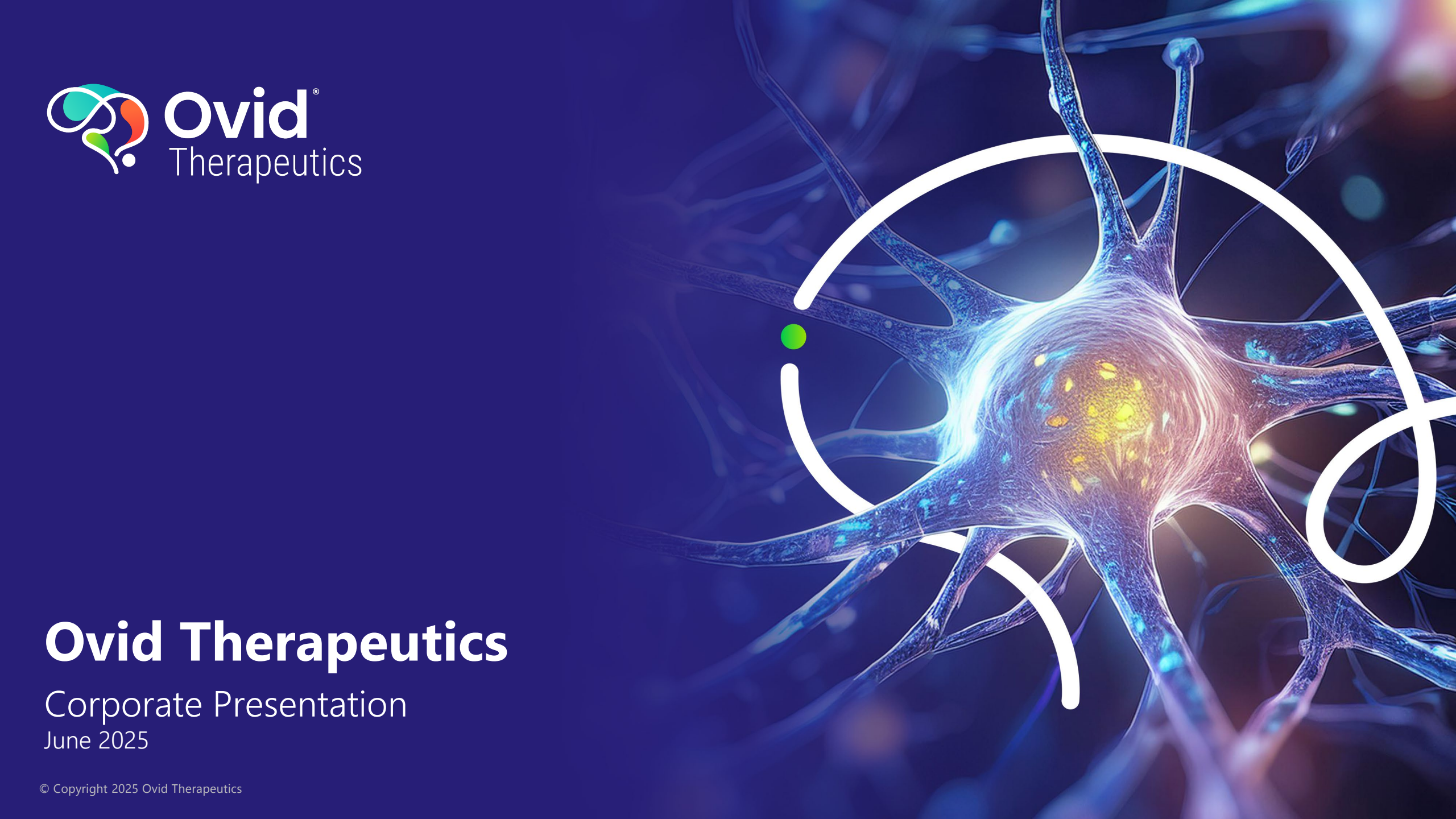


Ovid Therapeutics

Corporate Presentation

June 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, the potential therapeutic benefits of Ovid's current or future product candidates and pipeline programs; Ovid's expectations regarding the duration of its cash balance, and the expectation that it will support the advancement of Ovid's operations and development program; statements regarding the expected timing of the initiation, completion, and results and data of Ovid's clinical studies; expected timing of IND submission for OV4041 oral; the outcome of Ovid's evaluation of the results of recently completed competitor trials and its impact to the OV888/GV101 program; expectations regarding patent term extension for OV329; the potential use, development and therapeutic opportunity of OV329, OV350 and other compounds from Ovid's library of direct activators of KCC2; the likelihood that data, including safety and tolerability data, for OV329 will 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 May 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.

OUR FOCUS

Foundational biological targets

implicated in neuronal hyperexcitability with broad potential therapeutic utility

Precision small molecules

highly specific targeted 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

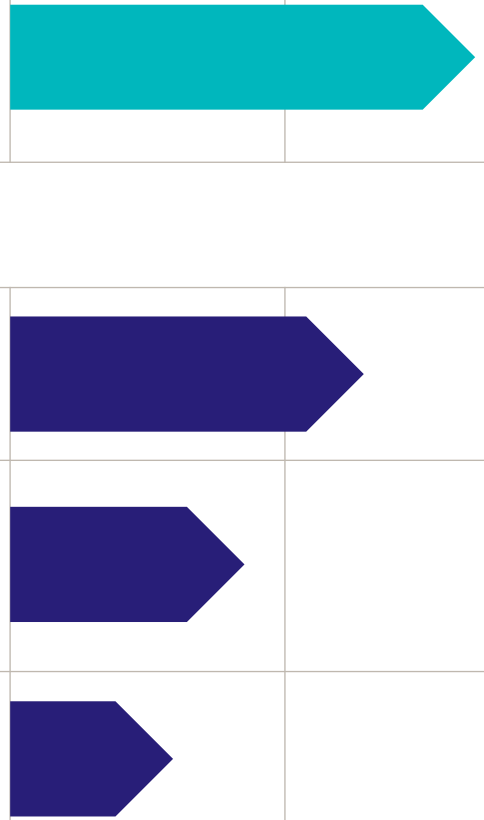
Cash balance of $43 million

expected to fund operations into second half of 2026[1]

1. As of March 31, 2025

Differentiated pipeline, with multiple near-term milestones

Programs	Indication opportunities	Preclinical	Phase 1	Phase 2	Anticipated milestones*
OV329					
GABA-aminotransferase inhibitor	Drug resistant adult epilepsies (DREs) & developmental epileptic encephalopathies (DEEs)				Phase 1 topline Q3 2025 (includes biomarkers of target engagement and potential clinical effect) Phase 2a initiation Q1 2026; topline Q1 2027
KCC2 direct activator portfolio					
OV350 IV	First-in-human potential proof of mechanism				Phase 1 initiated Q1 2025 Safety, tolerability and PK Q4 2025
OV4071 oral	Psychosis assoc. with Parkinson's disease and Lewy body dementia				IND-enabling studies in progress Phase 1 initiation expected in Q2 2026 Patient Phase 1b initiation in Q3 2026; topline results in Q1 2027
OV4041 oral	Generalized anxiety disorder or Rett syndrome				Initiating IND-enabling late 2025 IND submission late H2 2026



* Subject to Ovid capital runway or partnerships.

