorder and Rett syndrome				Initiating IND-enabling late 2025 IND submission late H2 2026

OV329: A next-generation, potential best-in-class GABA-aminotransferase (AT) inhibitor

Precision inhibition
of neuronal hyperexcitability, including drug-resistant epilepsies (DREs)

Validated mechanism
for seizures, with differentiated safety from 1st generation

Clinical data
Phase 1 showed safety, tolerability and pharmacodynamic activity
Phase 2 initiation Q2 2026

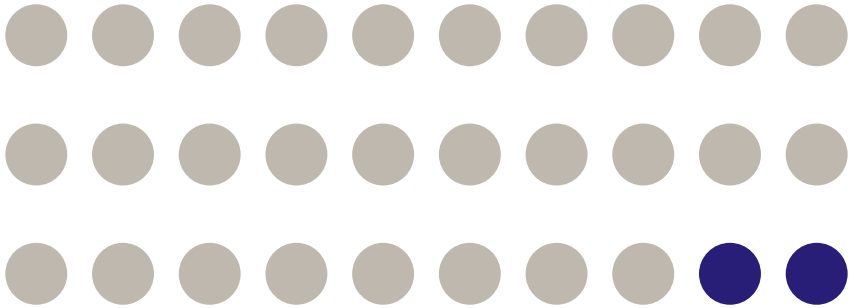
IP through 2041
(composition of matter; assumes five-year patent term extension)
10 patent families

A potential medicine for people living with treatment-resistant focal seizures

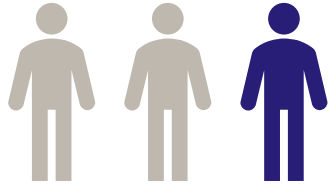
New mechanisms of action are needed in epilepsy

Drug resistant epilepsy (DREs)

30 anti-seizure medications (ASM) approved in 15 years, but only 2 with novel mechanisms[1]



A crowded treatment landscape and yet—
>1 in 3 patients live with uncontrolled seizures



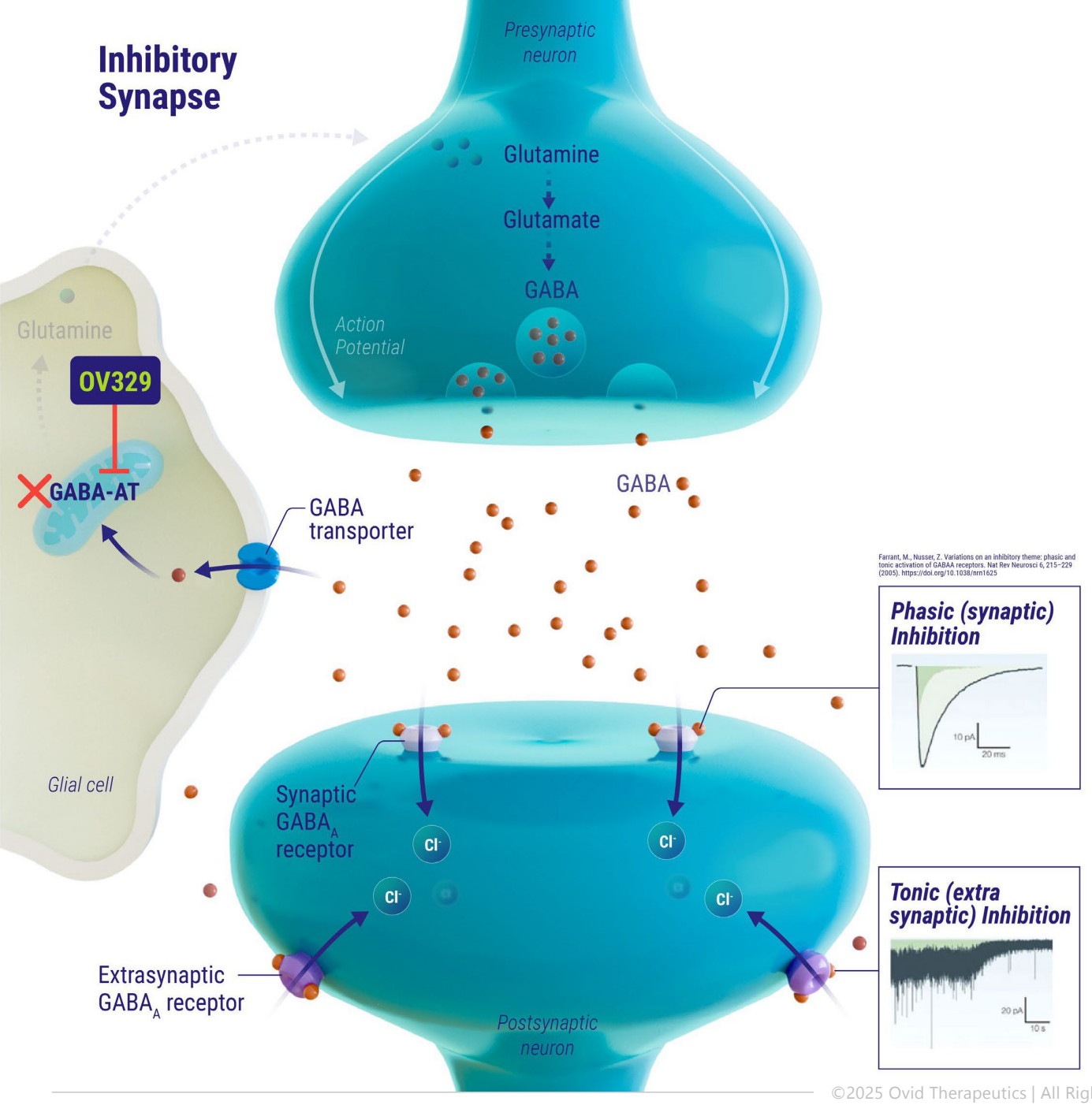
47% of epilepsy patients in the U.S. report poly-pharmacy use taking 5 medications on average[2]

1. Each dot = 1 FDA-approved ASM; Purple dots are Fycompa and Cenobamate
2. Terman SW, Aubert CE, Hill CE, et al. Polypharmacy in patients with epilepsy: a nationally representative cross-sectional study. Epilepsy Behav. 2020;111:10726

Challenges of current GABA-modulating medicines

Safety	**1st-generation GABA-AT inhibitor had a poor safety profile**	• Vigabatrin has no therapeutic index • Irreversible retinal changes & vision loss in some patients • Copious dosing (2 – 3 grams) to achieve anti-convulsant effects
Poor tolerability	**Surge in GABA in synapse creates Vigabatrin (VGB) tolerability challenges**	• Sedation • Dizziness
Durability of effect	**Short-acting**	• Some benzodiazepines have limited pharmacodynamic (PD) duration

Significant opportunity exists for an efficacious, well-tolerated ASM that elevates natural levels of GABA



OV329 optimally tunes GABA; potentially enhancing tolerability and efficacy

Inhibits GABA-AT, the enzyme that degrades the inhibitory neurotransmitter, GABA[1]

Increases GABA in the synapse & extra-synaptic regions delivering phasic & tonic inhibition[2]

Creates an inhibitory neural environment that averts the tolerability issues associated with surging GABA in the synapse

1. Silverman RB. Chem Rev. 2018 Apr 11;118(7):4037-4070. doi: 10.1021/acs.chemrev.8b00009. Epub 2018 Mar 23. PMID: 29569907; PMCID: PMC8459698
2. Colmers P et al. eNeuro 27 June 2024, 11 (7) ENEURO.0137-24.2024; https://doi.org/10.1523/ENEURO.0137-24.2024

