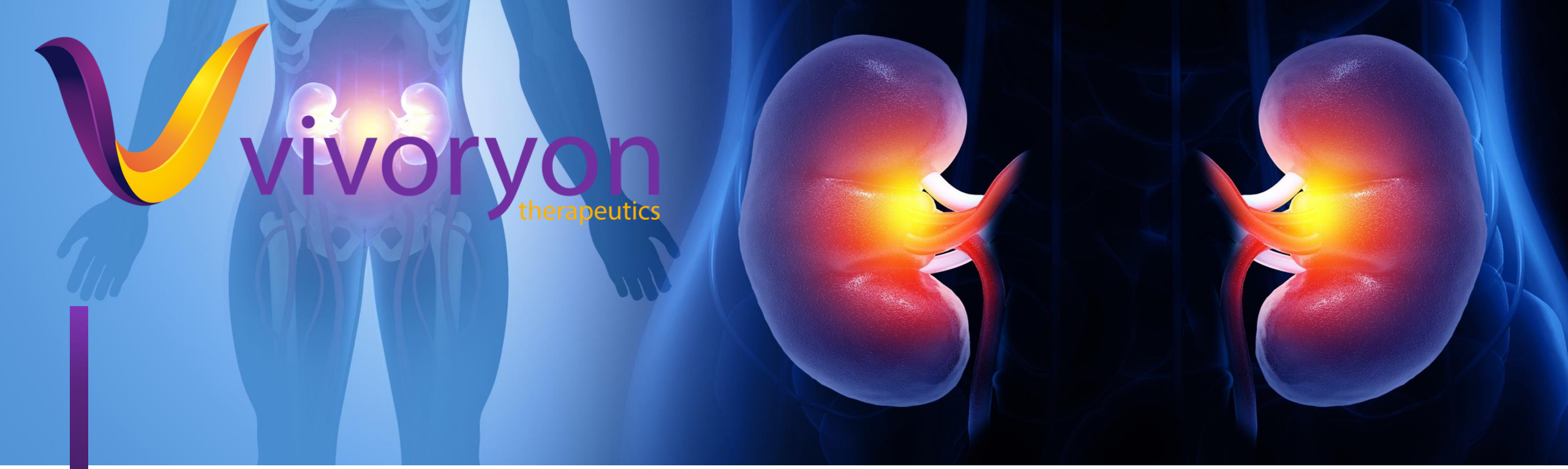

Innovation to Improve Kidney Health Outcomes
Lead Program: Varoglutamstat in Diabetic Kidney Disease

December 2025

Vivoryon Therapeutics N.V.

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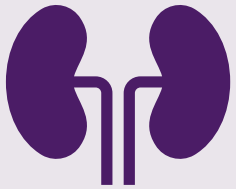
Forward-looking statements speak only as of the date they are made, and we do not undertake any obligation to update them in light of new information or future developments or to release publicly any revisions to these statements in order to reflect later events or circumstances or to reflect the occurrence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

Vivoryon's approach and expertise positions company for future growth and supports development of innovative oral therapies for kidney disease



Transforming the treatment of kidney disease

High medical need

Therapies needed that can stabilize or improve kidney function and prevent progression to kidney failure / end-stage kidney disease (ESKD)

Unique oral product profile shown to improve kidney function

Varoglutamstat improves kidney function and reduces key drivers of inflammation and fibrosis, setting it apart from other therapies

Program de-risked by compelling data

Statistically significant and clinically meaningful impact on kidney function demonstrated in two independent Phase 2 studies[1]

Significant opportunity for value creation

Clearly defined commercial strategy in promising therapeutic space, active big Pharma players, attractive time to market and long IP runway to 2044+*

*[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, *potential for Hatch-Waxman extension up to 5 years*

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

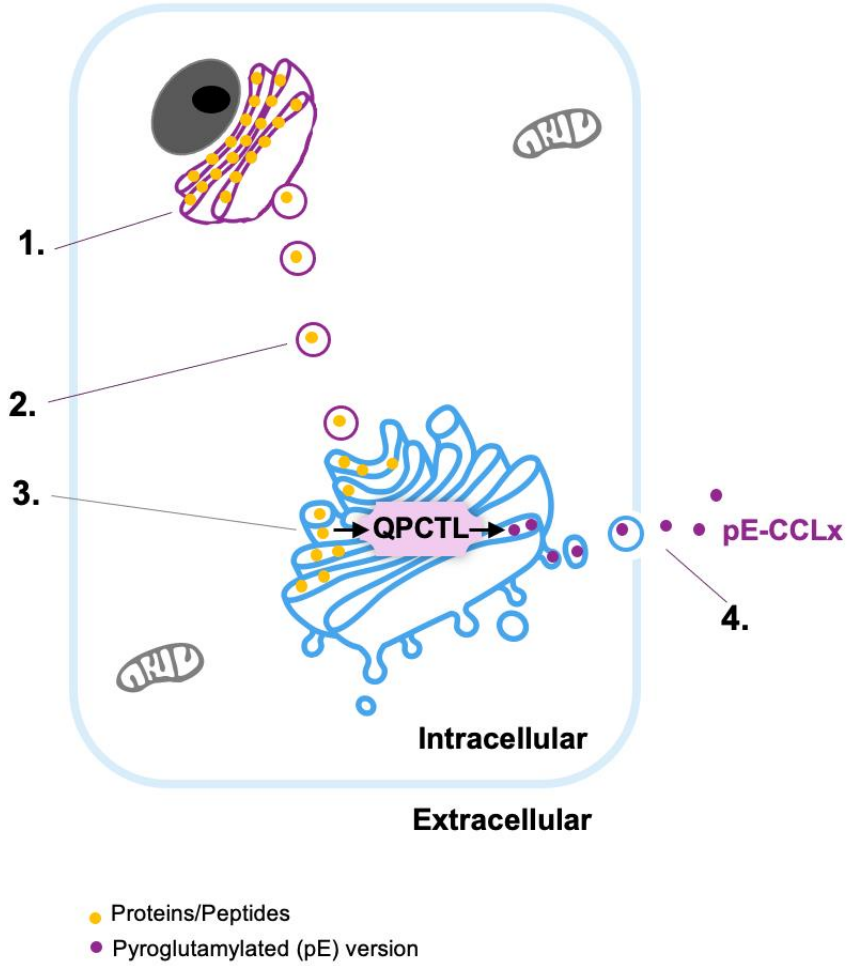
Vivoryon has identified QPCTL, an enzyme that creates pro-inflammatory pE-versions of key inflammatory proteins, as a promising target with potential to stabilize disease

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

Varoglutamstat's target QPCTL plays a crucial role in protein maturation and inflammation

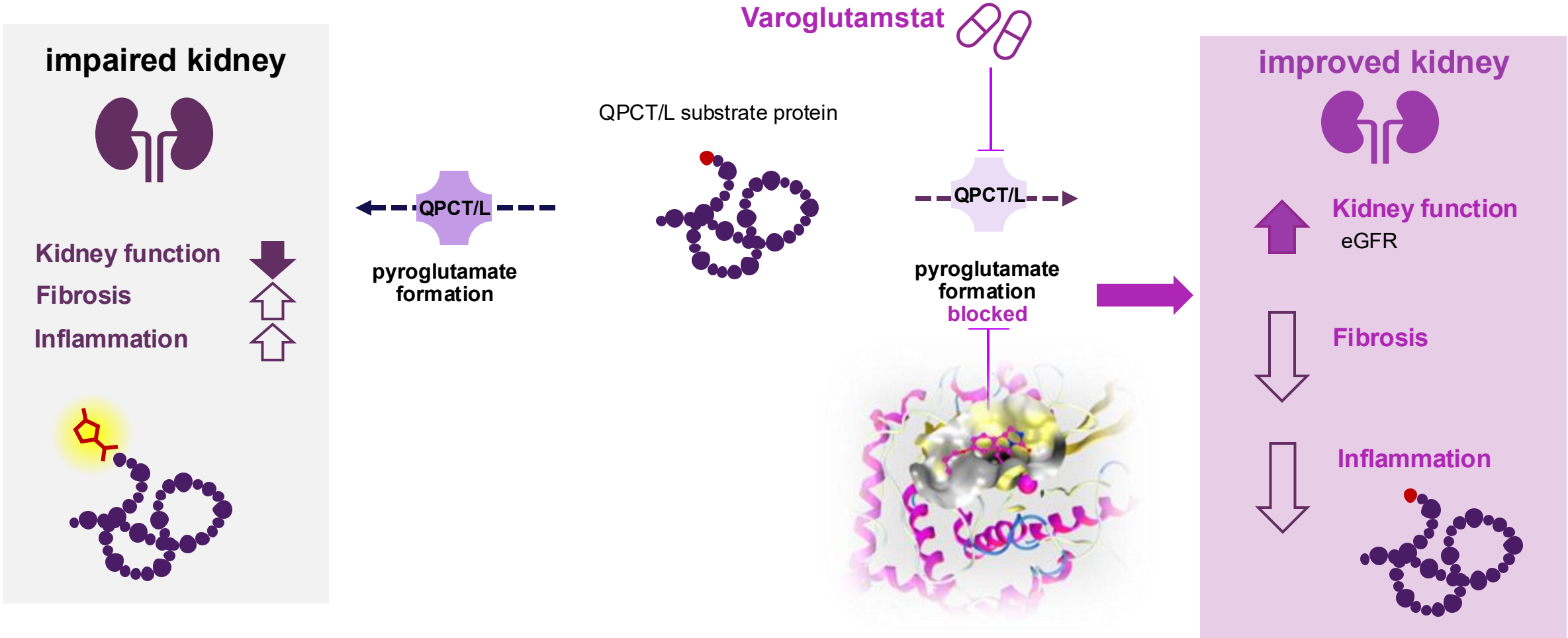


Chronic kidney tissue damage can lead to higher expression of QPCTL which can accelerate inflammation and organ fibrosis, organ dysfunction and finally kidney failure

1. Peptides including pro-inflammatory chemokines are synthesized on the endoplasmic reticulum (ER)
2. These are transported to the Golgi apparatus where they undergo further maturation / modification
3. The enzyme QPCTL resides in the Golgi and mediates pyroglutamylation, a crucial step in enhancing the potency and stability of certain proteins
4. Pyroglutamylation leads to excretion of more mature, potent and resilient proteins from the cell, including chemokines such as pE-CCL2 and pE-CCL13

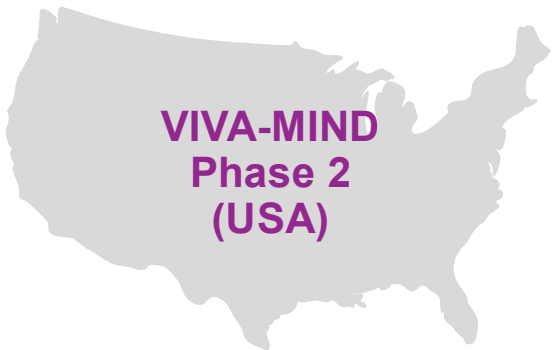
pE-CCLx – various pE chemokines; Notes: Graphic is for illustrative purposes only; not to scale.

Pyroglutamated peptides produced by QPCT/L are a central part of the pro-inflammatory/pro-fibrotic pathways in kidney disease



Cynis et.al. EMBO, 2011; Cynis et al, Int. J Exp Path, 2013, Kanemitsu et al., 2021; Vivoryon company data, 2024;
QPCT/L - Glutaminyl-peptide cyclotransferase / -like protein; Notes: Graphic is for illustrative purposes only.

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 & VIVA-MIND

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	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 wks (mean) / 96 wks (max.)	46 wks (mean) / 72 wks (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 IDNCT04498650; VIVA-MIND: ClinicalTrials.gov IDNCT03919162; BID: twice daily; eGFR: estimated glomerular filtration rate, based on serum 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%.

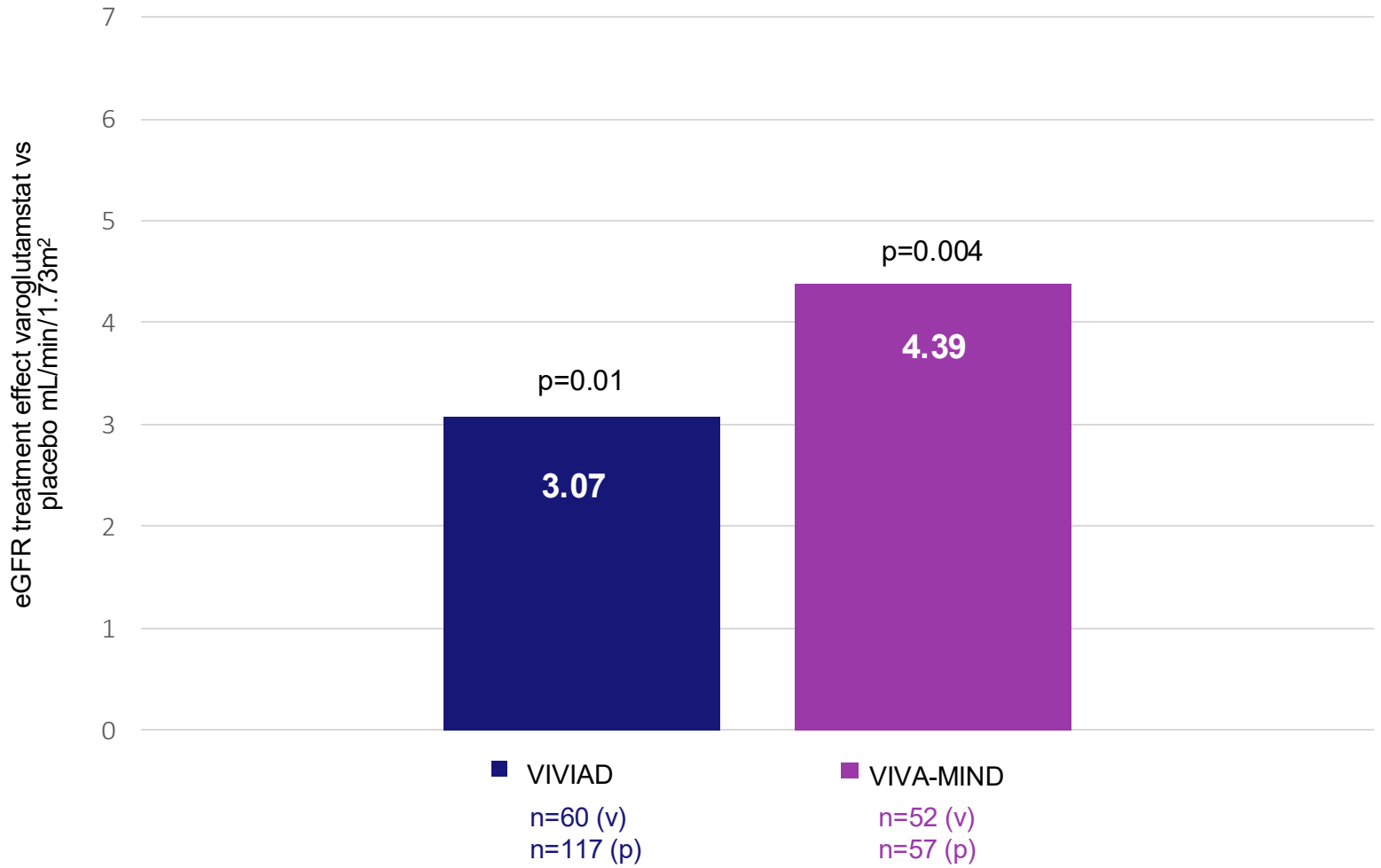
VIVIAD and VIVA-MIND both show a statistically significant and clinically meaningful improvement in eGFR over baseline

