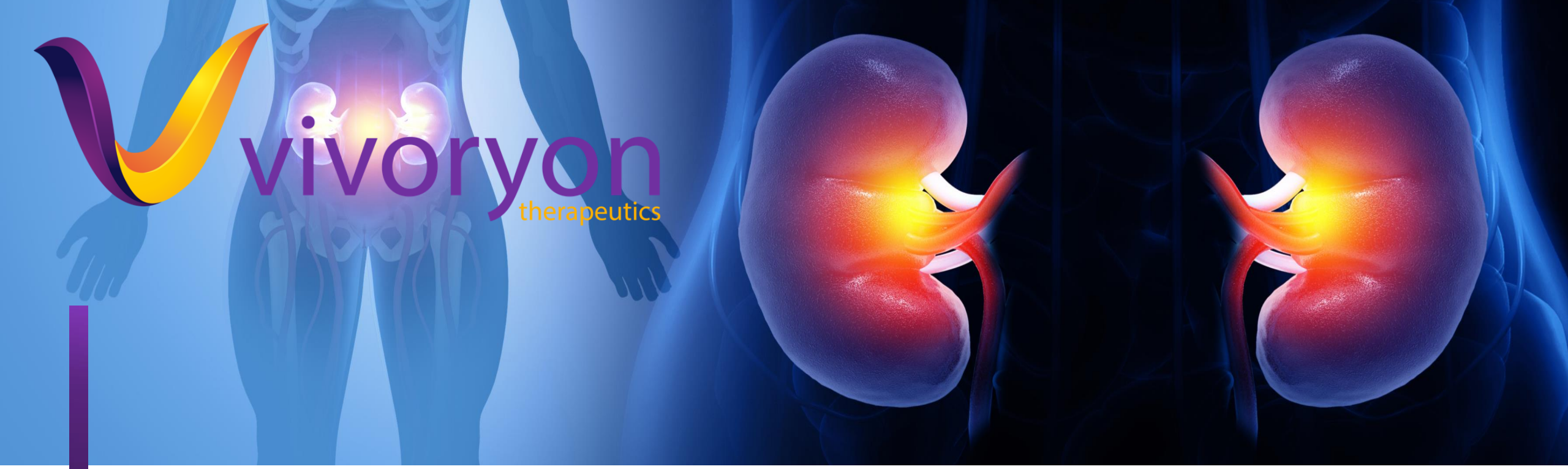

Half Year 2024: Financial Results & Operational Update

Progress Marked by Compelling Kidney Function Data and Execution of Strategy to Advance Varoglutamstat in Kidney Disease

September 12, 2024

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Forward-looking statements speak only as of the date they are made, and we do not undertake any obligation to update them in light of new information or future developments or to release publicly any revisions to these statements in order to reflect later events or circumstances or to reflect the occurrence of unanticipated events or otherwise, except as required by applicable law.

Key highlights

A major step forward in kidney disease emerging from a setback in AD

- **Continued strong progress in transitioning strategy driven by compelling kidney function data**
- **Rigorous scientific research laid foundation for opportunity in inflammatory/fibrotic diseases**
- **Deeper understanding of varoglutamstat's MoA and VIVIAD results: QPCTL is an attractive target in kidney disease**
- **Comprehensive body of evidence underpins plan to advance into Phase 2 in DKD**
- **Pursuing actionable plan to establish presence in kidney disease**

H1 2024 was marked by continuous progress and successfully transitioning our strategy, led by compelling kidney function data

VIVIAD Phase 2 results revealed exciting kidney function data; Alzheimer's data not as hoped

- Missed primary & secondary endpoints in early AD
- Strong and consistent statistically significant effect of varoglutamstat observed on pre-specified, validated regulatory kidney function endpoint (eGFR[1])
- Substantially higher eGFR treatment effect observed in post-hoc diabetes subgroup[2]
- Excellent tolerability & safety profile supporting potential opportunities in additional indications

Advancing development in DKD and exploring orphan kidney disorders

- Varoglutamstat's unique product profile could change the course of kidney disease
- Planning a new Phase 2 study with varoglutamstat in diabetic kidney disease (DKD)[3]
- Biomarker analysis and non-clinical / MoA studies ongoing to support prioritization of additional indications

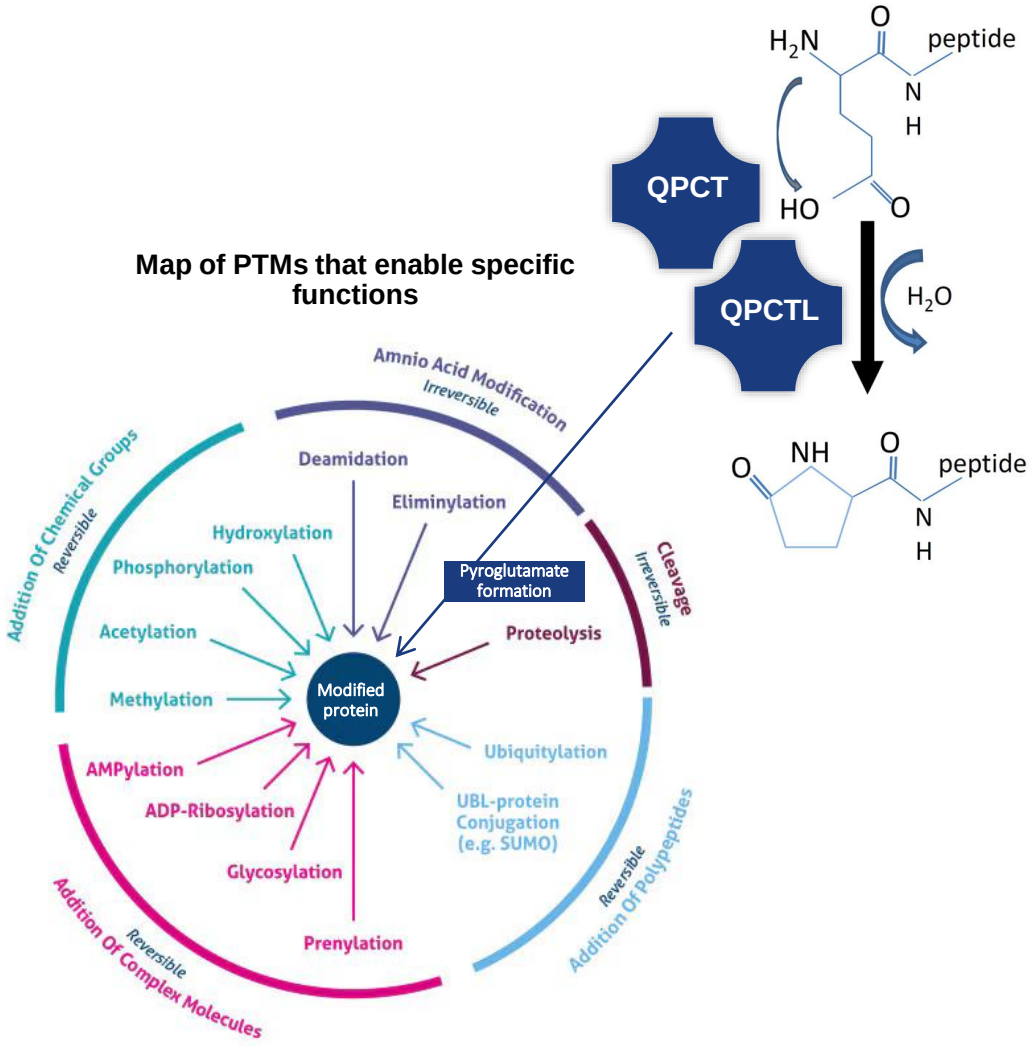
Continuing to understand AD results

- PK/PD in depth analysis
- Additional biomarker analysis
- Analyze data from VIVA-MIND AD study by year end 2024

