Corporate Presentation

November 2026



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Forward-looking statements

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This presentation discusses product candidates that are under clinical study and which have not yet been fully approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied to the extent it has not yet been approved.

The trademarks included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of such products.

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Important Safety Information

Indication: TRUTAKNA® (atacicept-vymj) is indicated to reduce proteinuria in adults with primary immunoglobulin A nephropathy (IgAN) at risk for disease progression.

This indication is approved under accelerated approval based on reduction of proteinuria. It has not been established whether TRUTAKNA slows kidney function decline over the long-term in patients with IgAN. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory clinical trial.

Important Safety Information

Contraindications: TRUTAKNA is contraindicated in patients with serious hypersensitivity to atacicept-vymj or any excipients of TRUTAKNA.

Warnings and Precautions

Immunosuppression and Increased Risk of Infections: TRUTAKNA suppresses the immune system by reducing antibody production, which may increase the risk of infections. Patients with chronic infection or recurring infections may have an increased risk of serious infection. In clinical trials, infections were reported in 32% of TRUTAKNA patients compared with 28% of placebo patients.

Before initiating TRUTAKNA, assess patients for active infections. Delay TRUTAKNA administration in patients with active infection until the infection resolves or is adequately treated. Monitor patients for signs and symptoms of infection during treatment with TRUTAKNA. If a serious infection develops, consider interrupting TRUTAKNA until the infection is controlled.

The concomitant use of TRUTAKNA and other immune-modulating therapies has not been evaluated. Concomitant use of TRUTAKNA with drugs that affect the immune system, including systemic corticosteroids, may increase the risk of infection.

Immunosuppression and Immunization Risk: TRUTAKNA may interfere with the immune response to vaccines and increase the risk of infection from live vaccines. Prior to initiating treatment with TRUTAKNA, complete all age-appropriate immunizations. Live vaccines are not recommended within 30 days prior to initiation or during treatment with TRUTAKNA as safety of coadministration has not been established.

Adverse Reactions: The most common adverse reactions (≥5%) in patients treated with TRUTAKNA and placebo, respectively, were infections (32% vs 28%) and local administration reactions (30% vs 5%). The most common infection was upper respiratory tract infection (12% vs 9%), and the most common local administration reactions were injection site reaction (19% vs 2%) and injection site erythema (6% vs 1%).

Use in Specific Populations

Pregnancy: Available data on TRUTAKNA used in pregnant women exposed during clinical trials are insufficient to evaluate for a drug-associated risk of major birth defects, miscarriage, or other adverse maternal or fetal outcomes.

Based on the mechanism of action, TRUTAKNA may cause immunosuppression in the in utero-exposed infant. Consider the potential clinical impact of TRUTAKNA exposure in infants exposed in utero. Pregnant women exposed to TRUTAKNA, or their healthcare provider, should report TRUTAKNA exposure by calling 1-833-633-8372.

Pediatric Use: The safety and effectiveness of TRUTAKNA in pediatric patients have not been established.

You may report side effects to the FDA at 1-800-FDA-1088 or www.fda.gov/medwatch. You may also report side effects to Vera Therapeutics at 1-833-MED-VERA or medinfo@veratx.com.

Please see full Prescribing Information for additional Important Safety Information.

This presentation includes data not in the FDA-approved Prescribing Information for TRUTAKNA and discusses investigational uses of TRUTAKNA that are not approved. No representation is made as to the safety or effectiveness of TRUTAKNA for the uses under investigation.

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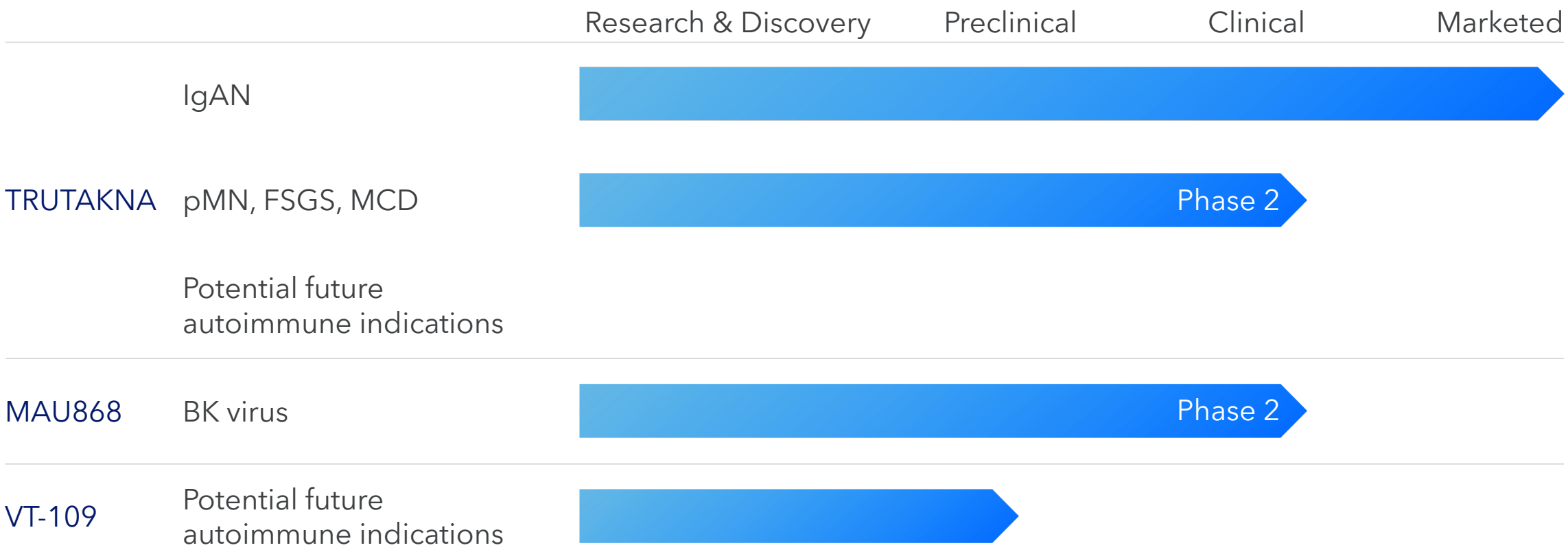
- VERA (Nasdaq) is a San Francisco-based biotechnology company with world-class development and commercialization leadership
- First commercial product: TRUTAKNA, a first-in-class BAFF and APRIL inhibitor that could transform treatment of autoimmune disease
- FDA granted accelerated approval in IgAN
- Phase 3 final efficacy analysis presented at ASN Kidney Week 2026 opening plenary session, simultaneously published in *The New England Journal of Medicine*
- Submitted sBLA for potential full U.S. approval in mid-2027
- Aligned with EU regulators on plan for full MAA submission in Q1 2027



APRIL, A proliferation-inducing ligand; BAFF, B-cell activating factor, also known as B lymphocyte stimulator (BLyS); IgAN, immunoglobulin A nephropathy; MAA, marketing authorization application; sBLA, supplemental Biologics License Application.

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Vera pipeline



Vera holds worldwide, exclusive rights to develop and commercialize TRUTAKNA, VT-109, and MAU868

FSGS, focal segmental glomerulosclerosis; MCD, minimal change disease; pMN, primary membranous nephropathy.

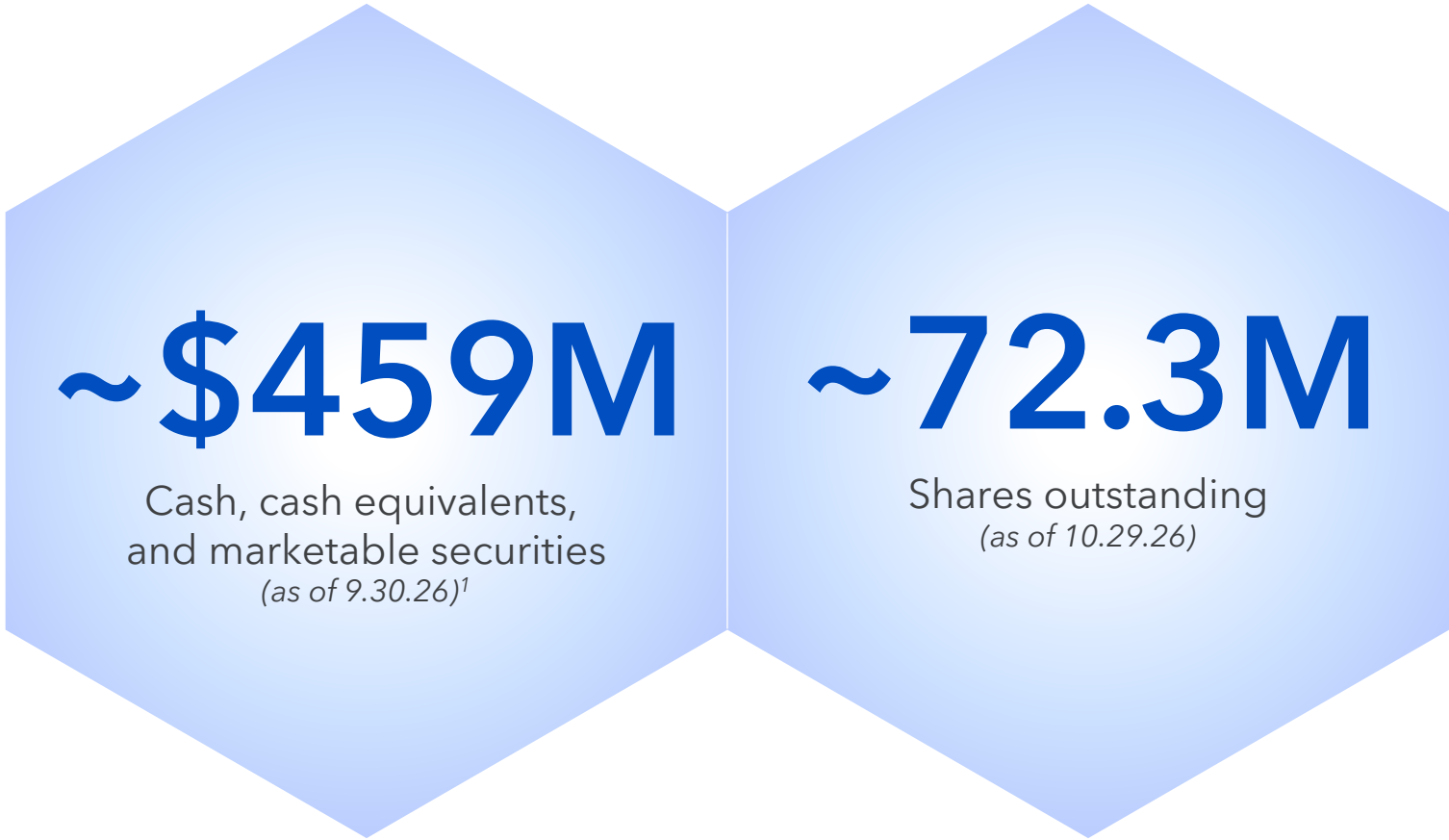
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Management team: Successful clinical and commercial track record



