



Corporate Presentation

January 12, 2026

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; 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 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 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).

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Medicines to quell conditions caused by neural hyperexcitability

Fundamental 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

expected to fund multiple value-driving catalysts

Focused high-value pipeline with differentiated mechanisms of action

Programs	Indication opportunities	Preclinical	Phase 1	Phase 2	Anticipated milestones
GABA-aminotransferase 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				Established human safety, PK, tolerability and potential electrophysiology biomarker (qEEG)
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 (Initiationmid-2026) Patient Phase 1b initiations (Q3 2026) Topline PoC results in PDP/LBD (mid-2027)
OV4041 oral	Generalized anxiety disorder and Rett syndrome				Initiating IND-enabling late 2025 IND submission late H2 2026

*Ovid refocused resources on chronic oral formulations, including OV4071 and OV4041, and will not advance OV350 IV further in the clinic

Timelines pending feedback from regulatory discussions Ketamine challenge to initiate pending PK characterization in Phase 1 study
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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

Biomarker validated; advancing to Phase 2

In active discourse with regulators; expect to initiate Phase 2 in Q2 2026

IP through 2041

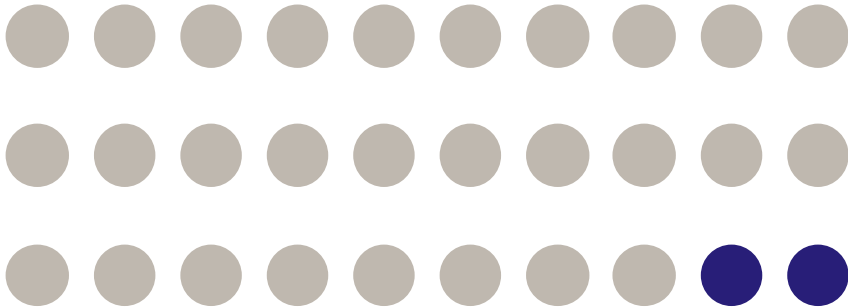
(composition of matter; assumes five-year patent term extension)
10 patent families

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

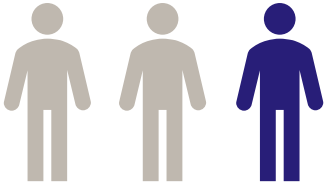
New mechanisms of action are needed in epilepsy

Drug resistant epilepsy (DREs)

30 anti-seizure medications (ASM) approved in 15 years, but only 2 with novel mechanisms¹



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 average²

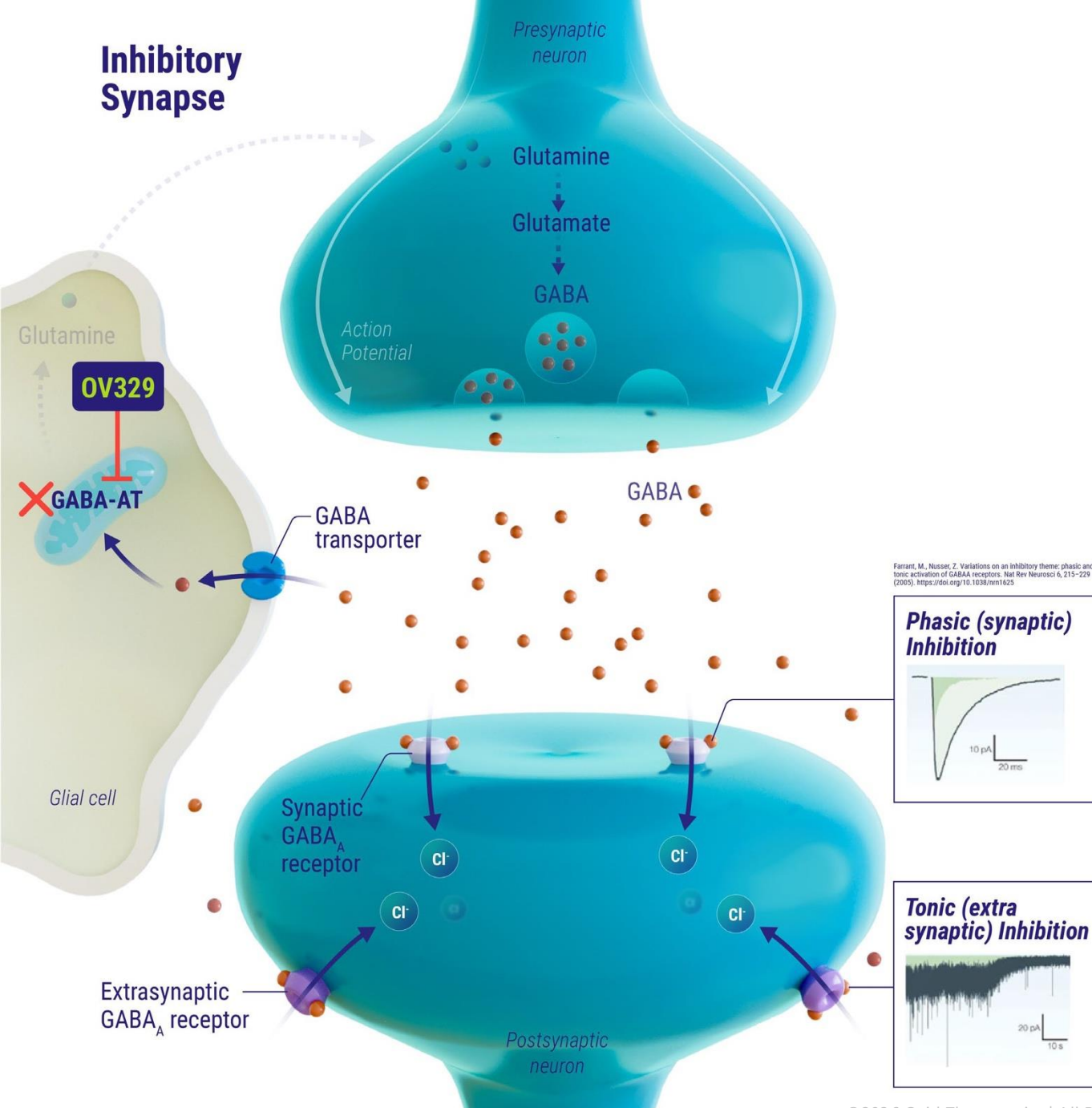


1. Each dot = 1 FDA-approved ASM; Purple dots are Fycompa and Cenobamate

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



OV329 optimally tunes GABA; potentially enhancing tolerability and efficacy

Inhibits GABA-AT, the enzyme that degrades the inhibitory neurotransmitter, GABA¹

