

Ovid Therapeutics

Corporate Presentation

JUNE 2024

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Forward looking statement

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 including, without limitation, statements regarding timing for the the potential opportunity for soticlestat; the timing of potential regulatory filings and or regulatory decisions for soticlestat, and the potential for milestone and royalty payments from Takeda for soticlestat, statements regarding the timing for additional data readouts from Takeda evaluating soticlestat for Lennox-Gastaut and Dravet syndromes; discussions between Takeda and regulators related to the approval of soticlestat; success of Takeda's trials in soticlestat, Ovid's expectations regarding the duration its cash runway, and the expectation that it will support the advancement of Ovid's pipeline and Ovid's potential future business development opportunities; the timing and development of Ovid's product candidate pipeline and achievement of expected near- and long-term milestones; the potential therapeutic benefits of Ovid's current or future product candidates and pipeline programs; the potential development and therapeutic opportunity of OV888; and the potential safety, selectivity and potency of OV888 and other ROCK2 inhibitors; the potential use of OV888 and other ROCK2 inhibitors to treat cavernous cerebral malformations and other rare central nervous system disorders; the potential timing of the pivotal formulation for OV888; the potential timing of clinical studies for OV888 and the resulting data; the reporting of data for the potential Phase 1 study in healthy volunteers for OV329; the potential use of OV329 to treat rare and treatment-resistant forms of epilepsy and seizures; the clinical and regulatory development of OV329, including the anticipated timing of clinical trials of OV329; the likelihood that data for OV329 will support future development and therapeutic potential; the potential development of OV350 and other KCC2 compounds in the Company's library; the potential timing of a potential IND filings for OV329 and OV350; the suitability of the Company's library of novel, direct KCC2 transporter activators for a range of formulations and administrations that would make it possible to pursue both chronic and acute epilepsies; Ovid's business development intentions; the success of any licensing or partnering opportunities; the success, timing, ability to attract and maintain strategic collaborations; the clinical and regulatory development and potential commercialization of soticlestat, OV888, OV329, OV350, or any of Ovid's other current or future product candidates and pipeline programs and market opportunities. You can identify forward-looking statements because they contain words such as "will," "may," "plan," "believes," intends," "anticipates," "design", "target," "seek," "expects," "demonstrates," "observe," 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 fact nor guarantees or assurances of future performance. Important factors 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, risks related to Ovid's ability to achieve its financial objectives, the risk that Ovid may not be able to realize the intended benefits of its technology or its business strategy, risks related to 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 risks to 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 Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission (SEC) on May 14, 2024, and in future filings Ovid makes with the SEC. Any forward-looking statements contained in this press release 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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NOVEL BIOLOGICAL TARGETS

Modulated by potential first-in-class or best-in-class mechanisms of action

HARNESSING A SCIENTIFIC REVOLUTION

Applying highly specific small molecules for the central nervous system (CNS)



DIFFERENTIATED PIPELINE

Intended to treat rare epilepsy & brain conditions

4× 2025 CLINICAL & REGULATORY MILESTONES

From Ovid's pipeline expected by 2025

Potential to be a leading rare epilepsy & brain disorder company

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Differentiated pipeline with cadence of value-driving events

PROGRAMS	INDICATION/TARGET	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	ANTICIPATED MILESTONES
ROCK2 PLATFORM & OV888[1] Selective ROCK2 inhibitor Collaboration with: GRAVITON BIOSCIENCE CORPORATION	Cerebral cavernous malformations Undisclosed rare CNS indications					• MAD completed; safety findings in H1 2024 • Phase 2 initiating in H2 2024
OV329 ORAL GABA-aminotransferase inhibitor	Oral formulation for chronic treatment of epilepsies					• Phase 1 readout in H2 2024
OV329 INTRAVENOUS GABA-aminotransferase inhibitor	IV formulation for acute treatment of seizures					• IND in H2 2024[2]
KCC2 PLATFORM & OV350 KCC2 transporter activator	Resistant epilepsies and other neurological conditions					• IND for OV350 in H2 2024[2]
SOTICLESTAT Cholesterol 24-hydroxylase inhibitor Out-licensed to: Takeda	Dravet syndrome		COMPLETE			• Topline results available • Reviewing totality of data with health regulators
	Lennox-Gastaut syndrome		COMPLETE			

UNDISCLOSED INDICATIONS OUTSIDE SEIZURES THAT MAY PROVIDE BUSINESS DEVELOPMENT OPPORTUNITY

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Financial summary & upcoming milestones

Cash balance $90.3 million

As of March 31, 2024

Expected to support runway into H1 2026

Shares outstanding

70.9 million

As of May 10, 2024

Pipeline Milestones

	Key Milestones	Anticipated Timing
OV888	Phase 1 MAD study Top Line results	H1 2024
OV888	Initiate Phase 2 signal-finding trial in CCM	H2 2024
OV329	Results of Phase 1 SAD/ MAD with MRS and TMS biomarkers	H2 2024
OV329	IND for IV formulation submitted	H2 2024
KCC2 & OV350	IND for IV formulation submitted	H2 2024

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Soticlestat

Sold rights back to Takeda Pharmaceuticals

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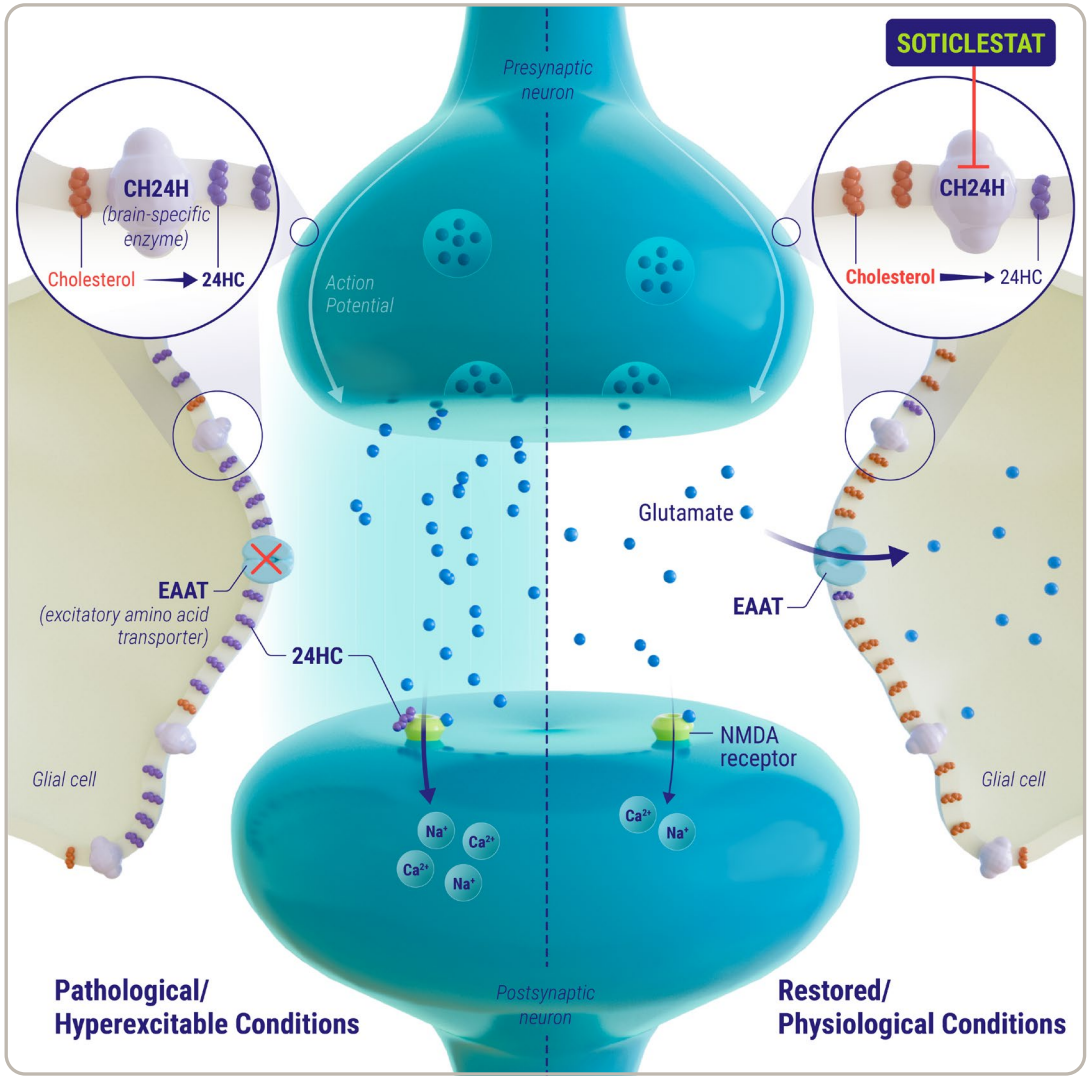
Overview

