

Maze Therapeutics

Harnessing the power of human genetics to transform the lives of patients

November 2026

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the “safe harbor” provisions of the Private Securities Litigation Reform Act of 1995. These forward-looking statements reflect the current beliefs and expectations of management. All statements other than statements of historical fact are statements that could be deemed forward-looking statements, including, without limitation, statements concerning the company’s future plans and prospects, any expectations regarding the safety, tolerability, efficacy or mechanism of action of MZE829, MZE782 and other candidates under development, the ability of MZE829 to treat APOL1-mediated Kidney Disease (AMKD) or other indications, the ability of MZE782 to treat Chronic Kidney Disease (CKD), Phenylketonuria (PKU) or other indications, the market size of AMKD, CKD and PKU, the company’s competitive positioning, the planned design and timing of the company’s clinical trials, data results and further development of MZE829, MZE782 and other therapeutic candidates, potential pipeline expansion, the potential benefits of our out-licensed candidates, including potential milestone payments, the company’s expected cash runway, and the sufficiency of the company’s cash and cash equivalents to fund its operating expenses and capital expenditure requirements. In addition, when or if used in this presentation, the words “may,” “could,” “should,” “anticipate,” “believe,” “estimate,” “expect,” “intend,” “plan,” “predict” and similar expressions and their variants, as they relate to the company may identify forward-looking statements. Forward-looking statements are neither historical facts nor assurances of future performance. Although the company believes the expectations reflected in such forward-looking statements are reasonable, the company can give no assurance that such expectations will prove to be correct. Readers are cautioned that actual results, levels of activity, safety, performance or events and circumstances could differ materially from those expressed or implied in the company’s forward-looking statements due to a variety of factors, including risks and uncertainties related to the company’s ability to advance MZE829, MZE782 and its other therapeutic candidates, obtain regulatory approval of and ultimately commercialize the company’s therapeutic candidates, the timing and results of preclinical studies and clinical trials, the company’s ability to fund development activities and achieve development goals, its ability to protect its intellectual property, general business and economic conditions, and risks related to the impact on its business of macroeconomic conditions, including inflation, volatile interest rates, tariffs, instability in the global banking sector, and public health crises. Further information on potential risk factors that could affect the company’s business and its financial results are detailed under the heading “Risk Factors” included in the company’s annual and quarterly reports and other filings filed from time to time with the U.S. Securities and Exchange Commission, including the company's Annual Report on Form 10-K and Quarterly Reports on Form 10-Q. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. These forward-looking statements speak only as of the date of this presentation and Maze undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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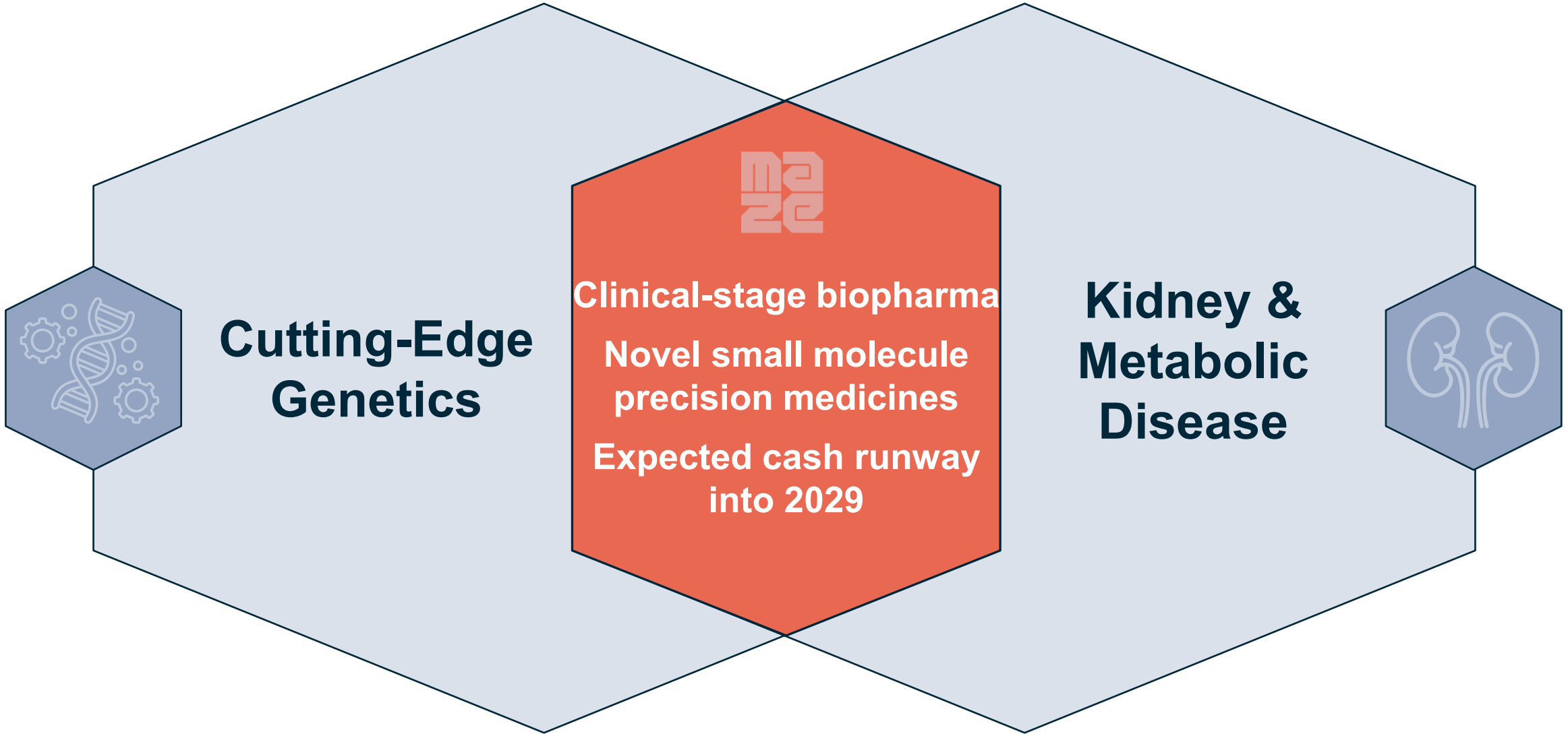
Maze Therapeutics: Harnessing the power of human genetics to transform the lives of patients



Focused Pipeline in Kidney & Metabolic Diseases

Program	Target	Indication	Target Validation	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Catalysts
MZE829	APOL1	APOL1-mediated Kidney Disease (AMKD)	HORIZON Phase 2 enrollment complete						Additional HORIZON data in early 2027
MZE782	SLC6A19	Phenylketonuria (PKU)	CIPheR Phase 2 enrolling						Topline data in 2027
		Chronic Kidney Disease (CKD)	Phase 2 ready						Initiate Ph2 in 1H 2027
Research & Discovery	Multiple	Kidney & Metabolic Diseases, including obesity							
MZE001 / S606001	GYS1	Pompe Disease	SHIONOGI Phase 2 enrolling						

Expected cash runway into 2029 based on the current business plan



MZE829

APOL1-Mediated Kidney Disease (AMKD)

MZE829: Addressing Critical Needs in AMKD

Disease Burden

Earlier Disease Onset: APOL1 high-risk variants linked to CKD onset before age 50[1]

Accelerated Progression: Dialysis initiation ~10 years earlier than non-APOL1 CKD patients[2]

Suboptimal Outcomes with SoC: Patients with AMKD progress rapidly to ESKD despite treatment with available therapies[3]

MZE829 Opportunity

Best-in-class Potential: Selective APOL1 inhibitor with potential to modify disease course

Dual APOL1 Inhibition: Disrupts channel function by inhibiting pore assembly and blocking ion flux for broader disease modulation

Long-Term Impact: Potential to delay progression to ESKD



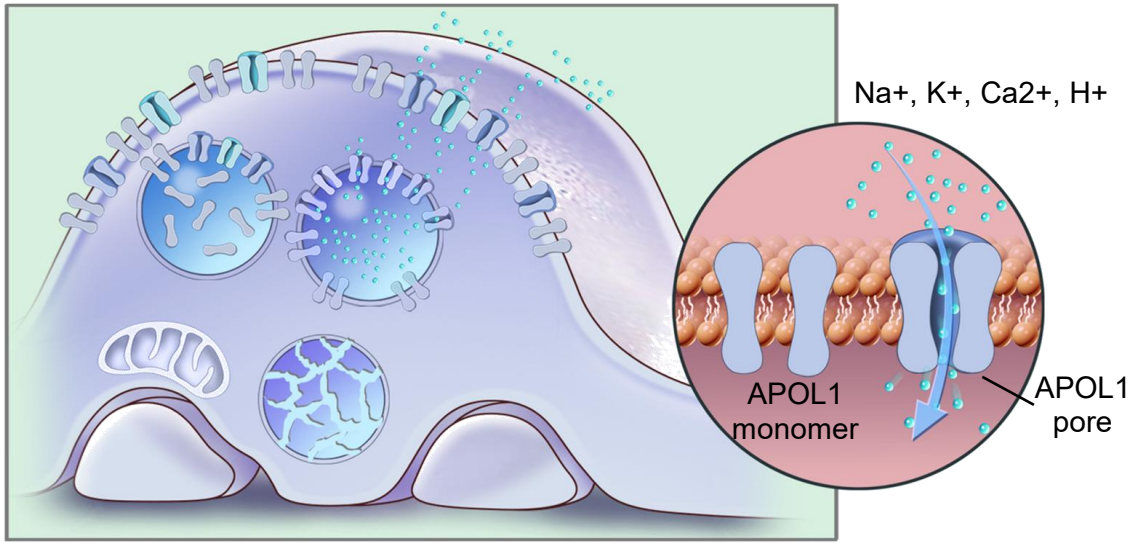
1. Freedman BI et al. APOL1 renal-risk variants are associated with earlier onset of end-stage kidney disease and with faster progression of nephropathy in African Americans. Kidney Int. 2015;87(1):120-129. 2. Freedman BI, Cohen AH. Hypertension-attributed nephropathy: what's in a name? Nat Rev Nephrol. 2016;12(1):27-36. 3. Nadkarni GN et al. APOL1 genotype and risk of kidney disease progression and mortality: A prospective cohort study. Lancet. 2022;399(10343):2031–2039.
SoC: standard of care; ESKD: end-stage kidney disease

MZE829 Dual Mechanism: Disrupts APOL1 Pore Assembly and Inhibits Pore Function

APOL1-Driven Podocyte Death and Kidney Damage

Toxic APOL1 pores are increased in podocytes

Increased number of toxic pores disrupts podocyte function and leads to podocyte death

