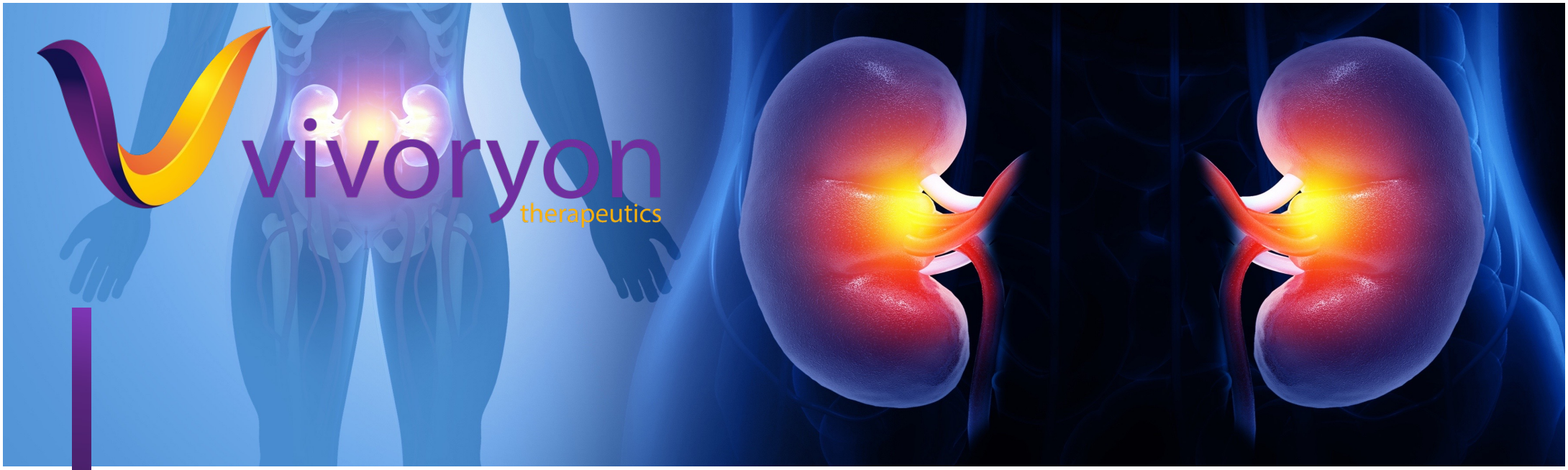

Changing Outcomes in Kidney Disease

Virtual R&D Update with KOL Speakers

February 18, 2025

Vivoryon Therapeutics N.V.

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SPEAKERS

Host	**Anne Döring, CFA** CFO Vivoryon Therapeutics
	Tobias Huber, MD Director III. Department of Medicine UKE Hamburg-Eppendorf
	Michael Schaeffer, PhD CBO Vivoryon Therapeutics
	Kevin Carroll, PhD CEO KJC Statistics
	Frank Weber, MD CEO Vivoryon Therapeutics

Anne Döring, CFA

CFO
Vivoryon Therapeutics

Inhibiting QPCTL has potential to halt the progressive course of kidney disease through unique approach to tackle inflammation and fibrosis

Huge unmet medical need

Current treatments do not stabilize / improve kidney function leaving significant risk of ESRD (dialysis, transplant) or cardiovascular event

Inflammation a key underlying driver

Inflammation and fibrosis have long been known as key drivers of disease yet attempts to develop effective therapeutics selectively targeting key pathways have had limited success

Targeting QPCTL to unlock inflammatory approach

Vivoryon has identified QPCTL, an enzyme that creates pro-inflammatory pE-versions of key inflammatory proteins, as a promising target with potential to stabilize disease

Varoglutamstat

- Oral, selective QPCTL inhibitor
- Significantly improved kidney function[1] in two independent Phase 2 studies[2]
- Unprecedentedly large and sustainable effect size over two years

[1] Measured by estimated glomerular filtration (eGFR): rate based on creatinine, calculated using modification of diet in renal disease (MDRD) method;
[2] VIVIAD and VIVA-MIND Phase 2 studies in early Alzheimer's disease (AD) included prospectively defined measures of kidney function as safety and other exploratory endpoints.
QPCTL - Glutaminyl-peptide cyclotransferase-like protein

Tobias B. Huber, MD

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

New scientific results of varoglutamstat in kidney disease

Tobias B. Huber | 18th February 2025

Disclosures

Consultancy agreements: Alexion, AstraZeneca, Bayer, Beren Therapeutics, Boehringer-Ingelheim, DaVita, Euroimmun, Fresenius Medical Care, Nipoka, Novartis, Pfizer, Renovate, Retrophin-Travere, Sanofi, Vera Therapeutics, Vifor, Vivoryon Therapeutics

Research funding: Amicus Therapeutics, Fresenius Medical Care, Euroimmun, Vivoryon Therapeutics

Editorial boards: Kidney International, Nature Review Nephrology

Patents: EP23154267.1 “AAV2-vector variant for targeted transfer of genes”
EP23154266.3 “AAV9 capsid variant for targeted gene transfer”
EP23195461. “Novel solution for ex-vivo organ storage during machine perfusion”

President of the International Society of Glomerular Disease

Vivoryon has evaluated varoglutamstat's effect on kidney function in two independent randomized double-blind placebo-controlled Phase 2 studies

Similarities and differences between VIVIAD and VIVA-MIND studies

Parameter	VIVIAD (Europe)	VIVA-MIND (U.S.)
Patient selection	Mild AD, mean age 68 yrs	Mild AD, mean age 72 yrs
No. of patients treated	N=259	N=109
Varoglutamstat dose investigated	300 and 600 mg BID	600 mg BID
Dose escalation period	Slow: 600 mg start week 13	Fast: 600 mg start week 9
Treatment duration	76 weeks (mean) / 96 weeks (max.)	46 weeks (mean) / 72 weeks (max.)
eGFR[1] sampling	Every 12 weeks plus week 4	Every 12 weeks plus weeks 4, 8,16
No. of patients with diabetes	N=32 (12.4%)	N=16 (14.7%)

Kidney function, measured using eGFR, was a pre-specified safety / exploratory endpoint

VIVIAD: ClinicalTrials.gov ID NCT04498650; VIVA-MIND: ClinicalTrials.gov ID NCT03919162; BID: twice daily; eGFR: estimated glomerular filtration rate, based on creatinine samples and calculated using the modification of diet in renal disease (MDRD) method; AD: Alzheimer's disease; Diabetes subgroup defined as patients having at baseline either medical history of diabetes (type 1 or 2, and glucose tolerance impaired, hyperglycaemia in VIVA-MIND) and/or comedication with drugs used in diabetes and/or untreated with a HbA1c > 6.5%.

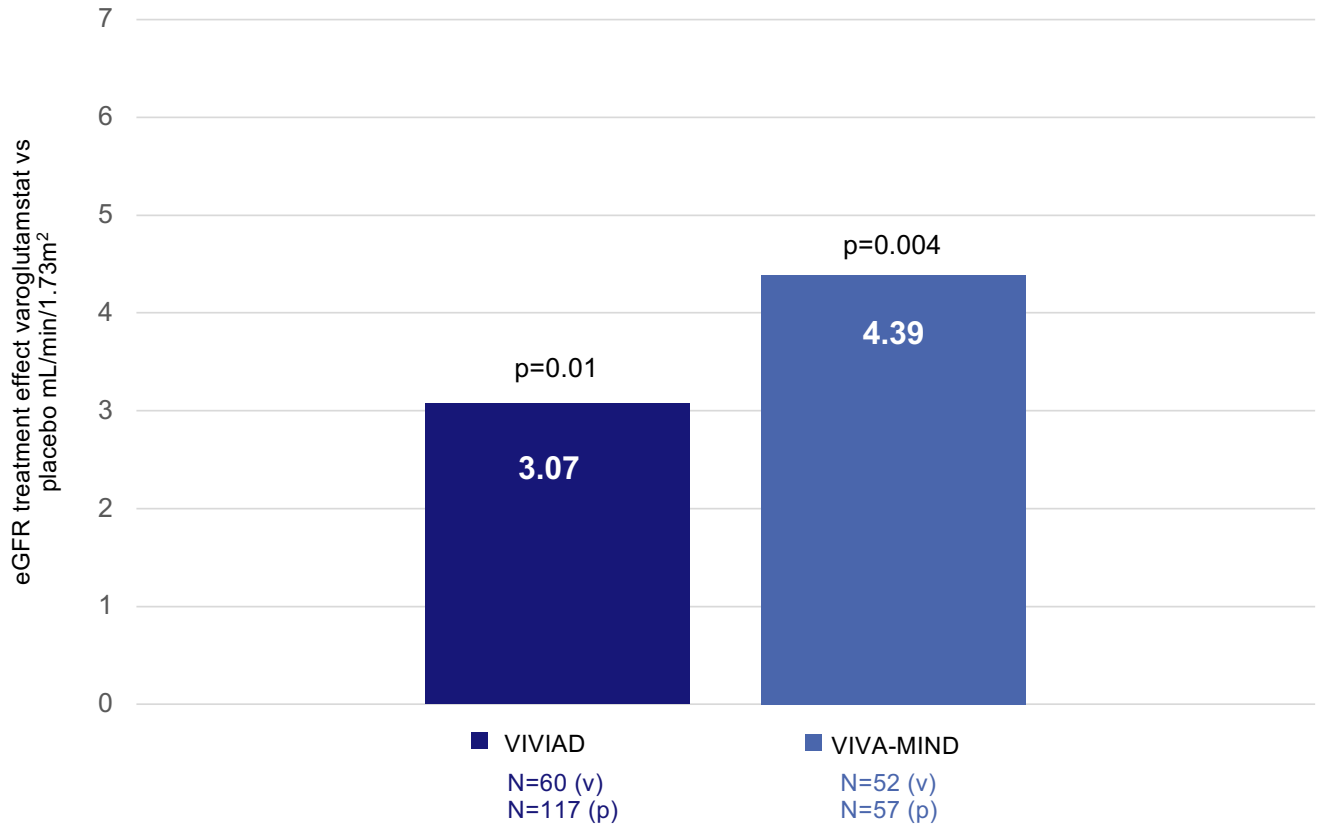
VIVIAD and VIVA-MIND both show a statistically significant and clinically meaningful improvement in eGFR over baseline

eGFR results (MDRD); all patients randomized to 600 mg BID varoglutamstat (v) and placebo (p)

eGFR treatment effect:

Difference between varoglutamstat and placebo (LSmean change from baseline)

