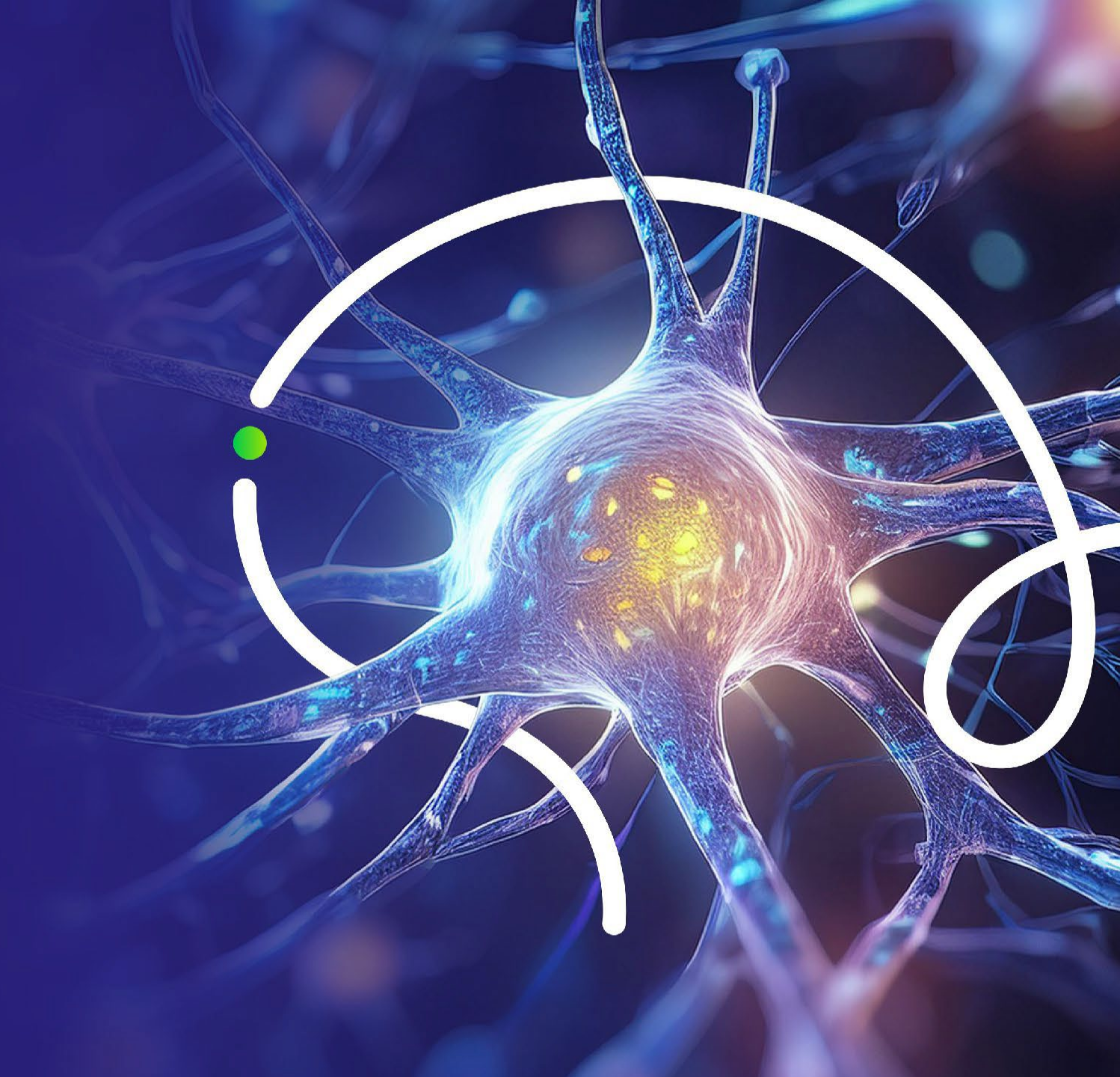
Ovid Therapeutics

OV329 Phase 1 Topline Results

October 3, 2025



Forward-looking statements

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

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

Agenda

Overview

Jeremy Levin DPhil, MB BChir, Chairman & Chief Executive Officer

OV329: Topline biomarker and safety results

Meg Alexander President & Chief Operating Officer

Questions and answers

Jeremy Levin DPhil, MB BChir, Chairman & Chief Executive Officer
Meg Alexander President & Chief Operating Officer
Jeffrey Rona Chief Business & Financial Officer
Zhong Zhong PhD, Chief Scientific Officer

Jeremy Levin, DPhil, MB BChir

Chairman & Chief Executive Officer

Overview

TOPLINE PHASE 1

Biomarker & safety results

Questions & answers

OUR FOCUS

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

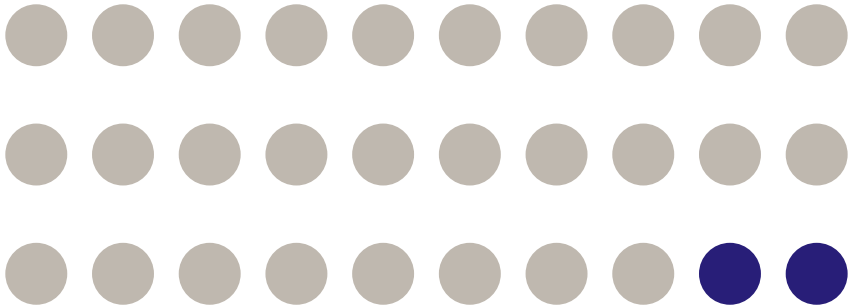
Focused high-value pipeline with differentiated mechanisms of action

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				Phase 1 topline findings (Q4 2025) To include safety, tolerability and PK results
OV4071 oral	Psychosis assoc. with Parkinson's disease and Lewy body dementia and schizophrenia				Regulatory submission & clearance (Q1 2026) Phase 1 initiation (Q2 2026) Proof-of-mechanism ketamine study (mid-2026) Patient Phase 1b initiations (mid 2026) Topline PoC results in PDP/LBD and SCZ (Q1 2027)
OV4041 oral	Generalized anxiety disorder and Rett syndrome				Initiating IND-enabling late 2025 IND submission late H2 2026

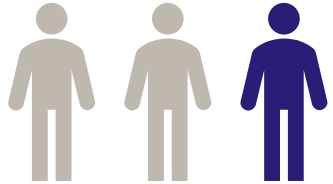
New mechanisms of action are needed in epilepsy

Drug resistant epilepsy (DREs)

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



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



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

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

Challenges of current GABA-modulating medicines

Safety

1st-generation GABA-AT inhibitor, vigabatrin, had a poor safety profile

- No therapeutic index
- Irreversible retinal changes & vision loss in some patients
- Copious dosing (2 – 3 grams) to achieve anti-convulsant effects