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

eGFR treatment effect:

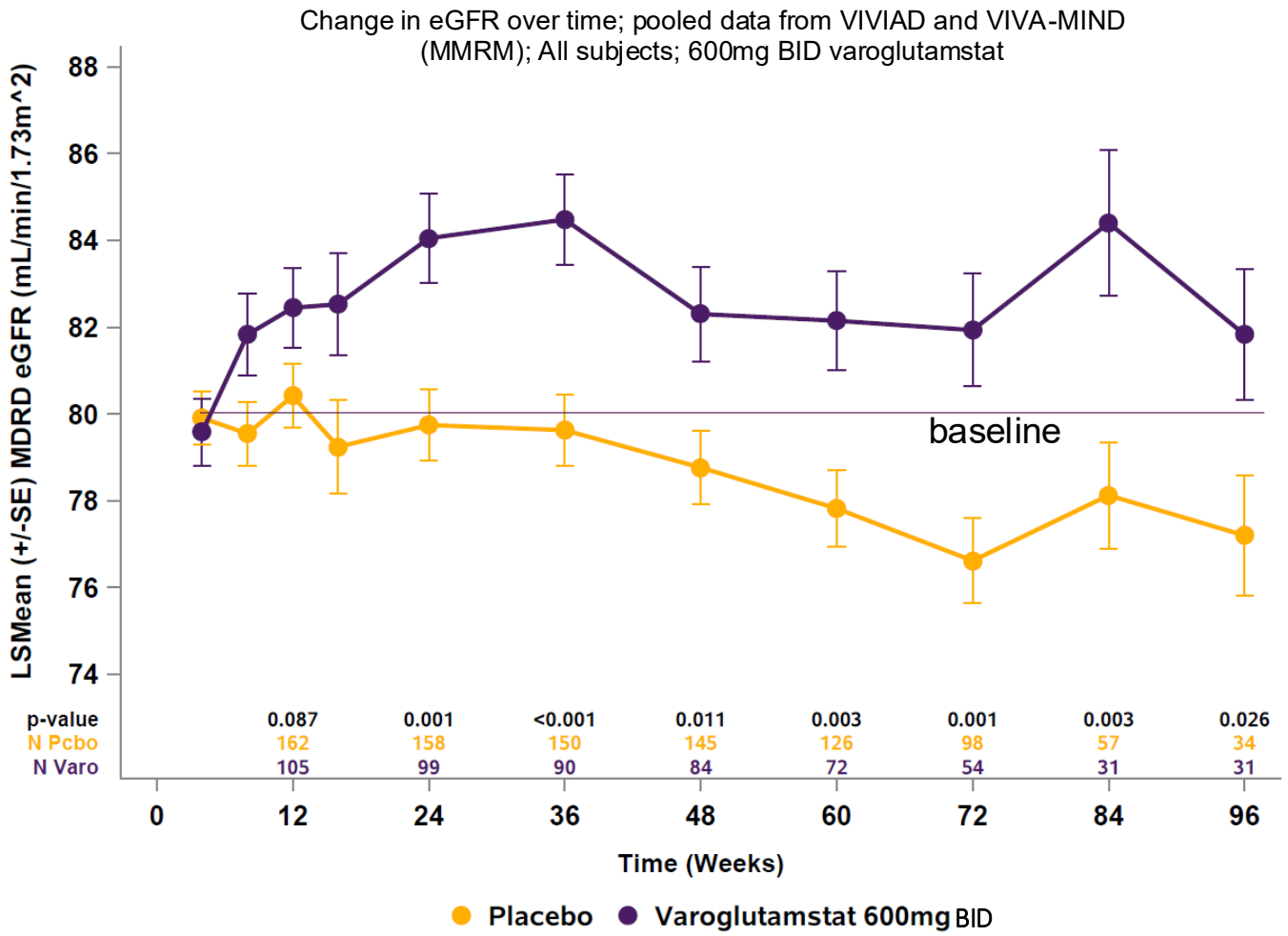
Difference between varoglutamstat and placebo (LSmean change from baseline)

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)

Compelling beneficial effect on kidney function in subjects treated with varoglutamstat compared to placebo



Varoglutamstat

- Clear and consistent separation of curves after 24 weeks
- Effect stable and maintained above baseline for 2 years
- Placebo patients decline mildly

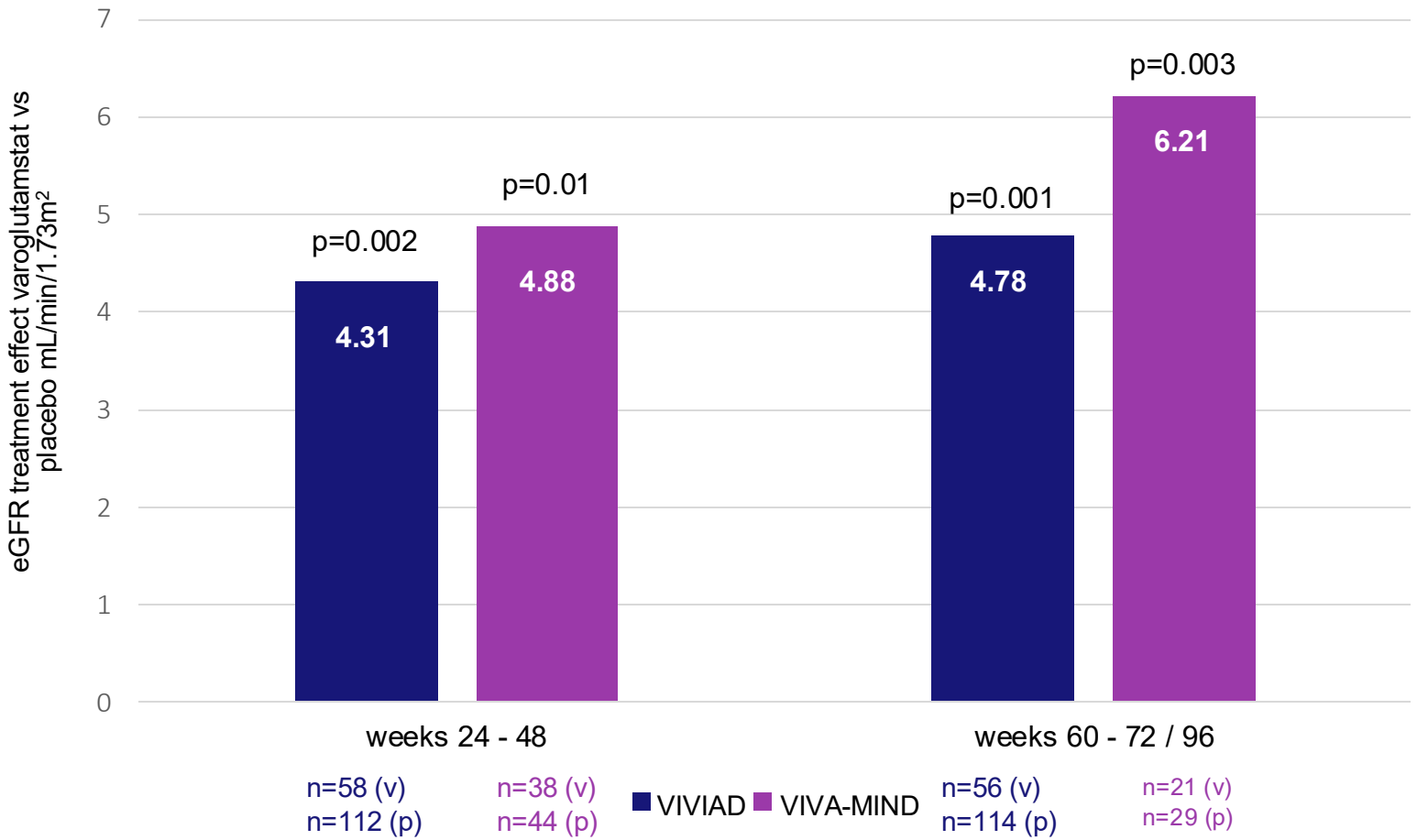
eGFR: estimated glomerular filtration rate, based on serum creatinine and calculated using the modification of diet in renal disease (MDRD) method. BID: twice daily. eGFR data were analyzed over time using MMRM (mixed model for repeated measures) modelling including fixed effect terms for study, treatment, visit window and treatment-by-visit interaction as well as baseline and study using data from patients randomized to 600mg BID and placebo of all visits on treatment, for VIVIAD (4–96 wks) and VIVA-MIND (4–72 wks). Within subject error modelled using an unstructured covariance matrix.

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)



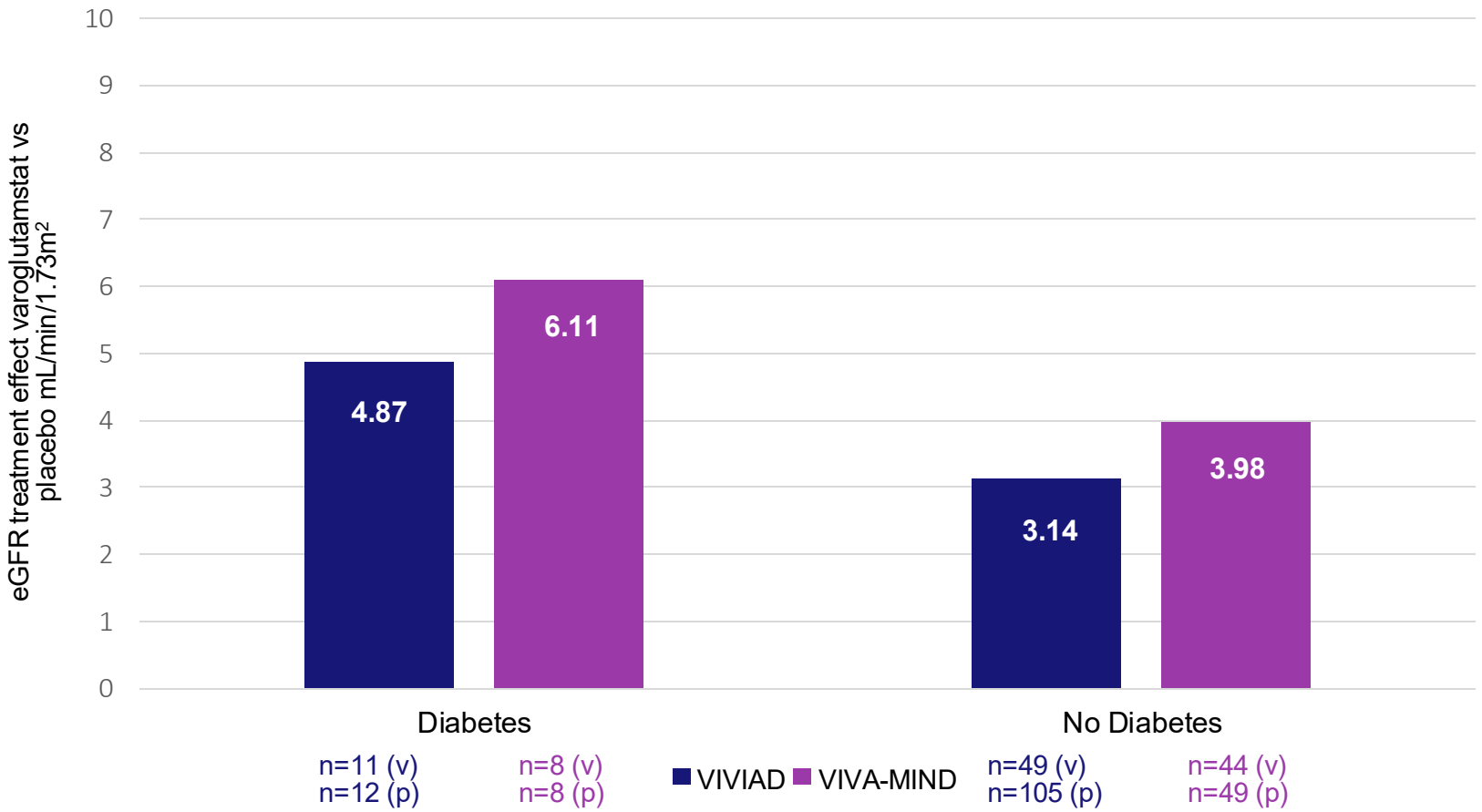
eGFR: estimated glomerular filtration rate, based on creatinine and calculated using the modification of diet in renal disease (MDRD) method. eGFR data were analysed over time using MMRM (mixed model for repeated measures) modelling including fixed effect terms for treatment, visit window and treatment-by-visit 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); LSmean: least squares mean

