



ORIGIN Phase 3 Update

November 6, 2025

Forward-looking statements

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Certain data in this presentation are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities or differences.

This presentation discusses product candidates that are under clinical study and which have not yet been 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.

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- San Francisco biotechnology company founded in 2016; led by world-class development and commercialization team
- Atacicept potential **first-in-class mechanism** (dual BAFF/APRIL inhibitor) to transform treatment of autoimmune disease
- IPO on Nasdaq May 2021; >\$1.5B raised to date
- Positive IgAN Phase 3 results; BLA submission Q4 2025; **US market launch projected in 2026** subject to FDA approval

APRIL, A proliferation-inducing ligand; BAFF, B-cell activating factor; BLA, Biologics License Application; IgAN, immunoglobulin A nephropathy.



Atacicept Projected Catalysts

		Catalyst	2025	2026
 IgAN		Phase 3 full enrollment		
		Phase 3 primary endpoint		
		BLA submission		
		Projected US launch ¹		
 IgAN		Clinical results		
 IgAN, PMN, FSGS, MCD		Clinical results		

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

1. Subject to US FDA approval.
FSGS, focal segmental glomerulosclerosis; MCD, minimal change disease; PMN, primary membranous nephropathy.

Strong Financial Position

~\$497M

Cash, cash equivalents,
and marketable securities
(as of 9.30.25)

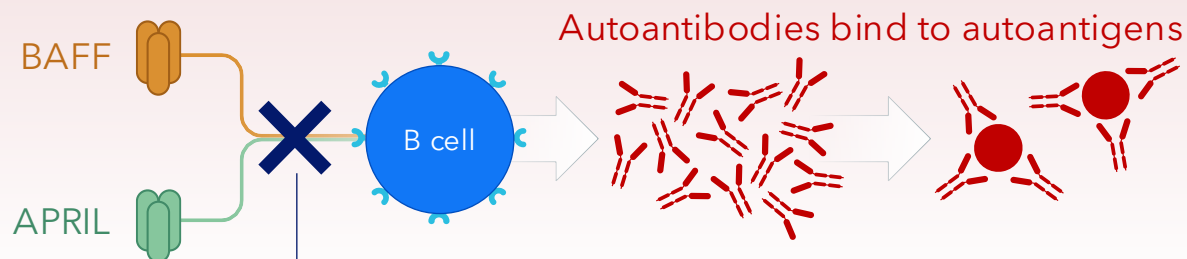
~63.9M

Shares outstanding
(as of 10.30.25)

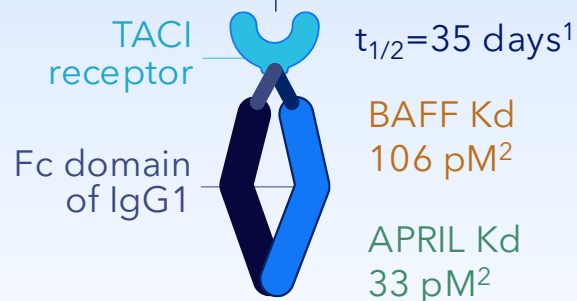
Additional \$425M non-dilutive capital available through Oxford Facility

B cell modulation via dual BAFF/APRIL inhibition represents a potential paradigm shift in how patients with autoimmunity will be treated

Autoimmune disease



- Autoantigens and autoantibodies mediate autoimmune disease
- B cells are source of autoantibodies → target cell of interest for therapeutic intervention
- B cells fueled by two cytokines, BAFF and APRIL