Increases GABA in the synapse & extra-synaptic regions delivering phasic & tonic inhibition²

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 to treat a broad population of epilepsies with 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

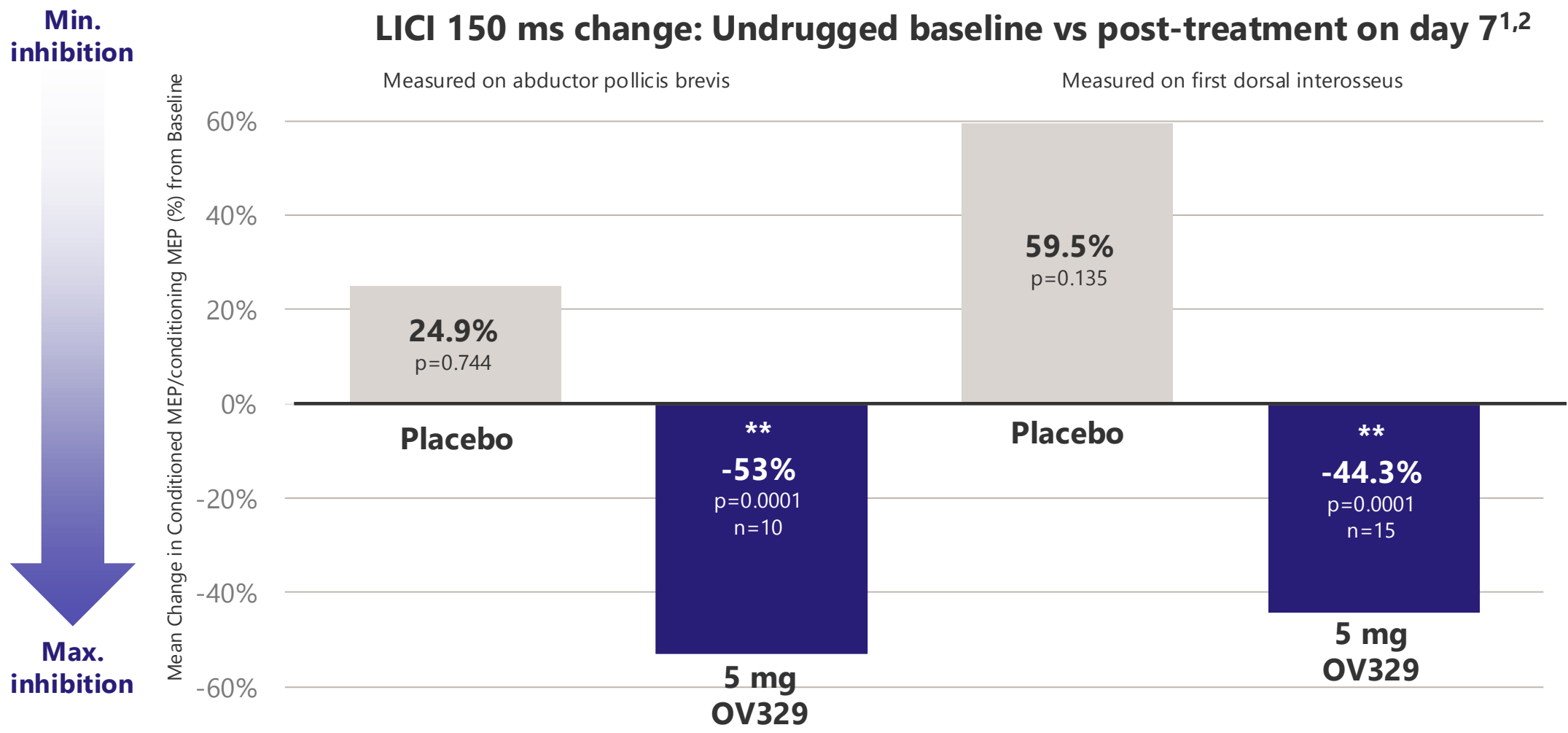
1. Pierantozzi M et al. Effect of Vigabatrin on motor responses to transcranial magnetic stimulation: an effective tool to investigate in vivo GABAergic cortical inhibition in humans. Brain Research, Nov. 26, 2004. In this study, vigabatrin was dosed at 50mg/kg. in keeping with the recommended therapeutic 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%

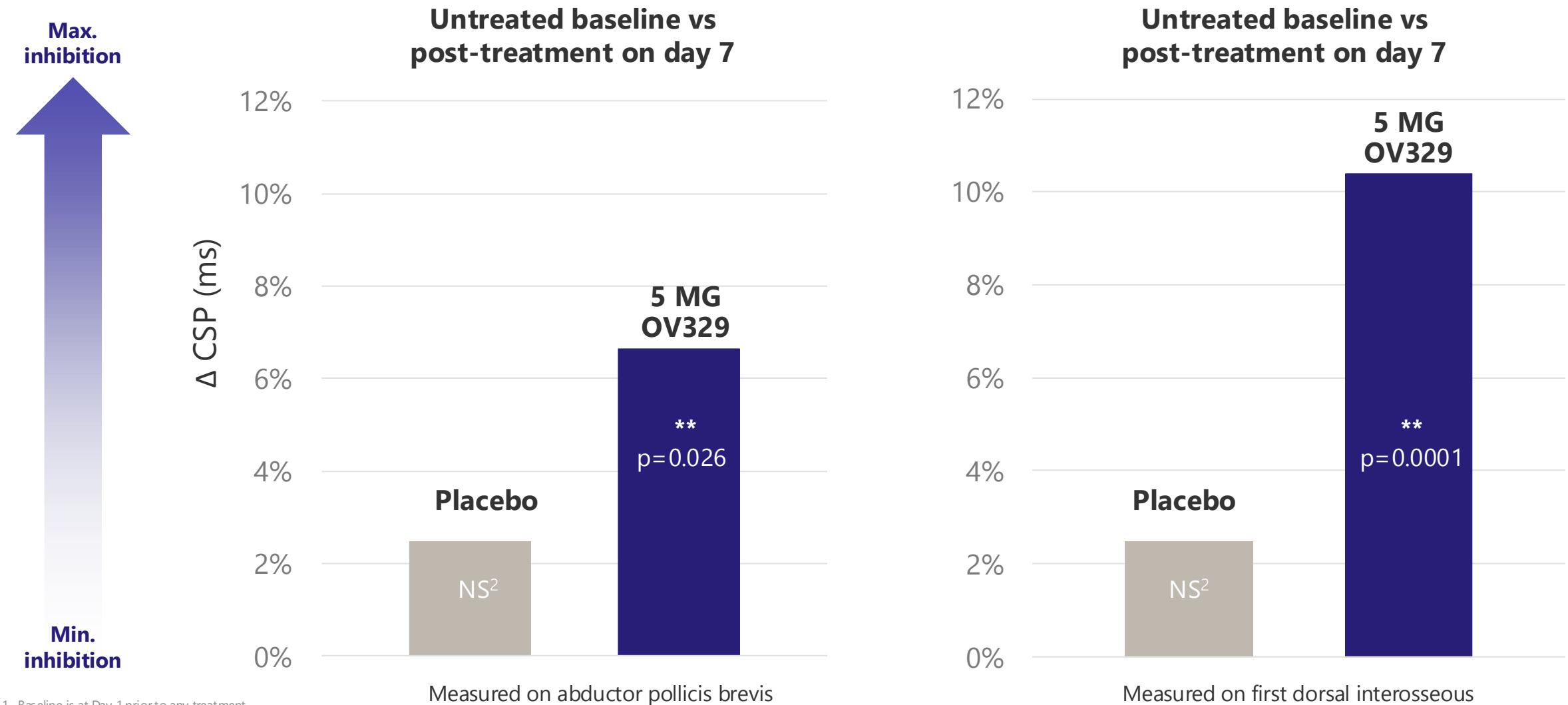
OV329 demonstrated statistically significant increases in cortical inhibition exceeding vigabatrin's inhibition