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)



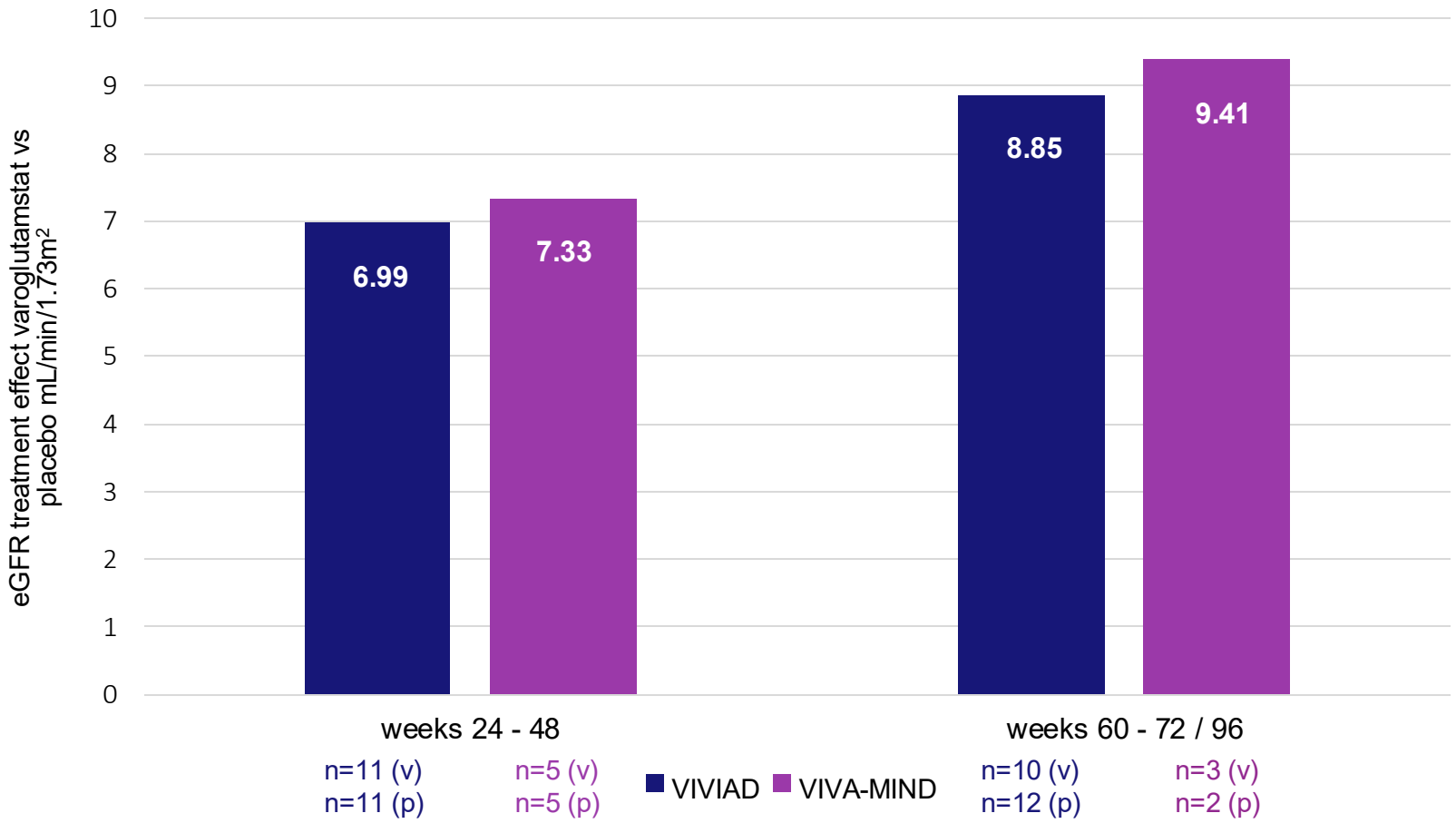
eGFR: estimated glomerular filtration rate, based on creatinine and calculated using the modification of diet in renal disease (MDRD) method. eGFR data were analysed over time using MMRM (mixed model for repeated measures) modelling including fixed effect terms for treatment, visit window and treatment-by-visit 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). Diabetes patients identified as defined on slide 9; LSmean: least squares mean

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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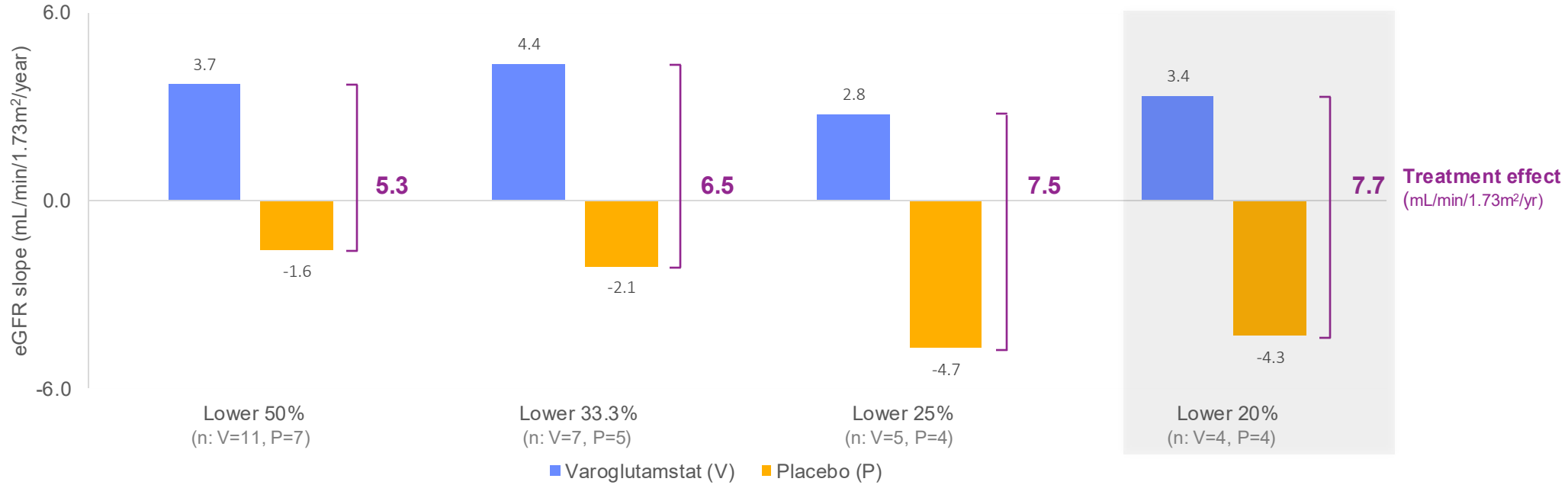
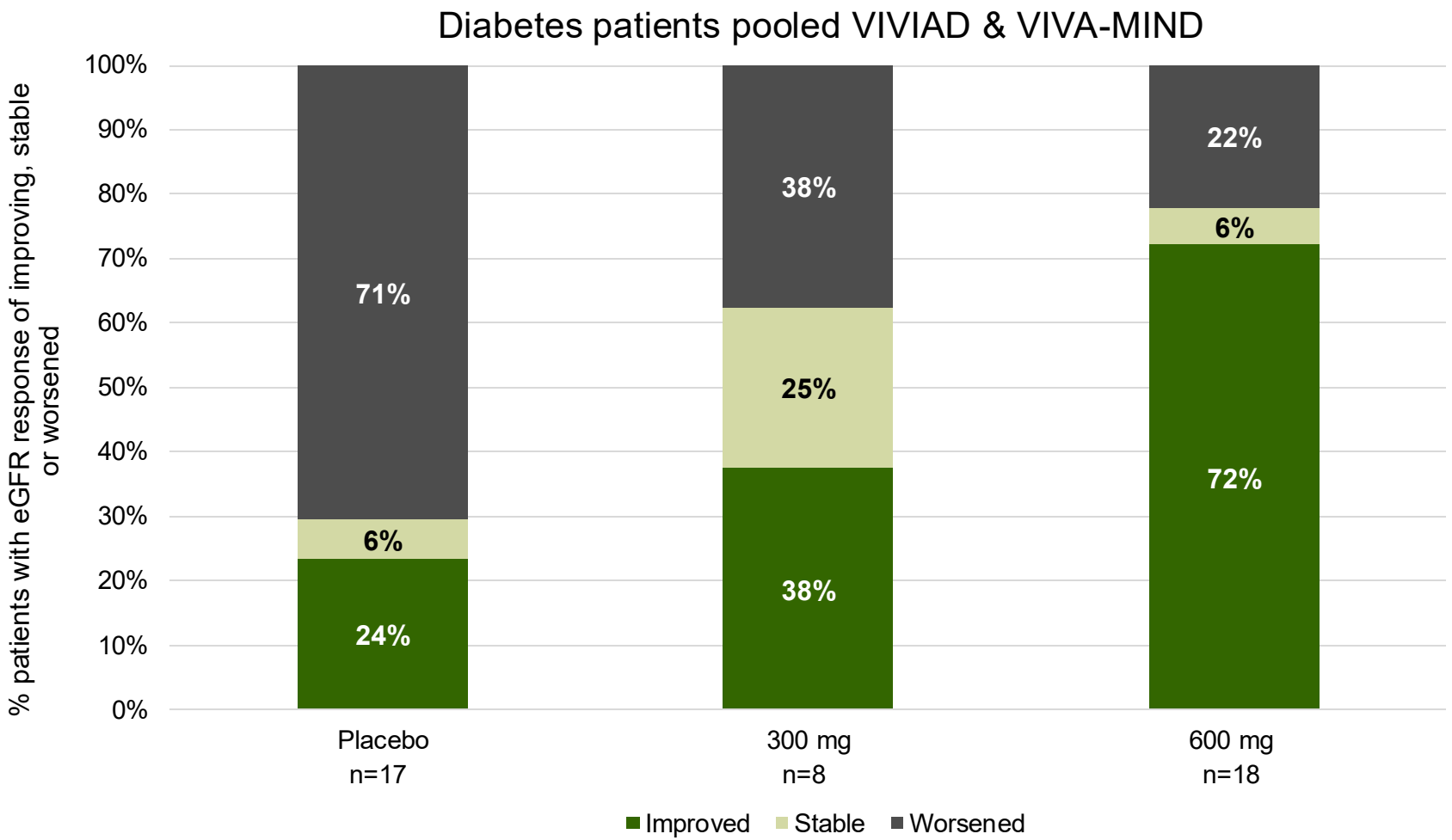
Varoglutamstat showed consistent and strong improvement of eGFR in patients with lower baseline eGFR values and diabetes

Participants with diabetes & reduced kidney function
(baseline eGFR levels in lower percentiles within the diabetes subgroup*;
Treatment effect (annualized rate of change in eGFR / eGFR slope) 600 mg BID varoglutamstat vs. placebo)



eGFR slope (mL/min/1.73m²/year): Random coefficients and MMRM calculations on pooled data from VIVIAD and VIVA-MIND in diabetic patients in the lower percentile.
**Diabetes subgroup defined as patients having at baseline either medical history of diabetes (type 1 or 2, and glucose tolerance impaired, hyperglycaemia) and/or comedication with drugs used in diabetes and/or untreated with a HbA1c > 6.5%; table data rounded to one decimal place; number of patients in the varoglutamstat treatment group (V) or the placebo group (P) at week 12*

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

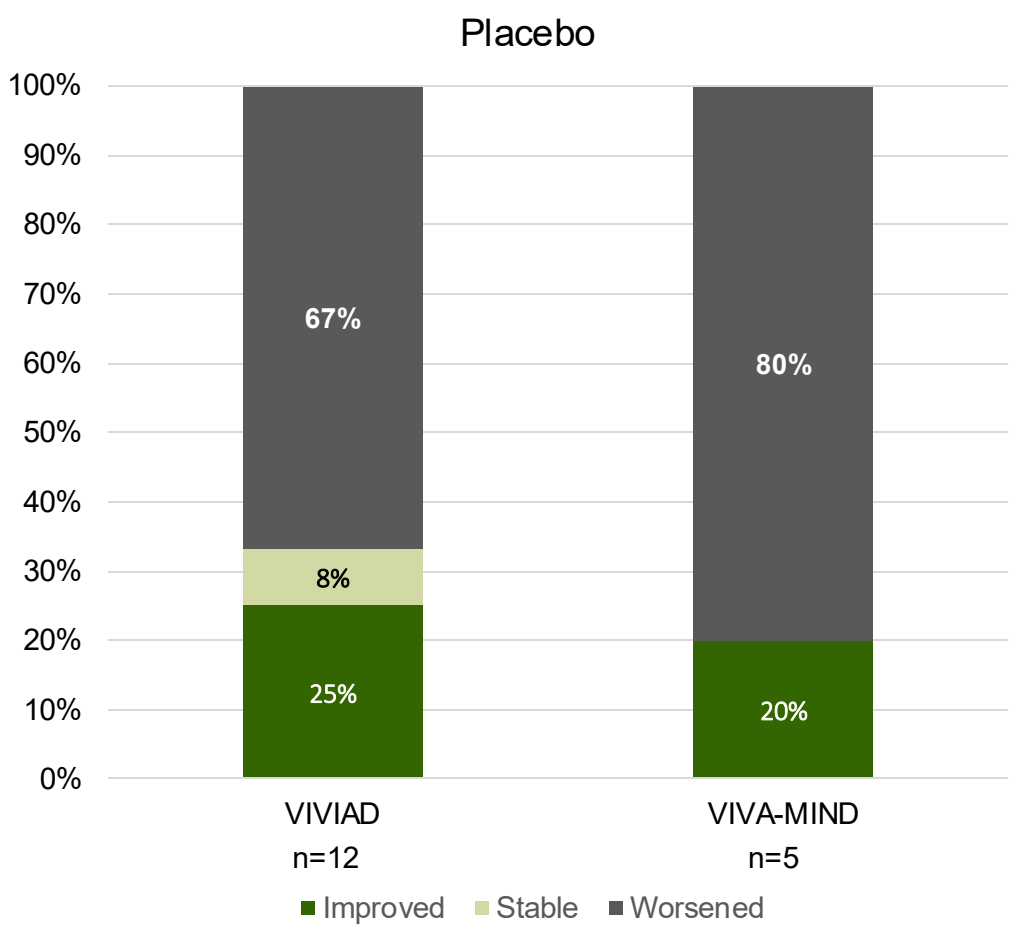
Response analysis (proportional odds)

