Full Year 2023 Results & Strategic Update Webcast and Conference Call

April 24, 2024

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AGENDA

01 INTRODUCTION

02 VAROGLUTAMSTAT IN ALZHEIMER'S DISEASE

03 VAROGLUTAMSTAT IN KIDNEY / FIBROTIC & INFLAMMATORY DISEASE

04 2023 FINANCIALS

05 OUTLOOK & STRATEGIC PRIORITIES

06 Q&A

Prioritizing resources to maximize value from varoglutamstat & pipeline following negative topline results from VIVIAD study in early AD

2024 – Significant post-period events

- **VIVIAD in early AD** – topline data reported in March: study missed primary & secondary endpoints; discontinuing VIVA-MIND study in H2 2024; stopping VIVALONG study preparations
- **VIVIAD kidney analysis** – revealed statistically significant effect on prospectively defined kidney function endpoint (eGFR[1])
- **Board changes** – New CFO A. Doering; K. Sathiyanandarajah & M. A. Karsdal stepping down as Non-Executive Directors

2023 – Key highlights

- Continued varoglutamstat development as planned (VIVIAD progressed; VIVA-MIND expanded)
- Secured additional capital (EUR 25 million)
- Transitioned and expanded executive team
- Unveiled growth strategy beyond Alzheimer's disease (AD)

Shift in strategic focus towards inflammatory and fibrotic disorders, from AD

- Explore varoglutamstat's potential in diseases affecting kidney function[2]
- Continue to explore QPCT/L inhibitors in inflammatory/fibrotic diseases

- Assess potential of a higher dose of varoglutamstat in early AD[2]

- Selectively assess early pipeline opportunities
- Actively pursue funding and business development opportunities
- Prudent cash runway management; current cash runway to now extend into Q2 2025

[1] Estimated glomerular filtration rate. [2] Additional clinical studies would require additional funding and/or partnership.

Detailed VIVIAD analysis suggests no consistent effect of varoglutamstat up to 600mg BID in early AD, but a significant effect on kidney function at 600mg BID

Early AD

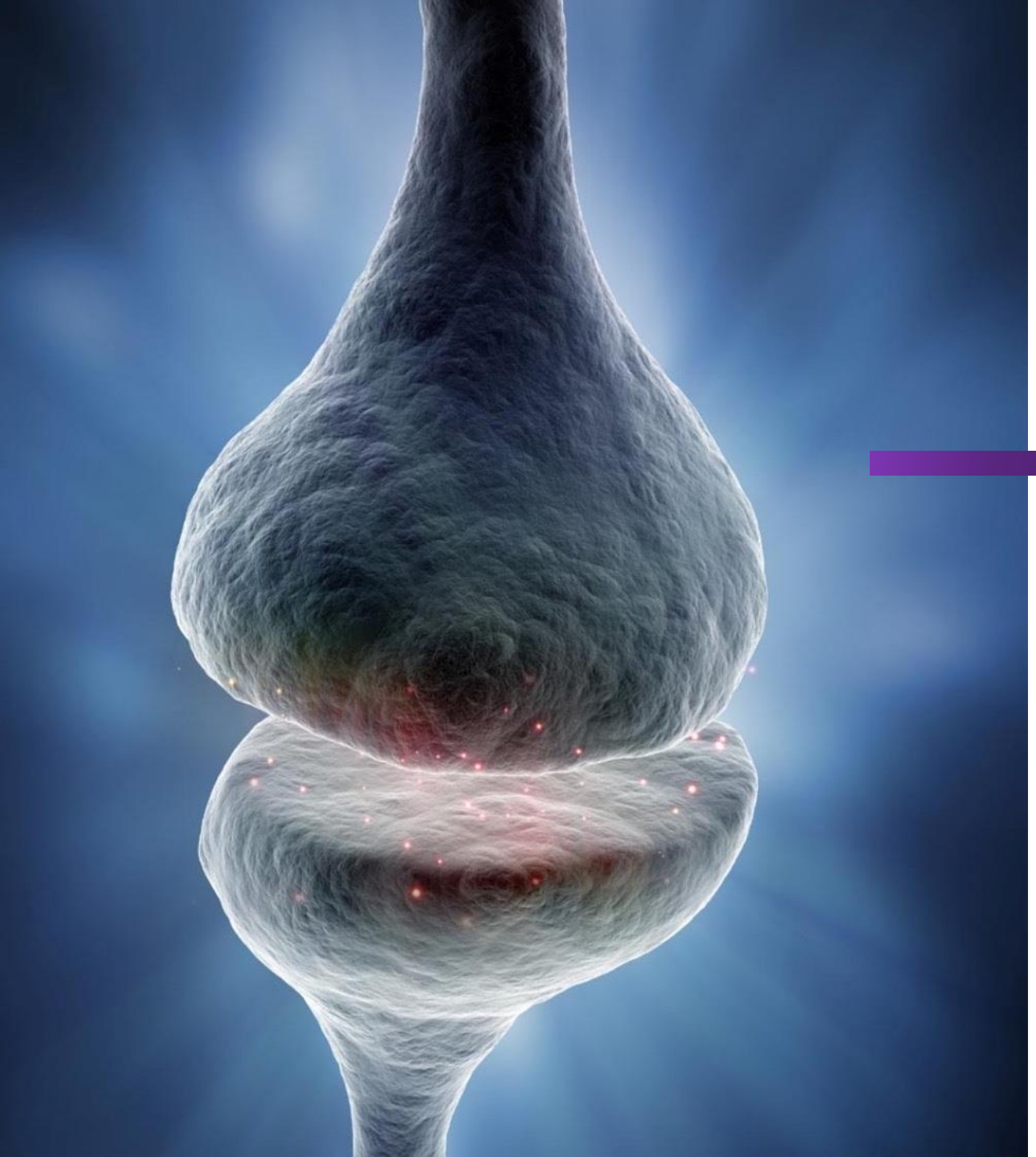
- No statistical effect of varoglutamstat up to 600mg BID on primary or secondary endpoints in early AD
- Statistically significant difference observed for varoglutamstat on exploratory endpoints for executive function, letter fluency test, and WAIS IV coding, at week 48
- Well-tolerated with low discontinuation rates due to adverse events and no evidence of symptomatic ARIAs in clinical setting