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Strong financial position



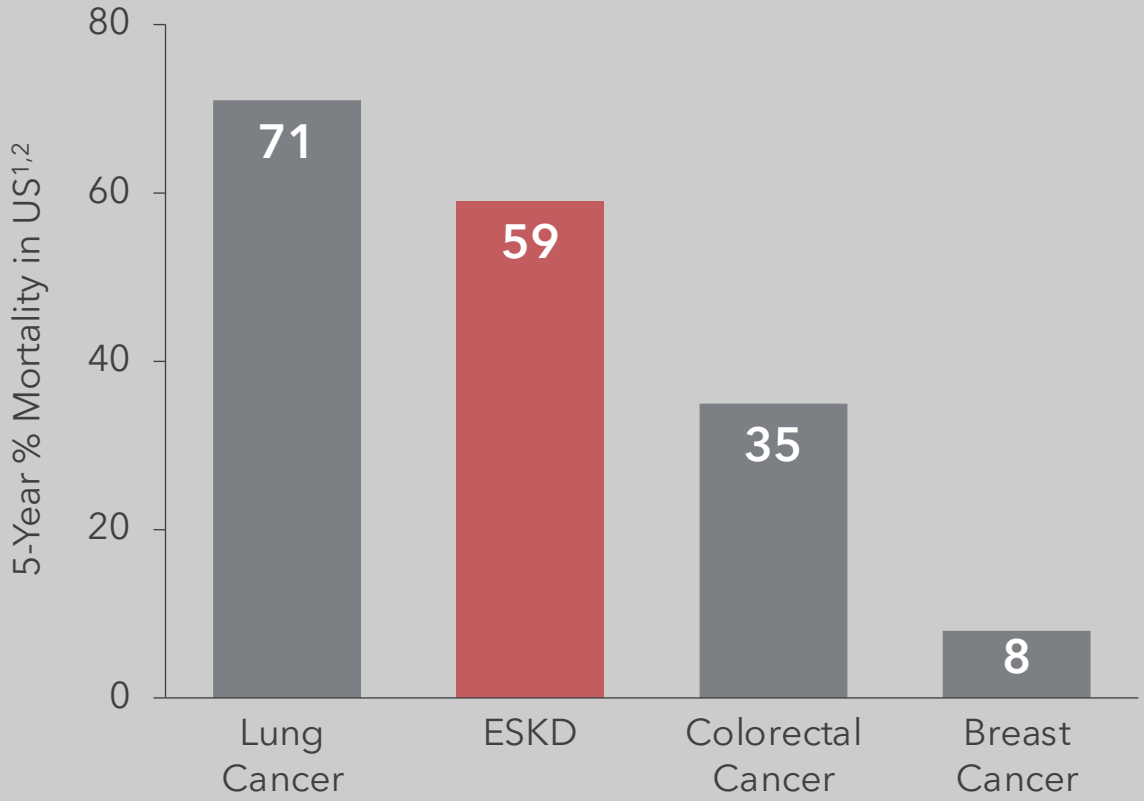
Additional $350M non-dilutive capital available through Oxford Facility, subject to satisfaction of certain conditions

1. Pro forma unaudited estimate includes ~$384M of cash, cash equivalents and marketable securities as of September 30, 2026 and additional $75M drawn under its debt facility in late October, adjusting solely to reflect the October borrowing as if it had occurred on September 30.

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Inhibition of immune complex formation in IgAN may offer the potential to avoid end-stage kidney disease during a patient's lifetime



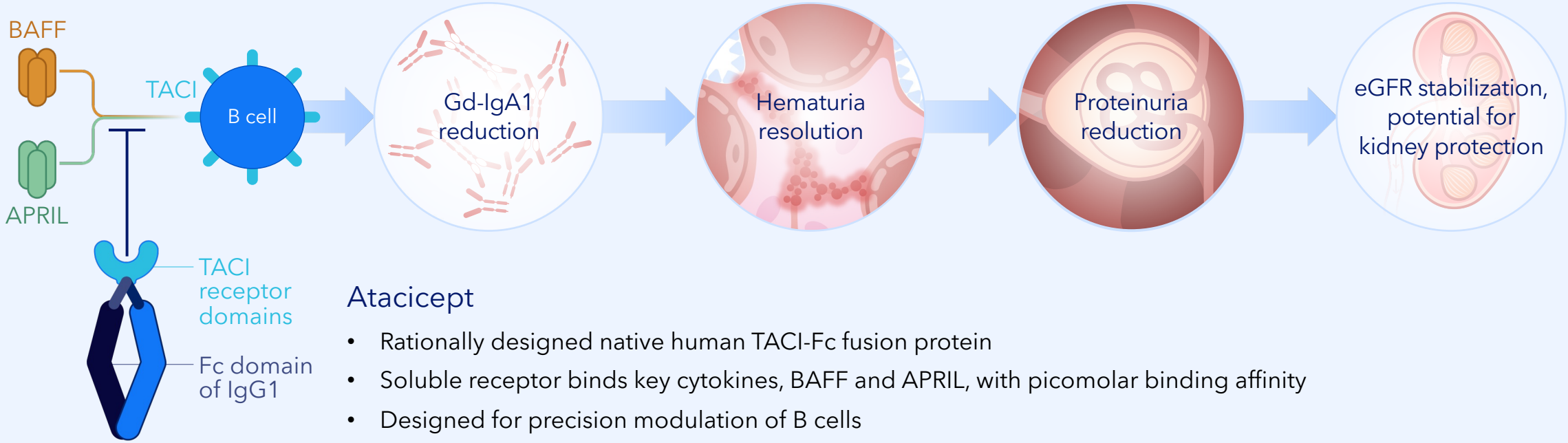


ESKD, end stage kidney disease.
1. US CDC Cancer Statistics based on cancers diagnosed from 2015 to 2021 and follow-up of patients through Dec 31, 2021; 2. Thurlow JS, et al. Am J Nephrol 2021 (data for ESKD resulting from all kidney disease).

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Atacicept: Rationally designed fusion protein conceived in the era of modern biotechnology

IgAN is a disease of B cell origin with kidney pathology



Atacicept

- Rationally designed native human TACI-Fc fusion protein
- Soluble receptor binds key cytokines, BAFF and APRIL, with picomolar binding affinity
- Designed for precision modulation of B cells

Treatment goal: reduce rate of eGFR loss to **<1** mL/min/year (physiological rate)[1]

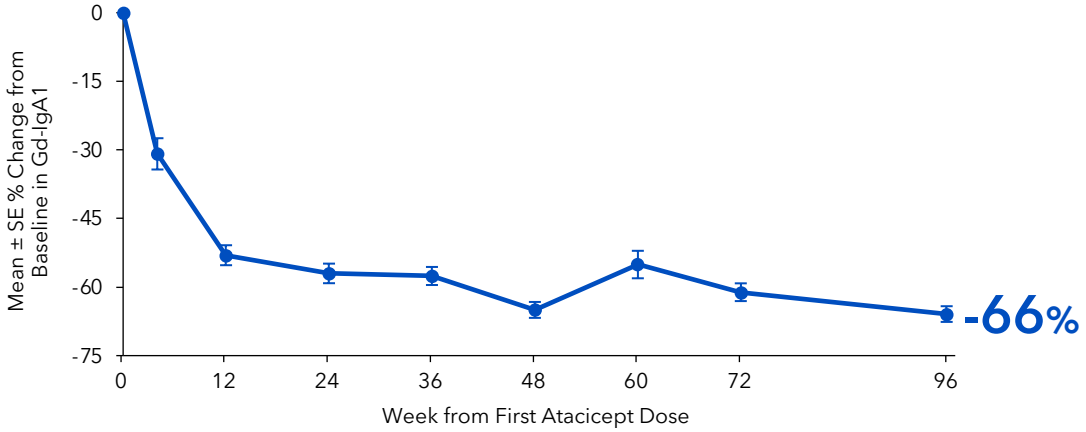
Landmark trials of CKD therapies reported **~16–55%** risk reductions in composite kidney disease outcomes[2]

CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; Fc, fragment crystallizable region; Gd-IgA1, galactose-deficient immunoglobulin A1; TACI, transmembrane activator and calcium-modulator and cyclophilin ligand interactor.
1. KDIGO IgAN and IgAV Work Group. Kidney Int. 2025;108(4S):S1-S71. 2. Barratt J, et al. N Engl J Med 2026 doi/full/10.1056/NEJMoa2610103. These data are from multiple trials and not a head-to-head clinical trial; such data may not be directly comparable due to differences in composite kidney outcome definitions, study protocols, conditions and patient populations.

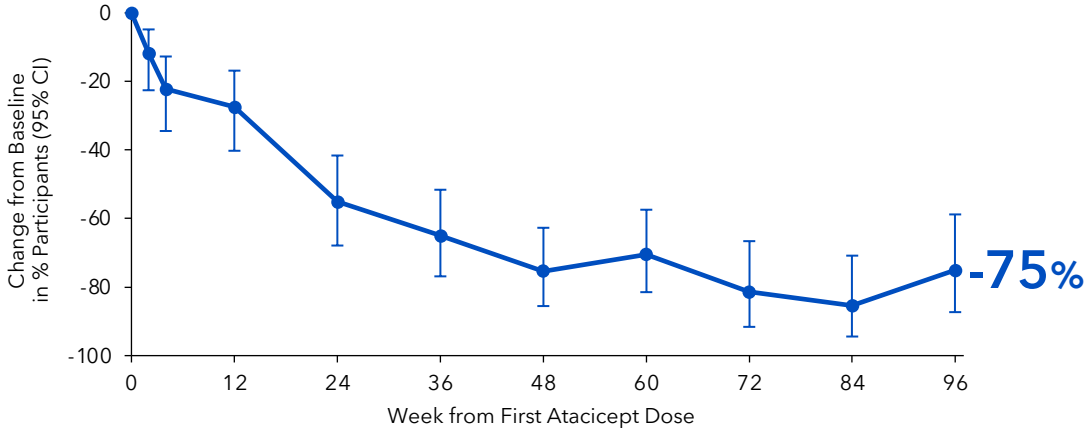
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ORIGIN Phase 2b long-term results appear consistent with disease modification