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 results
Phase 1 safety and biomarkers Q3 2025; Phase 2 initiation Q1 2026

IP through 2041
(assumes five year patent term extension)

10 patent families

Delivering precision inhibition for people living with treatment-resistant focal & generalized seizures

A significant unmet need for a safe and well-tolerated GABA-AT inhibitor

50 million

living with epilepsy across the globe[1]

30% are treatment resistant

meaning failure of two or more drugs[3]

2.4 million

adults living with epilepsy in the US[2]

47% of epilepsy patients in the U.S. report poly-pharmacy use

taking on average five medications[4]

1 World Health Organization epilepsy fact sheet updated in February 2023. https://www.who.int/news-room/fact-sheets/detail/epilepsy
2 Terman SW, Aubert CE, Hill CE, et al. Polypharmacy in patients with epilepsy: a nationally representative cross-sectional study. Epilepsy Behav. 2020;111:107261
3. Beghi E. The epidemiology of epilepsy. Neuroepidemiology. 2020;54(2):185-191. 3) Epilepsy fast facts. Centers for Disease Contr Easl and Prevention
4. Terman SW, Aubert CE, Hill CE, et al. Polypharmacy in patients with epilepsy: a nationally representative cross-sectional study. Epilepsy Behav. 2020;111:10726

Unmet need in drug-resistant epilepsies & OV329 opportunity

Challenges of existing GABA-enhancing medicines

- **Short-acting**
  - Inhibition not sustained
- **Sedating**
- **High dose burden** of 1st generation medicine
  - Sabril (vigabatrin) starting dose is 3 grams, OV329 is ~1000-fold LESS drug
- **Safety challenges of 1st generation**
  - Vigabatrin causes irreversible visual toxicity
  - Black Box and REMS
  - Routine ocular monitoring required

Intended OV329 target product profile*

Optimally tuned inhibition at low doses

- Durable inhibition and anti-convulsant activity

Sustained seizure reduction

- Strong reduction effect across focal & other seizure types

Improved safety profile

- No evidence of ocular changes in humans
- No monitoring requirement expected on label
- No expected titration
- Not sedating in humans
- No anticipated drug-drug interactions

1. *Intended label reflected; profile will need to be demonstrated through future clinical development and patient studies

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

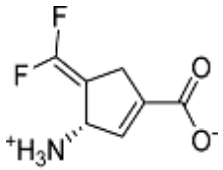
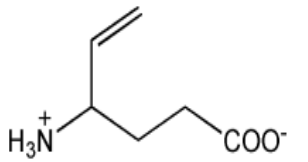
Initial intended indication

Focal seizures in adults with drug resistant epilepsy

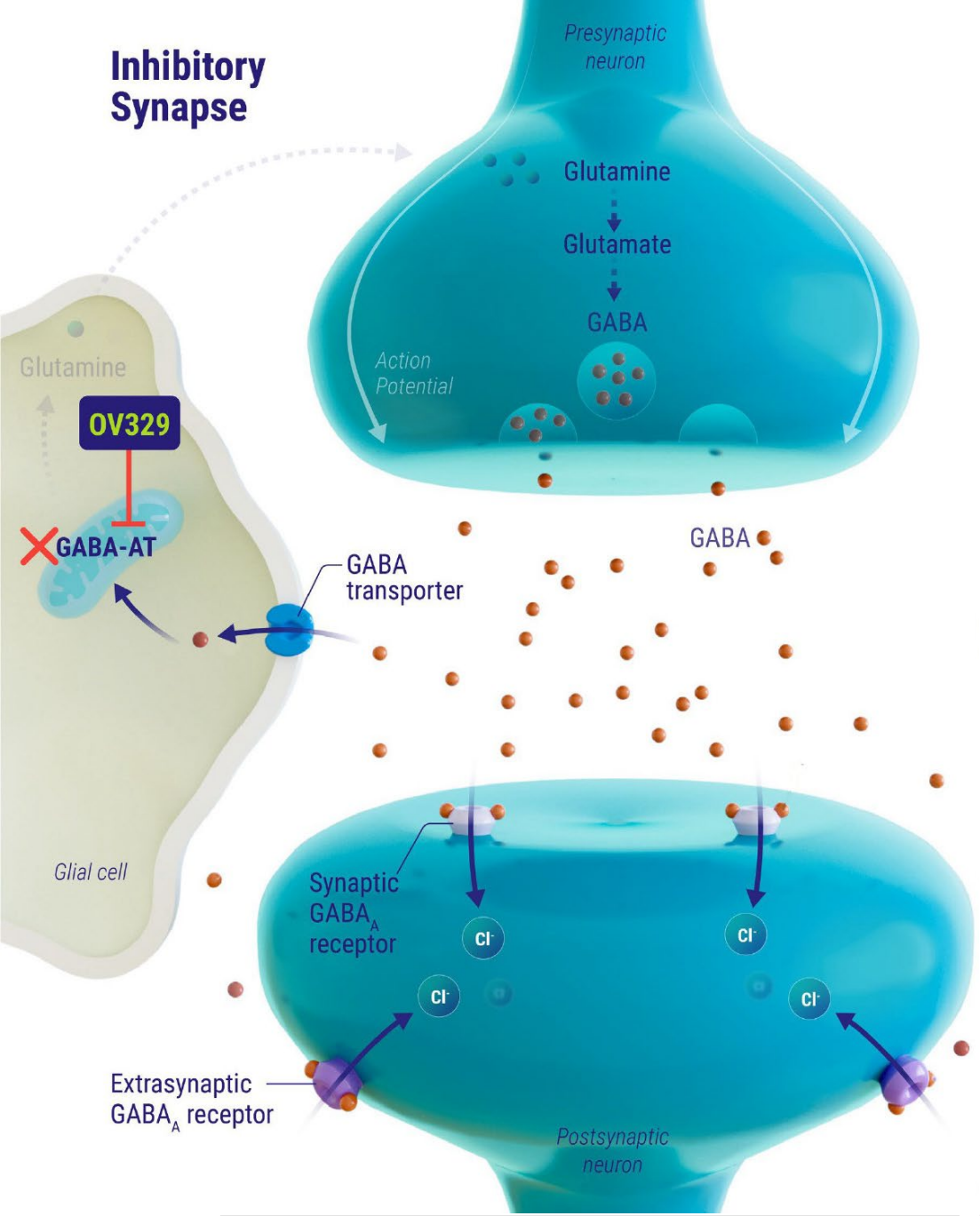
Potential follow-on indications

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

OV329 rationally designed to deliver potential best-in-class profile

	OV329	Vigabatrin
Molecule		
Potency (IC_{50})[1]	~0.1 - 0.3 mM	~60 – 100 mM
Exposure	$T_{1/2}$ ~1.0 Hour with prolonged PD duration	$T_{1/2}$ >5.0 Hours
Mechanism of enzyme inhibition	Electrostatic (irreversible) (more sophisticated chemistry, primarily enamine pathway) [2]	Covalent modification of GABA-AT (irreversible)
Therapeutic index[3,4]	✓ No ocular changes in humans	✗ Visual toxicity at therapeutic dose
Inhibition delivered	Phasic & tonic inhibition (synaptic & extrasynaptic)	Toxicity limits ability to achieve phasic & tonic inhibition

1. 1. J Med Chem. 2012 Jan 26; 55(2): 567–575; 2. J Am Chem Soc. 2018 Feb 14; 140(6): 2151–2164
2. 2. Feja et al. OV329, a novel highly potent γ-aminobutyric acid aminotransferase inactivator, induces pronounced anticonvulsant effects in the pentylenetetrazol seizure threshold test and in amygdala-kindled rats. Epilepsia. 2021 Dec;62(12):3091-3104. doi: 10.1111/epi.17090. Epub 2021 Oct 7.
3. 3. As seen in clinical trials to date.
4. 4. Ovid data on file. Therapeutic index measured in Sprague Dawley rats, a proxy model used to assess ocular safety.