Poor tolerability

Surging GABA in the synapse creates tolerability challenges

- Sedation
- Dizziness

Durability of effect

Short-acting

- Some benzodiazepines have limited PD duration

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

Meg Alexander
President & Chief Operating Officer

Overview

TOPLINE PHASE 1
Biomarker & safety results

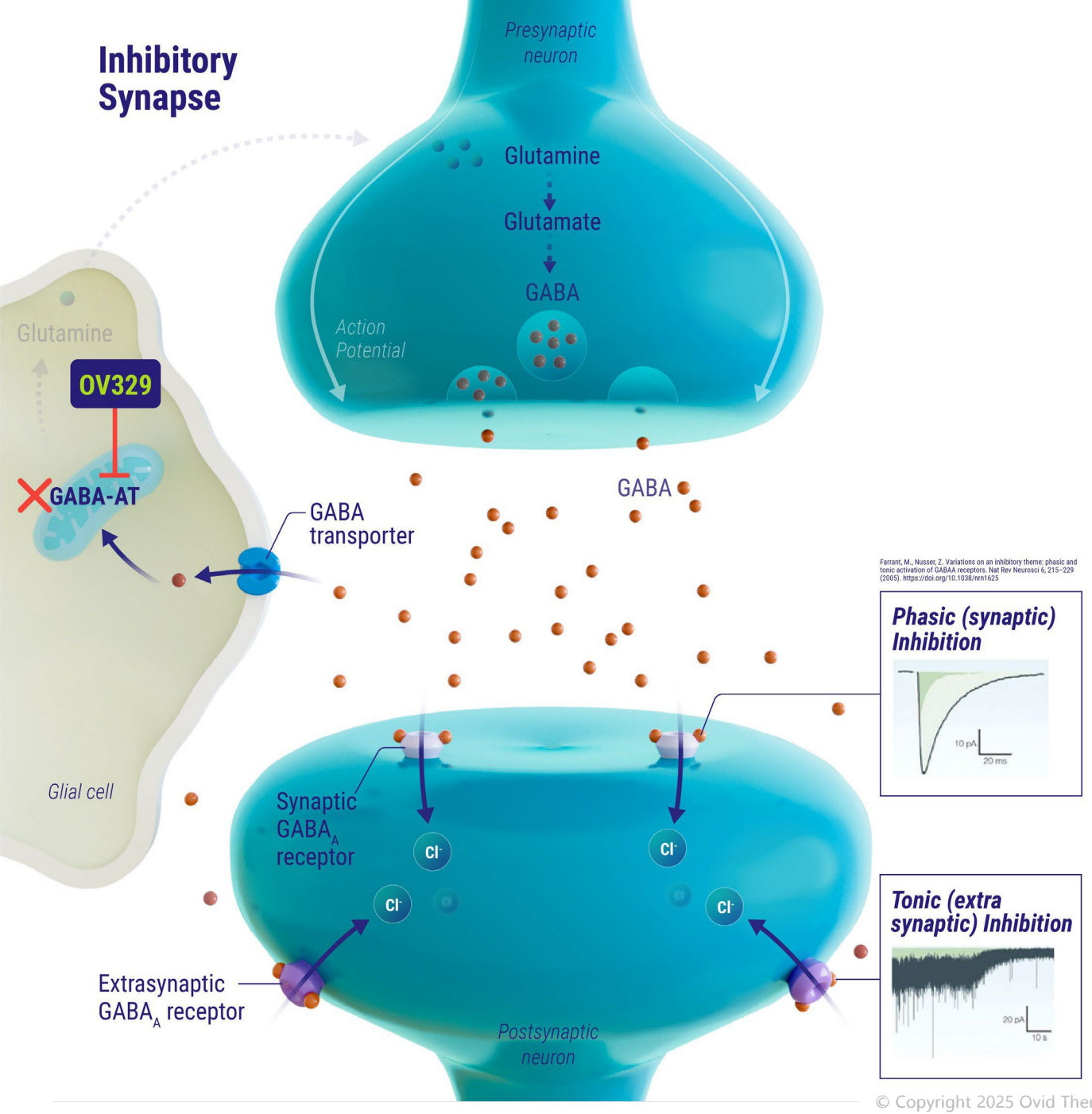
Questions & answers

Results overview

- 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)

Supports rapidly advancing into a Phase 2a trial

1. FDA Medical Review(s) for Sabril (vigabatrin) 2009. US FDA. Accessdata.fda.gov



OV329 optimally tunes GABA; potentially enhancing tolerability and efficacy

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

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

Creates an inhibitory neural environment that averts tolerability issues

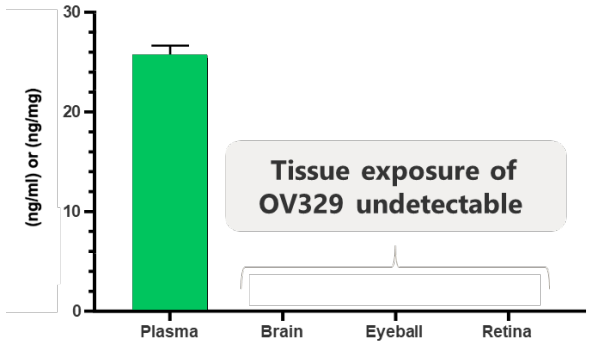
1. Silverman RB. Chem Rev. 2018 Apr 11;118(7):4037-4070. doi: 10.1021/acs.chemrev.8b00009. Epub 2018 Mar 23. PMID: 29569907; PMCID: PMC8459698
2. Colmers P et al. eNeuro 27 June 2024, 11 (7) ENEURO.0137-24.2024; https://doi.org/10.1523/ENEURO.0137-24.2024

Extensive preclinical characterization supports efficacy & ophthalmic safety

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

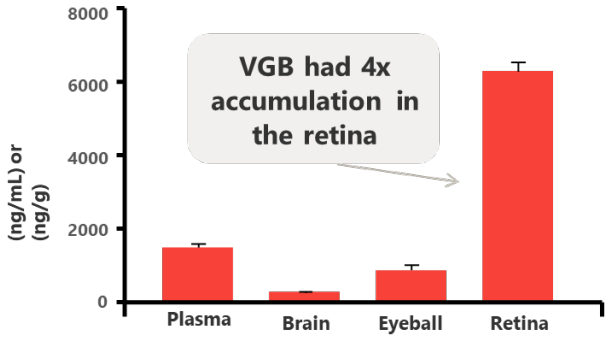
OV329

Mean Plasma and Tissue Concentration 5.0 mg/kg/day



VIGABATRIN

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



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

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

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

OV329 offers a differentiated target product profile

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

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

Phase 1 SAD/MAD design

Objective

Establish pharmacodynamic, pharmaco-kinetic activity, safety and tolerability of OV329 in healthy volunteers to inform Phase 2 dosing

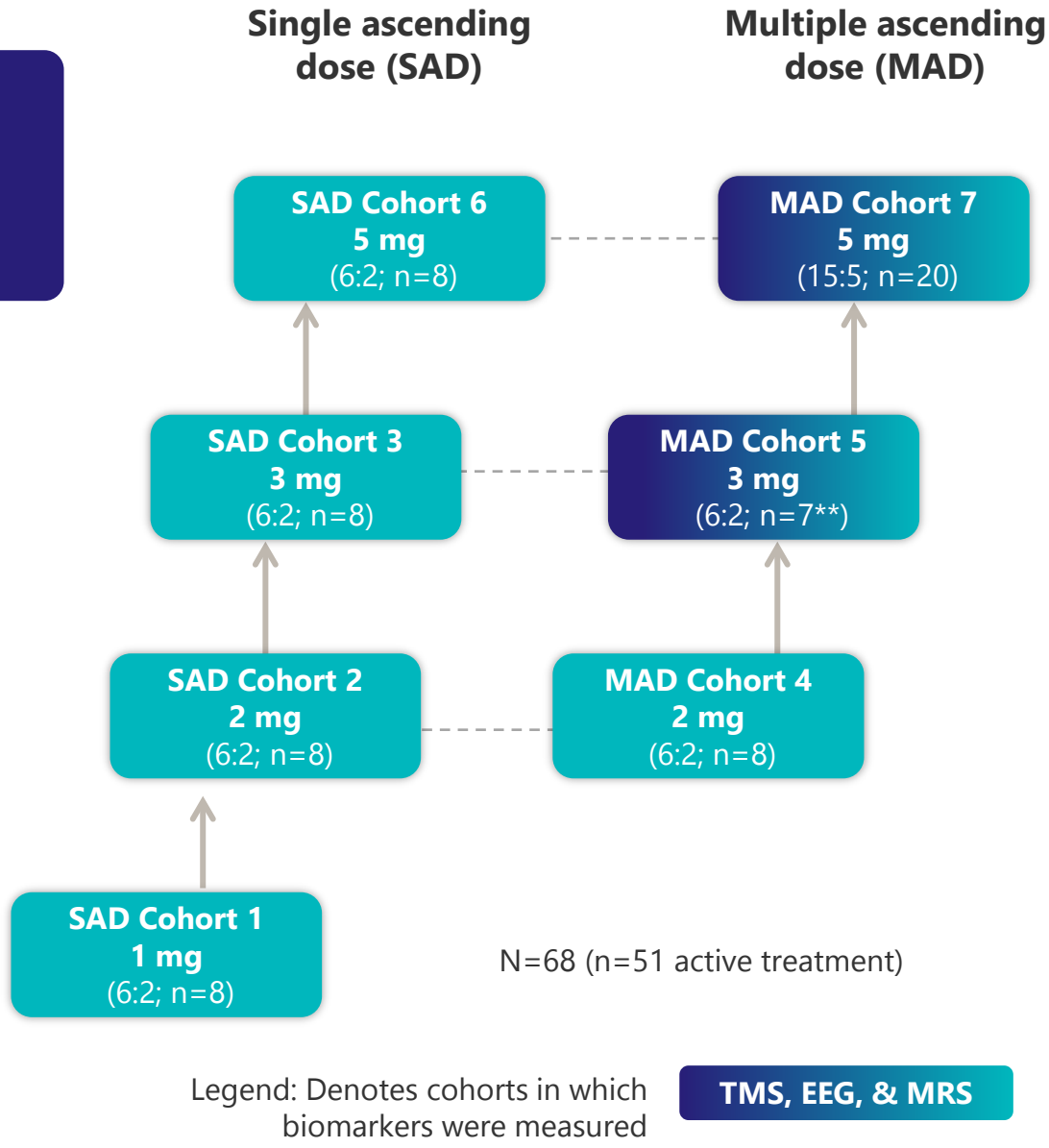
Ophthalmic safety metrics

- Best corrected visual acuity
- Fundus photography
- Indirect dilated ophthalmoscopy
- Automated threshold visual field perimetry
- Optical coherence tomography