- Soticlestat is a potential novel inhibitor of cholesterol 24-hydroxylase that is thought to control seizures by reducing:
  - Over-activated glutamatergic signaling; neuronal hyperexcitability
  - Inflammation
- Ovid co-developed soticlestat with Takeda from preclinical trials through to Phase 2
- Sold rights back to Takeda in 2021*
- Takeda conducted & completed two, Phase 3 trials:
  - SKYWAY in Lennox-Gastaut syndrome
  - SKYLINE in Dravet syndrome
- If Takeda secures regulatory approval for soticlestat, Ovid may receive potential milestones and royalties



*Ovid sold its 50% share of rights back to Takeda

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Topline SKYLINE & SKYWAY results

	skyline STUDY DRAVET SYNDROME	skyway STUDY LENNOX-GASTAUT SYNDROME
Primary endpoint	• Soticlestat narrowly missed primary endpoint of reduction from baseline in convulsive seizure frequency vs. placebo (p-value= 0.06) • Soticlestat showed, in a pre-specified subgroups of patients, significant treatment effects on the primary efficacy endpoint,	• Soticlestat missed its primary endpoint in reduction in major motor drop seizure
Secondary endpoints	6 key secondary endpoints showed clinically meaningful and significant results (all p-values ≤ to 0.008) • Responder rate • Caregiver and clinician global impression of improvement scales • Seizure intensity and seizure duration scales	• Some secondary endpoints showed significant treatment effects
Safety	Favorable safety and tolerability profile consistent with prior trials	
Pooled analysis with Phase 2	• Soticlestat showed reduction from baseline in convulsive seizure frequency compared to placebo (p-value=0.001)	• N/A

Takeda moving forward to discuss the totality of the data with regulatory authorities

1. Takeda data on file

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OV888
(GV101)

A highly selective ROCK2 inhibitor for potential treatment of cerebral cavernous malformations (CCM) & other CNS disorders

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OV888 (GV101)
Highly selective ROCK2 inhibitor for rare neurological conditions

POTENTIAL OPPORTUNITY

Potential 1st and best-in-class disease modifying treatment for CCM

Well characterized safety profile in humans

Large addressable population with no pharmacological treatment

Mechanism applicable for other CNS conditions

STATUS

Completed Phase 1 safety study

- Pivotal gel capsule formulation
- Healthy volunteer study
- No SAEs observed

Potential 1st indication: CCM

~70x more selective for ROCK2 than NRL-1049[1]

Intend to initiate patient signal finding trial in H2 2024

1. Based on analysis of OV888 and NRL-1049 biochemical assays

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Cerebral cavernous malformations

Presentation

- Mulberry-shaped abnormal blood vessels in the brain and or spinal cord
- Vessels have thin, irregularly formed walls that can leak blood over time
- 2nd most common intracranial vascular malformation in humans[1,2]
  - 1 in 500 people in the U.S. have at least one cavernous malformation in their brain
- Symptoms include seizures, headaches, hemorrhage, and functional neurological deficits[2]
- Diagnosis is typically classified as either sporadic or familial

Morbidity & mortality

- ~30% of patients who have a hemorrhage will suffer moderate to severe disability[3]
- Overall morbidity was 26% after a mean follow up of 1.9 years since the first hemorrhage[5]
- One hemorrhage is a significant risk factor for future bleeds[4]

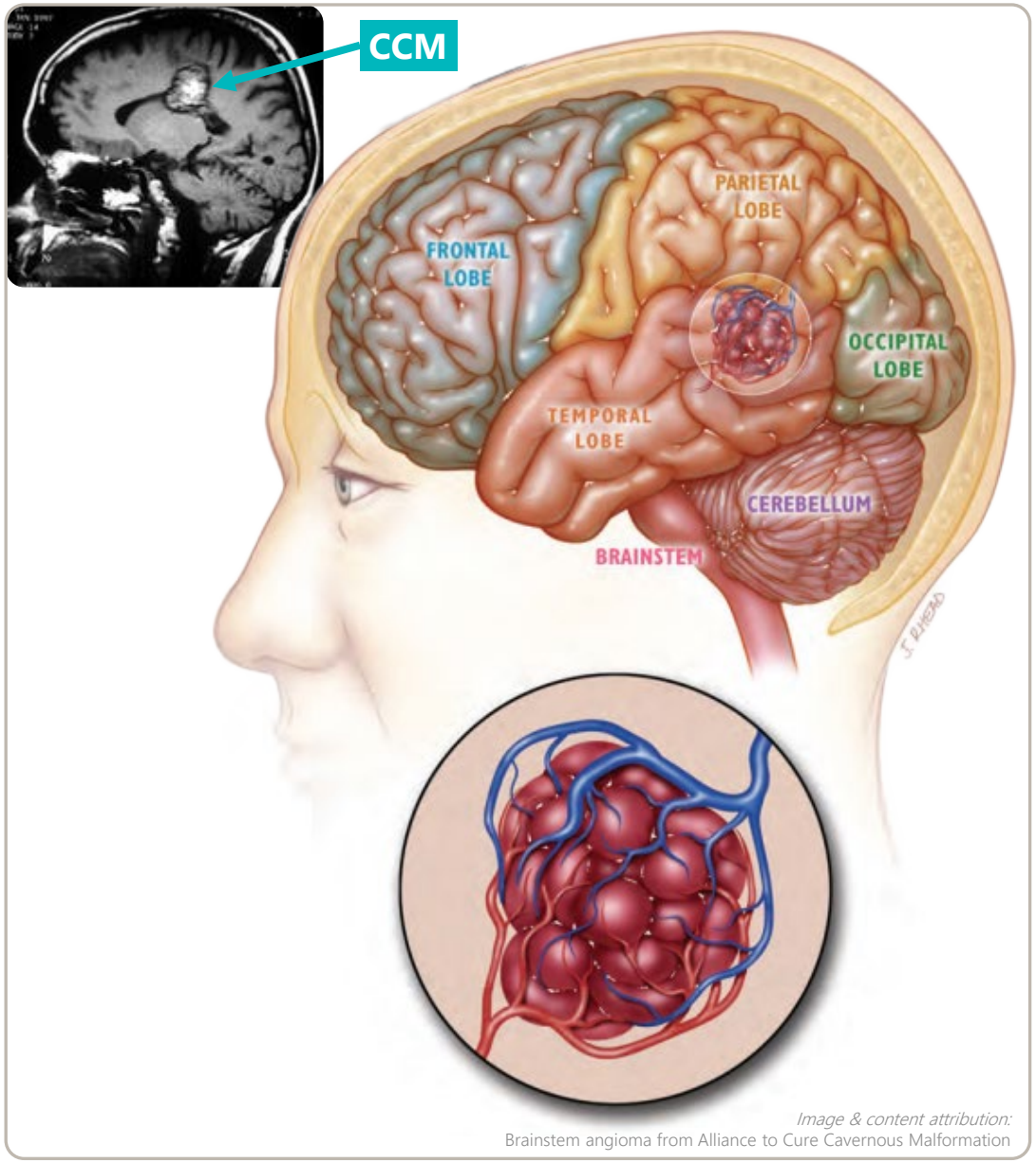

Image & content attribution:
Brainstem angioma from Alliance to Cure Cavernous Malformation