Mechanism of action

- Validated mechanism of action
- Inhibits GABA-AT, the enzyme that catabolizes the inhibitory neurotransmitter, GABA
- Increases GABA in the synapse & extrasynaptic regions
- Believed to optimally tune GABA to create an inhibitory neural milieu

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 Phase 1 trial design

Establish safety & tolerability of GABA-AT inhibitor OV329

Exploratory biomarkers measuring biological effect and target engagement

- Transcranial magnetic stimulation (TMS)
- Magnetic resonance spectroscopy (MRS)
- Electroencephalography (EEG)

Endpoints

- Pharmacokinetics
- Safety and tolerability
- Ophthalmic safety metrics & monitoring (30 days)
  - Best corrected visual acuity
  - Fundus photography
  - Indirect dilated ophthalmoscopy
  - Automated threshold visual field perimetry
  - Optical coherence tomography (OCT)

Excellent safety observed to date

- No serious adverse events
- Adverse events mild and transient (e.g., headache)
- Cleared for 28-day daily patient dosing

Current cohort

- Higher dose cohort added to optimize Phase 2 dosing opportunities
- Larger patient enrollment to support ability to show biomarker signal

1. Ovid data on file

OV329 has differentiated PK, PD & ocular profile in preclinical findings

- ✓ Penetrates the brain, as it is found in animal plasma and brain tissues[1]
- ✓ Anti-convulsant activity across an extensive range of seizure models
- ✓ >100x more potent than vigabatrin in animal studies[1]
- ✓ Undetectable in the retina, eye and brain tissue (vigabatrin shown to accumulate 4x in the retina)[2]
- ✓ Rapid tissue clearance properties and half-life (1.5 hours) compared to vigabatrin (4 hours)[1]
- ✓ Established therapeutic index; vigabatrin produces ocular toxicity at therapeutic dose

1. Mukherjee, J., et al. (2023). Blocking of GABA-AT Activity Selectively Alters Tonic and Phasic Inhibition. Poster presented at the 2023 American Epilepsy Society Conference
2. Tsai, J., et al. (2024). Evaluation of the Potential Accumulation of OV329 in the Brain, Retina, and Eye Following Continuous Infusion. Poster presented at the 2024 Epilepsy Pipeline Conference

Anti-convulsant activity demonstrated in 9 seizure models

Seizure reduction seen in chronic & acute seizure models

	i.v. (ivPTZ)	NMDA-Induced Infantile Spasm model	Audiogenic Seizure	Amygdala Kindled	Corneal Kindled	Intrahippocampal Kainate Model of Mesial-Temporal Lobe Epilepsy (MTLE)	Intraamygdala Kainate Model of Mesial-Temporal Lobe Epilepsy (MTLE)	Lithium-Pilocarpine	Dravet *Scn1a* $^{A1783V/WT}$
Injury Model	Acute/ seizure	Acute/ seizure	Acute	Chronic/ epilepsy	Chronic/ epilepsy	Chronic/ epilepsy	Epilepsy prevention/ modification	Acute/ seizure	Chronic/ Genetic epilepsy
Clinical Correlate	Nonconvulsive Seizures (e.g., absence, myoclonic)	Infantile spasms	Generalized seizures	Chronic focal to bilateral tonic-clonic seizure/ Pharmacoresistant seizures	Chronic focal to bilateral tonic-clonic seizure	Focal Mesial temporal lobe epilepsy/ Pharmacoresistant seizures; Status Epilepticus	Focal Mesial temporal lobe epilepsy/ Pharmacoresistant seizures; Status Epilepticus	Like human, rodents exhibit EEG abnormalities, convulsions, and cognitive impairment	Spontaneous seizures, higher rate of SUDEP. Hyperthermia-induced Pharmcoresistant seizures.
Species	Rat	Mouse	Mouse	Rat	Mouse	Mouse	Mouse	Rat	Mouse
Dosing	Acute (5, 20, 40 mg/kg i.p.)	Acute (0.0025, 0.01, 0.1, 1 mg/kg, p.o.)	Acute (0.01, 0.05, 0.1 mg/kg, p.o.)	Acute (30, 40 mg/kg, i.p.)	Acute (1, 3, 10, 20, 30, 40, 60 mg/kg, p.o.)	Acute, single dose (0.01, 0.1, 1, 10 mg/kg, p.o.; 10 mg/kg, i.p.) Subacute (8 days q.d.) 0.3, 1.0 and 3.0 mg/kg/day (p.o.)	Acute (40 mg/kg p.o.)	Acute (15mg/kg, IV)	Repeat (10mg/kg x 4d, IP)
Activity	+	+	+	+	+	+	+	+	+

1. Ovid data on file

Differentiated safety in retinal model required by regulators

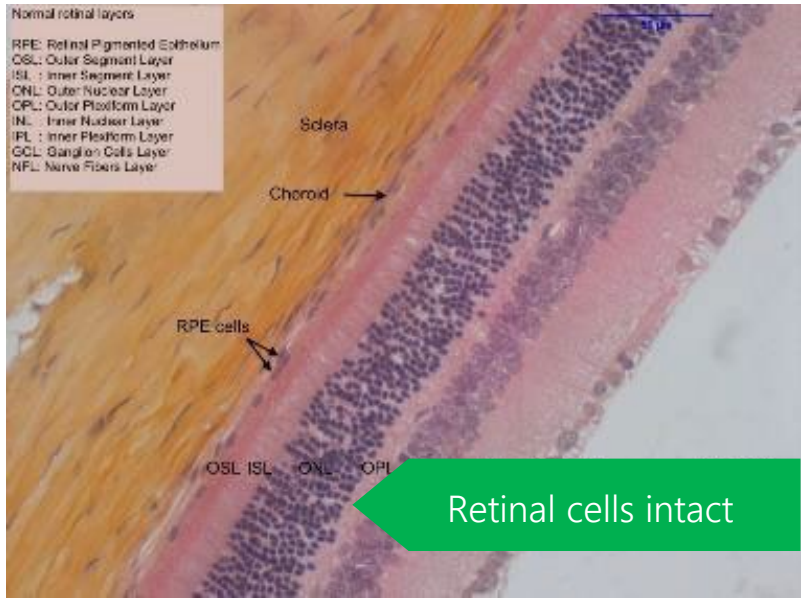
Anti-convulsant activity in 9 animal seizure models