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



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

OV329 was well-tolerated with no treatment-related SAEs¹

Phase 1 summary of potentially treatment-related and treatment-emergent AEs¹

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*

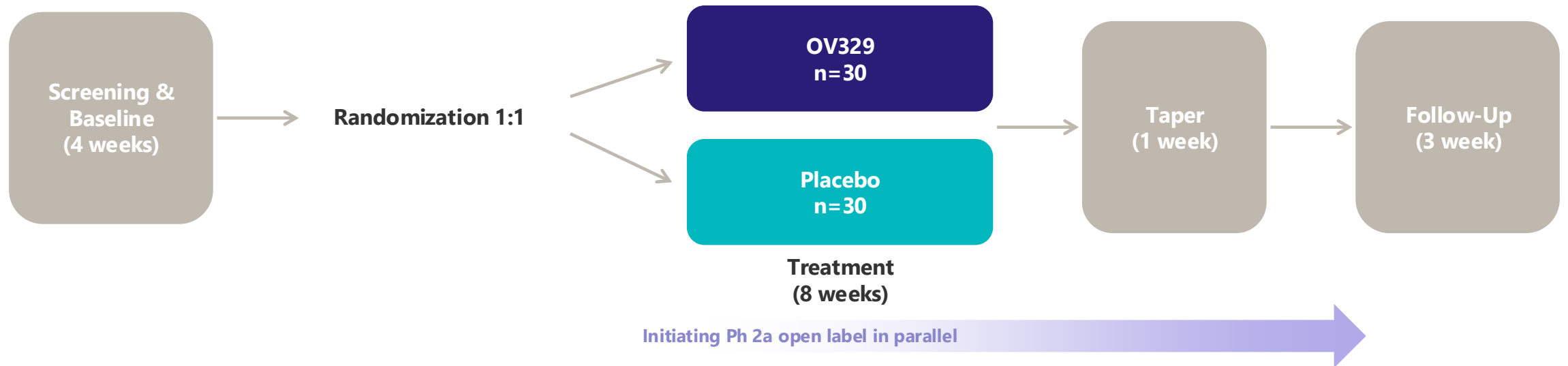
- Extensive ophthalmic testing closely monitored for clinical and structural (sub-clinical) changes
- No treatment related effects were noted in any of the metrics tested

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

OV329 Phase 2 study design

Randomized, placebo-controlled trial in adult patients with treatment-resistant focal epilepsy



Trial design

- Global multi-site study
- Patients 18 - 55 years of age
 - Treatment-resistant focal aware or awareness impaired motor onset seizures
 - Stable concomitant anti-seizure medications
 - 4 or more countable seizures per 28 days in screening

Key Endpoints

Safety: Incidence and severity of treatment-emergent adverse events (TEAEs)

Efficacy:

- Change from baseline in monthly seizure frequency
- Responder rate
- Time to pre-randomized monthly baseline seizure frequency
- Clinical Global Impression of Improvement (CGI-I)

Anticipated clinical & regulatory next steps

Regulatory

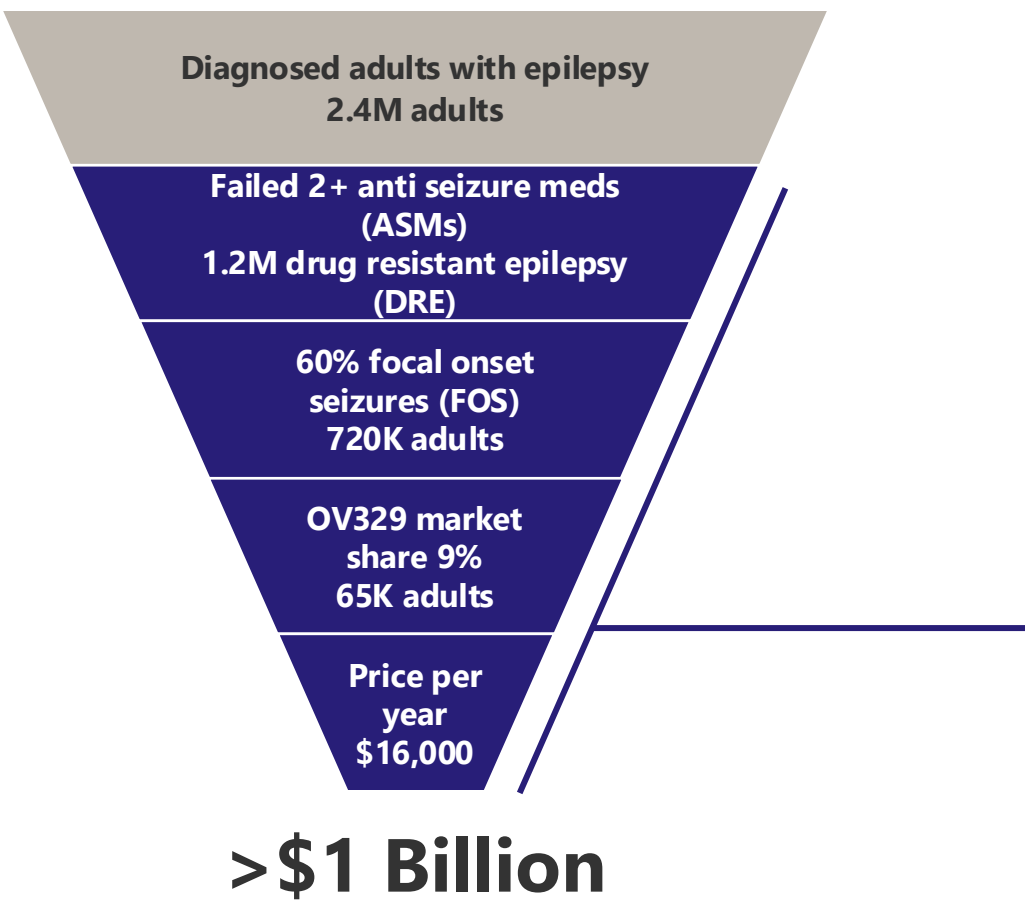
- Engaging in pre-Phase 2 meetings and scientific advice with global regulatory bodies