OV329 offers a differentiated target product profile

- Quell hyper-excited neurons by optimally tuning synaptic and extra-synaptic GABAergic inhibition
- Potent, lower dosing compared to VGB
  (est. OV329 human doses: 5-7 mg vs 2-3 g for VGB)
- Oral, once daily dosing
- Sustained reduction of focal seizures
- Competitive seizure reduction efficacy and potential best-in-category safety profile
- No titration anticipated

A potential profile to treat a broad population of epilepsies that experience focal onset seizures and disorders driven by excess neural excitation

Phase 1 results support rapidly advancing OV329 into a Phase 2a trial

- Strong safety profile with few treatment-related adverse events; no ophthalmic safety findings
- Multiple positive biomarker results support inhibitory activity and therapeutic potential
- Inhibitory effects on par or exceed therapeutic doses of vigabatrin (which has historically shown 50% or greater reduction in FOS frequency and potential for seizure freedom)[1]
- Engages and inhibits the target and elevates levels of GABA in the brain
- Demonstrates a clear responder rate relative to drug exposure
- Potential for a blockbuster drug with patent protection through 2041 (with PTE)

1. FDA Medical Review(s) for Sabril (vigabatrin). (2009). U.S. Food and Drug Administration. [accessdata.fda.gov]

Multiple biomarkers confirm OV329's inhibitory activity

Measure of inhibition

- Well accepted biomarkers to study GABAergic drugs
- Quantitative clinical measure of cortical inhibition
- Most optimal way of measuring inhibitory activities short of seizure trial

Comparability relative to established ASMs

- Measured against historical performance of Tx doses of VGB in healthy humans
- Incorporated confirmatory and negative control biomarkers based upon historic performance of VGB

Methods

- All biomarkers assessed on two muscle groups to ensure consistency of signal
  - First dorsal interosseous
  - Abductor pollicis brevis
- Requires a large N size to show dose responsiveness

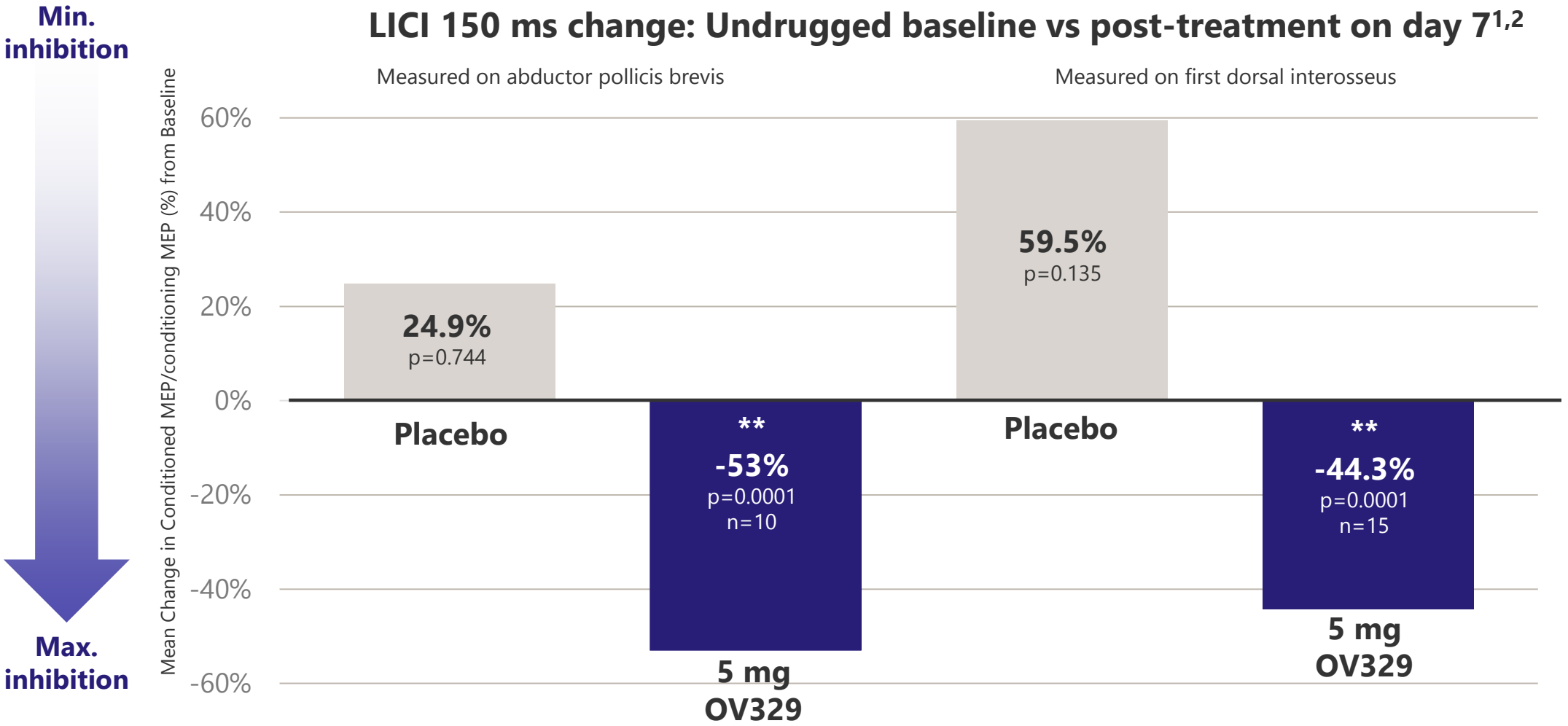
OV329 showed similar or better pharmacodynamic effects as vigabatrin in confirmatory biomarker measures

TMS metric	Vigabatrin therapeutic dose (50 mg/kg) Measured in healthy volunteers[1,2]	OV329 3 mg and 5 mg doses Measured in healthy volunteers
Paired-pulse long-interval intracortical inhibition (LICI) 150 ms	↑ Improved ~35%	↑ **Improved inhibition by 44-53%[3]**
Paired-pulse long-interval intracortical inhibition (LICI) 200 ms	✕ No change as expected for GABAergic drugs	✕ **No change as expected for GABAergic drugs[3]**
Cortical silent period (CSP) as measured in two separate studies	↑ Increased 19% in one study[1] ✕ No change in another study[2]	↑ **Increased 6-19%[4]**

Note: Information compares similar studies and methodologies; a head-to-head trial was not conducted

1. Pierantozzi M et al. Effect of Vigabatrin on motor responses to transcranial magnetic stimulation: an effective tool to investigate in vivo GABAergic cortical inhibition in humans. Brain Research, Nov. 26, 2004. In this study, vigabatrin was dosed at 50 mg/kg. in keeping with the recommended therapeutic dose of 2,500 – 3,000 mg in patients daily
2. Ziemann U et al. Effects of antiepileptic drugs on motor cortex excitability in humans: A transcranial magnetic stimulation study. Annals of Neurology, 1996. In this study, vigabatrin was given in a single dose of 2,000 mg.
3. Note: 6 subjects were unavailable for APB findings because of inconsistent capture, of which 5 were active, one was placebo
4. When participants who were "overstimulated" by motor evoked potentials (MEPs) are removed; results are stronger – in 3 mg cohort the mean prolongation of the CSP was 19%

OV329 demonstrated statistically significant increases in cortical inhibition exceeding vigabatrin's inhibition