Pharmaco-kinetics

Biomarker assessments explore pharmacodynamic activity & target engagement

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



**one person withdrew because of a family matter

Participant demographics

	SAD					MAD			
	Cohort 1 1 mg	Cohort 2 2 mg	Cohort 3 3 mg	Cohort 6 5 mg	Placebo	Cohort 4 2 mg	Cohort 5 3 mg	Cohort 7 5 mg	Placebo
Male, n(%)	3 (50.0)	2 (33.3)	5 (83.3)	4 (66.7)	4 (50.0)	4 (66.7)	4 (66.7)	10 (6.7)	6 (66.7)
Female, n(%)	3 (50.0)	4 (66.7)	1 (16.7)	2 (33.3)	4 (50.0)	2 (33.3)	2 (33.3)	5 (33.3)	3 (33.3)
N	6	6	6	6	8	6	6	15	9
Age (years)	40.2 ± 2.9	37.5 ± 11.3	37.0 ± 7.5	36.8 ± 13.4	34.0 ± 9.0	31.2 ± 8.9	27.0 ± 7.1	29.7 ± 6.8	30.8 ± 10.2
Height (cm)	165.9 ± 12.2	172.6 ± 6.9	181.2 ± 7.7	174.4 ± 9.4	169.3 ± 12.8	172.2 ± 9.3	170.3 ± 13.1	173.0 ± 9.2	175.1 ± 8.8
Weight (kg)	80.2 ± 10.1	81.4 ± 9.8	88.1 ± 14.0	76.5 ± 15.0	78.7 ± 12.2	75.0 ± 11.7	76.3 ± 16.9	80.5 ± 17.8	78.8 ± 20.8
BMI (kg/m^2)	29.4 ± 4.1	27.2 ± 2.8	26.7 ± 2.7	24.9 ± 2.8	28.0 ± 4.1	24.9 ± 3.0	26.3 ± 4.6	26.7 ± 3.9	25.5 ± 6.0

OV329 was well-tolerated with no treatment-related SAEs

Summary of potentially treatment-related and treatment-emergent AEs

Dosage	Adverse Event	OV329	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. N= number of participants
2. (x "number") = number of events

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

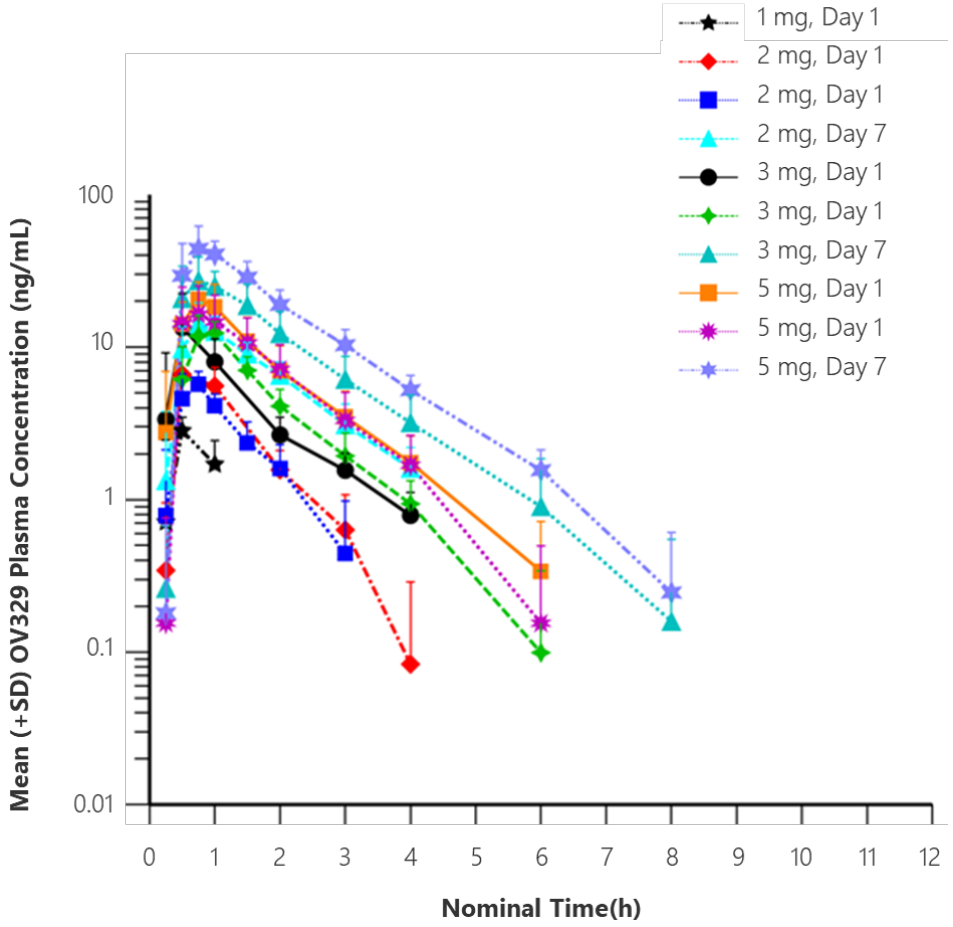
Ophthalmic metric*	Result
Best corrected visual acuity	No treatment related effects
Fundus photography	No treatment related effects
Indirect dilated ophthalmoscopy	No treatment related effects
Automated threshold visual field perimetry	No treatment related effects
Optical coherence tomography	No treatment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

1. *Assessments from baseline to Day 30

Phase 1 results showed predictable pharmacokinetic profile aligned with pharmacology strategy

- Dose linear increases in AUC and Cmax demonstrated in repeat dosing
- Consistent renal clearance after single and repeated dosing
- Steady state achieved by Day 3
- Tmax 1 hour

OV329's irreversible binding and potency inhibits the GABA-AT enzyme with low daily doses and maintains suppression due to slow turnover of the enzyme in the CNS



Biomarker results

Overview

TOPLINE PHASE 1
Biomarker & safety results

Questions & answers

What does TMS tell us about inhibition?

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

Multiple biomarkers confirm OV329's inhibitory activity that meets or exceeds VGB

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

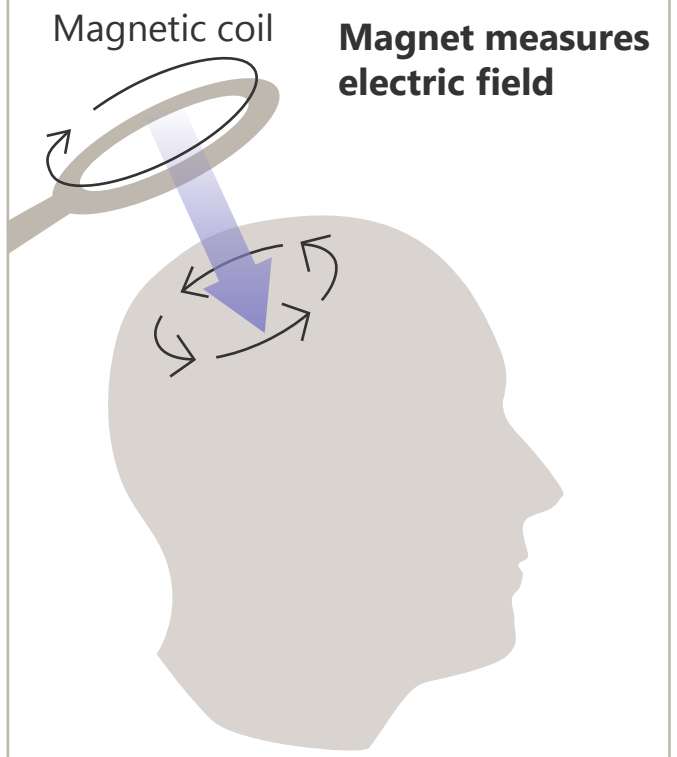
TMS metric	Vigabatrin therapeutic dose (50 mg/kg) Measured in healthy volunteers[1,2]	OV329 3 mg and 5 mg doses Measured in healthy volunteers
Paired-pulse long-interval intracortical inhibition (LICI) 150 ms	↑ Improved ~35%	↑ **Improved inhibition by 44-53%[3]**
Paired-pulse long-interval intracortical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

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

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

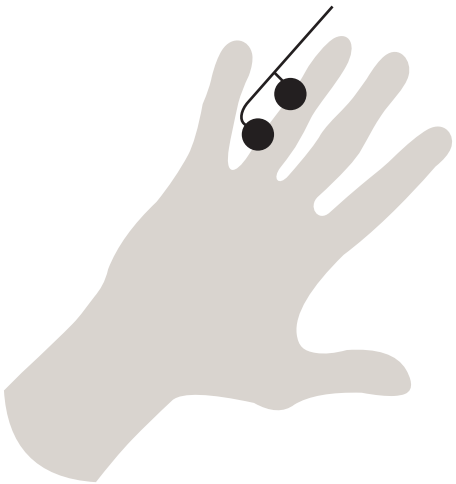
LICI methodology

Measures cortical activity by pairing two separate higher-intensity stimuli with a (long) interval of time between the two stimuli



Magnet measures electric field

Electrodes placed on target muscles* to measure motor response

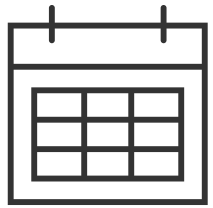


Paired-pulse motor-evoked potential (stimuli) delivered at varying time intervals