Kidney function

- Statistically significant improvement of kidney eGFR from baseline with varoglutamstat 600mg BID
- Treatment effect sustained for up to two years
- Effect size versus placebo was stronger in patients with risk factors for kidney disease

- Evaluating if dose of varoglutamstat 600mg BID was potentially too low to have a beneficial effect on early AD
- Assess potential of varoglutamstat in doses higher than 600mg BID in early AD

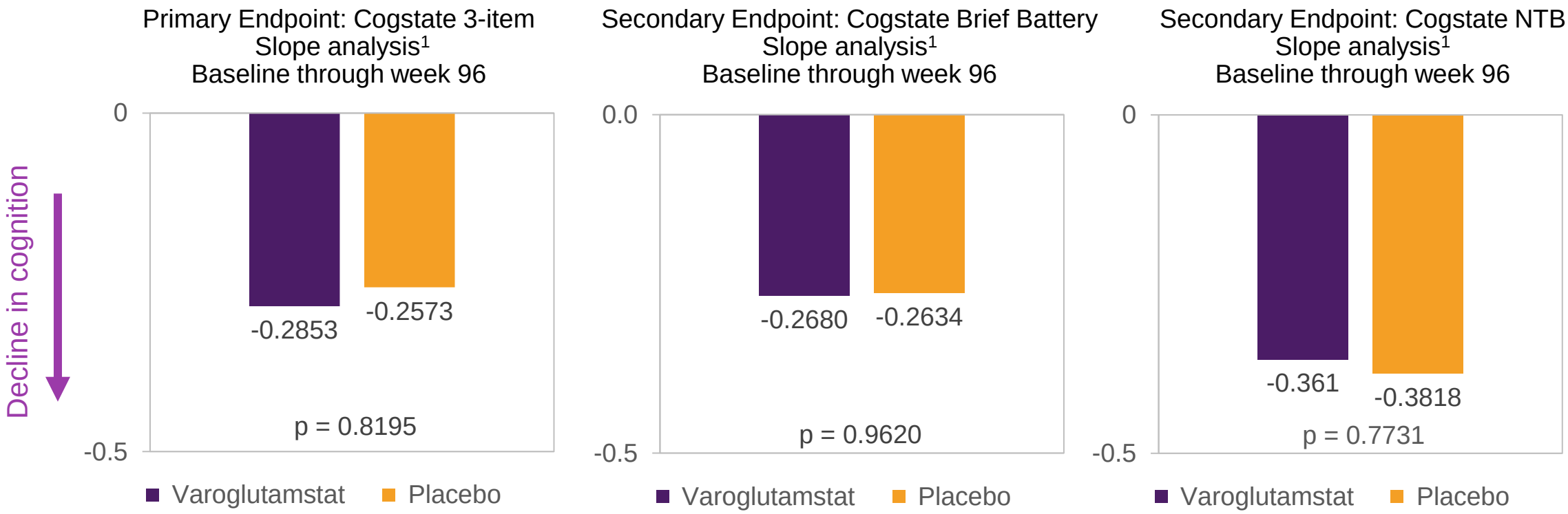
- Supports mechanism of action of QPCT/L inhibition
- Demonstrates varoglutamstat is a potent and well tolerated inhibitor of the QPCT/L enzyme family



VAROGLUTAMSTAT IN ALZHEIMER'S DISEASE (AD)

VIVIAD PHASE 2B – INITIAL DATASETS

VIVIAD did not demonstrate a statistically significant change in cognition with varoglutamstat up to 600mg BID compared to placebo

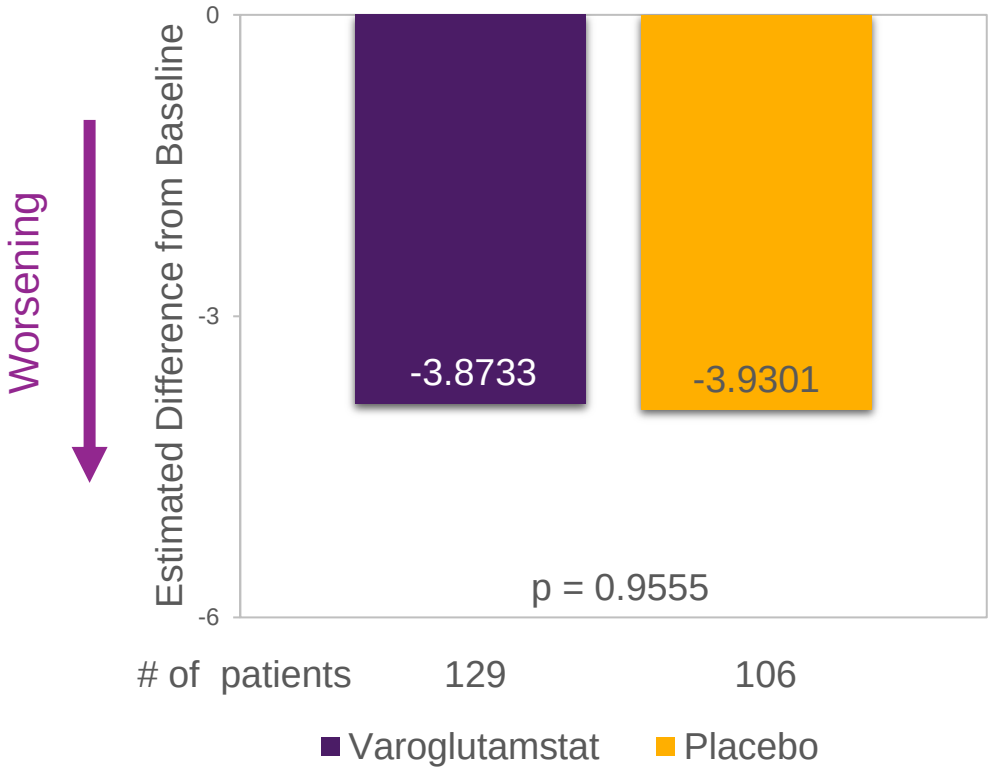


Progressive decline in cognition from baseline for treatment and placebo arms was statistically significant for all 3 endpoints (p<= 0.003) demonstrating sensitivity of the endpoints and confirming patient selection