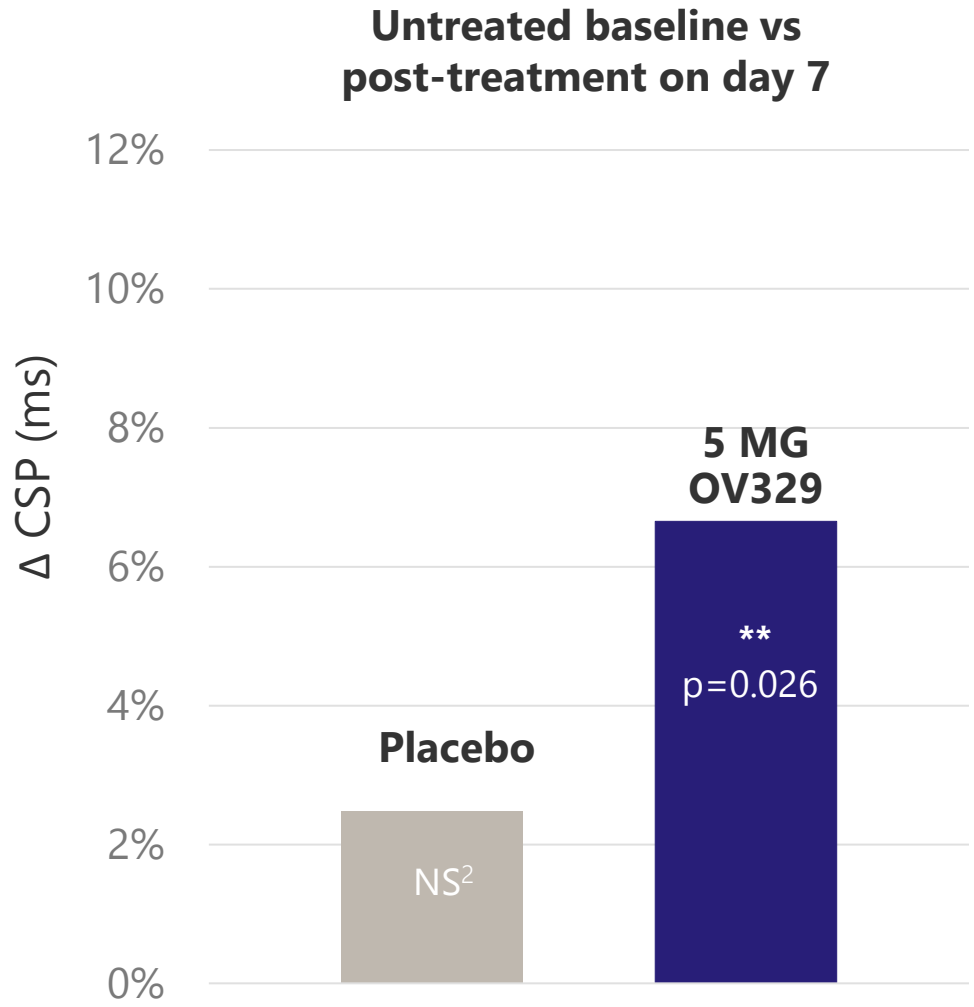
1. Baseline is at day 1 prior to treatment
2. Pierantozzi et al. Brain Res. 2004 Nov 26;1028(1):1-8

Note: Information compares similar studies and methodologies; a head-to-head trial was not conducted

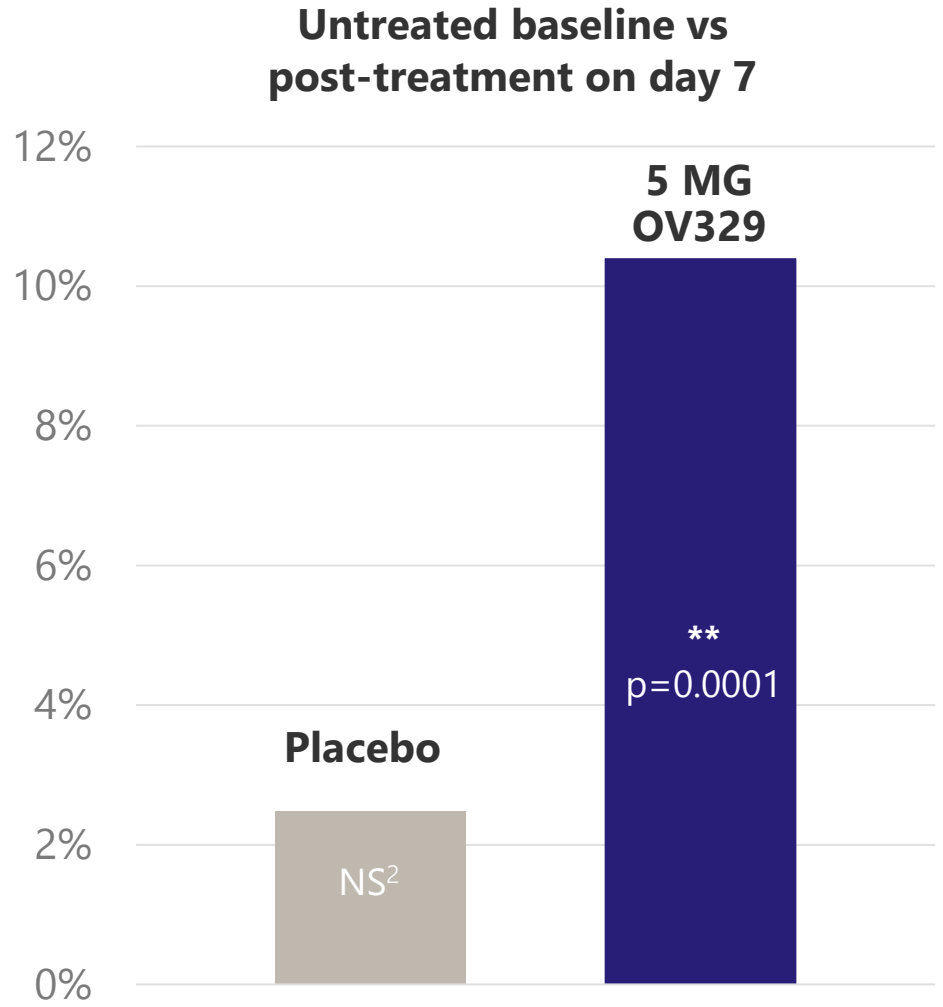
OV329 increases inhibition as measured by the CSP

Max. inhibition

Min. inhibition



Measured on abductor pollicis brevis



Measured on first dorsal interosseous

1. Baseline is at Day 1 prior to any treatment
2. Not statistically significant

OV329 was well-tolerated with no treatment-related SAEs[1]

Phase 1 summary of potentially treatment-related and treatment-emergent AEs[1]

Dosage	Adverse Event	OV329 (n=51)	PBO	Severity	Drug Related	Outcome
2 mg MAD	Headache	n=1	n=0	Mild (grade 1)	Possibly related (x2)	Recovered or resolved (x2)
	Drowsiness	n=1	n=0	Mild (grade 1)	Possibly related	Recovered or resolved
5 mg MAD	Metallic taste	n=1	n=0	Mild (grade 1)	Related	Recovered or resolved

- The most common potentially treatment-related AEs across treated and placebo groups were mild (grade 1) and transient.
- The most frequent AE seen was cannula site reactions at the site of blood draws (n=13), all of which were mild to moderate and were ruled not related to treatment.