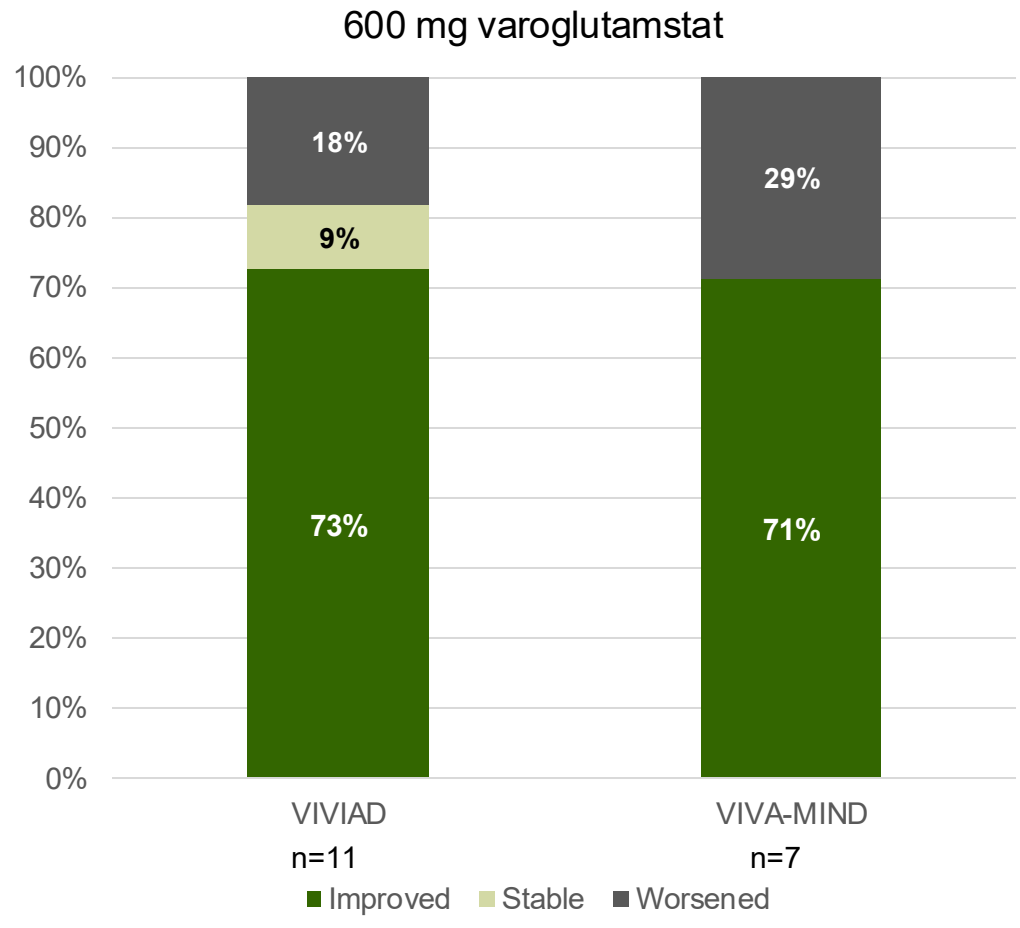
300 mg vs placebo
2.91, 95% CI (0.55, 15.53), p=0.2106

600 mg 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



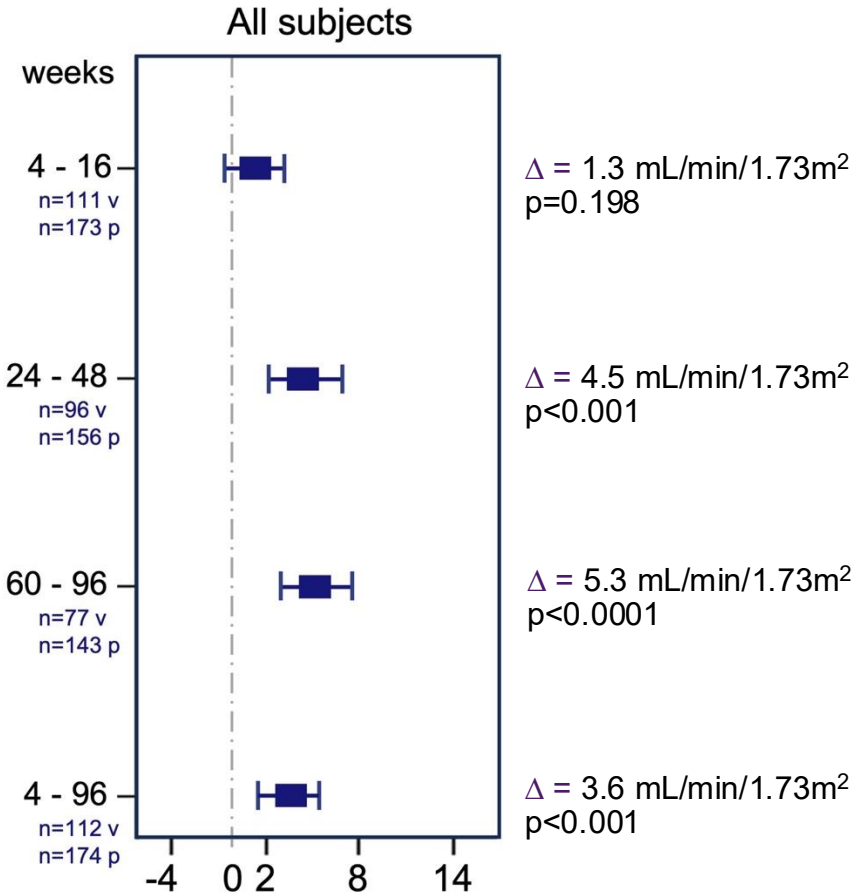


Classification of eGFR response (change mean eGFR (week 12-EOT) vs. baseline, mL/min/1.73m^2):
Improved: ≥ 2 mL above baseline, Stable: ≥ 0 - < 2 mL above baseline, Worsened: < 0 mL below baseline

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

VIVIAD and VIVA-MIND: Meta-analysis shows strong effect on eGFR

Difference of change from baseline between varoglutamstat (v) and placebo (p) of eGFR (MDRD)



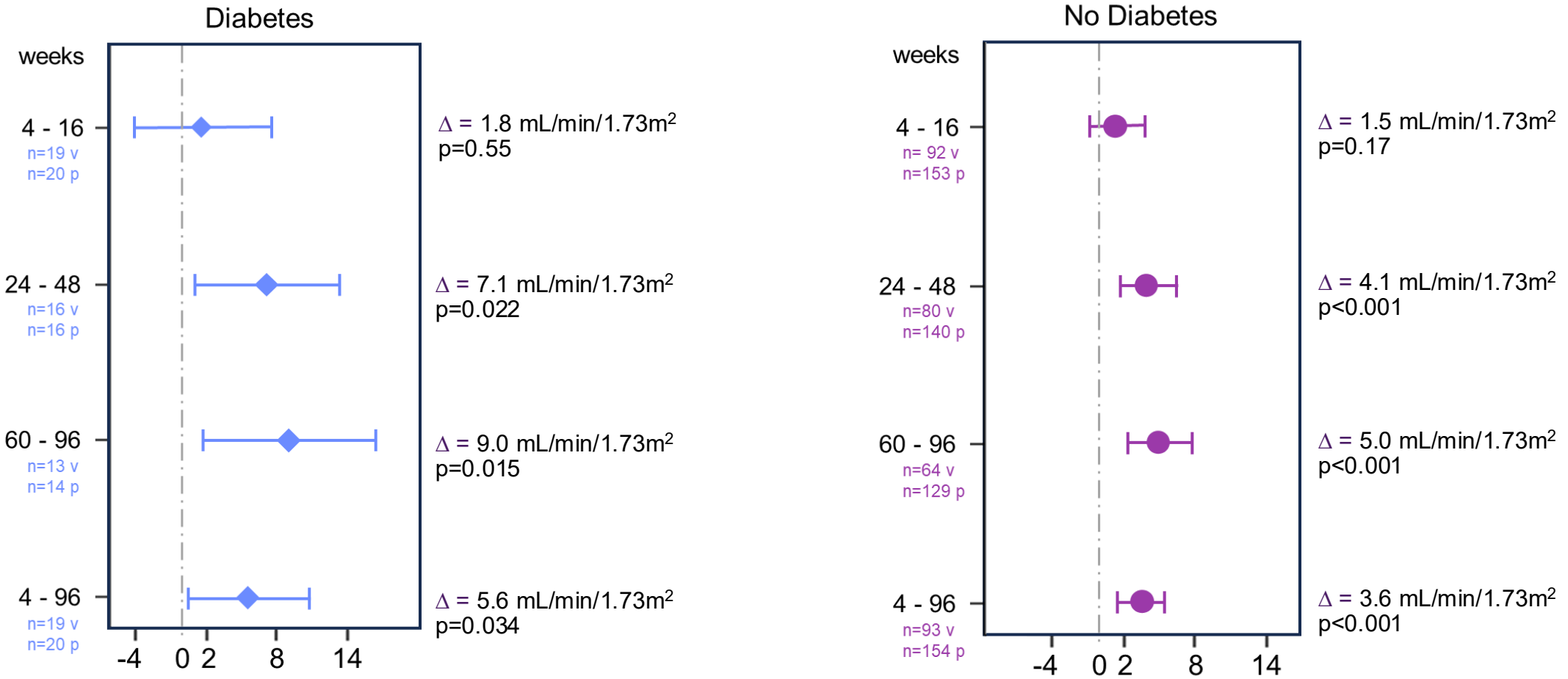
Treatment effect and 95% confidence intervals (mL/min/1.73m²)
0: No treatment effect; > 0: Improvement of eGFR (MDRD);
n: Number of patients in the varoglutamstat (v) and placebo (p) group

- Meta-analysis includes all patients on placebo and all patients randomized to 600 mg varoglutamstat BID of both studies (patients randomized to 300 mg BID in VIVIAD not included)
- Improvement of eGFR – kidney function is demonstrated in the total population
- Difference of change from baseline between varoglutamstat and placebo becomes significant at week 24
- Treatment effect is maintained for 2 years

VIVIAD Phase 2b in early AD included investigation of kidney function (eGFR) over up to 96 weeks; VIVA-MIND Phase 2 study in early AD included investigation of kidney function (eGFR) over up to 72 weeks; eGFR: estimated glomerular filtration rate based on creatinine, calculated using modification of diet in renal disease (MDRD) method; Meta analysis of change from baseline (MMRM) in varoglutamstat-treated patients vs. placebo

VIVIAD and VIVA-MIND: Meta-analysis shows a larger effect size in diabetes versus non-diabetes patients

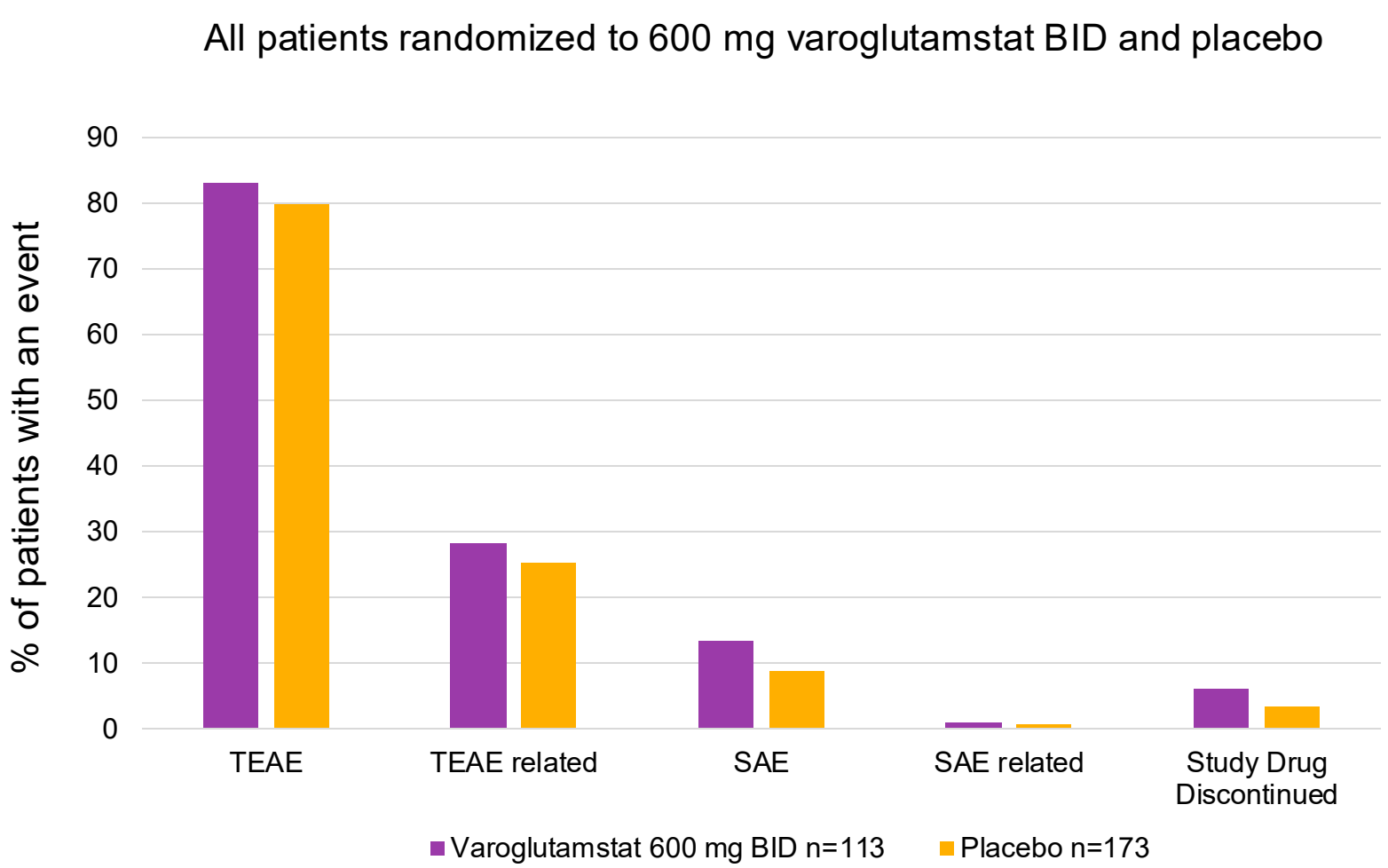
Difference of change from baseline between varoglutamstat (v) and placebo (p) of eGFR (MDRD)