[1] Estimated Glomerular Filtration Rate (eGFR); [2] Defined as patients having at baseline either medical history of diabetes (type 1 or 2) and/or comedication with drugs used in diabetes and/or untreated with a HbA1c > 6.5%; [3] Subject to additional funding/partnerships

Selectively preventing post-translational modification is key to tackling pathologically altered cellular processes

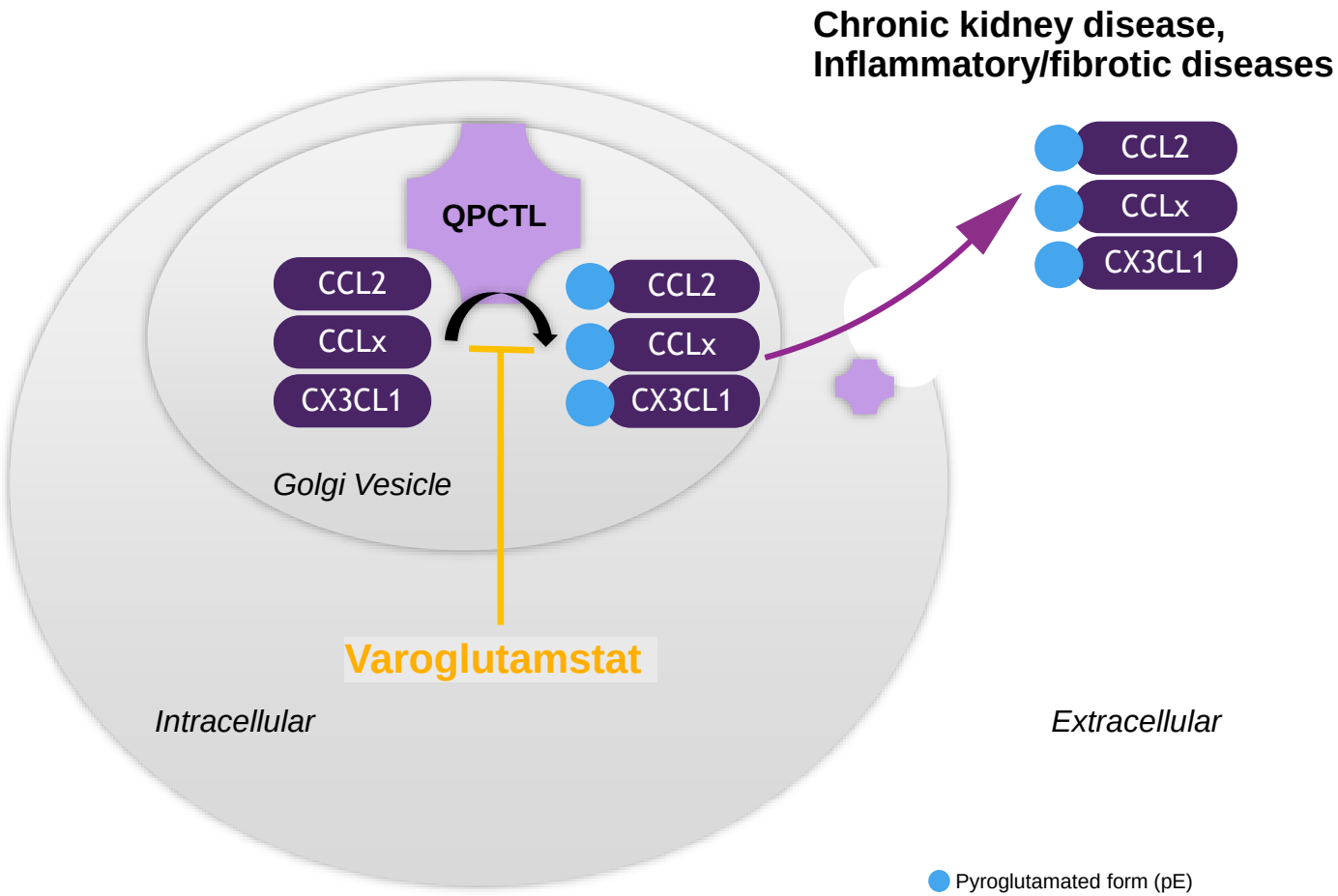
- Post-translational modification (PTM) occurs both physiologically and in disease settings, it is a crucial process to functionalize proteins
- Many different PTMs are catalyzed by enzymes that have become known drug targets, e.g. kinases, proteases, or methylases
- Human glutaminyl cyclase enzymes (QPCT/QPCTL) catalyze pyroglutamate (pE) formation
- pE peptides have emerged as a central element in different diseases including neurodegenerative, inflammatory and fibrotic diseases, as well as cancer



Schematic adapted from Ribet & Cossart, FEBS Letters, 2010

Varoglutamstat is designed to prevent inflammatory and fibrotic processes by blocking pyroglutamate formation on key disease drivers

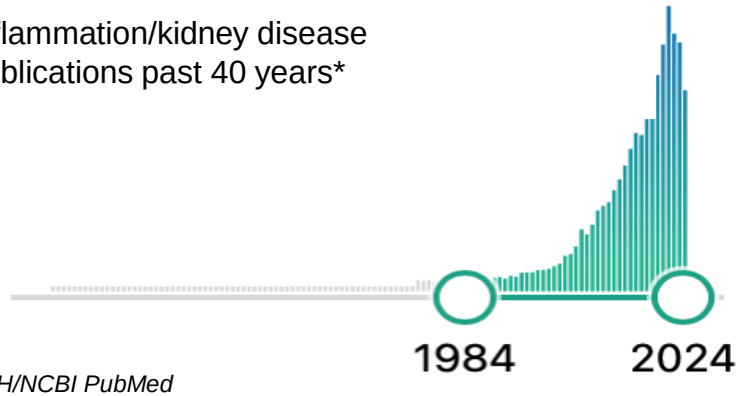
- Varoglutamstat is a highly potent, oral small molecule inhibitor of human QPCTL which is localized intracellularly in Golgi vesicles
- QPCTL catalyzes a PTM that converts N-terminal glutamine or glutamate into pyroglutamate (pE) which is crucial for protein activity, stability, and certain protein-protein interactions
- QPCTL substrates include certain chemokines which are modified by pE formation within the cell and then secreted
- QPCTL target chemokines are highly expressed in kidney, liver, specialized epithelial cells, endocrine cells, fibroblasts and immune cells
- Several groundbreaking findings on the application of QPCTL inhibitors in inflammation & NAFLD published by Vivoryon[1,2,3]



[1] Cynis et.al. EMBO, 2011; [2] Cynis et al, Int. J Exp Path, 2013; [3] Kehlen et.al. Biosci Rep, 2017;
Notes: Graphic is for illustrative purposes only ; CCLx – other potential pE chemokines.