1. Serious Adverse Events (SAEs); Adverse Events (AEs)

No ophthalmic safety findings or retinal changes associated with OV329*

- Extensive ophthalmic testing closely monitored for clinical and structural (sub-clinical) changes
- No treatment related effects were noted in any of the metrics tested

Ophthalmic metric*	Result
Best corrected visual acuity	No treatment related effects
Fundus photography	No treatment related effects
Indirect dilated ophthalmoscopy	No treatment related effects
Automated threshold visual field perimetry	No treatment related effects
Optical coherence tomography	No treatment related effects

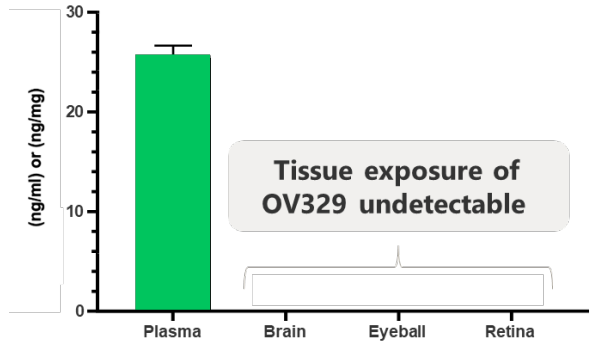
1. *Assessments from baseline to Day 30

Extensive preclinical characterization of OV329 supports anti-convulsant activity & ophthalmic safety

No accumulation detected in the eye or retina compared to vigabatrin[1,2]

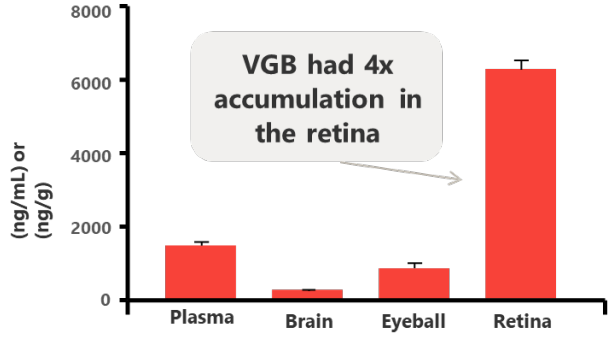
OV329

Mean Plasma and Tissue Concentration 5.0 mg/kg/day



VIGABATRIN

Mean Plasma and Tissue Concentration at 80.0 mg/kg/day



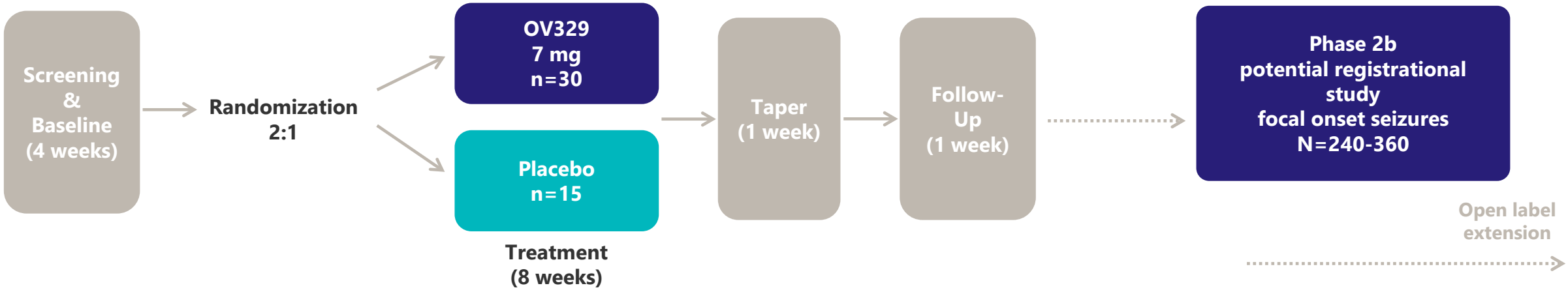
Anticonvulsant activity demonstrated in 9 chronic & acute seizure models[2]

- i.v. (ivPTZ)
- NMDA-induced infantile spasm
- Audiogenic seizure
- Amygdala kindled
- Corneal kindled
- Intrahippocampal kainate model of mesial temporal lobe epilepsy (MTLE)
- Intra-amygdala kainate model of MTLE
- Lithium pilocarpine
- Dravet *Scn1a*$^{A1783V/WT}$

1. Zhong et al. OV329 does not accumulate in mouse retina: A pharmacokinetic comparison with vigabatrin . Poster presented at the 2024 American Epilepsy Society.
2. Walters et al. Pharmacol Res Perspect. 2019 Jan 7:7(1)
3. Tsai J., et al. (2023). Preclinical Data Supporting the Efficacy of OV329, A Next-Generation GABA Aminotransferase Inhibitor, Against Seizures. Poster presented at the 2023 Epilepsy Pipeline Conference.

Proposed Phase 2a trial design in patients with focal epilepsy

Evaluating the safety, tolerability, and anti-seizure effect of OV329



Trial design

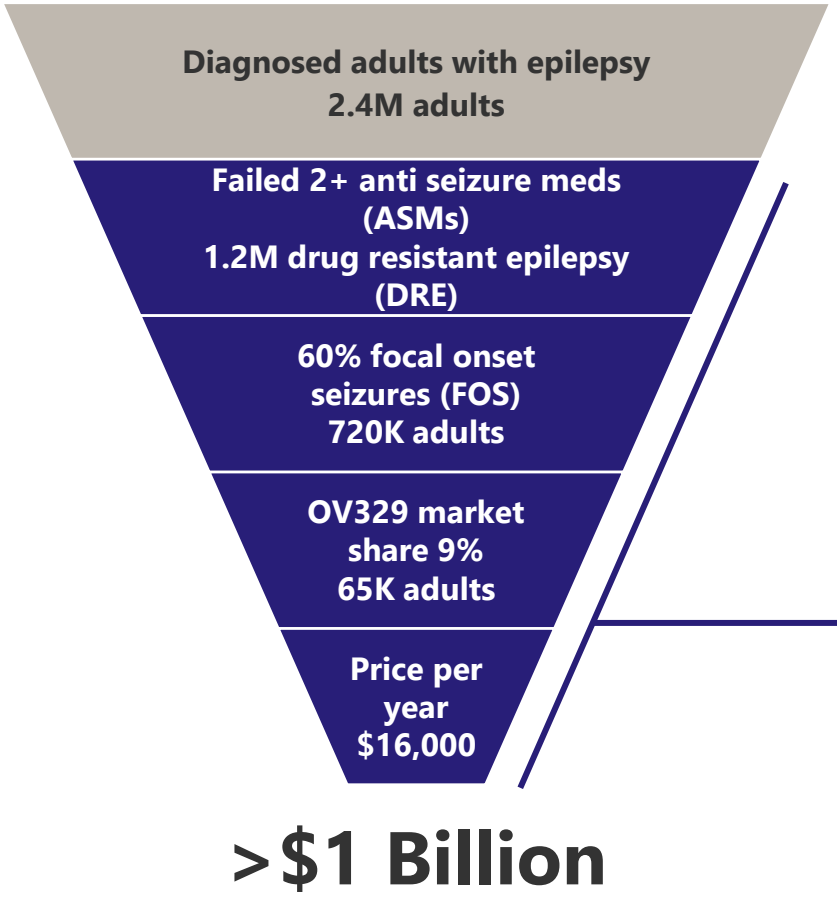
- Multi-site study
- Patients 18-55 years of age
- Diagnosed treatment refractory focal aware or awareness impaired motor onset seizures
- Concomitant 0-3 ASMs that have been stable for ≥1 mo

Outcome measures

- Safety (including ophthalmic, neurological and neurophysiology exams)
- Pharmaco-kinetics
- Efficacy:
  - % change from baseline in monthly seizure frequency
  - Responder rate
  - Time to pre-randomized monthly baseline seizure frequency
  - Seizure-free days
  - Clinical Global Impression of Improvement (CGI-I)