Treatment effect and 95% confidence intervals (mL/min/1.73m^2)
0: No treatment effect; > 0: Improvement of eGFR (MDRD);
n: Number of patients in the varoglutamstat (v) and placebo (p) group

VIVIAD Phase 2b in early AD included investigation of kidney function (eGFR) over up to 96 weeks; VIVA-MIND Phase 2 study in early AD included investigation of kidney function (eGFR) over up to 72 weeks; eGFR: estimated glomerular filtration rate based on creatinine, calculated using modification of diet in renal disease (MDRD) method; Meta analysis of change from baseline (MMRM) in varoglutamstat-treated patients vs. placebo

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



Extensive safety package
(# / duration)

Pharmacology / Phase 1

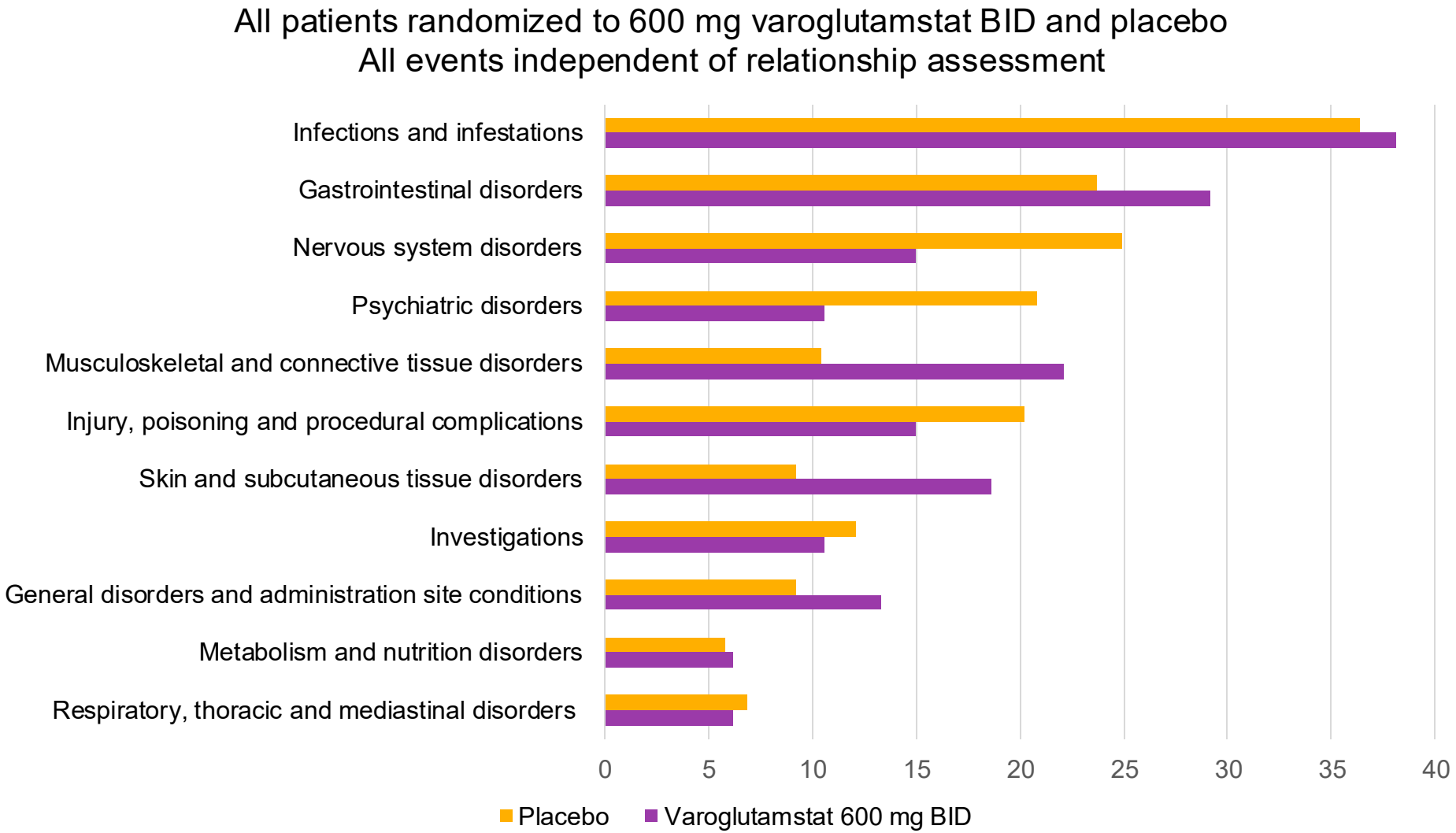
- Phase 1 study: large trial with 205 subjects
- Human ADME / mass balance study completed

Phase 2 double-blind, placebo-controlled

- Phase 2a study: 120 patients, 12 weeks
- VIVIAD Phase 2b study: 259 patients, avg. treatment duration ~80 weeks
- VIVA-MIND Phase 2 study: 109 patients treated, avg. treatment duration ~46 weeks

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

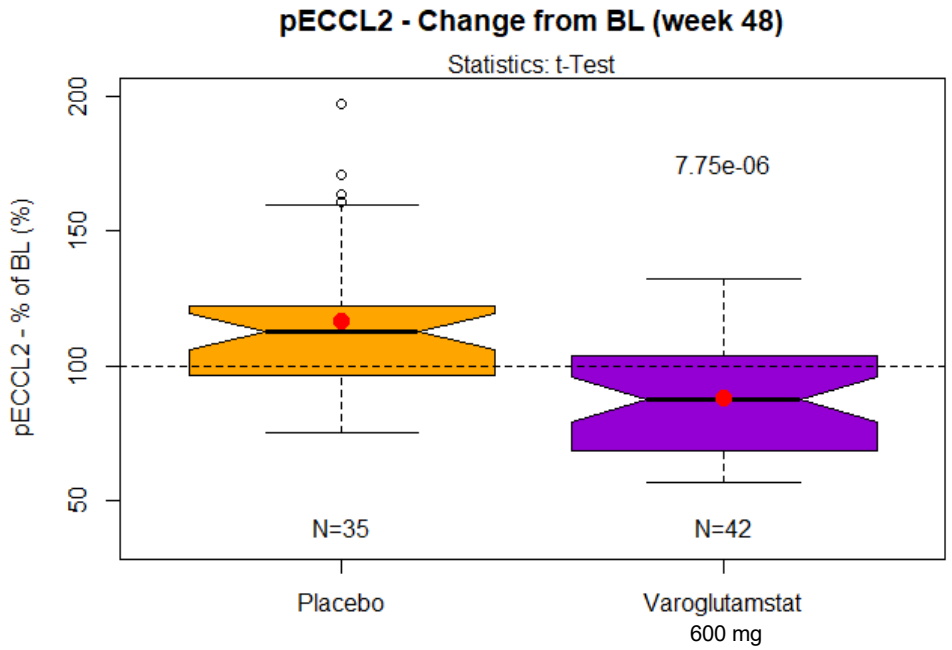
Pooled safety analysis VIVIAD and VIVA-MIND: TEAE by system organ class

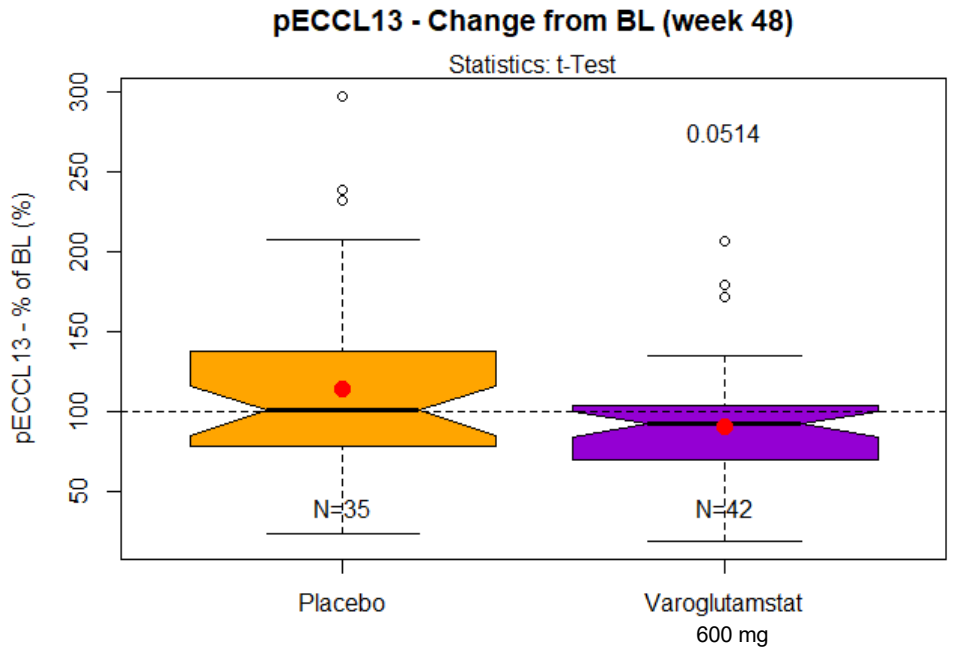


TEAE: Treatment Emergent Adverse Event; A 5% cut-off (aggregated) was applied.

Inhibition of QPCTL by varoglutamstat effectively reduces pro-inflammatory cytokines pE-CCL2 and pE-CCL13 in plasma

- Measurement of VIVIAD plasma samples[1] using new, highly sensitive, liquid chromatography-mass spectrometry (LC/MS)-based assay revealed a reduction in pE-CCL2 and pE-CCL13 levels
- Statistically significant, dose-dependent reduction of pE-CCL2, consistent with previous analyses