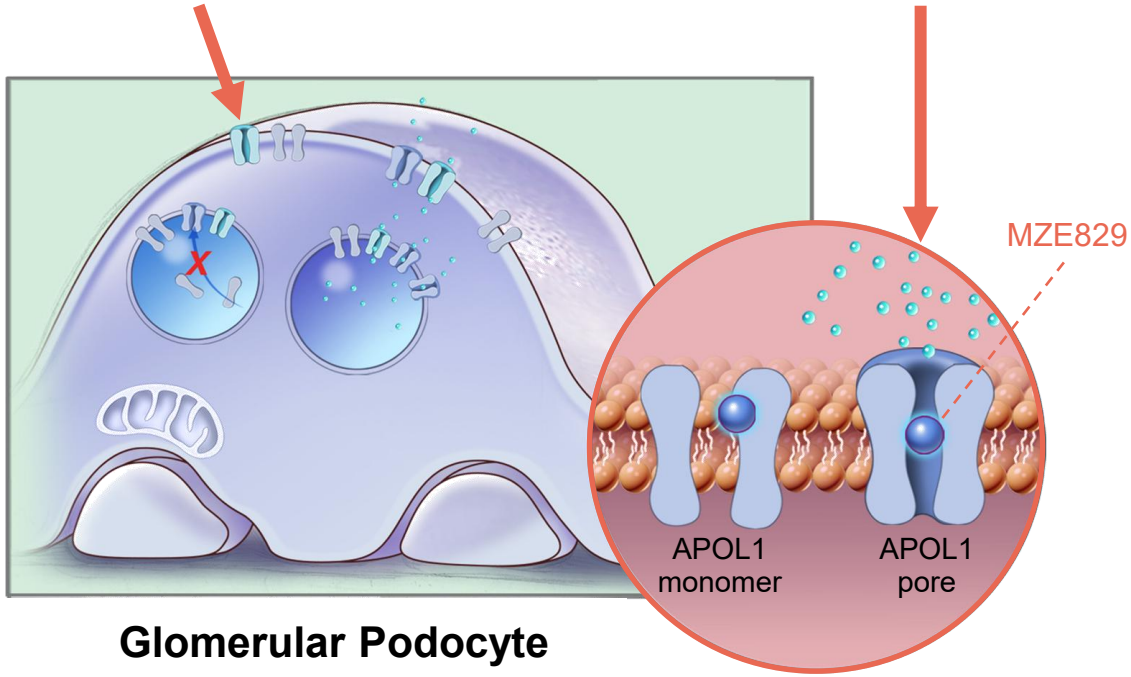


Glomerular Podocyte

MZE829 Dual Mechanism of Action

1 MZE829 blocks pore formation

2 MZE829 blocks pore function

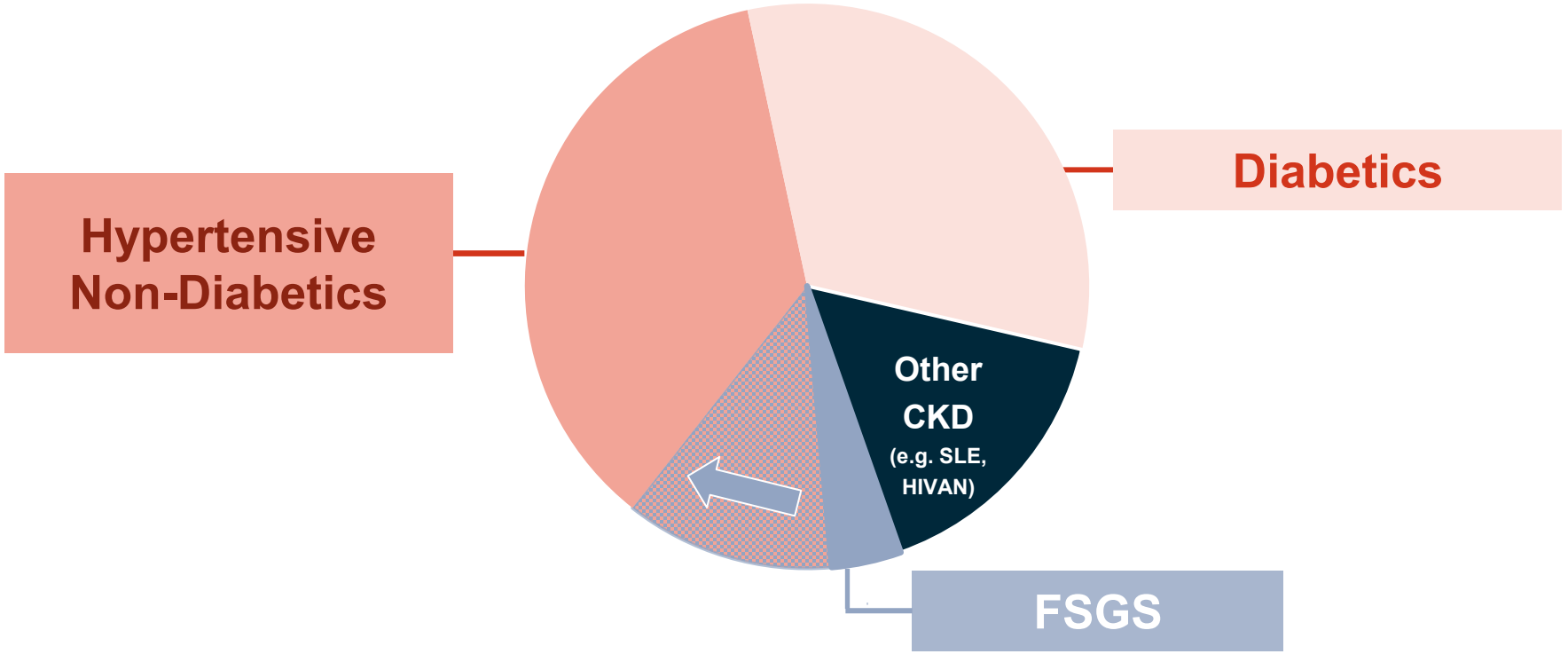


Glomerular Podocyte

AMKD Represents Large Market Opportunity Without Approved Therapies

At least 250,000

Estimated AMKD patients in U.S.



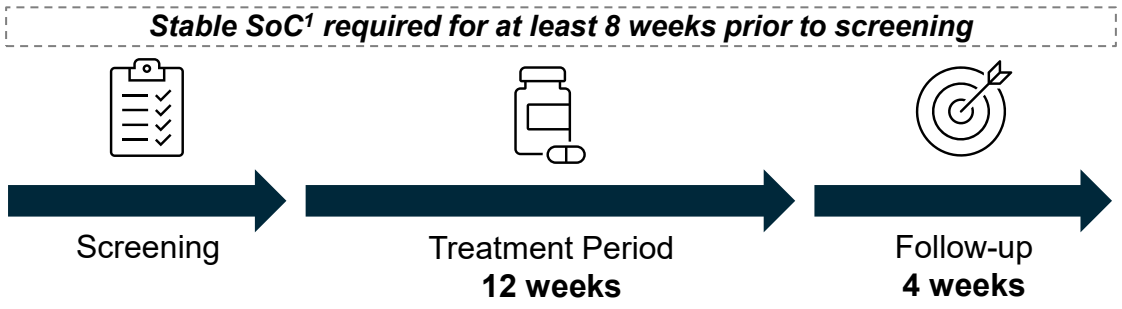
No approved therapies currently exist for AMKD despite its prevalence and severity

SLE = systemic lupus erythematosus, HIVAN = HIV-associated nephropathy, FSGS = Focal Segmental Glomerulosclerosis

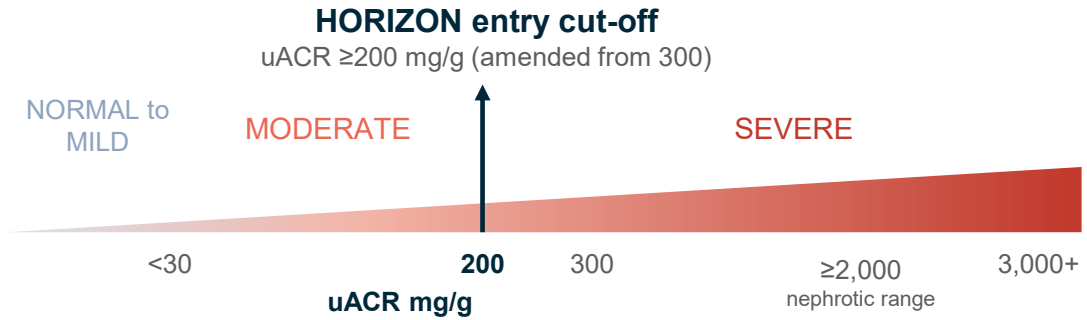
MZE829 Phase 2 HORIZON Study

Enrollment complete; data from all three cohorts expected in early 2027

Patient Timeline



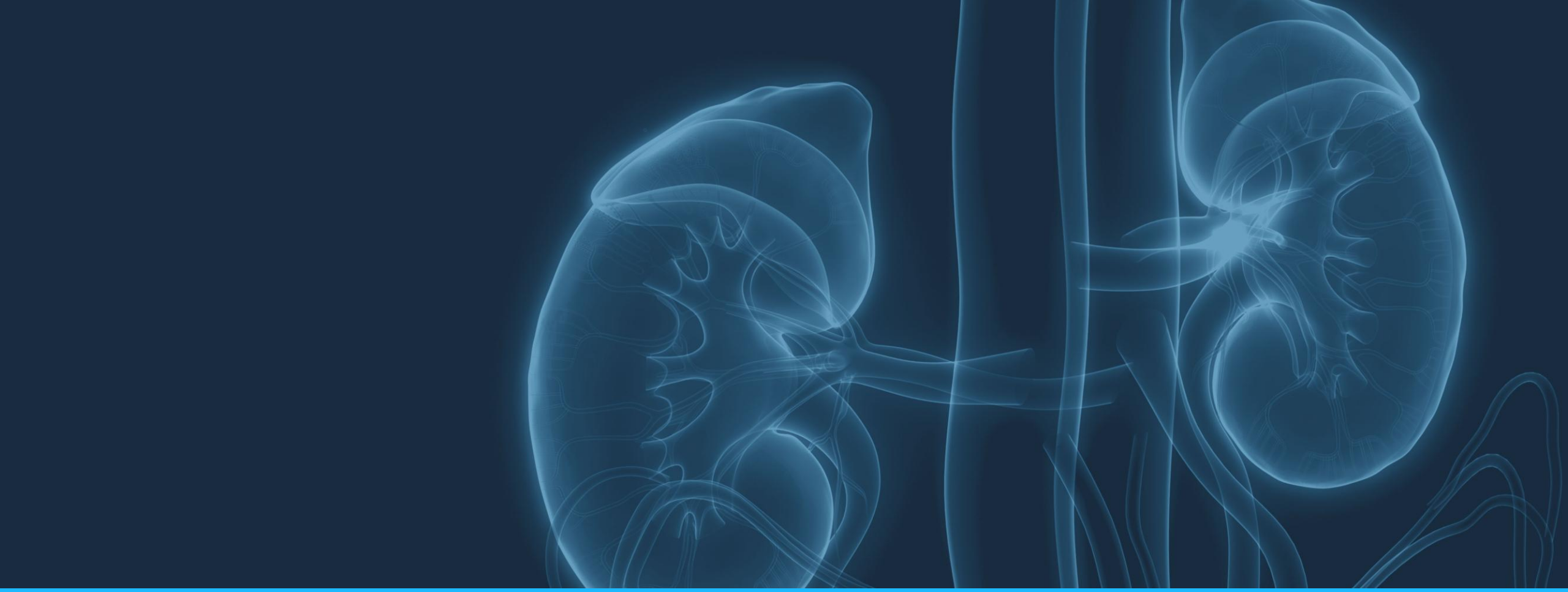
uACR and Disease Severity



Study Elements

- MZE829 once-daily 250 mg oral
- Open-label basket study in three cohorts:
  - AMKD without diabetes
  - AMKD with diabetes
  - FSGS without diabetes[3]
- All patients have two copies of the APOL1 risk variants
- Stable background CKD regimen (RAASi +/- other SoC, e.g., SGLT2i, MRA) for at least 8 weeks prior to study
- In-clinic/home-health visits every ~4 weeks
- Enrollment complete: >50 patients across three cohorts, exceeding the target of 10–15 per cohort
- **Endpoints:**
  - Primary: safety, tolerability
  - Secondary: uACR[2], PK
  - Exploratory: uPCR[2]