Total population,
600 mg BID patients only,
all visits



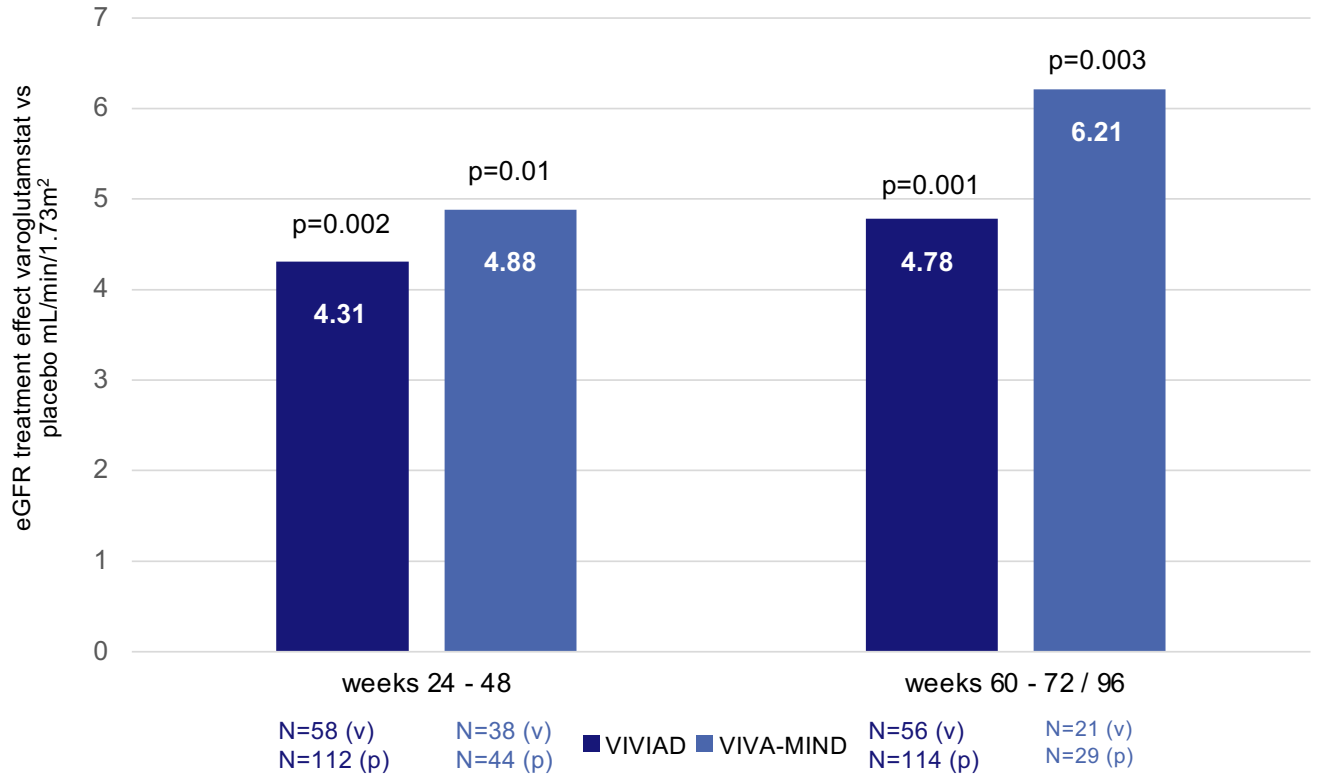
eGFR: estimated glomerular filtration rate, based on creatinine and calculated using the modification of diet in renal disease (MDRD) method. eGFR data were analysed over time using MMRM (mixed model for repeated measures) modelling including fixed effect terms for treatment, visit window and treatment-by-visit interaction using data from patients randomized to 600 mg and all visits on treatment, for VIVIAD (4 – 96 weeks) and VIVA-MIND (4 – 72 weeks)

Consistent improvement in kidney function and effect size across distinct treatment periods in both studies

Sensitivity analysis; all patients randomized to 600 mg BID varoglutamstat (v) and placebo (p)

eGFR treatment effect:

Difference between varoglutamstat and placebo (LSmean change from baseline)



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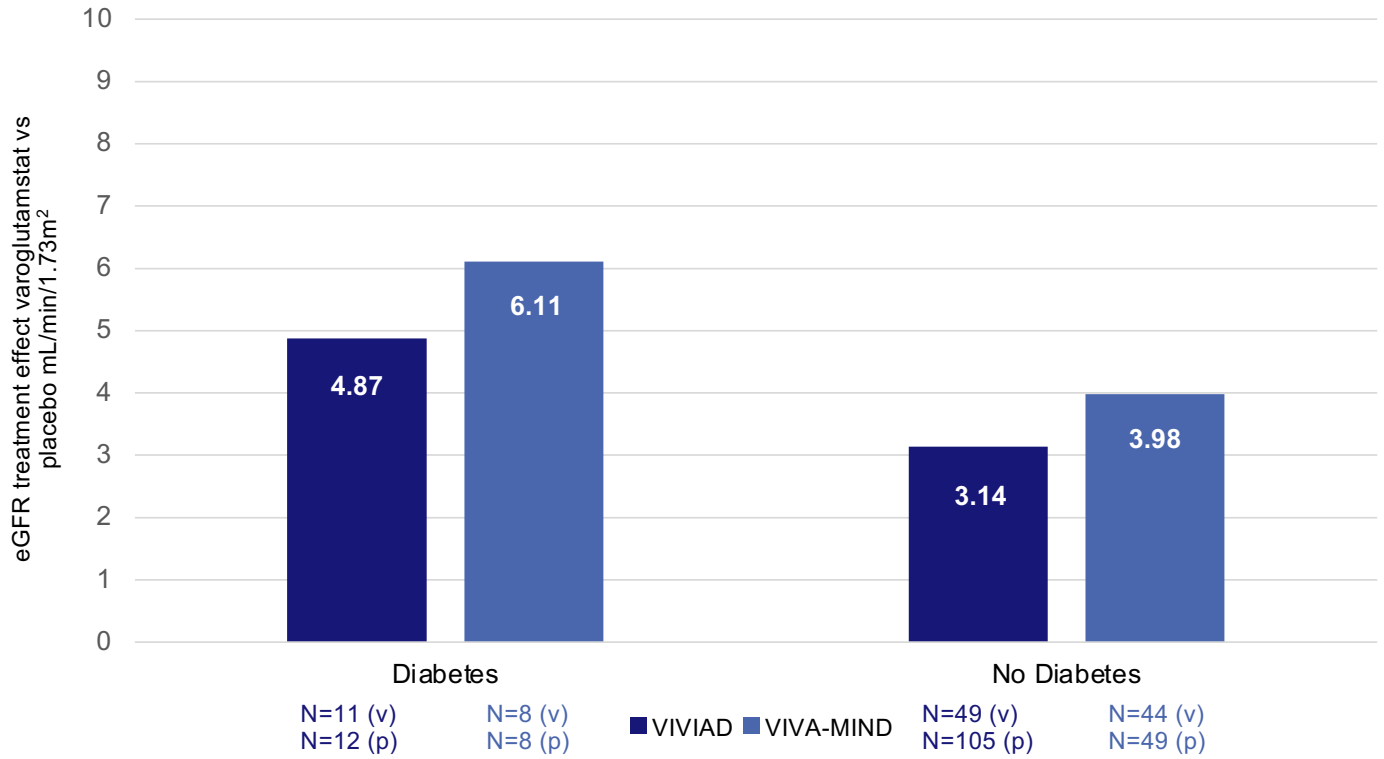
Results are nearly identical between studies when comparing treatment effect in patients with or without diabetes, with consistently higher effect in diabetes

Subgroup analysis; with and without diabetes; 600 mg BID varoglutamstat (v) and placebo (p)

eGFR treatment effect:

Difference between varoglutamstat and placebo (LSmean change from baseline)



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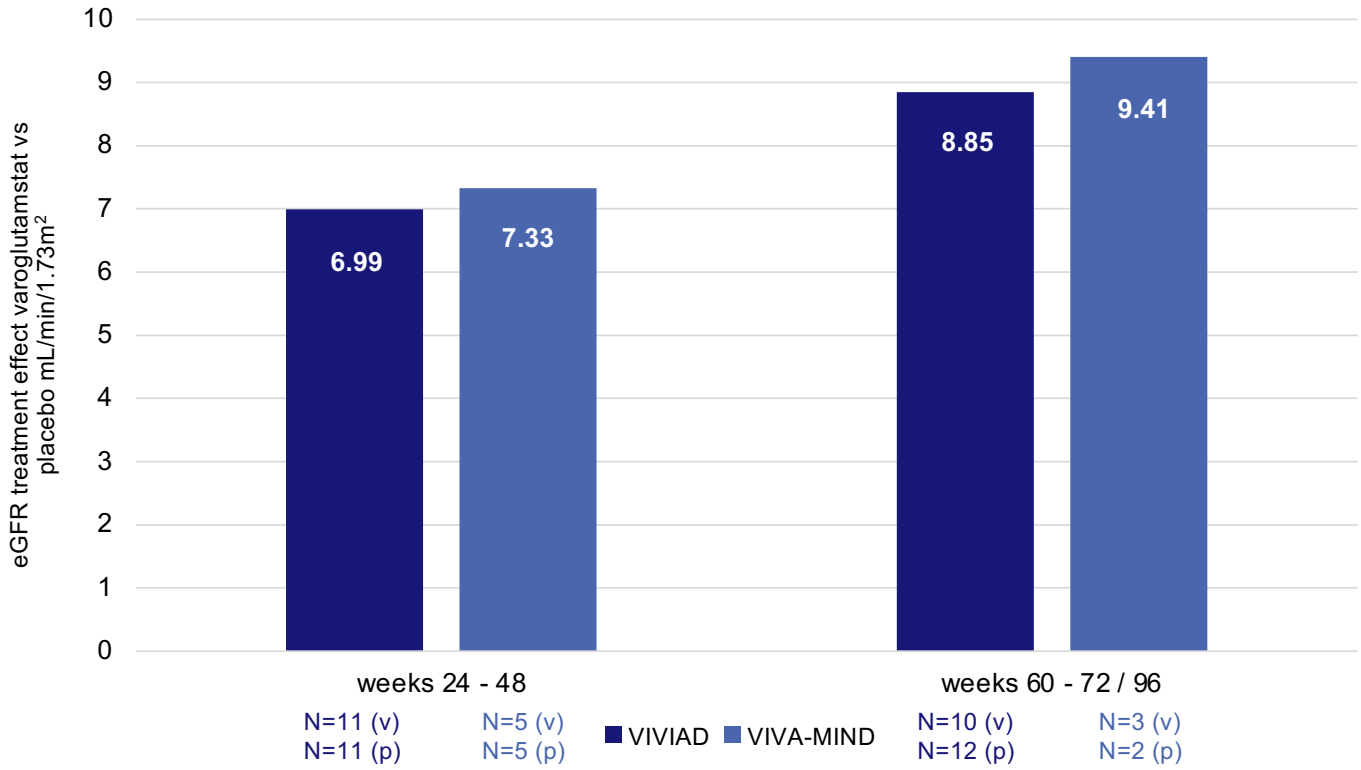
Consistent and very strong efficacy signal and large treatment effect observed in both studies in patients with diabetes at different timepoints

Subgroup analysis; patients with diabetes; 600 mg BID varoglutamstat (v) and placebo (p)

eGFR treatment effect:

Difference between varoglutamstat and placebo (LSmean change from baseline)