GABA-mediated inhibition

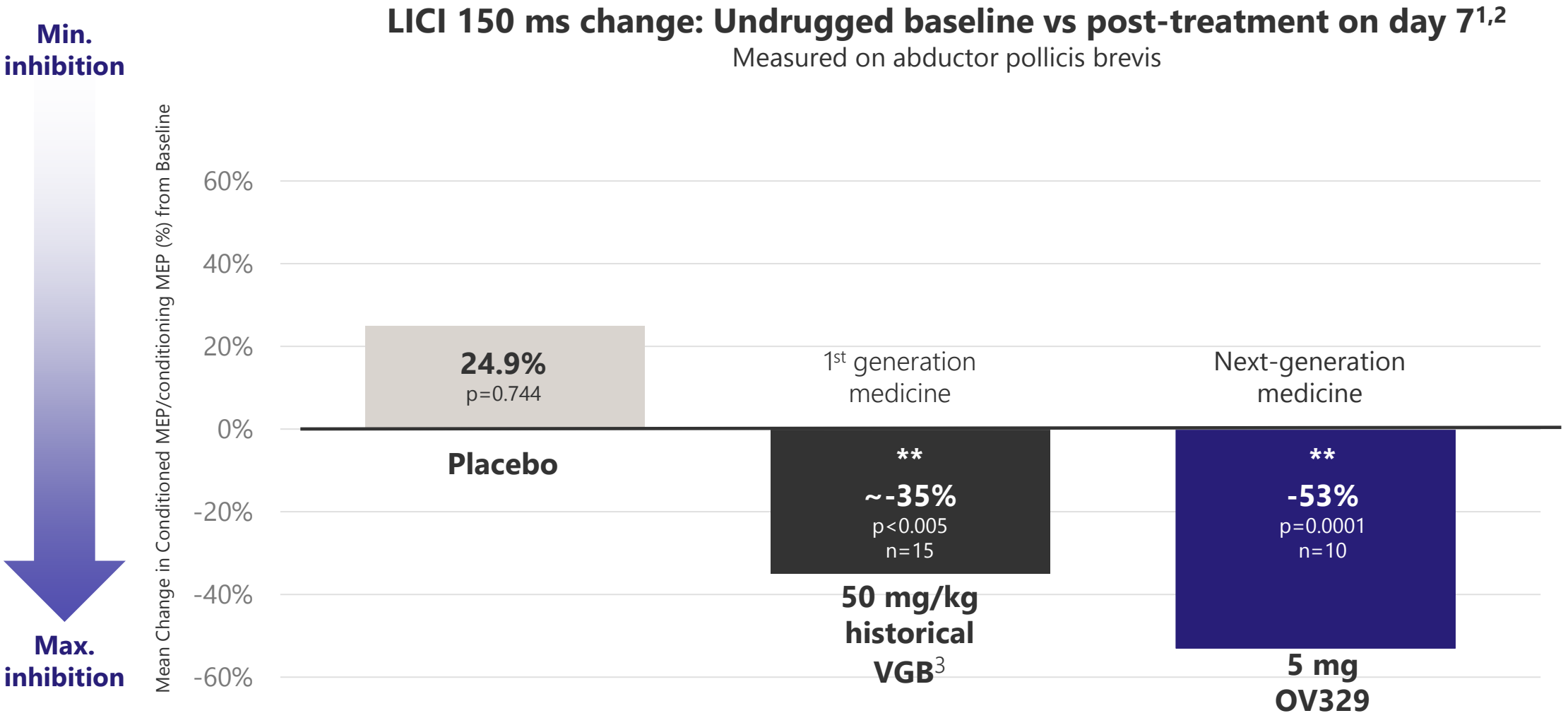
Motor response

Participants' mean response measured pre-treatment and after 7 days of daily dosing



*First dorsal interosseous (FDI) and abductor pollicis brevis (APB)

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



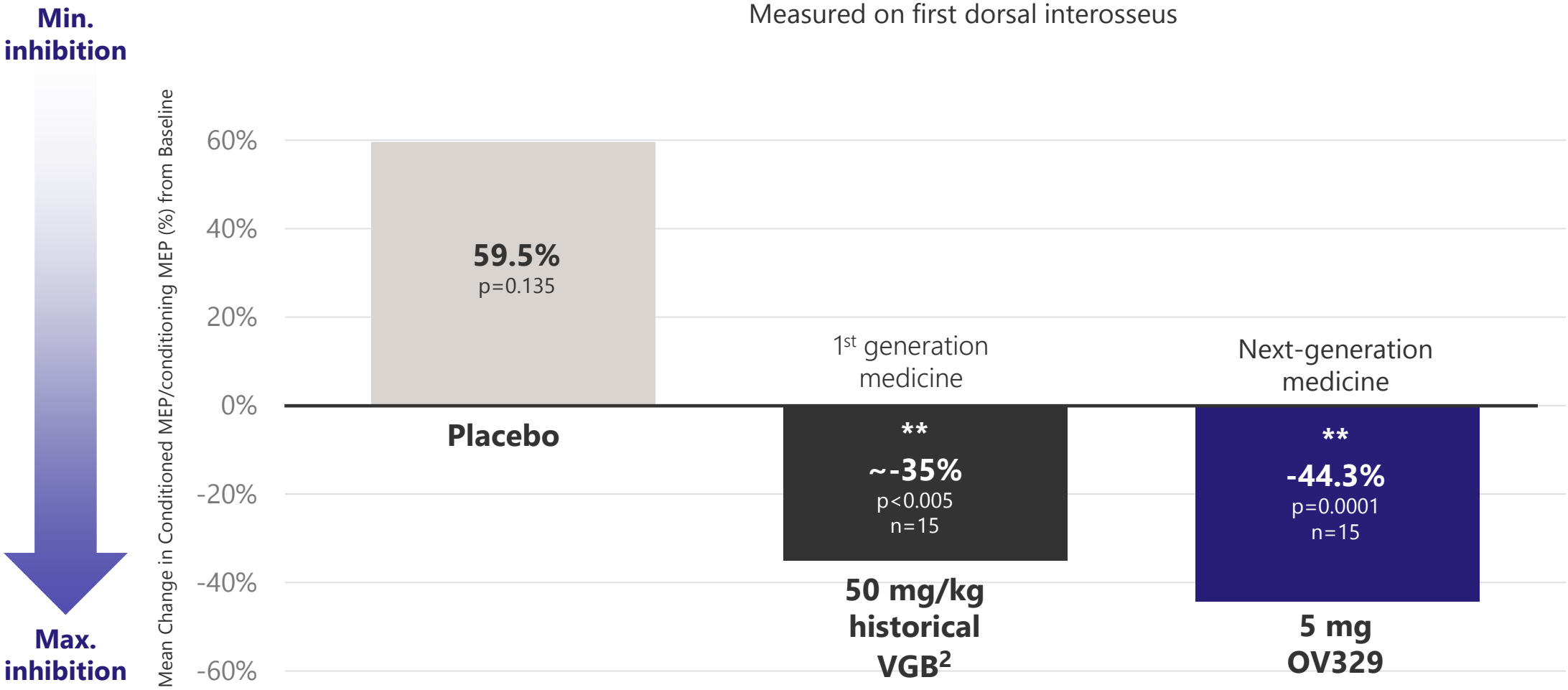
1. Baseline is at day 1 prior to treatment
2. Note: 6 subjects were unavailable for APB findings because of inconsistent capture, of which 5 were active, one was placebo
3. Pierantozzi et al. Brain Res. 2004 Nov 26;1028(1):1-8

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

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

LICI 150 ms change: Undrugged baseline vs post-treatment on Day 7[1,2]

Measured on first dorsal interosseus



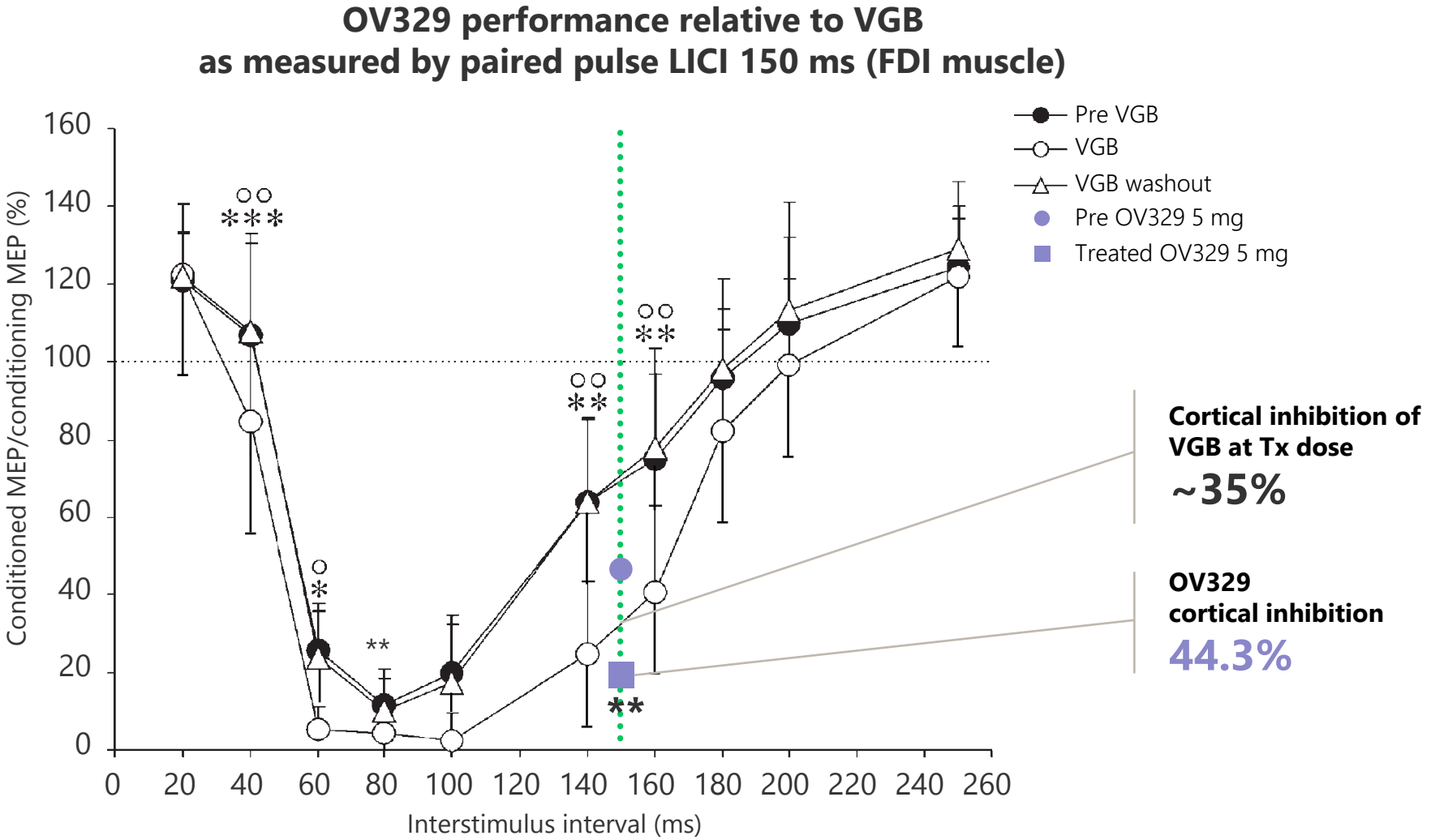
1. Baseline is at day 1 prior to treatment
2. Pierantozzi et al. Brain Res. 2004 Nov 26;1028(1):1-8