Key pre-clinical findings support development of QPCTL inhibitors in kidney and liver disorders with high unmet medical need

Inflammation/kidney disease publications past 40 years*



*NIH/NCBI PubMed

Clinical Practice: Mini-Review

Inflammation in Diabetic Kidney Disease

Perez-Morales et al. 2019

> *This new pathogenic perspective of DKD as an inflammatory condition leads to novel horizons, such as the potential role of inflammatory signaling pathways and their downstream products as emerging biomarkers and promising therapeutic targets*

Pharmacological inhibition of the chemokine CCL2 (MCP-1) diminishes liver macrophage infiltration and steatohepatitis in chronic hepatic injury

Baeck et. al. 2012

Inhibition of Glutaminyl Cyclases alleviates CCL2-mediated inflammation of non-alcoholic fatty liver disease in mice

Cynis et al., 2013

N-terminal pyroglutamate formation in CXC3CL1 is essential for its full biologic activity

Kehlen et. al. 2017

The Role of Chemokines and Chemokine Receptors in Diabetic Nephropathy

Chang et. al. 2020

Chronic treatment with the (iso-)glutaminyl cyclase inhibitor PQ259 is a novel and effective approach for glomerulonephritis in chronic kidney disease

Kanemitsu et. al. 2021

REVIEW ARTICLE

CCL2-CCR2 signaling axis in obesity and metabolic diseases

Wu et. al. 2024

Cynis et.al. Int, J of Exp Pathol, 2013; Baeck et.al., Gut, 2012; Chang et.al. Int J Mol Sci., 2020; Wu & Ma, J Cell Physiol, 2024; Kanemitsu et al., Naunyn Schmiedebergs Arch Pharmacol, 2021; Kehlen et al, Biosci Rep, 2017; Perez-Morales et al., Nephron, 2019

Understanding the different outcomes in VIVIAD

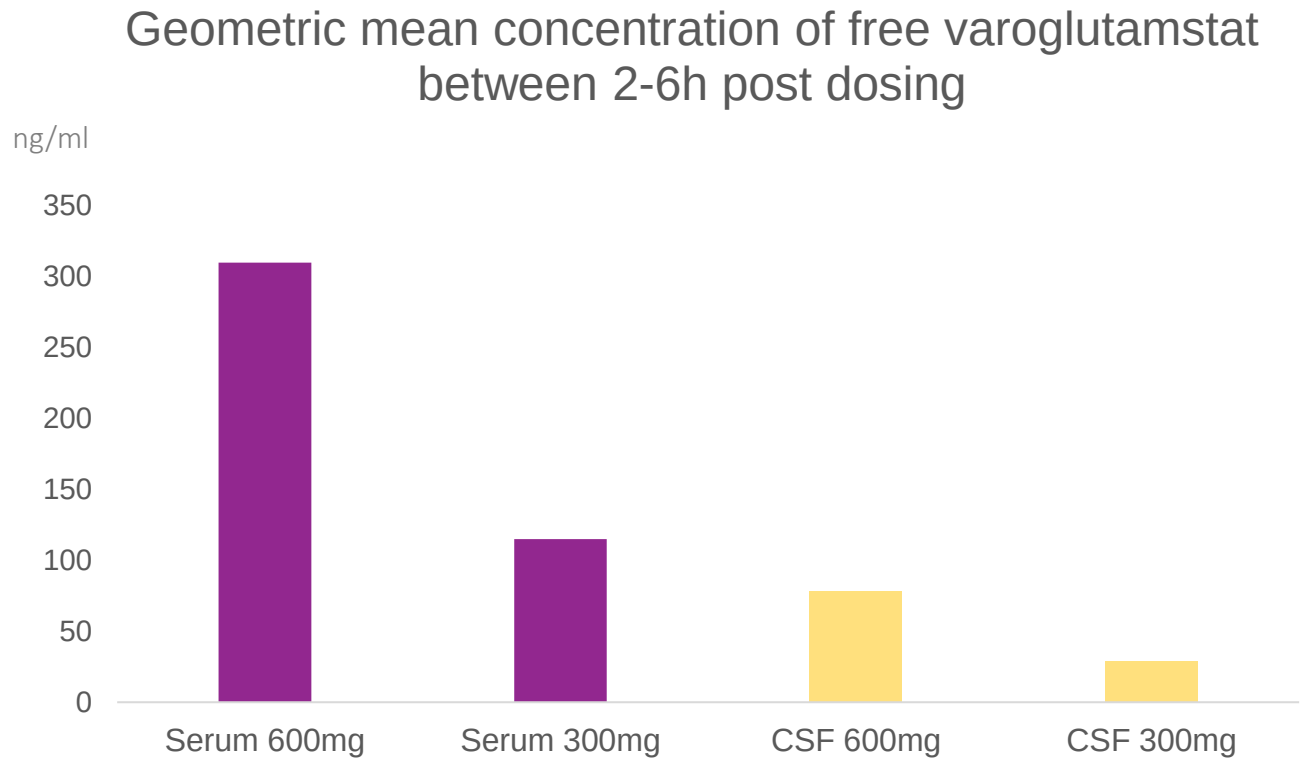
Conducted stepwise analysis of the VIVIAD results to better understand the difference in outcomes between kidney and AD:

1. Concentration of varoglutamstat in serum and CSF at 300 and 600 mg
2. Inhibition of the **extracellular QPCT (QC) enzyme** in serum and CSF
3. Inhibition of the **intracellular QPCTL (iso-QC) enzyme** in serum and CSF
4. Reduction of pE-Abeta 40 in the CSF (target peptide for AD - initial results)

1 Concentration of varoglutamstat in serum and CSF at week 48

Key conclusions

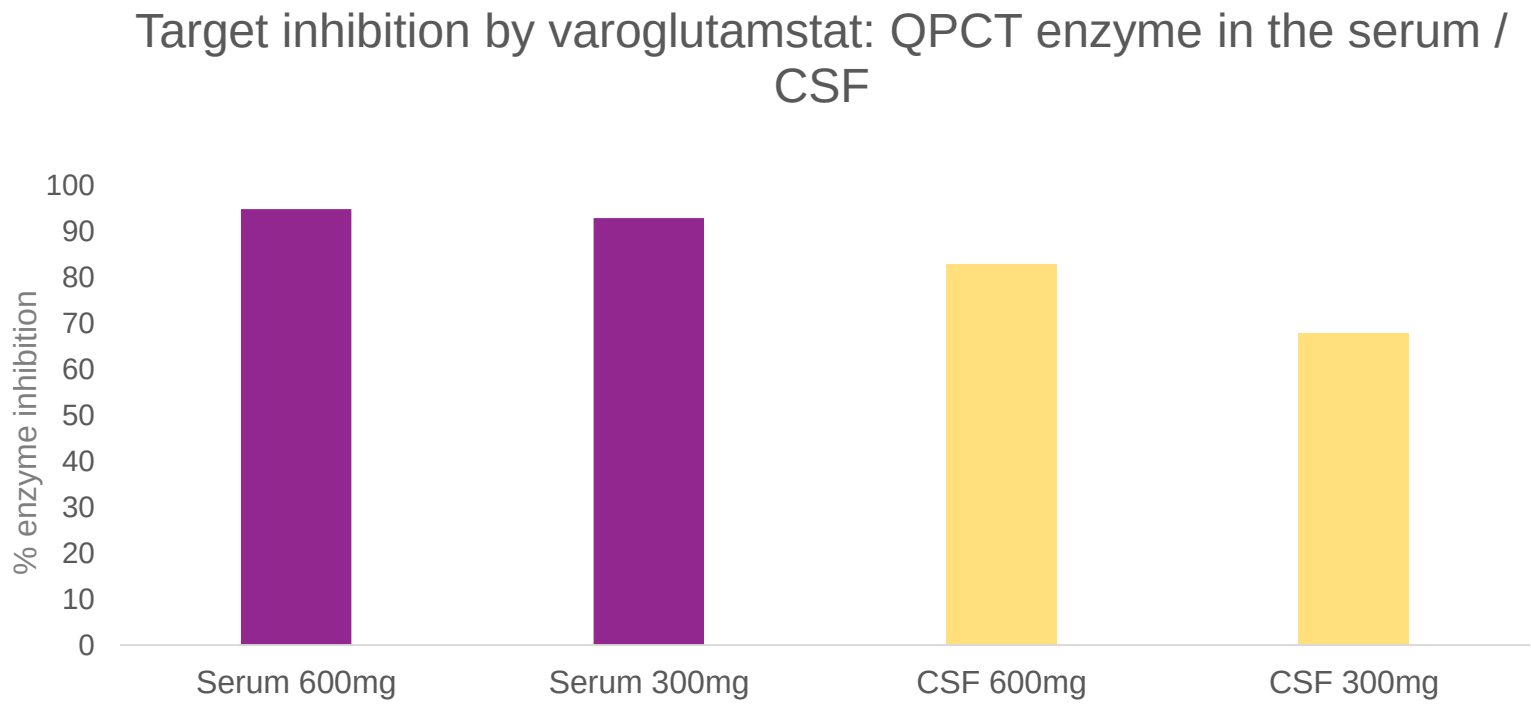
- The blood brain barrier reduces the concentration of varoglutamstat in the CSF compared to serum
- Dose-dependent concentration of varoglutamstat is fully consistent with previously published results