- Represent a wide range of seizures
- Clinically active at low doses

Ocular safety profile differentiation

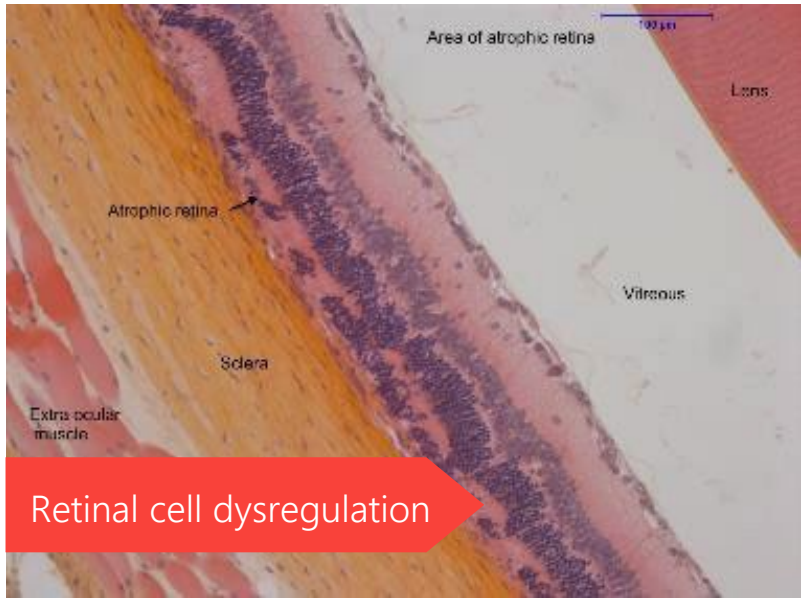
- Demonstrated in 2 models & toxicology
- Unique potency, binding & clearance profile likely related to lack of accumulation in eye

OV329 3.0 mg/kg
(Therapeutic dose)



No ocular effects seen in 3 mg/kg q.d. in rats

vigabatrin 300 mg/kg
(Therapeutic dose)



Ocular changes in more than half of rats treated (300 mg/kg)

45-day study

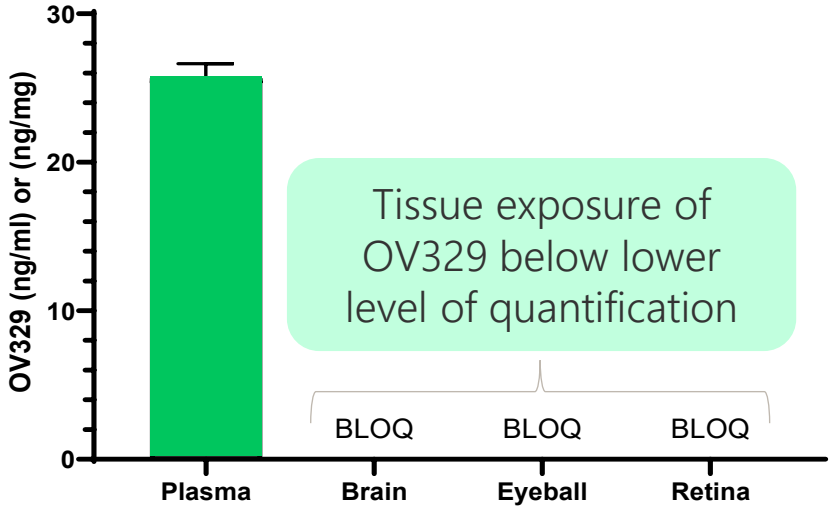
Ovid data on file

Head-to-head ocular accumulation study of OV329 versus vigabatrin

Lack of retinal accumulation of OV329 supports differentiated ocular safety profile vs. vigabatrin[1]

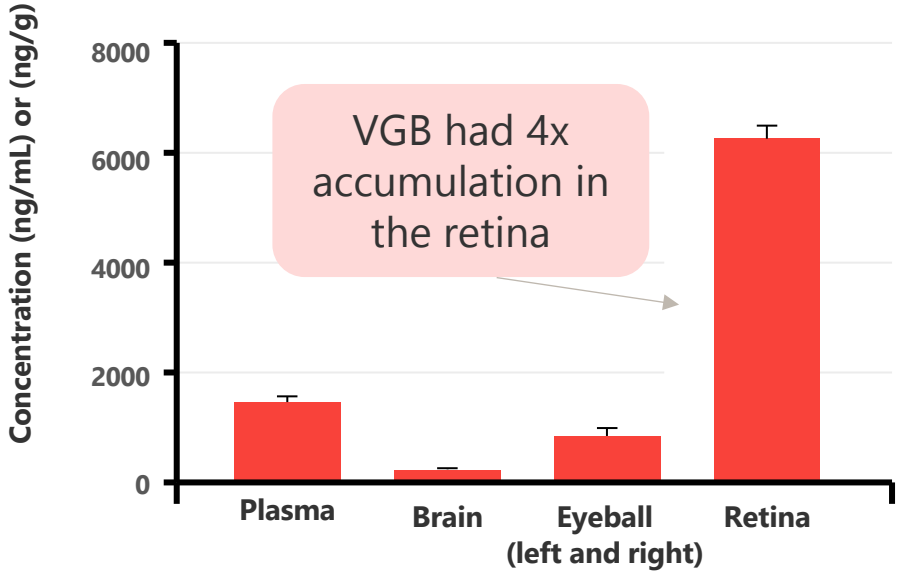
OV329

Mean Plasma and Tissue Concentration 5.0 mg/kg/day



VIGABATRIN

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



FINDINGS[1]

- OV329 penetrated the brain, was present in the plasma and then cleared the tissue
- No accumulation detected of OV329 in the eye or retina
- 4x greater exposure of vigabatrin in retina as compared to plasma
- Suggests vigabatrin, but not OV329, preferentially partitions into the retina when plasma exposure is kept at a relatively constant level

1. Tsai, J., et al. (2024). Evaluation of the Potential Accumulation of OV329 in the Brain, Retina, and Eye Following Continuous Infusion. Poster presented at the 2024 Epilepsy Pipeline Conference.

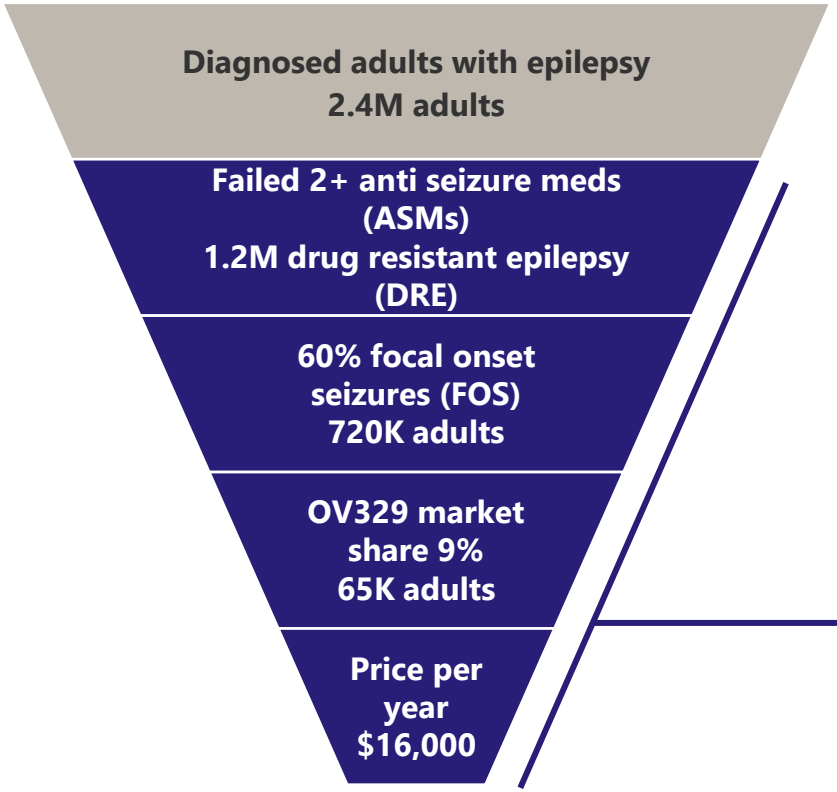
Comprehensive biomarker strategy evaluating OV329