1. SoC = standard of care
2. uACR = mg urine albumin / g urine creatinine; uPCR = mg urine total protein / g urine creatinine
3. FSGS diagnosed by kidney biopsy



Initial Results

HORIZON: Key takeaways from initial data results

- Demonstrated clear uACR reduction in patients with AMKD
- Data supports pivotal study initiation in FSGS, and in broad AMKD
- Promising initial results in AMKD with diabetes support further development in this population

HORIZON: Summary of initial data read out in March 2026

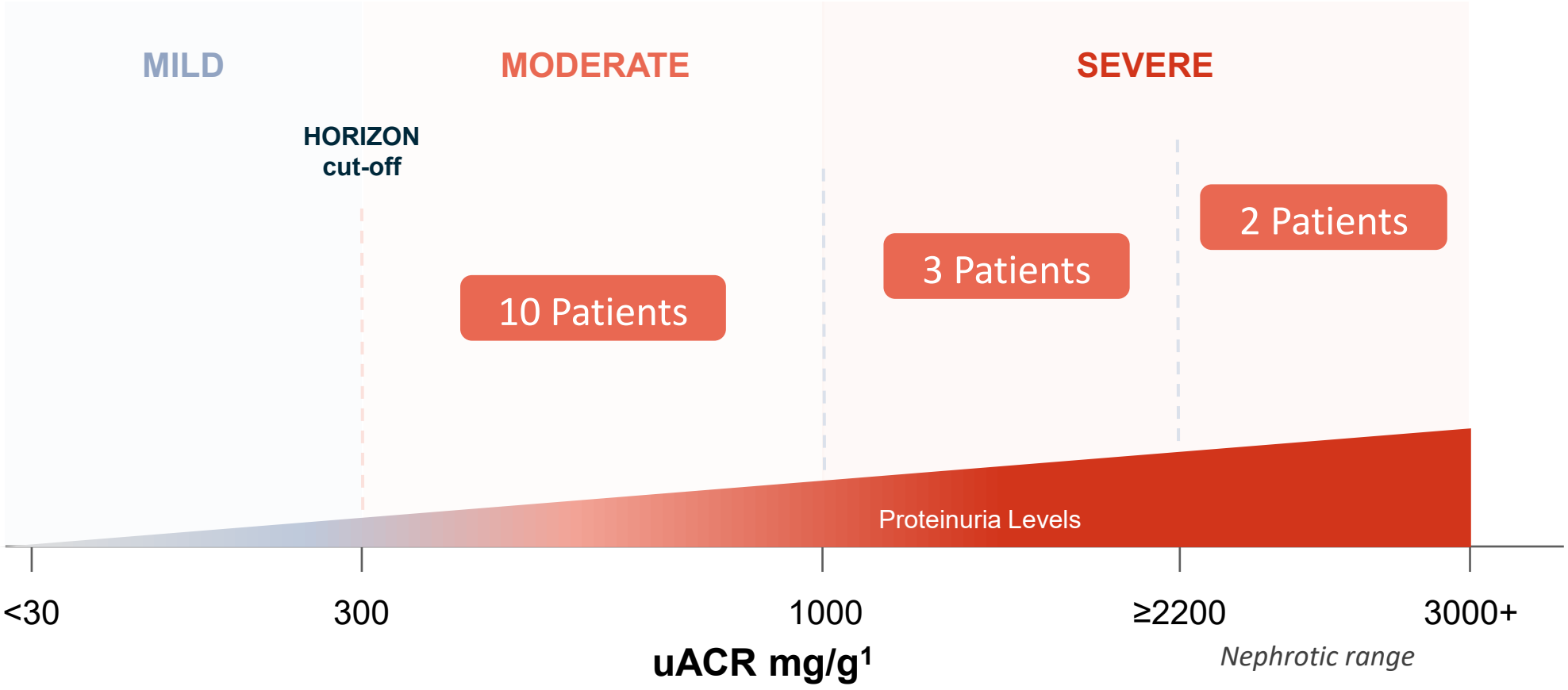
Study Design	• Open-label basket study in adults with proteinuric chronic kidney disease (CKD) and APOL1 high-risk genotype • 1st clinical study in genetically defined, broad AMKD population, including patients with less severe proteinuria and patients with diabetes
Dosing	• MZE829 250 mg oral once-daily
Safety & Tolerability	• MZE829 well-tolerated and consistent with Ph 1 safety profile
Clinical Proof-of-Concept in Broad AMKD	• MZE829 demonstrated clinically meaningful mean reduction of 36% in uACR (proteinuria) across 12 evaluable patients • 49% mean reduction seen in AMKD patients without diabetes • Promising early data in AMKD patients with diabetes
Best-in-class potential in FSGS	• FSGS patients showed a 62% mean reduction in uACR, reinforcing MZE829's differentiation through its dual mechanism of action

Demographics Overview

Characteristic	Total AMKD (N=15) [1]	AMKD without diabetes (N=8)	AMKD with diabetes (N=7)
Age — yr	49.1±12.0	46.4±13.4	52.3±10.3
Sex — no. (%)			
Female	7 (46.7)	4 (50.0)	3 (42.9)
Male	8 (53.3)	4 (50.0)	4 (57.1)
APOL1 genotype — no. (%)			
G1/G1	3 (20.0)	1 (12.5)	2 (28.6)
G1/G2	9 (60.0)	6 (75.0)	3 (42.9)
G2/G2	3 (20.0)	1 (12.5)	2 (28.6)
Body Mass Index [2]	33.4±6.7	32.9±6.0	34.1±8.0
uACR (mg/g) [3]	1113.7±812.8	1371.0±946.1	819.7±555.2
uPCR (mg/g) [3]	1724.3±1169.9	2046.2±1299.8	1356.5±962.7
eGFR (mL/min/1.73 m^2) [4]	41.9±13.5	42.2±13.8	41.7±14.4
# with reported FSGS	5	5	0

1. 12 patients included in efficacy analysis; 3 patients did not meet 80% compliance for efficacy analysis
2. BMI expressed as kg/m2 x 703; mean ± SD shown
3. uACR / uPCR at baseline was calculated as mean of 3 consecutive first morning voids during screening period; mean ± SD shown
4. eGFR based on CKD-EPI equation

Phase 2 study included patients with moderate to severe AMKD



1. uACR at baseline was calculated as mean of 3 consecutive first morning voids during screening period

Safety Overview – Key Takeaways

MZE829 well-tolerated and consistent with Ph 1 safety profile

- No Serious Adverse Events (SAEs)
- 5 patients had treatment-related adverse events (TRAEs): all mild or moderate
- One patient had a TRAE leading to treatment discontinuation due to mild nausea
- No clinically relevant changes in vital signs, laboratory tests and ECGs

TRAEs seen in 2 or more patients	
	MZE829 (N=15)
Headache	2 (13%)
Diarrhea	2 (13%)

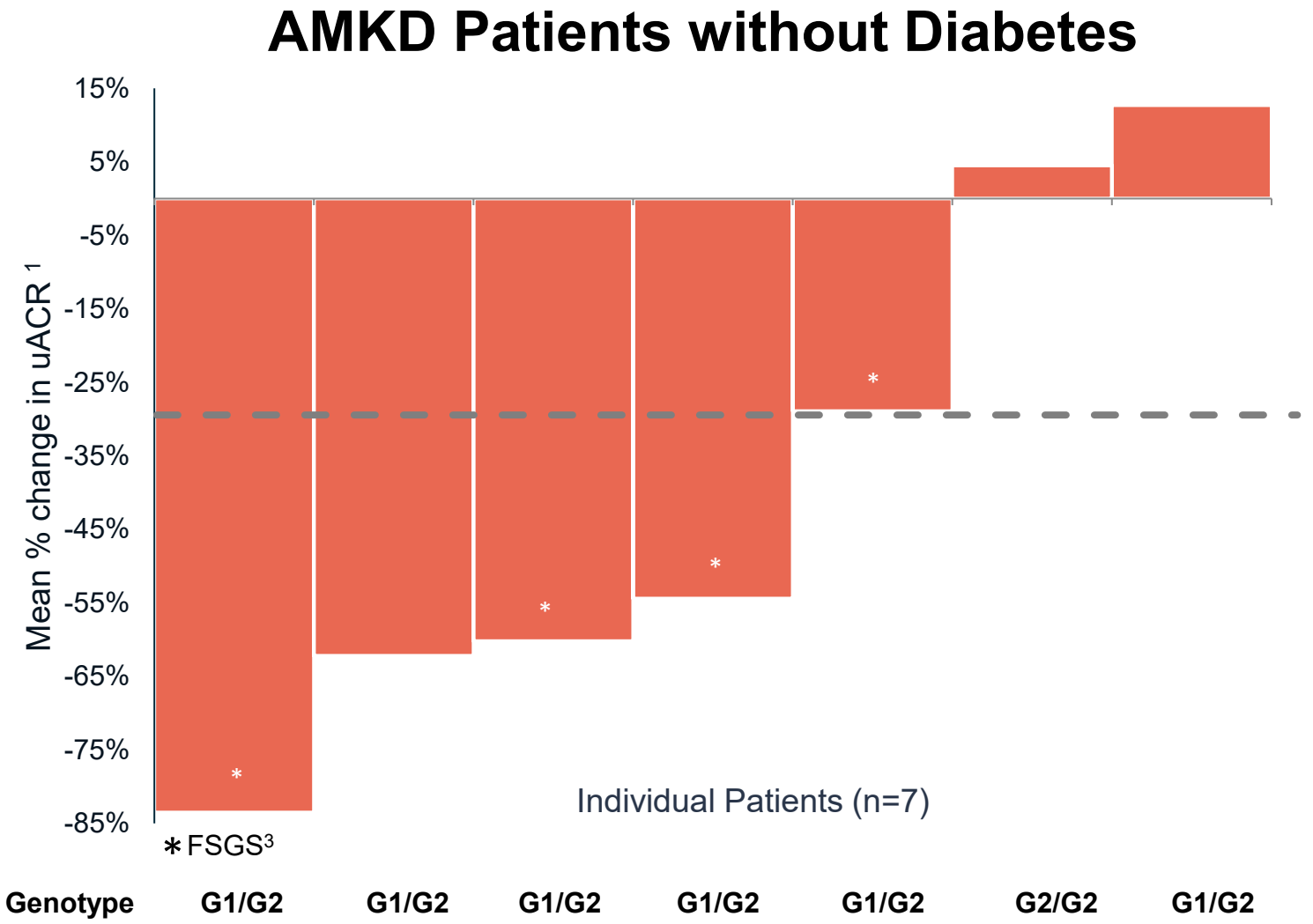
Summary of Adverse Events

AE Category no. (%)	Total AMKD (N=15)[1]	Non-Diabetic (N=8)	Diabetic (N=7)
Adverse event	10 (66.7)	6 (75.0)	4 (57.1)
TEAE based on severity	10 (66.7)	6 (75.0)	4 (57.1)
Mild	5 (33.3)	4 (50.0)	1 (14.3)
Moderate	4 (26.7)	2 (25.0)	2 (28.6)
Severe	1 (6.7)	0	1 (14.3)
Treatment-related TEAE	5 (33.3)	4 (50.0)	1 (14.3)
Mild	3 (20.0)	3 (37.5)	0
Moderate	2 (13.3)	1 (12.5)	1 (14.3)
Severe	0	0	0
Serious adverse event	0	0	0
TEAE leading to treatment discontinuation	1 (6.7)	1 (12.5)	0
Most common TRAEs			
Diarrhea	2 (13.3)	1 (12.5)	1 (14.3)
Headache	2 (13.3)	0	2 (28.6)