2 Inhibition of the **extracellular QPCT (QC) enzyme** in serum and CSF: measurement of QPCT activity / calculation of target inhibition

Key conclusion

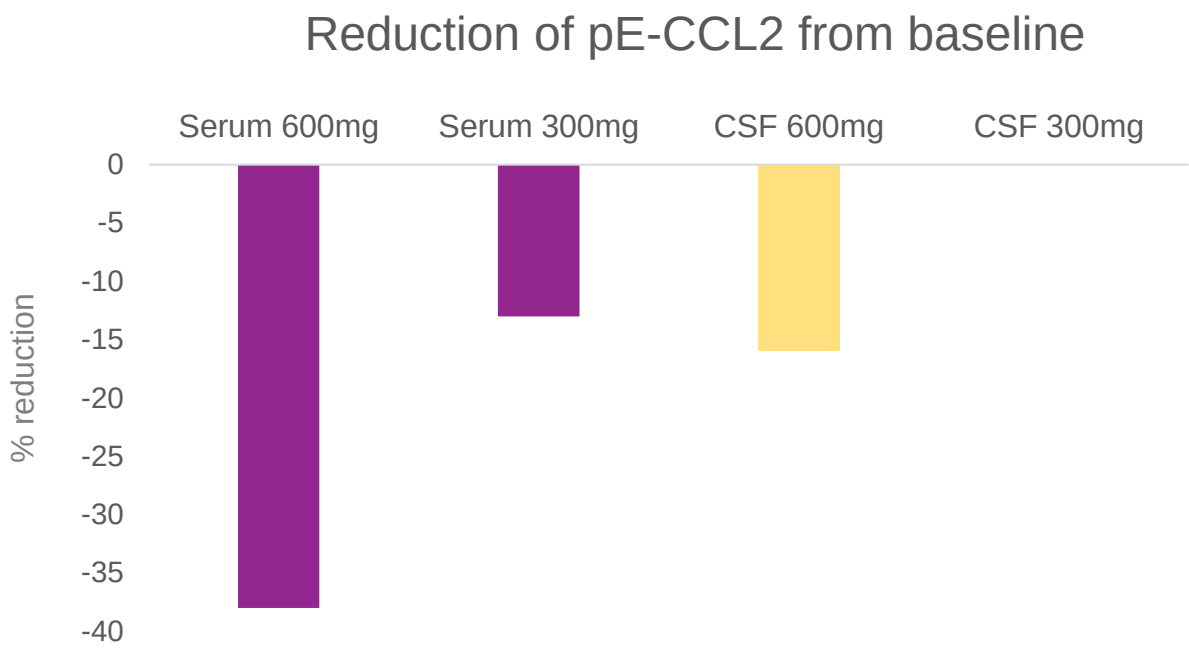
- Strong and significant inhibition of the **extracellular QPCT enzyme** at all doses in the serum and CSF consistent with previously published results



3 Inhibition of the **intracellular QPCTL (iso-QC) enzyme** measured by reduction of pE-CCL2 formation at week 48

Key conclusions

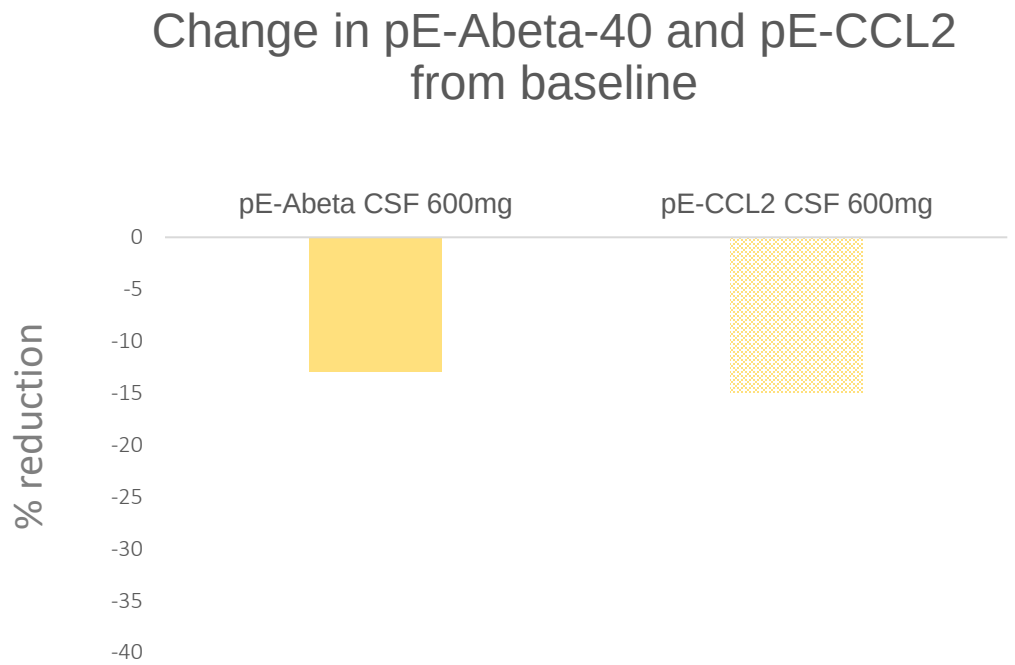
- **Strong inhibition of the intracellular QPCTL at 600mg** BID varoglutamstat demonstrated by statistically significant ($p<0.0001$) and large reduction of pE-CCL2 observed in the serum
- **Minor inhibition of the intracellular QPCTL at 300mg** BID varoglutamstat; pE-CCL2 reduction of only around 15% in the serum and similar to 600mg BID in the CSF
- No effect observed 300mg BID in the CSF