Clinical

- Anticipate initiating Phase 2 randomized placebo controlled study in Q2 2026 – to establish safety and seizure reduction effect; responder rate
- Concurrently, initiating open-label seizure effect study (to inform dosing and pivotal trial powering)
- Seek to correlate biomarker and electrophysiology to clinical responses

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

Substantial opportunity within the DRE segment



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 Phase 2 Planned	XEN1101	XCOPRI
		Fycompa [®] perampanel	BRIVIACT [®] (brivaracetam)C
Reimbursement & coverage dynamics remain favorable. Polypharmacy well covered			

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

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 showed good safety and predictable pharmacokinetics

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

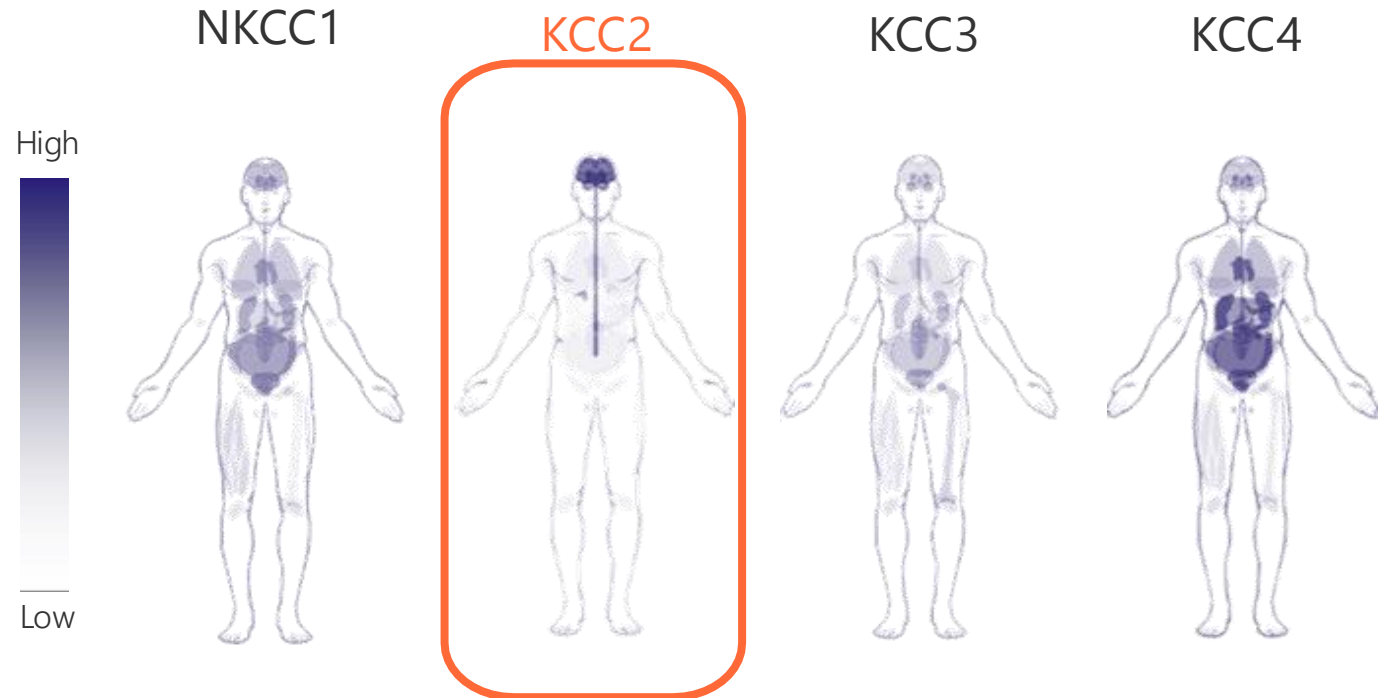
“Master switch” for neurological disorders

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>

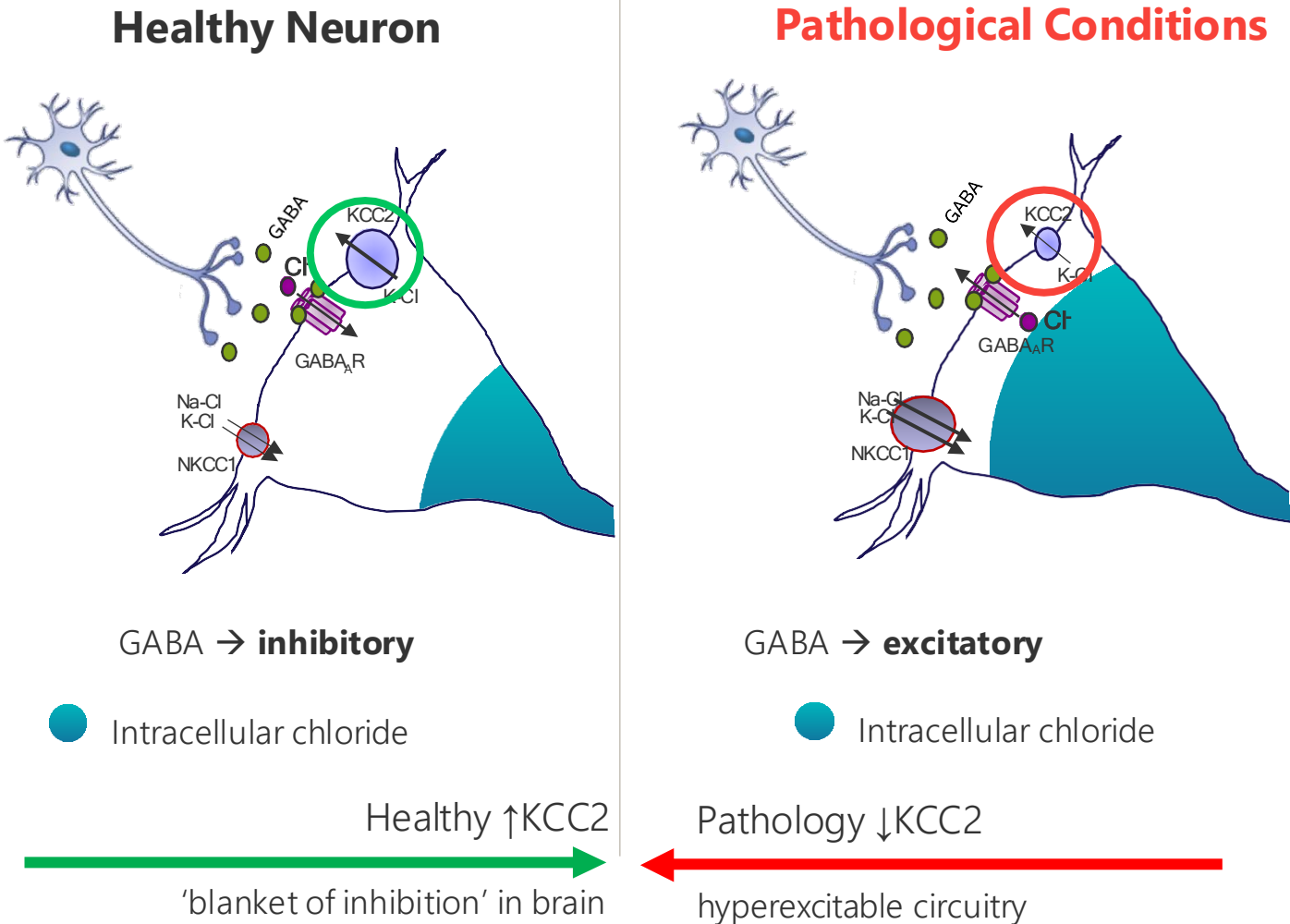
KCC2 is the main regulator of GABA inhibition by maintaining neuronal chloride homeostasis