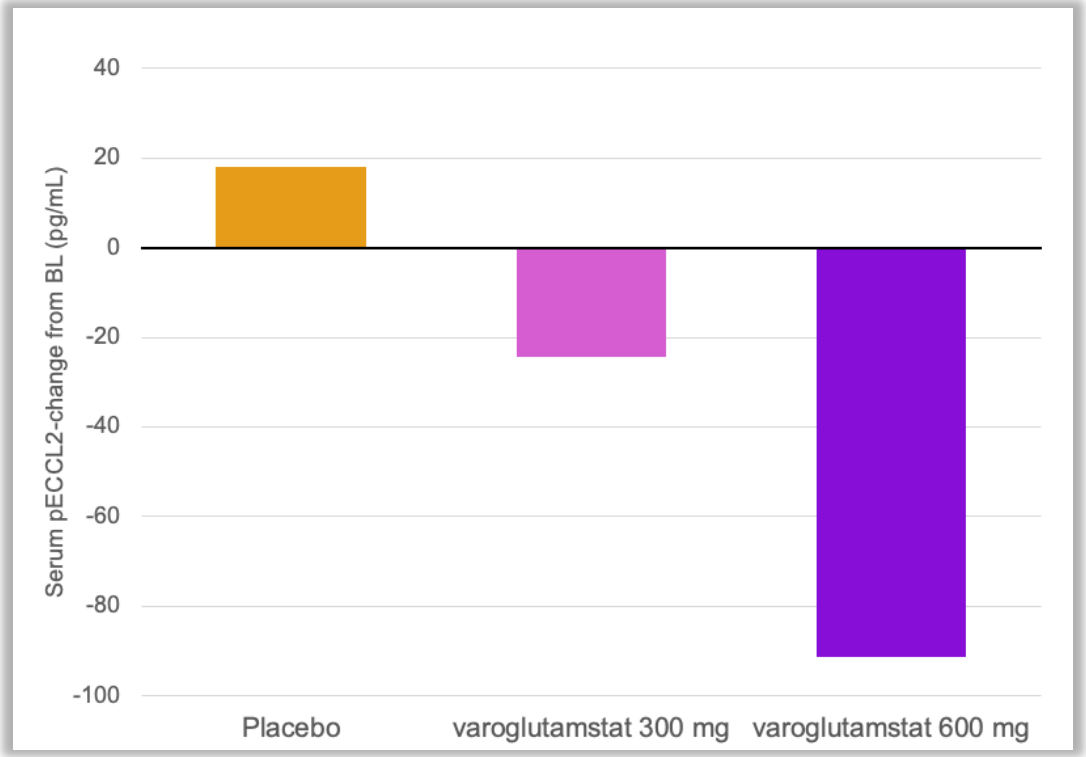




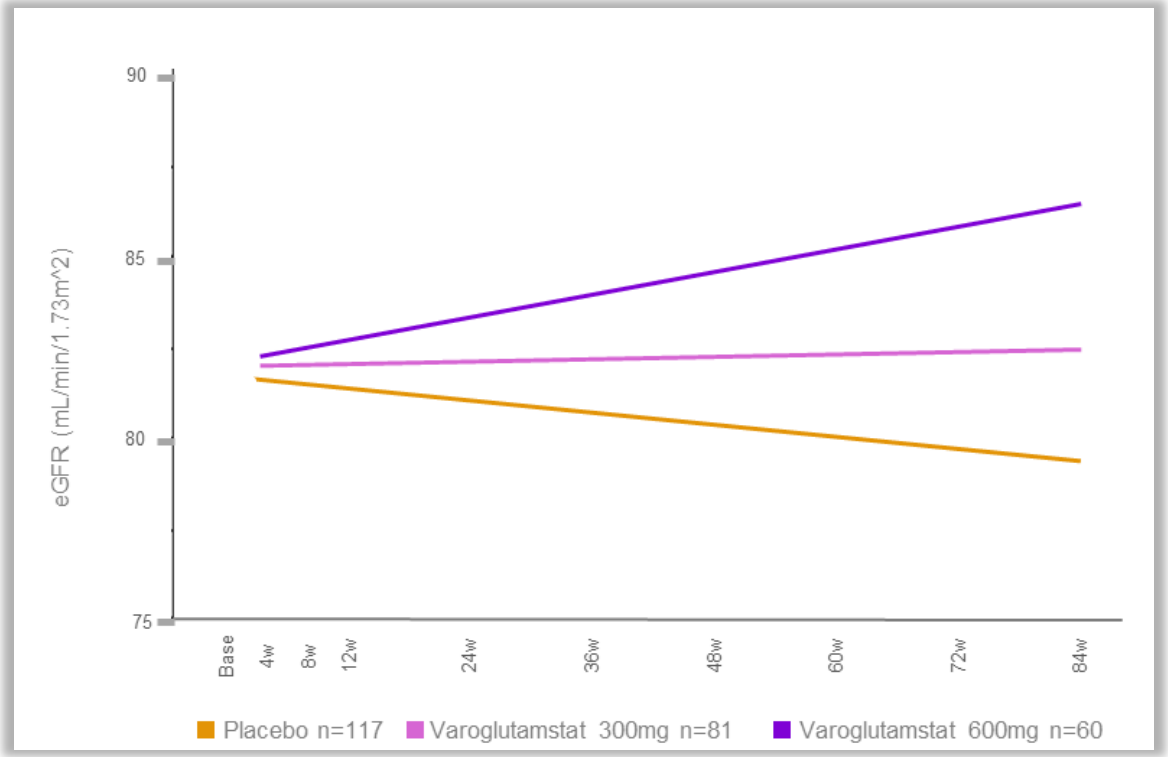
[1] *Data obtained from VIVIAD biomarker sample set with available screening at baseline and week 48 based on informed consent*

Dose-dependent reduction of pE-CCL2 correlates with improvement of eGFR

Median reduction in pE-CCL2 levels at week 48 compared to baseline with varoglutamstat
(total population)

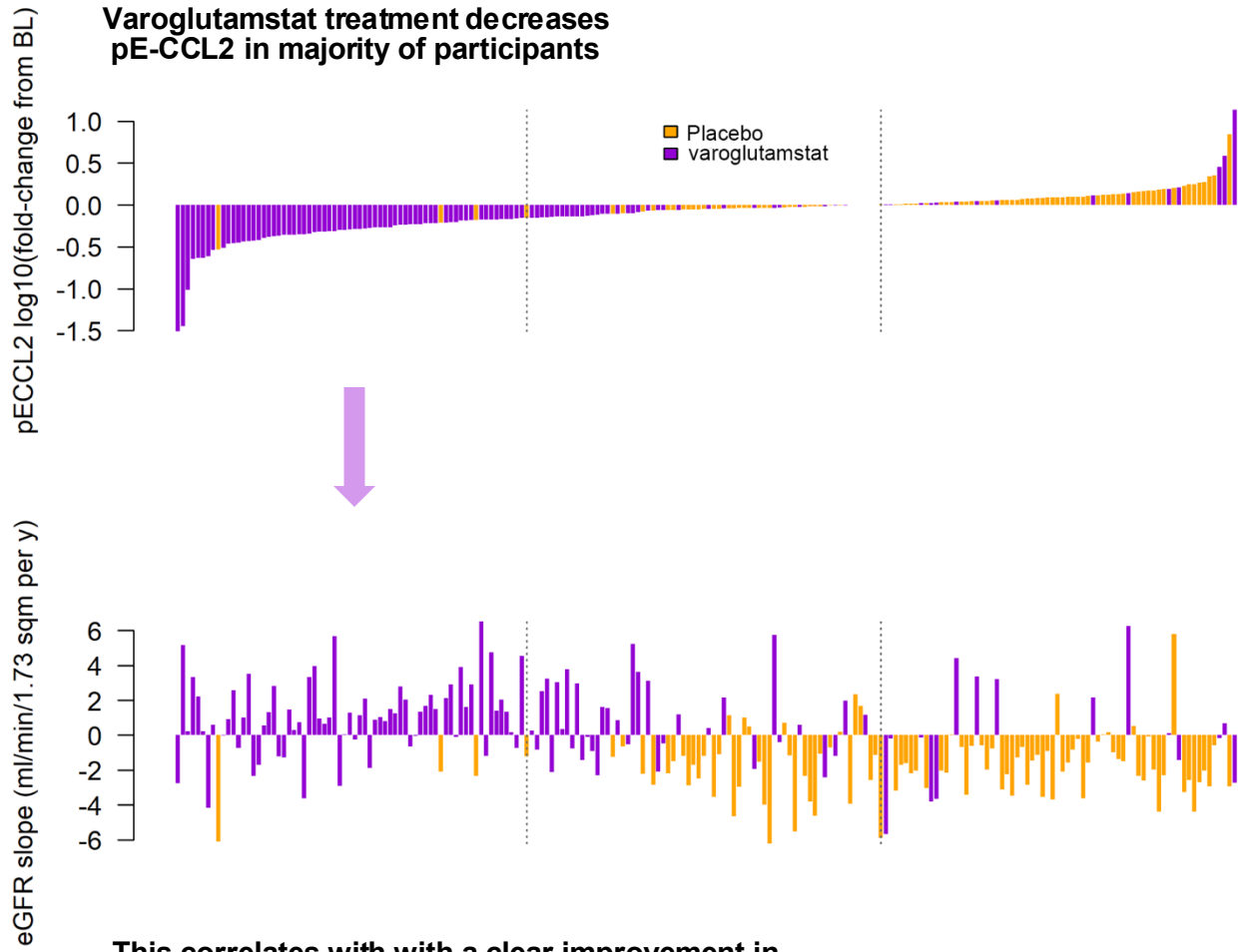


Varoglutamstat effect on kidney function outcomes
(total population; change in eGFR over time slope analysis (MDRD)[1])

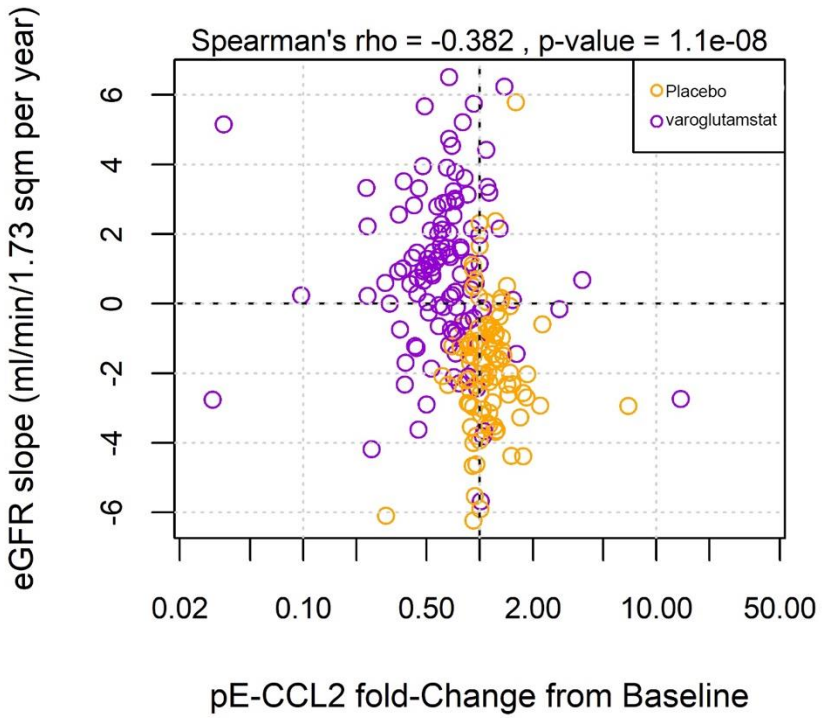


[1] *Chart shows change in eGFR (estimated glomerular filtration rate) over time based on creatinine and calculated using modification of diet in renal disease (MDRD) method; Data obtained from VIVIAD*

Improvement of eGFR correlates with reduction of pE-CCL2 on individual patient level, in line with total population data



Statistically significant correlation between change in pE-CCL2 serum levels at week 48 and the slope in eGFR over time



Data obtained from VIVIAD, all patients with available baseline and week 48 pE-CCL2 analysis (210 participants)

New diabetic kidney disease preclinical model results corroborate effects of varoglutamstat on inflammation, fibrosis and kidney function

- Evaluated varoglutamstat in an established preclinical model specific for DKD (reninAAV-accelerated DKD model in single kidney db/db mice)
- QPCT/L inhibition with varoglutamstat resulted in statistically significant reduction in inflammation (CD11c), fibrosis (glomerulosclerosis) and plasma creatinine, supporting an improvement in kidney function
- These data are consistent with prior data showing a similar effect of varoglutamstat on key kidney disease biomarkers in the ADI/CKD model

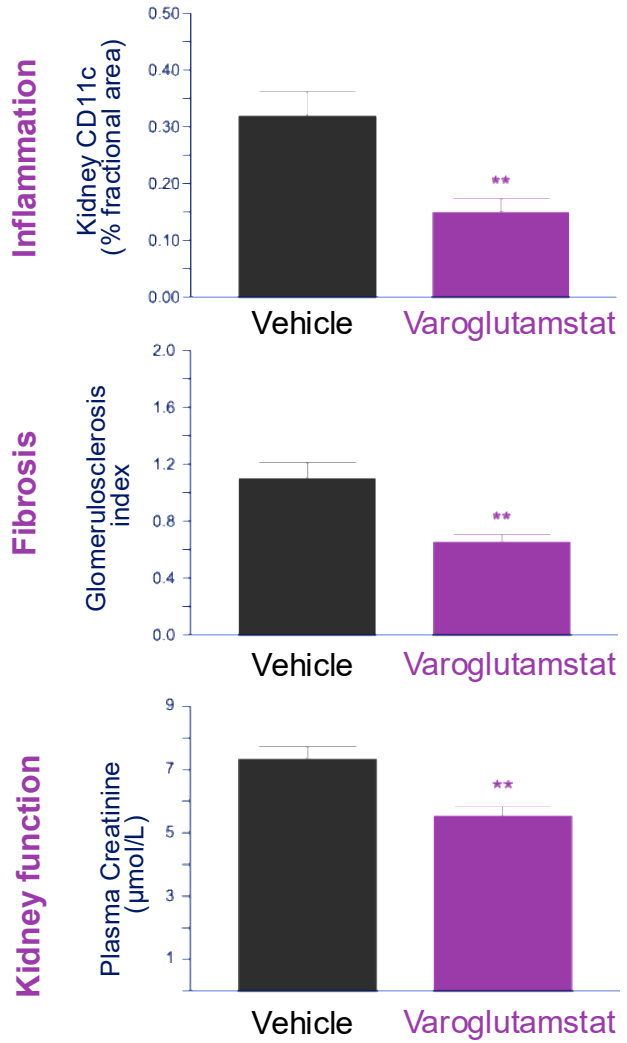


*Chart data: Values expressed as mean ± 95%CI, N = 13 to 17 per group, Dunnett's test one-factor linear model. **: P < 0.01 compared to vehicle*

Impressive synergistic effects of dapagliflozin plus varoglutamstat - pronounced modulation of inflammatory and fibrotic mechanisms in CKD mouse model

Results pave the development path of QPCT/L inhibitors in combination with SGLT-2 inhibitors

- Efficacy observed on top of SGLT-2 inhibitors derisk the DKD / CKD clinical development program substantially
- Magnitude of effect of QPCT/L inhibition together with SGLT-2 inhibition shows trend towards normalization of pathological findings across multiple outcome parameters
- Once daily similar efficacy vs. twice daily in pre-clin models supports investigation of once daily in clinical trial
- Ideal combination partner for patients treated with SGLT-2 inhibitors with strong synergistic effect observed
- Due to outstanding effect observed we have filed patents for combination of QPCT/L inhibitors with SGLT-2 inhibitors