4 Reduction of pE-Abeta-40 in the CSF (target peptide for AD - initial results)

Key conclusions

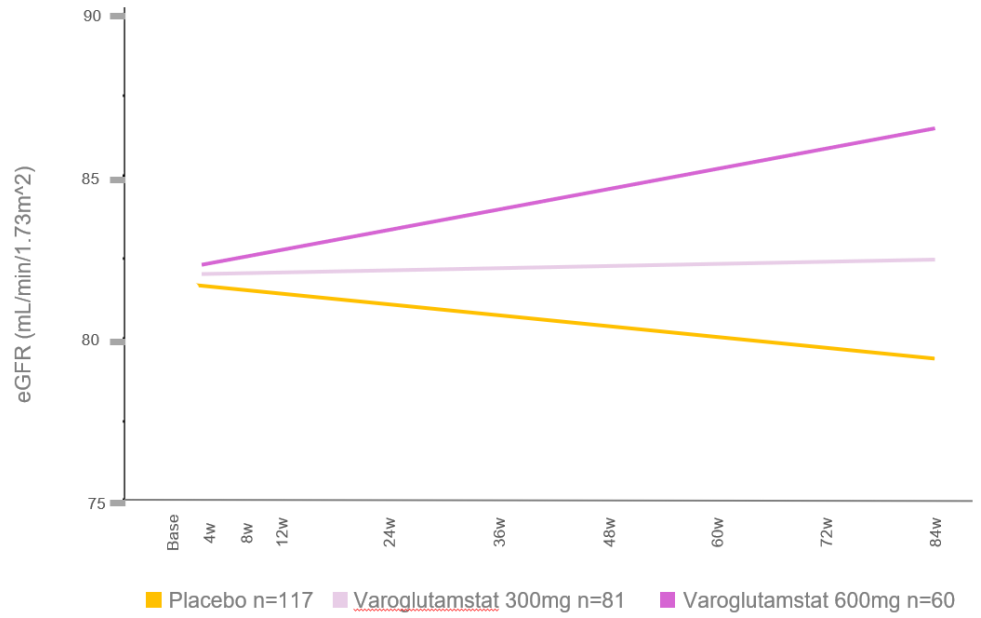
- A 13% reduction of pE-Abeta in the CSF was observed in a first set of samples (n=36) at a dose of 600mg BID
- pE-Abeta reduction was not sufficiently high
- The effect size of pE-Abeta reduction is comparable to the reduction of pE-CCL2 at 600mg BID in the CSF (~15%) - pE-CCL2 is produced intracellularly



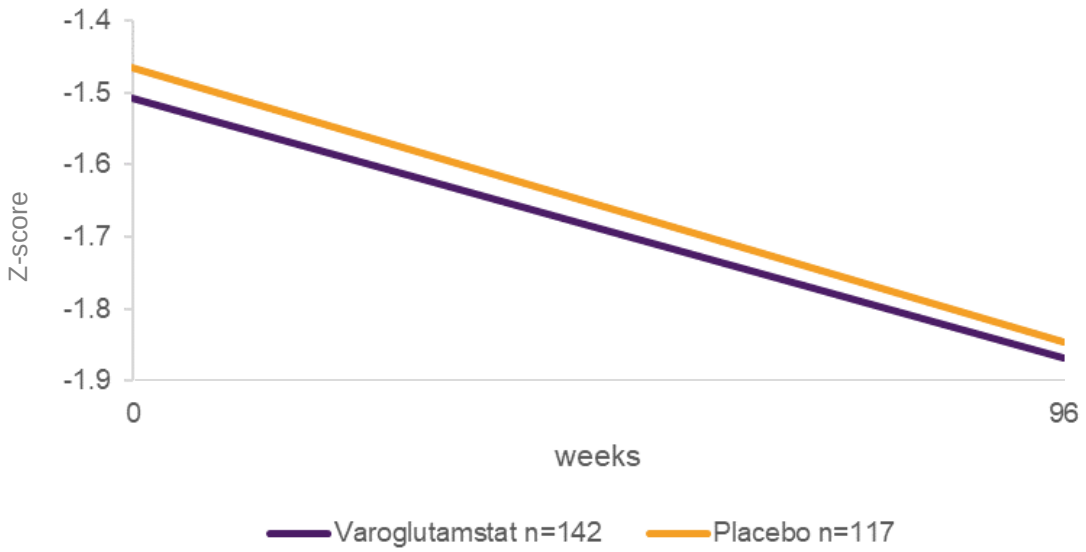
- New assay development for pE-Abeta-40 in CSF matrix using ultrasensitive NULISA technology by Alamar
- Analysis of VIVIAD initial clinical sample set (n=36, 4 groups of 9 subjects)

Intracellular inhibition of pE peptides is the key driver for efficacy

Varoglutamstat effect on kidney function outcomes
Change in eGFR over time (measured using MDRD)[1]



Varoglutamstat effect on AD outcomes
Change in complete Cogstate NTB score over time[2]



High varoglutamstat concentrations lead to clear pE-peptide reduction and result in a profound effect on kidney function

Lower concentrations yield only small effects on pE-formation with no effect on AD and kidney outcomes

[1] Chart shows change in eGFR (estimated glomerular filtration rate) over time based on creatinine and calculated using modification of diet in renal disease (MDRD) method.
[2] Chart shows secondary endpoint of Complete Cogstate NTB score including CBB plus CFT, LFT, immediate and delayed shopping list (slope analysis).

Results provide deeper understanding of VIVIAD data and confirm QPCTL is an attractive target in kidney disease

The VIVIAD study provides clear evidence:

- Concentration profiles of varoglutamstat intensively studied in Phase 1 were confirmed in VIVIAD (PK)
- Varoglutamstat strongly inhibits extracellular QPCT (QC) at all doses tested but no correlation with clinical benefits in AD or kidney
- **Clinically beneficial effects only observed at varoglutamstat concentrations which inhibit the intracellular QPCTL (iso-QC)**
  - 600mg BID showed clear reduction of pE-CCL2 in the serum and a strong effect on eGFR
  - 300mg BID showed a minor effect on pE-CCL2 in the serum and a minor effect on eGFR
  - Varoglutamstat concentrations in the CSF at 600mg BID show a minor reduction on pE-Abeta and pE-CCL2 and no effect on AD
- Initial results suggest that the majority of pE-Abeta is produced intracellularly in the brain cells and not in the CSF / interstitial fluid around plaques

In sum, these findings suggest that intracellular QPCTL is more important in driving efficacy

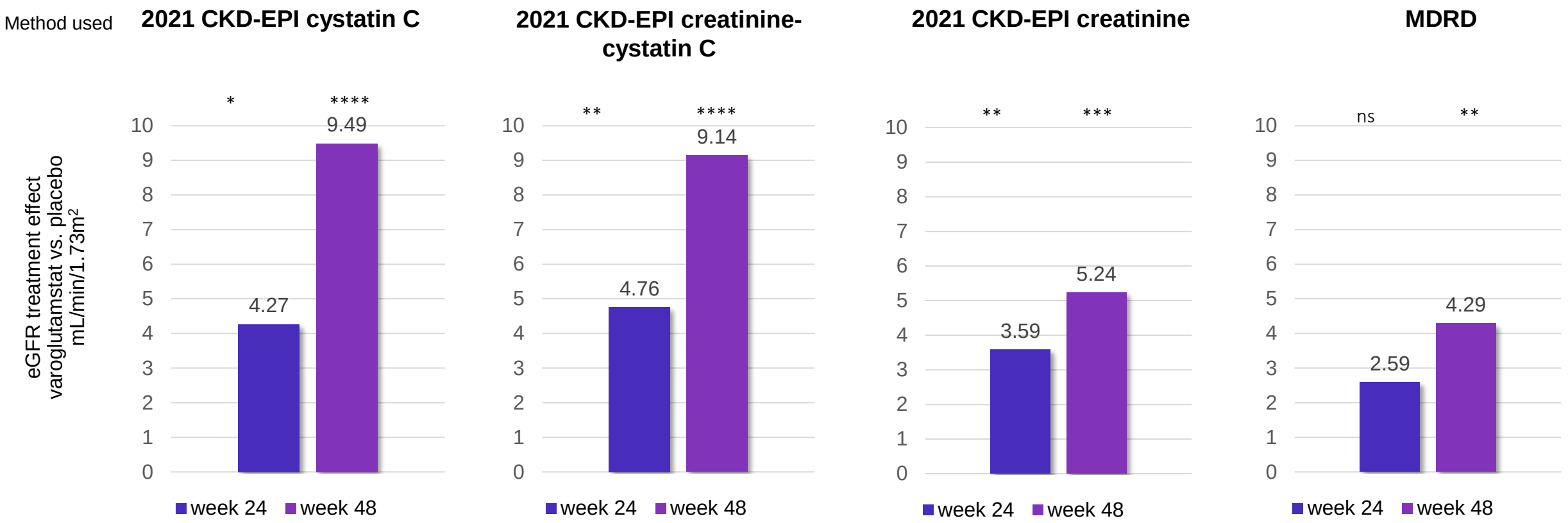
Comprehensive body of evidence underpins strategic shift