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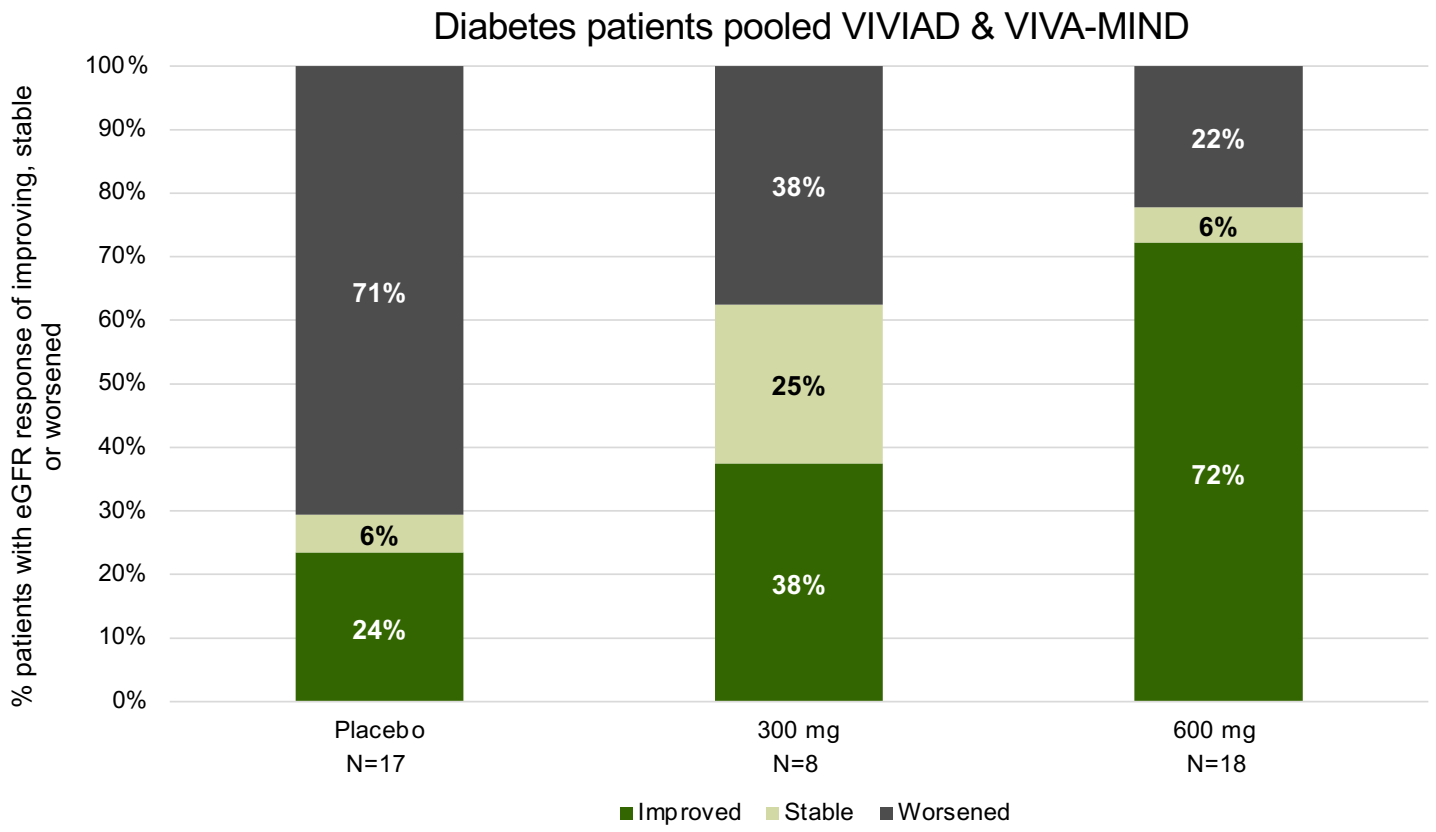
Responder analysis: kidney function predominantly improved or stabilized in varoglutamstat treated patients compared to a decline in the placebo group



Classification of eGFR response
(change vs. baseline, mL/min/1.73m^2)

- Improved: ≥ 2 mL above baseline
- Stable: ≥ 0 - < 2 mL above baseline
- Worsened: < 0 mL below baseline

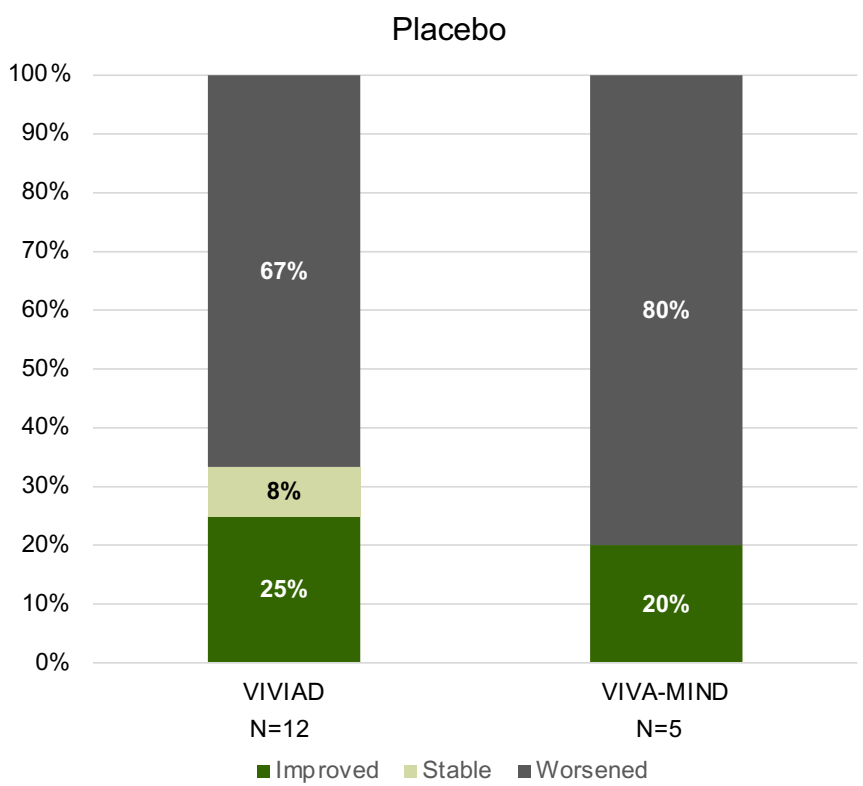
Response analysis (proportional odds)

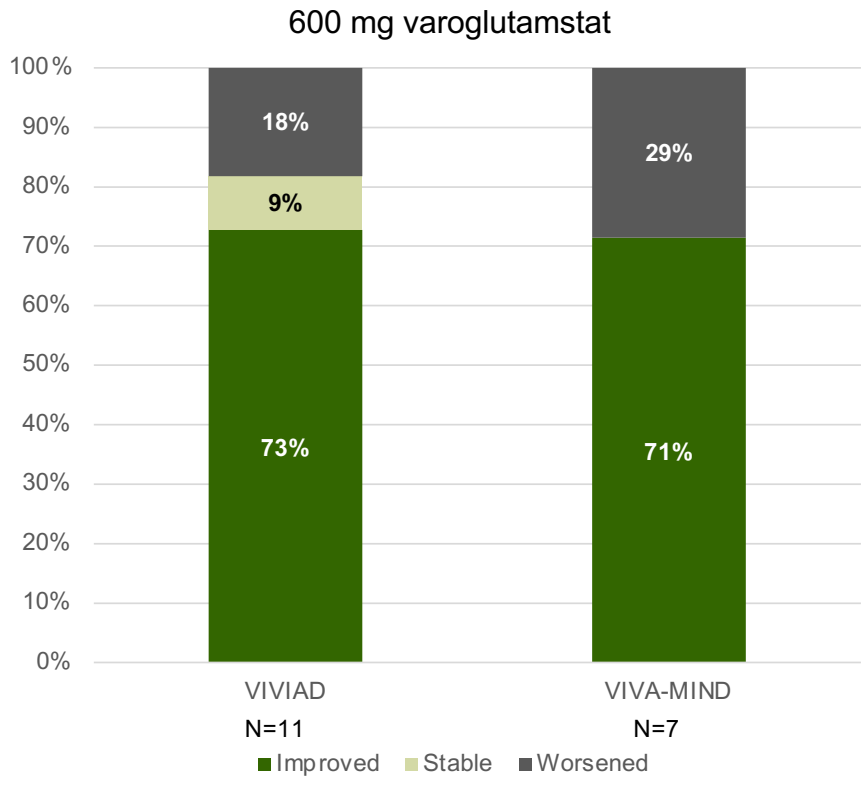
300 mg vs placebo
2.91, 95% CI (0.55, 15.53), p=0.2106

600mg vs. placebo
9.20, 95% CI (2.14, 39.50), p=0.028

Data based on mean eGFR (week 12 – EOT) vs. baseline; combined data from VIVIAD and VIVAM-MIND studies by dose; average treatment duration in VIVIAD was 76 weeks (70 weeks in the diabetes subgroup) and in VIVA-MIND was 46 weeks; diabetes subgroup as defined on slide 9; CI: confidence interval. Some figures do not sum to 100% due to rounding

Sensitivity analysis: side by side comparison of responder analysis in diabetes patients shows high consistency between studies in diabetes patients