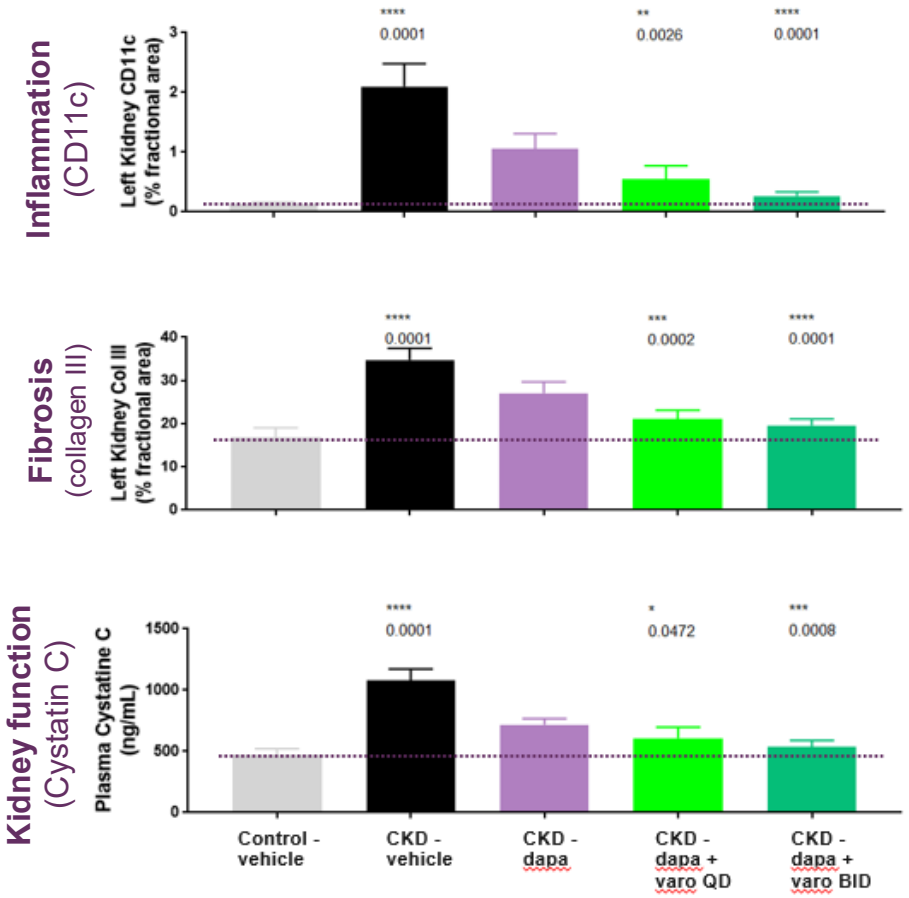


Chart data: Values expressed as mean ± 95%CI, N = 8 to 16 per group, Dunnett's Test, comparison to CKD – Dapagliflozin group; QD: once daily; BID: twice daily; varo: varoglutamstat; dapa: dapagliflozin

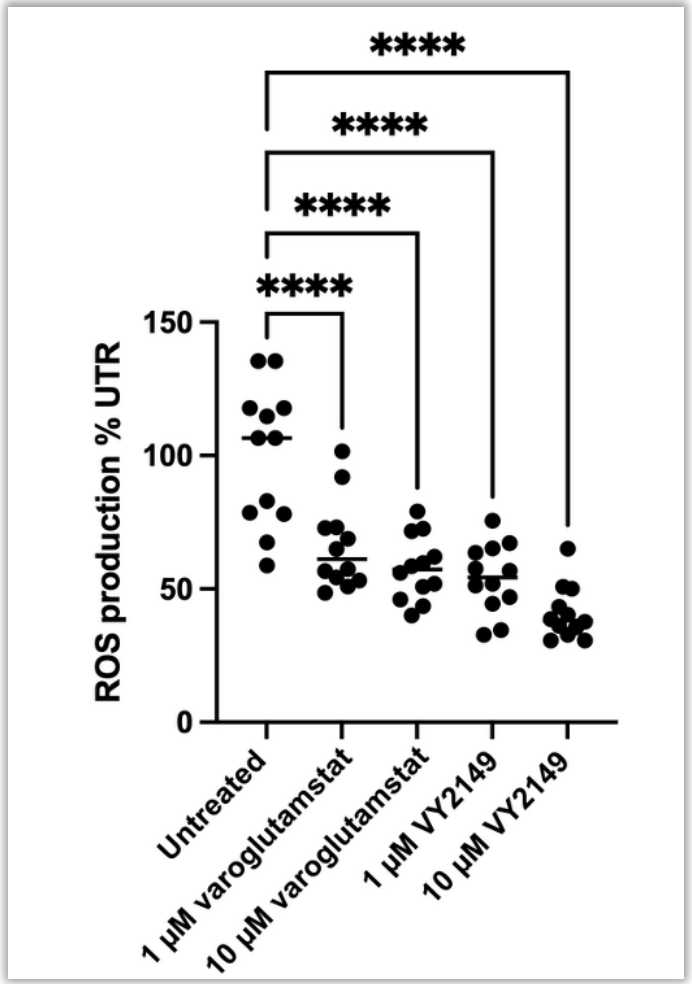
QPCTL inhibitors reduce oxidative stress in Fabry disease kidney cells

About Fabry disease

- Rare genetic disorder affecting more than 1/15,000 people[1], resulting from a deficiency of the enzyme alpha-galactosidase A
- Leads to accumulation of certain metabolic products inside cells of the kidney (podocytes), heart and other organs
- This triggers a cellular stress response including generation of reactive oxygen species (ROS) which are major drivers in Fabry nephropathy
- Existing therapies (e.g. ERT, oral chaperones) have limited efficacy especially in advanced disease leaving a significant need for therapies targeting underlying molecular mechanisms[2]

New pre-clinical data

- Vivoryon has investigated the effect of two QPCT/L inhibitors, varoglutamstat and VY2149 on Fabry podocytes
- Promising preliminary data show a significant dose-dependent reduction of ROS production, establishing a basis for further research in rare disease applications



ERT= Enzyme replacement therapy; ROS = Reactive oxygen species, % UTR = percentage of untreated control

[1] *https://www.orpha.net/en/disease/detail/324* ; [2] *Thompson et al., 2025, Biomedicines*

Varoglutamstat: Potential to become a convenient new oral therapy to transform the treatment of kidney disease

Medical Need

Therapies that can stabilize or improve kidney function for majority of patients

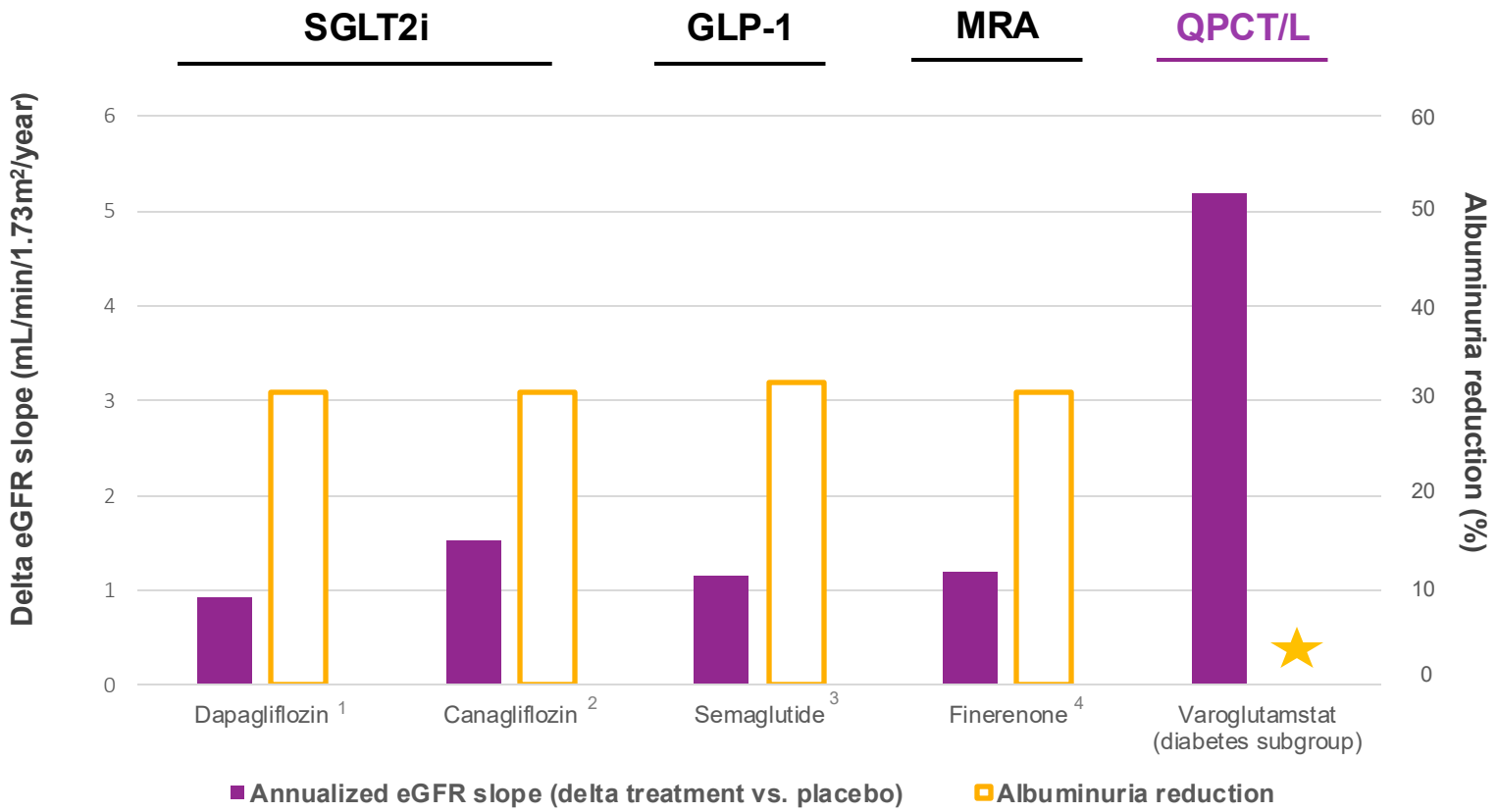
Opportunity

Varoglutamstat is a first-in-class **single agent** oral compound that has been shown to **stabilize and partially recover** kidney function

- Clear development path to market
- Future program based on robust available data
  - ✓ Statistically significant and clinically meaningful improvement in eGFR
  - ✓ Effects observed in two independent Phase 2 studies
  - ✓ Most promising subgroup identified: Substantially larger effect size in participants with diabetes
  - ✓ Excellent safety profile consistent across two years of study duration
  - ✓ Highly synergistic effect on top of current DKD SoC
  - ✓ Planned Phase 2b in DKD stage 3b/4 to evaluate effect in target population[1]
  - ✓ Further potential in certain rare diseases that impact kidney function, e.g. Fabry disease and Alport Syndrome

[1] *Subject to funding / partnership; DKD: Diabetic kidney disease; SoC: standard of care*

Effect size substantially higher than observed with current standard of care (SGLT2i / GLP-1)

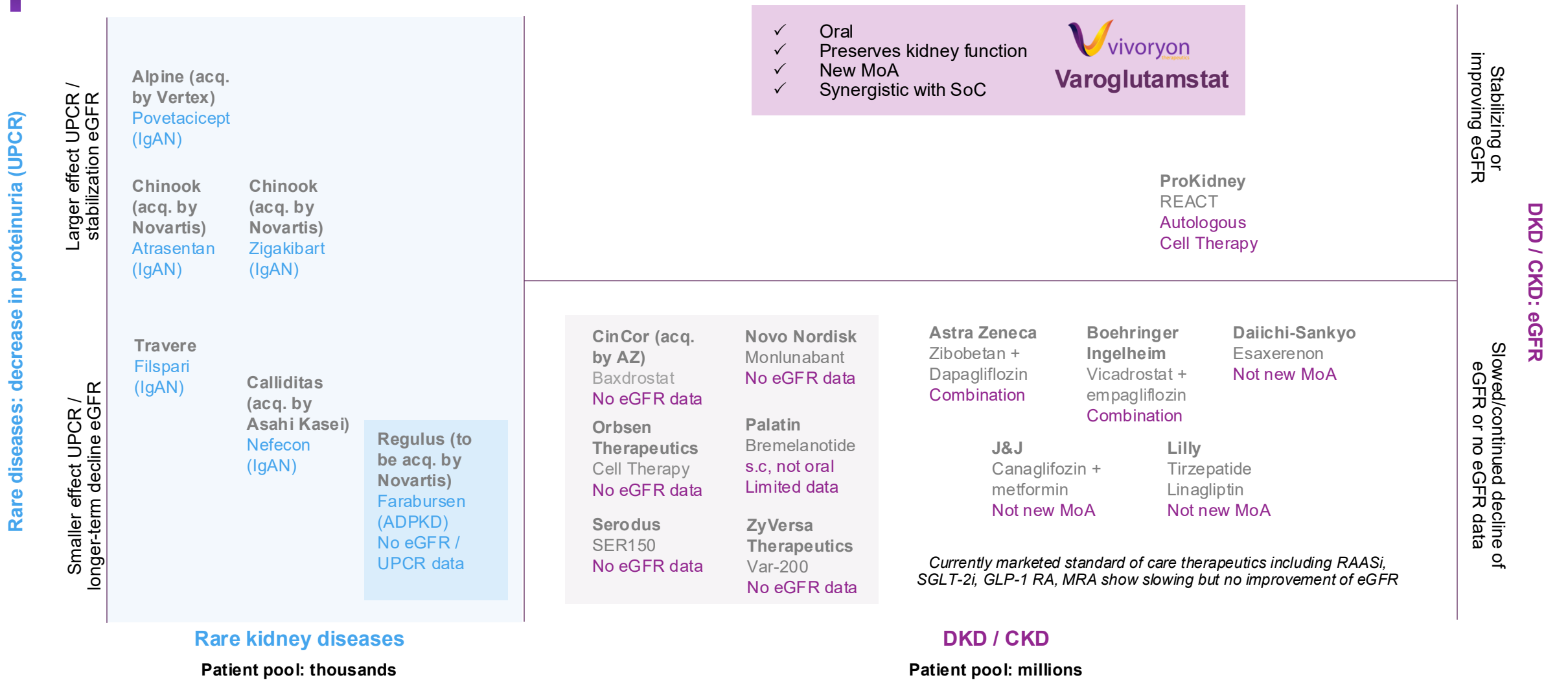


★ Conducted qualitative assessment, no increase in albuminuria observed; analysis of albuminuria planned for next Phase 2b study