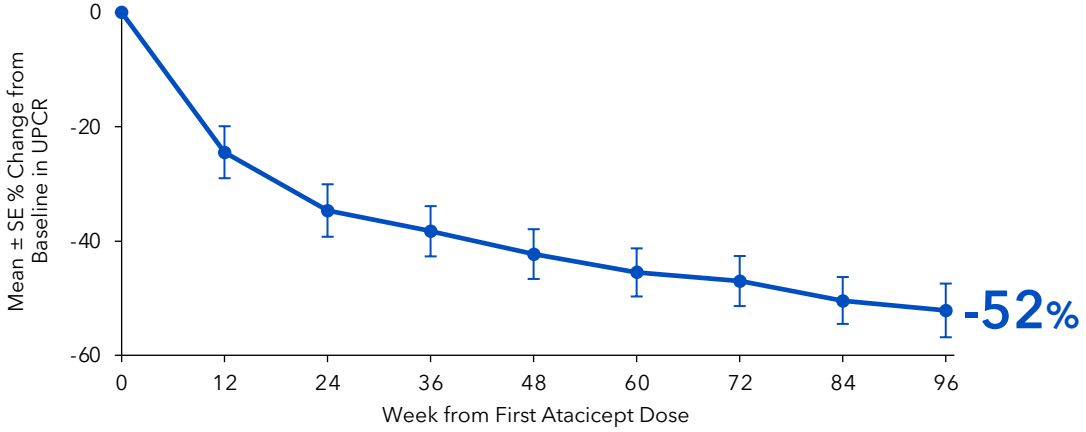
Reduction in Gd-IgA1



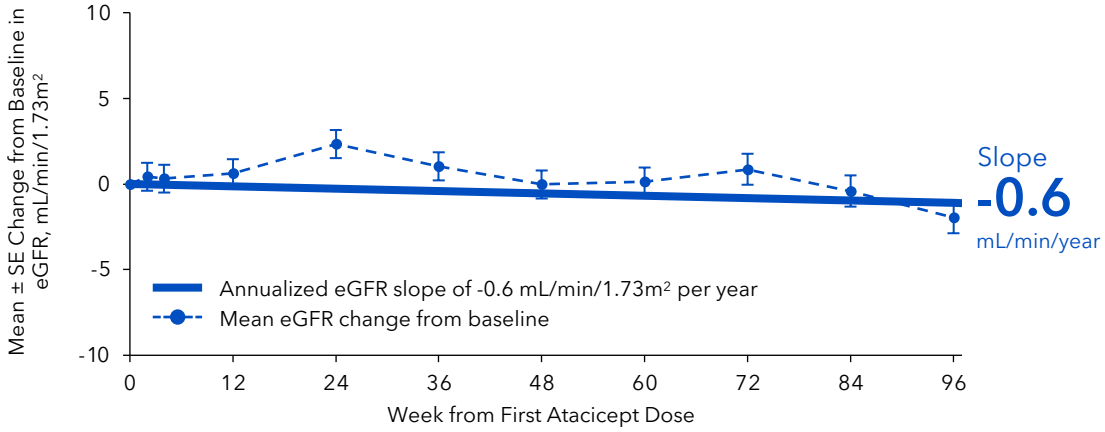
Resolution of hematuria



Reduction in proteinuria



Stabilization of eGFR



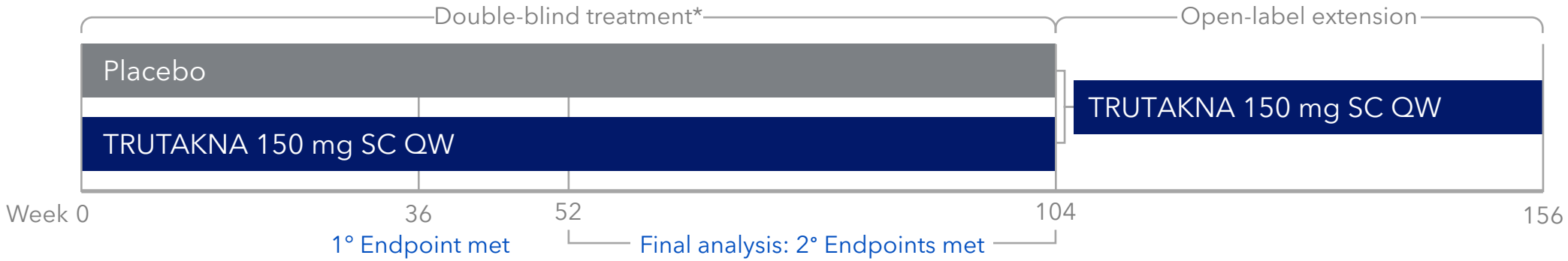
Data includes all participants receiving any active dose at any time point, with baseline (week 0) defined as last available measurement prior to first active dose. Results through 96 weeks were exploratory endpoints and included open-label treatment. CI, confidence interval; UPCR, urine protein creatinine ratio.

Barratt J, et al. J Am Soc Nephrol 2025.

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ORIGIN 3 trial design

Multinational, randomized, placebo-controlled trial of TRUTAKNA, self-administered at home via weekly 1-mL SC injection



Key Inclusion Criteria

- Patients ≥18 years old with biopsy-proven IgAN and high risk of disease progression
- UPCR-24h ≥1.0 g/g or UP ≥1.0 g/24h
- eGFR ≥30 mL/min/1.73m^2
- Stable and optimized RASi for ≥12 weeks, use of SGLT2i allowed
- Blood pressure ≤150/90 mmHg

Key Exclusion Criteria

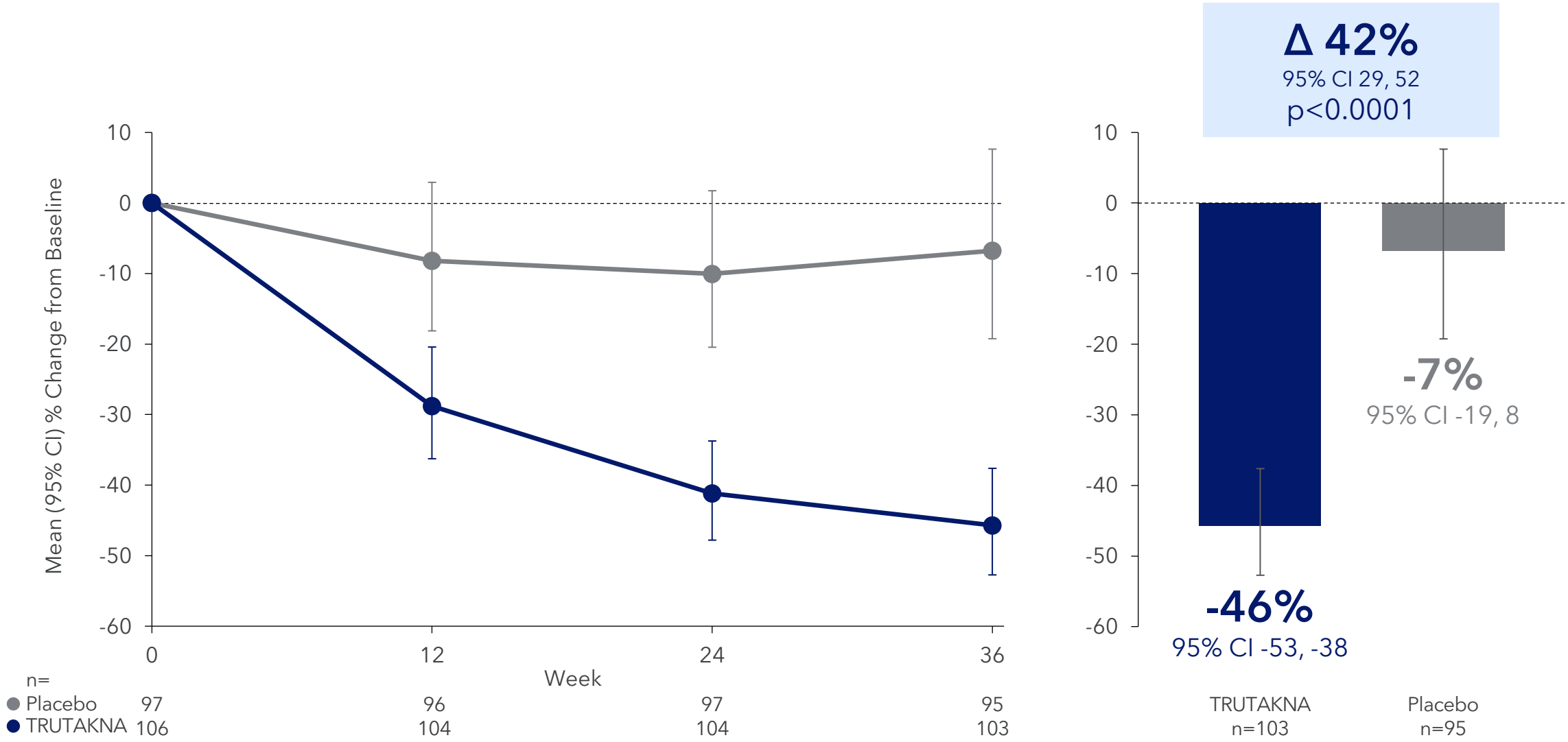
- IgAN secondary to another condition
- Nephrotic syndrome within 6 months of screening
- Evidence of rapidly progressive glomerulonephritis (loss of ≥50% of eGFR) within 3 months of screening

Endpoints

- Primary: UPCR-24h % change from baseline at 36 weeks
- Secondary endpoints tested in a prespecified, fixed-sequence hierarchy:
  - Key: eGFR change from baseline at 52 weeks
  - Annualized eGFR slope through 104 weeks
  - UPCR-24h % change from baseline at 52 weeks
  - Gd-IgA1 % change from baseline at 52 weeks
  - Hematuria resolution at 52 weeks
  - Time to first sustained ≥30% eGFR decline through 104 weeks
  - Time to first composite kidney-disease progression event through 104 weeks
- Safety

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Primary endpoint: UPCR reduction



Interim Analysis Set (IAS) included the first 203 randomized participants who received ≥1 dose of trial drug. Change from baseline in natural-log transformed UPCR at Week 36 was analyzed using a mixed-effects model with repeated measurement (MMRM).

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TRUTAKNA was generally well tolerated in ORIGIN 3 interim analysis

Participants, %	TRUTAKNA n=214	Placebo n=214
Most common adverse events (≥5%)		
Infections	32	28
Upper respiratory tract infection	12	9
Local administration reactions	30	5
Injection site reaction	19	2
Injection site erythema	6	1
Serious adverse events[1]	0.5	5
Adverse events leading to drug discontinuation[2]	1	4
Serious or severe infections	0	1
Opportunistic infections	0	0
Hypersensitivity reactions	4	7
Adverse events leading to death	0	0

- Most adverse events were mild or moderate in severity and resolved without treatment interruption or discontinuation
- Rate of serious adverse events was lower in the atacicept group compared with placebo
- No clinically relevant hypogammaglobulinemia
- No clinically significant effect of anti-drug antibodies on PK, PD, safety or efficacy
- No deaths occurred

Analysis of safety population (all participants randomized and treated) as of interim data cut on 15-May-2025.
1. TRUTAKNA: cholecystitis, determined by site investigator to be unrelated to treatment; Placebo (n=1 each): gastroenteritis, lower respiratory tract infection, pneumonia, pyelonephritis, IgA nephropathy, renal impairment, acute myocardial infarction, transplant rejection, hyponatremia, osteonecrosis, ovarian epithelial cancer, carotid artery aneurysm, hypertension, acute cholecystitis. 1 placebo serious adverse event was deemed related to study drug.
2. Discontinuations in the 2 TRUTAKNA participants were due to eczema and erythema.

