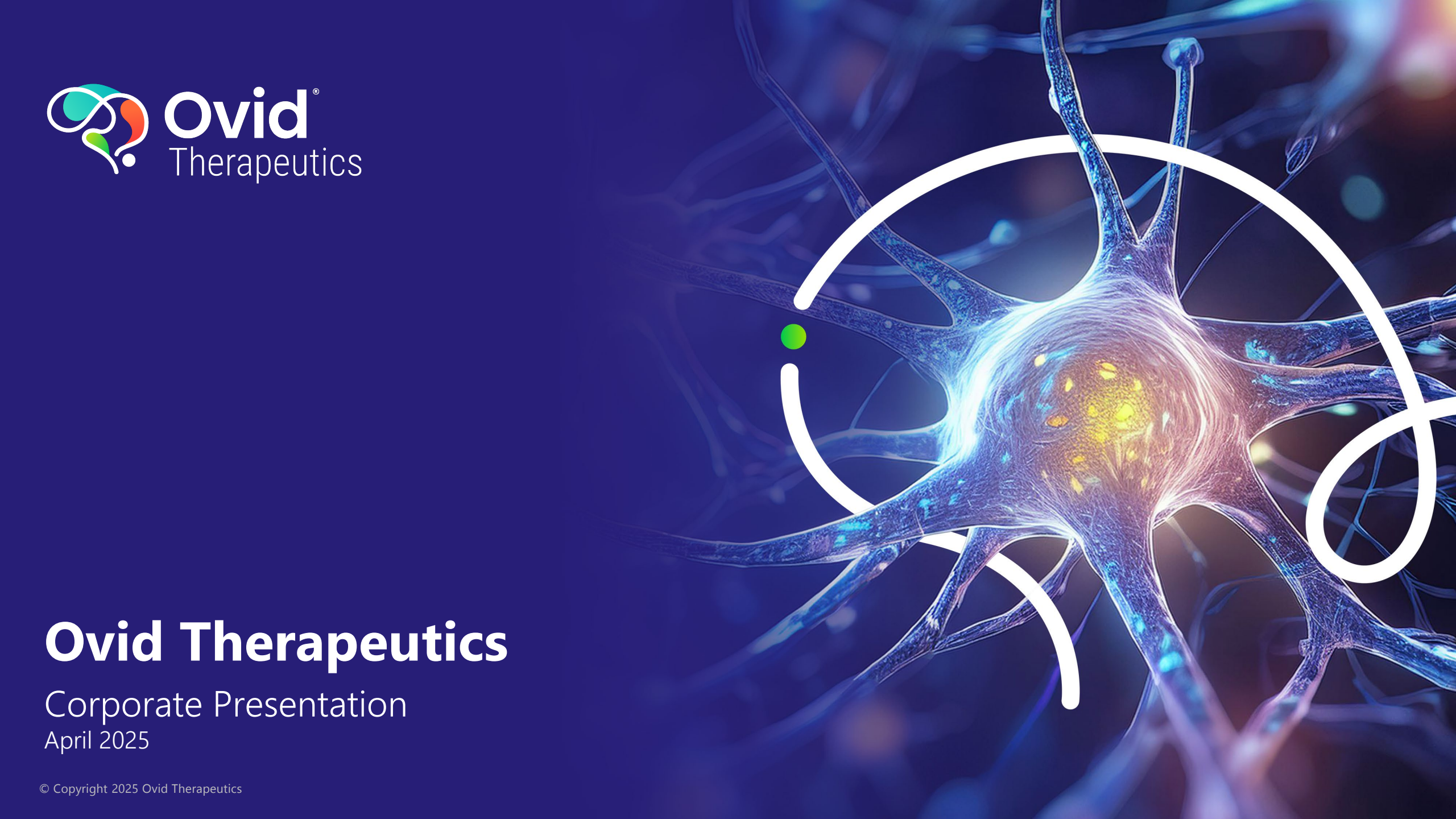

Ovid Therapeutics

Corporate Presentation

April 2025

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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; and Ovid's or any of its partners' abilities to meet anticipated deadlines and milestones. 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 Annual Report on Form 10-K filed with the Securities and Exchange Commission (SEC) on March 11, 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.

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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 $53.1 million

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

1. As of December 31, 2024

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Differentiated pipeline, rapidly advancing in the clinic

Programs	Indication opportunities	Preclinical	Phase 1	Phase 2	Anticipated milestones[1]
OV329					
GABA-aminotransferase inhibitor	Drug resistant adult epilepsies (DREs) & developmental epileptic encephalopathies (DEEs)				Phase 1 topline Q3 2025 (includes clinical effect & target engagement biomarkers) Phase 2a initiation Q1 2026; topline Q1 2027
KCC2 direct activator portfolio					
OV350 IV	Proof of mechanism				Regulatory submission filed in Q4 2024 Phase 1 initiated Q1 2025; topline Q4 2025 Potential proof of mechanism in patients: Q4 2026
OV4071 oral	Psychosis assoc. with Parkinson's disease and Lewy body dementia				Currently in IND-enabling studies Phase 1 initiation Q2 2026 Phase 1b initiation Q3 2026; topline results Q1 2027
OV4041 oral	Generalized anxiety disorder or Rett syndrome				Initiating IND-enabling studies late 2025 IND submission late H2 2026

Ovid has a Phase 2-ready ROCK2 inhibitor, OV888, in collaboration with Graviton Bioscience. This program was cleared by regulators to initiate Phase 2 but is currently paused while Ovid monitors the outcome of regulatory interactions by competitors.