Tool	Magnetic resonance spectroscopy (MRS)	Transcranial magnetic stimulation (TMS)	Electroencephalogram (EEG)
Measures	**Neurotransmitter concentrations**	**Cortical excitability and brain circuit function**	**Brain waves linked to inhibitory (GABAergic) activity**
Metrics* (positive & negative controls)	• GABA concentrations in the medial parietal lobe	• Cortical silent period (CSP) • Paired-pulse long-interval intracortical inhibition (LICI) • Paired-pulse short-interval intracortical inhibition (SICI) • Resting motor threshold (rMT) • Intracortical facilitation (ICF)	High and low frequencies
What it suggests	✓ **Target engagement** as measured by increased in GABA concentrations using modern MRI "fitting" technology	✓ **Pharmacodynamic activity** on $GABA_A$, $GABA_B$ receptors and overall enhancing brain inhibition ✗ No excitatory (glutamatergic) activity	✓ **GABAergic pharmacodynamic** effect as shown by changes in high frequency brain activity

1. * All metrics compare post-OV329 values with baseline

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

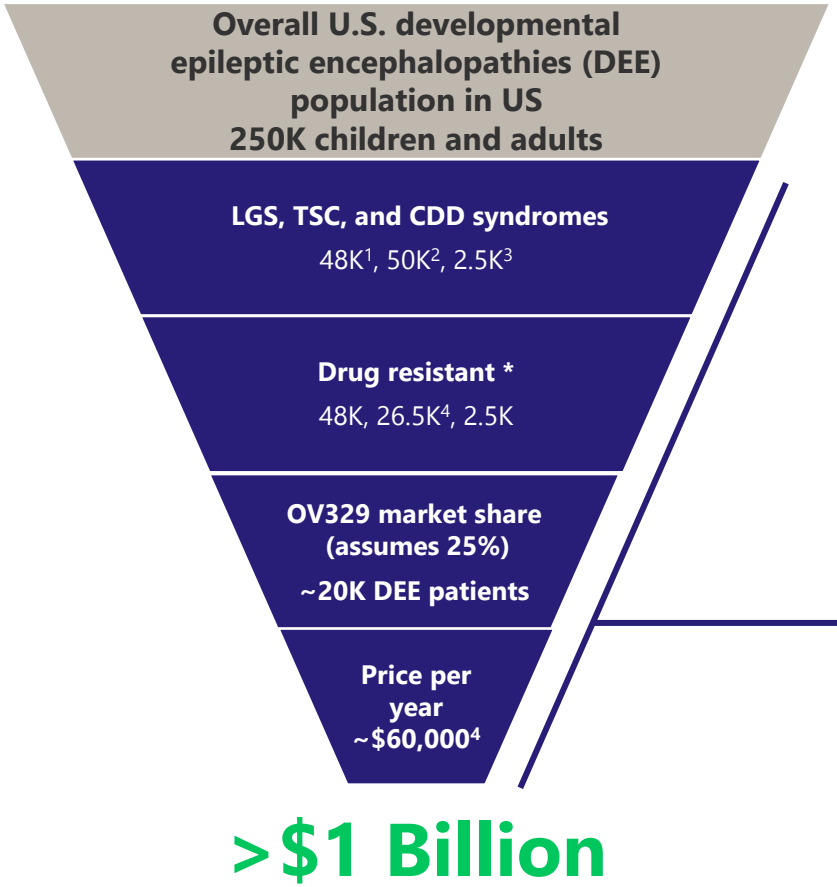
1L	Carbamazepine	Levetiracetam	Oxcarbazepine
	Phenytoin	Eslicarbazepine	Lacosamide
2L	Clobazam	Lamotrigine	Topiramate
	Rufinamide	Zonisamide	
Refractory + polypharmacy (~40%)	OV329 *In Phase 1*	XEN1101	XCOPRI
		Fycompa perampanel	BRIVIACT (brivaracetam)

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

Continued unmet need across a range of rare epilepsies; limited treatment options indicated for LGS, TSC, CDD with drug resistant seizures

Substantial opportunity within the DEE segment



>$1 Billion

	TSC	LGS	CDD
1L	Vigabatrin Valproate	Valproate Clobazam	Valproate Clobazam
2L	Clobazam Levetiracetam Lamotrigine Carbamazepine	Felbamate Lamotrigine Topiramate Rufinamide	Levetiracetam Topiramate Vigabatrin
3L (Failed 2 ASMs- prior authorization criteria)	Epidiolex® (cannabidiol)	Epidiolex® (cannabidiol) Fintepla® (fenfluramine)	Ztalmy® (ganaxolone) oral suspension 50 mg/mL Epidiolex® (cannabidiol)
DEE options in development	OV329 *In phase 1* Bexicaserin *Phase 2 completed*		
Non-pharm. Options	Surgical Resection Vagus Nerve Stimulation Ketogenic Diet		

1. https://www.tscalliance.org/understanding-tsc/what-is-tsc/
2. https://www.lgsfoundation.org/about-lgs-2/how-many-people-have-lgs/https://pmc.ncbi.nlm.nih.gov/articles/PMC3065368/
3. https://www.epilepsy.com/causes/genetic/cdkl5-disorder
4. Assuming weight based dosing 50% 25kg and 50% 55kg. Analgoue pricing Fintepla ranges from 60-130K and epidiolex 35k-70K

* assuming all patients will become resistant at some point during treatment; TSC 80% with seizure and 65% resistant

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

Portfolio of unique KCC2 direct activators
Potassium chloride cotransporter 2 (KCC2)

Fundamental target "master switch" in neuronal hyperexcitability

Potential first-in-class direct activators

OV350 initiated Phase 1 Q1 2025

IP through 2046 (assumes five year patent term extension)

9 method-of-use patents filed

KCC2 maintains inhibitory/excitatory balance & normalizes neuronal activity[1]

"Master switch" for a broad spectrum of neurological disorders

where hyperexcitability accelerates disease progression

Precise MoA exclusively expressed in the brain

To rebalance network hyperexcitability & mitigate safety effects associated with other potassium chloride transporters that are broadly expressed

Final "common pathway"

Downstream of many genetic and acquired causes of neurological dysfunction and central to maintain GABAergic inhibitory tone

Impossible to "over modulate"

Energetically challenging to over-extrude chloride from the neuron which should contribute to a cleaner profile