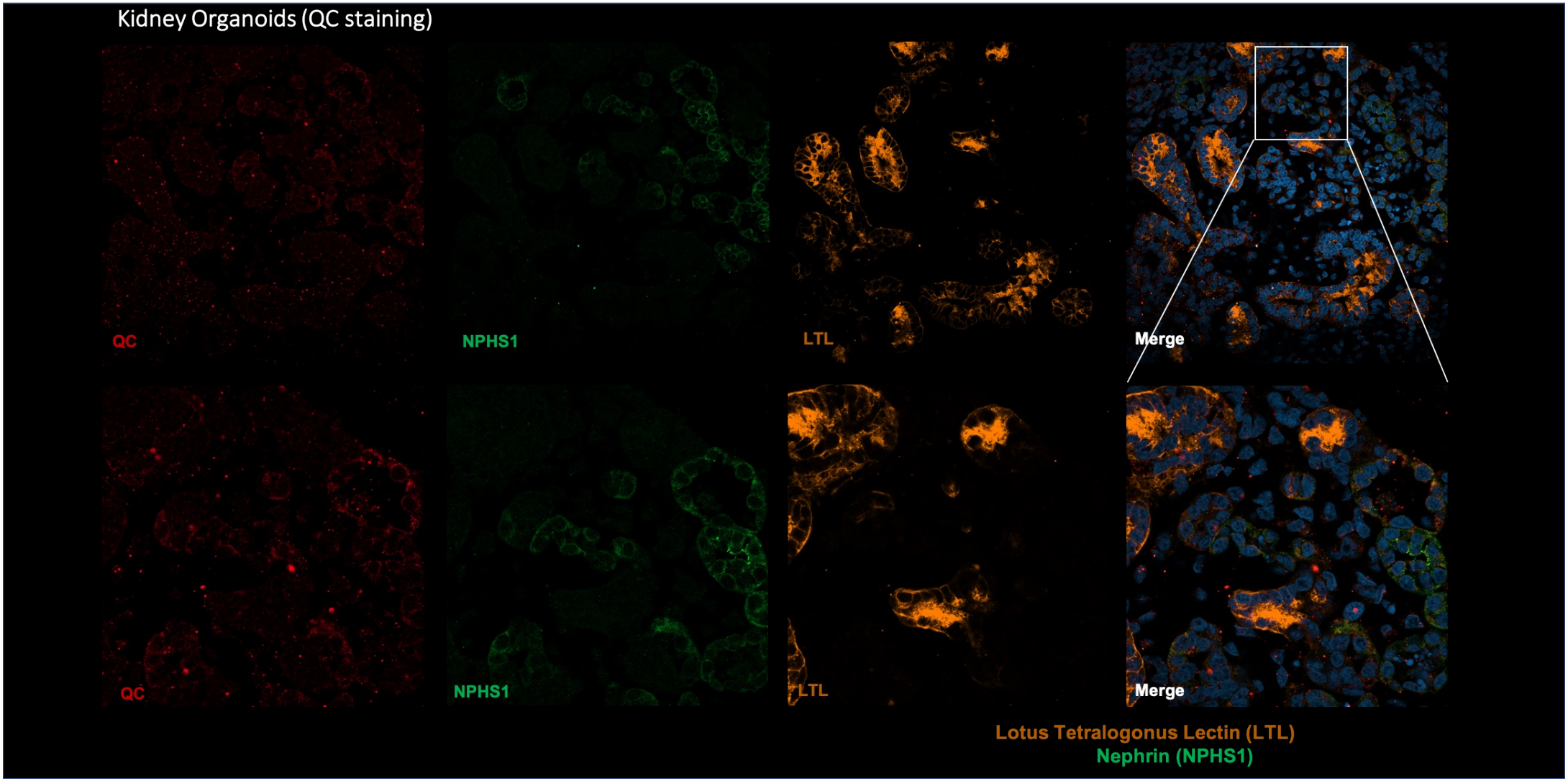




Classification of eGFR response (change mean eGFR (week 12-EOT) vs. baseline, mL/min/1.73m^2):
Improved: ≥ 2 mL above baseline, Stable: ≥ 0 - < 2 mL above baseline, Worsened: < 0 mL below baseline

Data based on mean eGFR (week 12 – EOT) vs. baseline; data from VIVIAD and VIVAM-MIND studies 600 mg BID dose vs. placebo; average treatment duration in VIVIAD was 76 weeks (70 weeks in the diabetes subgroup) and in VIVA-MIND was 46 weeks; diabetes subgroup as defined on slide 9

Localization of QC in human kidney organoids



Significant need for therapies that can stabilize / improve kidney function

Inhibition of QPCTL represents a novel target to address unmet needs in kidney disease

Need

- Current standard of care reduces risk of CKD progression by approx. 1/3
- Significant remaining risk of disease progression or premature death in a growing population
- Significant burden for patients and healthcare providers
- Urgently need therapies that reduce or reverse risk of progression in CKD/DKD and rare kidney disorders

Michael Schaeffer, PhD

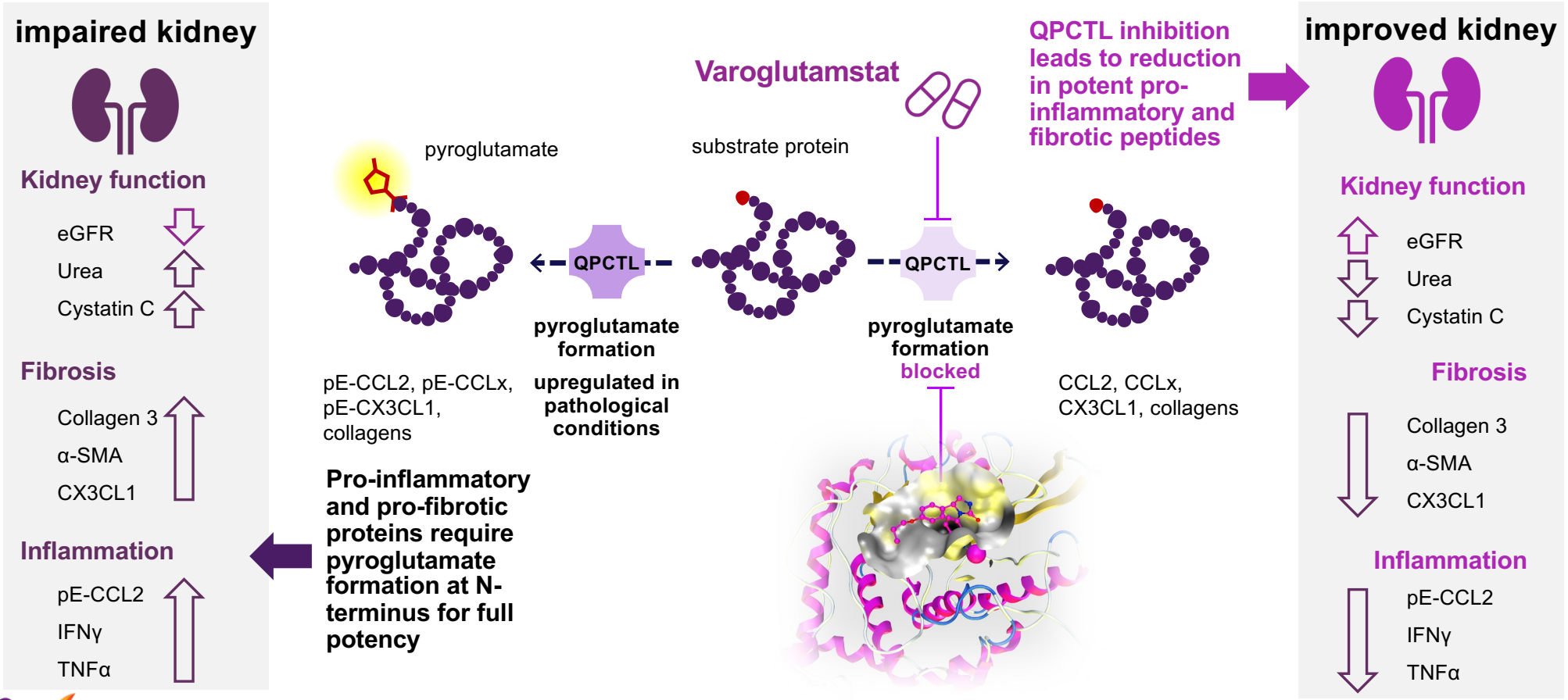
CBO
Vivoryon Therapeutics

A convenient new treatment option to fill the existing gap in kidney diseases

Varoglutamstat has the potential to stabilize/counteract continuous decline in kidney function

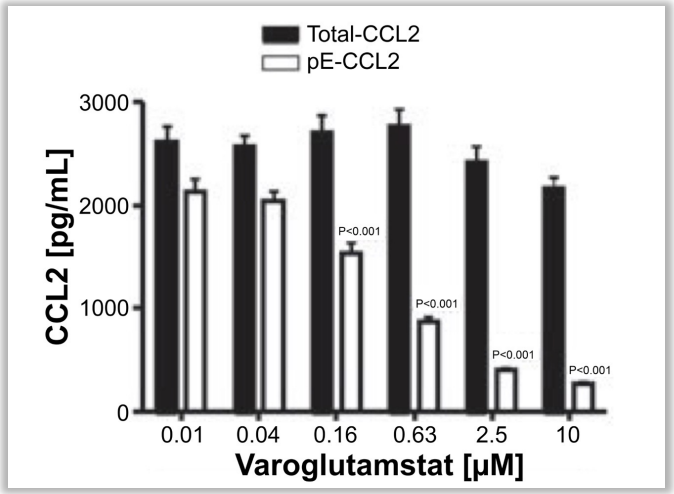
- Single agent oral compound
- First-in-class mechanism of action addressing key pathways in inflammation / fibrosis
- Statistically significant and clinically meaningful improvement of eGFR in two independent double-blind placebo-controlled studies
- Effect size substantially larger in diabetes population vs. non-diabetes population
- Clearly differentiated profile with >70% patients showing improvement or stabilization of eGFR in diabetes subgroup
- Excellent safety profile consistent across two years of study duration
- MOA and safety make varoglutamstat suitable for treatment on top of SOC and potential combinations with other therapeutics

Groundbreaking discovery: Inhibition of QPCTL reduces kidney inflammation and fibrosis, and improves pathophysiology and kidney function

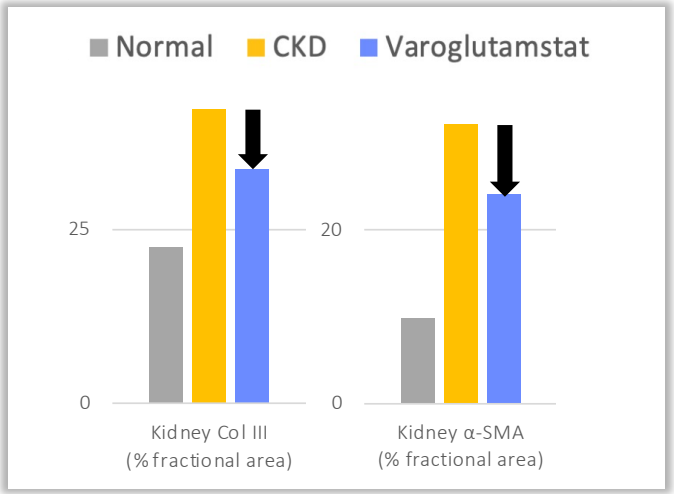


Kehlen et.al. Biosci Rep, 2017; Cynis et.al. EMBO, 2011; Cynis et al, Int. J Exp Path, 2013, Kanemitsu et al., 2021; Vivoryon company data, 2024; QPCTL - Glutaminyl-peptide cyclotransferase-like protein. pE-CCLx – other potential pE chemokines. Notes: Graphic is for illustrative purposes only.

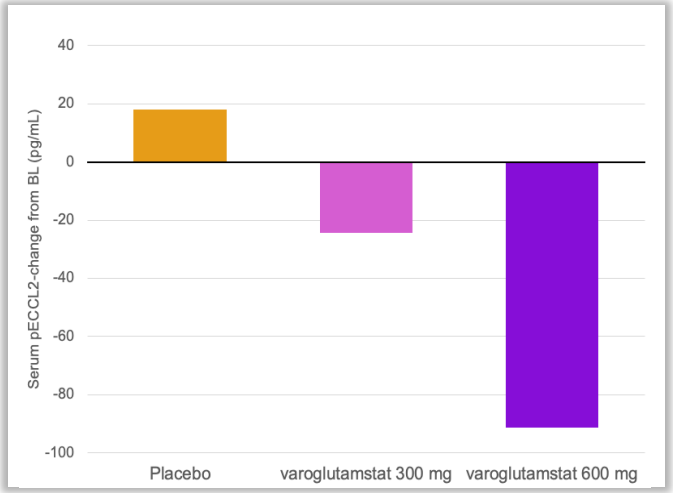
Robust evidence demonstrating inhibition of intracellular QPCTL decreases activity of pro-inflammatory cytokines and kidney fibrosis



Decrease of pE-CCL2 levels by QPCT/L inhibitor application. LPS-stimulation of RAW264.7 cells. Analysis of varoglutamstat effect on total-CCL2 and pE-CCL2.



Histological changes show improvement of kidney Col-III and α-SMA. Adenine-induced mouse model of CKD.