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1. Some milestones are subject to Ovid's ability to raise additional capital and/or enter additional 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

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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

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OV329, a next-generation GABA-AT inhibitor

Challenges of current GABA-enhancing medicines:

- Short-acting (inhibition not sustained)
- Sedating
- High dose burden of 1st gen
  - Sabril (vigabatrin) starting dose is >1,000 mg, OV329 is ~1,000 less
- Safety concerns
  - Vigabatrin causes visual toxicity, with a Black Box and REMS

Intended OV329 target product profile*

Efficacy and potency

- Durable inhibition and anti-convulsant activity
- Biological activity at 5x margin

Safety

- No evidence of ocular changes in humans
- No monitoring requirement expected on label
- No expected titration
- No sedation seen in humans
- No anticipated drug-drug interactions

Potential to treat

- Developmental epileptic encephalopathies
- Adult drug-resistant epilepsies
- Other conditions in which neuronal hyperexcitation is implicated, such as post-operative pain

Multiple $1B Market Opportunities

1. *Will need to be demonstrated through future patient study, continuing long-term toxicology and further clinical development

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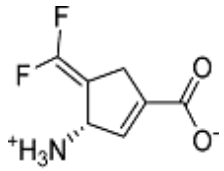
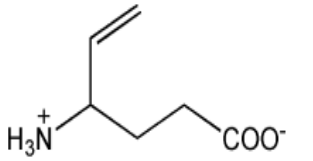
Factors contributing to OV329 differentiation vs vgb

	OV329	Vigabatrin
Molecule		
Potency	IC_{50}: ~0.1 - 0.3 μM	IC_{50}: ~60 – 100 μM
Exposure characteristics	Reduced exposure ($T_{1/2}$ ~1.0 Hour) coupled with prolonged PD duration	$T_{1/2}$ >5.0 Hours Both R (inactive) & S (active) enantiomers
Mechanism of enzyme inhibition	Electrostatic (irreversible) (more sophisticated chemistry, primarily enamine pathway) [2]	Covalent modification of GABA-AT (irreversible) (early generation chemistry, primarily Michael addition pathway)[1]
Purity	Stereoisomer	Mixture of active (S) and inactive (R) enantiomers
Inhibition	Phasic + tonic (synaptic & extrasynaptic)	Primarily phasic (synaptic)
Therapeutic index (as measured in Sprague Dawley rats, a proxy model used to assess ocular safety)	✓	✗ None – toxicity seen at therapeutic dose
Teratogenic	No	Yes (rats, mice and potentially in humans)

»» Improved potency (100-1,000x), PK/PD and established therapeutic index in animals[2]

1. J Med Chem. 2012 Jan 26; 55(2): 567–575; 2. J Am Chem Soc. 2018 Feb 14; 140(6): 2151–2164
2. Feja et al. OV329, a novel highly potent γ-aminobutyric acid aminotransferase inactivator, induces pronounced anticonvulsant effects in the pentylenetetrazole seizure threshold test and in amygdala-kindled rats. Epilepsia. 2021 Dec;62(12):3091-3104. doi: 10.1111/epi.17090. Epub 2021 Oct 7.
Ovid data on file

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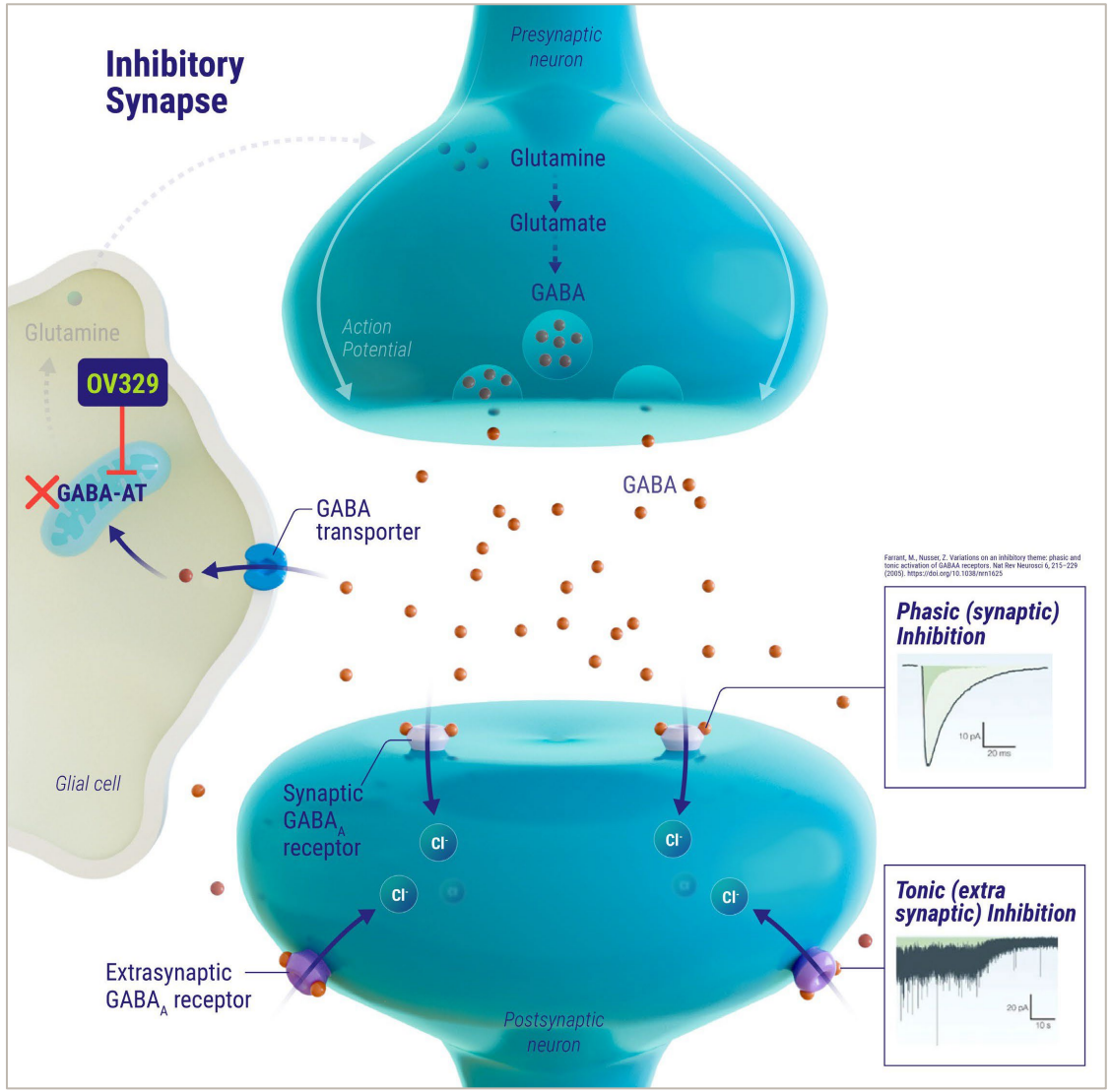
GABA-AT inhibition a validated mechanism of action