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Vera Therapeutics Announces Alignment with U.S. FDA on Earlier ORIGIN Phase 3 Analysis to Support Potential Full Approval for Atacicept in Adults with IgA Nephropathy

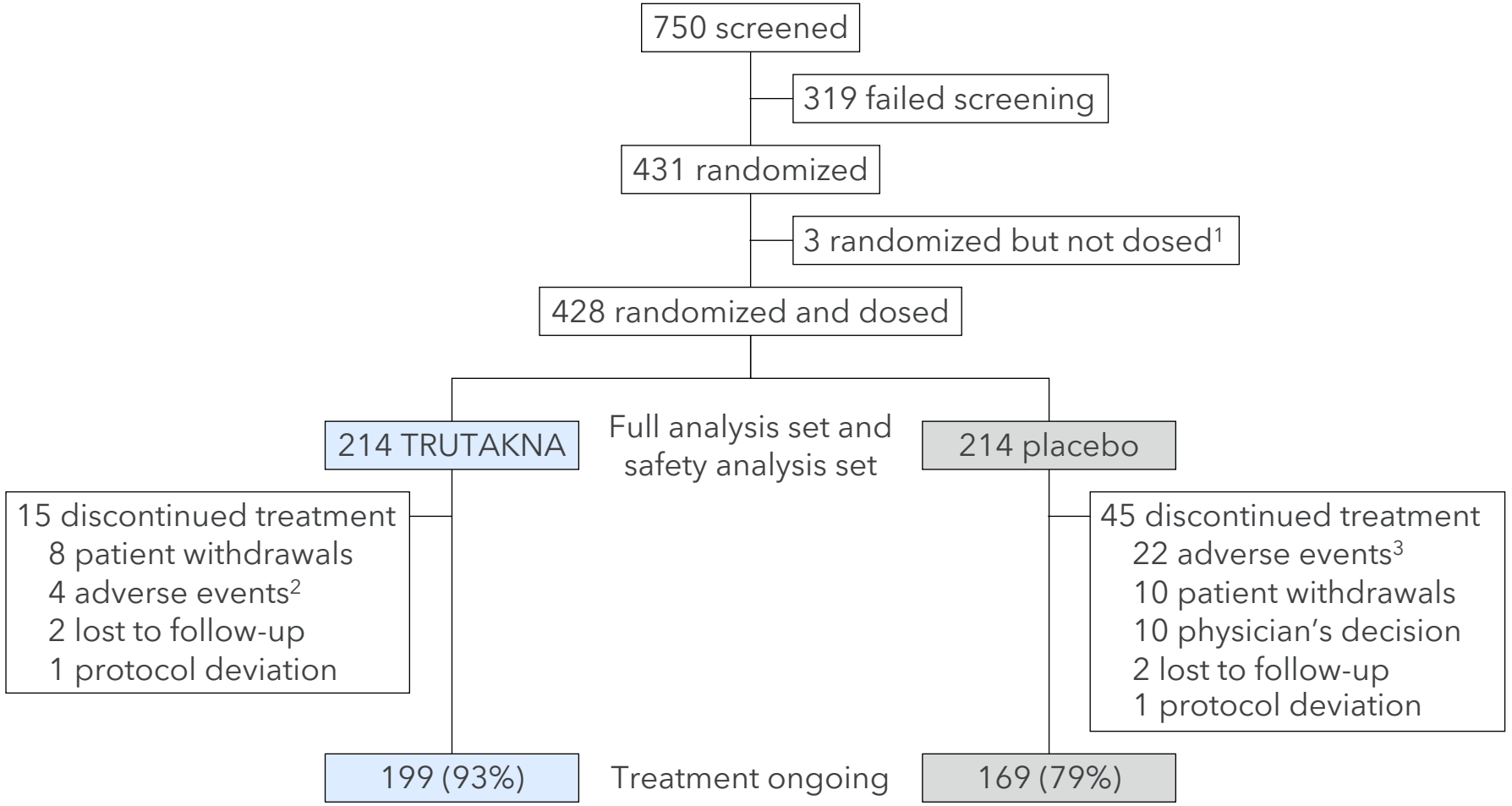
- Revised ORIGIN 3 eGFR analysis now planned for Q3 2026, pulled forward from 2027
- Pending positive eGFR analysis, plans to submit a supplemental Biologics License Application (sBLA) in Q4 2026, with potential full approval in 2027

BRISBANE, Calif., June 02, 2026 (GLOBE NEWSWIRE) -- Vera Therapeutics, Inc. (Nasdaq: VERA), a biotechnology company focused on developing and commercializing transformative treatments for patients with serious immunological diseases, today announced it has aligned with the U.S. Food and Drug Administration (FDA) on a revised, earlier ORIGIN 3 eGFR analysis plan to support full approval for atacicept in adults with IgAN. The eGFR results are now expected in the third quarter of 2026. Pending these results, Vera Therapeutics plans to submit an sBLA for full approval in the fourth quarter of 2026.

Alignment with the FDA on a revised eGFR analysis plan follows a recent workshop hosted by the National Kidney Foundation which included clinicians, researchers, regulators, and patient advocates. In addition, the alignment with the FDA has been supported by the eGFR results from the ORIGIN Phase 2b trial of atacicept in IgAN.

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Final efficacy analysis: Patient disposition



Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.
As of data cut on 10-Jun-2026.

1. 2 were randomized to atacicept and 1 to placebo, but were not dosed due to randomization error, physician decision, or patient withdrawal.
2. The adverse events leading to discontinuation in the atacicept group were splenic abscess, erythema, rash, and injection-site reaction.
3. The adverse events leading to discontinuation in the placebo group were chronic kidney disease (n=3), proteinuria (n=2), renal impairment (n=2), acute kidney injury, end-stage renal disease, IgA nephropathy, renal failure, pneumonia, pyelonephritis, upper respiratory tract infection, skin lesion, high-grade B-cell lymphoma, ovarian epithelial cancer, carotid artery aneurysm, Parkinson's disease, aplastic anemia, parophthalmia, blood creatinine increased, osteonecrosis, and aortic dissection.

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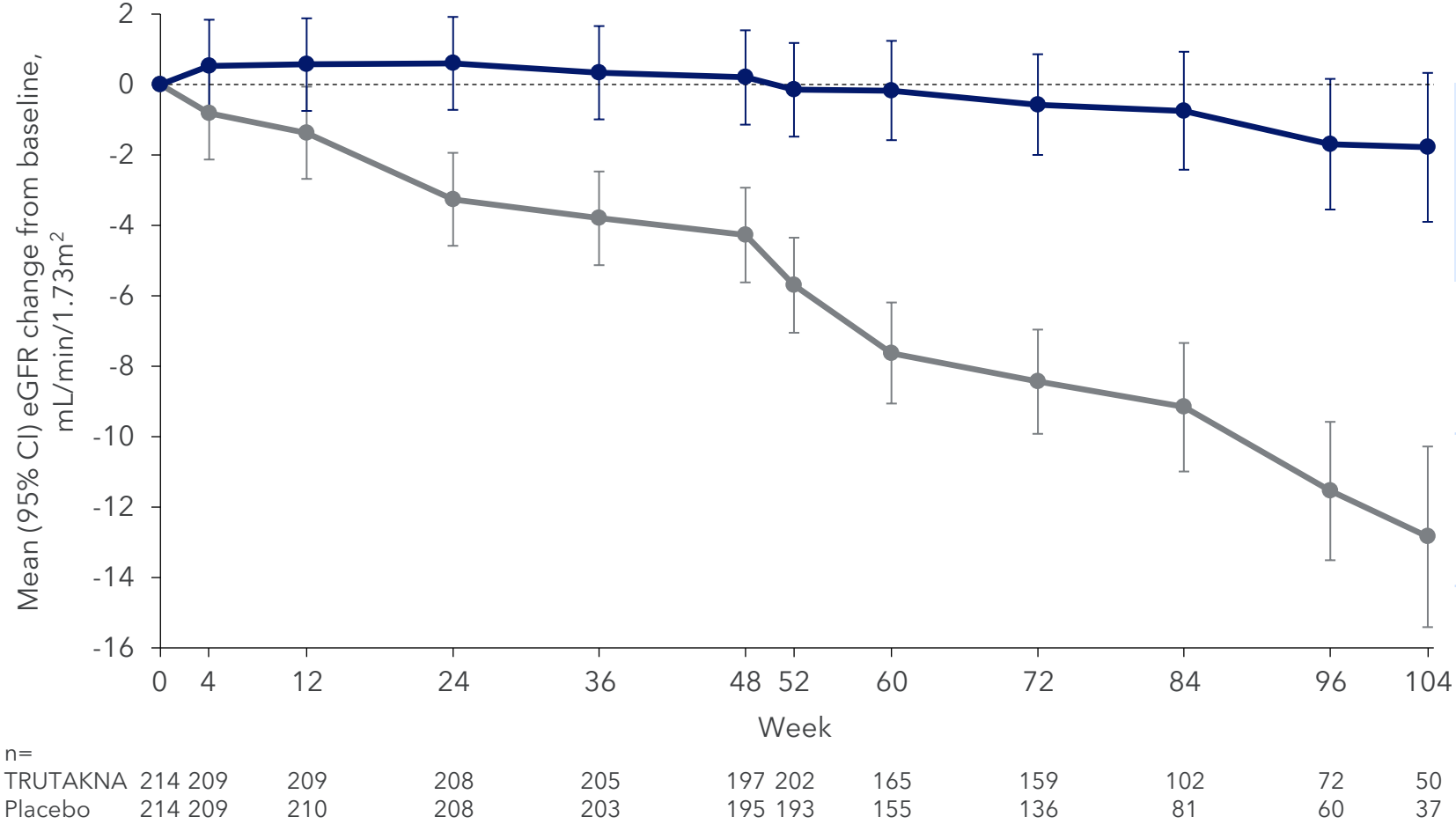
Baseline demographics and characteristics

	TRUTAKNA n=214	Placebo n=214	Total n=428
Age, years, median (range)	41 (18, 74)	41 (18, 70)	41 (18, 74)
Male sex, n (%)	122 (57)	132 (62)	254 (59)
Race, n (%)			
Asian	117 (55)	114 (53)	231 (54)
White	94 (44)	95 (44)	189 (44)
Other	3 (1)	5 (2)	8 (2)
Region, n (%)			
Americas, Europe, Australia	110 (51)	108 (50)	218 (51)
Asia	104 (49)	106 (50)	210 (49)
eGFR, mL/min/1.73m^2, mean ± SD	65 ± 27	63 ± 27	64 ± 27
UPCR by 24h urine, g/g, mean ± SD	1.6 ± 0.9	1.6 ± 1.0	1.6 ± 1.0
Proteinuria, g/day, mean ± SD	2.2 ± 1.1	2.2 ± 1.2	2.2 ± 1.1
Systolic blood pressure, mmHg, mean ± SD	124.1 ± 11.1	124.6 ± 11.0	124.3 ± 11.0
Diastolic blood pressure, mmHg, mean ± SD	79.7 ± 8.3	78.4 ± 8.1	79.0 ± 8.2
Time since biopsy, years, mean ± SD	2.6 ± 2.6	2.3 ± 2.4	2.4 ± 2.5
SGLT2i use, n (%)	134 (63)	129 (60)	263 (61)

Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.