1. Zafar A, et al Stroke. 2019 May;50(5):1294-1301. doi: 10.1161/STROKEAHA.118.022314. PMID: 30909834; PMCID: PMC6924279 ;
2. Caton MT, Stat Pearls Publishing; https://www.ncbi.nlm.nih.gov/books/NBK538144/.;
3. Santos, A.N., et al. Sci Rep 13, 15490 (2023). https://doi.org/10.1038/s41598-023-42594-0
4. Dammann P, et al. World Neurosurg. 2016;91:73–80. doi: 10.1016/j.wneu.2016.03.080
5. Ma, Li Et al. Stroke, 2020 https://doi.org/10.1161/STROKEAHA.120.029942

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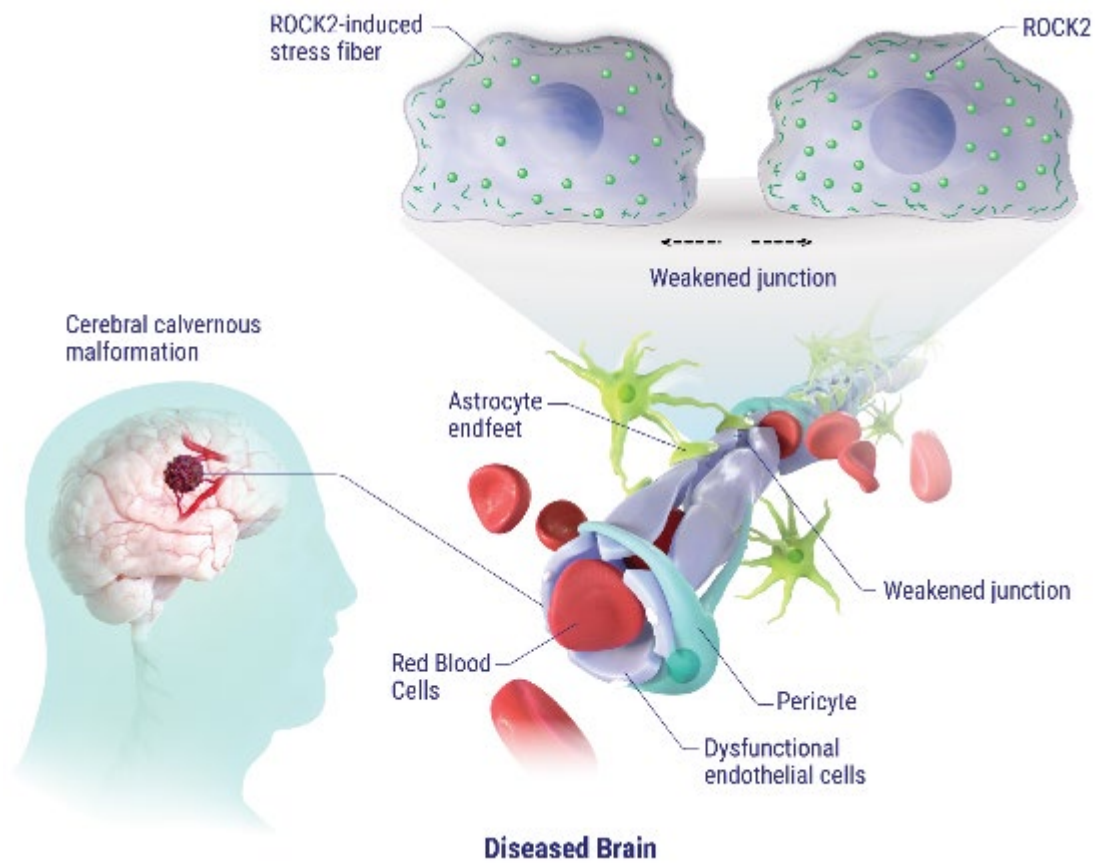
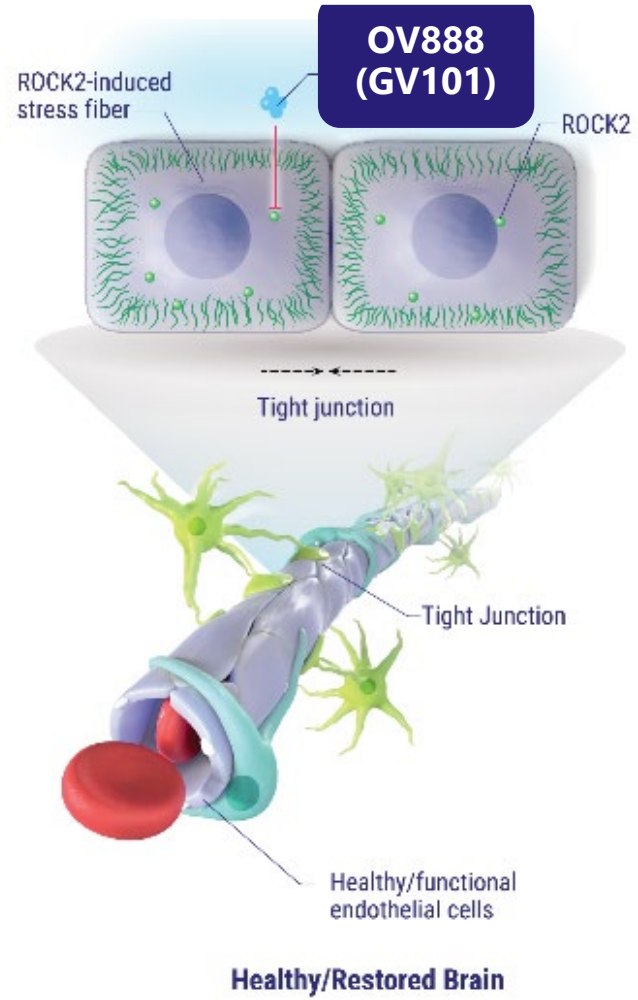
Proposed mechanism of action of OV888 (GV101)

Pathological State



Endothelial cytoskeleton abnormalities & weakened cell junctions can lead to lesions and bleeds

Proposed Mechanism of Action



A. L. Borilkova, et. al (2010) Journal of Biological Chemistry, Vol 285. 11760 and O. Pertz, et al (2006) Nature, Vol 440:1069