Unique, well-tolerated MoAs are rewarded in epilepsy, even in a competitive DRE market

Substantial opportunity within the DRE segment



>$1 Billion

Key: Voltage-gated Ion Channel Target | GABAergic | Synaptic Target | Other / Mixed / Unknown

Focal / Partial seizures

1L	Carbamazepine	Levetiracetam	Oxcarbazepine
	Phenytoin	Eslicarbazepine	Lacosamide
2L	Clobazam	Lamotrigine	Topiramate
	Rufinamide	Zonisamide	
Refractory + polypharmacy (~40%)	OV329 *In Phase 1*	XEN1101	XCOPRI
		Fycompa perampanel	BRIVIACT (brivaracetam)

Reimbursement & coverage dynamics remain favorable. Polypharmacy well covered

1. J Med Chem. 2012 Jan 26; 55(2): 567-575
2. J Am Chem Soc. 2018 Feb 14; 140(6): 2151-2164

Broader lifecycle opportunity for a safe GABA-AT inhibitor is significant

Initial intended indication

Potential follow-on indications

Focal seizures in developmental epileptic encephalopathies → Post-surgical & peri-operative pain → Substance abuse and alcohol withdrawal

Portfolio of direct activators of potassium chloride cotransporter 2 (KCC2)

Potentially transformational target that is a fulcrum rebalance inhibitory/excitatory signaling in many diseases of the brain

Potential first-in-class direct activators

OV350 (IV) Phase 1 topline readout estimated for Q4 2025

OV4071 (oral) Phase 1 initiation expected in Q2 2026

IP through 2046 (Composition of matter patent - assumes five-year patent term extension)

9 method-of-use patents filed

KCC2 normalizes inhibitory/excitatory balance in neurons and maintains GABA's inhibitory tone1

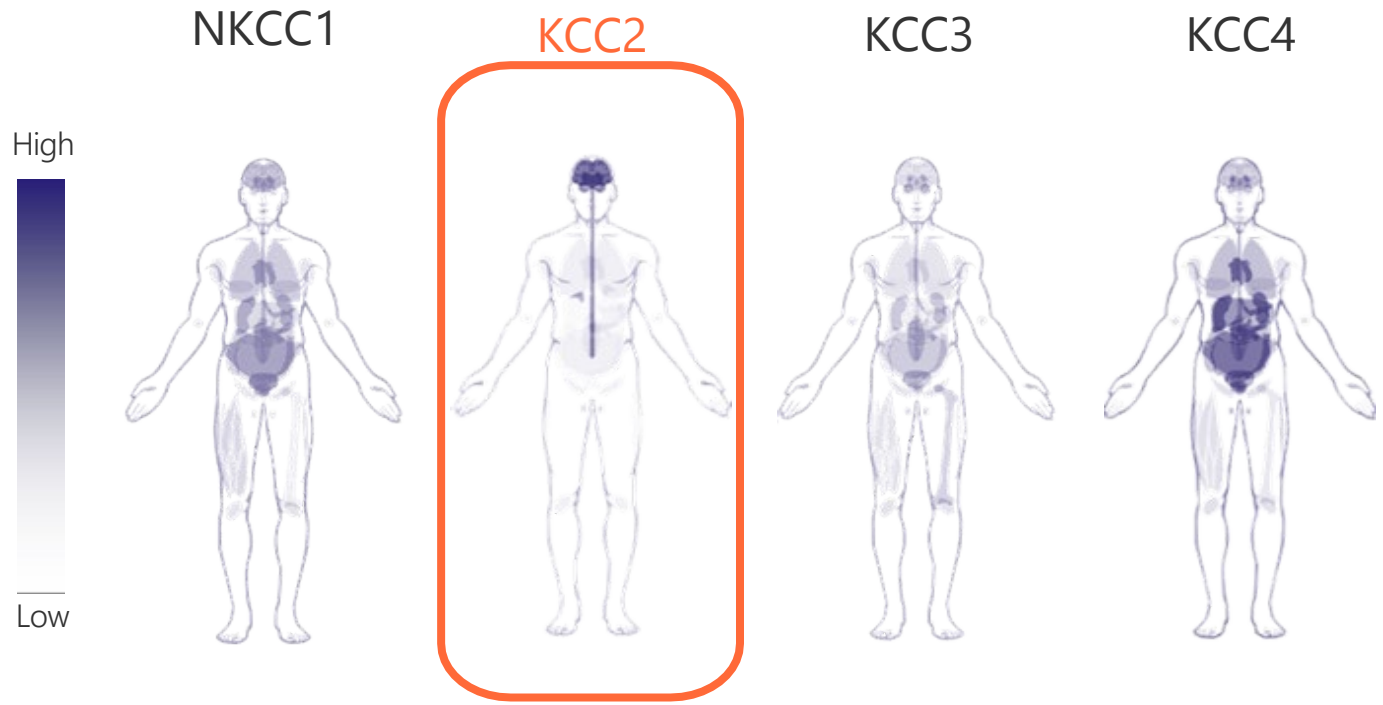
"Master switch" for neurological disorders

Where hyperexcitability is core to pathophysiology

Safety inherent in target

- Unlike enzymes, KCC2 cannot push beyond electrochemical equilibrium
- Driven by electrochemical gradients (K^+ and Cl^-)
- Self-limiting activation is intrinsic to KCC2 and supports safety in chronic use

KCC2 protein expression is confined to the CNS



KCC2 is an isoform of the potassium–chloride cotransporter family, specifically the neuron-specific member encoded by SLC12A5

1. Pressey et al. "Chloride transporters controlling neuronal excitability", https://doi.org/10.1152/physrev.00025.2021

KCC2 is the main regulator of GABA inhibition by maintaining neuronal chloride homeostasis

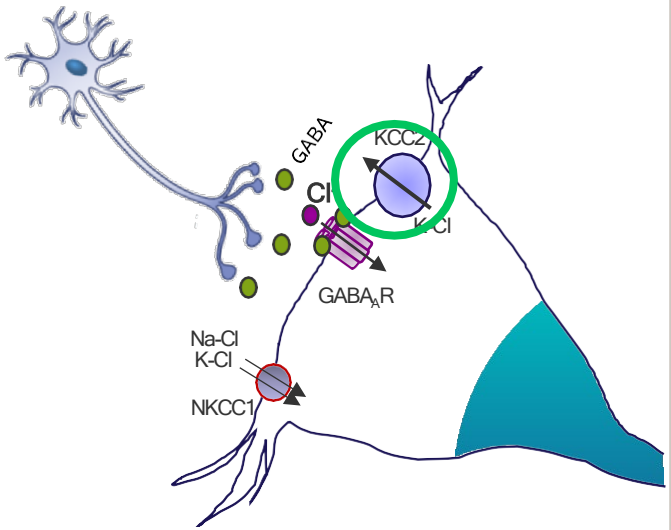
Regulates chloride gradient

Manages the passive inward entry of Cl^- through activated GABA channels

Maintaining low intracellular chloride is critical for hyperpolarization

KCC2 activation: Restores inhibition
'A battery to recharge interneurons'