VIVIAD Phase 2b kidney data highlights (all patients):

- Robust effects on eGFR independent of analytical method, formula for calculations and substrate either creatinine or cystatin C in the total population of VIVIAD
- Persistency of effect until last dose on treatment for up to 2 years
- Excellent tolerability & safety profile based on over 400 subjects in Phase 1 and Phase 2 studies
- Hyperfiltration unlikely to be a potential mode of action due to: no worsening of proteinuria, reduction of diastolic blood pressure and mean arterial blood pressure, and slow onset of action (3-6 months on full dose until full effect)
- Dose response connected to a plausible mechanism of action; 300mg BID not sufficiently potent
- Results in patients consistent with animal study outcomes

Kidney function effects are consistent and robust across various validated analysis methods for eGFR assessment

Sensitivity analysis: baseline adjusted treatment effect of varoglutamstat versus placebo



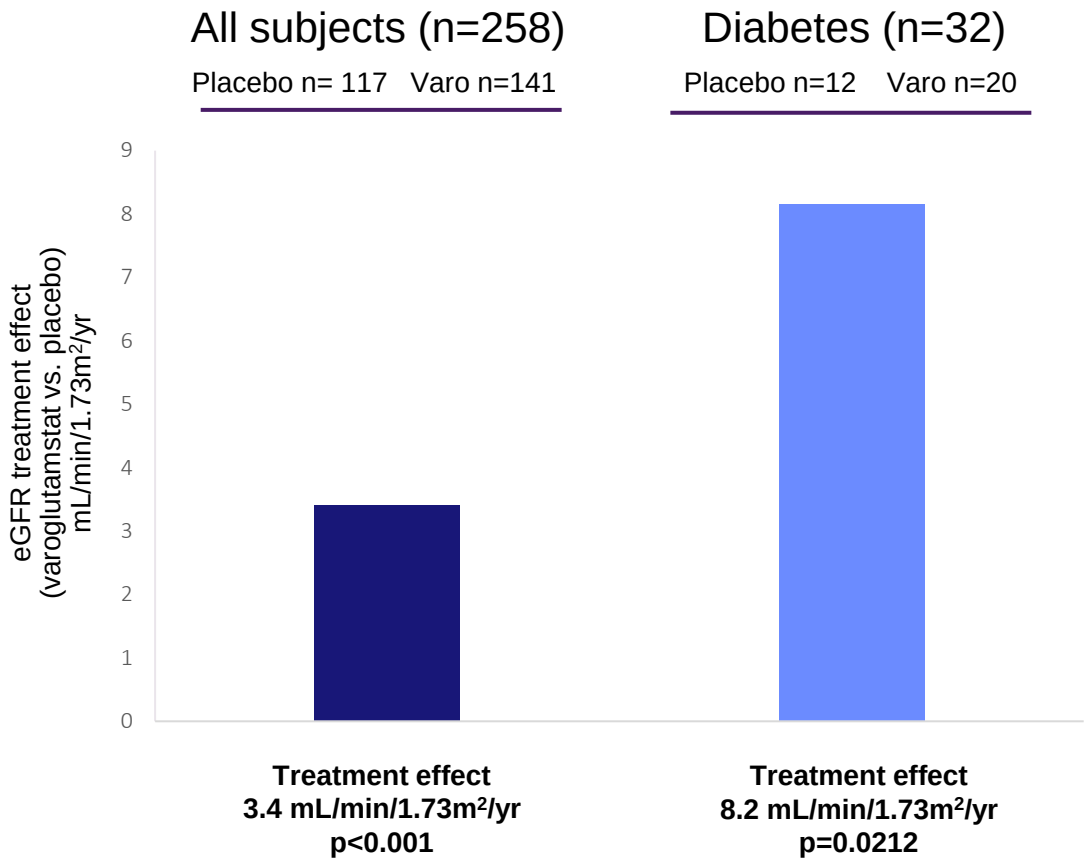
* significant difference (with p<0.05) ** significant difference (with p<0.01) *** significant difference (with p<0.001) **** significant difference (with p<0.0001)

CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration); eGFR assessed by cystatin C and additional creatinine measurement (Atellica CH analyser)

Effect size more than doubled in diabetes subgroup

eGFR measured using creatinine and MDRD

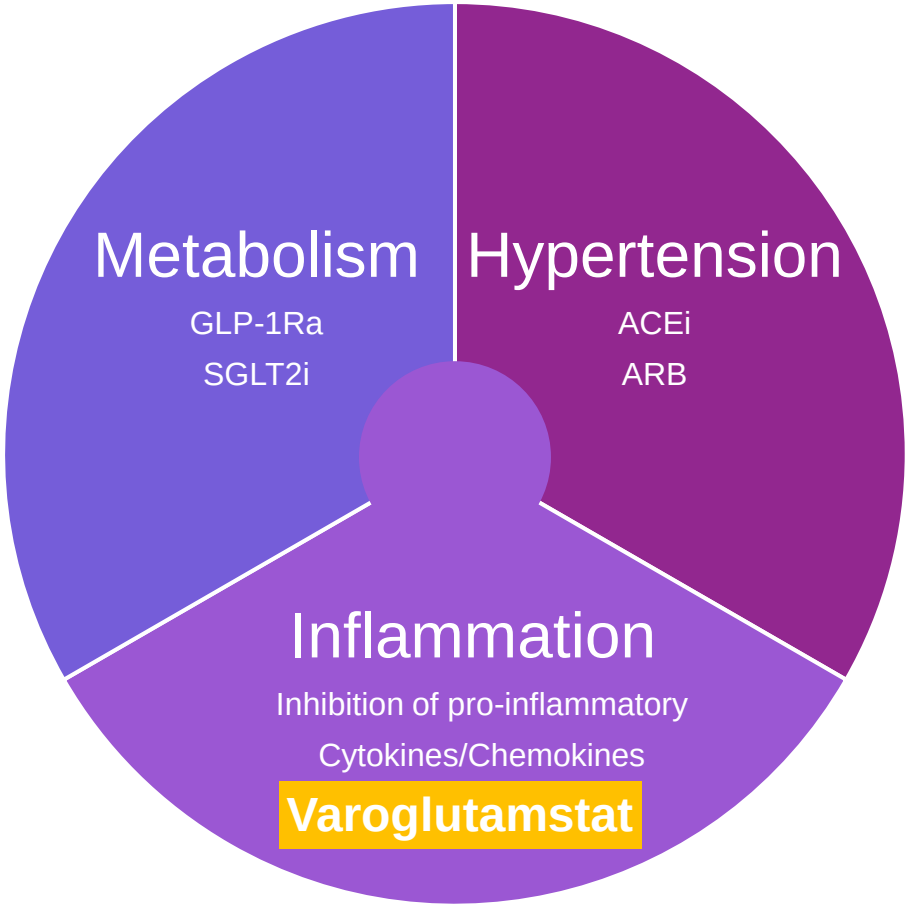
- ✓ **Treatment effect in diabetes subgroup[1] was substantially higher than in the overall patient population**
  - Improvement of eGFR (slope): > 8 mL/min/1.73m^2/yr (p=0.02)
- ✓ **Additional health benefits observed in patients with diabetes versus placebo group [2]**
  - Trend for improving transaminases: - 6 units average at week 48
  - Mild weight loss: - 4 kg at week 48
  - Lowering of diastolic blood pressure: - 6 mmHg at week 48
  - Lower mean arterial blood pressure: - 5 mmHg at week 48
- ✓ **Comparable adverse event (AE) rates vs. total population**
- ✓ **Data support further advancing varoglutamstat in DKD**