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TRUTAKNA treatment resulted in a *physiologic* rate of eGFR loss vs *pathologic* rate of eGFR loss with placebo



	Mean change from baseline[1] at 52 weeks	Mean change from baseline[1] at 104 weeks	Annualized eGFR slope[2]
Placebo-adjusted difference	**Δ5.6** (3.7, 7.5) p<0.0001	**Δ11.1** (7.7, 14.4) p<0.0001	**Δ5.0** (3.6, 6.5) p<0.0001
TRUTAKNA	**-0.1** (-1.4, 1.2)	**-1.8** (-3.9, 0.3)	**-0.6** (-1.6, 0.4)
Placebo	**-5.7** (-7.0, -4.4)	**-12.8** (-15.4, -10.3)	**-5.6** (-6.6, -4.6)

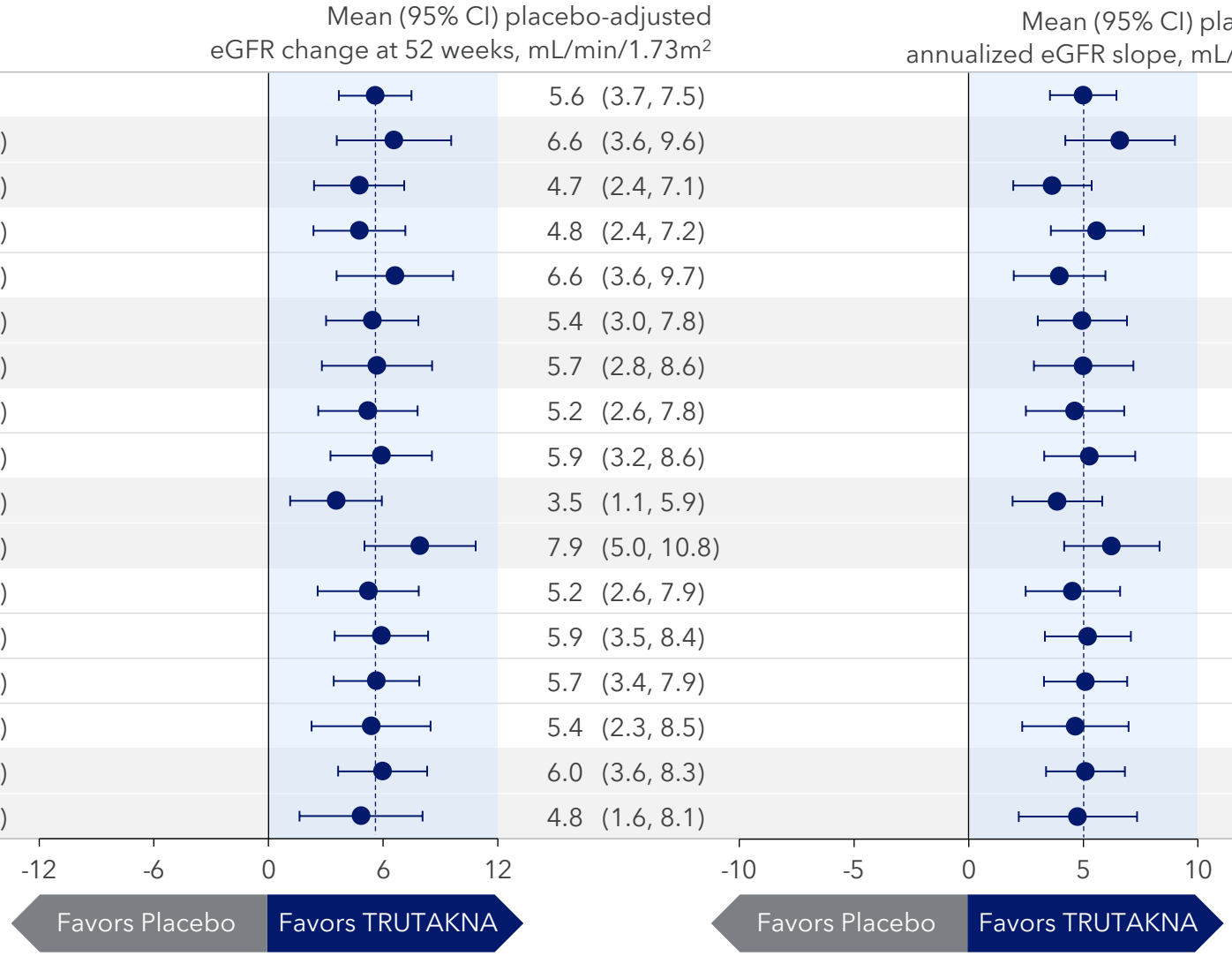
Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.

1. Change from baseline in eGFR was analyzed using a mixed model for repeated measures (MMRM). eGFR change from baseline at 52 weeks was the prespecified key secondary endpoint using an analysis of data through 52 weeks. The eGFR change from baseline at 104 weeks was a prespecified exploratory endpoint using an analysis of data through 104 weeks, and the p-value is nominal.
2. Annualized eGFR slope through 104 weeks was a prespecified secondary endpoint and analyzed using a mixed model with random intercept and random slope (MMRIRS).

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TRUTAKNA eGFR benefit observed across all prespecified subgroups

		n (%)	Mean (95% CI) placebo-adjusted eGFR change at 52 weeks, mL/min/1.73m^2	Mean (95% CI) placebo-adjusted annualized eGFR slope, mL/min/1.73m^2/yr
Overall		428	5.6 (3.7, 7.5)	5.0 (3.6, 6.5)
Age, years	<40	189 (44)	6.6 (3.6, 9.6)	6.6 (4.2, 9.0)
	≥40	239 (56)	4.7 (2.4, 7.1)	3.7 (2.0, 5.4)
Sex	Male	254 (59)	4.8 (2.4, 7.2)	5.6 (3.6, 7.6)
	Female	174 (41)	6.6 (3.6, 9.7)	4.0 (2.0, 6.0)
Region	Americas, Europe, Australia	218 (51)	5.4 (3.0, 7.8)	5.0 (3.0, 6.9)
	Asia	210 (49)	5.7 (2.8, 8.6)	5.0 (2.9, 7.2)
Race[1]	White	189 (44)	5.2 (2.6, 7.8)	4.7 (2.5, 6.8)
	Non-white	238 (56)	5.9 (3.2, 8.6)	5.3 (3.3, 7.3)
BL UPCR, g/g	<1.5	233 (54)	3.5 (1.1, 5.9)	3.9 (1.9, 5.8)
	≥1.5	195 (46)	7.9 (5.0, 10.8)	6.3 (4.2, 8.3)
BL eGFR, mL/min/1.73m^2	<45	128 (30)	5.2 (2.6, 7.9)	4.6 (2.5, 6.6)
	≥45	300 (70)	5.9 (3.5, 8.4)	5.2 (3.3, 7.1)
	<60	227 (53)	5.7 (3.4, 7.9)	5.1 (3.3, 6.9)
	≥60	201 (47)	5.4 (2.3, 8.5)	4.7 (2.3, 7.0)
SGLT2i use at BL	Yes	263 (61)	6.0 (3.6, 8.3)	5.1 (3.4, 6.8)
	No	165 (39)	4.8 (1.6, 8.1)	4.8 (2.2, 7.4)



Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.
Change from baseline in eGFR was analyzed using an MMRM. Annualized eGFR slope was analyzed using an MMRIRS. The confidence intervals for the subgroup analyses were not adjusted for multiplicity and should not be used in place of hypothesis testing; thus, no conclusions regarding individual subgroups can be made.
1. Race was not reported for one participant.

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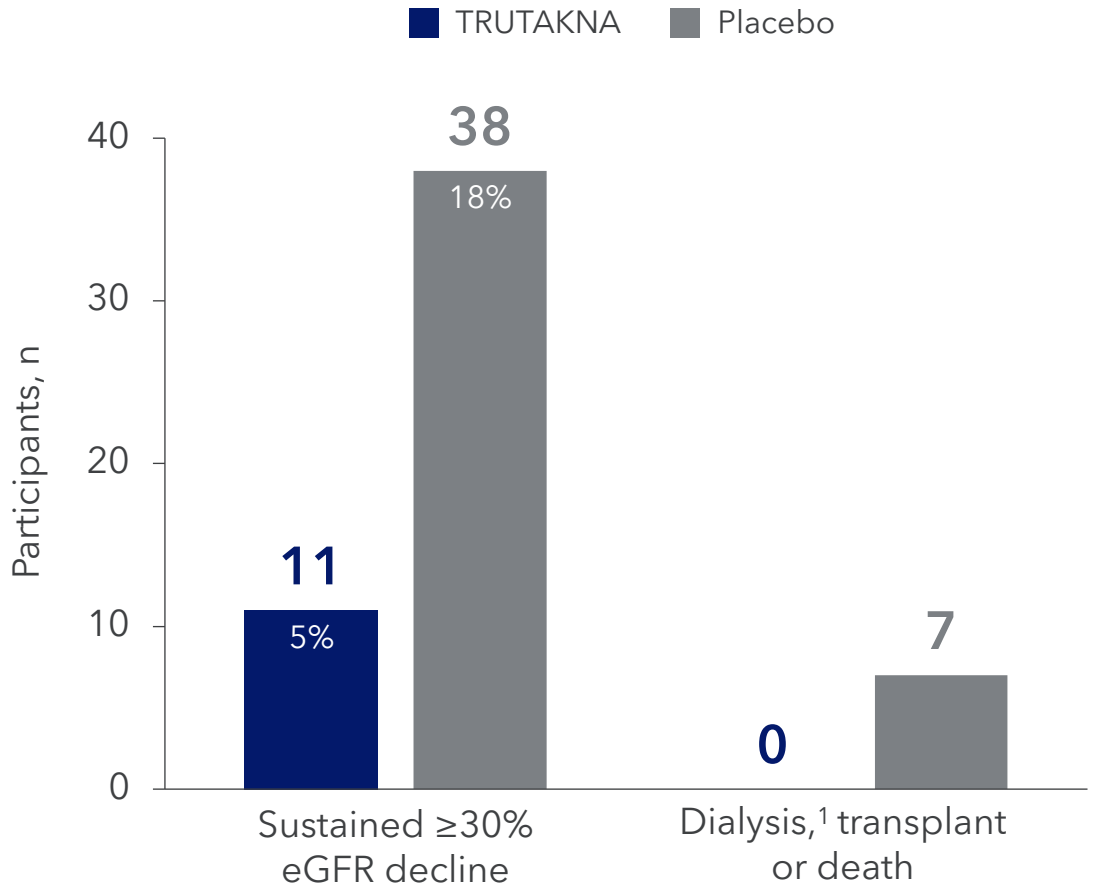
TRUTAKNA achieved **76%** risk reduction in the composite kidney disease progression endpoint

Composite kidney disease progression through 104 weeks

Hazard ratio	Risk reduction
0.24	**76%**
(95% CI 0.12, 0.48)	
p<0.0001	

A composite kidney disease progression event was defined as ≥1 of the following:

- Death
- Kidney transplant
- Chronic dialysis ≥30 days
- eGFR <15 mL/min/1.73m^2 sustained for ≥30 days
- ≥30% eGFR decline sustained for ≥30 days

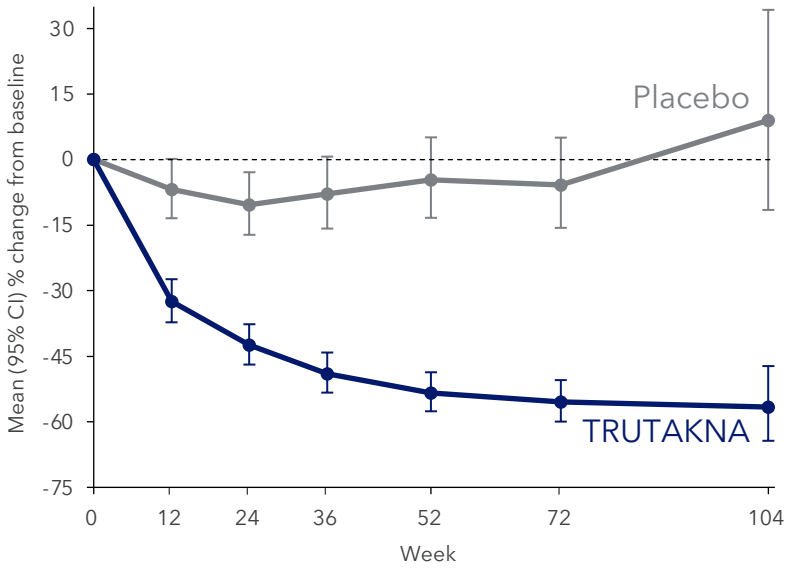


Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.
Hazard ratio, 95% CI, and the likelihood ratio test p-value were calculated based on Cox proportional hazard model.
All patients who had composite events first experienced a sustained ≥30% decline in eGFR.
1. Does not include one placebo participant who received transient dialysis for acute kidney injury.