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’



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

Avoids tolerability & safety issues that have been associated with other potassium chloride transporters

Prevents unintended modulation of off-target signaling cascades

Maintain physiological limits on hyperpolarization

All Ovid molecules confirmed as direct activators of KCC2

The potential of KCC2 direct activation

**A fulcrum in neural
excitation/inhibition;
fundamental
biological target**

**Highly selective, precision
small molecules**

**Expansive therapeutic
applicability**

**Preferable tolerability
& safety (no sedation)**

**Potential for oral, injectable
and IV administration**

**Only confirmed
direct activators**

**Replenishing portfolio of unique molecules
to fuel a CNS franchise in disorders with great unmet need**

Ovid development-stage portfolio

Stage	OV350 (tool program)	OV4071	OV4041	OV5000 & OV6000 Series
	Phase 1 completed	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	• Clean safety results, with no treatment related SAEs • Predictable pharmacokinetics • Exploratory qEEG results supportive of central activity • Supports and informs oral portfolio	• 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

Disease model activity

Prominent antipsychotic activity

Anticonvulsant activity

Neuroprotective & anti-inflammatory signals

OV350
(IV tool program)

Safety

No evidence of sedation

Supportive safety package

No treatment related serious adverse events in Phase 1 trial

OV350 IV showed a good safety profile in Phase 1

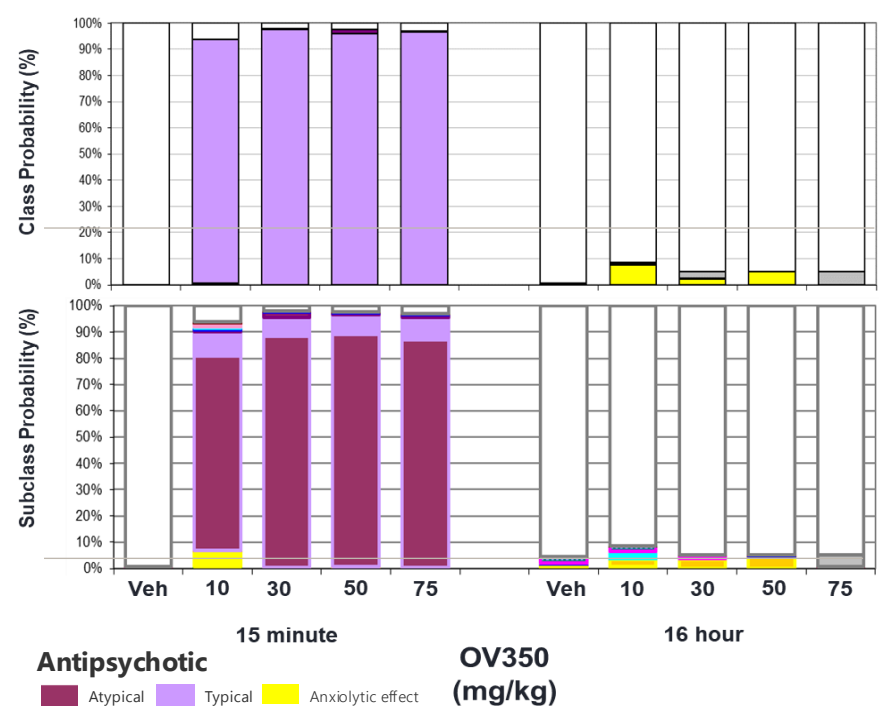
- ✓ Evaluated as tool program in an exploratory randomized, placebo-controlled, single-ascending dose study
 - 16 healthy participants (6=active, 2=placebo cohort)
 - Doses 50 mg and 100 mg administered by IV infusion
- ✓ Showed good safety profile, with no treatment-related laboratory findings, no safety findings, and no treatment-related serious adverse events (SAEs)
- ✓ Pharmacokinetics were as predicted
- ✓ Exploratory qEEG results suggest:
 - Central activity
 - Spectral power consistent with expected physiological effects of KCC2 modulation
 - Activity aligned with expected drug exposure in the brain

Results support advancement of the Company's KCC2 portfolio, including the first oral direct activator, OV4071, which is on track for Phase 1/1b regulatory submission expected in Q1 2026