Healthy Neuron



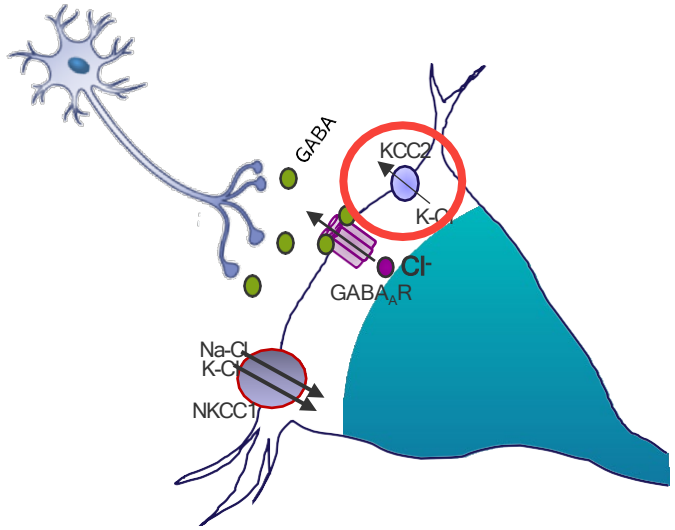
GABA → **inhibitory**

Intracellular chloride

Healthy ↑KCC2

'blanket of inhibition' in brain

Pathological Conditions



GABA → **excitatory**

Intracellular chloride

Pathology ↓KCC2

hyperexcitable circuitry

1. Pressey et al. "Chloride transporters controlling neuronal excitability", https://doi.org/10.1152/physrev.00025.2021
2. Illustrations adapted from publication by Phan Q. Duy, Miao He, Zhigang He & Kristopher T. Kahle (2020) Preclinical insights into therapeutic targeting of KCC2 for disorders of neuronal hyperexcitability, Expert Opinion on Therapeutic Targets, 24:7, 629-637, DOI:10.1080/14728222.2020.1762174

Activating KCC2: A lynchpin to restore GABA's inhibitory tone in a broad range of CNS conditions

Initial intended indications

Psychoses in Parkinson's disease & Lewy body dementia*

Schizophrenia*

Other indications

Neurodegenerative disorders

- Alzheimer's agitation
- Huntington's disease*

Neurodevelopmental disorders

- Rett syndrome*
- Autism
- SLC12A5-related early infantile epileptic encephalopathy (EIMFS)

Psychiatric & Other

- Schizophrenia*
- Bipolar disorder
- Depression
- Anxiety
- Obsessive compulsive disorder
- Neuropathic pain*
- Audiogenic trauma
- Epilepsies*
- Spinal cord injury–related spasticity
- Post-stroke spasticity

*Denotes indications in which Ovid has disease biology evidence supporting its KCC2 direct activators

Direct activation matters

Advantages

Bind directly to KCC2 transporter—modulating function independently of upstream pathways

Effective regardless of phosphorylation, brain-derived neurotrophic factor (BDNF) signaling, or disease-driven dysregulation

Enable dose-controlled, rapid, predictable pharmacodynamics

Profile optimization

Avoid tachyphylaxis and downstream adaptation seen in indirect pathways

Prevents unintended modulation of off-target signaling cascades

Maintain physiological limits on hyperpolarization

All Ovid molecules confirmed as direct activators of KCC2

Ovid data on file

Ovid development-stage portfolio

	OV350	OV4071	OV4041	OV5000 & OV6000 Series
Stage /Anticipated Milestones	Phase 1 results est. Q4 2025	Trial initiation projected Q2 2026	IND est. Q4 2026	Leads confirmed
Formulation	**Intravenous**	**Oral & injectable formulations**	**Oral & injectable formulations**	**Oral and injectable formulations (long lasting)**
Strategic Rationale	**Class safety & tolerability**	**PoC: Psychosis in Parkinson's disease & Lewy body disease**	**Generalized anxiety disorder Rett syndrome**	**Broad CNS indications chronic applications**
Characteristics	• Antipsychotic & anticonvulsant activity • No sedation • Rapid acting	• Antipsychotic • 20x potency over OV350 • 60x EC50 reached in repeat dosing • Excellent tolerability • Consistent plasma exposure • No sedation	• Anxiolytic • Antipsychotic • Anticonvulsant activity	• Low cLogP • High solubility • Good brain exposure • Consistent CNS biology as OV4000s • Additional formulation options

Ovid data on file

OV350 is a first-in-class KCC2 direct activator

Specificity

Directly binds to KCC2 (surface plasmon resonance and thermal shift assay)

No off-target effects on kinases regulating KCC2 activity (PKC, SPAK, WNK)

Penetrance

Small molecule (MW<500)