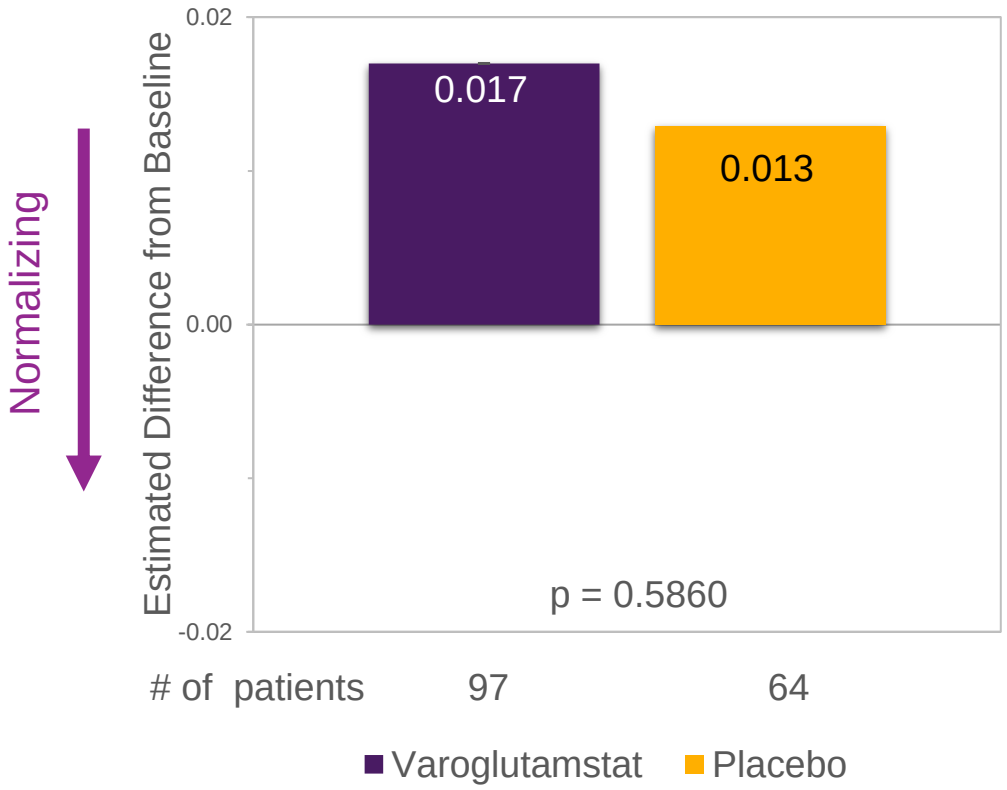
[1]Change in Z-score through week 96, estimates by MMRM model. Cogstate NTB (neuropsychological test battery): Working memory & attention combined score, primary endpoint (3-item scale: Identification, Detection, One Back), Cogstate Brief Battery, secondary endpoint (CBB, 4-item scale: 3-item scale plus One Card Learning), Complete Cogstate NTB, secondary endpoint (8-item scale)

VIVIAD did not demonstrate a statistically significant effect on activities of daily living and EEG theta power of varoglutamstat up to 600mg BID

A-IADL-Q Scores
Change from Baseline at week 48



EEG Global Relative Theta Power (4-8 Hz)
Change from Baseline at week 48



A-IADL-Q =Amsterdam-IADL-Questionnaire

VIVIAD analysis reveals no consistent effect of varoglutamstat at 300mg and 600mg BID on most pre-specified and exploratory AD endpoints

Pre-specified subgroup analysis

- No clinically meaningful and no statistically significant differences between varoglutamstat 300mg/600mg BID and placebo for
  - MMSE baseline severity
  - APOE4 status
  - pTau baseline concentration in the CSF
  - AD treatment naive versus concomitant AD treatment
  - Functional impairment at baseline

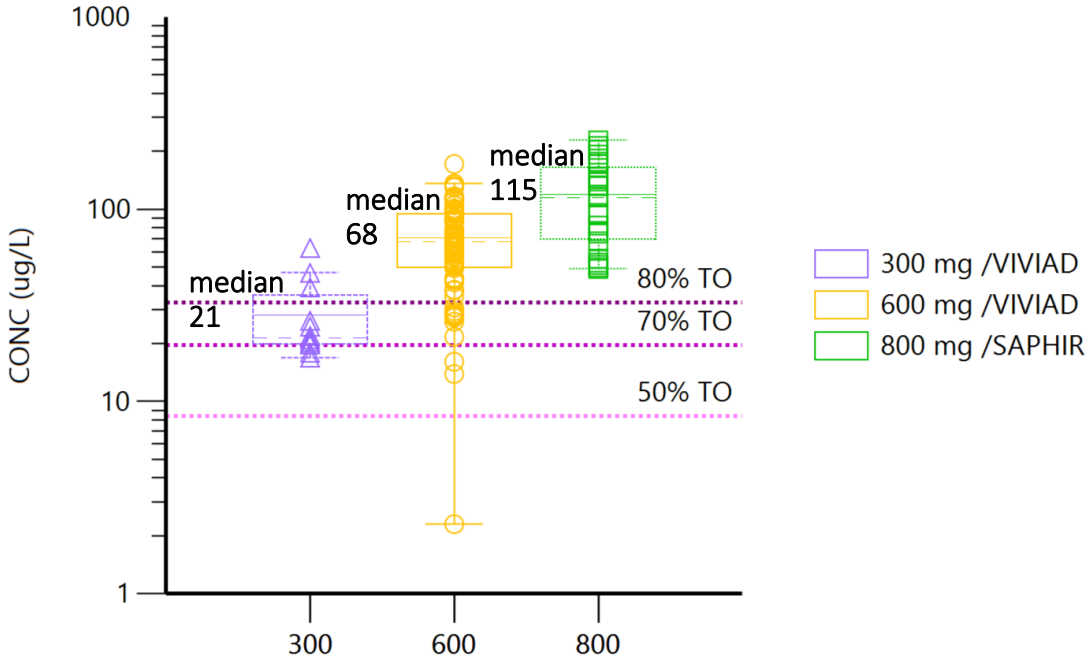
Exploratory endpoints

- No clinically meaningful and no statistically significant changes between varoglutamstat up to 600mg BID and placebo for any cognitive domain*
- No statistically significant changes on any EEG or CSF biomarker endpoints including pTau, YKL-40 and neurogranin
- Significant difference in favor of varoglutamstat up to 600mg BID in WAIS-IV coding test (p=0.0257) and letter fluency test (p=0.0138) at week 48