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

OV329 delivered cortical inhibition that matched or exceeded a therapeutic dose of VGB

Min. inhibition

Max. inhibition



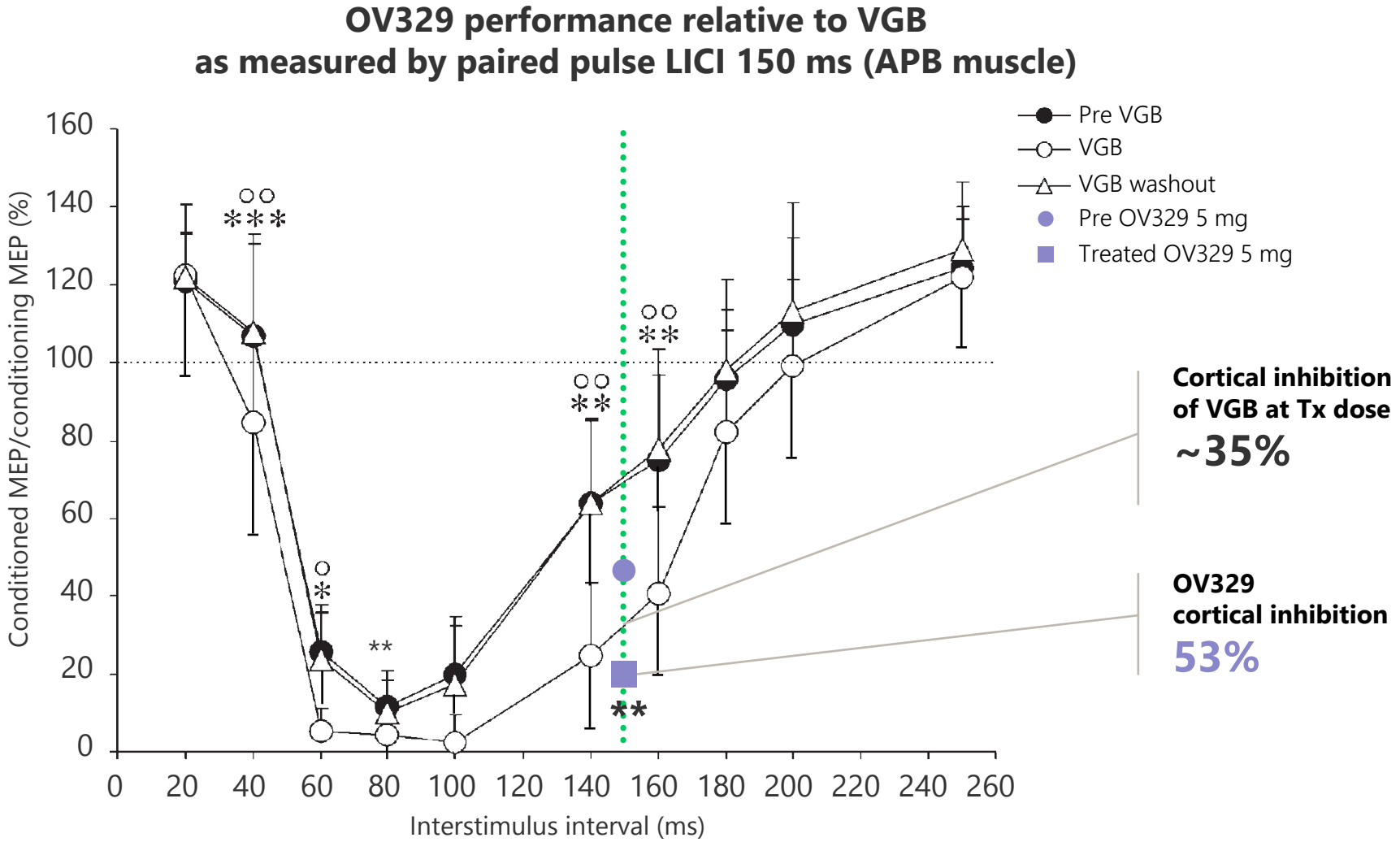
1. OV329 results as measured using FDI muscle
2. Pierantozzi et al. Brain Res. 2004 Nov 26;1028(1):1-8.
3. *p<0.05, **p<0.01, ***p<0.001

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

OV329 delivered cortical inhibition that matched or exceeded a therapeutic dose of VGB

Min. inhibition

Max. inhibition

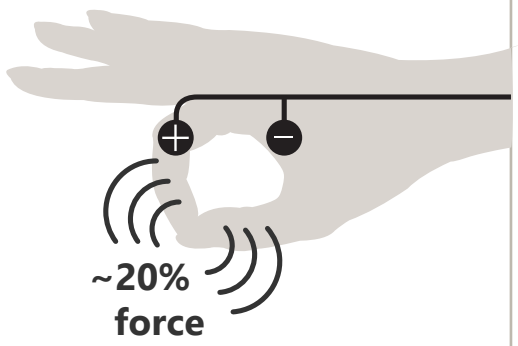


1. OV329 results as measured using FDI muscle
2. Pierantozzi et al. Brain Res. 2004 Nov 26;1028(1):1-8.
3. *p<0.05, **p<0.01, ***p<0.001

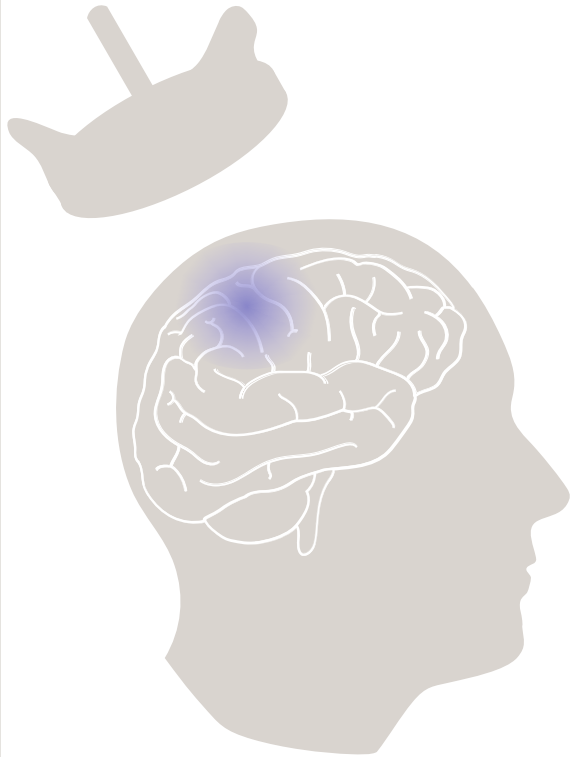
Note: Information compares similar studies and methodologies; a head-to-head trial was not conducted

CSP methodology[1]