OV350 elicits potent, robust, rapid and reversible antipsychotic activities

Profile similar to atypical antipsychotic in SmartCube® screen

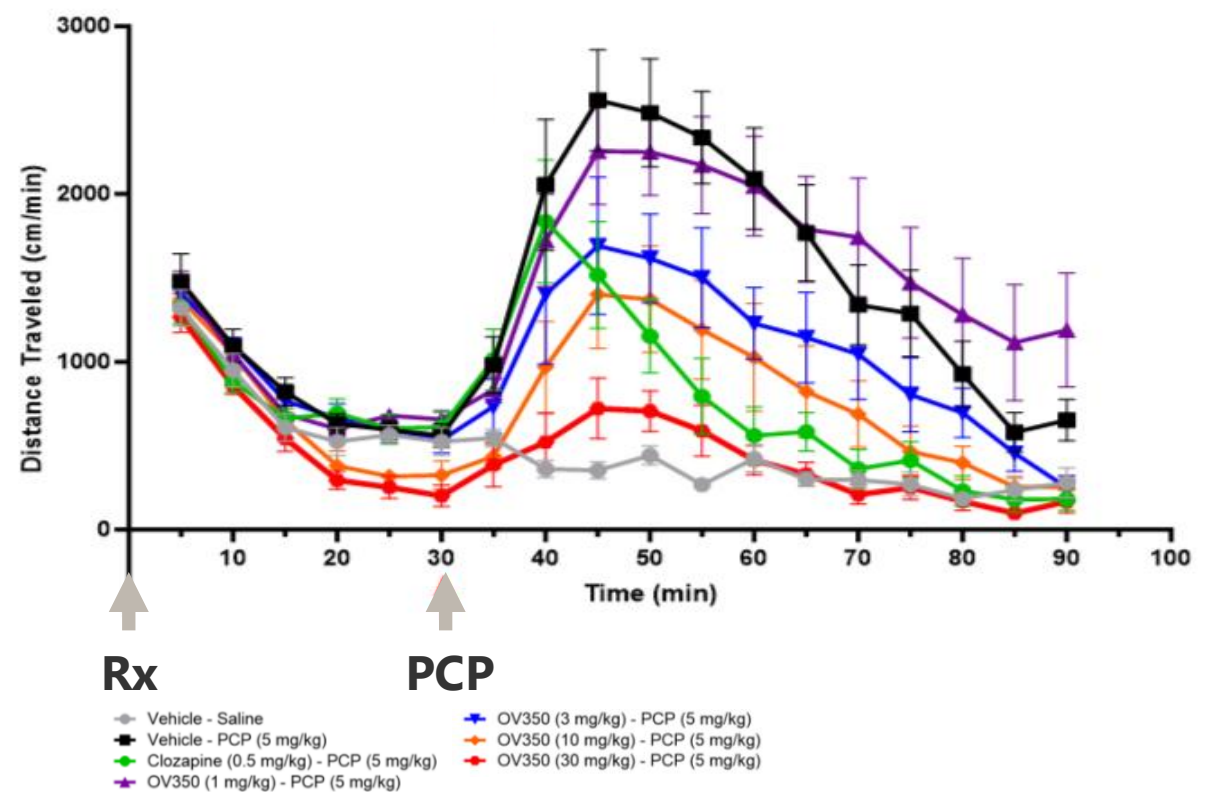
- Onset of effect within 15 minutes
- Dose response starting at 10 mg/kg; disappears in 16 hours
- No sedation or other adverse behavior effects observed up to 75 mg/kg
- Minor anxiolytic effects observed (yellow)



Ovid data on file

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



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)