eGFR – estimated glomerular filtration rate based on creatinine and calculated using modification of diet in renal disease (MDRD) method based on clinical data from the VIVIAD study, creatinine was measured at 12-week intervals throughout the study

Targeting inflammation offers a third complementary element to existing therapeutic approaches for DKD

- Activation of pro-inflammatory proteins and pathways is a hallmark of DKD pathogenesis[1]
- Varoglutamstat directly attenuates pro-inflammatory signaling by destabilizing key inflammatory molecules
- Varoglutamstat's mode of action is different from drugs acting on SGLT2 or GLP-1, indicating that progressors on current and future SoC could benefit from varoglutamstat
- In line with need to identify novel targets and approaches directly aiming to suppress inflammatory pathways[2]



[1] Ruiz-Ortega, Nature Rev Nephrol, 2020; [2] Rayego-Mateos, Kidney International, 2023;
SoC: standard of care; SGLT2i, sodium-glucose cotransporter-2 inhibitor; GLP-1Ra, glucagon-like peptide 1 receptor agonist.

Planning to conduct a Phase 2 study in advanced stage 3b/4 diabetic kidney disease (DKD) on top of standard of care (SoC)[1]

Expected to meet industry needs and address key questions, interim analysis at month 15

Draft design

- Stage 3b/4 DKD patients with > 100mg/g albuminuria / proteinuria
- All patients on standard of care medicines (SoC)
- Randomized 1:1 varoglutamstat BID (dose 600mg BID) vs. placebo
- Treatment duration per patient 15 months

Up to 120 patients treated for 15 months to detect a difference of 5ml eGFR slope With 80% power (sensitivity for smallest treatment difference is 3.5ml)

Key endpoints

- eGFR slope
- UA(p)CR (albuminuria)
- Biomarkers inflammation/fibrosis metabolism incl. NAFLD / NASH
- Safety

Potential timelines

Frequent measurement of eGFR (q 4 weeks)

Screening and recruitment	Randomization (SOT)	*Interim analysis at 15 months*	EOT	Down-titration	Follow up
◆ Approx. 9-12 months ◆ Sites in US and Europe	Up-titration 8 weeks		week 60	8 weeks	4 weeks

Total duration ~ 2.5 years

Typical trial cost approx. EUR 10 – 12mn[2]

[1] Clinical trial subject to further funding / partnership; [2] Based on typical industry costs for Phase 2 in similar indications.

Varoglutamstat: a unique target product profile in DKD with the potential to improve kidney outcomes

Efficacy

Kidney function improvement:

- Robust effect with clear improvement of eGFR over baseline independent of baseline eGFR (range studied to date 45 -120ml/min)

Additional effects in diabetes subgroup:

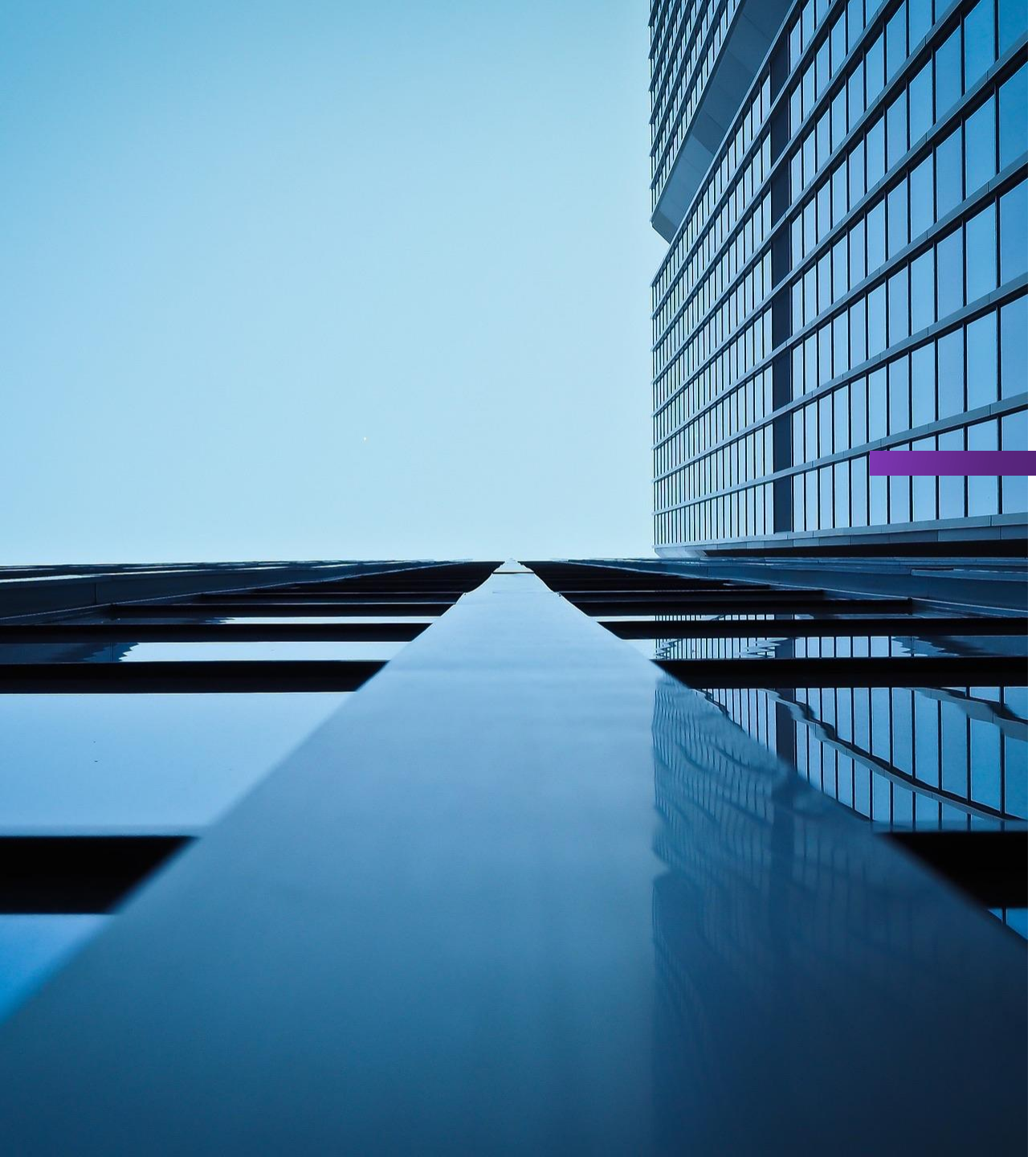
- Slight reduction of diastolic and mean arterial pressure (which is favorable for any cardiovascular outcome and event rate)
- Reduction of liver transaminases (which is favorable for frequent comorbidity of NAFLD / NASH in DKD patients)
- Mild weight reduction (which is favorable for overall and cardiovascular outcomes)