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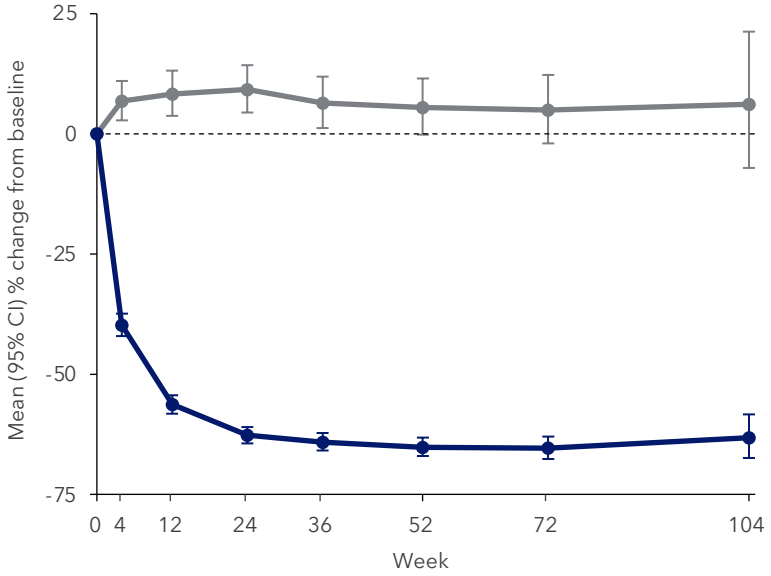
TRUTAKNA reduced UPCR, Gd-IgA1, and hematuria through 104 weeks

UPCR reduction[1]



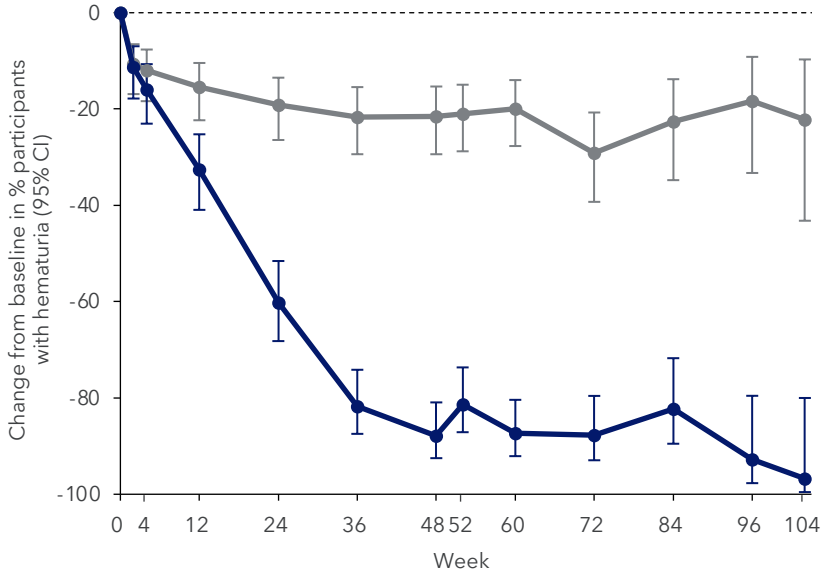
	52 weeks	104 weeks
Placebo	-5%	+9%
TRUTAKNA	-53%	-57%
Placebo-adjusted reduction	Δ51% p<0.0001	Δ60%

Gd-IgA1 reduction[2]



	52 weeks	104 weeks
Placebo	+3%	+6%
TRUTAKNA	-66%	-63%
Placebo-adjusted reduction	Δ67% p<0.0001	Δ65%

Hematuria resolution[3]



	52 weeks	104 weeks
Placebo	-21%	-22%
TRUTAKNA	-81%	-97%
	p<0.0001	

Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.
The 52-week estimates were prespecified secondary endpoints using analyses of data through 52 weeks, and 104-week estimates were prespecified exploratory endpoints using analyses of data through 104 weeks.
1. UPCR % reduction was obtained from the geometric mean ratios, where the natural log-transformed ratio to baseline in UPCR (based on 24-hour urine collection) was analyzed using an MMRM model. 2. Gd-IgA1 % reduction was obtained from the geometric mean ratios, where the natural log-transformed ratio to baseline in Gd-IgA1 was analyzed using an MMRM model. 3. The logistic regression model with repeated measurement was used to analyze the proportion of participants achieving hematuria resolution at each postbaseline visit.

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TRUTAKNA was well tolerated with a favorable safety profile, and generally comparable to placebo

Participants, n (%)	TRUTAKNA n=214	Placebo n=214
Adverse events	160 (75)	167 (78)
Serious adverse events[1]	15 (7)	33 (15)
Adverse events leading to drug discontinuation	4 (2)	22 (10)
Adverse events of infections and infestations	110 (51)	112 (52)
Serious or severe infections and infestations	6 (3)	8 (4)
Opportunistic infections	0	0
Study drug related adverse events[2]	75 (35)	38 (18)
Adverse events associated with injection site reactions[3]	54 (25)	16 (7)
Hypersensitivity reactions	21 (10)	23 (11)
Adverse events leading to death	0	1 (0.5)

- Most adverse events were mild or moderate in severity and resolved without treatment interruption or discontinuation
- Injection site reactions were mild or moderate
- Fewer serious adverse events were reported in the TRUTAKNA group compared with placebo
- No clinically relevant hypogammaglobulinemia reported
- No opportunistic infections reported
- No deaths occurred in the TRUTAKNA group
- No clinically meaningful effect of anti-drug antibodies on PK, PD, safety or efficacy

Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.
Analysis of safety population (all participants randomized and treated) as of data cut on 10-Jun-2026.

1. In the placebo patients with serious adverse events, 7 had events characterized as kidney and urinary disorders. Additional laboratory-related events included decreased eGFR in 2 patients and increased blood creatinine in 1. There were no kidney/urinary or kidney-related laboratory serious adverse events in the atacicept group.
2. Most study drug related adverse events were mild to moderate injection site reactions that did not lead to discontinuation.
3. Injection site reactions among TRUTAKNA recipients were largely characterized by injection site erythema, hematoma, and pruritis.

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ORIGIN 3 final efficacy analysis summary

- ORIGIN 3 demonstrates that TRUTAKNA has the potential for comprehensive disease modification in IgAN
- TRUTAKNA treatment resulted in a physiologic rate of eGFR loss compared to a pathologic rate of eGFR loss with placebo
- ORIGIN 3 met all prespecified endpoints, including placebo-adjusted eGFR and composite kidney disease progression benefits
- TRUTAKNA was well tolerated with a favorable safety profile, and generally comparable to placebo
- Results published in *The New England Journal of Medicine*[1]
- Study participants are transitioning to open-label atacicept for the remainder of the trial
- Submitted sBLA to U.S. FDA to support potential full approval in 2027; aligned with EU regulators on plan for full MAA submission in Q1 2027

These results have not been reviewed by FDA and are not part of the approved Prescribing Information.

1. Barratt J, et al. N Engl J Med 2026. doi/full/10.1056/NEJMoa2610103.

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PIONEER is evaluating atacicept in antibody mediated glomerular diseases

IgAN

Adults with biopsy-proven
- IgAN
- IgAVN
- recurrent IgAN post-transplant

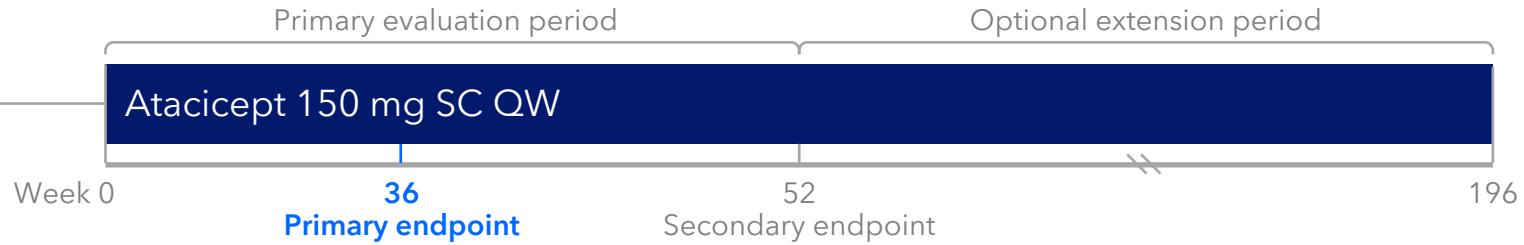
eGFR ≥20 mL/min/1.73m²
24h-UPCR-24h ≥0.5 g/g

Children* (age ≥2 to <18 years) with biopsy-proven IgAN / IgAVN

pMN

Adults with biopsy-proven pMN
Anti-PLA2R ≥25 RU/mL
eGFR ≥30 mL/min/1.73m²
24h-UPCR ≥1.5 g/g

MCD/FSGS



Inclusion criteria

- Stable RASi required; stable SGLT2i, ERA, MRA allowed
- Blood pressure ≤160/≤90 mmHg
- Weight ≥40 kg at screening*

Exclusion criteria

- ≥50% loss of eGFR within 3 months of screening
- Mixed or multiple glomerulopathies at biopsy
- Severe kidney impairment (eGFR <15 mL/min/1.73m²), or receiving dialysis or kidney replacement therapy
- Secondary causes of glomerular disease

Primary endpoints

- Change from baseline in UPCR at Week 36
- Safety and tolerability

Secondary endpoints

- Change from baseline in eGFR at Weeks 36 and 52
- Change from baseline in disease-specific antibodies