- GABA is the most common inhibitory neurotransmitter in the central nervous system
- GABA aminotransferase (GABA-AT) is responsible for degrading GABA[1]
- OV329 substantially reduces GABA-AT activity, increasing GABA in the synapse & extra-synaptic region[2]
- Moderates excessive neuronal excitation to reduce seizure activity



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

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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)

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

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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

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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	+	+	+	+	+	+	+	+	+

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Differentiated safety in retinal model required by regulators

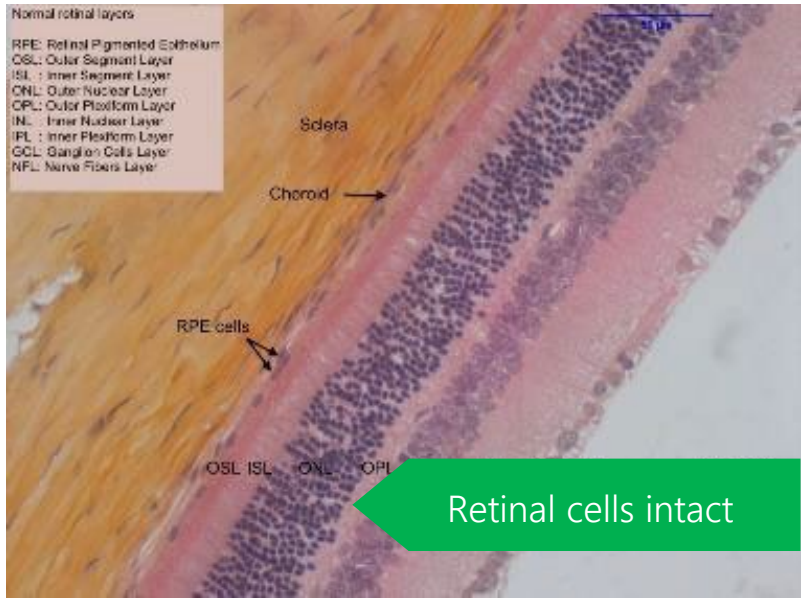
Anti-convulsant activity in 9 animal seizure models

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

Ocular safety profile differentiation

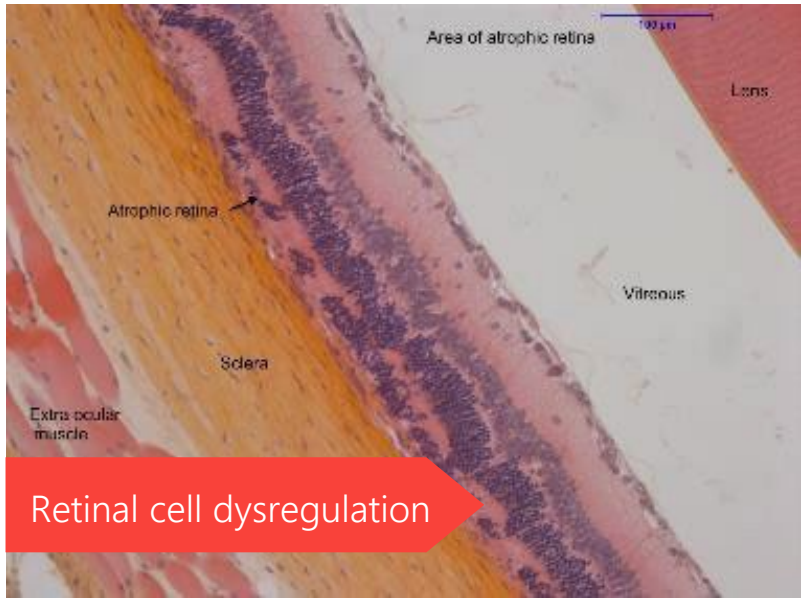
- Demonstrated in 2 models & toxicology
- Unique potency, binding & clearance profile likely related to lack of accumulation in eye