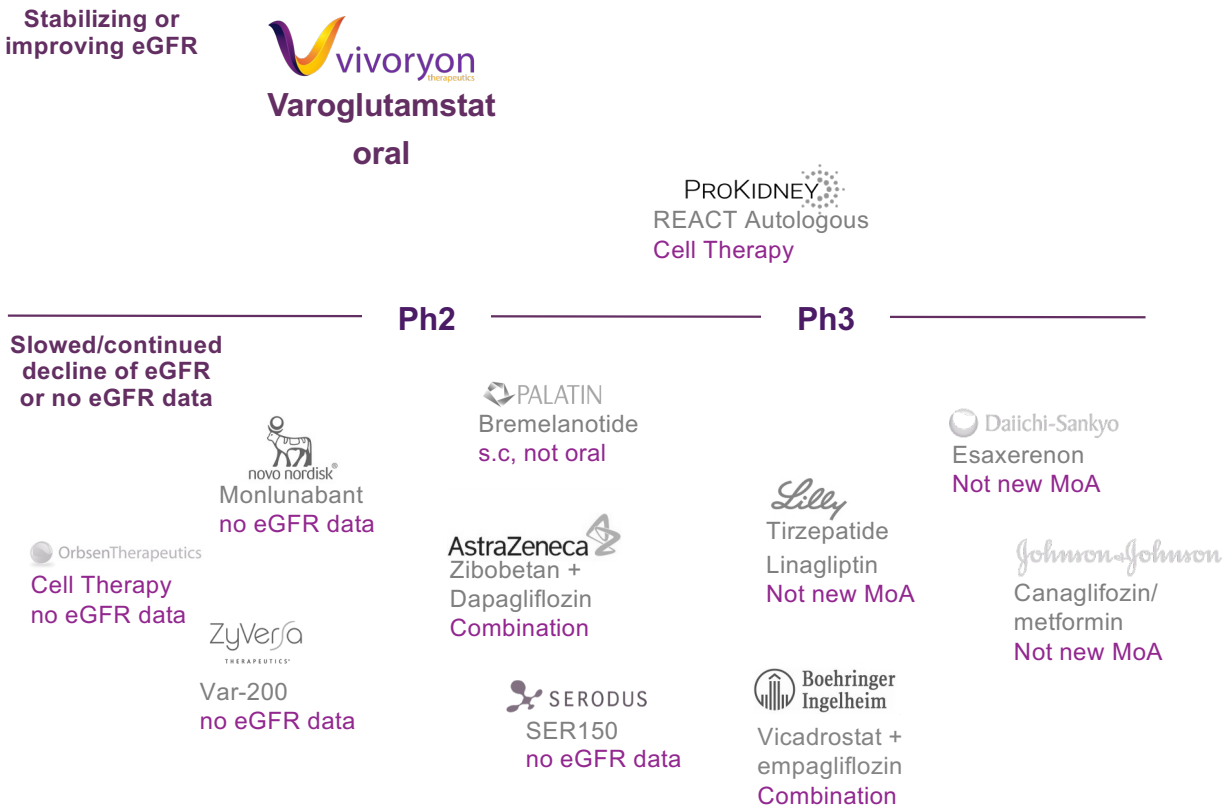


Median reduction in pE-CCL2 levels compared to baseline with varoglutamstat. VIVIAD, total population, at week 48.

Company data, and adapted from Cynis et al., Int. J Exp Path, 2013

Vivoryon's varoglutamstat is well-positioned in competitive landscape

Key characteristics Varoglutamstat	
oral	✓
Novel MoA First-in-class QPCTLi	✓
Single agent with potential use in combination	✓
Demonstrated long-term effect on eGFR	✓
Long-term Safety	✓



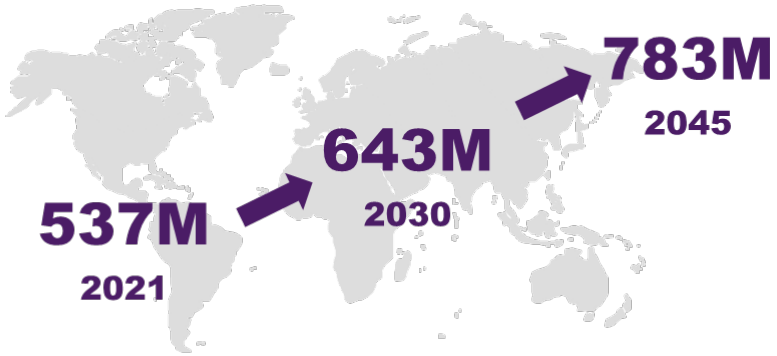
Currently marketed standard of care therapeutics including RAASi, SGLT-2i, GLP-1 RA, MRA show slowing but no improvement of eGFR

eGFR: estimated glomerular filtration rate; RAASi: renin-angiotensin-aldosterone system inhibitor; SGLT-2i: sodium-glucose co-transporter 2 inhibitor; GLP-1 RA: Glucagon-like peptide-1 receptor agonists; MRA: mineralocorticoid receptor antagonist; above graphic for representation purposes only, includes select development candidates

Initial target market represents an attractive patient opportunity with potential label expansion to earlier stages of DKD / CKD

Diabetes is a significant and growing global challenge

(adults aged 20-79 years with diabetes, worldwide)[1]

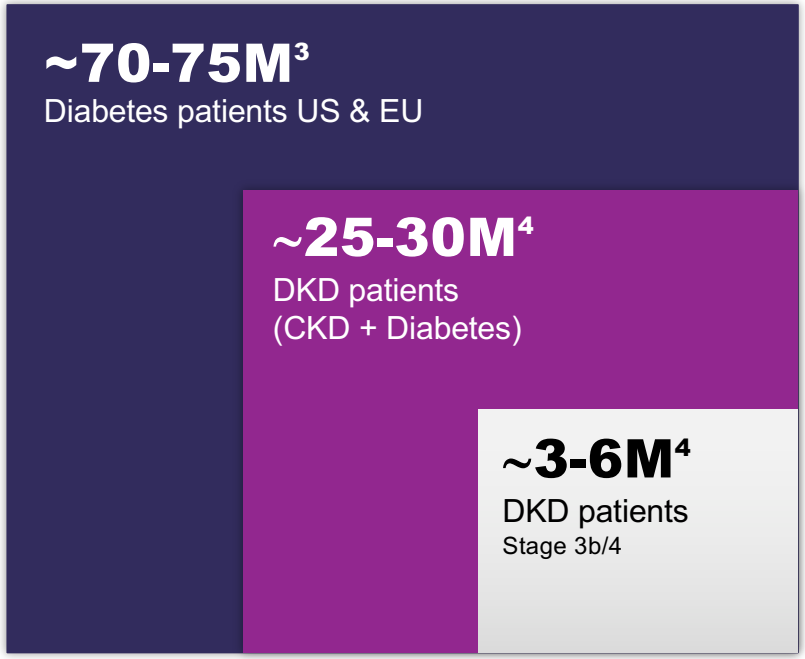


~40%

of people with diabetes may develop diabetic kidney disease (DKD)[2]

1 in 10

people with diabetes may end up with end-stage kidney disease[2]

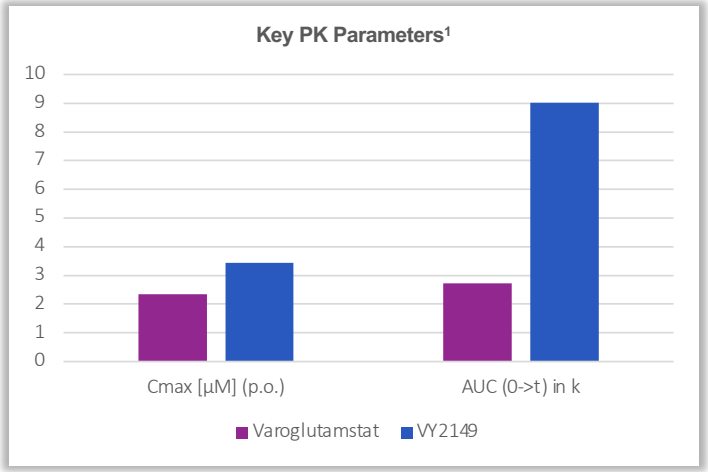


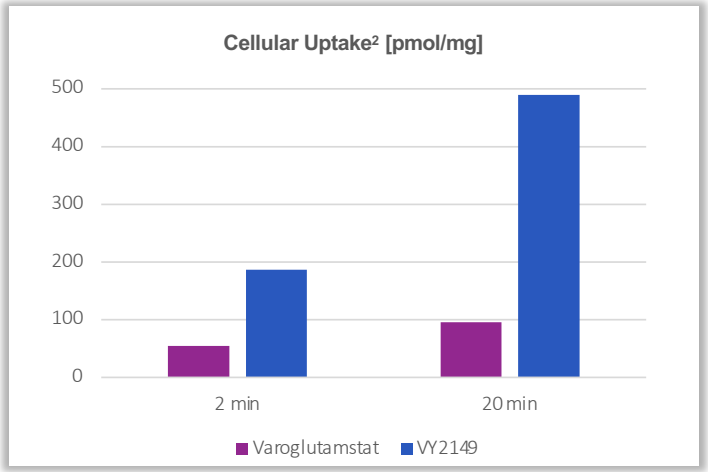
Total prevalent population

[1]International Diabetes Federation (IDF) Atlas 2021; [2]Qazi et al., EMJ Nephrol, 2022; [3]CDC National Diabetes Statistics Report 2024; Eurostat 2017; CDC Chronic Kidney Disease in the United States, 2023; Brück et al., J Am Soc Nephrol, 2015; Sundström et al., The Lancet, Regional Health Europe, 2022; [4]Prevalent population assumptions based on internal analyses using a combination of public sources and management estimates, including Wu et al., BMJ Open Diabetes Research and Care, 2016; Feng et al., Kidney Med, 2022, CDC Kidney Disease Surveillance System (NHANES); This information may prove to be inaccurate because this information cannot always be verified with complete certainty due to the limits on the availability and reliability of raw data, the voluntary nature of the data gathering process and other limitations and uncertainties.