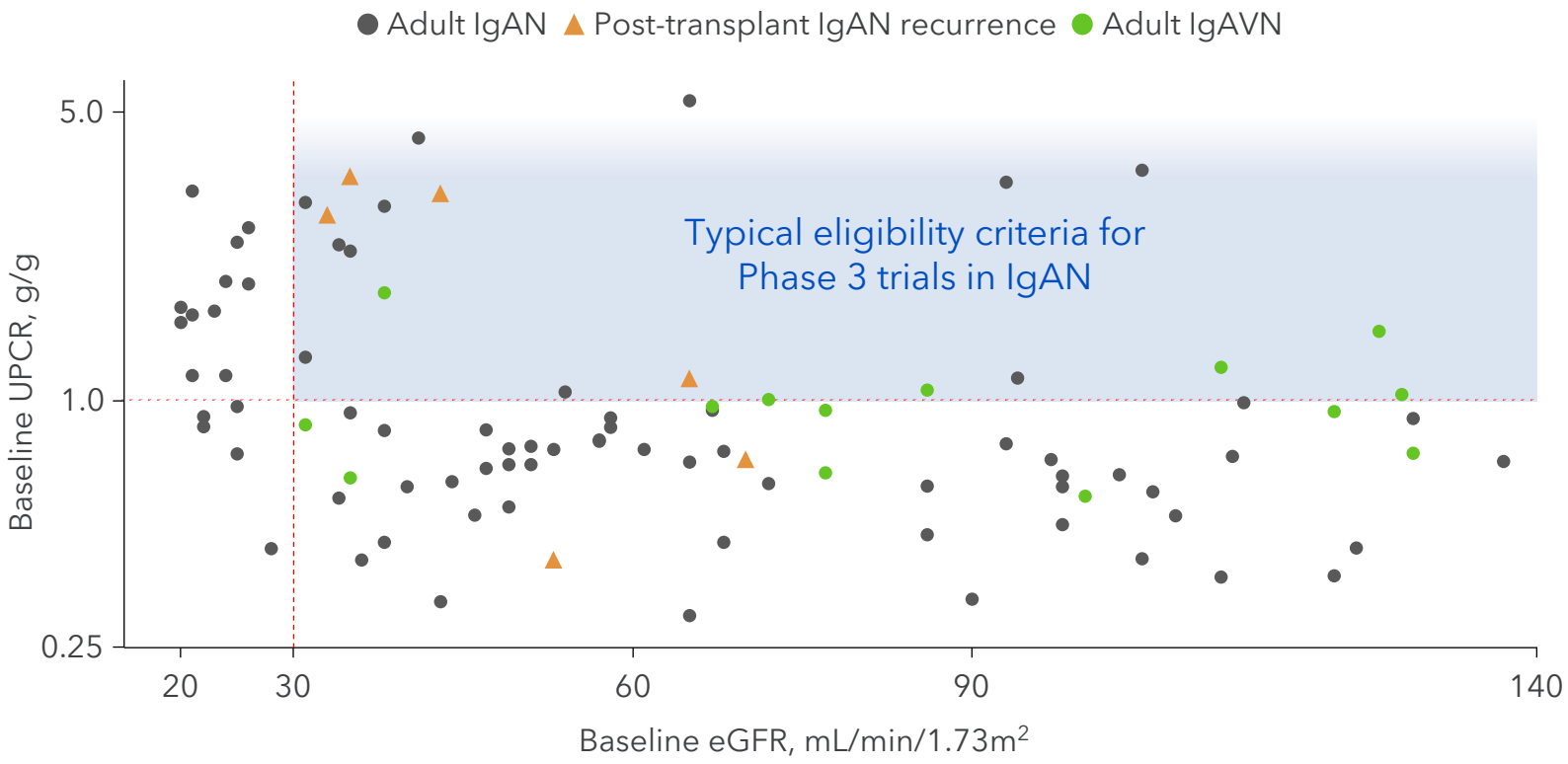
*Currently enrolling children age ≥10 years and weighing ≥40 kg. For children ages ≥2 to <10 years and weighing <40 kg, enrollment will open pending interim analysis.
ERA, endothelin receptor antagonist; FSGS, focal segmental glomerulosclerosis; IgAVN, immunoglobulin A vasculitis with nephritis; MCD, minimal change disease; MRA, mineralocorticoid receptor antagonist; PLA2R, phospholipase A2 receptor; pMN, primary membranous nephropathy; RU, relative unit.

Atacicept is not approved by any health regulatory agency for use outside of adults with IgAN and thus no conclusions of safety or efficacy can be made.

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PIONEER enrolled adults with IgAN with baseline characteristics distinct from ORIGIN 3 and other Phase 3 trials

Baseline characteristic	IgAN, n=101
Age, years, median (range)	41 (18-88)
Male sex, n (%)	62 (61)
Asian race, n (%)	23 (23)
White race, n (%)	67 (66)
Hispanic/Latino ethnicity, n (%)	10 (9.9)
eGFR, mL/min/1.73m^2, mean ± SD	64 ± 32.8
<30 mL/min/1.73m^2, n (%)	16 (16)
UPCR-24h, g/g, mean ± SD	1.2 ± 1.04
<1.0 g/g, n (%)	65 (64)
≥3.5 g/g, n (%)	6 (6)
SGLT2i use at baseline, n (%)	80 (79)
ERA use at baseline, n (%)	8 (8)

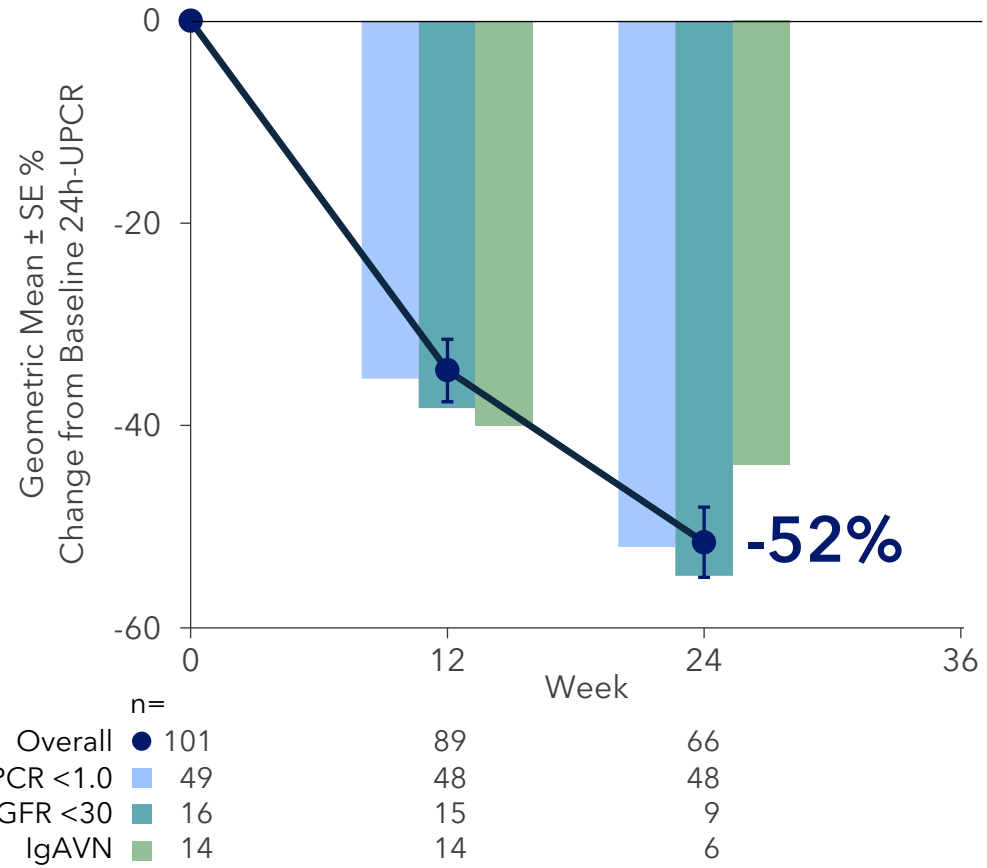


PIONEER includes participants with baseline eGFR and UPCR levels beyond the ranges evaluated in ORIGIN 3

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Proteinuria reduction in PIONEER IgAN cohorts

Baseline characteristic	UPCR <1.0 g/g	eGFR <30	IgAVN
N, (% overall)	49 (49)	16 (16)	14 (14)
Age, years, median (range)	41 (18–58)	50 (24–88)	31 (19–59)
Male sex, n (%)	33 (67)	11 (69)	4 (29)
Asian race, n (%)	11 (22)	2 (13)	3 (21)
White race, n (%)	35 (71)	13 (81)	8 (57)
Hispanic/Latino ethnicity, n (%)	5 (10)	1 (6)	1 (7)
eGFR, mL/min/1.73m^2, mean ± SD	73.2 ± 30.0	23.5 ± 2.9	85.7 ± 35.1
UPCR-24h, g/g, mean ± SD	0.7 ± 0.2	1.6 ± 0.7	1.0 ± 0.3
SGLT2i use at baseline, n (%)	40 (82)	15 (94)	10 (71)



Similar to proteinuria reductions demonstrated in ORIGIN 3: -46% change from baseline in atacicept arm at Week 36

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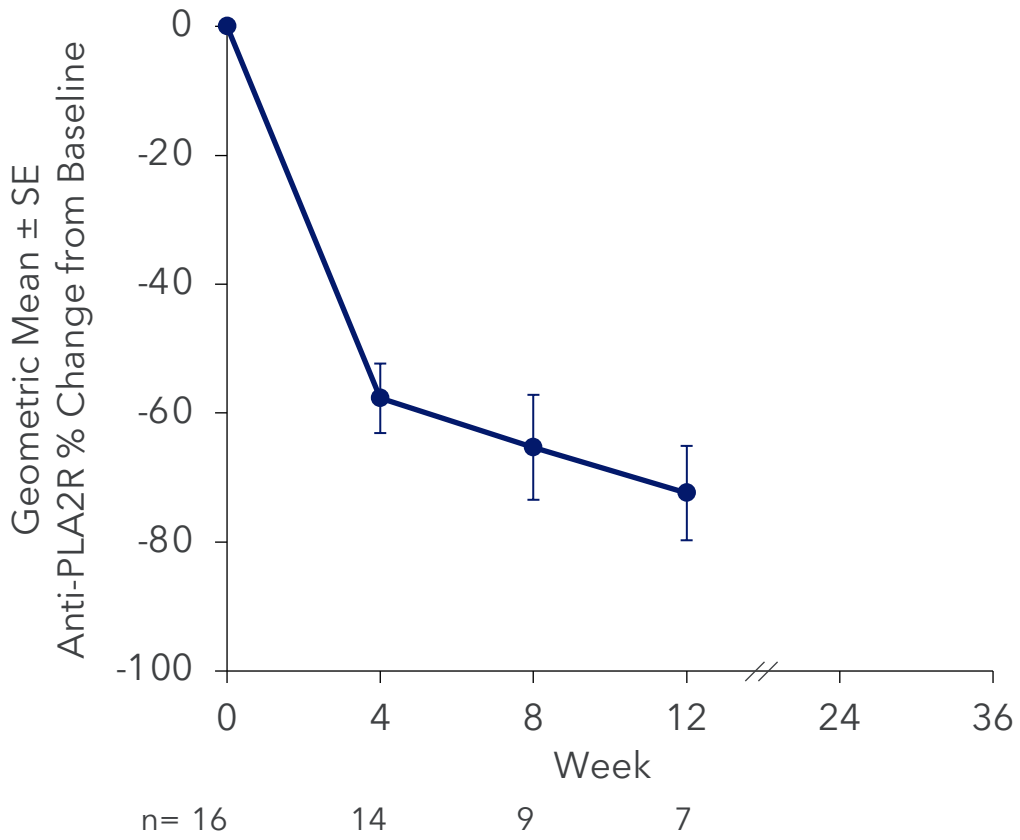
PIONEER pMN baseline characteristics

	Adults with pMN n=16
Age at screening, years, median (range)	57 (37–70)
Male sex, n (%)	12 (75)
Asian race, n (%)	0
White race, n (%)	13 (81)
Hispanic/Latino ethnicity, n (%)	2 (13)
Anti-PLA2R antibody, RU/mL, median (IQR)	83 (55–208)
eGFR, mL/min/1.73m^2, mean ± SD	72 ± 27
24h UPCR, g/g, mean ± SD	4.5 ± 2.7
≥3.5 g/g, n (%)	11 (69)
Serum albumin, g/dL, mean ± SD	3.2 ± 0.5
SGLT2i use at baseline, n (%)	9 (56)