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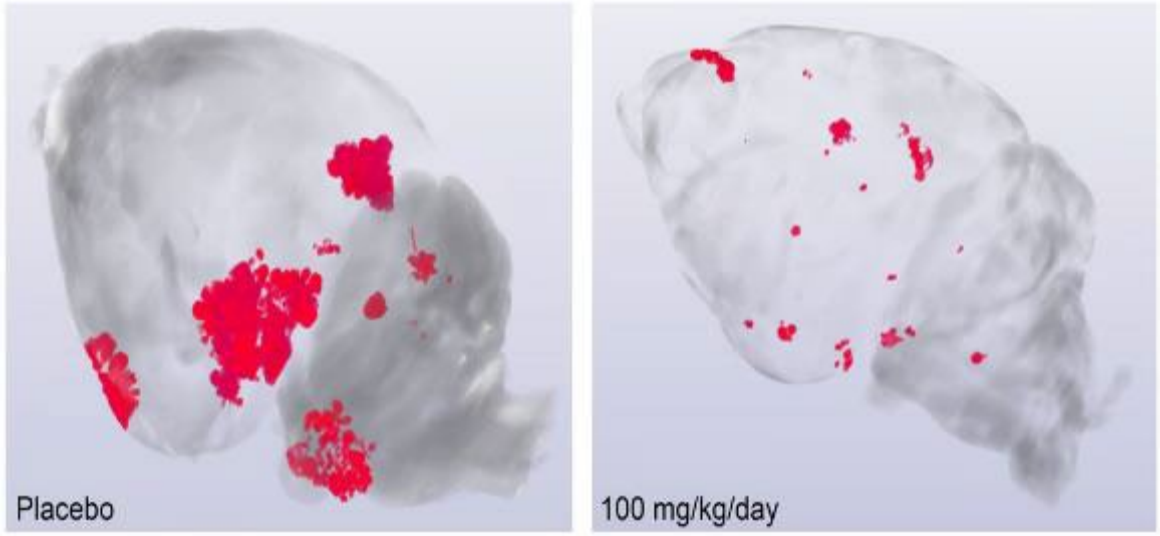
Preclinical validation for ROCK2 inhibition in cerebral cavernous malformations

- ROCK is hyperactive in brain capillary endothelial cells in patients with CCM[1]
- Inhibiting ROCK2 seeks to repair the endothelial cell defect to reduce the permeability of the blood–brain barrier[2]
- Preclinical studies have shown ROCK2 inhibitors[3]
  - Reduce lesion size and genesis
  - Restore the barrier function of endothelial cells
  - Reverse hyperactivation of ROCK

ROCK2 inhibition appeared to reduce the lesion volume and lesion sites



*Micro-CT imaging of the brain of ccm1$^{+/-}$ mice
Dosed with: ROCK2 inhibitor BA-1049

In CCM transgenic mice, ROCK2 inhibitor reduced the leakiness of lesions, prevented the growth and formation of lesions, and slowed disease progression[3]

1. 1. Stockton RA et al. J Exp Med. 2010 Apr 12;207(4):881-96. doi: 10.1084/jem.20091258.
2. 2. Niego B, et al. PLoS One. 2017 May 16;12(5):e0177332. doi: 10.1371/journal.pone.0177332. PMID: 28510599; PMCID: PMC5433693.
3. 3. McKerracher L, et al. Stroke Res. 2020 Jun;11(3):365-376. doi: 10.1007/s12975-019-00725-8. Epub 2019 Aug 24. PMID: 31446620; PMCID: PMC7036327.

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OV888's potential as a potent, selective, best-in-class ROCK2 inhibitor

	ROCK2 IC_{50} (uM)	ROCK1 IC_{50} (uM)	Selectivity (ROCK1/ROCK2)	BBB Penetrant	Off-Target Effects	Indication
OV888 OVID/GRAVITON	**0.002-0.02**	**12.1**	**1071 fold**	✓		**CCM**
KD025[1] Kadmon Pharmaceuticals	0.105	24	~80 - 100 fold	×		Graft vs host disease
NRL-1049[2] (Neurellis)	0.24 – 0.73	3.9 – 10.2	13.9 – 16 fold	✓	✓	CCM

Chart based on biochemical assays; not head-to-head studies

Molecules with pan-ROCK inhibition have shown off-target effects

1 Ann Clin Transl Neurol. 2014 Jan; 1(1): 2–14. Published online 2013 Nov 19. doi: 10.1002/acn3.19
2 https://patents.google.com/patent/US10106525B2/ena

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OV888 (GV101): Phase 1 topline results

OVERVIEW

- OV888 (GV101) capsule formulation
- Double-blinded, placebo controlled
- Male/female healthy adult volunteers
- 7-day consecutive dosing:
  - MAD: 100 mg, 200 mg, 400 mg, and 600 mg
- Objective to measure:
  - Safety
  - Tolerability
  - Pharmacokinetics (PK)

✓ RESULTS

- ✓ Met objective: Well-tolerated at all tested doses
- ✓ Target engagement achieved
- ✓ Exposure & half-life supportive of once daily dosing
- ✓ Results:
  - No SAEs (0); no participants met preset stopping criteria
  - All AEs were mild and resolved
    - Most common reported AE was headache (23%); which was transient and resolved
  - Asymptomatic laboratory results rated grade 2 or higher in at least 5% of subjects:
    - Clinically insignificant increases in total bilirubin without direct hyperbilirubinemia, with no concurrent liver enzyme elevations, reported in 30% of participants;
    - Creatine phosphokinase elevations without muscle pain or weakness were reported in 7% of participants
    - All participants normalized during the study period

NEXT STEPS: Expect to initiate Phase 2 proof of concept study for the potential treatment of CCM in H2 2024

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Source: Ovid & Graviton data on file

Unmet need in CCM is substantial and no pharmacological therapies exist

Addressable CCM symptomatic population[1]

7 major markets

~400K

CCM patients[2]

United States

~150K-175K

CCM patients[2]

Disease modifying therapies are needed

- Symptoms from lesions can result in neurological deficits & potentially lead to severe disability
- Only available therapy is surgical resection of the CCM (when possible)
- Prior CCM hemorrhage carries significant risk of additional hemorrhage[3]

1. Addressable population is defined as symptomatic patients and patients with an incidental finding
2. Ovid 2022 global market research
3. Ma L et al. Stroke. 2020 Oct;51(10):2997-3006. doi: 10.1161/STROKEAHA.120.029942. Epub 2020 Sep 21. PMID: 32951540.

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OV329

A next-generation GABA-aminotransferase inhibitor for the potential treatment of resistant seizures

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GABA-aminotransferase inhibitor programs for potential treatment of chronic and acute refractory seizures

POTENTIAL OPPORTUNITY

- Unique molecule for a validated target, GABA aminotransferase (GABA-AT)
- Intended to deliver optimal levels of GABA to achieve:
  - Sustained seizure suppression
  - Improved safety
  - Preferable (lower) dosing
- 7 preclinical models demonstrate OV329's anti-convulsant activity
- Designed to supplant vigabatrin (VGB) which had a safety issue at the therapeutic index
- OV329 is believed to be >100x more potent than VGB in preclinical studies[1]

STATUS

2 formulations

- Oral – chronic refractory seizures
- Intravenous (IV) – acute refractory seizures