Atacicept

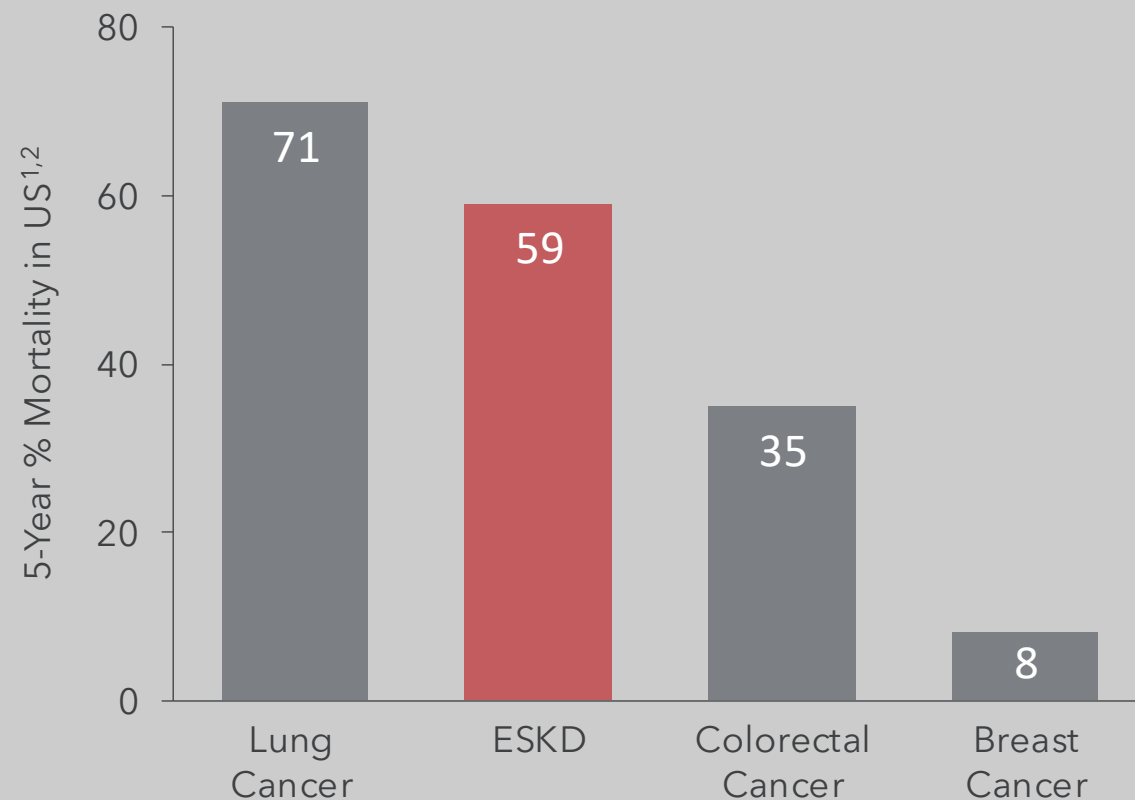
- Rationally designed therapeutic of modern biotechnology
- Native TACI-Fc fusion protein: soluble receptor binds BAFF and APRIL with picomolar binding affinity
- Offers the promise of precision modulation of B cells and autoantibodies

Fc, fragment crystallizable; IgG1, immunoglobulin G1; Kd, dissociation constant; TACI, transmembrane activator and calcium-modulator and cyclophilin ligand.