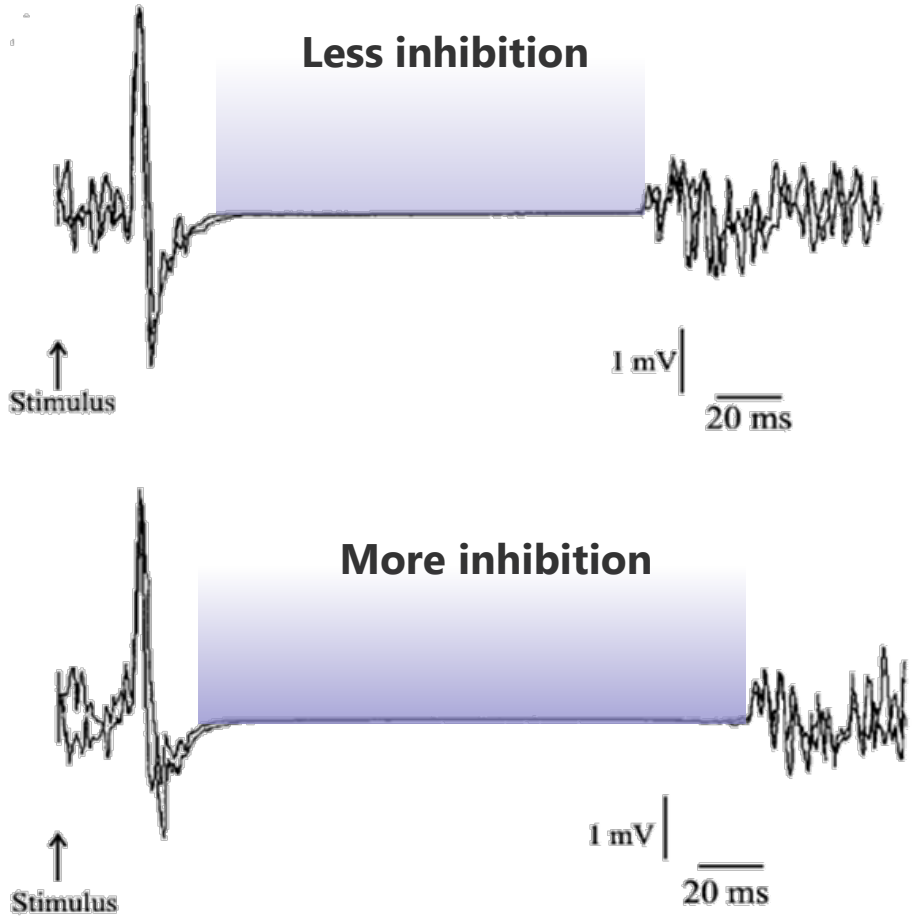
Uses muscle exertion to measure cortical activity



Measures electrical field using magnet



Single stimuli (MEP)



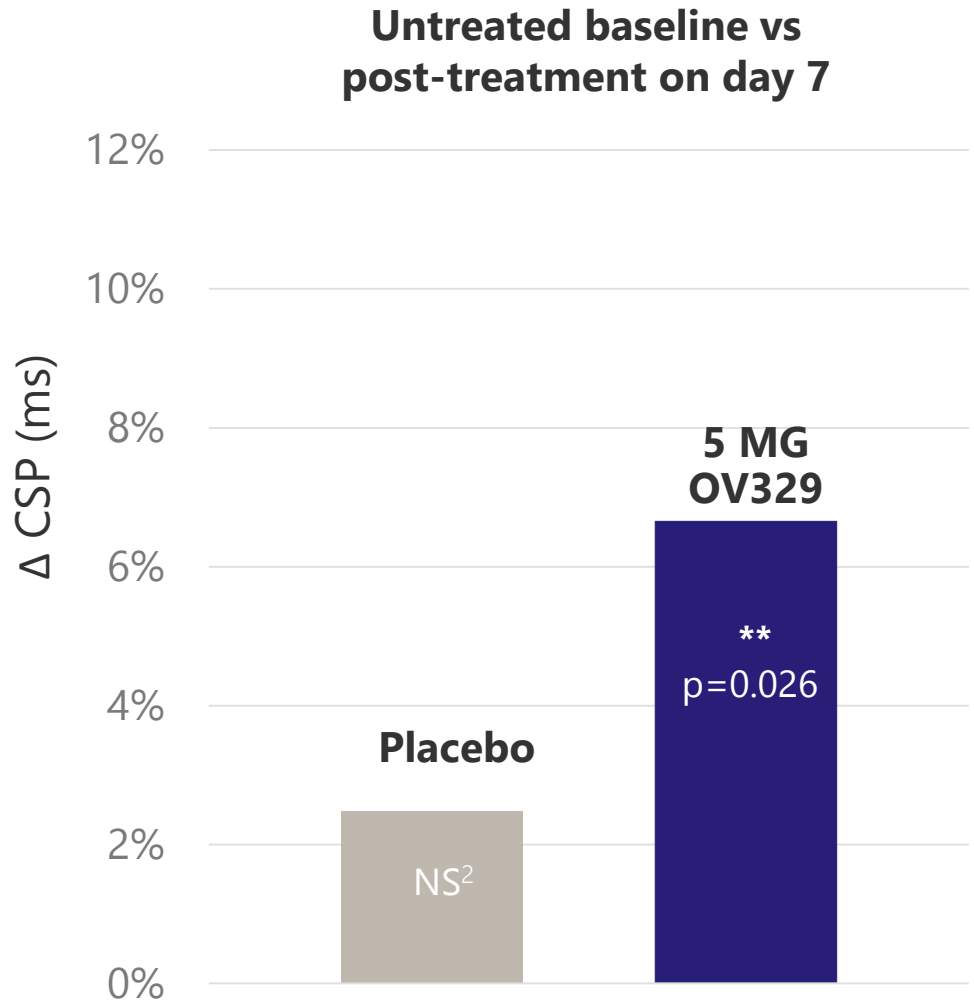
Meaningful result is treatment-related prolongation of the CSP, which is indicative of inhibitory activity

1. Edwards D, Davila Perez P, Horvath J, Rotenberg A, Pascual-Leone A. A Practical Manual for Transcranial Magnetic Stimulation. Springer; 2024. doi:10.1007/978-3-031-62304-2.
Pierantozzi et al. (2004), Brain Res; 1028(1):1–8.

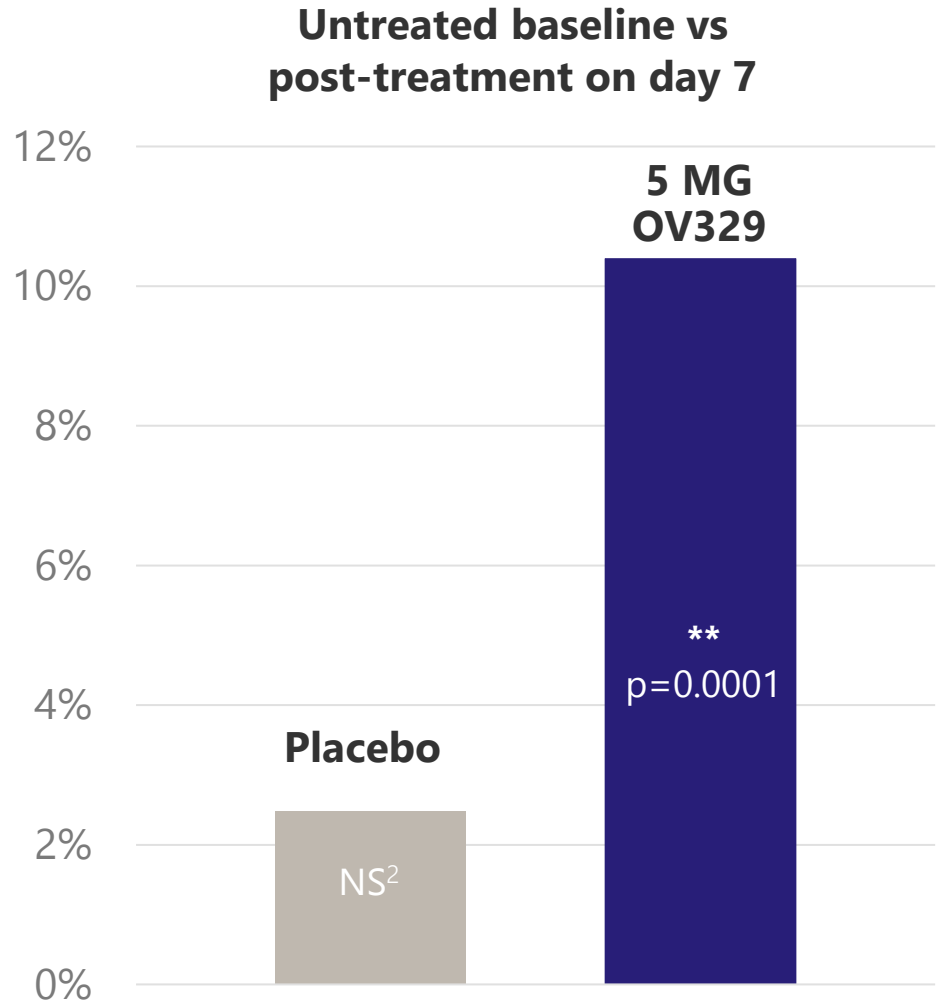
OV329 increases inhibition as measured by the CSP

Max. inhibition

Min. inhibition



Measured on abductor pollicis brevis



Measured on first dorsal interosseous

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

MRS results

Methodology[1]

- Seek signs of brain penetration and target engagement based upon change in GABA concentration in the brain
- Measure GABA levels in the medial parietal lobe (within 24 hours of the last day of dosing) relative to participants' pre-treated and post-treatment baseline
- Applies state-of-the art statistical models to avoid counting overlapping neuro-metabolite signals

Results

3 mg MAD n=6	Placebo n=1[2]	5 mg MAD n=14[3]	Placebo n=4
5.65%	-1.62%	7.13%	0.24%

- Suggest OV329 is getting into the brain and increasing GABA levels relative to participants' natural baselines
- Significant variability was seen in subjects' baseline GABA levels (to be expected)
- Findings were not statistically significant