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

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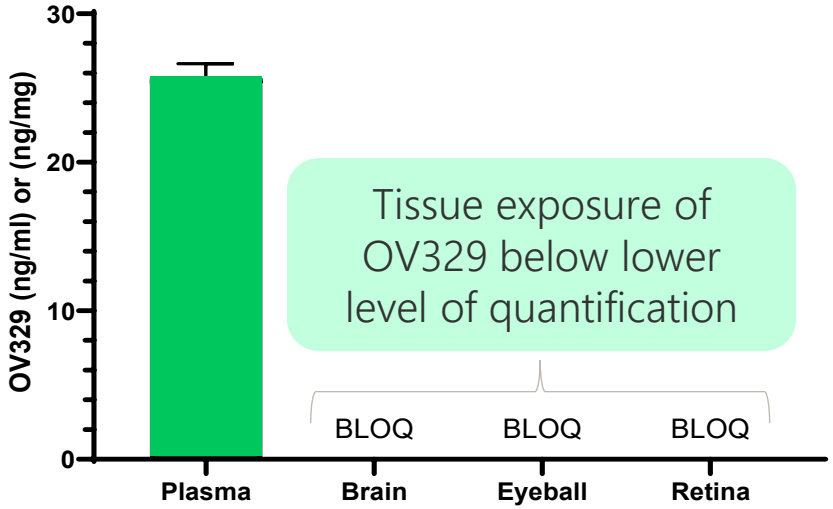
Head-to-head ocular accumulation study of OV329 versus vigabatrin

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

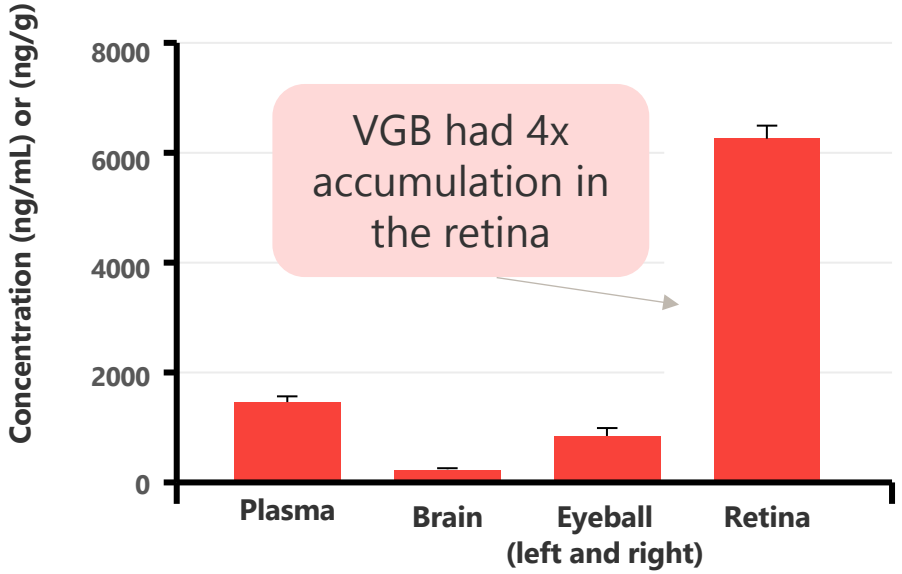
OV329

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.

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Biomarker strategy

Biological activity

Transcranial magnetic stimulation (TMS)

Therapeutic dose of VGB increased two key parameters[1,2]

- **Long-interval intracortical inhibition (LICI) 150**
  A lengthened time interval between two stimuli suggesting increased intercortical inhibition
- **Cortical silent period (CSP)**
  Longer CSPs suggest increased inhibition

TMS is among the best predictive tools for ASMs that we have in drug development (short of a patient trial).

Target engagement

Magnetic resonance spectroscopy (MRS)

Can measure GABA levels in multiple areas of the brain

Experts recommend measuring GABA levels in the medial parietal lobe (guidelines and best practice)

Ambient and quantifiable levels of GABA increasing in treated participants, suggesting that GABA-aminotransferase (GABA-AT), the enzyme that catabolizes GABA, is being inhibited by OV329

Consistent response

Trend across post-treatment participants
Seek to see directional changes across the majority of treated participants relative to their untreated baseline

1. Tsuboyama M, Kaye HL, Rotenberg A. Biomarkers obtained by transcranial magnetic stimulation of the motor cortex in epilepsy. Front Integr Neurosci. 2019;13:57. doi:10.3389/fnint.2019.00057.
2. Pierantozzi M, Marciani MG, Palmieri MG, Brusa L, Galati S, Caramia MD, 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 Res. 2004;1028(1):1-8. doi:10.1016/j.brainres.2004.06.009

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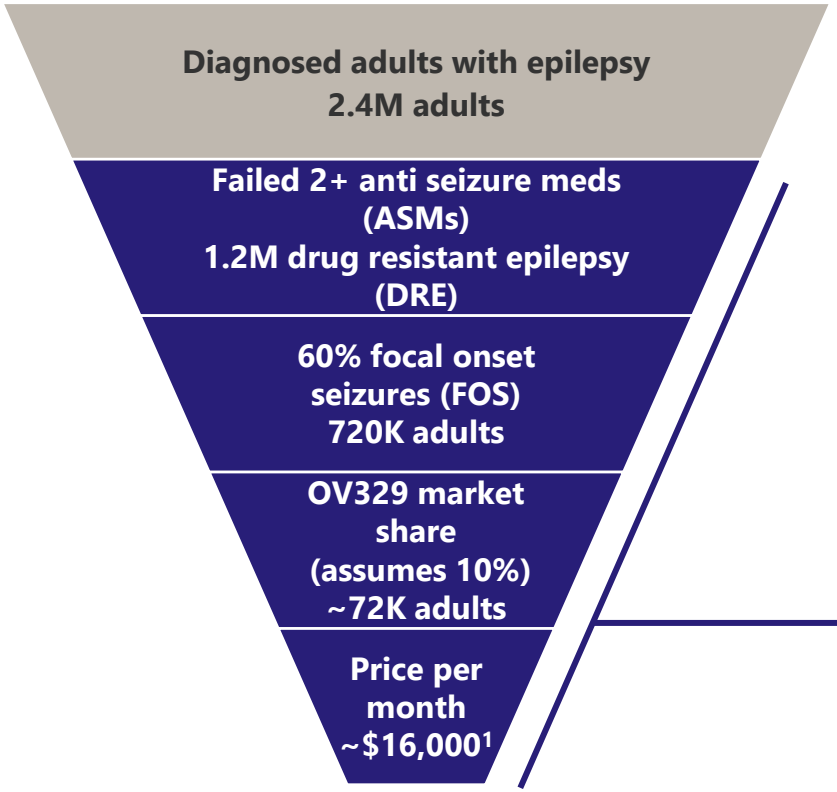
Unique MoAs are rewarded in epilepsy - even in a competitive DRE market