1. Willen D, et al. Eur J Drug Metab Pharmacokinet 2020; 2. Vera data on file.

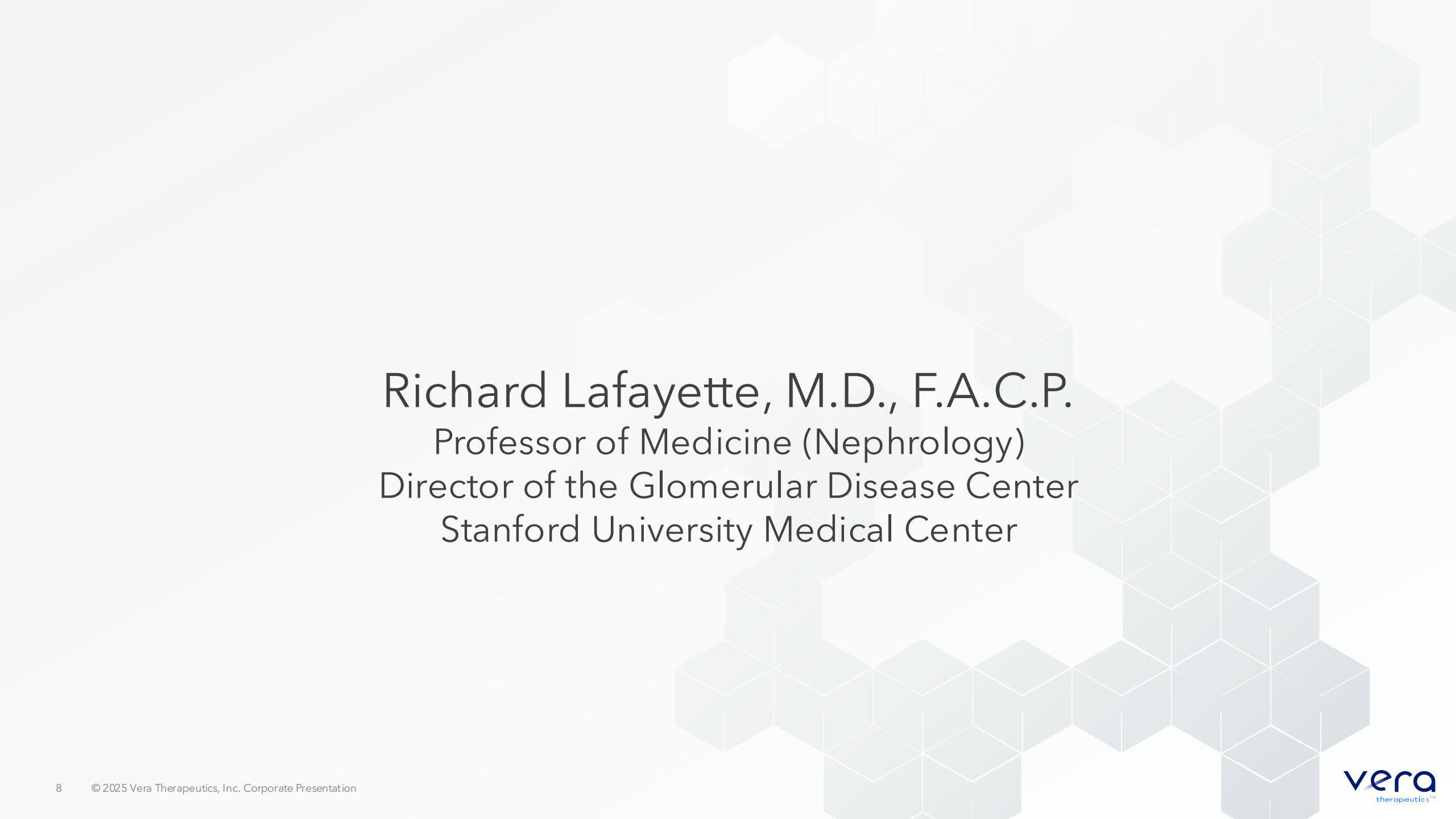
Inhibition of immune complex formation in IgAN offers the potential to avoid end stage kidney disease during a patient's lifetime

**ESKD 5-year mortality
comparable to cancer**



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).



Richard Lafayette, M.D., F.A.C.P.
Professor of Medicine (Nephrology)
Director of the Glomerular Disease Center
Stanford University Medical Center

IgAN: Most common primary glomerular disease worldwide



Estimated worldwide incidence of **2.5/100k** people per year in adults¹



Predominantly diagnosed in **young adults**, with most patients presenting with demonstrable CKD at time of diagnosis²⁻⁵



At least **50%** of patients progress to **kidney failure or death within 10 to 20 years** of diagnosis^{2,3}



2025 KDIGO Guideline recommends treatment goal of **reducing rate of loss of kidney function** to the physiological state (<1 mL/min/year for most adults)⁶