Excellent brain penetration

Potential for biomarker

OV350

Disease model activity

Prominent antipsychotic activity

Anticonvulsant activity

Neuroprotective & anti-inflammatory signals

Safety

No evidence of sedation in animals

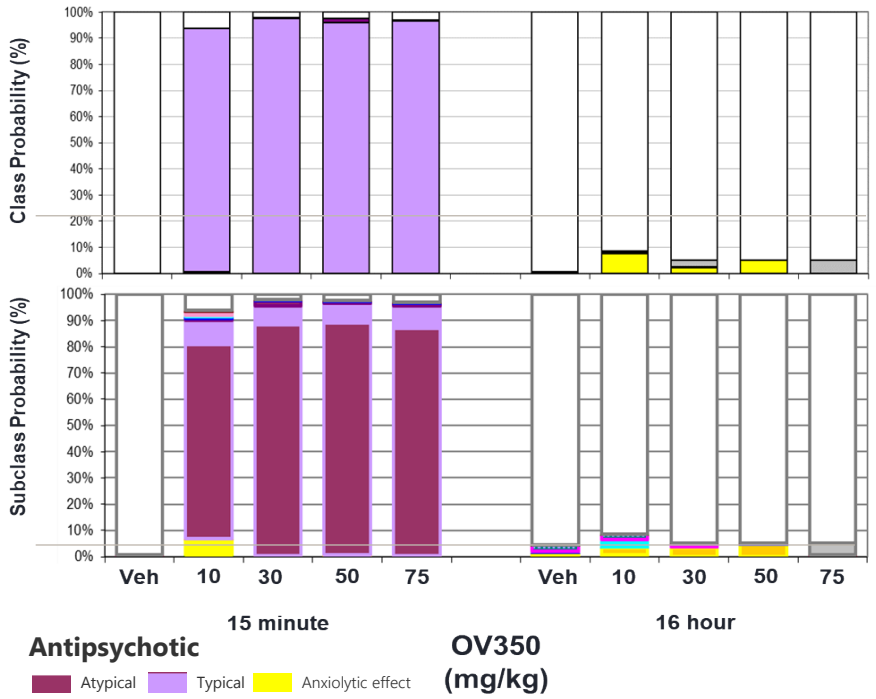
Supportive safety package

Ovid data on file

OV350 elicits potent, robust, rapid and reversible antipsychotic activities

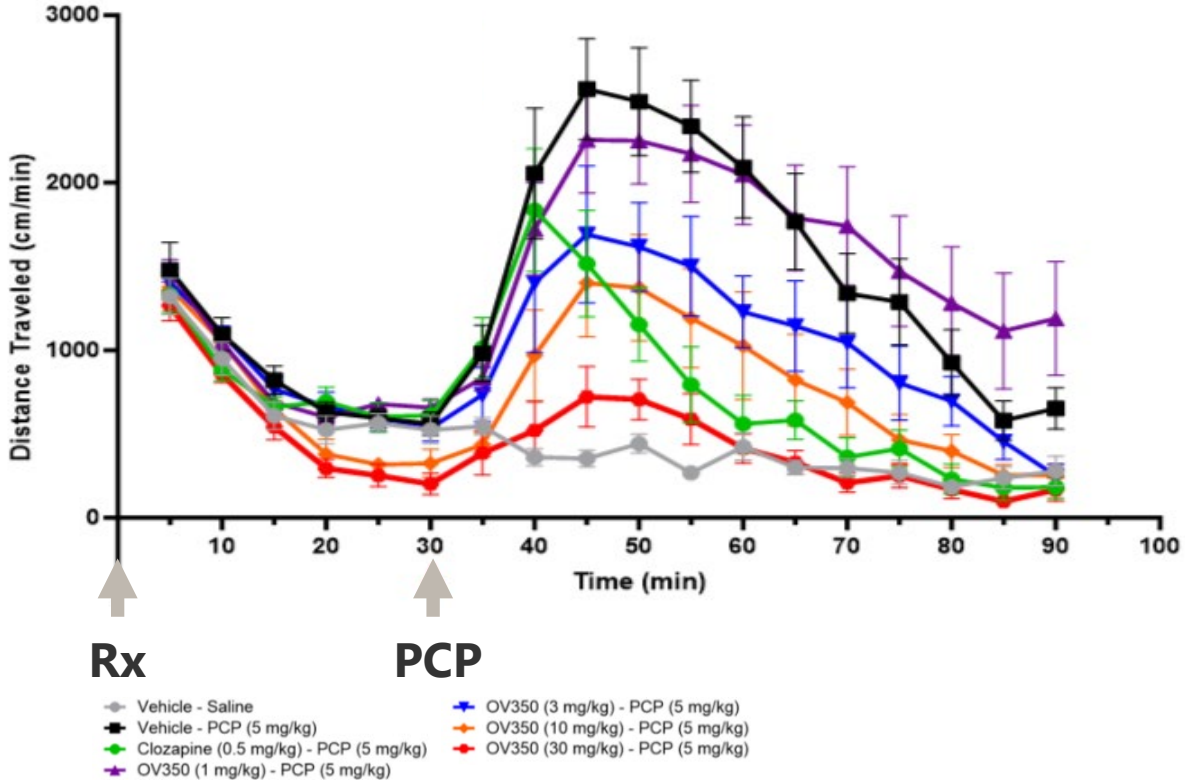
Profile similar to atypical antipsychotic in SmartCube® screen

- Onset of effect within 15 minutes
- Dose response starting at 10 mg/kg; disappears in 16 hours
- No sedation or other adverse behavior effects observed up to 75 mg/kg
- Minor anxiolytic effects observed (yellow)



Phencyclidine-induced psychosis (PCP) model

- OV350 dose-dependently inhibited PCP induced hyperlocomotion
- Model is characterized by: Confusion, excitation, aggression, paranoia, hallucinations, and can be experimentally measured by hyperlocomotion
- No evidence of sedation



Ovid data on file

Overview: OV4071, an oral KCC2 direct activator

Pharmacology

- Orally available
- Highly potent: 20x potency over OV350
- 60x EC50 reached in repeat dosing
- Consistent plasma exposure
- Highly efficient CNS penetration: brain:plasma > 1:1

Pharmacodynamics

- Strong profile comparable to atypical antipsychotic in phenotypic screens without sedation
- Strong effect in mouse model of hyperlocomotion induced by a psychotomimetic
- Improvement in Huntington's disease model

Completing IND-enabling studies

- Rat 14-day daily oral DRF cleared MTD with limited histopathology
- Rat 28-day oral GLP study – in life completed
- Dog DRF ongoing and GLP toxicology (already completed non GLP dog toxicology including histopathology)

Clinical Plan

- 1st indication: Psychosis in Parkinson's disease and Lewy Body dementia
- Regulatory filings expected 1Q 2026
- Begin P1 HV study expected 2Q 2026
- Topline patient data expected 1Q 2027