Safety data includes all patients that received at least one dose and either completed treatment or completed the Week 12 assessment

1. N represents number of patients

Significant Reductions Noted in AMKD Patients without Diabetes, Including Those with FSGS



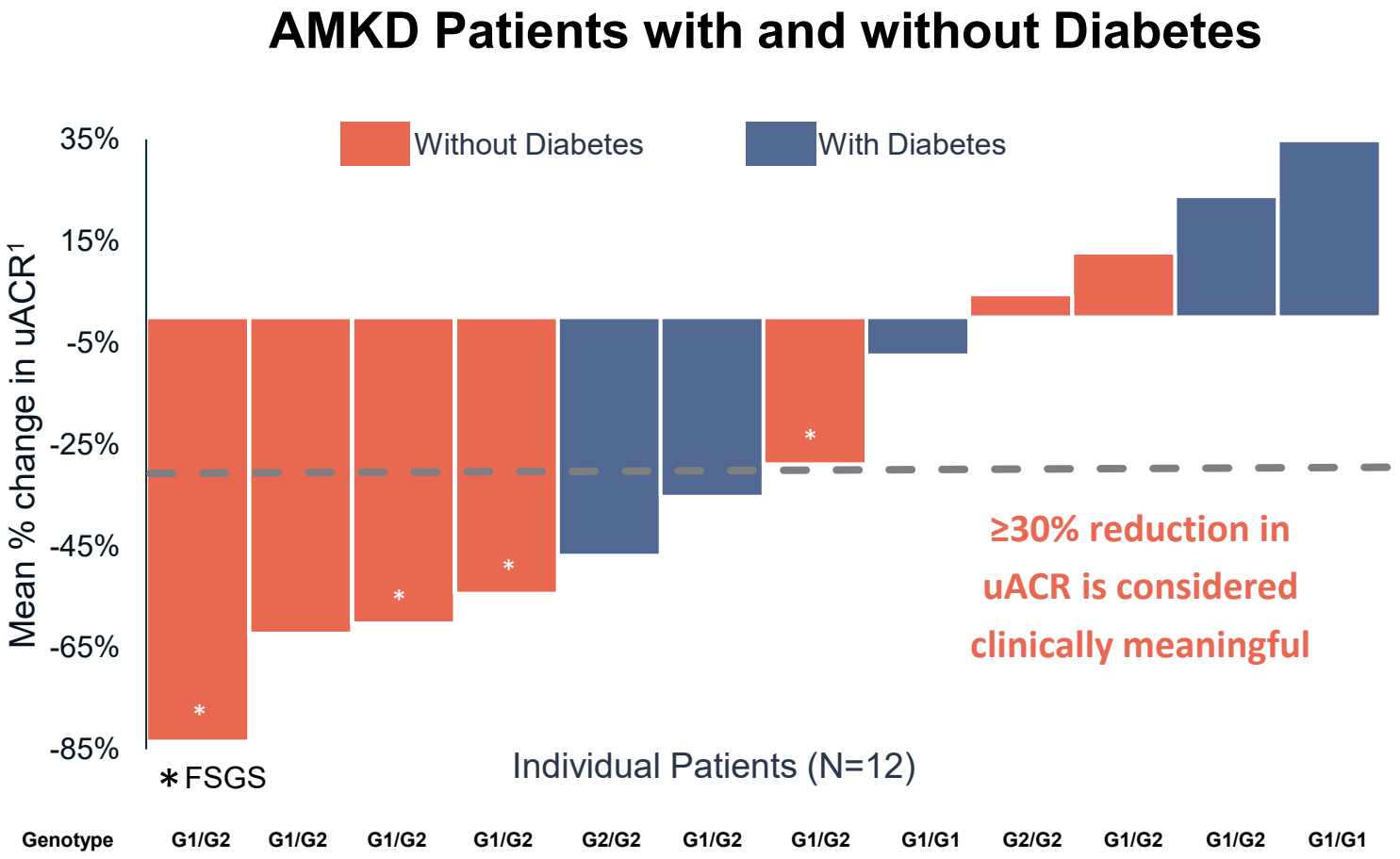
Mean uACR reduction in patients without diabetes at 12 weeks: **49%**[2]

Response rate at ≥30% uACR reduction: **57%**[2]

Mean uACR reduction in FSGS subset: **62%**[2]

1. uACR at baseline was calculated as mean of 3 consecutive first morning voids during screening period
2. AMKD without diabetes uACR -49% (90% CI -68.6 %, -16.0%); Response rate 57% (90% CI 23%, 87%); FSGS -62% (90% CI -81.4%, -21.6%);
3. 1 of the 5 FSGS patients did not meet 80% compliance in the study

MZE829 demonstrates clinical proof-of-concept in evaluable AMKD patients



Mean uACR reduction for both cohorts at 12 weeks: **36%**[2]

Response rate at ≥30% uACR reduction: **50%**[2]

Early promising data in AMKD patients with diabetes

1. uACR at baseline was calculated as mean of 3 consecutive first morning voids during screening period
2. AMKD with and without diabetes uACR -36% (90% CI -53.3% , -11.4%); response rate 50% (90% CI 25.0%, 75.0%);

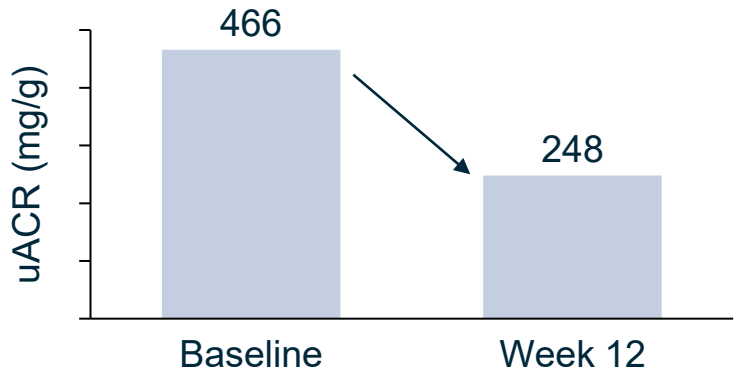
Promising early data in AMKD patients with diabetes

Patient with Early Disease Onset

G2/G2

- 37-year-old male
- Stage 2 CKD
- Type 2 diabetes
- Hypertension

Background Meds	• Dulaglutide (GLP-1RA) • Nifedipine (anti-hypertensive) • Hydrochlorothiazide (diuretic)



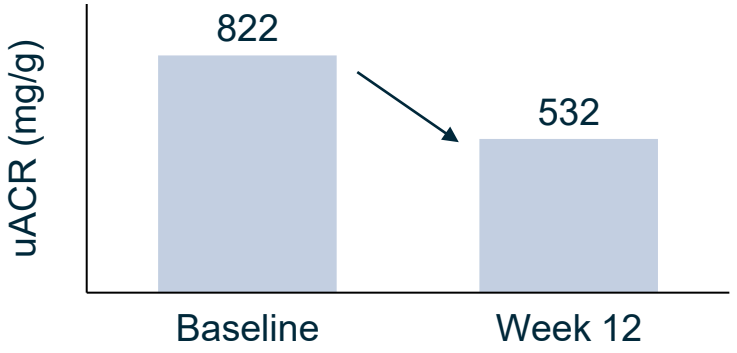
47% uACR Reduction

Patient with Multiple Comorbidities

G1/G2

- 63-year-old male
- Stage 3 CKD
- BPH
- Type 2 diabetes
- Hypertension
- Osteoarthritis
- Hyperlipidemia
- Hyperparathyroidism
- Gout

Background Meds (9 Total)	• Empagliflozin (SGLT2i) • Finerenone (MRA) • Amlodipine (anti-hypertensive) • Chlorthalidone (diuretic) • Other



35% uACR Reduction

MZE829 Next Steps

Complete HORIZON

- Targeting data updates in early 2027 on three 10-15 patient cohorts:

AMKD without diabetes	AMKD with diabetes	AMKD with FSGS

- Study and protocol amendments enacted:
  - uACR threshold lowered to 200 mg/g
  - Number of FSGS patients capped
  - Allowed patients to take with food

Initiate Pivotal Program

- Targeting pivotal trial initiation in 1H 2027, subject to regulatory feedback
- Expecting to incorporate learnings from PARASOL project in AMKD

2026: Focused on clinical execution and pivotal study preparation

MZE782



Phenylketonuria (PKU)

Topline proof-of-concept data in patients expected 2027

MZE782: Oral Therapy with the Potential to Transform PKU Management

Disease Burden

- **Limited Efficacy of PAH Activators:** Only 10 - 20% of patients with classic PKU respond to treatment[1, 2]
- **Strict Dietary Burden:** Lifelong **low-Phe diet is difficult to maintain**[3]
- **Neuropsychiatric Burden:** Fluctuating Phe levels can **impair brain function**[4]

MZE782 Opportunity

- **Applicability Across PKU Spectrum:** Designed to benefit patients with classic, moderate and mild forms of the disease
- **Convenient, Well-Tolerated Oral Therapy:** Oral dosing may improve adherence and patient acceptance
- **Potential for Diet Liberalization:** May reduce dependence on strict low-Phe diets, improving quality of life and adherence

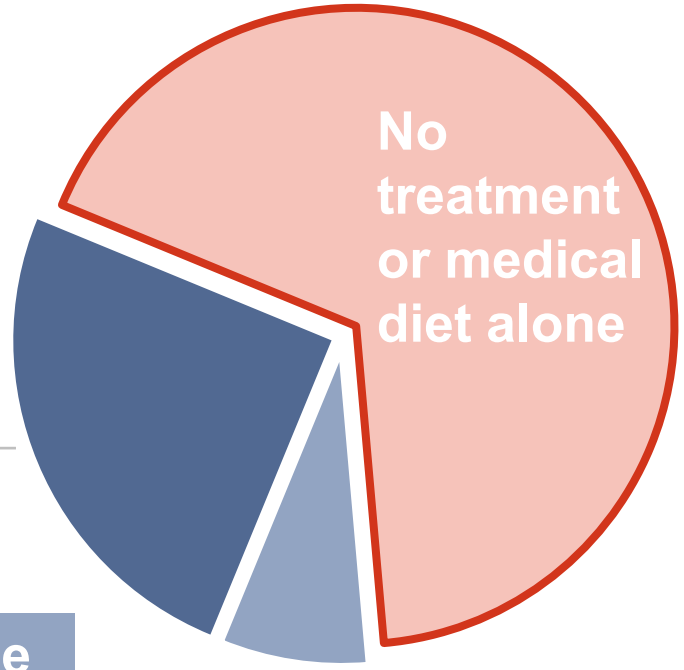


1. Trefz, F K et al. “Significance of genotype in tetrahydrobiopterin-responsive phenylketonuria.” Journal of inherited metabolic disease vol. 32,1 (2009): 22-6. 2. Longo, Nicola et al. “Long-term safety and efficacy of sapropterin: the PKUDOS registry experience.” Molecular genetics and metabolism vol. 114,4 (2015): 557-63. 3. National PKU Alliance (NPKUA).https://npkua.org. 4. Bilder DA et al. Neuropsychiatric comorbidities in adults with phenylketonuria: A retrospective cohort study. Mol Genet Metab. 2013;108(3):155–160.