*WAIS = Wechsler Adult Intelligence Scale; *episodic memory, executive function, working memory*

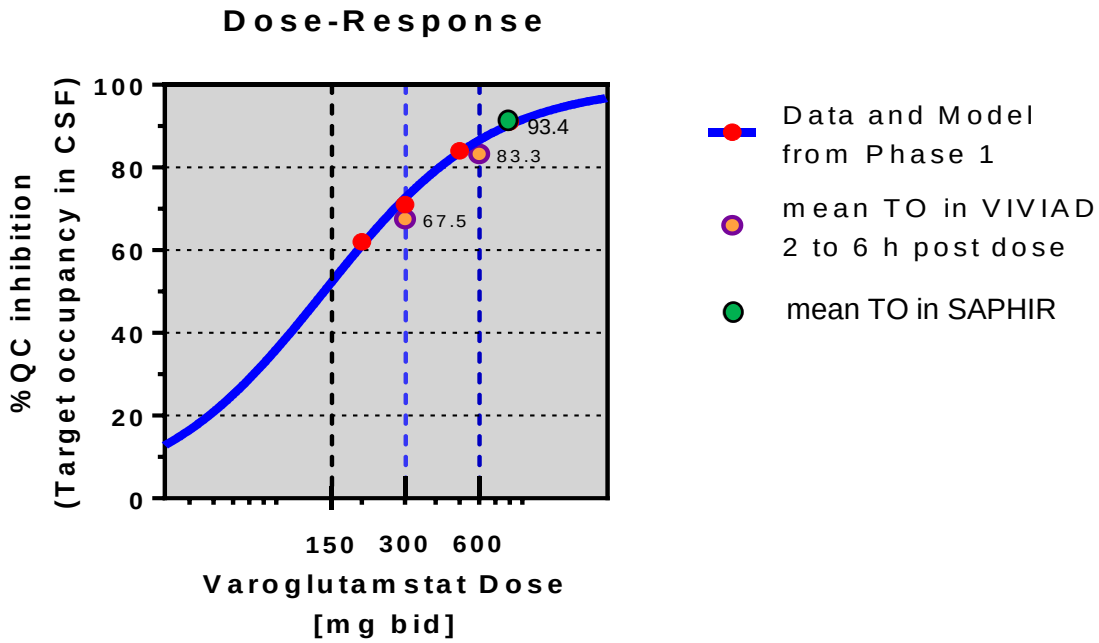
Drug concentration and QPCT/L (QC) inhibition measured in VIVIAD match previously published results

Varoglutamstat concentrations measured in CSF



VIVIAD @ week 48, SAPHIR@ week 12, Reference lines: estimated target occupancy (TO in %)in CSF

Target Occupancy (TO) in CSF



Dose-Response Curve derived from FIM data

CSF = cerebrospinal fluid; QC = glutaminyl cyclase

VIVIAD key safety data show varoglutamstat up to 600mg BID is well tolerated

- Rates of discontinuation in treatment group similar to placebo
- No difference between groups for treatment emergent adverse events
- Most common TEAEs are: COVID-19, diarrhea, dementia Alzheimer's type, headache, arthralgia*

Item	Varoglutamstat N (%)[1]	Placebo N (%)[1]	Total N (%)[1]
Patients randomized	142	117	259
Subjects who completed treatment	119 (83.8)	105 (89.7)	224 (86.5)
Subjects discontinued from treatment	23 (16.2)	12 (10.3)	35 (13.5)
- due to adverse events	6	4	10
- due to protocol deviation	1	0	1
- due to withdrawal	15	7	22
- due to physician decision	0	1	1
- other	1	0	1
Subjects with treatment emergent adverse events (TEAEs)			
- any TEAE	120 (84.5)	95 (81.2)	215 (83.0)
- any related TEAE	31 (21.8)	26 (22.2)	57 (22.0)
- serious TEAE	18 (12.7)	10 (8.5)	28 (10.8)
- serious related TEAE	2 (1.4)	0	2 (0.8)
- severe TEAE[2]	22 (15.5)	9 (7.7)	31 (12.0)
- severe related TEAE[2]	4 (2.8)	0	4 (1.5)
- fatal TEAE	0	0	0
Clinically diagnosed ARIA	0	0	0

[1] N=Number of patients, %=percentage of patients;
*[2] Severe treatment emergent AEs : with Grade >= 3 according to CTCAE; *most common TEAEs based on varoglutamstat 600mg BID*

VIVIAD data to inform future potential development of varoglutamstat in early AD at higher doses