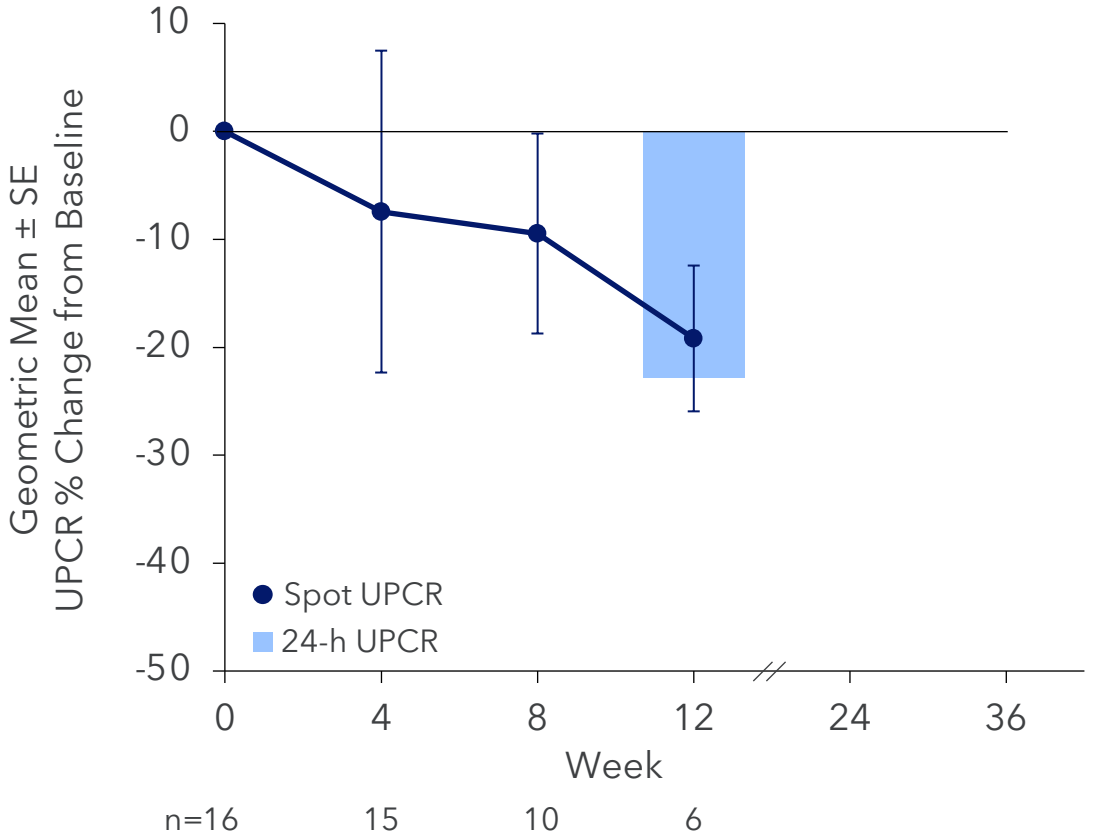
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Reduction in anti-PLA2R antibody and proteinuria are detected as early as 4 weeks

Anti-PLA2R antibody reduction



UPCR reduction



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Safety profile consistent with the overall atacicept clinical development program

Participants, n (%)	IgAN n=101	pMN n=16
Adverse events	82 (81)	12 (75)
Serious adverse events	4 (4)	2 (12)
Adverse events leading to drug discontinuation	0	0
Adverse events of infections and infestations	28 (28)	4 (25)
Serious or severe infections and infestations	2(2)	0
Opportunistic infections	0	0
Study drug related adverse events	39 (39)	7 (44)
Adverse events associated with injection site reactions[2]	22 (22)	2 (13)
Hypersensitivity reactions	3 (3)	1 (6)
Adverse events leading to death	0	0

- Most adverse events were mild or moderate in severity and resolved without treatment interruption or discontinuation
- No opportunistic infections
- Safety profile to date is consistent with the atacicept clinical development program

Analysis of safety population (all participants treated) as of data cut on 21-SEP-2026, All safety events presented are treatment-emergent.
1. Majority of study drug related adverse events were mild to moderate in severity and did not result in study drug interruption or discontinuation.
2. Injection site reactions were largely mild in severity and characterized by injection site erythema and pruritis.

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PIONEER interim results summary

IgAN

- PIONEER IgAN baseline characteristics distinct from ORIGIN 3
- Preliminary results demonstrate similar reduction in UPCR at 24 weeks as ORIGIN 3
- UPCR reductions generally consistent across cohorts with:
  - Baseline eGFR <30 mL/min/1.73m^2
  - Baseline UPCR <1.0 g/g
  - IgAVN

pMN

- Reductions in anti-PLA2R antibody levels were detectable as early as 4 weeks
- UPCR reduction was observed with spot and 24h urine collections in the first 12 weeks of atacicept treatment

The interim results support the continued investigation of atacicept in antibody-mediated glomerular diseases

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Estimated IgAN epidemiology

US IgAN Prevalence: ~0.04% of US Population[1]

~160K

+ ~40K
Low Risk
(~24% of patients)[2]

+ ~30K
Moderate Risk
(~20% of patients)[2]

~90K
High Risk
(~56% of patients)[2]

Phase 3 population



BAFF & APRIL inhibition

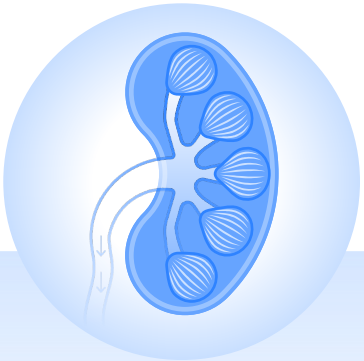
pioneer
Phase 2 study ongoing

Patient counts rounded to nearest 1,000.
1. Clearview Healthcare Partners Analysis 2021 with Vera internal estimates; 2. Pitcher D, et al. Clin J Am Soc Nephrol 2023. Low risk assumed to be 0–0.44 g/g UPCR, moderate risk assumed to be 0.44–0.88 g/g, high risk assumed to be >0.88 g/g; percentage of patients per proteinuria category in study population 1 applied to estimated US IgAN prevalence.

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IgAN market and TRUTAKNA have many hallmarks of an attractive commercial opportunity

IgAN Market



- Uptake of B-cell modulators have exceeded expectations; nephrology community expected to rapidly adopt disease-modifying therapies
- Large market with unmet need
- Growing IgAN population[1]
- Favorable payer mix[2]

TRUTAKNA

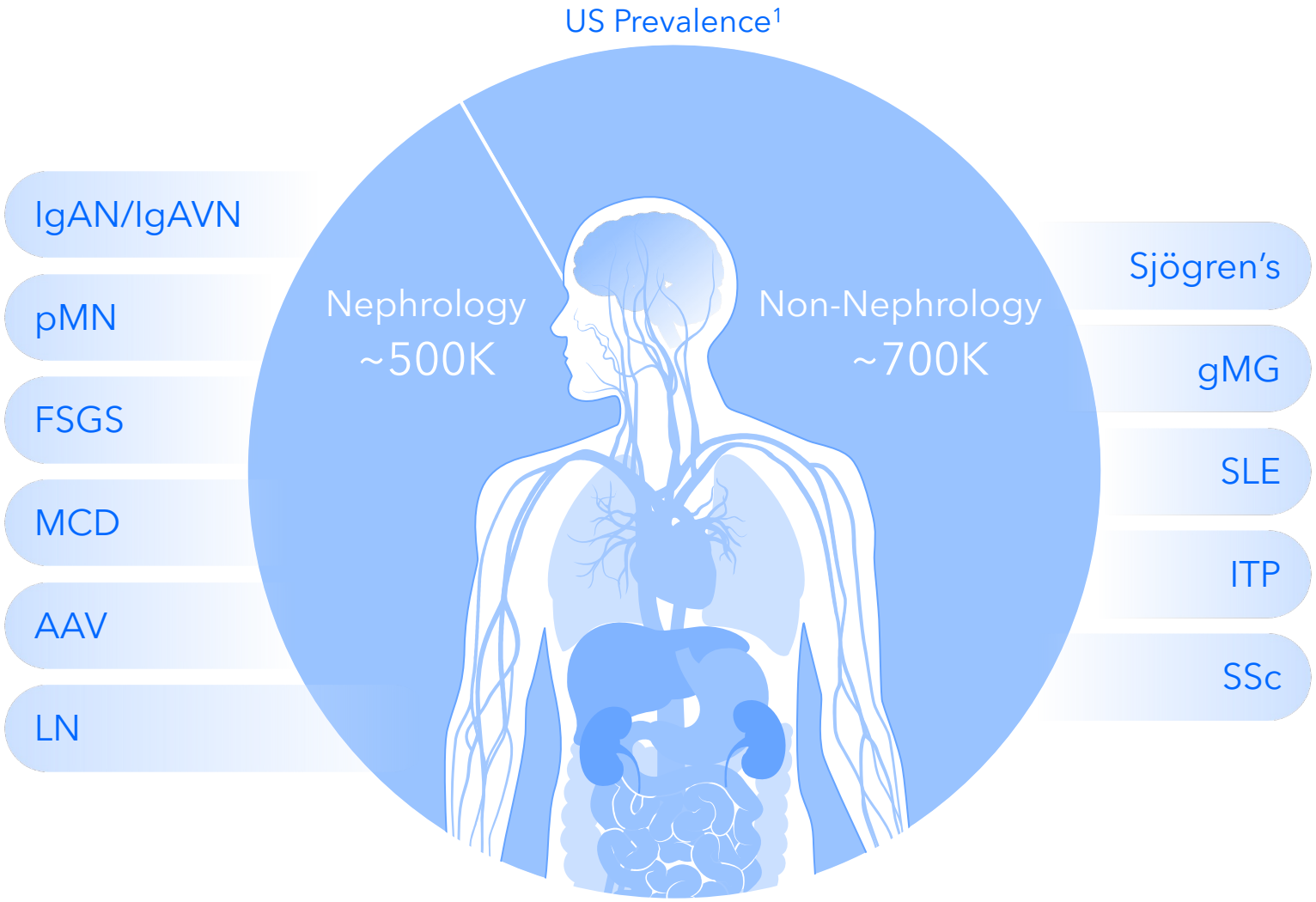


- Strong efficacy and safety profile
- Comprehensive data package considered compelling by nephrology community[3]
- Experienced commercial team actively promoting in the field
- Exciting lifecycle opportunities

1. ClearView Healthcare Partners Analysis; 2. Bluepath Solutions research conducted in Q4 2024; 3. Advisory boards and internal and external market insight gathering (n~115 nephrologists).

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Potential $10B+ U.S. market opportunity for TRUTAKNA in IgAN and pipeline autoimmune diseases[1]



1. Vera Therapeutics corporate estimates for peak year market opportunity and prevalence based on ClearView Healthcare Partners Analysis 2025.
AAV, anti-neutrophil cytoplasmic antibody-associated vasculitis; FSGS, focal segmental glomerulosclerosis; gMG, generalized myasthenia gravis; IgAVN, IgA vasculitis nephritis; ITP, immune thrombocytopenia; LCM, lifecycle management; LN, lupus nephritis; MCD, minimal change disease; pMN, primary membranous nephropathy; SLE, systemic lupus erythematosus; SSc, systemic sclerosis.

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TRUTAKNA projected catalysts

		Catalyst	2026	2027	2028
origin3	IgAN	US launch	✓		
		Phase 3 final efficacy analysis	✓		
		Projected full US approval[1]		Mid	
		Planned MAA submission		Q1	
		Planned J-NDA submission		2H	
origin extend	IgAN	Clinical results	✓	●	●
pioneer	IgAN, pMN, FSGS, MCD	Clinical results	✓	●	●

Vera holds worldwide, exclusive rights to develop and commercialize TRUTAKNA

1. Subject to U.S. FDA review of full clinical dataset and approval.
J-NDA, Japanese new drugs application.

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