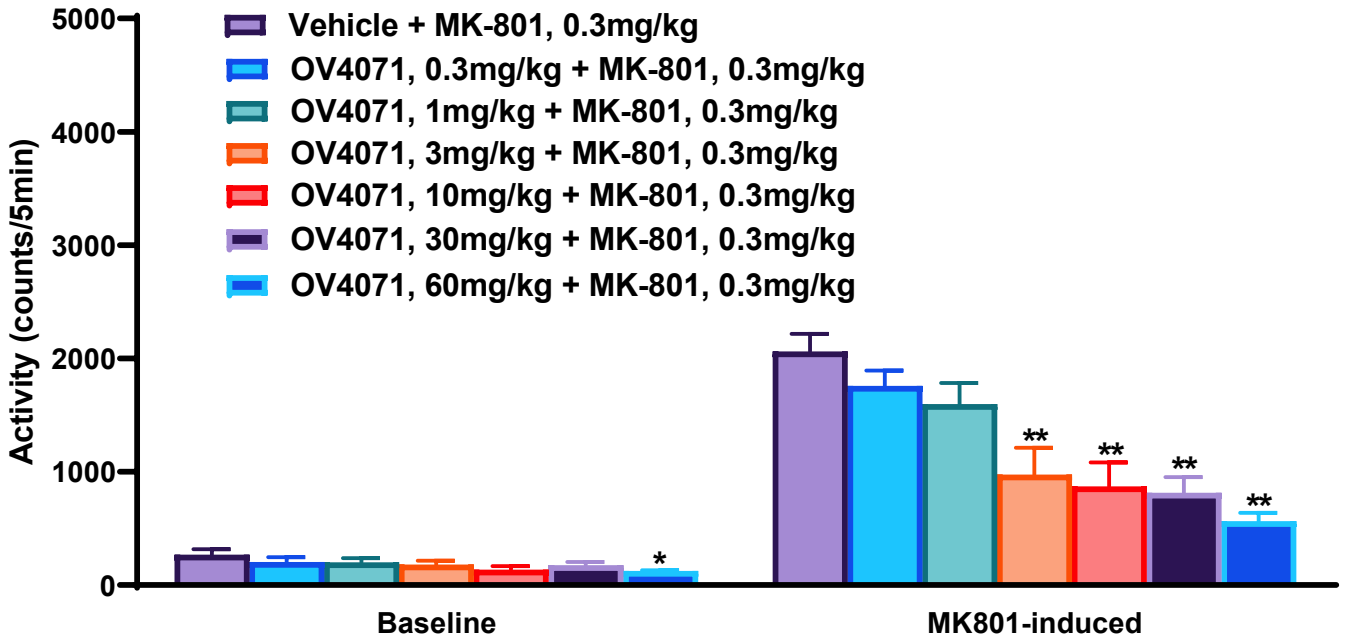
Ovid data on file

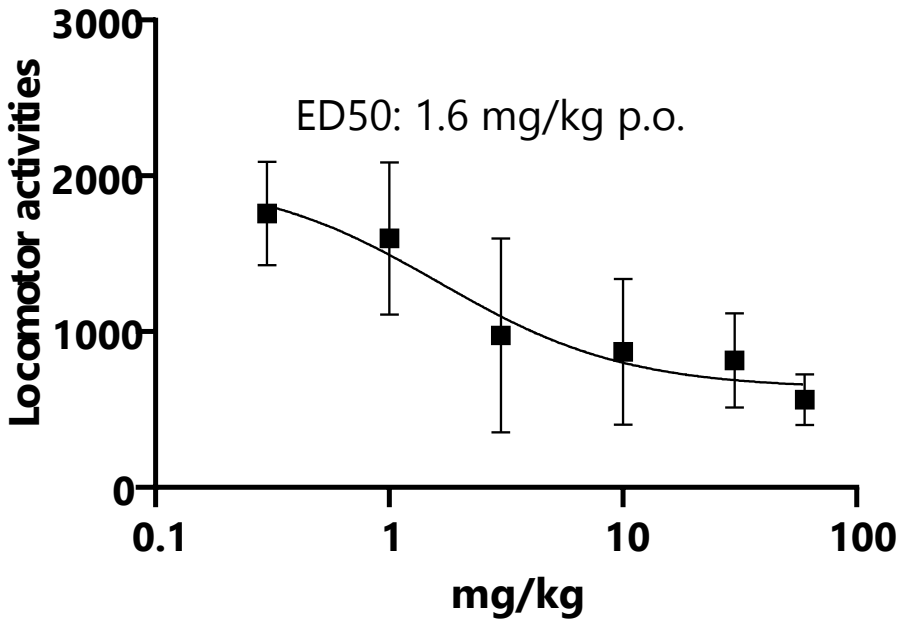
OV4071: IND-enabling progress

Selected Key Properties	Attribute	Status	Comments
Potency	60x EC50 reached in repeat dosing	✓	Highly potent, 20x potency over OV350
Direct activation of KCC2	0.64 µM	✓	Highly selective direct activator of KCC2, avoids unintended off-target downstream regulation
PK/PD	EC_{50}=44.2 ng/mL	✓	PK is linear and predictable with a dose-dependent PD effect
Brain penetration	Brain:plasma > 1:1	✓	Highly efficient CNS penetration
Efficacy	Dose-dependent reduction in multiple models of psychotic-like behavior	✓	Translatable models (PCP, MK-801) demonstrate potential clinical effect Comparable effect to pimavanserin and clozapine
DMPK	Oral, QD, 40 mg/day projected human dose	◔	Human PK prediction supports QD administration and 40 mg dose
Toxicology/ safety pharm	No biologically relevant off-target activity	✓	Rat and dog DRF complete; rat and dog GLP ongoing No sedation, no motor impairment, no genotox; no anticipated relevant DDI
Formulation	Oral	◑	Stability demonstrated Commercial-ready formulation in process
IP strategy	2046	✓	Composition of matter through 2046 (assumes PTE), 12 add'l method-of-use patents filed

Ovid data on file

MK801 schizophrenia psychosis model confirmed OV4071 robust, dose dependent signal





Dose-dependent pharmacodynamic effects of OV4071

Ovid data on file

Strategy for initial KCC2 indication – PD and LBD psychosis

Risks to mitigate

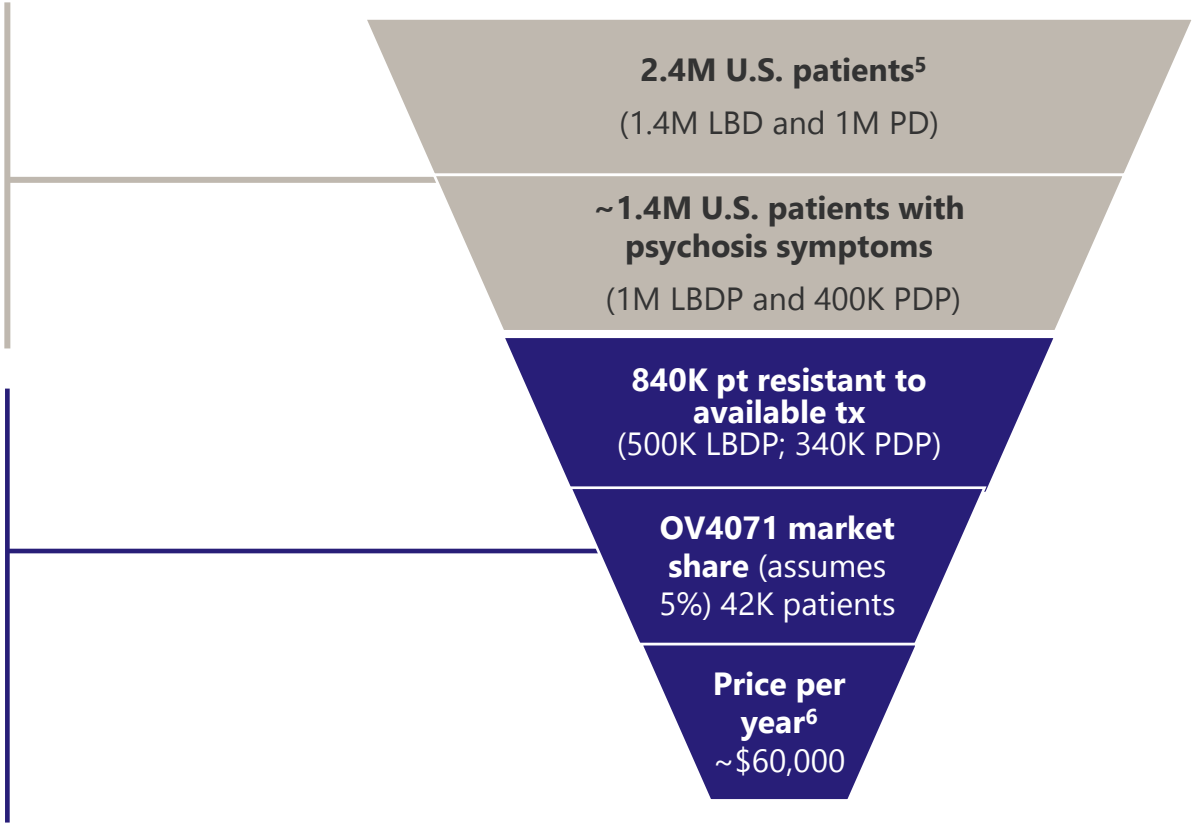
- Trial population heterogeneity
- Endpoint validation and variability
- Speed to enroll trials
- Regulatory risk
- Heavily genericized therapeutic areas

Strategy for 1st indication

- Confirm biological underpinnings of disease pathology (via synuclein assays) and reduce clinical heterogeneity (by focused I/E criteria)
- Deploy validated scales for Parkinson's and LBD as endpoints (SAPS-PD and SAPS-H+D)
- Recruit from existing patient population at Leiden CHDR
- Leverage pimavanserin development approach (approved on N = 185)
- Indications are "brand protected" because atypical antipsychotics contraindicated in PD/LBD psychosis;

A large opportunity for OV4071: Parkinson's disease (PD) psychosis and Lewy body dementia (LPD) psychosis

- LBD and PD prevalence growing as baby boomers age and life span increases
- Nearly all LBD patients (75%)[1] and up to half of PD (20-40%)[2] experience psychosis symptoms, requiring treatment
- In >50% of cases approved treatments have limited efficacy or have severe side effects/ contraindicated[3]
- Use of Nuplazid® (the SoC) is limited due to limited efficacy and a Boxed Warning[4]
- No approved treatment options for LBDP



~$2.5 Billion

1. Diederich et al, Nat Rev Neurol 5, 331–342 (2009). https://doi.org/10.1038/nrneurol.2009.62.
2. Parkingson Foundation: https://www.parkinson.org/understanding-parkinsons/non-movement-symptoms/hallucinations-delusions
3. Lewy Body Dementia Association: https://www.lbda.org/emergency-room-treatment-of-psychosis/ and https://www.lbda.org/treatment-options/ Nuplazid efficacy: https://www.nuplazidhcp.com/efficacy-data#67uQuSE2 ,31CdL3A5qcY1b
4. Nuplazid USPI: https://www.nuplazid.com/pdf/nuplazid-prescribing-information.pdf
5. LBD: https://www.lbda.org/about-lbd/ and PD: https://www.parkinson.org/understanding-parkinsons/statistics#:~:text=Nearly%20one%20million%20people%20in,90%2C000
6. https://www.nuplazidhcp.com/pdf/nuplazid-distribution-fact-sheet.pdf

Catalyst-rich pipeline set to deliver over the next 24 months