VIVIAD key learnings early AD

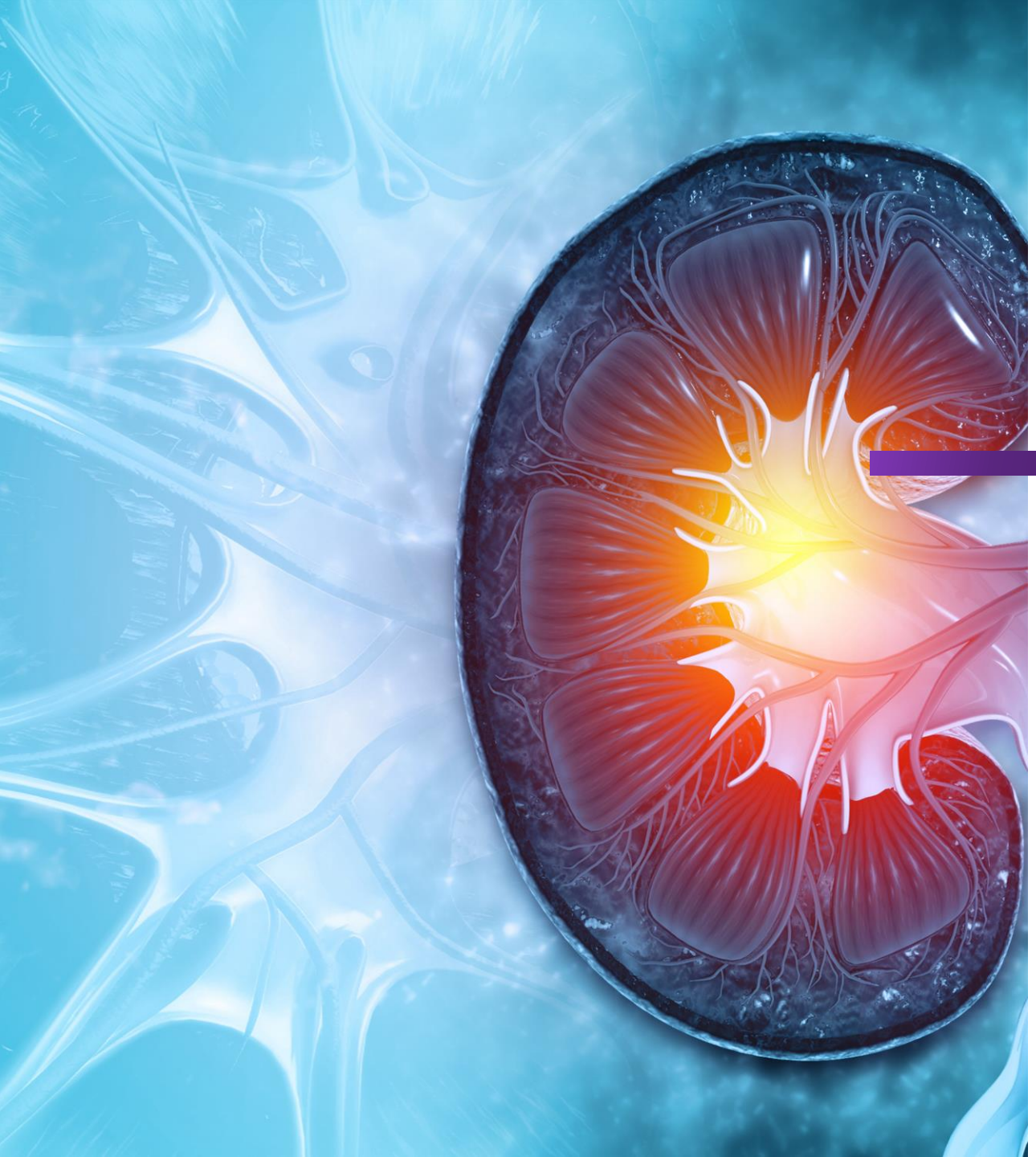
- Dose of varoglutamstat up to 600mg BID not effective in treatment of early AD
- PK and QPCT/L inhibition in plasma and CSF show that varoglutamstat is a potent inhibitor of QPCT and QPCTL
- Varoglutamstat at dose of 600mg BID was well tolerated
- Slower uptitration led to improved tolerability while maintaining favorable safety profile

Looking ahead in early AD

- Discontinue VIVA-MIND study in H2 2024; initial results with varoglutamstat at 600mg BID anticipated by end of 2024
- Integrate Phase 2 learnings from SAPHIR, VIVIAD and VIVA-MIND to further understand dose response with aim of refocusing development on higher doses
- Continue to investigate the science behind QPCT/L pathways

Key focus on assessing the potential of a higher dose of varoglutamstat in early AD*

Additional clinical studies would require further funding and/or partnership.



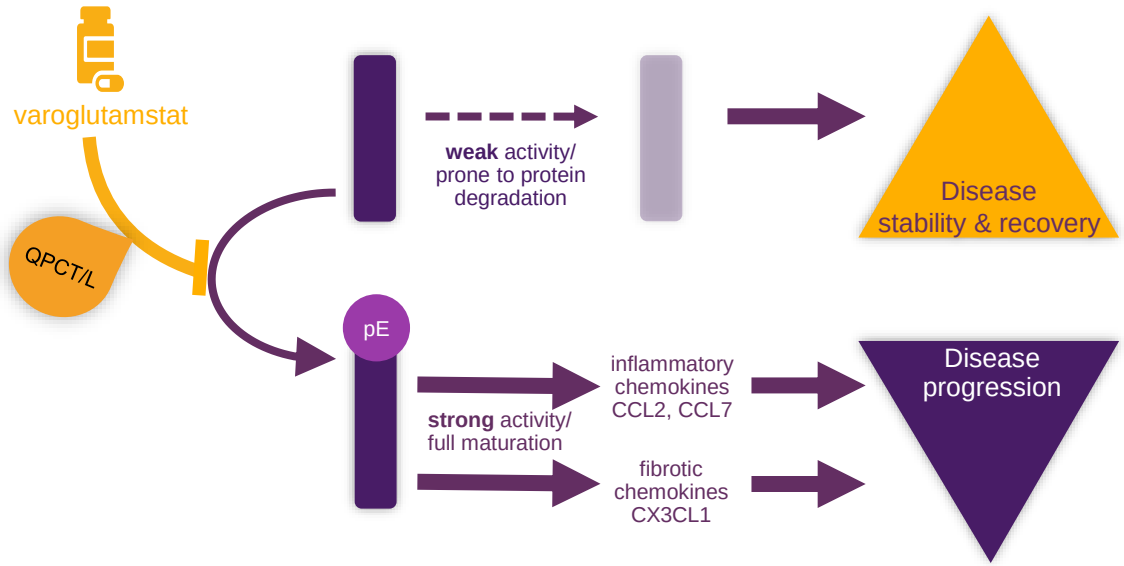
VAROGLUTAMSTAT EFFECT ON KIDNEY FUNCTION