Multiple indication opportunities

Phase 1 SAD/MAD with biomarkers

- Anticipated results H2 2024

IV formulation

- Investigational new drug authorization anticipated to be submitted H2 2024

1. Ovid data on file, presented at Eilat conference in 2022

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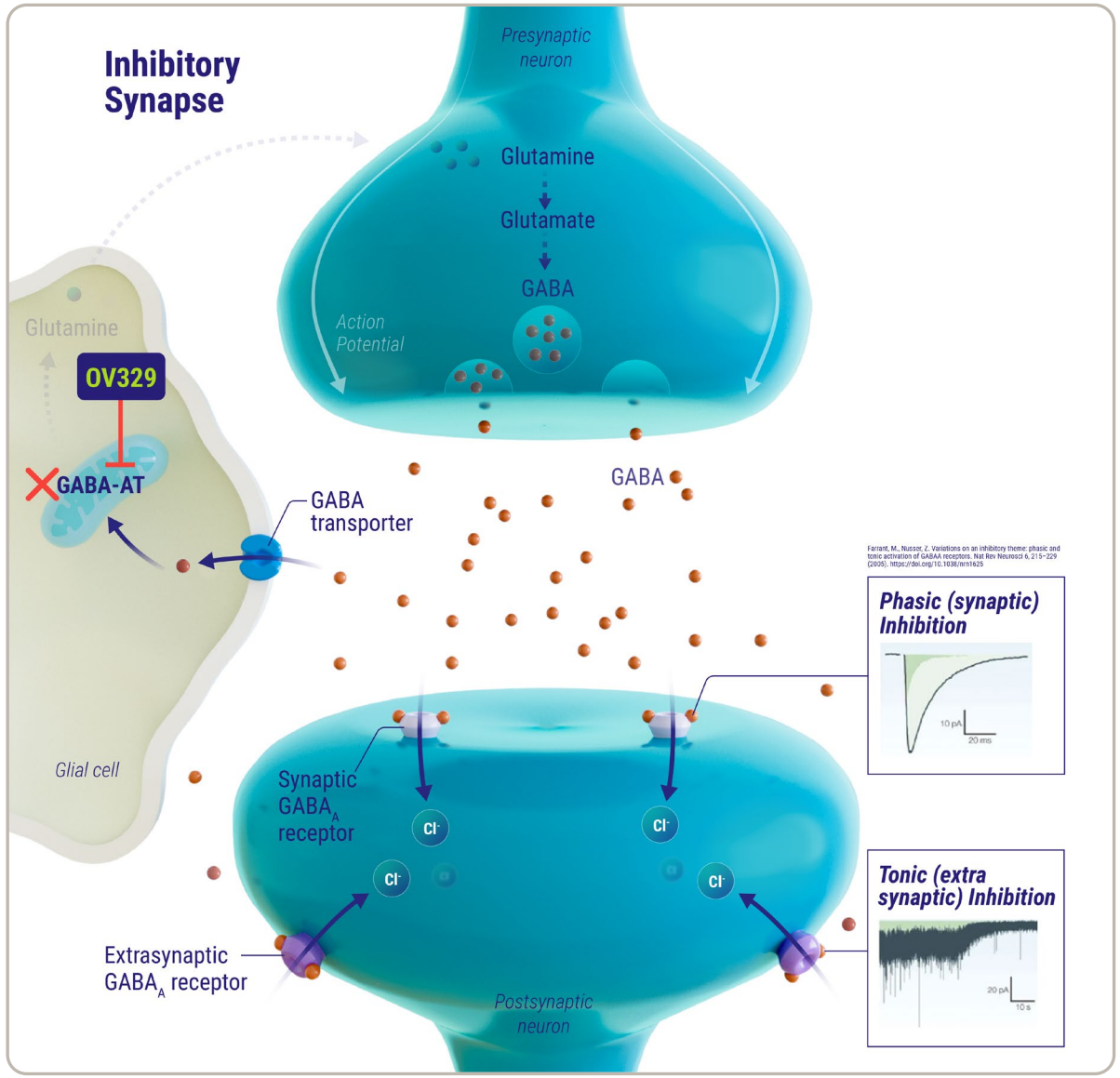
OV329 mechanism of action

- GABA is the major inhibitory neurotransmitter
- OV329 reduces GABA aminotransferase activity, a key enzyme responsible for degrading GABA[1]
  - Leading to the elevation of GABA levels
  - Curbing excessive neuronal excitation and seizures
- OV329 shows increases in phasic and tonic inhibitory neurotransmission at GABAergic synapses in preclinical models[2]
- Repeated low doses of OV329 demonstrate sustained seizure reduction and profound changes in tonic (extra-synaptic) inhibition in preclinical and animal models[3]



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. Feja, Malte 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 vol. 62,12 (2021): 3091-3104. doi:10.1111/epi.17090
3. Ovid Data presented at AES 2023

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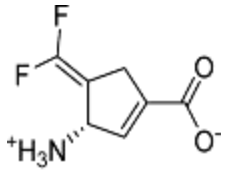
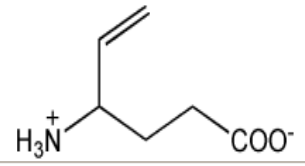
OV329 profile is differentiated from vigabatrin

	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 (more sophisticated chemistry, primarily enamine pathway) [2]	Covalent modification of GABA-AT (early generation chemistry, primarily Michael addition pathway) [1]
Purity	Only active, (S) enatiomer	Mixture of active (S) and inactive (R) enantiomers
Therapeutic window in Sprague Dawley rats (a proxy model used to assess ocular safety)	✓	✗ None – toxicity seen at therapeutic dose of 300 mg/kg

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

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Repeated low doses of OV329 showed a reduction in seizure frequency & duration in animal models

Mesial temporal lobe epilepsy (MTLE) model reflects:

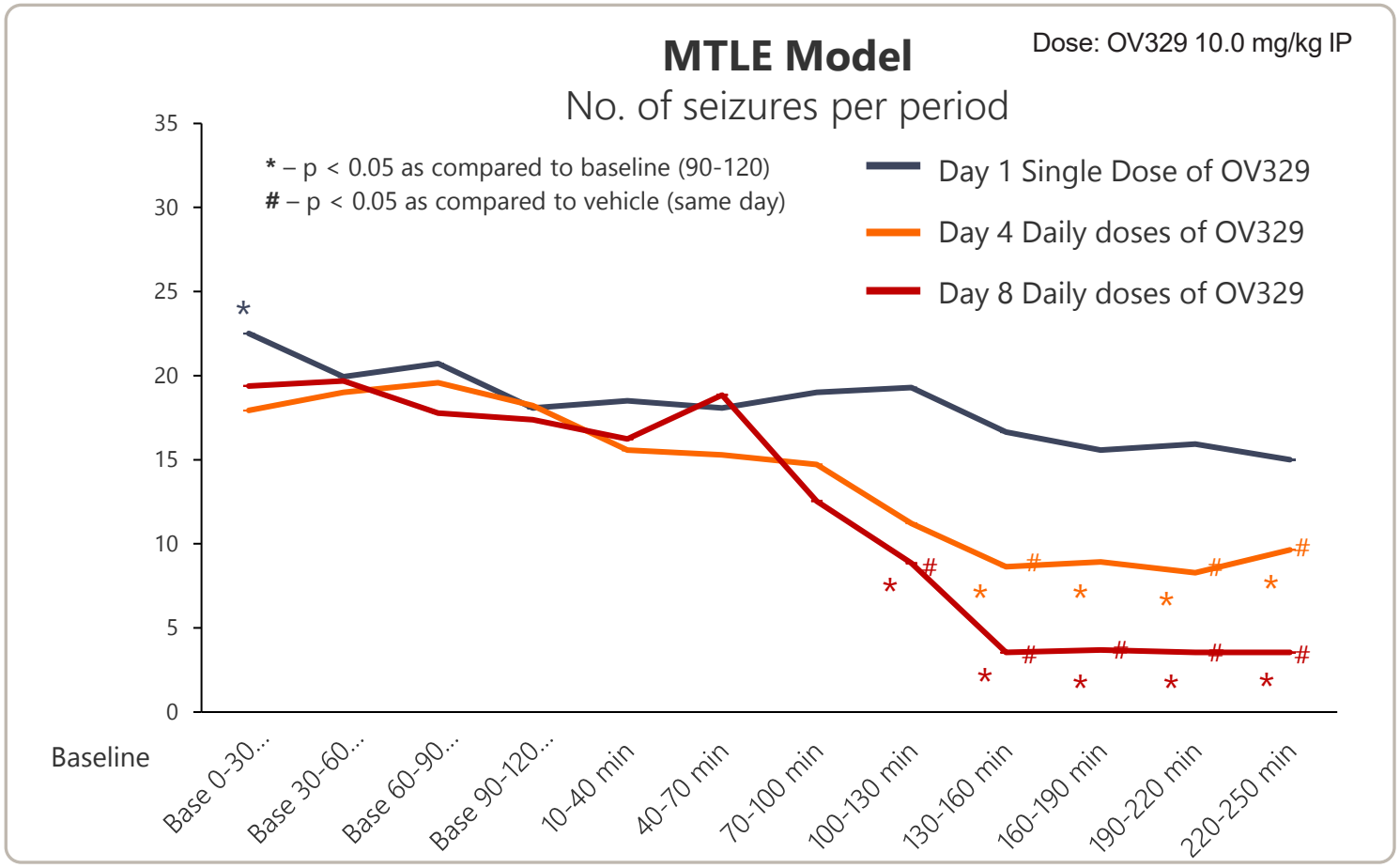
- The main pharmaco-resistant form of epilepsy[1]