Substantial opportunity within the DRE segment



>$1 Billion

	Generalized seizures	Focal / Partial seizures
1L	Valproate, Lamotrigine, Topiramate, Levetiracetam, Zonisamide, Ethosuximide	Carbamazepine, Levetiracetam, Oxcarbazepine, Phenytoin, Eslicarbazepine, Lacosamide
2L	Clobazam	Clobazam, Lamotrigine, Topiramate, Rufinamide, Zonisamide
Refractory Options (~30%)	OV329 *In Phase 1*; XEN1101; Fycompa perampanel; XCOPRI; BRIVIACT (brivaracetam) C	
Non-pharm. Options	Surgical Resection; Deep Brain Stimulation; Ketogenic Diet; Vagus Nerve Stimulation; Responsive Cortical Stimulation	

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

1. Analogue price based on XCOPRI price of $1,180 WAC per 30 count bottle https://www.sklifescienceinc.com/pdf/Connecticut_Prescriber_Discloser_Form.pdf

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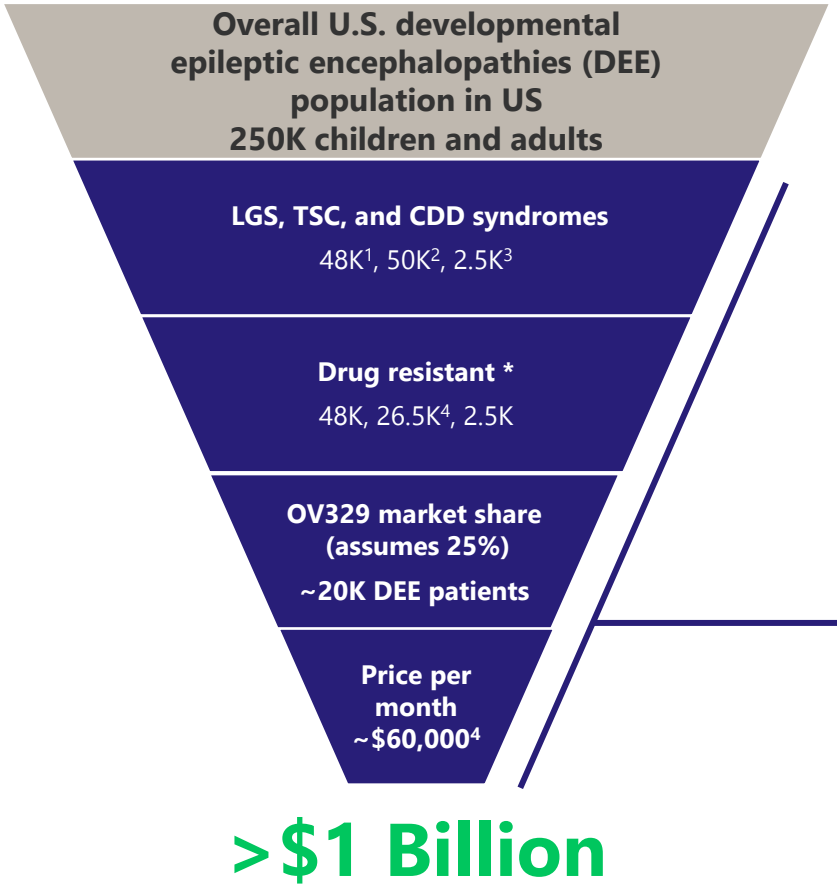
Continued unmet need across a range of rare epilepsies; limited treatment options indicated for LGS, TSC, CDD with drug resistant seizures

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

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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
1st-in-human
Q1 2025

IP through 2046 (assumes five year patent term extension)

9 method-of-use patents filed

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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