INITIAL DATA FROM VIVIAD

VIVIAD exploratory endpoint analyzing kidney function founded in scientific rationale

- Many kidney diseases are driven by inflammatory and fibrotic processes induced by a variety of stimuli including metabolic, vascular and autoimmune dysfunctions
- Many inflammatory and fibrotic pathways require formation of N-terminal pyro-glutamates (pE) for full activity
- pE versions of chemokines like CCL-2 and CX3CL1 (fractalkine) may contribute to renal diseases[1,2]
- QPCT/L inhibition has been shown to improve kidney function and reduce inflammation in glomerulonephritis CKD rat model via CCL2/CCR2 axis, using a Vivoryon compound[3]
- Research indicates a pathogenic role for the CX3CL1-CX3CR1 axis in acute and chronic renal diseases[4]

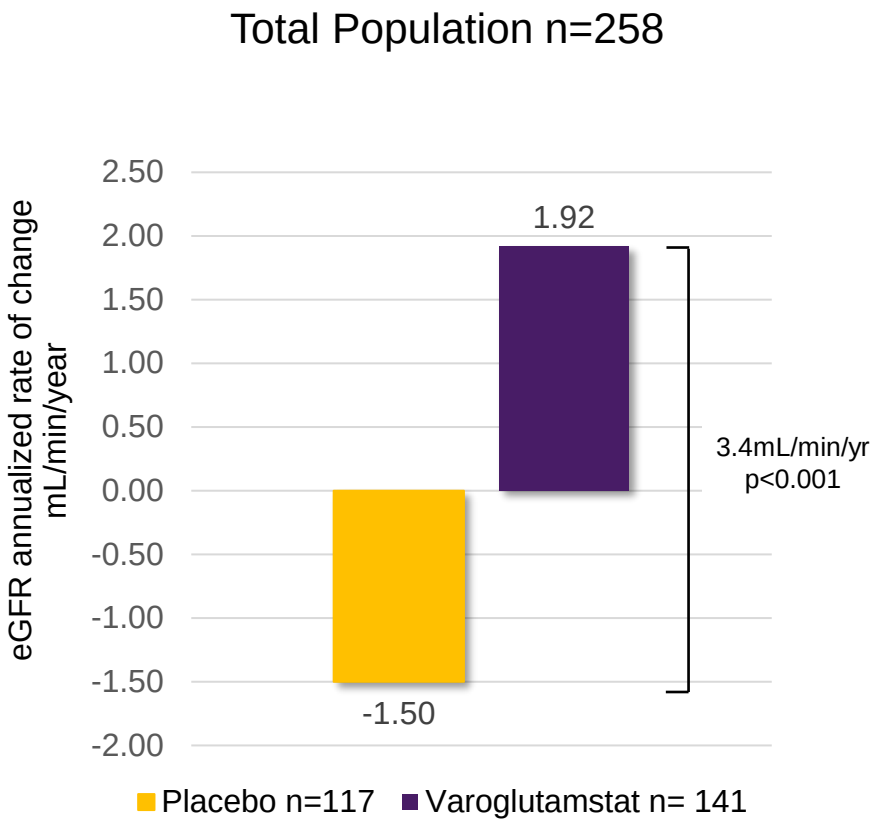
Varoglutamstat: Modulation of fibrotic and inflammatory processes on multiple layers

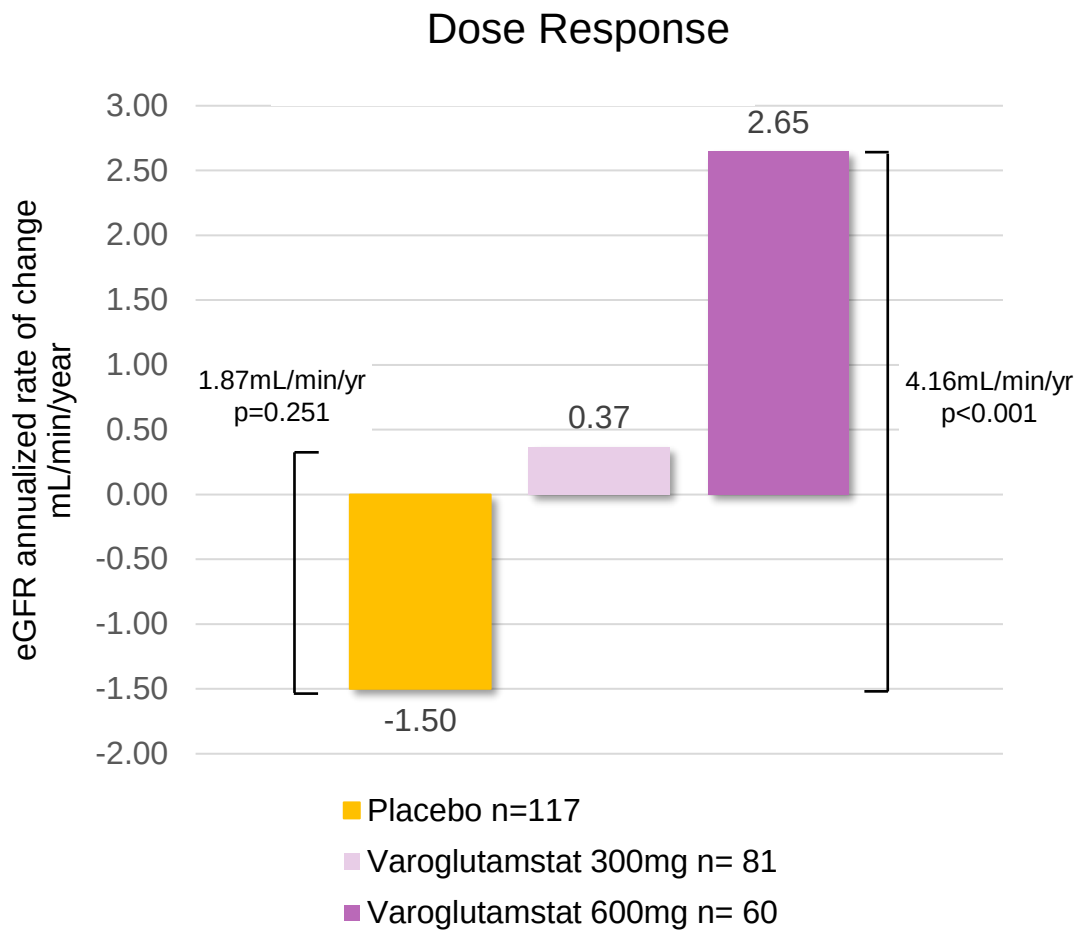


- VIVIAD protocol prospectively specified investigation of kidney function and measurement of biomarkers of kidney inflammation and fibrosis to explore the role of QPCT/L inhibition on kidney function
- While patients in VIVIAD were not selected for kidney function level, many of them have reduced kidney function due to age and or comorbidities like type 2 diabetes or hypertension