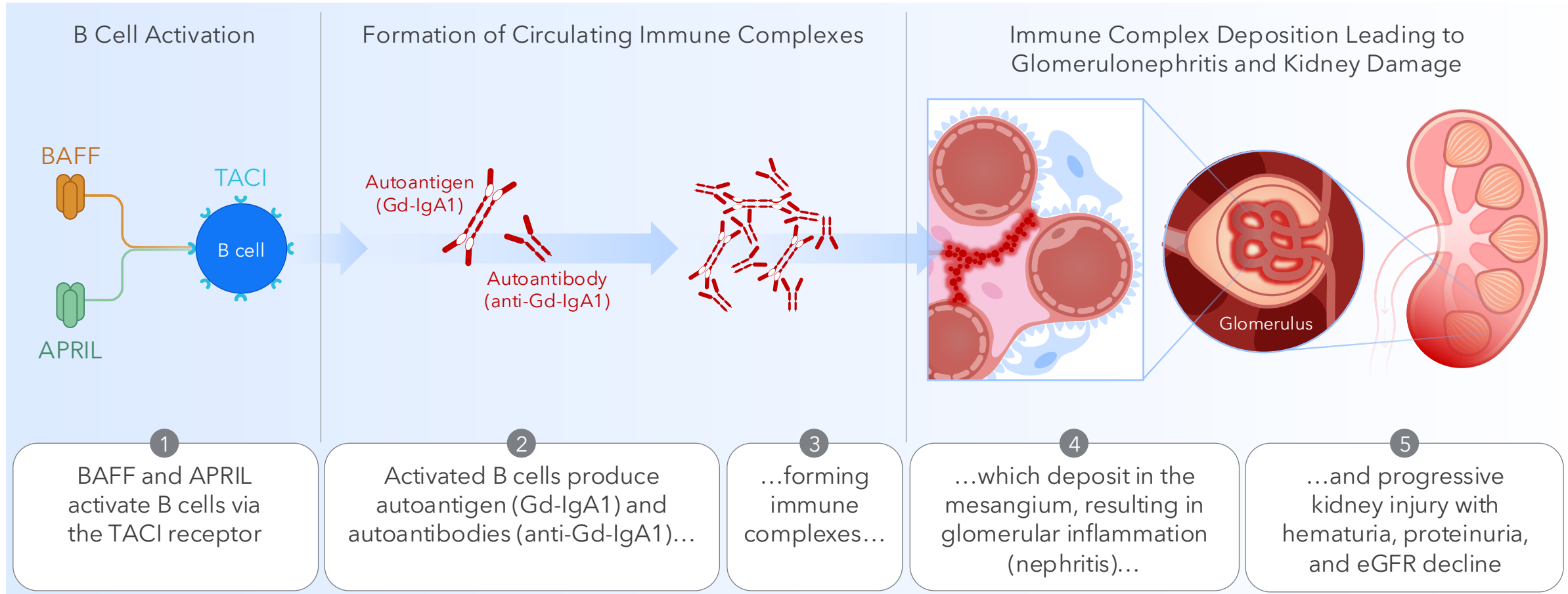
A **disease-modifying therapy** capable of stabilizing kidney function is an unmet need

CKD, chronic kidney disease.

1. McGrogan A, et al. Nephrol Dial Transplant 2011; 2. Kwon CS, et al. J Health Econ Outcomes Res 2021. 3. Pitcher D, et al. Clin J Am Soc Nephrol 2023. 4. Jarrick S, et al. J Am Soc Nephrol 2019. 5. Caster DJ, et al. Kidney Int Rep. 2023.