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

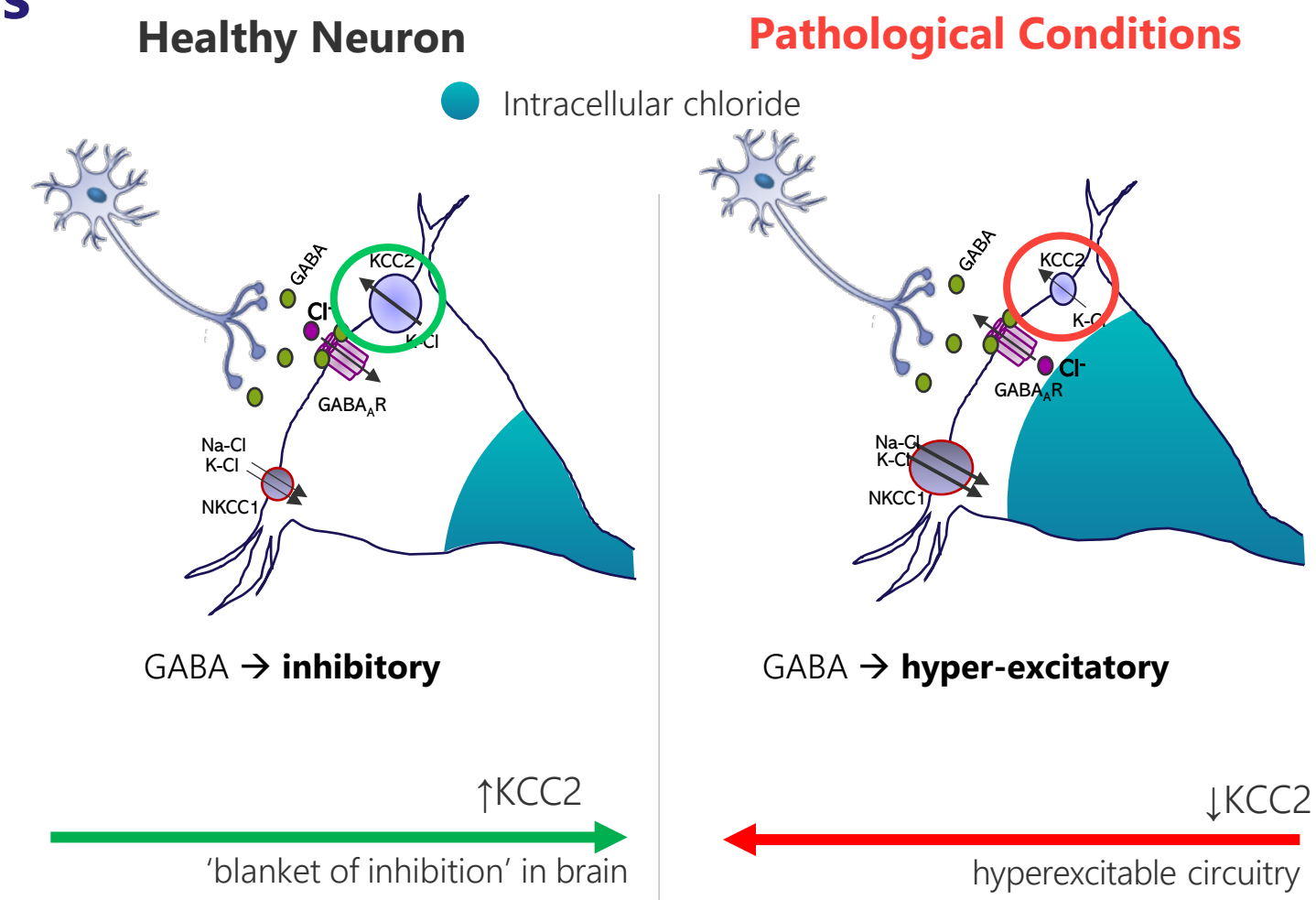
KCC2 regulates chloride gradient

- 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

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Franchise of KCC2 direct activators in development

OV350 & OV4071 behave similarly in phenotypic screens

	OV350	OV4071	OV4041
Formulation	Intravenous	Oral Intramuscular injection / SubQ	Oral Intramuscular injection
Indication	**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 (w/ qEEG biomarker) Potential proof-of-MoA in patients Q4 2026	Currently in IND-enabling Phase 1 initiation Q2 2026 Topline results Q1 2027	Initiating IND-enabling studies late 2025 IND submission late 2H 2026

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Entering human trials: 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

- ✓ Regulatory approval to initiate Phase 1
- ✓ Patient 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

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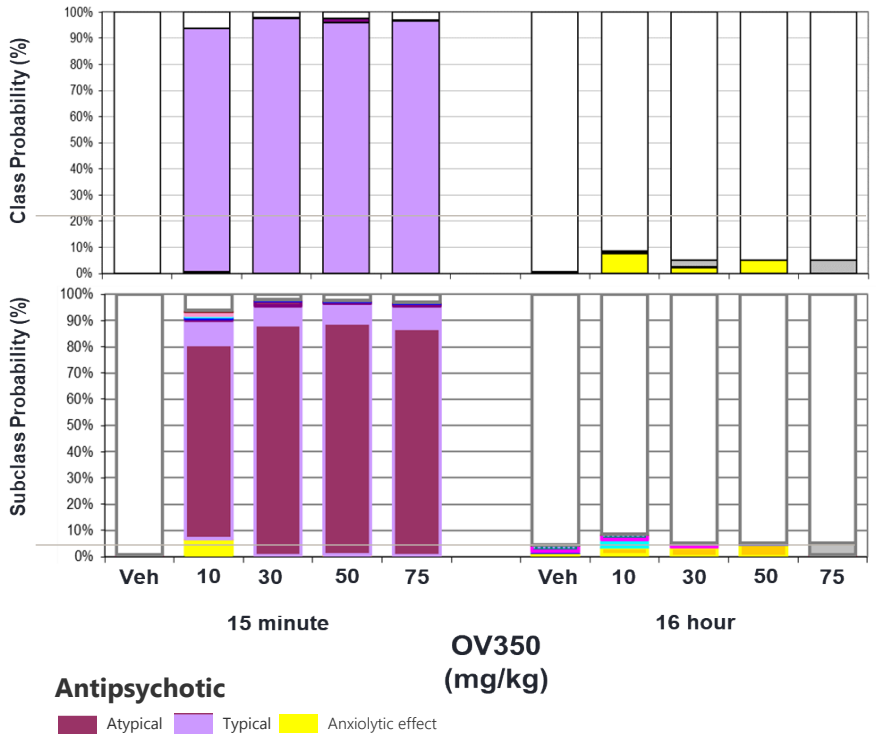
OV350 elicits potent, robust, rapid and reversible antipsychotic activities[1]