[1]Tesch GH, Am J Renal Physiol 2008; [2]Kehlen et.al , Biosci Rep, 2017; [3]Kanemitsu et.al., Naunyn-Schmiedeberg's Arch. of Pharmac., 2021; [4]Cormican S. et.al., Frontiers in Immu., 2021

eGFR slope analysis across 96 weeks in VIVIAD show statistically significant and clinically meaningful improvement for varoglutamstat 600mg BID

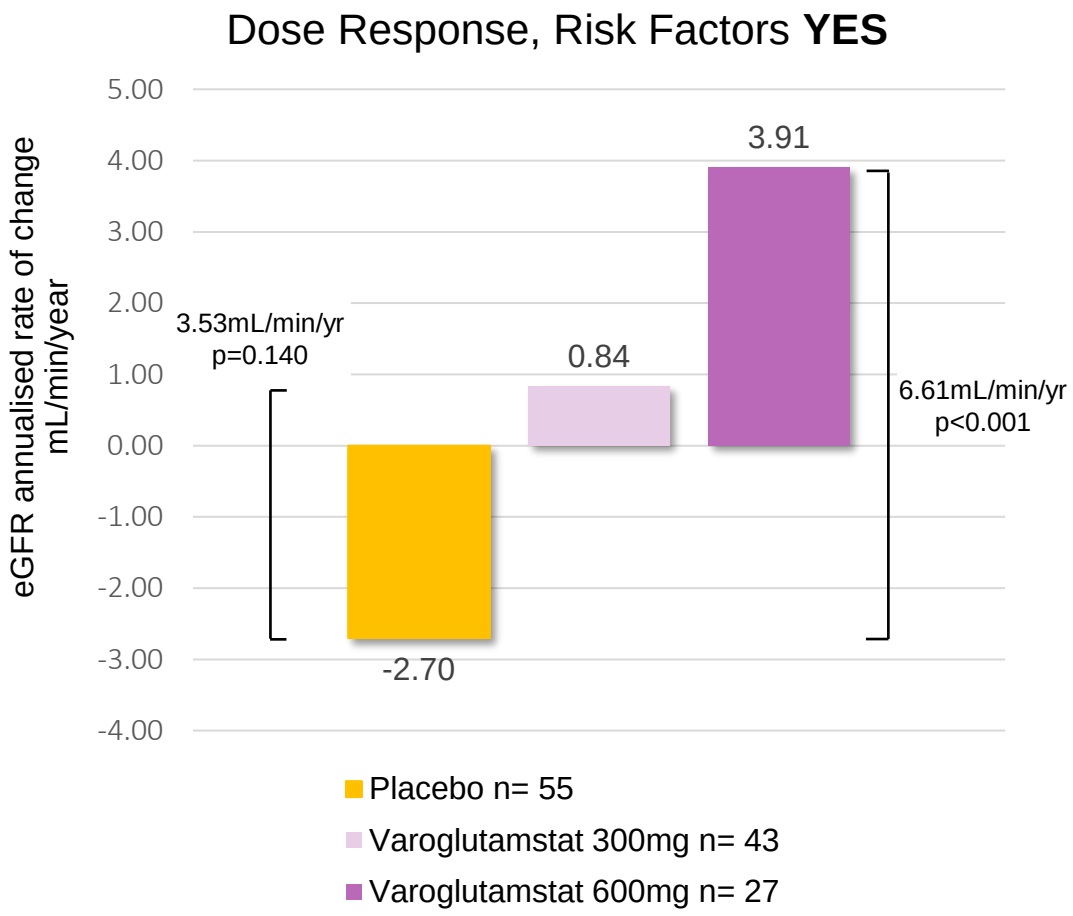




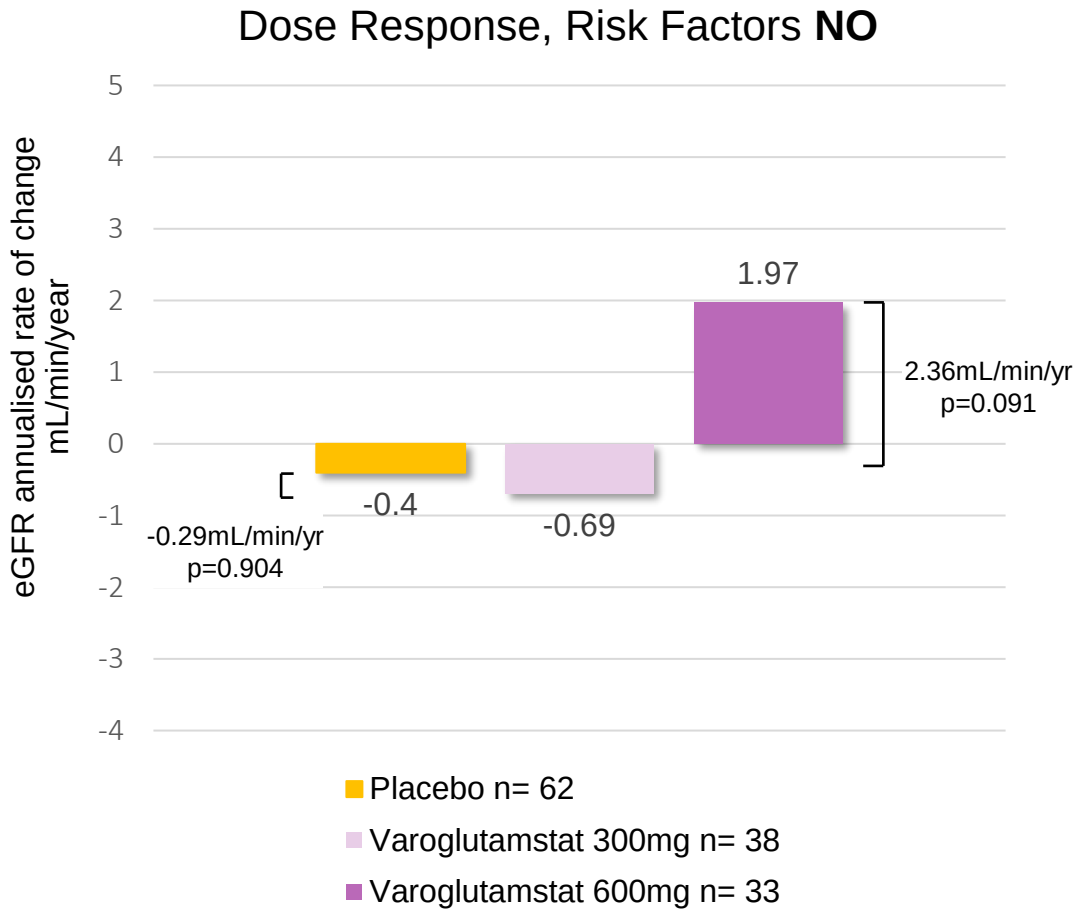
eGFR = estimated glomerular filtration rate based on creatinine