Repeat dosing leads to improved efficacy of seizure reduction:

- Seizure numbers
- Cumulative duration of focal seizures

6 other seizure models demonstrate OV329 anti-convulsant activity

- NMDA-Induced Infantile Spasm Model
- Corneal kindled
- Audiogenic seizure
- Amygdala kindled
- i.v. Pentylenetetrazol
- Kainic acid model



Supports potential chronic dosing for refractory seizures

1. Venceslas D, Corinne R. A Mesiotemporal Lobe Epilepsy Mouse Model. Neurochem Res. 2017 Jul;42(7):1919-1925. doi: 10.1007/s11064-017-2239-3. Epub 2017 Mar 23. PMID: 28332054.

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Multiple pre-clinical models in resistant seizures confirm OV329's anti-convulsant profile

	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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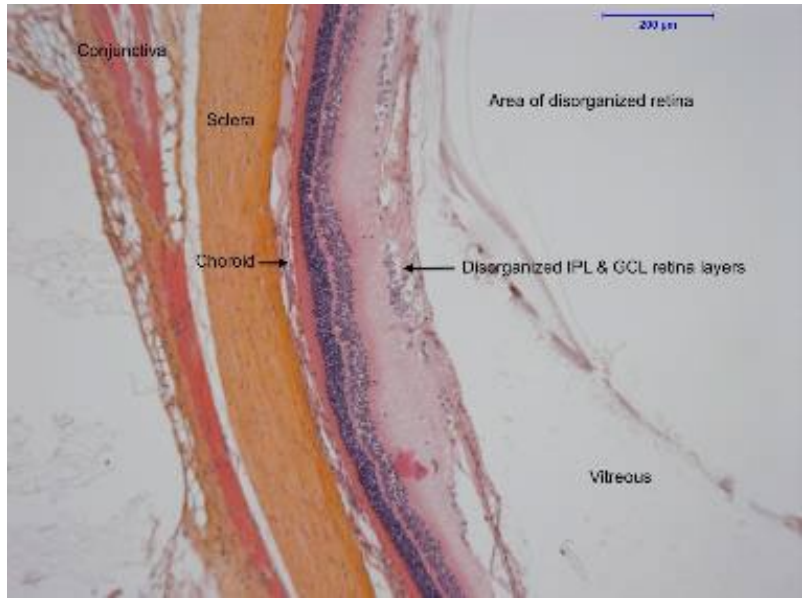
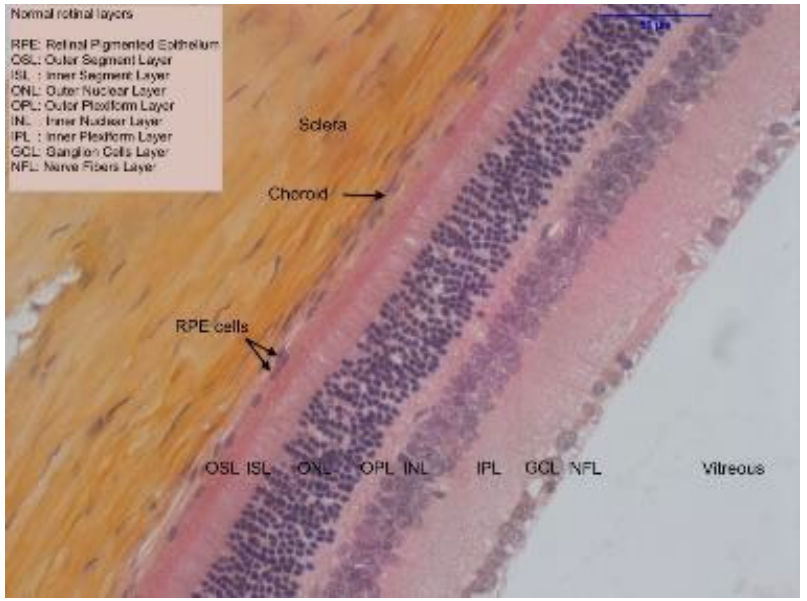
No ocular effects seen following chronic exposure with OV329 (3 mg/kg)

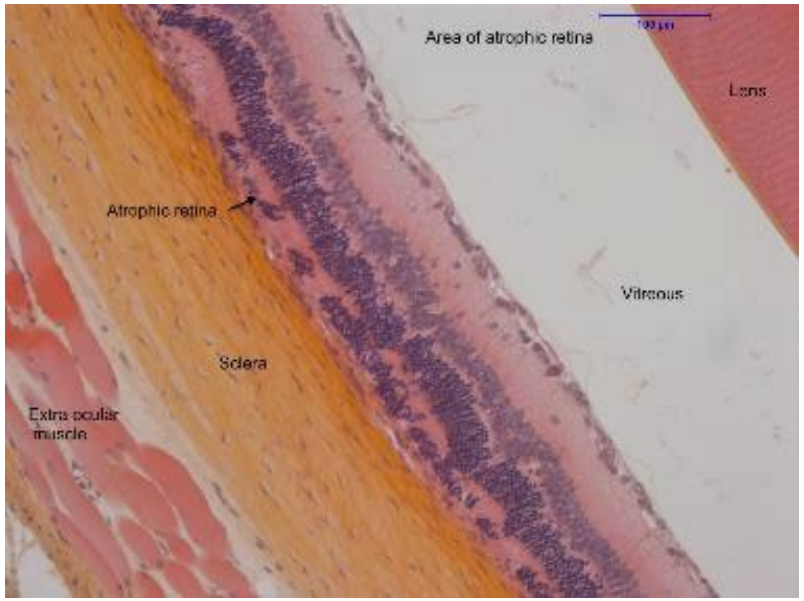
Vehicle



OV329 3.0 mg/kg



Vigabatrin 300 mg/kg



No ocular effects seen in 3 mg/kg OV329, q.d. and vehicle groups in rats at Day 45

Ocular effects seen in more than half of rats treated with Vigabatrin (300 mg/kg)

OVID, Data on file

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OV329 Development program

Oral – Chronic Dosing

- 3 SAD cohorts completed in healthy volunteers
  - MAD ongoing
- Two biomarkers included:
  - Transcranial magnetic stimulation (TMS)
  - Magnetic resonance spectrometry
- Conducting animal back-of-eye accumulation study to differentiate OV329 safety from vigabatrin
- Target readout late H2 2024

IV – Acute Dosing

- IV formulation created
- Anticipated IND submission in H2 2024
- Intent to initiate a Phase 1 trial in 2025