PKU Represents Significant Need

At least 60,000

Estimated PKU patients in key geographies



Potential Opportunity for MZE782

- Ability to work across the entire spectrum of PKU patients
  - Patients who are not well controlled by current therapies
  - Previously untreated patients with existing therapies

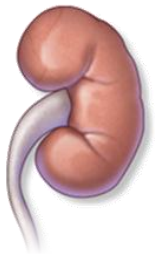
Note: Chart represents approximate breakdown of US patients

MZE782: Substrate Reduction Therapy for PKU

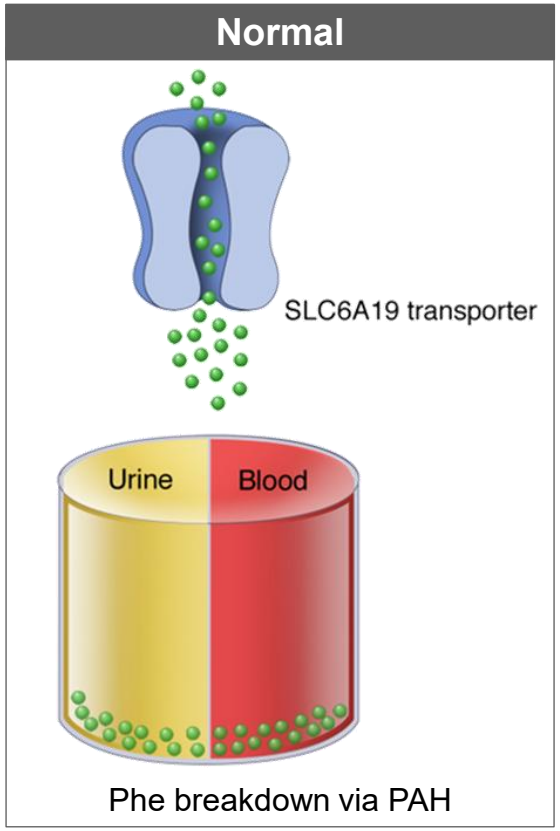
Leveraging Maze's expertise in developing oral substrate reduction therapies



Phe uptake in gut



Kidney Phe reabsorption



Potential for MZE782 to Work Across Entire Spectrum of Patients with PKU

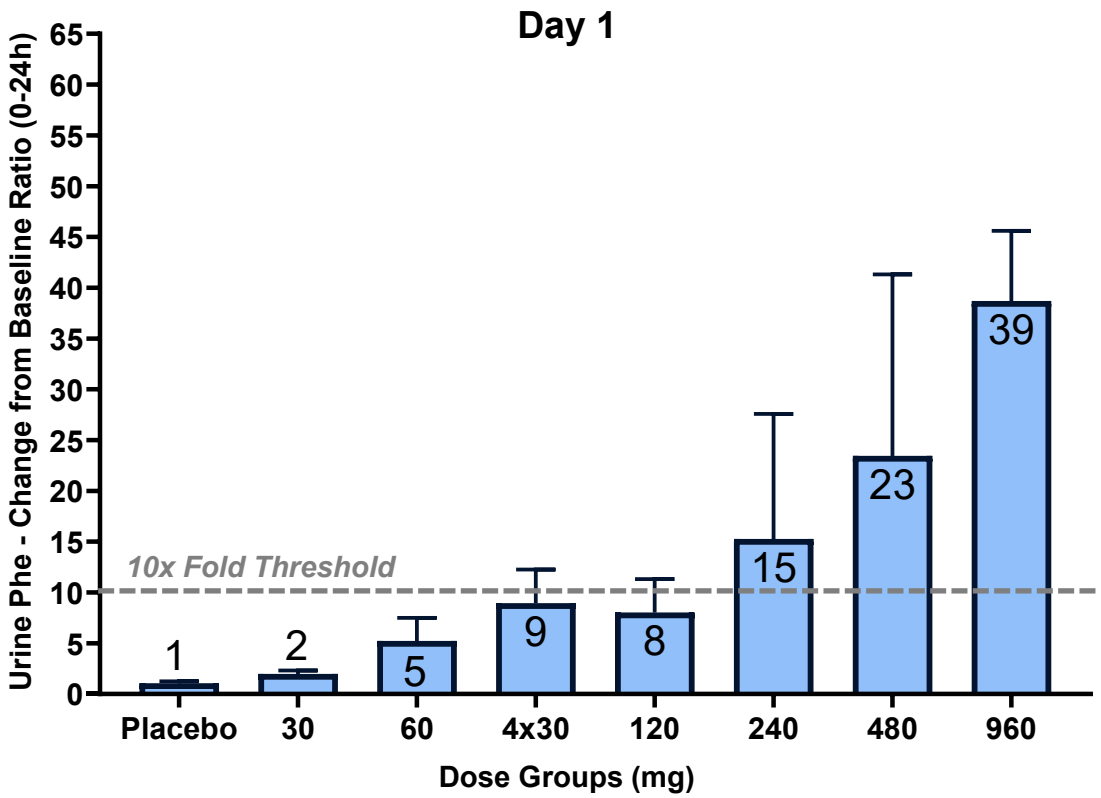
PAH = phenylalanine hydroxylase

Strong Phase 1 Data supportive of advancement into Ph 2

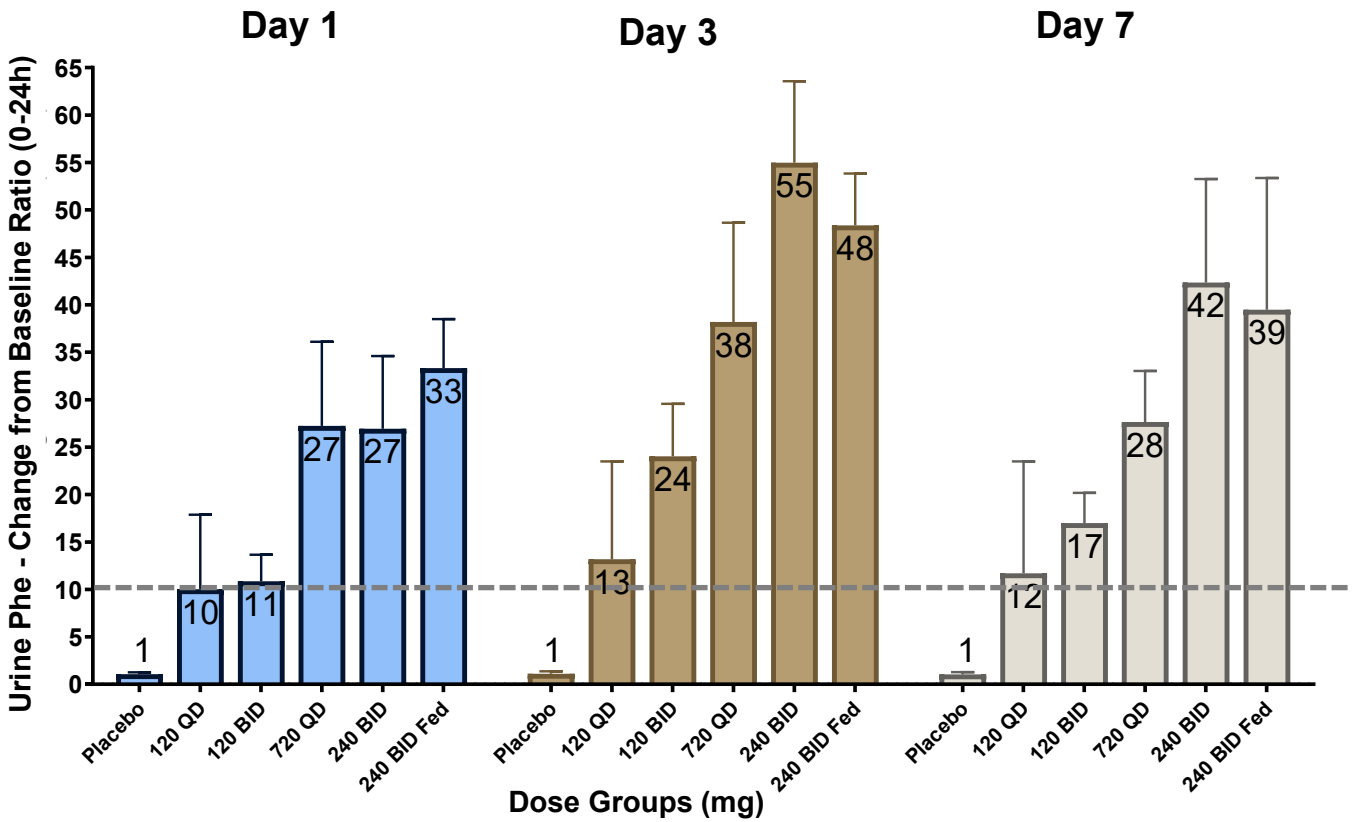
Study Design	• Randomized, double-blind, placebo-controlled study evaluating single ascending doses and multiple ascending doses of orally administered MZE782 in **112 healthy adult volunteers**
Dosing	• The single ascending doses, or SAD, ranged from 30 mg to 960 mg and the multiple ascending doses, or MAD, with dosing once or twice daily for seven days, ranged from 120 mg to 240 mg twice daily and 120 mg to 720 mg once daily
Safety & Tolerability	• MZE782 was **well tolerated** with an **excellent safety profile** at all dose levels evaluated
Pharmacokinetics	• MZE782 had **linear PK** across single doses of 30-960 mg with half-life ~11 hrs • Supportive of a once- or twice-daily dosing regimen to be evaluated in Phase 2.
Urinary Phe	• Consistent increases in urinary excretion of Phe observed with increasing doses and plasma exposures to MZE782 – Up to **42X increase in urinary Phe** at Day 7
Food Effect	• Moderate positive food effect observed only with single doses of MZE782 • When MZE782 administered twice daily, steady state **plasma exposures similar** with or without food administration

MZE782 Clinical Study Showed Phe Excretion Exceeding 40-Fold

Single Ascending Dose



Multiple Ascending Dose



Up to 39x increase in urinary L-phenylalanine excretion over 24h following MZE782 administration

Up to 42x increase in urinary L-phenylalanine excretion on Day 7 of MZE782 administration

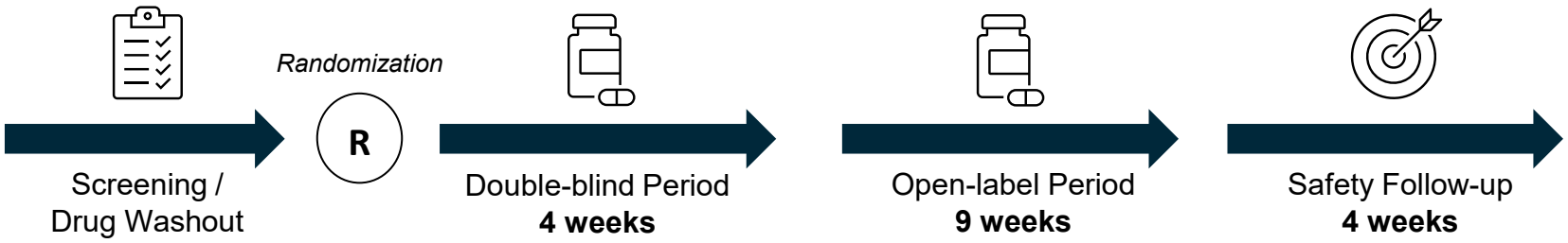
N=6 treated with MZE782 per cohort

Geometric Mean ± GSD values are plotted. Amount excreted (Ae) in urine pooled over specified time interval. Baseline is defined as the day before treatment (Day -1). Ratio=Ae post-dose/Ae at baseline. Ratio of 1 indicates no change. >1 indicates increase, <1 indicates decrease. e.g., Ratio of 10 indicates 10x increase over baseline.