1. 1. Methods leveraged updated best practices as defined in Big GABA 2017 consensus paper; readings secured using 7T MRIs
2. Only 1 placebo participant in the 3 mg MAD cohort (1 pbo dropped out due to a death in the family)
3. 2 subjects in the 5 mg MAD cohort had to be removed due to inconsistent data capture (1 placebo, 1 treated)

Exploratory EEG results

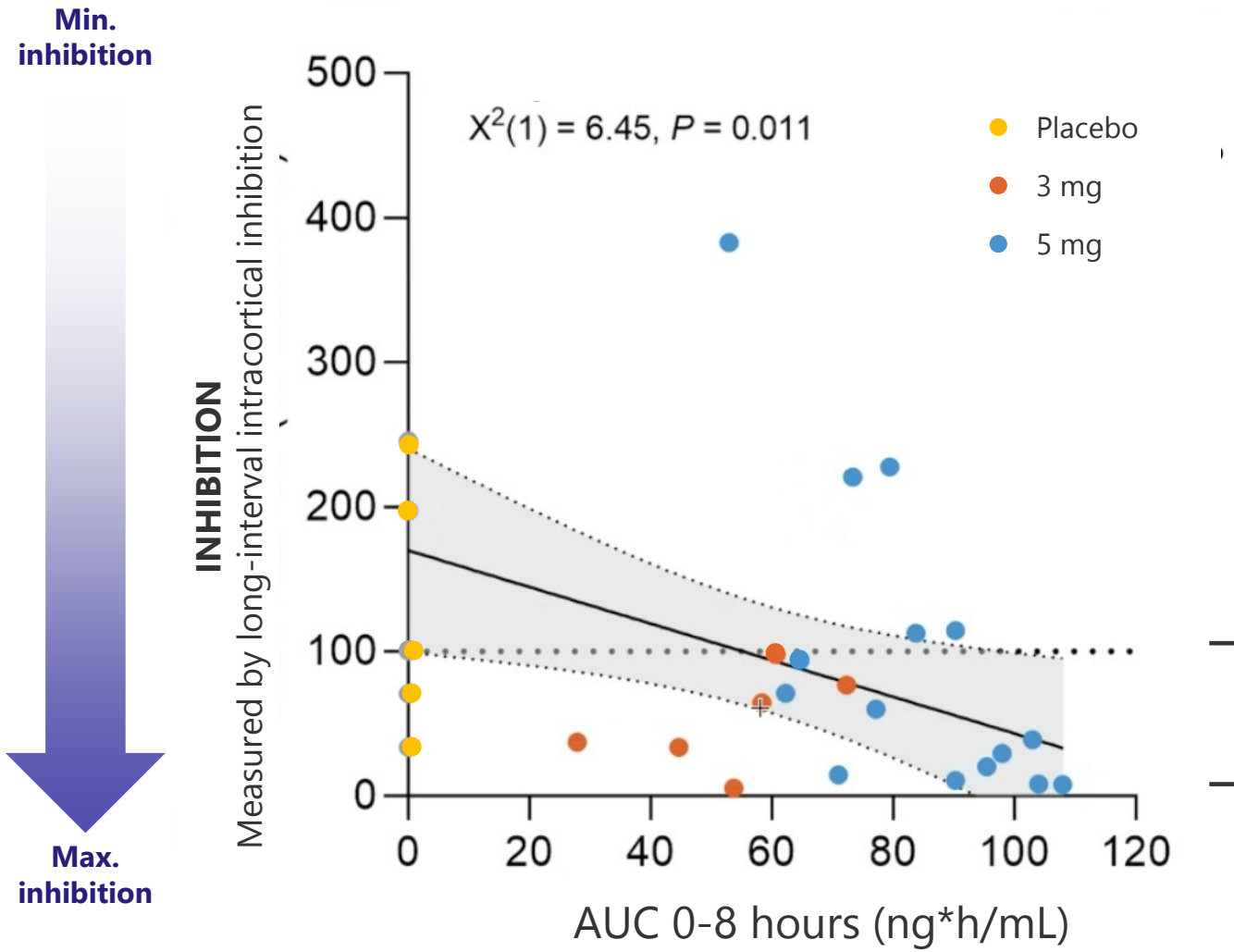
Methodology

- Eyes-open and eyes-closed resting EEG data was performed
- Absolute and relative power (ratio of absolute band power 1–80 Hz) of 6 frequency bands were measured from the parietal and occipital channels
  - Delta, 1–4 Hz
  - Theta, 4–7 Hz
  - Alpha, 8–12 Hz
  - Beta, 12–30 Hz
  - Low gamma, 30–45 Hz
  - Gamma, 55–80 Hz

Results

- Readings had significant noise
- 3 mg MAD cohort:
  - Demonstrated a statistically significant increase in beta + gamma power (n=5) [0.65, 0.77]; p=0.03 compared to pre-treatment
- 5 mg MAD cohort:
  - Post-hoc analysis demonstrated increased relative delta (EMD=4.1% [0.0, 8.1], p=0.040) and theta (EMD=4.6% [1.1, 8.1], p=0.013) power for eyes-open parietal electrodes post intervention compared to pre-treatment
  - There was a reduction in relative beta within placebo (EMD=-7.2% [-14.1, -3.9], p=0.039)

OV329 exposure in brain is highly correlated to inhibition and demonstrated a clear responder rate



Clear relationships between:

- Dose and brain exposure
- Brain exposure and cortical inhibition as measured by LICI 150 ms
- Results confirm Ovid internal Tx models
- Support advancement of 7 mg as maximal dose

Target brain exposure threshold of >80 ng*hr/mL highly correlated with inhibition

A potential best-in-class ASM profile

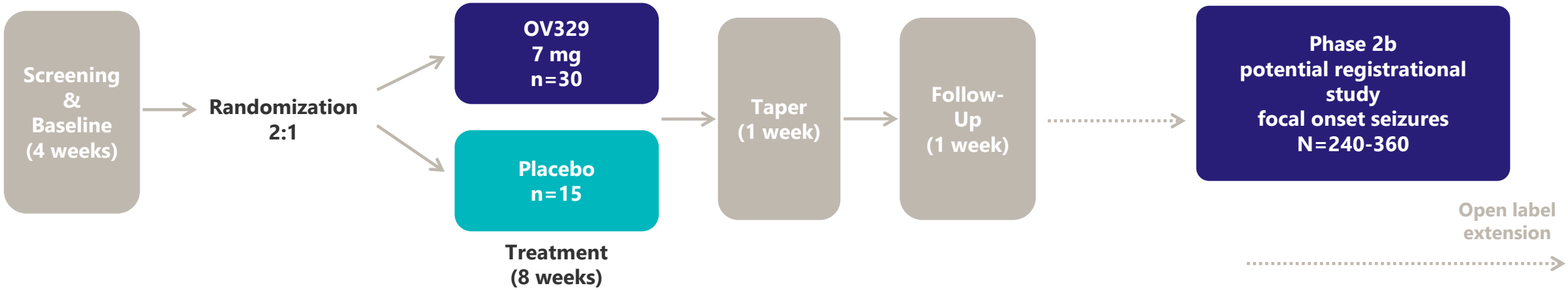
- Potential best-in-therapeutic area safety and tolerability profile relative to approved and development-stage ASMs
- Inhibition that is on par, or in excess of, therapeutic doses of VGB, providing confidence for patient clinical trials[1]
- Clear responder rate and predictable PK guides human dose selection
- Differentiated MoA in a field crowded with “me too” mechanisms
- No anticipated drug-drug interactions

Next steps: Phase 2a trial evaluating the anti-seizure effect of OV329 in patients with focal epilepsy

1. Pierantozzi M, Marciani MG, Palmieri MG, Brusa L, Galati S, Caramia MD, Bernardi G, Stanzione P. 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 Nov 26;1028(1):1-8. doi: 10.1016/j.brainres.2004.06.009. PMID: 15518635