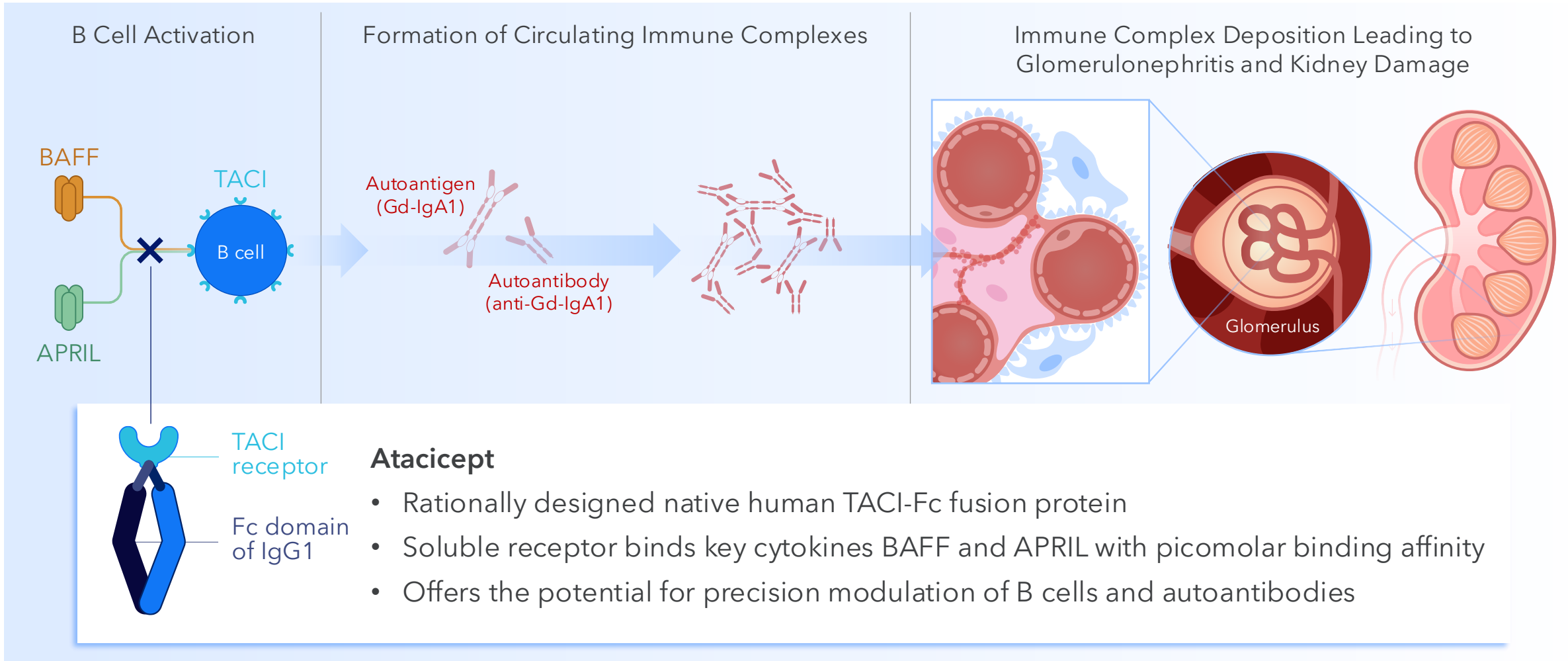
6. KDIGO IgAN and IgAV Work Group. Kidney Int. 2025.

IgAN is a disease of B cell origin with kidney pathology



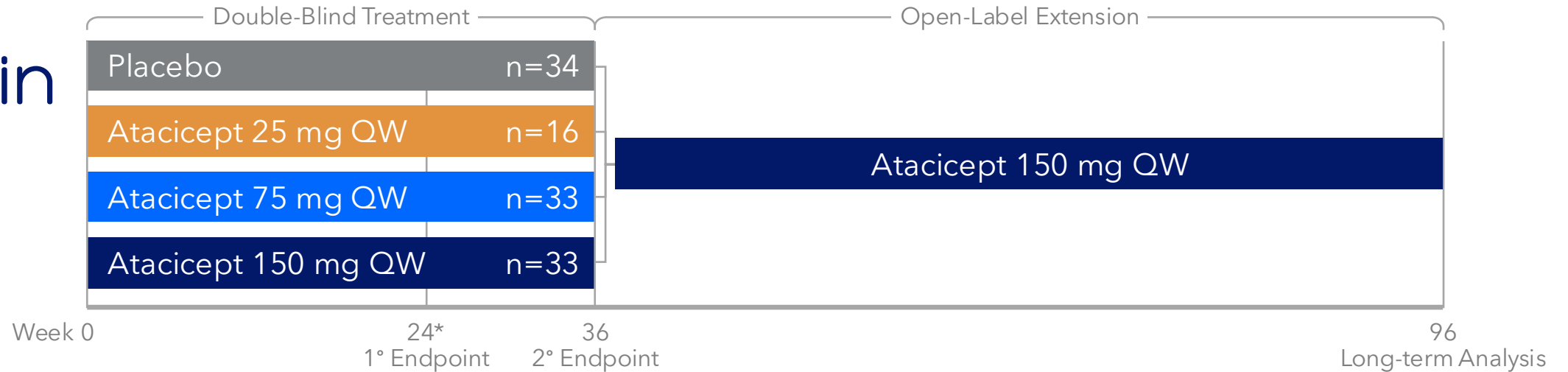
eGFR, estimated glomerular filtration rate; Gd-IgA1, galactose-deficient immunoglobulin A1.