MZE782 Phase 2 CIPheR in Patients with PKU

Topline proof-of-concept data in patients expected 2027

Patient Timeline



Study Elements

- MZE782 120 mg, 240 mg[1], or 240 mg with BH4 agent[2] twice-daily oral
- Double-blind, placebo-controlled, dose-ranging study
- Eligible patients
  - 18-75 years old
  - PKU diagnosis
  - Phe > 600 µMol/L (or > 360 µMol/L[2])

Endpoints

- Co-primary: safety, tolerability; absolute change in plasma Phe
- Secondary: MZE782 plasma levels, % plasma Phe reduction, % patients achieving plasma Phe ≤600, ≤360, and ≤120 µMol/L
- Exploratory: Urine Phe

1. 240 mg cohort will initiate after the 120 mg cohort completes the 4-week double-blind period and Safety Monitoring Committee review.
2. 240 mg with BH4 agent cohort will include patients with lower plasma Phe threshold and initiate after 240 mg cohort has been fully enrolled and all participants have completed Day 1.



MZE782

Chronic Kidney Disease (CKD)

Phase 2 initiation expected in 1H 2027

MZE782: Addressing Persistent Gaps in CKD Treatment

Disease Burden

- CKD is often **diagnosed late** with patients unaware of their condition until later stages of the disease[1]
- Inadequate response in a large subset of patients; **~25% of patients respond inadequately to SGLT2i therapies**[2]
- **SGLT2i treatment** associated with **increased risk of DKA and UTIs**, which may limit uptake or lead to discontinuation[3, 4]

MZE782 Opportunity

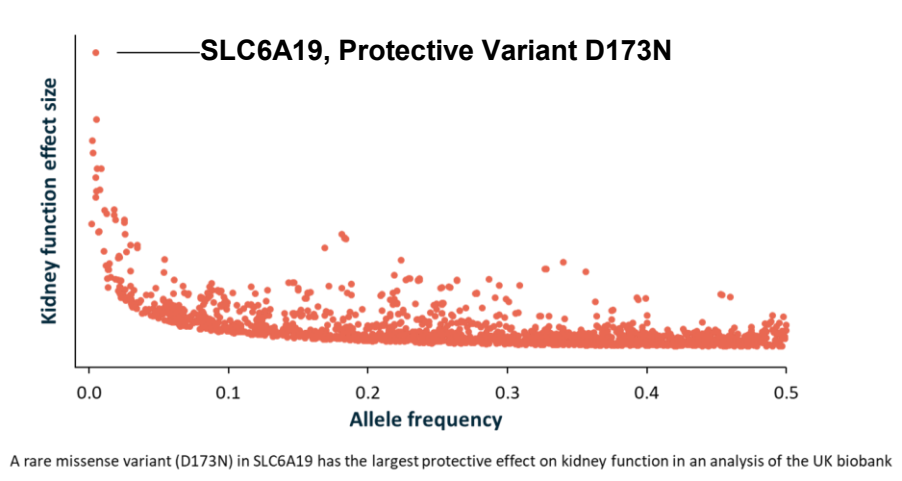
- **Monotherapy Potential:** To reduce proteinuria and slow progression to ESKD in patients inadequately controlled on SoC
- **Add-On Therapy:** Add-on potential to improve outcomes in combination with SoC and potentially reduce side effects
- **Targeted Innovation:** SLC6A19 inhibition offers a new mechanism in all CKD populations



1. Centers for Disease Control and Prevention (CDC). Chronic Kidney Disease in the United States, 2021. 2. Curovic et al. Diabetes Care 46:593, 2023. 3. Mende CW (2022) Chronic Kidney Disease and SGLT2 inhibitors: A Review of the Evolving Treatment Landscape. Adv Ther 39:148-164. 4. INVOKANA (canagliflozin) and FARXIGA (dapagliflozin) drug labels.
DKA: diabetic ketoacidosis; UTI: urinary tract infection

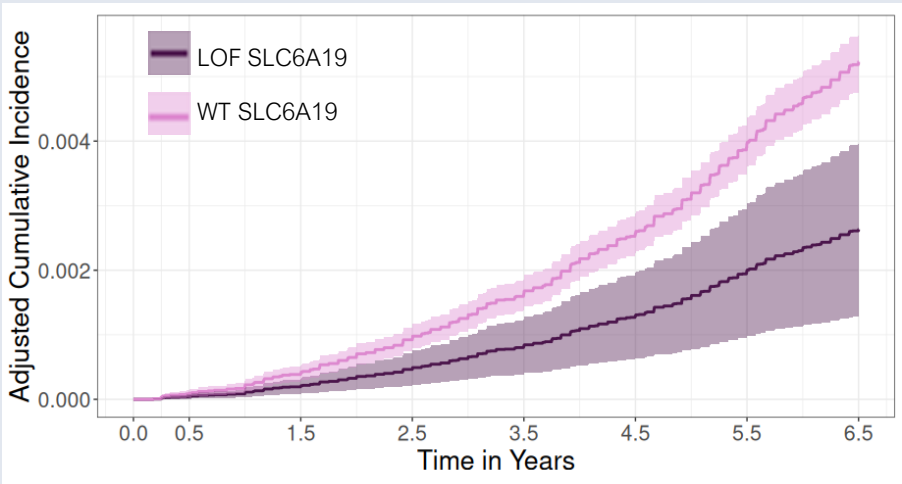
Genetic insights validate SLC6A19 as clinically meaningful CKD target

CKD Risk



A rare missense variant (D173N) in SLC6A19 has the largest protective effect on kidney function in an analysis of the UK biobank

CKD Progression

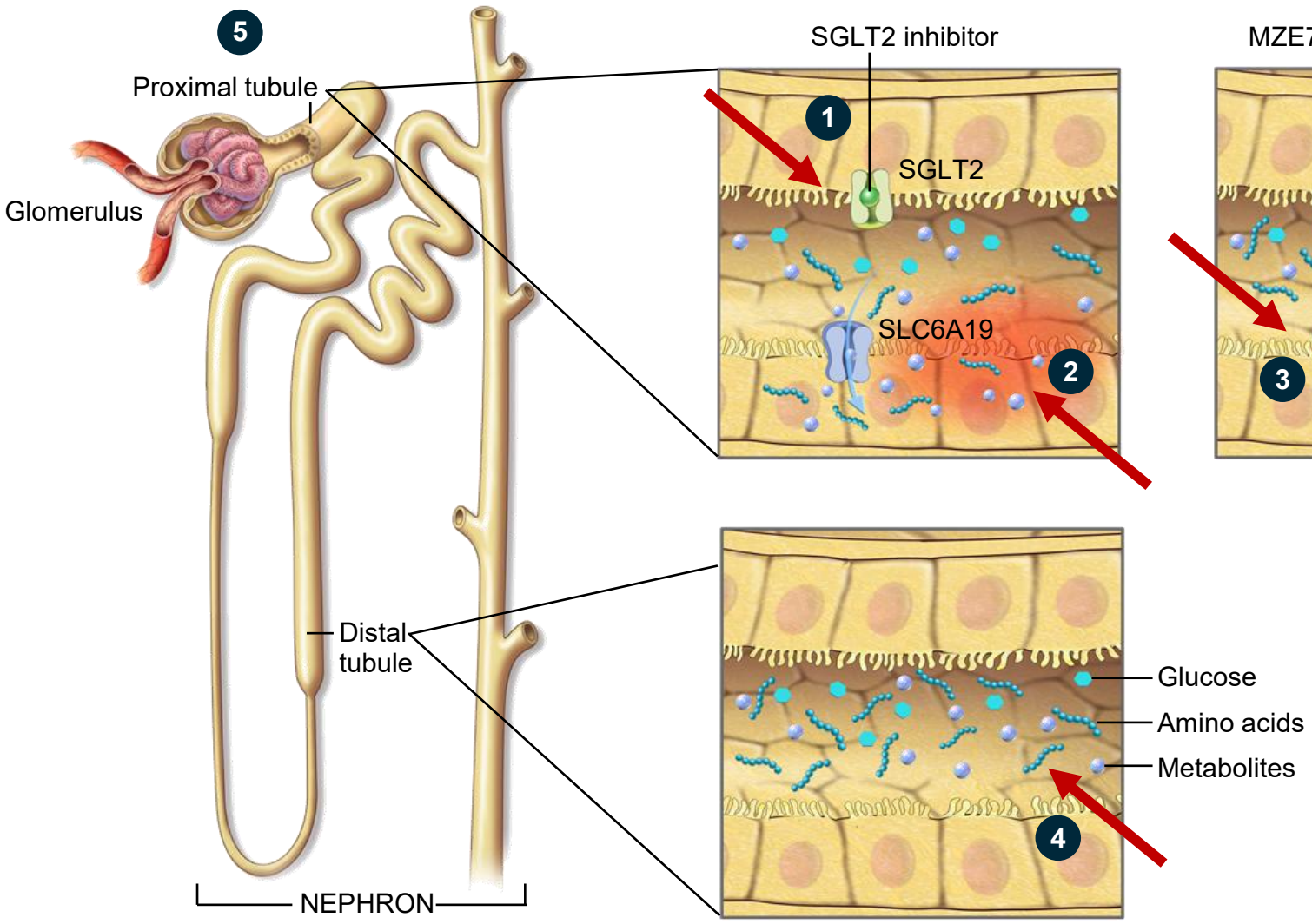


- **SLC6A19 emerged as a novel therapeutic CKD target** with a protective variant, D173N, based on analyses of independent human genetic datasets matched with longitudinal clinical data for kidney health
- **SLC6A19 LOF carriers** (i.e., heterozygous) **have ~50% lower risk of progression** to composite kidney endpoint
- Human genetics provides evidence that **inhibiting SLC6A19 is potentially safe**
- The genetic effect seen is accompanied by a <5-fold increase in urinary AA excretion

Maze internal data; Genetic ablation of SLC6A19 is protective against renal damage in mouse models of chronic kidney disease, presented at the American Society of Nephrology Kidney Week 2024. LOF: loss of function