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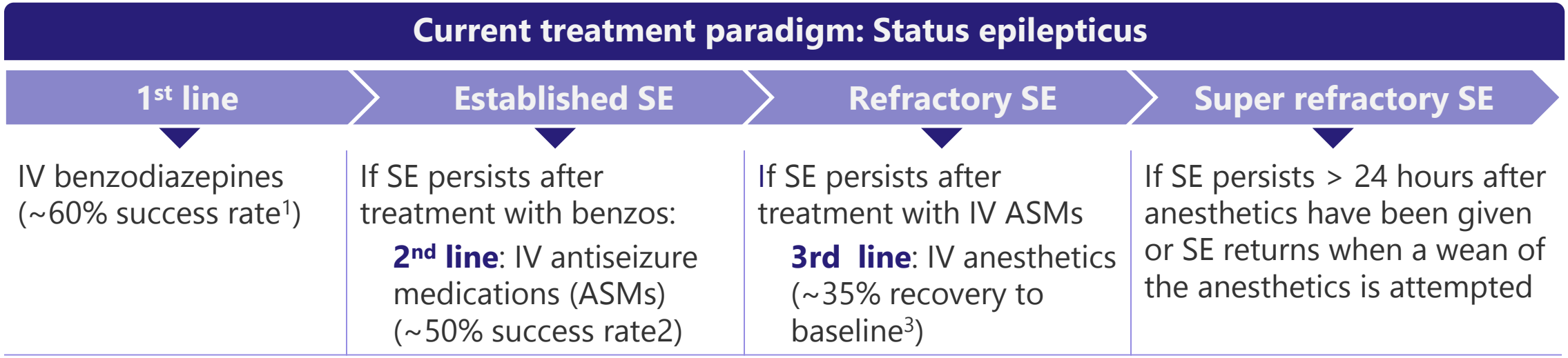
Significant unmet need & mortality in status epilepticus (SE)

SE defined as a seizure of 5+ minutes or more than 1 seizure in a 5-minute period, without a return to normal level of consciousness between episodes



1. Treiman et al., A comparison of four treatments for generalized convulsive status epilepticus. N Engl J Med. 1998 Sep 17;339(12):792-8. doi: 10.1056/NEJM199809173391202. PMID: 9738086.
2. Kapur et al.. Randomized Trial of Three Anticonvulsant Medications for Status Epilepticus. N Engl J Med. 2019 Nov 28;381(22):2103-2113. doi: 10.1056/NEJMoa1905795. PMID: 31774955; PMCID: PMC7098487.
3. Ferlisi M, Shorvon S. The outcome of therapies in refractory and super-refractory convulsive status epilepticus and recommendations for therapy. Brain. 2012 Aug;135(Pt 8):2314-28. doi: 10.1093/brain/aws091. Epub 2012 May 9. Erratum in: Brain. 2013 Jul;136(Pt 7):2326. PMID: 22577217.

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KCC2 library & OV350

Potential first-in-class direct activators of the KCC2 transporter

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KCC2 direct activator portfolio & OV350 for seizure and psychosis indications

POTENTIAL OPPORTUNITY

Unique library of direct activators of potassium chloride co-transporter 2 (KCC2)

Activators directly modulate intrinsic hyperexcitability of neurons via chloride extrusion[1]

Animal disease models have suggested:

- Anti-convulsant properties
- Anti-psychotic and anti-schizophrenic properties
- No sedative effects at expected therapeutic windows

Represents a potential franchise of neurotherapeutics

STATUS

1st IND from franchise expected to be submitted in H2 2024 (OV350) in psychiatric indication

Characterizing & formulating broad library of ~100 compounds

- Amenable to intravenous, oral and injectable formulations

1. Kahle KT, et al JAMA Neurol. 2014 May;71(5):640-5. doi: 10.1001/jamaneurol.2014.21. PMID: 24615367; PMCID: PMC4465580.

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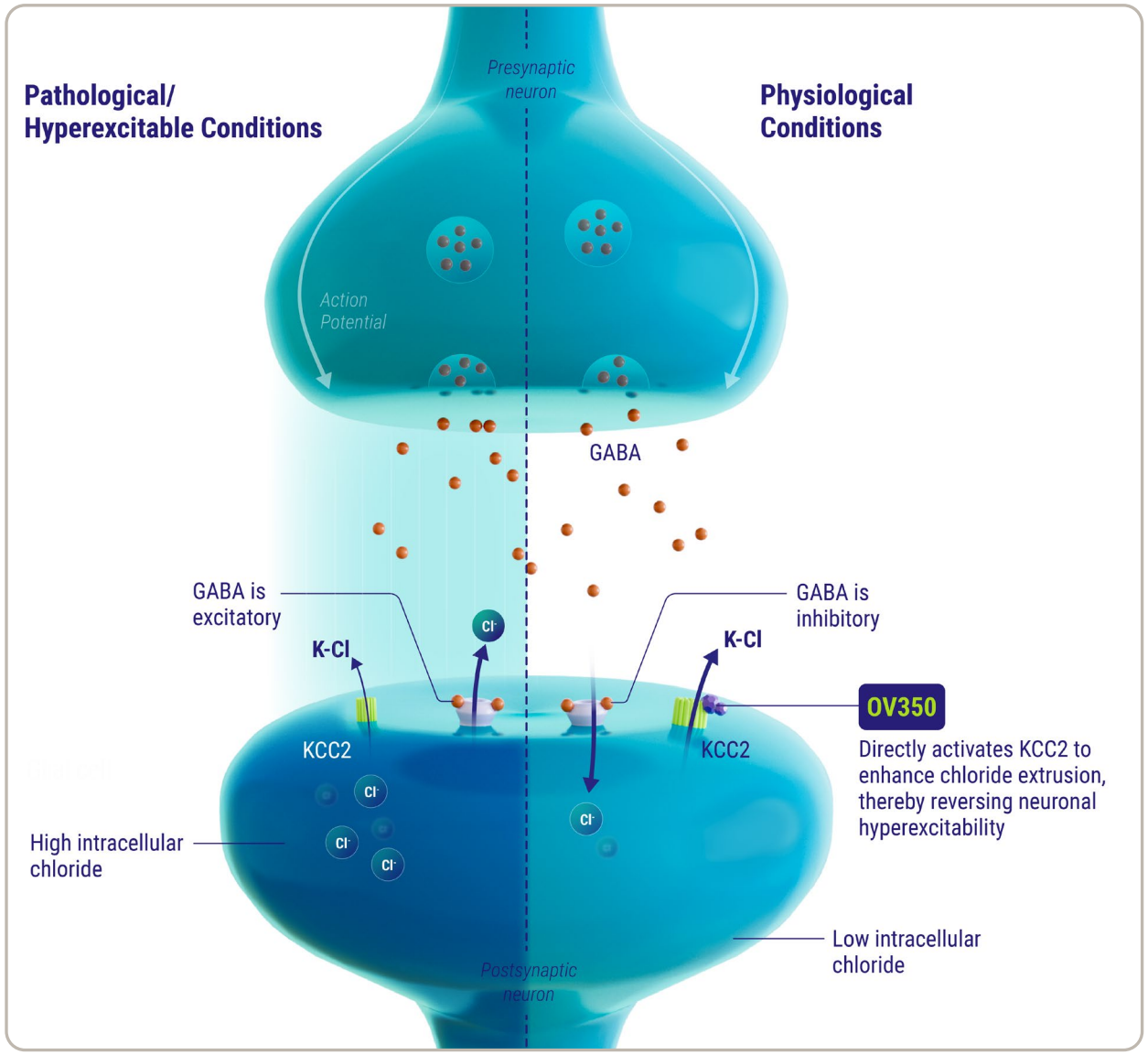
KCC2 is the main regulator of GABA inhibition by maintaining neuronal chloride homeostasis[1]