New development compound VY2149 shows improved cellular uptake, PK profile and superior outcomes in kidney animal studies

- Higher intracellular QPCTL inhibition translates to better activity, lower doses and the opportunity for once daily dosing
- Pre-clinical stage follow-on candidate VY2149, has shown improved molecular properties including
  - Improved peak concentration (Cmax) of VY2149 compared to varoglutamstat at comparable bioavailability upon oral dosing
  - Markedly increased overall drug exposure (AUC)
  - Significantly higher passive uptake into cells
- Assessment of once daily dosing for VY2149 in an animal model has shown strong effects on eGFR, creatinine, cystatin C levels and α-SMA levels and collagens





[1] *Single low dose (10mg/kg); p.o.= oral; PK = pharmacokinetics); AUC = Area under the curve; Cmax = peak concentration*

[2] *Passive uptake into HEK293 cells incubated for 2 vs. 20 min with 1 µM compound in medium (37°C); reported as pmol/mg protein of a reference protein.*

Pipeline focused on kidney disorders and inflammatory/fibrotic diseases

	Program	Approach	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Status
Inflammatory/fibrosis incl. kidney	DKD (Varoglutamstat/PQ912)	SMI QPCT/L	*POC in VIVIAD & VIVA-MIND results*					Preparing for Phase 2b DKD study
	Kidney orphan diseases (Varoglutamstat/PQ912)	SMI QPCT/L			Pre-IND			Pre-clinical orphan disease models
	Kidney disorders, fibrotic/inflammatory (VY2149)	SMI QPCT/L			Pre-IND			
	Fibrotic indications (NCE)	SMI Meprin			Research program			
Alzheimer's disease	Varoglutamstat (PQ912)	SMI QPCT/L						AD program: discontinued after negative topline data March 2024 (VIVIAD) & December 2024 (VIVA-MIND)
	Varoglutamstat (SIM0408, PQ912)	SMI QPCT/L	*CTA approval in China*					Partnered with Simcere in Greater China; under evaluation
	PBD-C06	mAb N3pE amyloid			Pre-IND			Partnered with Simcere in Greater China; under evaluation

DKD: diabetic kidney disease; SMI: small molecule inhibitor; IND: investigational new drug; NCE: novel chemical entity; CTA: Clinical Trial Application; mAb: monoclonal antibody

Kevin Carroll, PhD

CEO
KJC Statistics

Statistical considerations of varoglutamstat's effect on eGFR

Kevin Carroll, PhD

Meta-Analysis VIVIAD & VIVA-MIND

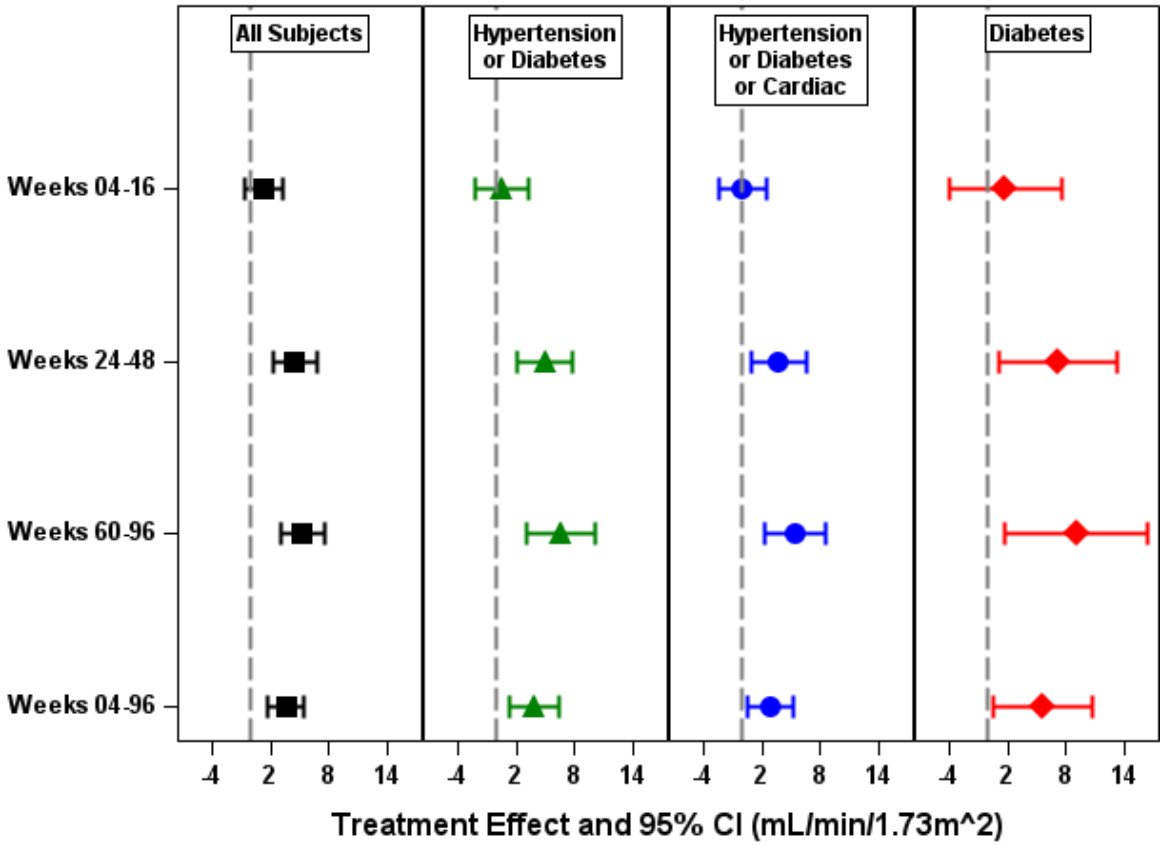
- Small N per trial, obvious approach is to combine the data via meta-analysis
- Not simple 'pooling', but stratified combination of treatment effects across trials
- Trial estimates are denoted $\hat{\theta}_i \sim N(\theta, v_i)$
- Fixed Effects Meta-Analysis [Homogeneity of Results]
  - $\hat{\theta}_F = \{\sum_i^k \hat{\theta}_i / v_i\} / \{\sum_i^k 1/v_i\}$ and $\hat{\theta}_F \sim N\left(\theta, \{\sum_i^k 1/v_i\}^{-1}\right)$
- Random Effects Meta-Analysis [Heterogeneity of Results]
  - $\theta_i \sim N(\theta, \tau^2)$ and $\hat{\theta}_i | \theta_i \sim N(\theta, v_i)$ $so\ that$ $\hat{\theta}_i \sim N(\theta, v_i + \tau^2)$
  - $\hat{\theta}_R = \{\sum_i^k \hat{\theta}_i / (v_i + \tau^2)\} / \{\sum_i^k 1/(v_i + \tau^2)\}$ and $\hat{\theta}_R \sim N\left(\theta, \{\sum_i^k 1/(v_i + \tau^2)\}^{-1}\right)$
  - Estimate τ^2 $from\ the$ chi-square test for heterogeneity

Meta-Analysis, Diabetic Subjects

Time Period	Trial	N Placebo	N Varoglutamstat 600 mg	Treatment Effect & 95% CI (mL/min/1.73m²)		P-value	Interaction P-value
Weeks 04-16	VIVA-MIND	8	8	3.548	(-3.773, 10.869)	0.3232	0.3451
	VIVIAD	12	11	-2.539	(-13.46, 8.378)	0.6415	0.3451
	Fixed	20	19	1.756	(-4.003, 7.514)	0.5502	0.3451
	Random	20	19	1.756	(-4.003, 7.514)	0.5502	0.3451
Weeks 24-48	VIVA-MIND	5	5	7.326	(-2.447, 17.099)	0.1361	0.9568
	VIVIAD	11	11	6.986	(-1.336, 15.307)	0.0967	0.9568
	Fixed	16	16	7.129	(1.053 , 13.205)	0.0215	0.9568
	Random	16	16	7.129	(1.053 , 13.205)	0.0215	0.9568
Weeks 60-96	VIVA-MIND	2	3	9.408	(-4.278, 23.095)	0.1736	0.945
	VIVIAD	12	10	8.849	(-0.066, 17.763)	0.0516	0.945
	Fixed	14	13	9.013	(1.779 , 16.247)	0.0146	0.945
	Random	14	13	9.013	(1.779 , 16.247)	0.0146	0.945
Weeks 04-96	VIVA-MIND	8	8	6.11	(-1.261, 13.480)	0.0991	0.8145
	VIVIAD	12	11	4.874	(-3.225, 12.972)	0.2255	0.8145
	Fixed	20	19	5.555	(0.421 , 10.689)	0.0339	0.8145
	Random	20	19	5.555	(0.421 , 10.689)	0.0339	0.8145

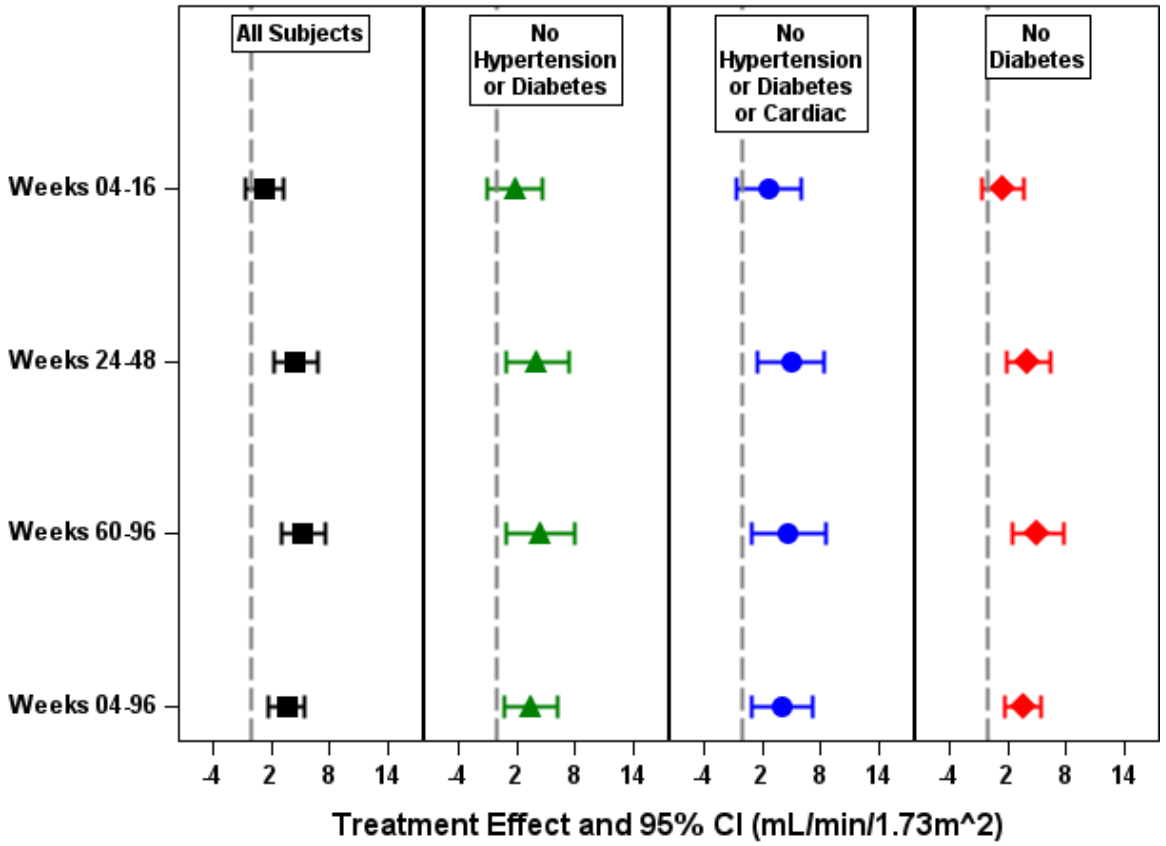
As of 15 Jan 2025

Meta-Analysis
All subjects and subgroups with risk factors for CKD



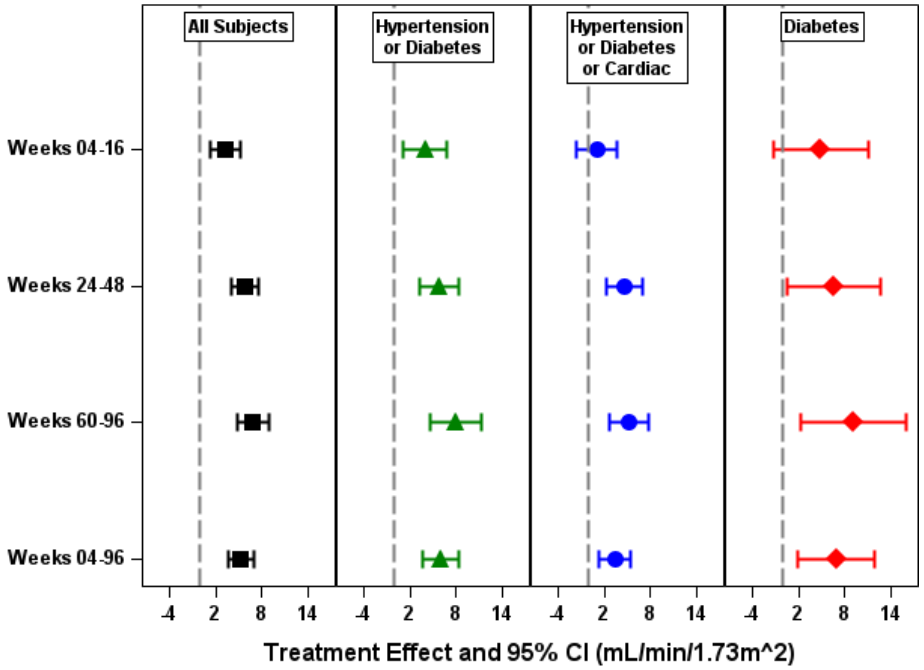
eGFR calculated with creatinine laboratory measurements as per protocol with MDRD formula