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



Phase 2b Trial Design

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



Inclusion Criteria

- Participants ≥ 18 years old with biopsy-proven IgAN and high risk of disease progression
- Stable and optimized RAASi for ≥ 12 weeks
- Use of SGLT2i allowed
- UPCr-24h > 0.75 g/g or UP > 0.75 g per 24h
- eGFR ≥ 30 mL/min/1.73 m²
- Blood pressure $\leq 150/90$ mmHg

Endpoints

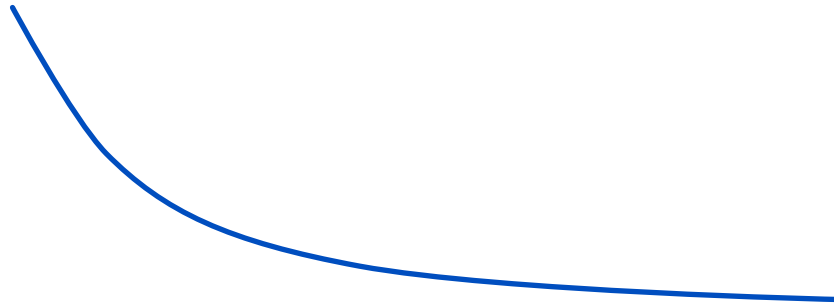
- Primary efficacy: UPCr-24h at week 24 achieved*
- Key secondary: UPCr-24h at week 36
- eGFR change up to week 96
- Gd-IgA1 change
- Hematuria resolution
- Safety

QW, once weekly; RAASi, renin-angiotensin-aldosterone system inhibitor; SC, subcutaneous; SGLT2i, sodium-glucose cotransporter-2 inhibitor; UPCr, urine protein:creatinine ratio.

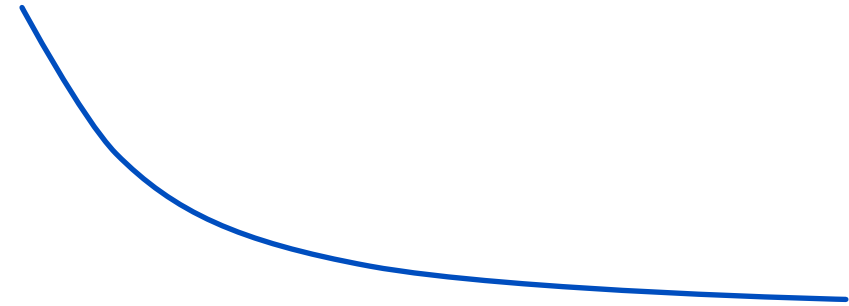
*The primary endpoint (mean difference between the combined 150- and 75-mg atacicept group versus placebo in UPCr-24h change from baseline at week 24 using the full analysis set population) was achieved ($p=0.037$).¹
1. Lafayette R, et al. Kidney Int. 2024.

A therapy that comprehensively addresses 2025 KDIGO treatment goals would...

Reduce immune complexes (Gd-IgA1)



Resolve inflammation (hematuria)