Safety & Administration

- Excellent tolerability and safety profile
- No worsening of proteinuria (potential improvement to be assessed in more advanced patients)
- Simple oral treatment of 600mg BID (morning and evening)

Position in therapy

- Treatment effect through anti-inflammatory / antifibrotic mechanism, complementary and non-redundant to current metabolic / glycemia based therapies
- Varoglutamstat has additional potential in orphan kidney disorders



FINANCIAL RESULTS & OUTLOOK

Financials reflect prudent spending while refocusing on kidney disease

In €k	Jun 30, 2024	Jun 30, 2023
Revenue	0	0
Research & Development expenses	(10,308)	(6,259)
General & Administrative expenses	(3,501)	(4,433)
Net loss for the period	(13,559)	(10,761)

In €k	Jun 30, 2024	Dec 31, 2023
Cash & cash equivalents	15,272	18,562*
Financial assets	0	10,165*

Cash runway into Q2 2025; H1 2024 captures investment in VIVIAD and VIVA-MIND which meaningfully ramp down in H2 2024 and beyond

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.*

Vivoryon: Mid-stage clinical biotech company with first-in-class QPCT/L inhibitor targeting high unmet need in kidney disease

Compelling efficacy and safety data in people at risk of kidney disease

- ✓ Statistically significant effect on kidney function in total VIVIAD population and diabetes subgroup, with robust safety data

Developed clinical plan targeting large unmet need in DKD

- ✓ Design of a placebo-controlled Phase 2 study in stage 3b/4 DKD patients on top of SoC

Strong scientific foundation and PK/PD data providing evidence for new MOA

- ✓ Elucidating varoglutamstat's intra / extracellular activity and QPTC/L inhibition capabilities along inflammatory and fibrotic pathways

Company activities aimed at securing all aspects to support future growth

- ✓ Current composition of matter patent to 2031[1]; new patent filings have potential to extend protection to 2044+
- ✓ Actively pursuing funding and business development opportunities to fully execute Phase 2 DKD study

A major step forward in kidney disease emerging from a setback in AD

[1] *with additional potential for Hatch-Waxman extension of up to 5 years.*

Up next: External views on varoglutamstat and opportunities in kidney disease

Virtual Kidney Disease KOL Event

Monday, September 30, 2024
at 3:00pm CEST (9:00am EDT)

- Expert presentations by seasoned KOLs followed by Q&A session
- Standard of care and existing medical need in kidney disease
- Market development and commercial potential in kidney disease
- Evidence generation and statistical principles in kidney disease drug development
- Special emphasis on diabetic kidney disease (DKD)

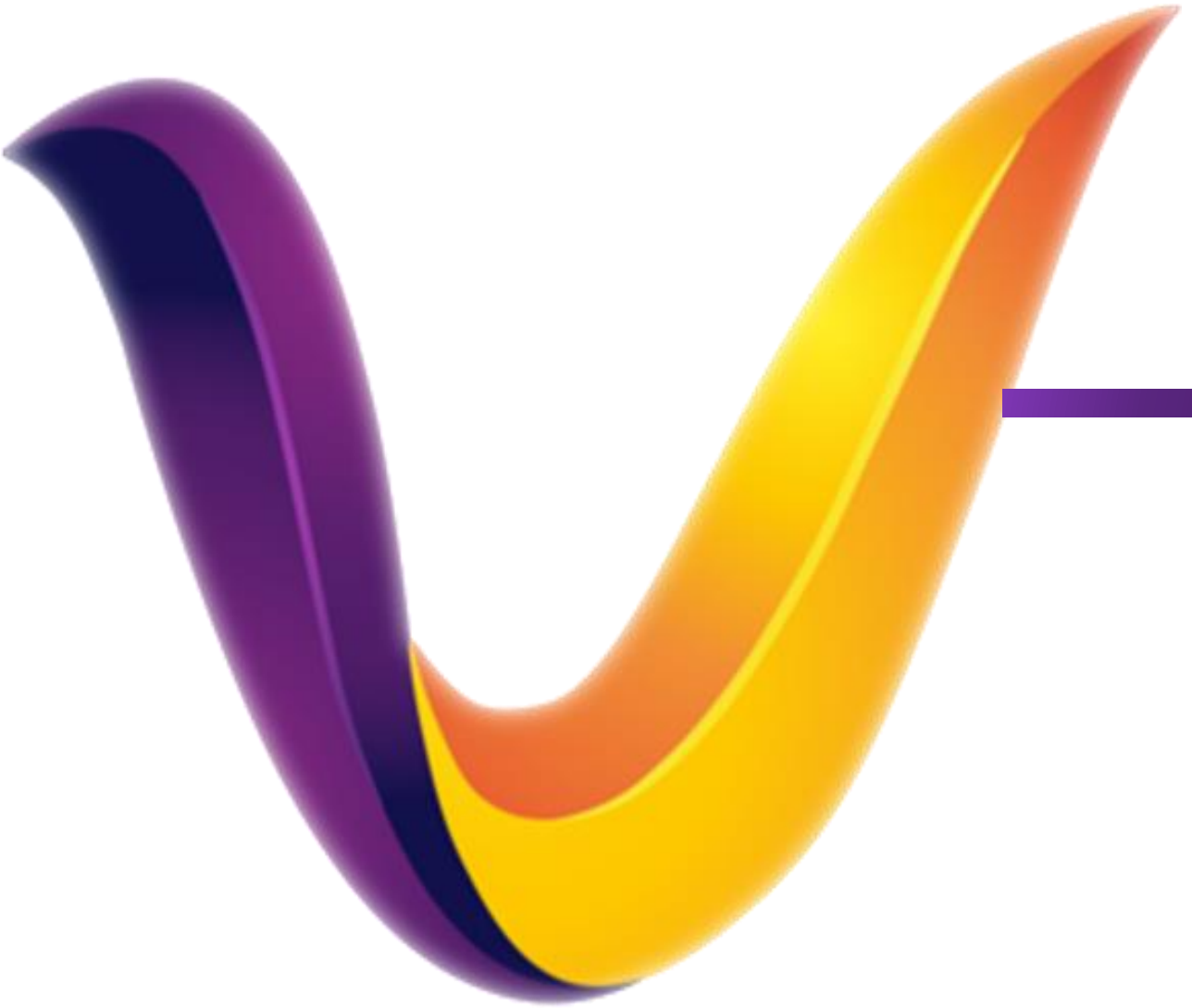
Featured Speakers:

Tobias B. Huber, M.D.
Chair of the Center of Internal Medicine and Director of the III. Department of Medicine, University Medical Center Hamburg-Eppendorf (UKE)

Florian Jehle
Chief Executive Officer, Vifor-FMC Renal Pharma

Kevin Carroll, Ph.D.
Chief Executive Officer, KJC Statistics

Host - Frank Weber, M.D.
Chief Executive Officer and Chief Medical Officer, Vivoryon Therapeutics



Q&A

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