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

KCC2 regulates GABA inhibition by maintaining neuronal chloride homeostasis[1]

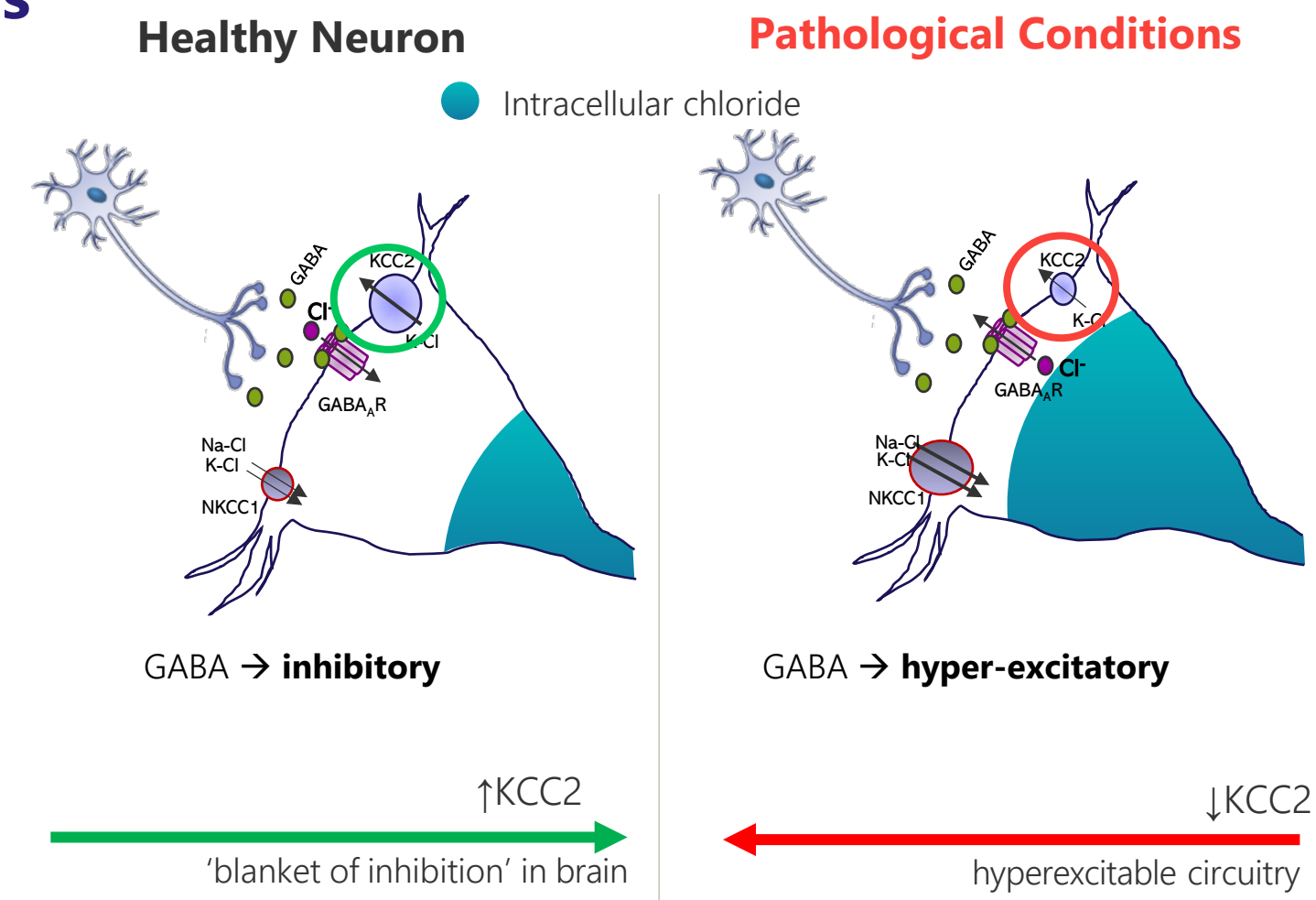
KCC2 regulates chloride gradient

- Chloride gradient is necessary for GABA-ergic inhibition at the synapse

Manages the passive inward flux of chloride through activated ion channels

Maintaining low intracellular chloride is required for:

- The efficacy of hyperpolarization
- Shunting inhibition mediated by GABAergic signaling in neurons



Activating KCC2 restores the brain's natural braking system, fixing network defects that cause excessive neural activity (hyperexcitability)

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

Franchise of KCC2 direct activators in development

OV350 & OV4071 behave similarly in phenotypic screens

	OV350	OV4071	OV4XXX
Formulation	Intravenous	Oral Intramuscular injection / SubQ	Oral Intramuscular injection
Indication	**First-in-human potential proof-of-mechanism**	**Psychosis in Parkinson's disease & Lewy body dementias**	**Generalized anxiety disorder Rett syndrome**
Characteristics (from disease & phenotypical models)	• Antipsychotic & anticonvulsant activity • No AEs • 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
Anticipated milestones	Phase 1 initiated Q1 2025 Safety, tolerability/PK completion Q4 2025	Currently in IND-enabling Phase 1 initiation Q2 2026 Topline results Q1 2027	Initiating IND-enabling studies late 2025/early 2026 IND submission late 2026

In Phase 1: OV350

Dose dependent exposure

- ✓ Strong activity in psychosis phenotypic & disease models
- ✓ Dose-exposure-PD relationship demonstrated in animals
- ✓ Behaves like atypical antipsychotics
- ✓ Outperforms to clozapine in phencyclidine-induced psychosis model

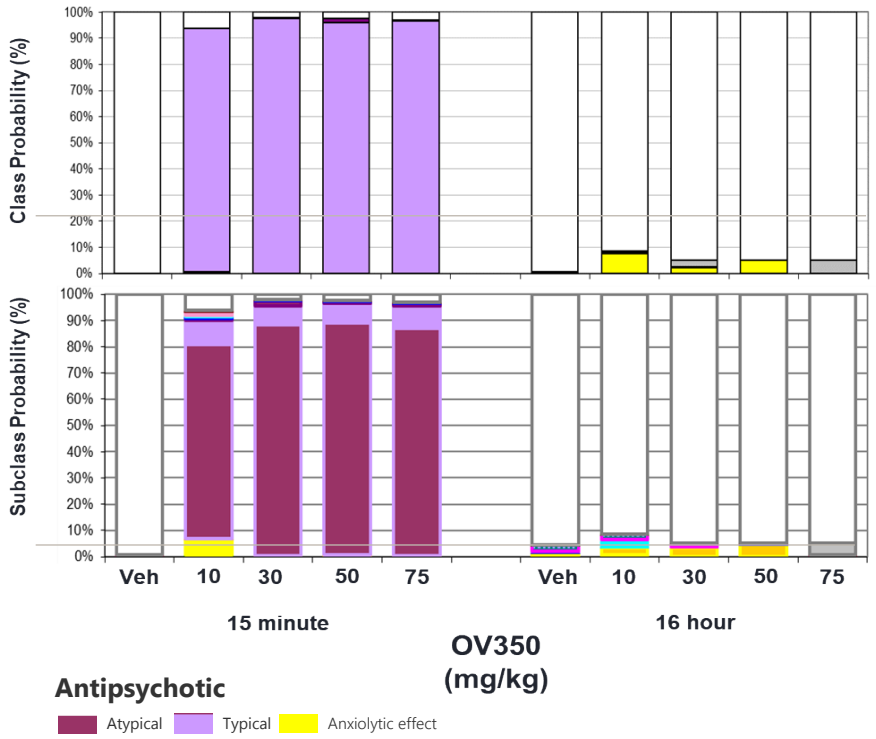
Safety & margins

- ✓ Started Phase 1 dosing Q1 2025
- ✓ Permissive safety package
- ✓ No observed adverse effect level (NOAEL) in rats & dogs
- ✓ No evidence of sedation
- ✓ Ames & in vitro mutagenesis tests negative (i.e., clean)
- ✓ DDI assessment completed with no concerns