300mg and 600mg cohorts defined as randomized

Varoglutamstat shows a statistically significant and clinically meaningful effect in patients with risk factors for CKD defined as type 2 diabetes or hypertension



300mg and 600mg cohorts defined as randomized



300mg and 600mg cohorts defined as randomized

CKD = Chronic kidney disease

Encouraging VIVIAD kidney function data shapes development path in kidney disease and strategic priorities for varoglutamstat

VIVIAD key learnings kidney disease

- Statistically significant and clinically meaningful improvement of kidney function vs. placebo and sustained improvement over baseline with varoglutamstat 600mg BID
- Strong dose response showing 600mg is more effective compared to 300mg
- Significant improvement of eGFR over baseline and vs. placebo in patients with type 2 diabetes and hypertension
- Results show QPTC/L inhibition can improve kidney function in an older patient group and those with kidney disease risk factors

Looking ahead in kidney disease

- Conduct additional biomarker analysis
- Assess data from additional subgroups
- Assess need for additional non-clinical studies to expand data package
- Continue to investigate the science behind QPCT/L pathways
- Create clinical development pathway for varoglutamstat in kidney disease*

Actively pursue funding and/or partnership opportunities for varoglutamstat in kidney disease

**Additional clinical studies would require further funding and/or partnerships.*

FINANCIAL RESULTS

Condensed statement of profit and loss

In €k	Dec 31, 2023	Dec 31, 2022	YoY in %
Revenue	(3,620)*	-	-
Research and development expenses	(17,637)	(20,224)	(13) %
General and administrative expenses	(8,600)	(8,908)	(3) %
Operating loss	(28,837)	(29,113)	(1) %
Finance result	261	758	
Income taxes	234	199	
Net loss for period	(28,342)	(28,156)	1 %
Loss per share (basic/diluted) (in EUR)	(1.12)	(1.28)	

Careful and selected spending through 2023, as Company worked towards VIVIAD read-out during first quarter of 2024

**Reflects reversal of revenue related to Simcere milestone receivable of EUR 3.6m concerning potential start by Simcere of Phase I study in Greater China; the initial revenue booking and this current reversal are both non-cash*

Key financial figures

In €k	Dec 31, 2023	Dec 31, 2022
Cash and cash equivalents	18,562*	26,555
Total assets	30,829	31,378
Total equity	26,282	26,506

In €k	Dec 31, 2023	Dec 31, 2022
Cash flows used in operating activities	(21,541)	(21,794)
Cash flows used in investing activities	(10,514)*	(13)
Cash flows from financing activities	24,157	33,381

Updated cash runway** to extend into Q2 2025, reflecting reduction in cash utilization and change in R&D focus towards inflammatory & fibrotic disorders

Further funding and/or partnerships required to support potential additional clinical studies and/or to extend runway beyond Q2 2025

Liquid funds in amount of EUR 10m have been deposited on a short-term basis with banks as term deposits. Thus, total liquid funds include cash & cash equivalents of EUR 18.6m and term deposits of EUR 10m.

OUTLOOK & STRATEGIC PRIORITIES

Outlook - Shift research & development focus towards inflammatory and fibrotic disorders such as kidney disease

Exciting potential opportunity for varoglutamstat in kidney disease

- Leverage very promising VIVIAD data to further explore varoglutamstat's potential in kidney disease
- Create development pathway in kidney disease

Assess feasibility of higher dose varoglutamstat in early AD

- Fully analyze VIVIAD results including biomarkers
- Discontinue VIVA-MIND in H2 2024
- Conclude Phase 2 program results in early AD
- Assess development pathway for higher dose

Continue to explore select early stage pipeline programs

- Focus on most promising QPCT/L inhibitors in inflammatory/fibrotic disorders
- Assess potential of meprin inhibitors and mAb

Corporate focus on prudent cash runway management and funding/BD

- Cash runway to now extend into 2Q 2025*
- Realign company strategy dependent on results of varoglutamstat
- Actively pursue funding and business development opportunities

**Reflects an overall reduction in cash utilization including the ramp down of spending on VIVIAD as it approaches its conclusion, the discontinuation of VIVA-MIND, the discontinuation of VIVALONG preparation activities as well as the streamlining of manufacturing costs and programs for API development.*

Q&A

VIVORYON THERAPEUTICS N.V.

Halle (Saale)
Weinbergweg 22
06120 Halle (Saale) Germany

Munich
Franz-Josef-Delonge-Str. 5
81249 München Germany

Info@vivoryon.com
+49 (0)345 555 99 00

www.vivoryon.com