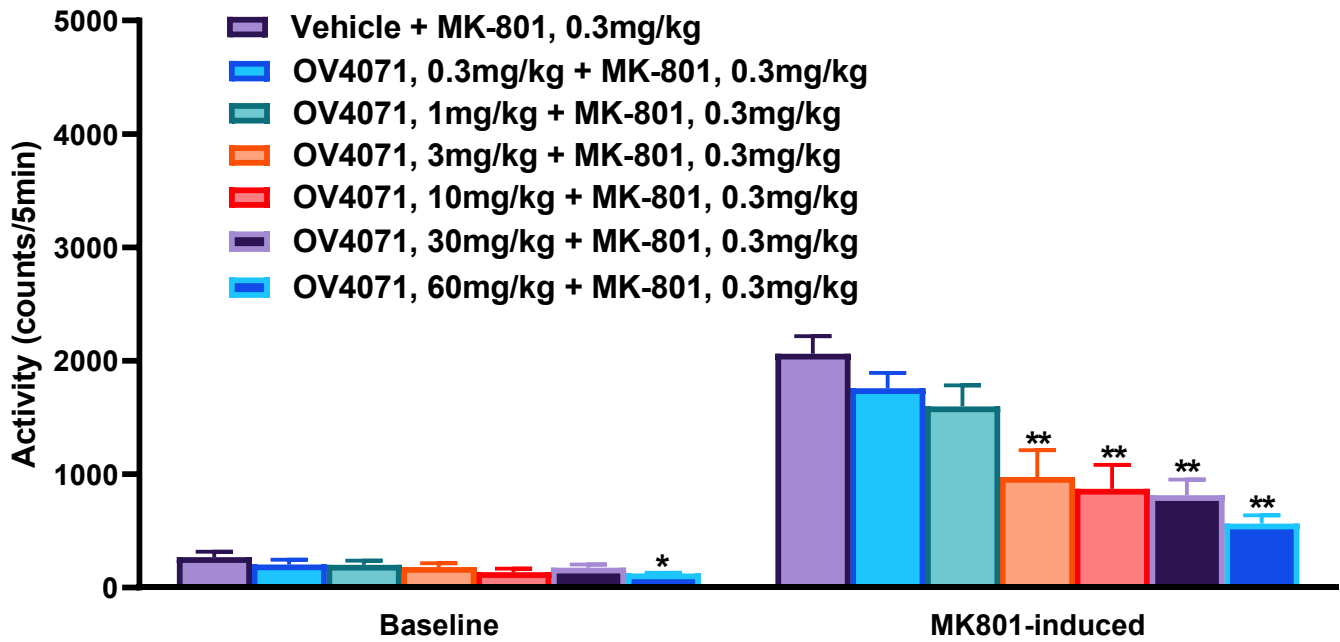
Clinical Plan

- 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

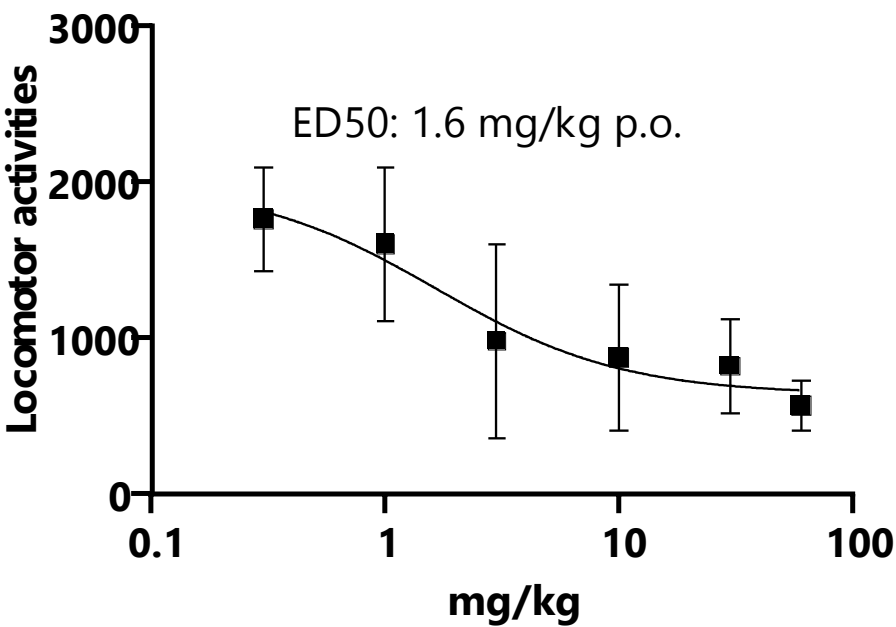
OV4071: IND-enabling progress

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

MK801 schizophrenia psychosis model confirmed OV4071 robust, dose dependent signal



Dose-dependent pharmacodynamic effects of OV4071



Mechanistic relevance in initial psychiatric indications

Neurodegenerative psychoses

- Visual hallucinations, especially in conditions like Parkinson's disease (PD) and schizophrenia, are linked to disruptions in the brain's GABAergic system, particularly in the visual cortex.
- Reduced GABAergic activity, or changes in GABAergic interneuron populations, can lead to imbalances in neural activity, potentially contributing to the misinterpretation of sensory input and the manifestation of visual hallucinations.
- In PD psychosis, research suggests that reduced GABA levels or altered GABAergic interneuron function in the visual cortex may be a key factor in the genesis of visual hallucinations
- GABA plays a crucial role in regulating neuronal activity.
- KCC2 activation enhances neurons' response to GABA and restores E/I balance in the visual cortex, and may provide a promising therapeutic option.

Schizophrenia

- Increasing evidence suggests a contributor to executive dysfunction in schizophrenia is reduced synaptic inhibition in principal cells of the pre-frontal cortex
- Several schizophrenia models have shown deficits in KCC2 in the pre-frontal cortex and were associated in changes with GABA reversal potential
- OV350, a KCC2 direct activator has been shown to rescue network and behavioral deficits in diseased animal models.
- Specifically, KCC2 direct activation improved:
 - Behavioral rescue in 22q11 DS deletion mice
 - Working memory in methamphetamine model
- KCC2 has demonstrated a profile comparable to atypical antipsychotics without sedation

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

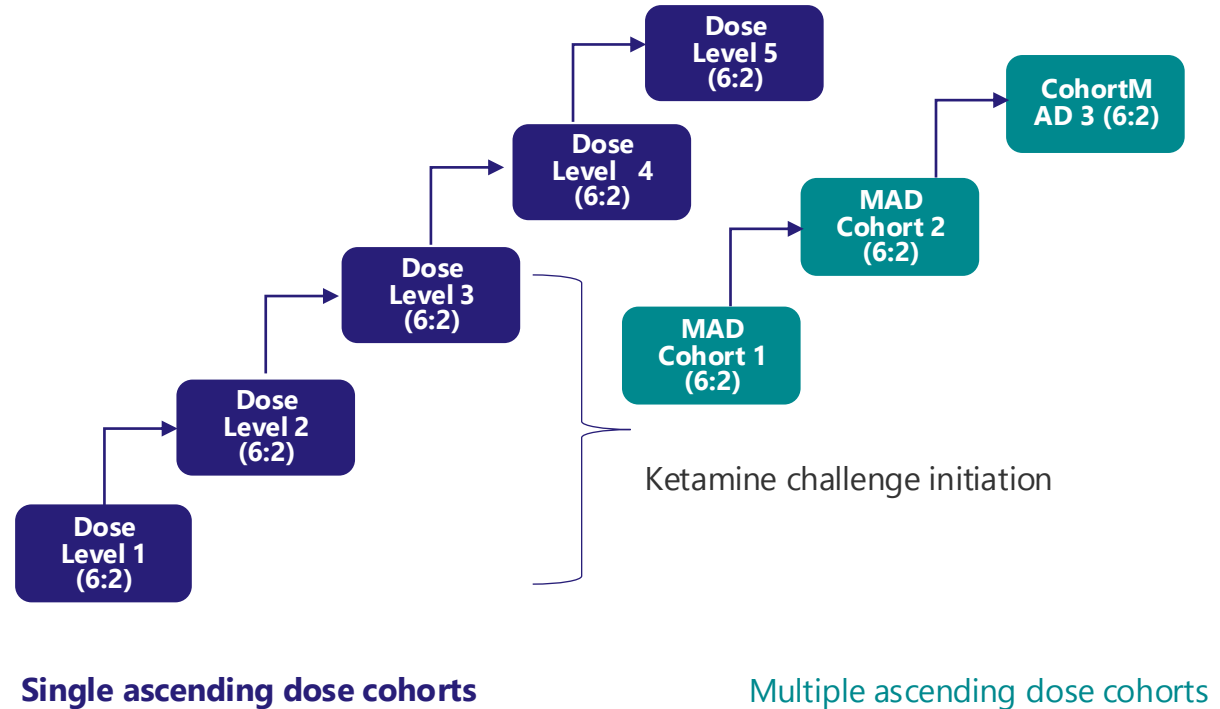
Strategy for 1st indication

- ✓ 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;