- Deficits in KCC2 activity have been linked to a variety of neurological disorders[2]
- OV350 shows direct activation of KCC2 transporter, improving:
  - Chloride extrusion
  - Neuronal inhibition in preclinical models



1. Mahadevan V, Woodin MA. J Physiol. 2016 May 15;594(10):2593-605. doi: 10.1113/JP271593. Epub 2016 Mar 31. PMID: 26876607; PMCID: PMC4865579.
2. Tomita K, et al. Jpn Dent Sci Rev. 2023 Dec;59:431-438. doi: 10.1016/j.jdsr.2023.11.001. Epub 2023 Nov 11. PMID: 38022385; PMCID: PMC10665825. 24:7, 629-637, DOI: 10.1080/14728222.2020.1762174

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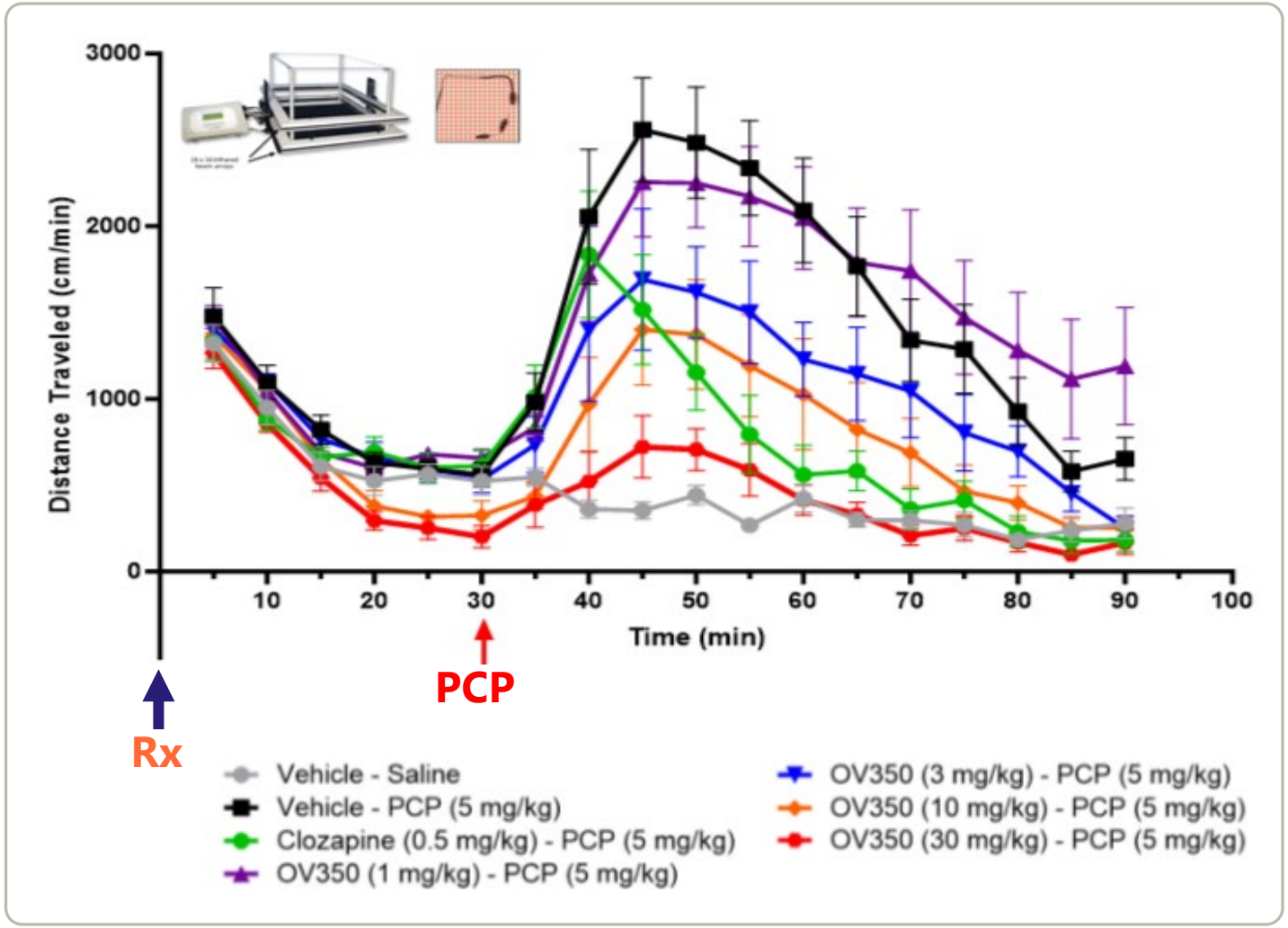
OV350 demonstrates anti-psychotic effects in a schizophrenia model



- Phencyclidine-induced psychosis (PCP) is characterized by:
  - Confusion, excitation, aggression, paranoia, hallucinations, and can be experimentally measured by hyperlocomotion
- OV350 demonstrated dose dependent responses and inhibited PCP induced hyperlocomotion
- OV350 appears to have anxiolytic effects without causing sedation

OVID data on file

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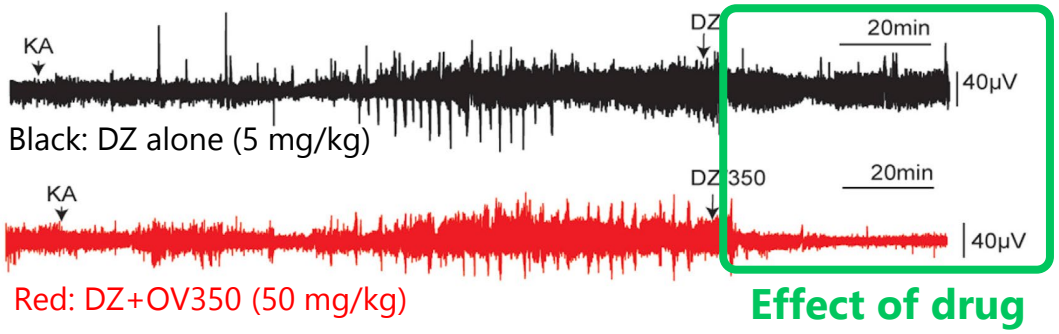
Preclinical POC for OV350 in epilepsy

OV350 potentially terminates ongoing and benzodiazepine refractory status epilepticus

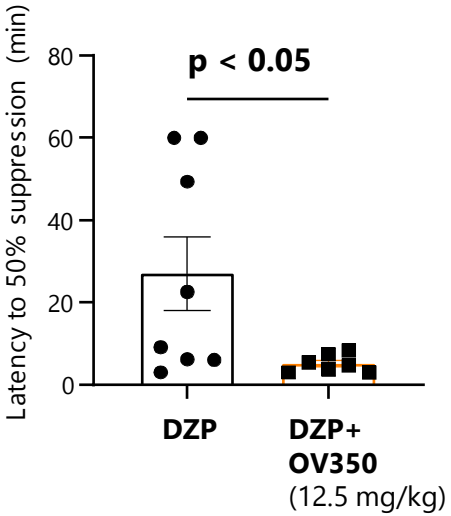
- OV350 demonstrated anti-seizure activities in kainate challenge model
- Mice implanted with electrodes to measure kainate induced seizures
- Demonstrated that OV350 at 50mg/kg and 12.5mg/kg
  - Restored diazepam activities in stopping seizures
- Supports development in Status Epilepticus using IV formulation of OV350/benzodiazepine combination

Induction of Status Epilepticus (SE)

EEG traces show SE cessation after OV350 intervention with DZ



Efficacy Confirmed at a Lower Dose



OV350 restores the activity of Diazepam in drug-refractory seizures

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Thank you

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