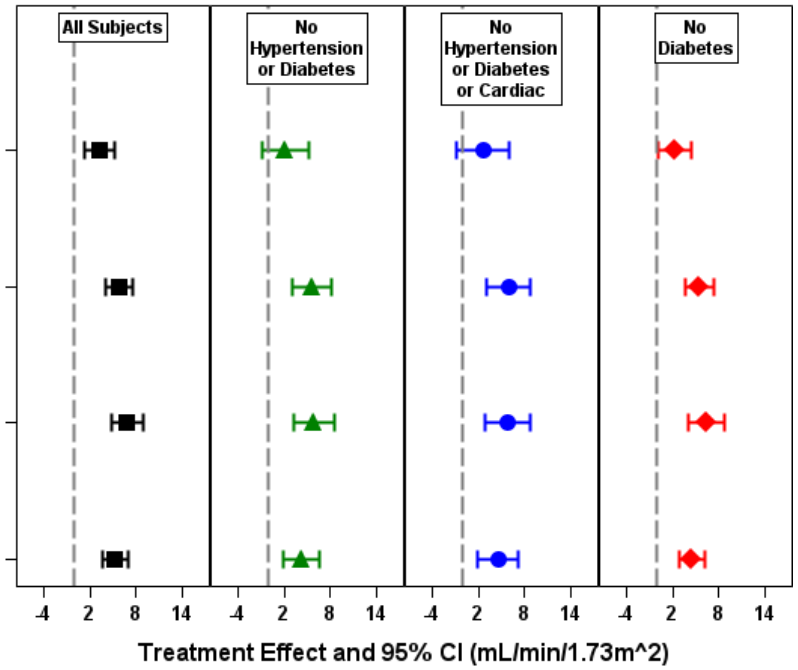
Meta-Analysis
All subjects and subgroups without risk factors for CKD



eGFR calculated with creatinine laboratory measurements as per protocol with MDRD formula

Meta-Analysis
Sensitivity Analysis: CKD-EPI remeasured creatinine





eGFR calculated with creatinine laboratory measurements performed after completion of the study with retained samples when available: remeasured; CKD-EPI formula

Phase II PoC Design in Diabetic Subjects

- Combined Study data for eGFR give :
  - Residual Error Variance 103 $\{mL/min/m^2\}^2$
  - Auto correlation = 0.43
  - SD mean eGFR over 18-24 weeks = 7.6 $mL/min/m^2$
- PoC Design
- N=40 per arm
- Primary Endpoint = average eGFR over 18-24 weeks
  - Assessment of eGFR @ 18, 21 and 24 weeks
- 80% power to for a hypothesized difference of 4.5 $mL/min/m^2$
- Minimum observed difference to yield $p<0.05$ 1-sided = 3 $mL/min/m^2$

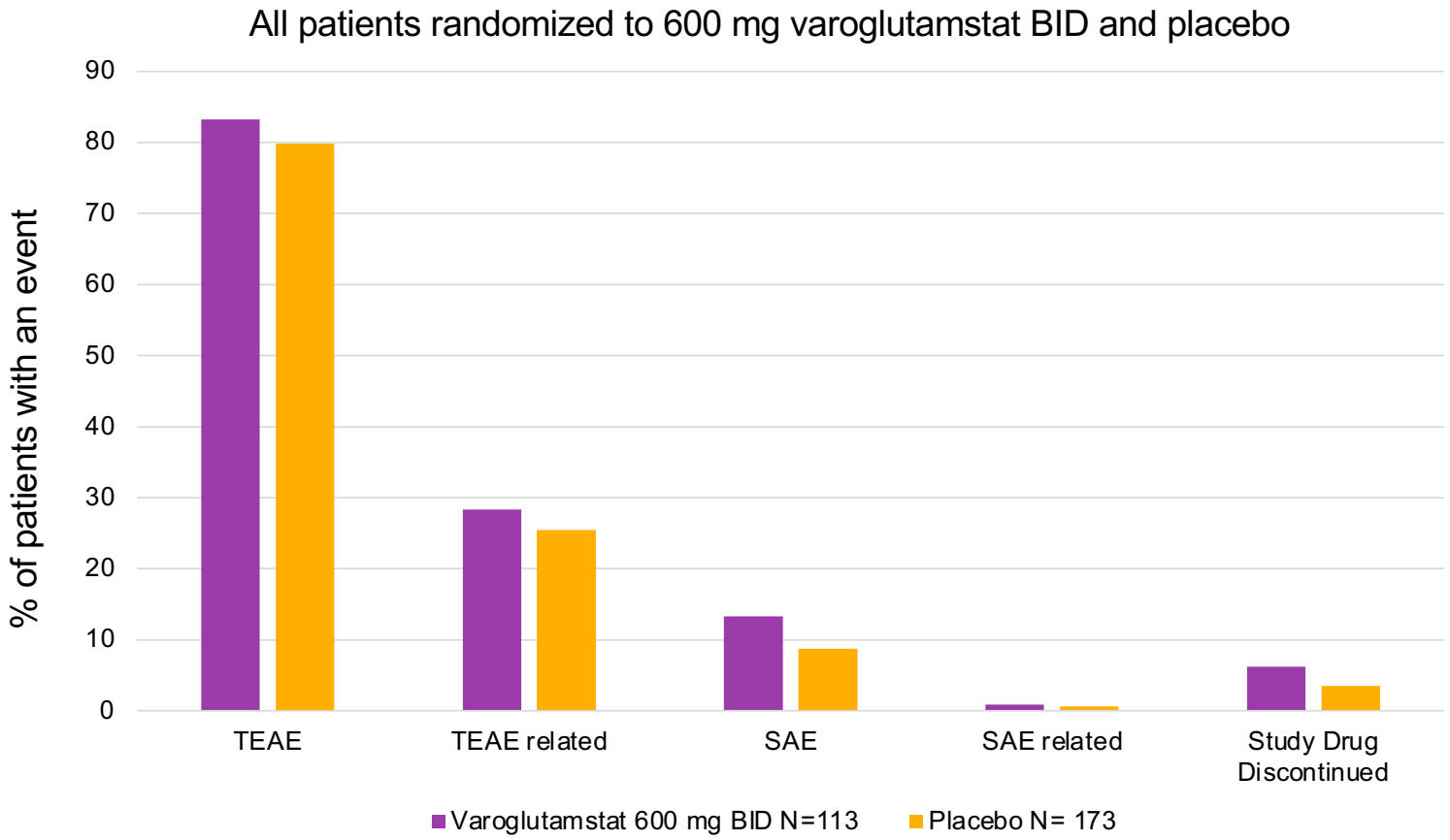
Phase III Design in Diabetic Subjects

- Endpoint: eGFR slope over 2 years
- VIVIAD data give:
  - Between slope variance component = 32.15 $\{mL/min/m^2\}^2$
  - Residual variance component after regression = 70.4 $\{mL/min/m^2\}^2$
- eGFR measured every 2 months
- N=150 per arm provides 90% power to test the hypotheses the reduction in the annualized rate of eGFR decline is 2.65 mL
  - Minimum observed difference to yield $p<0.05$ 2-sided = 1.6 $mL/min/m^2$

Frank Weber, MD

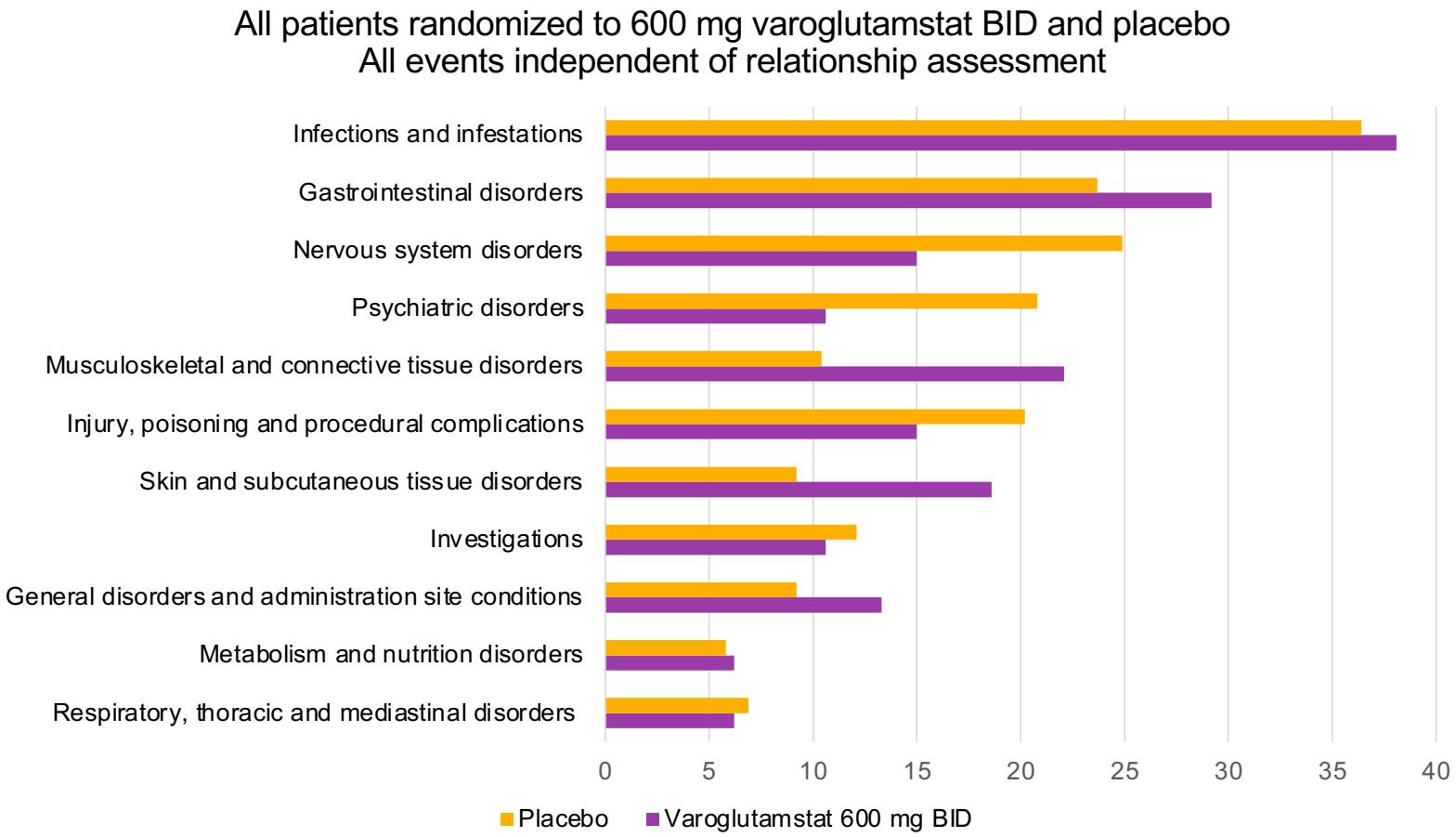
CEO
Vivoryon Therapeutics

Safety: pooled analysis of VIVIAD and VIVA-MIND
600 mg varoglutamstat is well tolerated