OV350 elicits potent, robust, rapid and reversible antipsychotic activities[1]

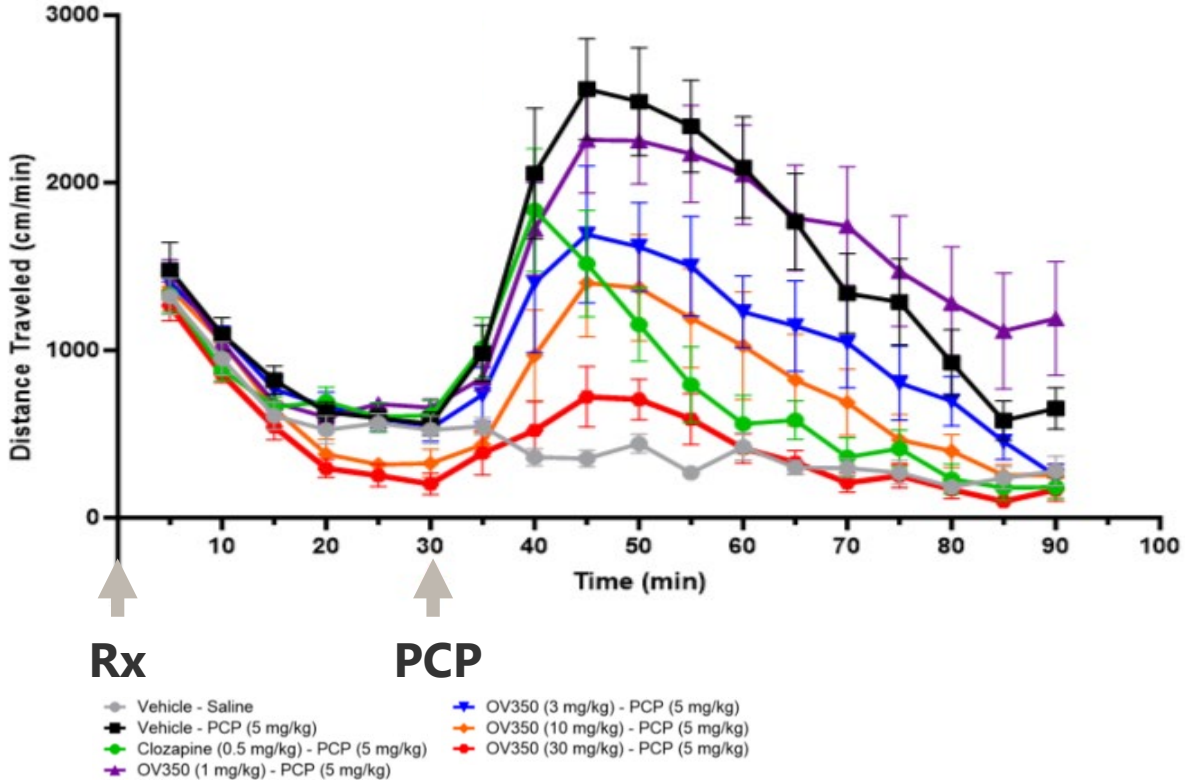
Behaves as atypical antipsychotic with a clean profile in SmartCube®

- 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

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



1. Ovid data on file

OV350 enables 1st in class characterization & rapid path to patients for oral KCC2 direct activators

- ✓ Phase 1 demonstrate acceptable safety (for a first-in-class approach)
- ✓ Exploratory quantitative EEG biomarker for translation
- ✓ Enables ability to potentially establish PK/PD relationship & apply insights to oral programs
- ✓ Characterize safety, tolerability, PK for non-CNS indications

Plan to initiate OV4071 (oral) program in Q2 2026, which behaves similarly to OV350 in phenotypic models

- ✓ Potential to establish proof-of-mechanism in patients via 1b
- ✓ Program currently in IND-enabling

IND-enabling studies: OV4071, an oral direct activator

Potent, dose dependent PK/PD

- >20x potency than OV350
- Strong activity in psychosis phenotypic & disease models
- Dose dependent response in animals

Safety & margins

- 60x in vivo EC50 reached in repeat dosing
- Excellent tolerability in repeat dosing
- Consistent plasma exposure
- Progressing in non-GLP toxicology studies