MZE782: Kidney Detoxification with Improved Filtration

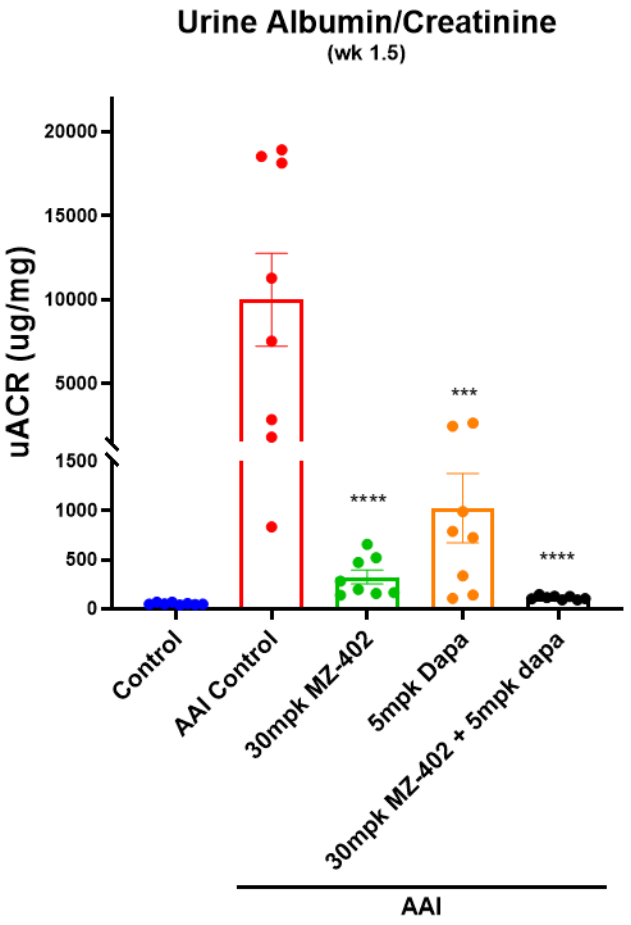
Complementary to - and potentially independent of - SGLT2 inhibition

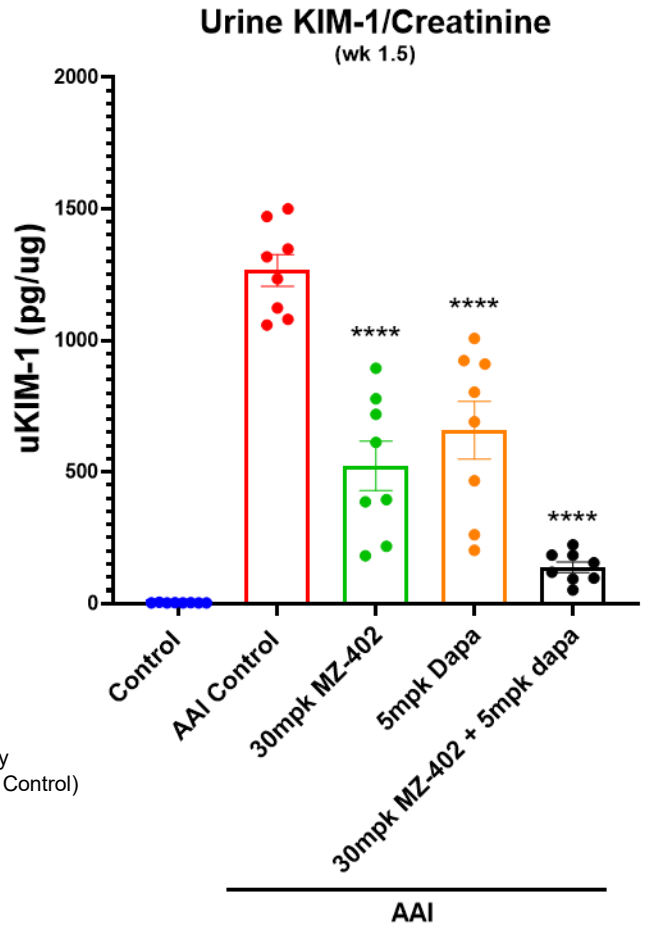


1. SGLT2 inhibitors reduce glucose and sodium reabsorption
2. SLC6A19 dysfunction allows harmful metabolites into kidney cells
3. MZE782 blocks this process, protecting cells from metabolite damage as well as reduces sodium reabsorption in the proximal tubule
4. Measuring amino acids in the urine can serve as a biomarker to test for target engagement
5. Measuring eGFR dip can serve as a biomarker for improved filtration

Demonstrated In Vivo Proof of Concept for SLC6A19 Inhibition in a Preclinical Model

SLC6A19 inhibition protects against albuminuria (uACR) and proximal tubule damage (KIM-1) through a potentially independent mechanism compared to SGLT2 inhibition





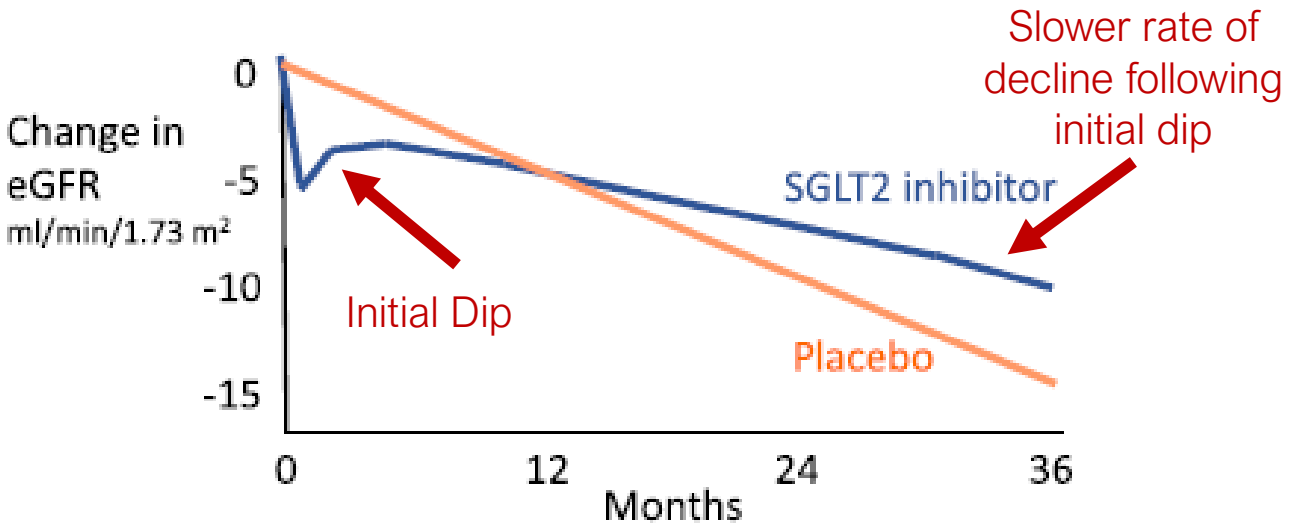
Mean ± SEM
Ordinary one-way
ANOVA (vs. AAI Control)
**** p<0.0001
*** p<0.0005

- First in vivo PoC of reduction in proteinuria with pharmacologic SLC6A19 inhibition in aristolochic acid injury (AAI) model
- Demonstrates complementary effect with SGLT2 inhibition
- Consistent with genetically-informed hypotheses that SLC6A19 inhibition could result in kidney benefit

Study ID: MZS-I023-25-021, week 1.5
KIM-1 = Kidney Injury Molecule-1, a protein biomarker that is specifically elevated in the urine and blood of individuals with kidney injury; PoC: proof of concept

Initial eGFR Dip on SGLT2i Therapy Shown to Predict Slower Chronic eGFR Decline in CKD

Typical eGFR Trajectory[1]



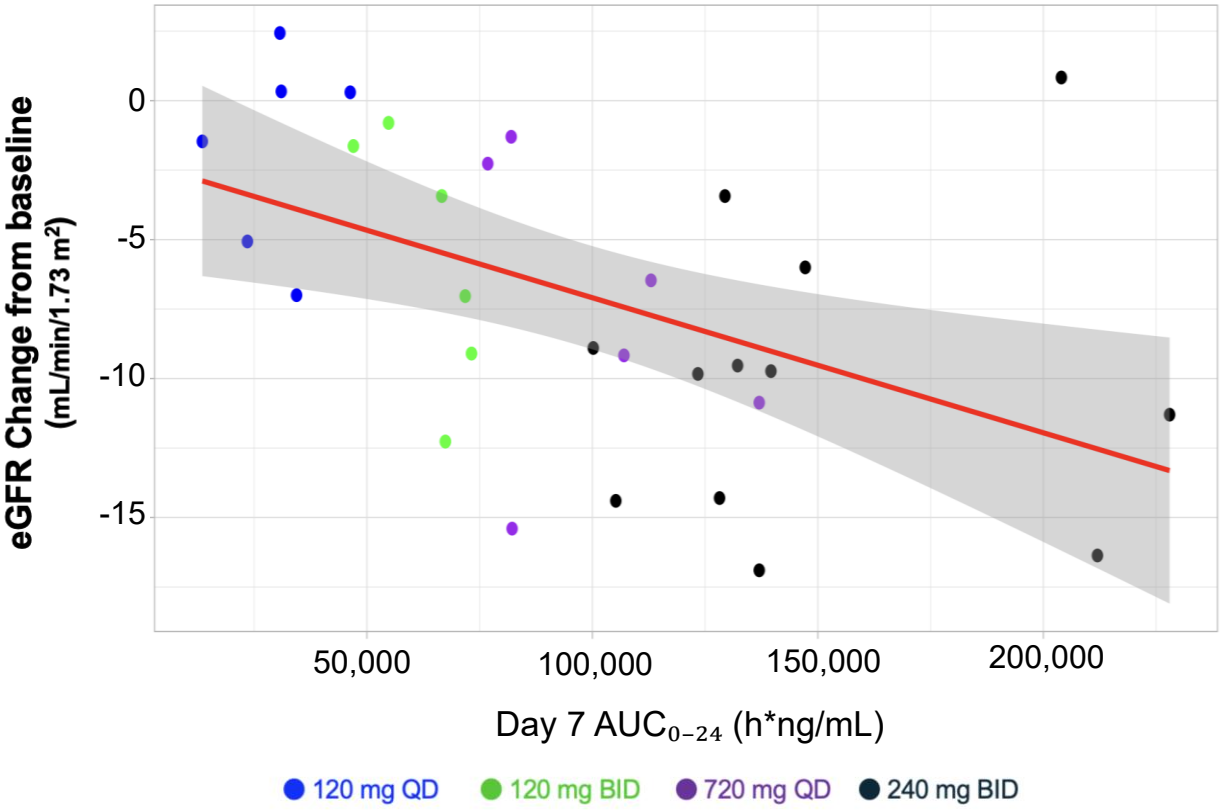
- Initial eGFR dip is a transient hemodynamic effect seen with renoprotective therapies[2,3]
- Observed with approved CKD therapies, including SGLT2 inhibitors[2,3]
- Moderate initial dip associated with slower chronic eGFR decline and better long-term kidney outcomes[2,3]
- Provides a pharmacodynamic readthrough supporting potential efficacy in CKD

Strengthens rationale for MZE782 to potentially improve CKD outcomes

1. Bailey, Clifford J et al. "Renal Protection with SGLT2 Inhibitors: Effects in Acute and Chronic Kidney Disease." Current diabetes reports vol. 22,1 (2022): 39-52. doi:10.1007/s11892-021-01442-z
2. Holtkamp, Frank A et al. "An acute fall in estimated glomerular filtration rate during treatment with losartan predicts a slower decrease in long-term renal function." *Kidney international* vol. 80,3 (2011): 282-7. doi:10.1038/ki.2011.79
3. Meraz-Muñoz, Alejandro Y et al. "eGFR Decline after SGLT2 Inhibitor Initiation: The Tortoise and the Hare Reimagined." Kidney360 vol. 2,6 1042-1047. 26 Mar. 2021, doi:10.34067/KID.0001172021