Proposed Phase 2a trial design in patients with focal epilepsy

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



Trial design

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

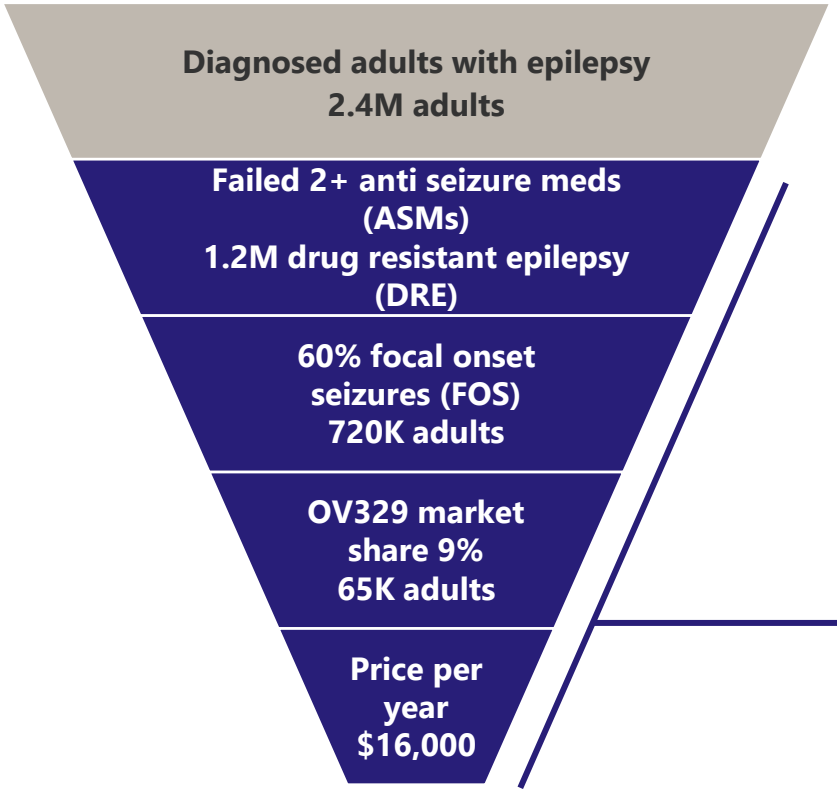
Outcome measures

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

Intend to conduct additional SAD/MAD 7 mg dosing cohort prior to initiation

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

Substantial opportunity within the drug-resistant epilepsy segment



>$1 Billion

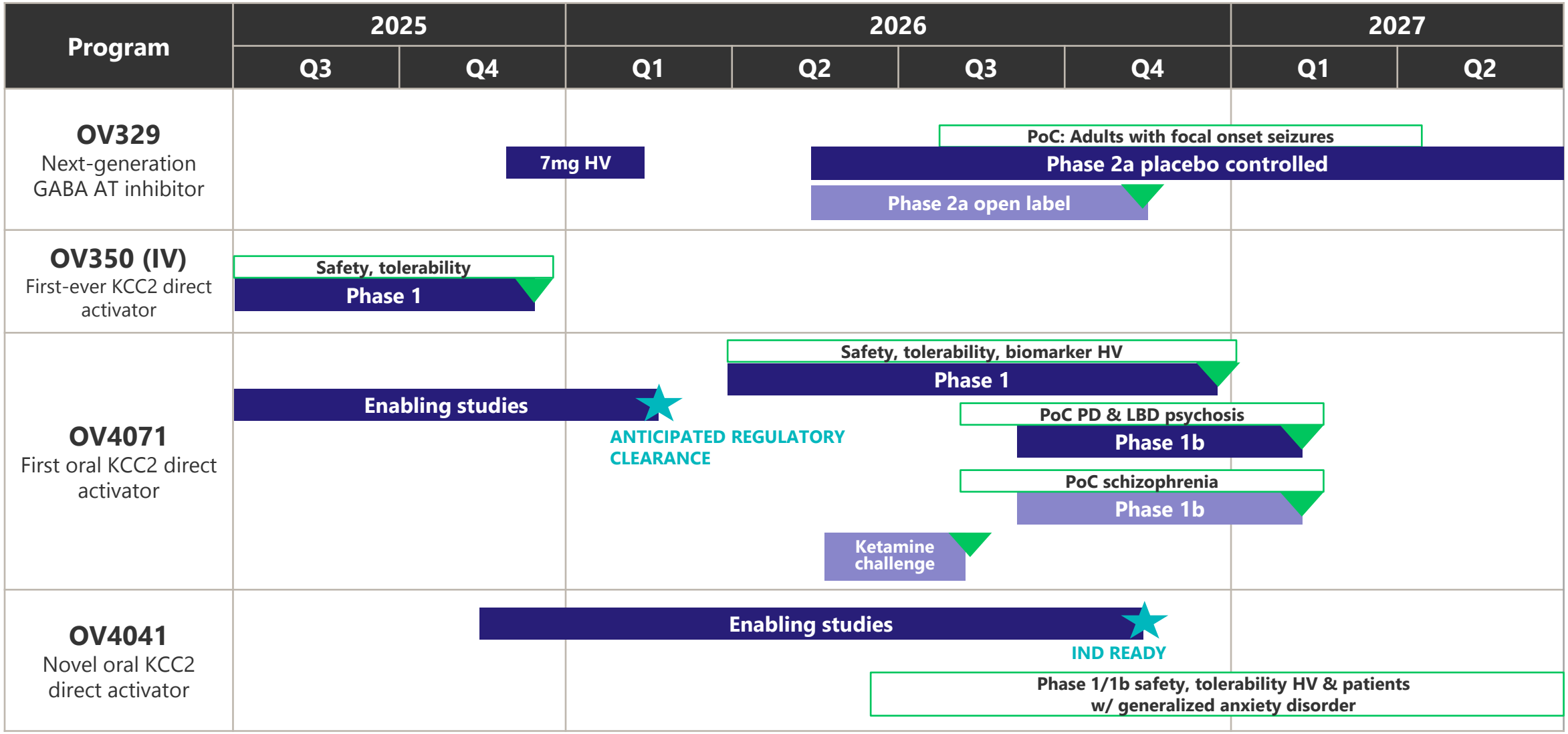
Key: Voltage-gated Ion Channel Target | GABAergic | Synaptic Target | Other / Mixed / Unknown

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

Catalyst-rich pipeline delivering over the next 24 months



Jeremy M. Levin, DPhil, MB BChir
Chairman &
Chief Executive Officer

Meg Alexander
President &
Chief Operating Officer

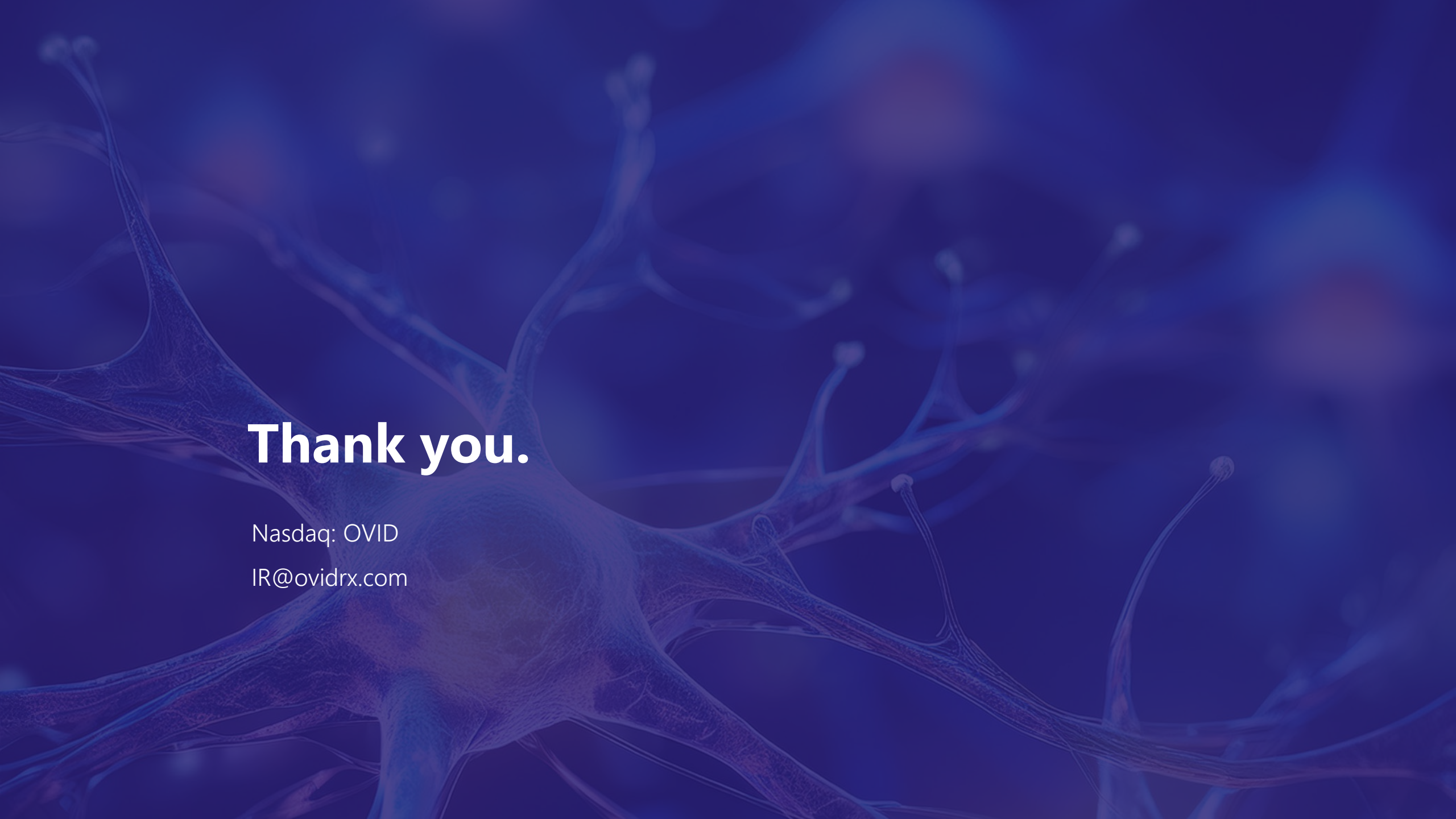
Jeffrey Rona
Chief Business
and Financial Officer

Zong Zhong, PhD
Chief Scientific Officer

Overview

TOPLINE PHASE 1
Biomarker & safety results

Questions & answers



Thank you.

Nasdaq: OVID

IR@ovidrx.com