TEAE: Treatment Emergent Adverse Event; SAE: Serious Adverse Event

Pooled safety analysis VIVIAD and VIVA-MIND: TEAE by system organ class

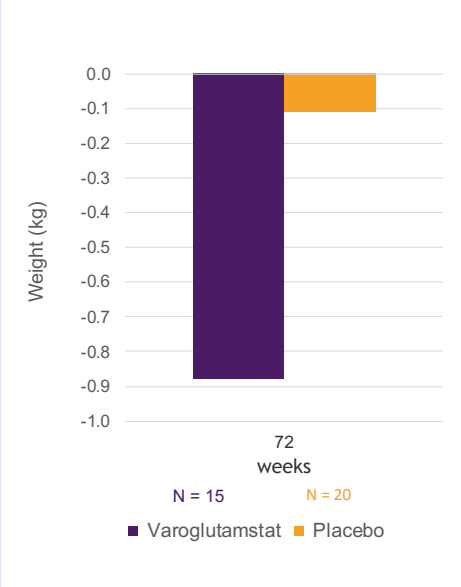


TEAE: Treatment Emergent Adverse Event; A 5% cut-off (aggregated) was applied.

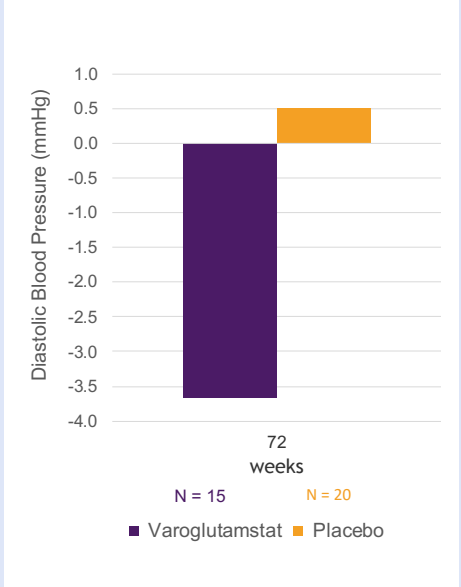
Additional health benefits observed in VIVA-MIND (total population*) at week 72

Effects on weight loss, blood pressure and liver enzymes show same trends as in VIVIAD

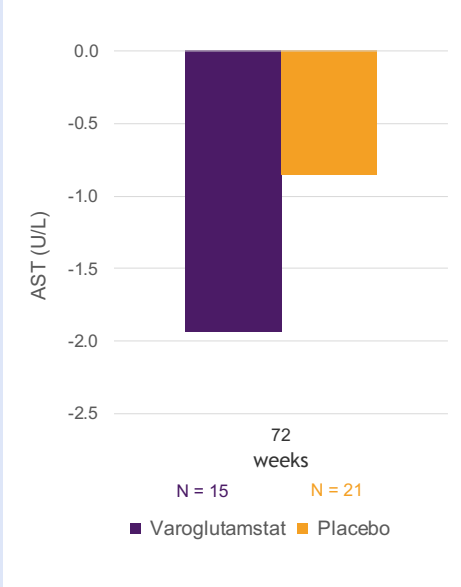
Change from baseline in **weight** (kg)

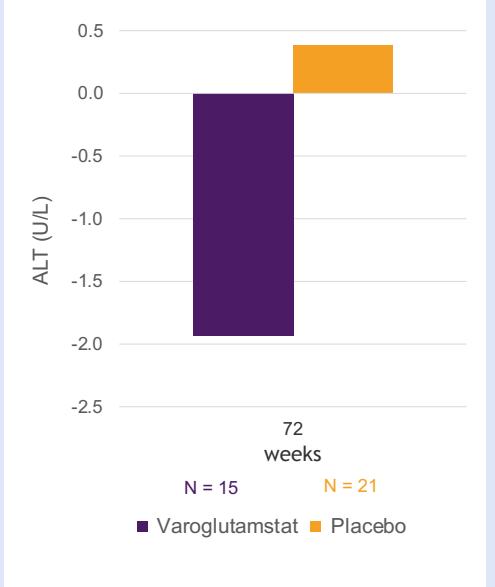


Change from baseline in **blood pressure** (mmHg)



Change from baseline in liver **transaminases** (IU/L)





** An analysis in the subgroup of patients with diabetes was not feasible with only 6 patients being treated for 48 weeks*

Phase 2b study objectives and timelines

Primary Objective:

Investigate the efficacy and safety of varoglutamstat on kidney function in patients with T2DM and CKD 3b and worse

Secondary Objectives

Explore the efficacy of a once daily dose of varoglutamstat

Generate further evidence of the mechanism of action

Generate data on the effect of varoglutamstat on frequently concomitantly affected organs in T2DM patients: liver, vasculature, bodyweight

Estimated timeline to topline readout of new Phase 2b study

Screening, recruitment & randomization
- ~9 months
- Sites in US and Europe

All patients treated for minimum of 30 weeks

Last patient reaches 30 weeks
- ~15 months

Primary endpoint topline results
- ~18 months

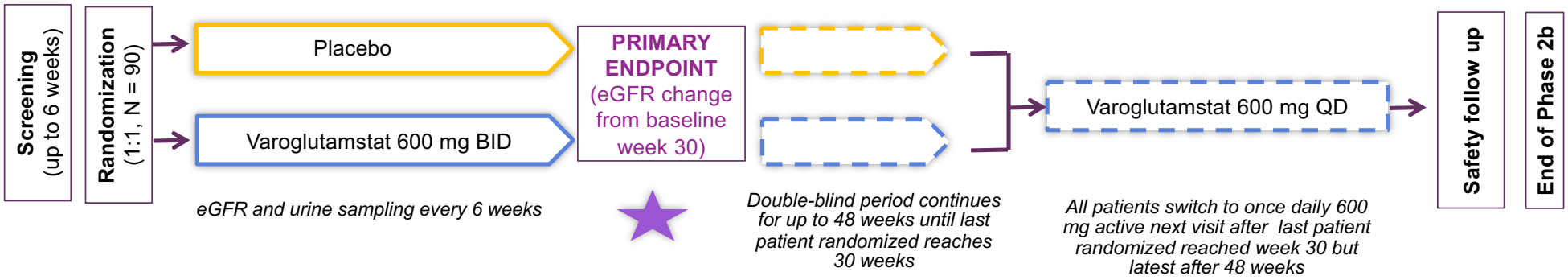
Further results and analysis

Once daily dose results

T2DM: type 2 diabetes mellitus, CKD: Chronic Kidney Disease

Double-blind placebo-controlled Phase 2b study in patients with T2DM and CKD stages 3b and worse on top of standard of care (SoC)[1]

Draft trial design based on robust data from VIVA-MIND and Phase 2 meta-analysis



Patient characteristics

- T2DM patients with Stage 3b+ CKD; all patients on standard of care medicines (SoC)

Endpoints

- **Primary:** eGFR change from baseline to last visit
- **Secondary:** UACR (albuminuria)
- **Exploratory:** Inflammatory, metabolic and fibrotic biomarkers liver transaminases, liver ultrasound (fibroscan)

Stratification

- By CKD severity
- Patients with SGLT-2 versus no SGLT-2
- Patients with GLP-1 versus without GLP-1

*[1] Clinical trial subject to further funding / partnership; [2] Based on typical industry costs for Phase 2 in similar indications; ^Baseline: average of two measurements during screening / baseline 6 weeks apart; *Month 6: mean of week 24 and week 30 result; BID: twice daily; QD: once daily; GLP-1: Glucagon-like peptide-1; SGLT-2: sodium-glucose co-transporter 2*

QPCTL inhibitors have a large market potential: Development opportunities across a range of diseases driven by underlying inflammation / fibrosis

DKD / CKD / earlier stages

Replication of a sustained improvement of kidney function in two independent Phase 2 studies[1]

Initial focus on stage 3b/4 DKD given high unmet need and large effect in diabetes subgroup

Opportunity to expand market potential by moving into earlier and later stage DKD / CKD

Rare kidney diseases

e.g. Alport / Fabry disease

Novel mode of action, effect on inflammatory markers and observed effect on kidney function holds promise for QPCTL inhibitors in certain rare diseases

Disorders progressing through inflammation & fibrosis

e.g. NAFLD

NAFLD is the most prevalent form of liver disease which may advance to metabolic dysfunction-associated steatohepatitis ("MASH") and cirrhosis

In vivo proof of concept in NAFLD mice[2]

[1] VIVIAD and VIVA-MIND Phase 2 studies in early Alzheimer's disease (AD) included prospectively defined measures of kidney function as safety and other exploratory endpoints, the primary and secondary endpoints in early AD were not met; [2] Cynis et al., 2013

Vivoryon: Poised to improve kidney health with varoglutamstat's novel mechanism of action and breakthrough clinical study results

Unique oral asset with MOA targeting inflammation

Developed first in class oral **QPCTL inhibitor;** only one in clinic to show **improvement in kidney function** in elderly population[1]

Compelling Phase 2 results replicated in two independent studies

Unprecedently large and **sustainable improvement** in kidney function, especially in 'diabetes' subgroup; **large long-term safety data base**

Actionable, risk-contained plan for Phase 2b study in DKD[2]

Next steps in target population founded on statistical insights from **robust, long-term Phase 2 data**

Extensive intellectual property portfolio[3]; pipeline of additional early-stage QPCTL inhibitors; experienced management team with track record in inflammation and business development

[1] VIVIAD and VIVA-MIND Phase 2 studies in early Alzheimer's disease (AD) included prospectively defined measures of kidney function as safety and other exploratory endpoints, the primary and secondary endpoints in early AD were not met; [2] Subject to funding / partnership; [3]Composition of matter patent protection expected to 2044+ based on current composition of matter patent to 2031 with additional potential for Hatch-Waxman extension of up to 5 years, new patent filings being evaluated

Q&A

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