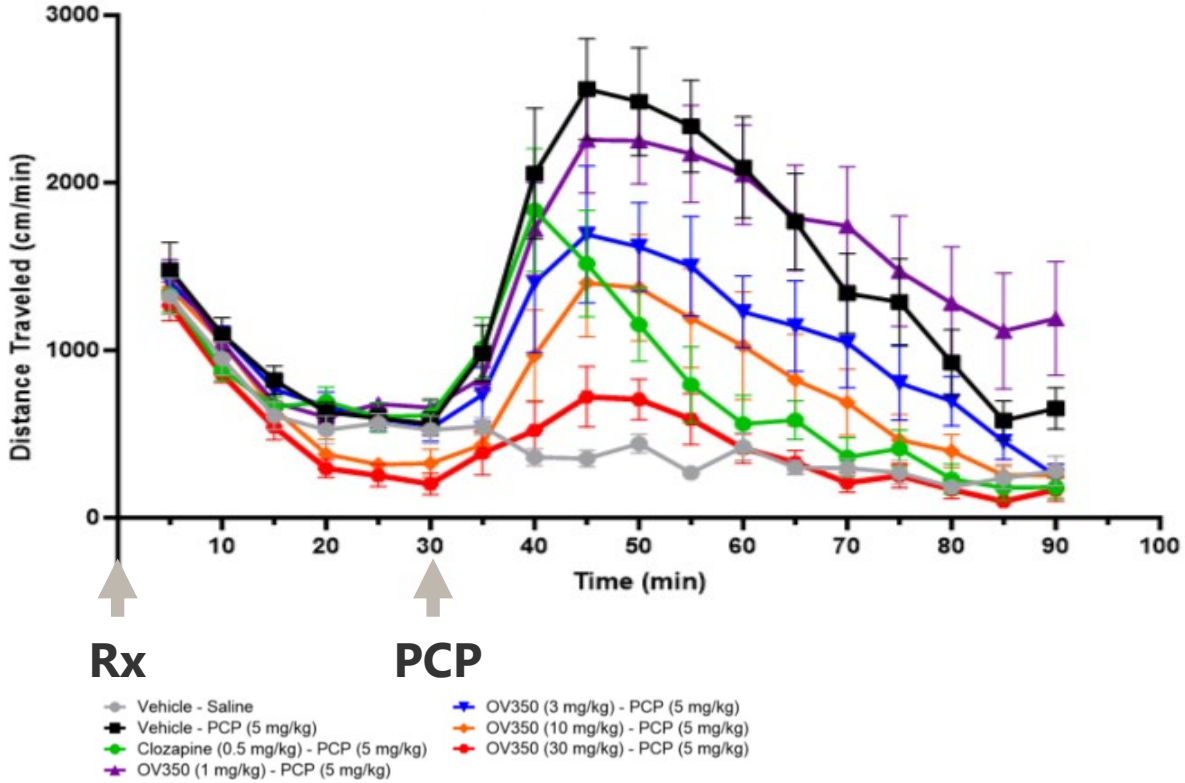
Behaves as atypical antipsychotic with a clean profile in SmartCube®

- 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



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OV350 enables rapid path to patients for oral KCC2 direct activators

- ✓ Existing regulatory path
- ✓ Phase 1 demonstrate acceptable safety (for a first-in-class approach)
- ✓ Quantitative EEG exploratory biomarker for translation
- ✓ Potential to establish proof-of-mechanism in patients

Initiate OV4071 (oral program) which behaves similarly to OV350 in phenotypic models

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Entering 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

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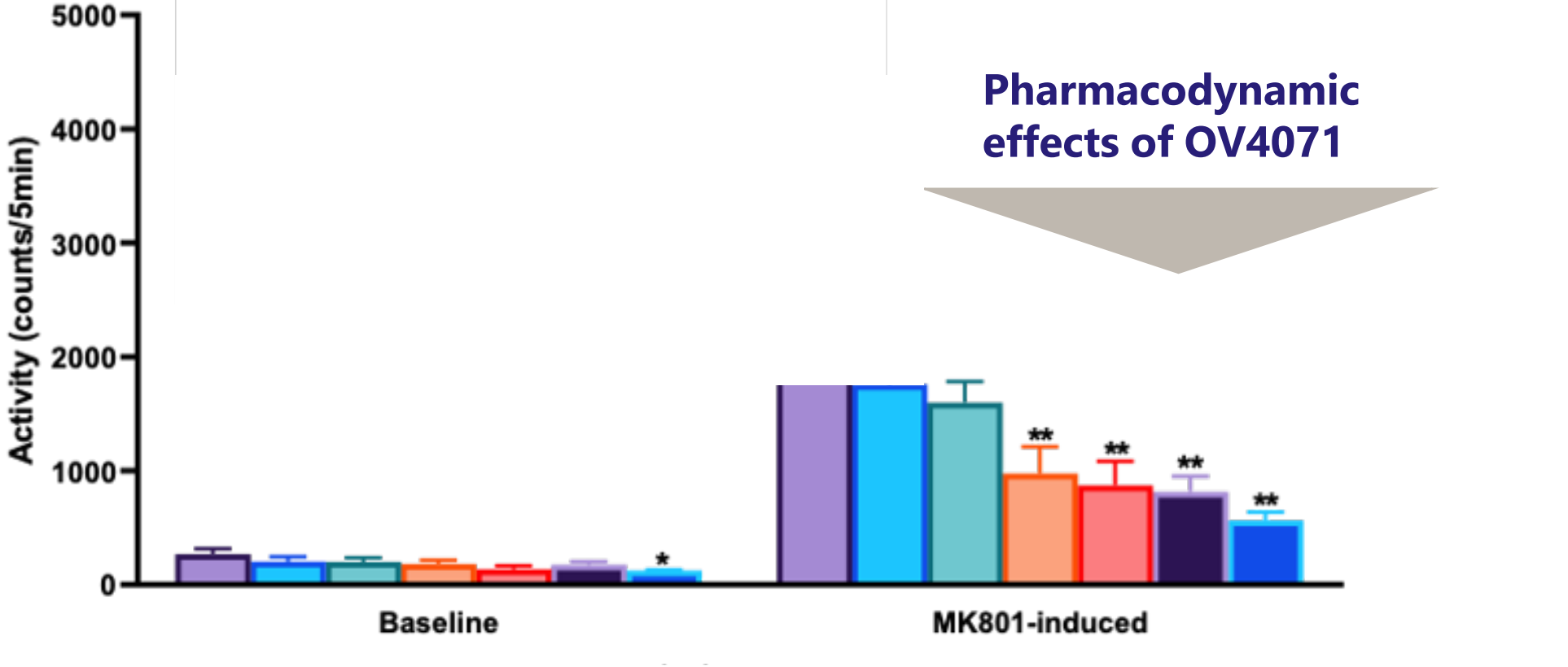
OV4071 dose dependent response in schizophrenia psychosis model