Note: data comparisons are for illustrative purposes and not from head-head-studies or comparable patient populations, timelines or methods; Data for varoglutamstat is from pooled slope analysis for VIVIAD / VIVA-MIND Phase 2 study, diabetes subgroup; SGLT2 – sodium glucose cotransporter-2 inhibitor class; GLP-1 Glucagon-like peptide class; MRA: mineralocorticoid receptor antagonist; QPCT/L – varoglutamstat inhibits the glutaminyl cyclases QPCT and QPCTL; eGFR: estimated glomerular filtration rate;

[1] Heerspink et al. N Engl J Med, 2020; [2] Perkovic et al., N Engl J Med, 2019;[3] Perkovic et al., N Engl J Med, 2024; [4] Bakris et al., N Engl J Med, 2020

Varoglutamstat's ability to stabilize and partially recover kidney function sets it apart in the kidney space and supports its potential to transform disease outcomes

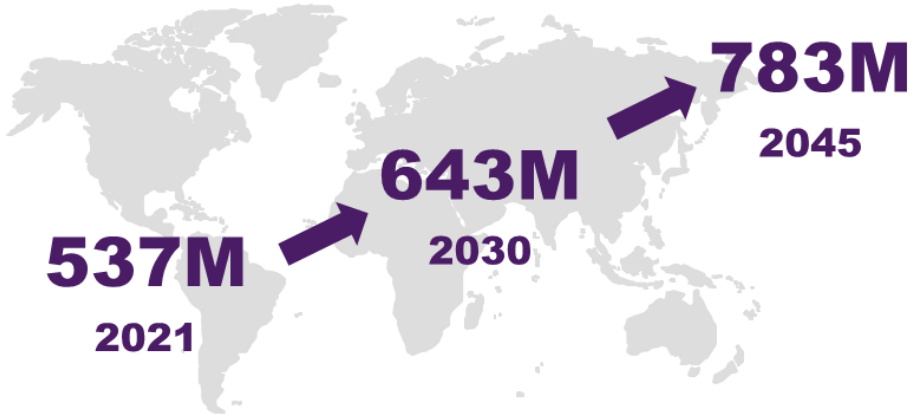


eGFR: estimated glomerular filtration rate; The above graphic includes select development / marketed drug candidates in the rare and CKD/DKD space and is for representation purposes only; data comparisons are for illustrative purposes and not from head-head-studies or comparable patient populations, timelines or methods; IgAN: IgA Nephropathy; ADPKD: autosomal dominant polycystic kidney disease; acq: acquisition / to be acquired by.

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]

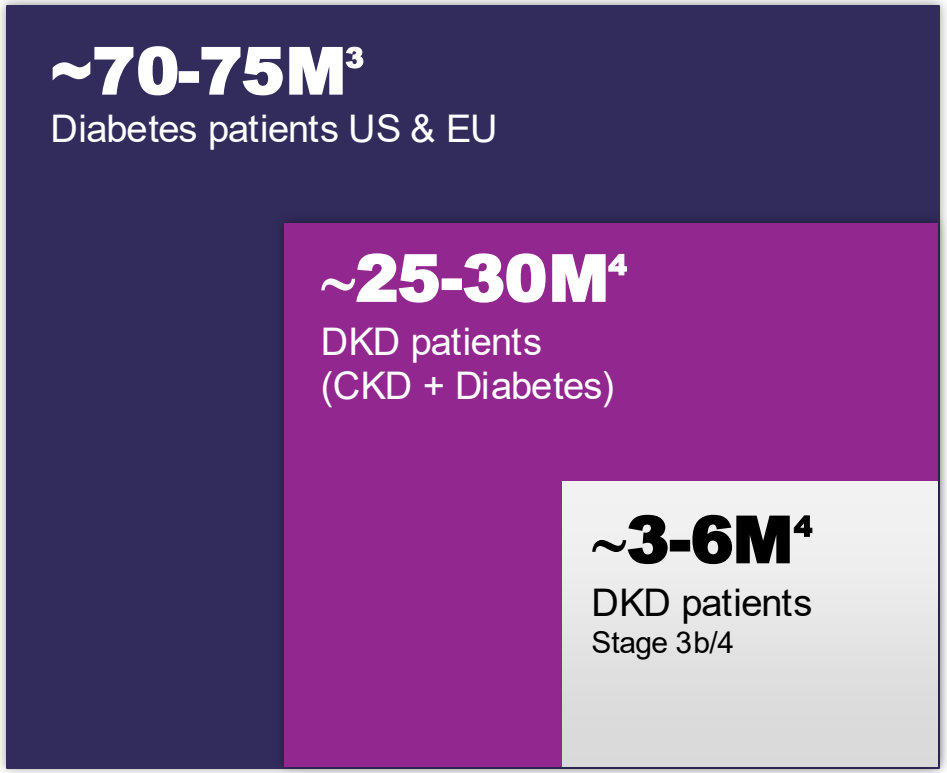


~40%

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 Study: Efficient study design to confirm the treatment effect in patients with advanced DKD[1]

Primary Goal

- Aiming to confirm the efficacy of varoglutamstat 600mg BID on eGFR in people with advanced diabetic kidney disease in an efficient and timely manner

Key Metrics and Considerations

- Double-blind randomized placebo-controlled multi-center study
- Patients with T2DM with stage 3b/4 CKD on top of SoC incl. SGLT2-i
- Adequately powered for meaningful data readout
- No. of patients: ~100 – 150
- Topline data ~24 months; design could include interim analysis at ~15 months to give earlier proof-of-concept[2]
- Typical trial cost approx. €12 -18m - dependent on patient number

[1] Draft trial considerations as part of scenario planning; study start and final trial design subject to additional financing / partnership; [2] timelines refer to from study start.
BID: twice daily; T2DM: type 2 diabetes mellitus; CKD: Chronic Kidney Disease; SoC: standard of care; SGLT2-i: sodium-glucose cotransporter-2 inhibitor

Summary: Varoglutamstat in DKD/CKD - building a robust body of evidence and advancing kidney disease program

2023

Laying the groundwork

- Decision to explore varoglutamstat in kidney disease
- VIVIAD study protocol amended to investigate effect of varoglutamstat on kidney function biomarkers

2024

Data-driven shift to DKD

- Exciting kidney function data observed in Ph 2b study (VIVIAD)[1]
- Substantially larger treatment effect in diabetes subgroup
- Benefit on kidney function confirmed in second Ph 2 study (VIVA-MIND)[1]
- Data presented at ASN Kidney Week and favorably received by experts

2025

Building evidence & Phase 2b DKD preparations

- Compelling kidney function data & meta-analysis from two Ph 2 studies - oral presentation at ERA 2025
- Novel U.S. composition of matter patent granted for varoglutamstat, expected exclusivity through 2044+[2]
- Preclinical synergistic effect for combination with an SGLT-2 inhibitor; new data supporting MoA in DKD model
- Preparations ongoing for Ph 2b of varoglutamstat in DKD
- Novel QPCT/L inhibitor VY2149 with improved profile nominated for development in DKD/CKD and rare diseases

[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] Potential for Hatch-Waxman extension of up to 5 years; SGLT-2: sodium glucose cotransporter-2; CKD/DKD: Chronic/diabetic kidney disease

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

Development opportunities for QPCT/L inhibitors range across many immune-mediated diseases characterized by inflammation and fibrosis

Broad potential for QPCT/L inhibitors: Multiple avenues to value generation

Leveraging proprietary QPCT/L platform and core expertise
to generate additional development and partnership opportunities

Primary focus

Diabetic kidney disease

Varoglutamstat shown to effectively reduce inflammation and fibrosis and to stabilize/ partially recover kidney function:

- **Diabetic kidney disease** (DKD, Phase 2b planned)[1]

Near-term opportunities

Rare kidney diseases

MoA and preclinical evidence provide strong rationale for exploring QPCT/L inhibitors beyond DKD, e.g.:

- **Fabry disease** (initial podocyte data)
- **Alport disease**
- **FSGS**
- **Cystic kidney diseases** (e.g. ADPKD)

Mid-term opportunities

Other immune-mediated diseases

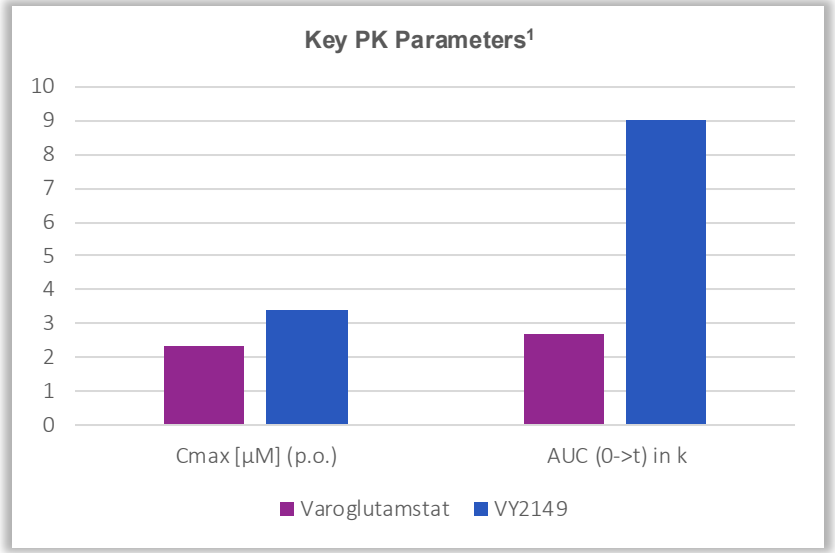
Strong evidence for benefit of QPCT/L inhibitors on inflammation and fibrosis supports expansion into other organ systems, e.g.:

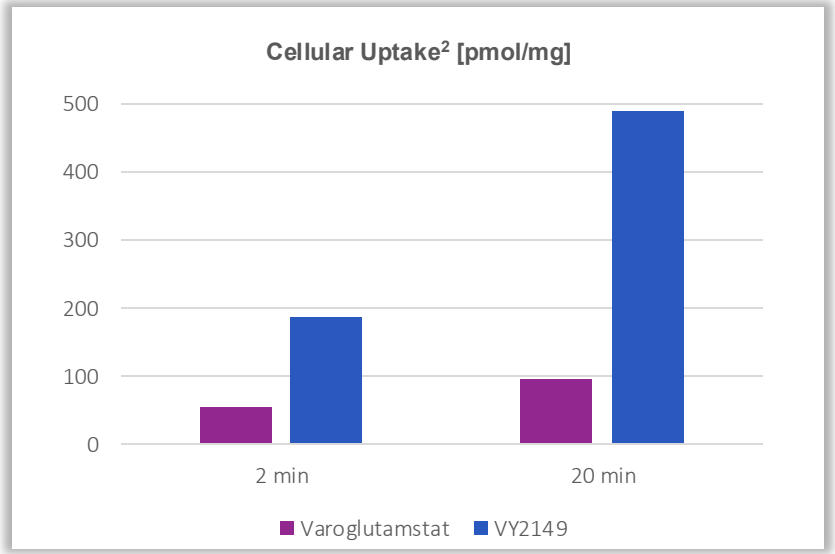
- **MASH / MASLD** (STAM mouse model: anti-inflammatory and anti-fibrotic effects)[2]
- **Cardiovascular diseases** (cuff-induced accelerated atherosclerosis in ApoE3*Leiden mice: reduction of cholesterol, pE-CCL2, infiltrated monocytes and neointima formation)[3]
- **IBD** (mouse model: ameliorated DSS-induced colitis symptoms)[4]
- **Septic arthritis** (Staph. aureus-induced mouse model: alleviated development and progression of experimental septic arthritis)[5]
- **MS** (MOG-induced EAE mouse model: protective effect against the development of EAE)[4]

[1] Subject to funding / partnership; [2] Cynis et al. 2013; [3] Cynis et al., 2011; [4] unpublished data; [5] Hellvard et al., 2013;
FSGS: Focal segmental glomerulosclerosis; MASH: Metabolic dysfunction-associated steatohepatitis;
MASLD: Metabolic dysfunction-associated steatotic liver disease; IBD: inflammatory bowel disease; MS: multiple sclerosis

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





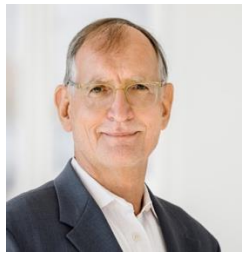
[1] *Single low dose (10mg/kg); p.o.= oral; PK = pharmacokinetics); AUC = Area under the curve; Cmax = peak concentration*

[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.*

A trusted company: Senior management team with a strong track record

Management

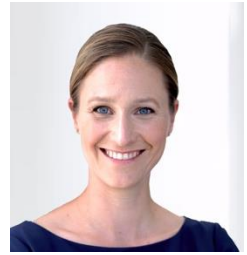
Frank Weber, MD
Chief Executive Officer



Marcus Irsfeld
Chief Financial Officer

Michael Schaeffer, PhD
Chief Business Officer

Julia Neugebauer, PhD
Chief Operating Officer



Non-executive Directors

Erich Platzer, MD, PhD
Chairman of the Board

Charlotte Lohmann

Claudia Riedl, PhD
Chair Audit Committee

Samir Shah, MD

Decades of collective experience in biopharma industry, e.g.:

First approved drug in pulmonary fibrosis

Successful development of biomarker driven oncology & diabetes programs

M&A and business development **expertise** from transactions with large biopharma

Know-how in life science research & development, biophysical and structure-based drug discovery

Strong financial, capital markets and legal **experience**

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

Addressing unmet needs in areas of high commercial potential

Mission is to improve **kidney health** and ultimately reduce rate of transplant / dialysis in **DKD/CKD/other** potential indications

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 trial 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+ with additional potential for Hatch-Waxman extension of up to 5 years

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