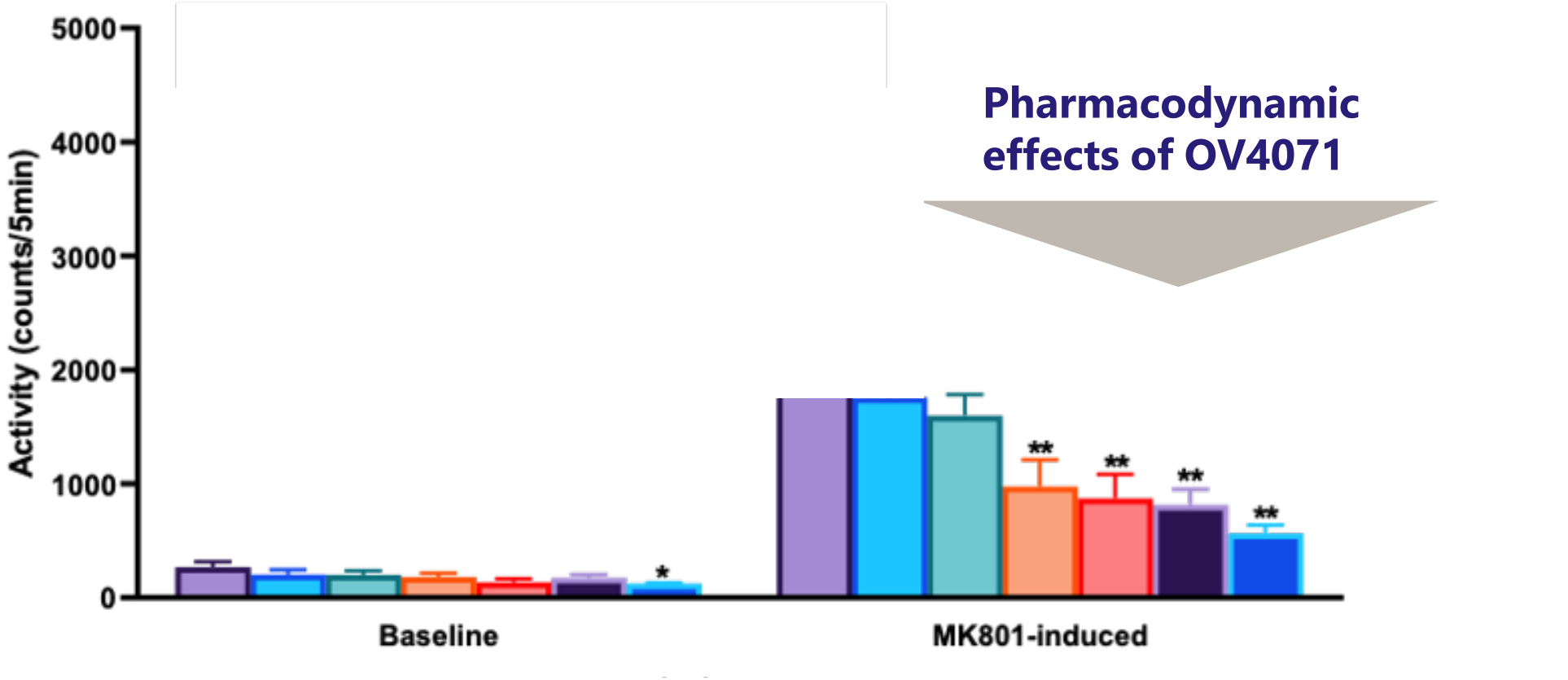
Large opportunity

- Similar to OV350
- Psychosis with Parkinson's disease, Lewy body dementia

1. Ovid data on file

OV4071 dose dependent response in schizophrenia psychosis model

Uses an NMDAR antagonist (MK801) to elicit dopamine release in the basal ganglia



1. Ovid data on file

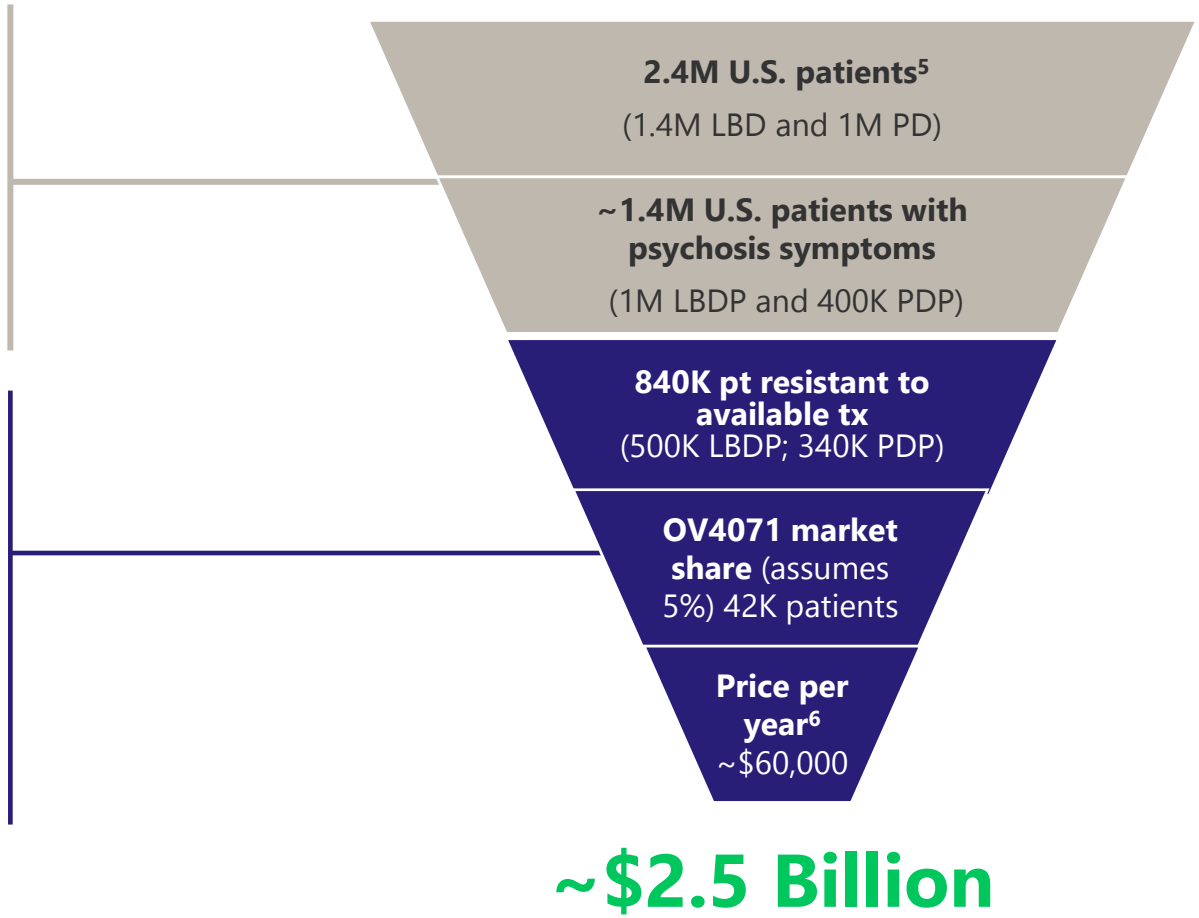
OV4071 indication represents a significant unmet need

	Psychoses associated with neuronal synuclein disorders (NSD)
Target population	Parkinson's disease psychosis Lewy body dementia psychosis (both NSDs)[1]
MoA relevance	Regulation of chloride balance is lost and GABA inhibitory signaling does not occur normally, KCC2 is downregulated in certain forms of psychosis[2]
Enrichment strategy	Neuronal synuclein disorder positive (can be confirmed via skin biopsy test & CSF) Screening for hyposmia
Unmet need	• Only 12% of patients respond to SoC (Nuplazid)[3] • Atypical antipsychotics contraindicated • High morbidity, mortality and cost • Highest risk of nursing home placement[4]
Value proposition	Most patients do not have a therapy that works >70% of PDP and LBD patients will experience psychosis symptoms[5,6]

1. Simuni et al. Lancet Neurol. 2024 Feb;23(2):178-190. doi: 10.1016/S1474-4422(23)00405
2. Hyde et al. J Neuroscci. 2011 Jul 27;31(30):11088–11095
3. Nuplazid USPI: https://www.nuplazid.com/pdf/nuplazid-prescribing-information.pdf
4. Goetz and Stebbins. Neurology. 1993 Nov;43(11):2227-9. doi: 10.1212/wnl.43.11.2227
5. Diederich et al, Nat Rev Neurol 5, 331–342 (2009). https://doi.org/10.1038/nrneurol.2009.62.
6. Lewy Body Dementia Association: https://www.lbda.org/emergency-room-treatment-of-psychosis/

A large opportunity for OV4071: Parkinson's disease psychosis and Lewy body disease 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#67uQuSEzX31CdL3A5qcY1b
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

Delivering a differentiated and rapidly advancing pipeline

- Potential for significant opportunity with OV329 in adult drug-resistant epilepsies or developmental epileptic encephalopathies (DEEs)
- Multiple, unique KCC2 programs enable the potential for optionality with co-development, transactions and relative to the Inflation Reduction Act

Topline data (biomarkers for clinical effect and target engagement, safety & tolerability)	Q3 2025
1st KCC2 direct activator entered human trials	Q1 2025
Oral KCC2 direct activator entering humans	Q2 2026