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



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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/

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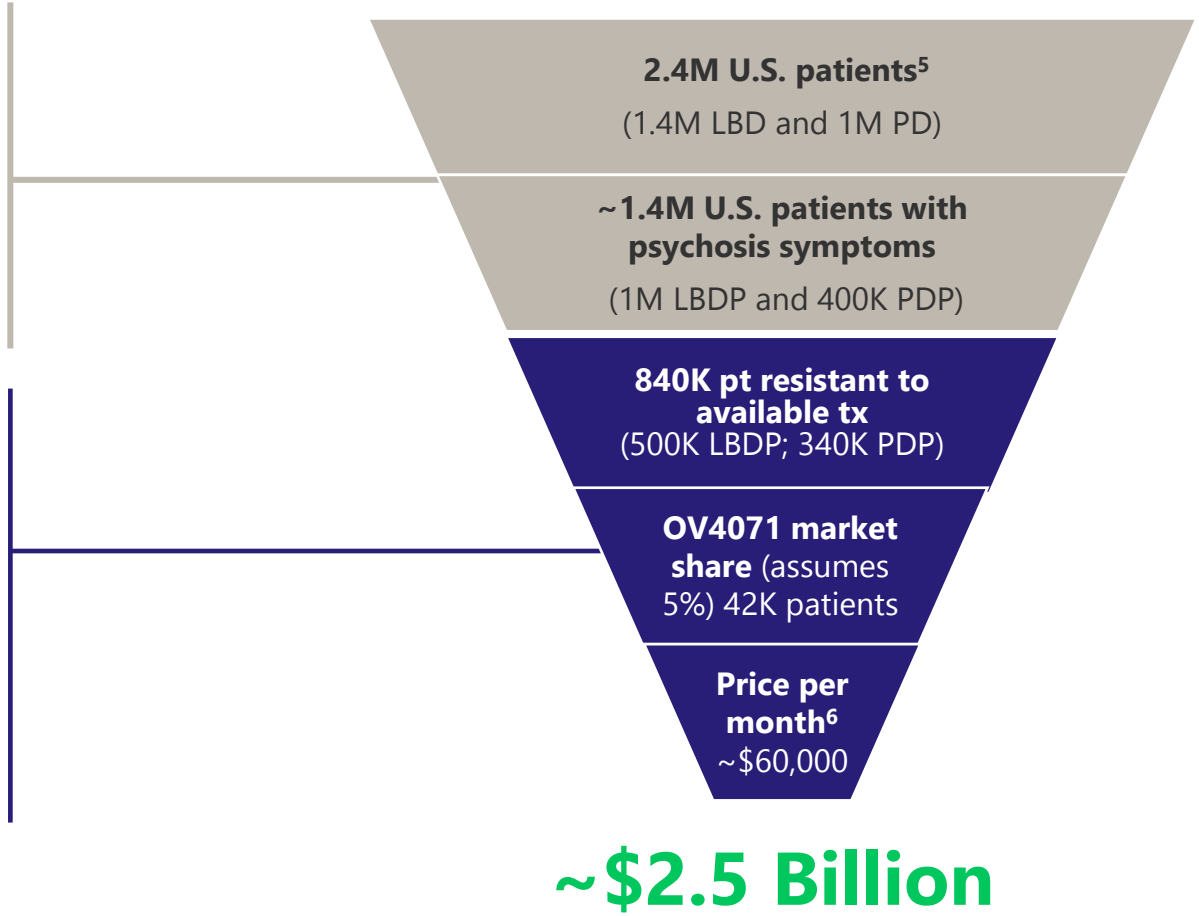
A large opportunity for OV4071: Parkinson's disease psychosis and Lewy body disease psychosis

- 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)

- Topline data anticipated Q3 2025 (biomarkers for clinical effect and target engagement, safety & tolerability)

Multiple, unique KCC2 programs enable the potential for optionality with co-development, transactions and relative to the Inflation Reduction Act

- 1st KCC2 direct activator entered human trials Q1 2025
- Oral KCC2 direct activator entering humans Q1 2026

Cash on hand as of December 31, 2024 to fund operations into the second half of 2026

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