Advancing the first oral KCC2 direct activator into the clinic

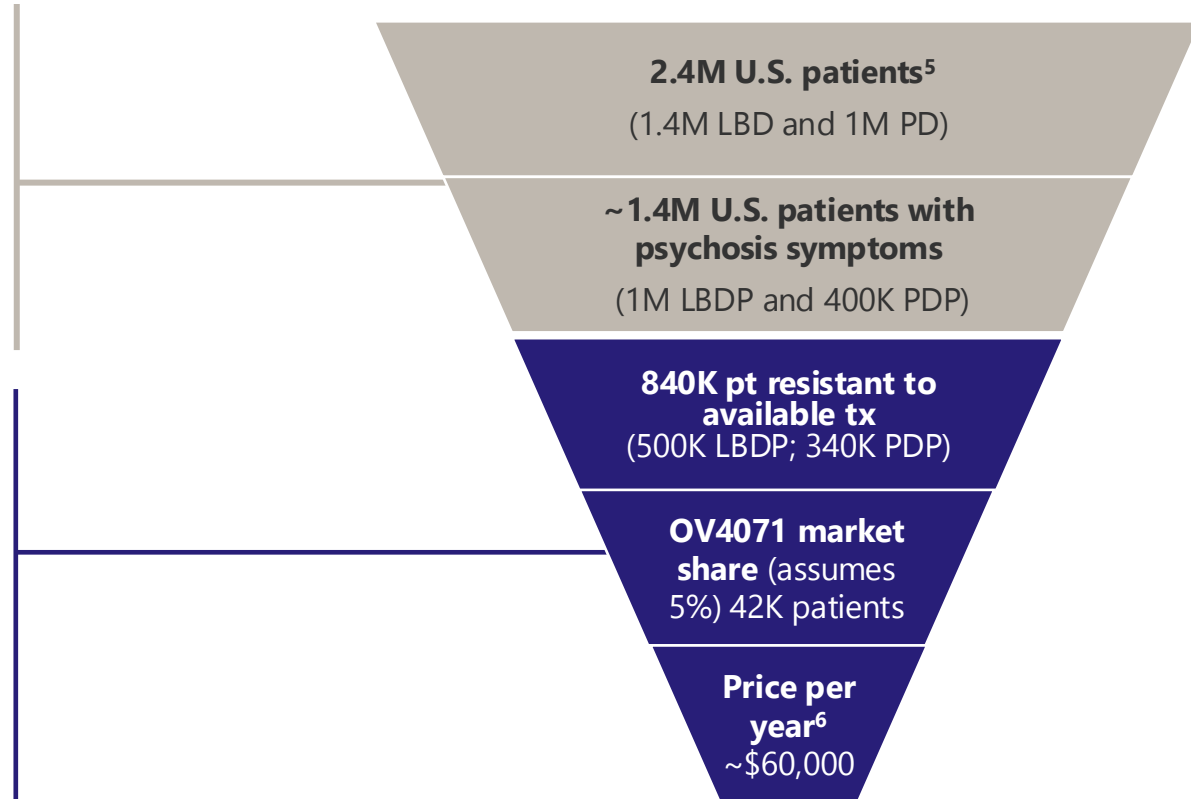
OV4071 Phase 1 SAD/MAD study design

- Healthy volunteer SAD/MAD
- Endpoints :
 - Safety and tolerability
 - Pharmacokinetic profile
 - Pharmacodynamic exploratory biomarkers including quantitative EEG, event-related potentials (ERPs) and saccadic eye movements (SEMs)
- Ketamine challenge cohort in parallel
 - After sufficient characterization of PK



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/ contraindicated³
- Use of Nuplazid® (the SoC) is limited due to limited efficacy and a Boxed Warning⁴
- 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. Parkinson Foundation: <https://www.parkinson.org/understanding-parkinsons/non-movement-symptoms/hallucinations-delusions>

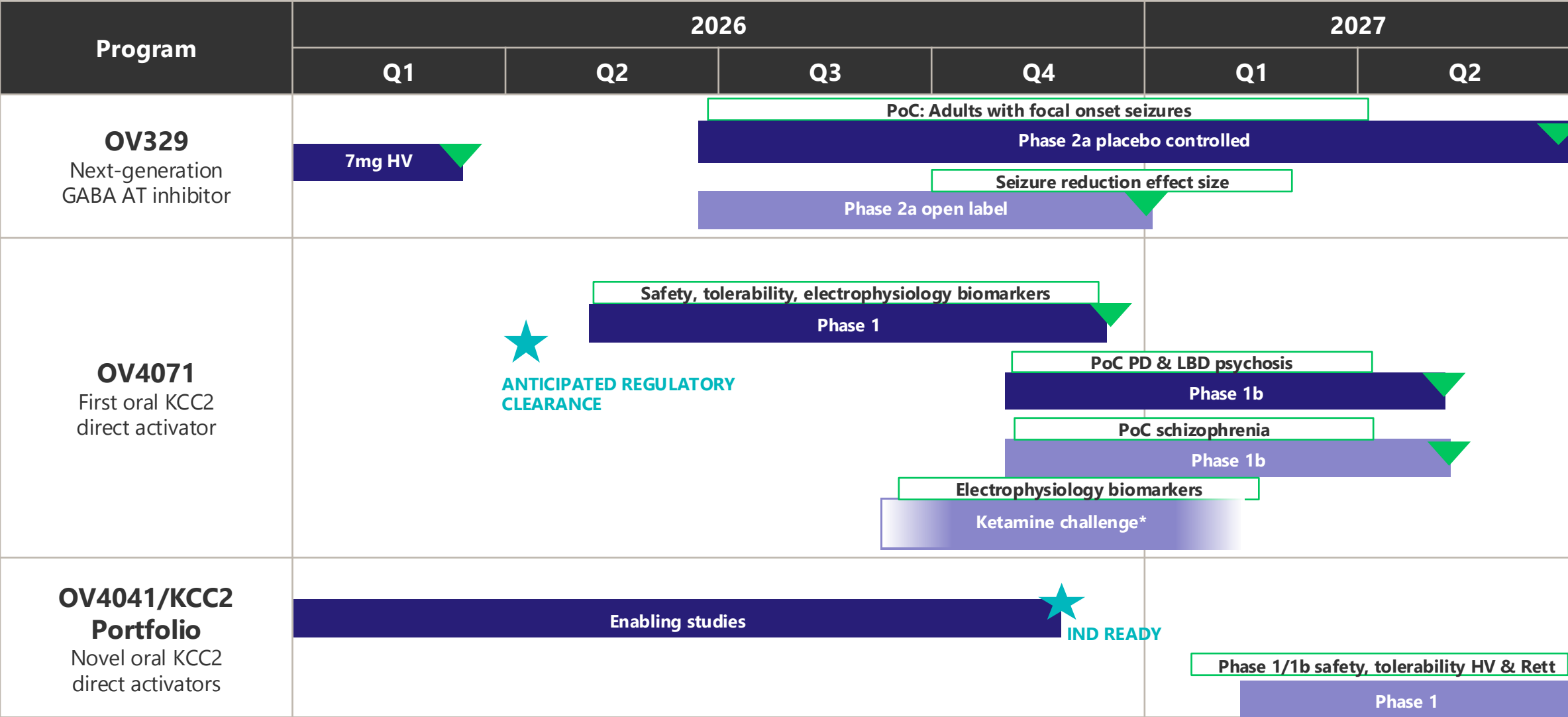
3. Lewy Body Dementia Association: <https://www.lbda.org/emergency-room-treatment-of-psychosis/> and <https://www.lbda.org/treatment-options/> Nuplazid efficacy: https://www.nuplazidhcp.com/efficacy-data#67uQuSEz_31CdL3A5qcY1b

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>

A catalyst-rich pipeline



Timelines pending feedback from regulatory discussions
*Ketamine challenge to initiate pending PK characterization in Phase 1 study



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