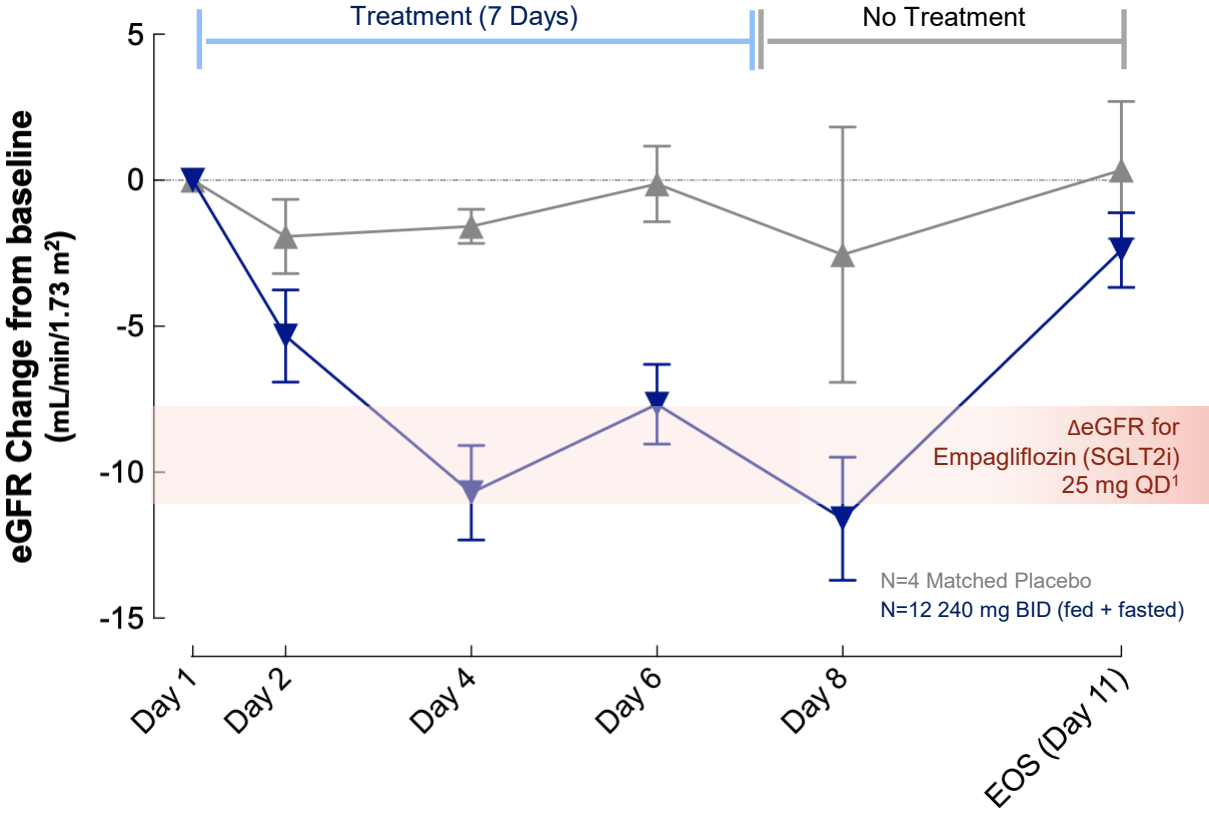
MZE782: Preliminary Clinical Data Support Potential Protective Mechanism in CKD; expected to start CKD Phase 2 in 1H 2027

Exposure Response: eGFR Change vs. MZE782 Exposure



Observed “dip” in eGFR generally consistent with exposure-response

240 mg BID vs. Matched Placebo



Early data appear to have comparable effect to other reno-protective therapies

eGFR derived using the 2021 CKD-EPI Creatinine Equation; . † Baseline eGFR = mean of the 3 pre-dose measurements ;
[1]Range of placebo-adjusted and non-adjusted % change from baseline eGFR (“eGFR dip”) at 4 weeks’ treatment with empagliflozin 25 mg QD on background ramipril in patients without significant renal impairment provided for approximate reference (Lytvyn Y, 2022). Dapagliflozin significantly lowers renal glucose threshold and reabsorptive capacity (TmG), driving glucosuria in both healthy and T2D subjects (DeFronzo et al., Diabetes Care 2013).

MZE782 Phase 2 CKD Proof-of-Concept: Broad Population with an Embedded Biomarker Signature



Design and Endpoints:

- Randomized, double-blind, placebo-controlled, BID
- Patients with CKD and Type 2 Diabetes
- Inclusive of patients on stable RAASi and/or SGLT2i
- Primary endpoints:
  - % reduction in uACR
  - Percentage of patients achieving ≥30% reduction in uACR

Biomarker signature (exploratory):

- Prognostic: higher baseline levels are associated with faster CKD progression in the GCKD[1] and other CKD cohorts
- Measured in all patients; not used to restrict enrollment
- Can potentially be used for patient stratification for subsequent clinical trials

[1] GCKD: German Chronic Kidney Disease study

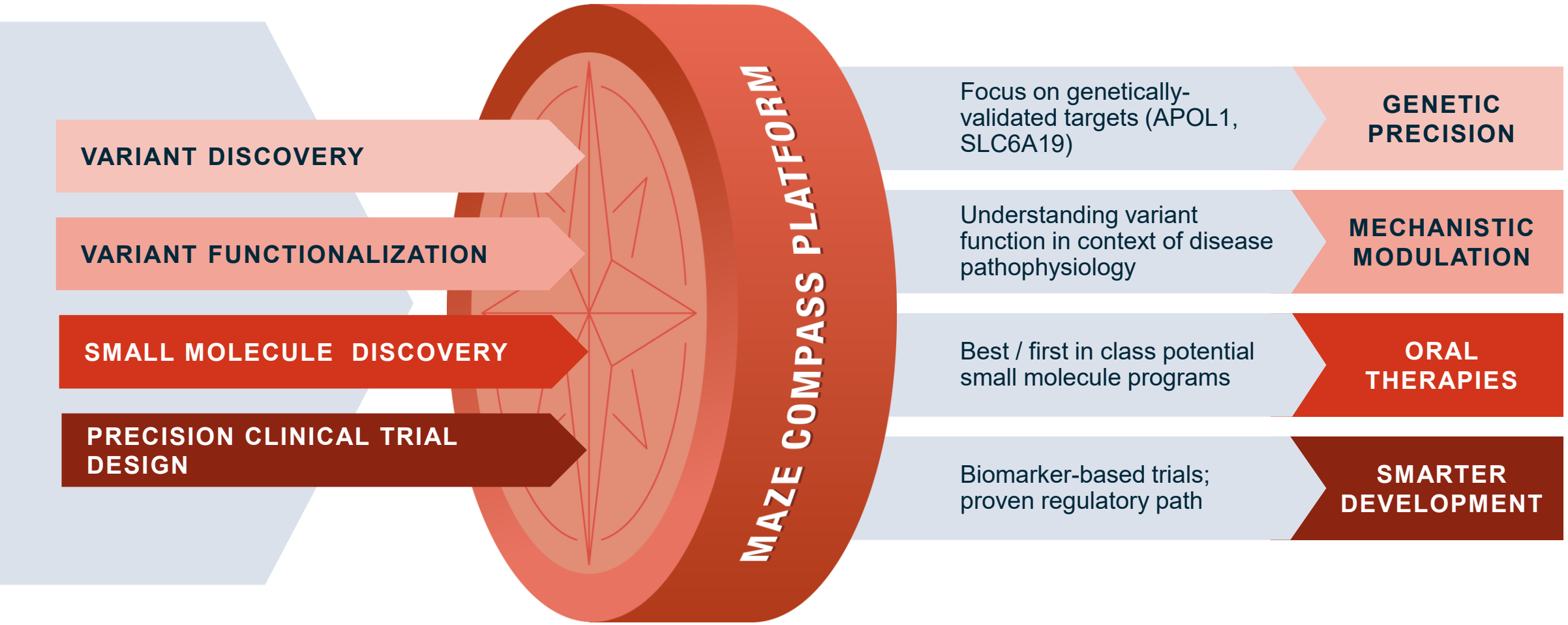


Research & Discovery

Kidney & Metabolic Diseases

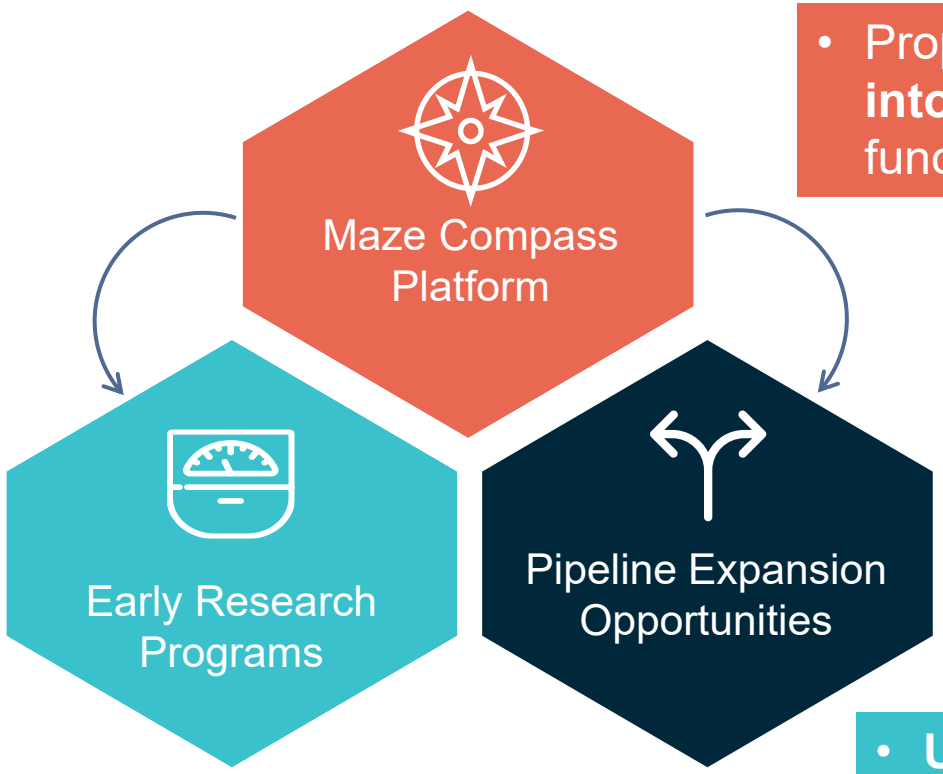
Unlocking Precision Pathways in Kidney & Metabolic Disease

Maze is built to turn complex genetics into targeted, oral medicines that change the course of disease



Navigating the Path to Precision Medicine

Compass platform accelerates precision medicine innovation through genetic insights, fueling an expanding pipeline in kidney & metabolic diseases, including obesity



- Proprietary Compass platform **translates human genetics into precision therapies** through variant discovery and functionalization to small molecule discovery and development

- Driving **lifecycle expansion** and the identification of **potential new indications**, including broader CKD populations for MZE829 (APOL1) and optimized use of MZE782 (SLC6A19) in CKD and PKU

- **Uncovering new kidney and metabolic disease candidates** through Compass-driven insights across our pre-clinical program initiatives

Key Near-Term Catalysts & Financials

Multiple paths to patients across AMKD, PKU and CKD, enabled by near-term data

Expected Key Catalysts

- **Early 2027:** MZE829 Phase 2 HORIZON data in AMKD
- **2027:** MZE782 Phase 2 topline data in PKU
- **1H 2027:** Initiate MZE782 Phase 2 PoC trial in CKD
- **1H 2027:** Initiate MZE829 pivotal program in AMKD

Financial Position

- **$477 million** cash and investments as of September 30, 2026
- Expected cash runway into 2029 based on the current business plan



Maze Therapeutics

Harnessing the power of human genetics to transform the lives of patients

November 2026