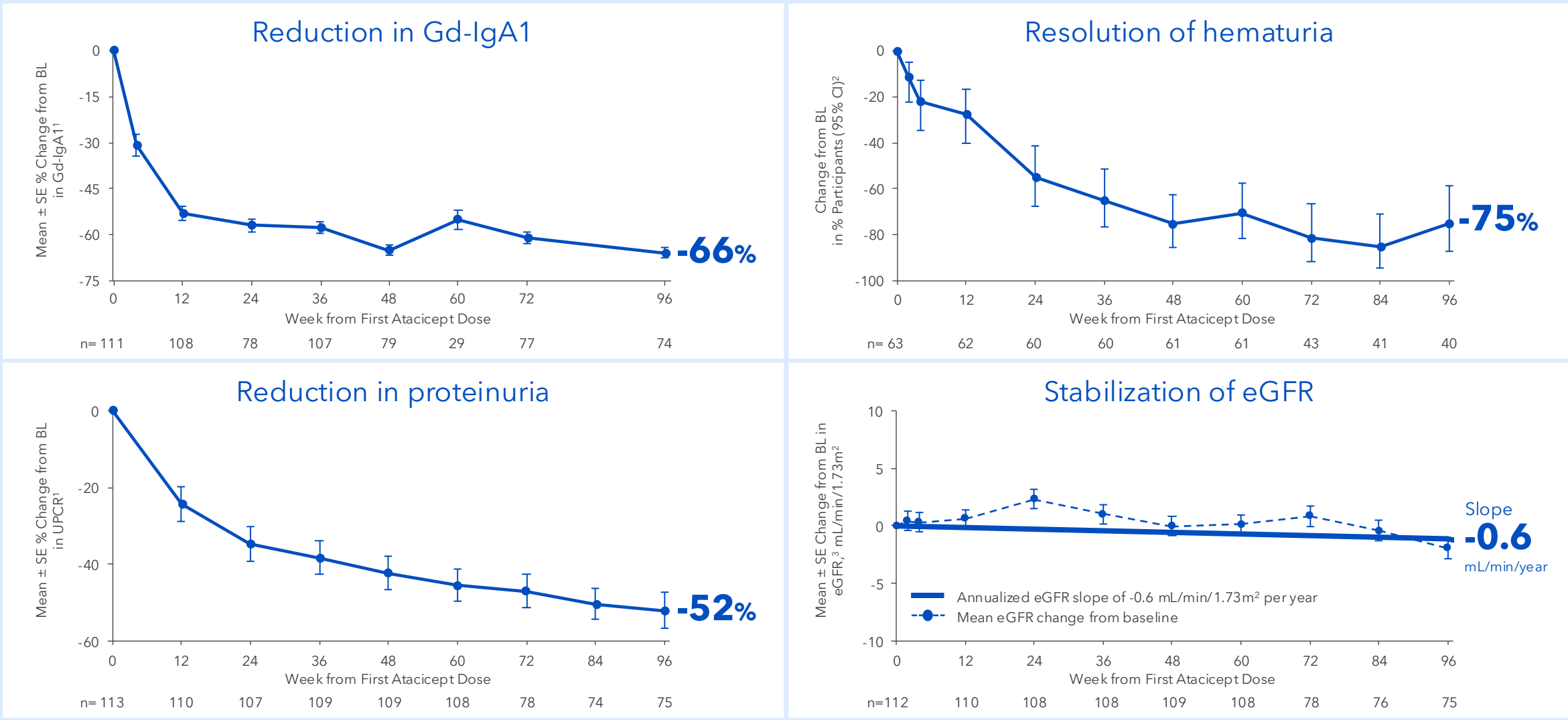
Reduce proteinuria



Stabilize eGFR



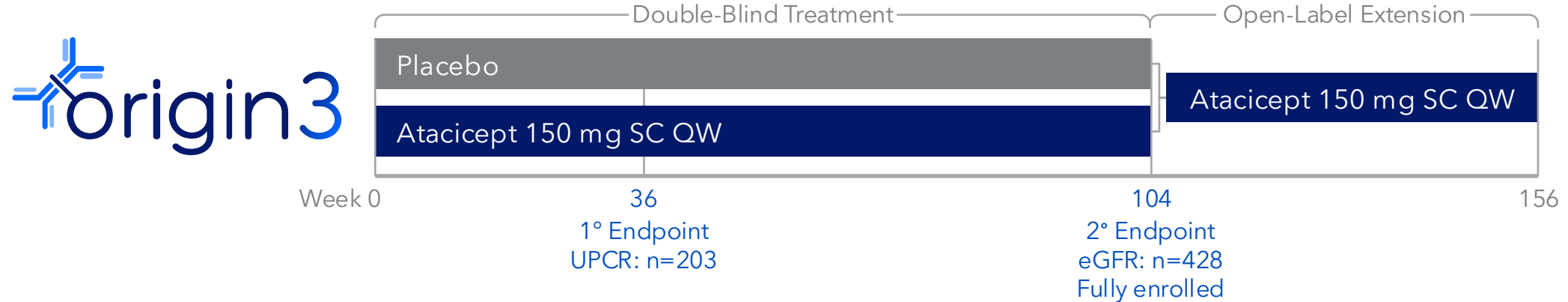
ORIGIN Phase 2b long-term results consistent with disease modification



Atacept group includes all participants receiving any atacept dose at any time point, with baseline (week 0) defined as last available measurement prior to first dose of atacept. CI, confidence interval. Barratt J, et al. J Am Soc Nephrol 2025.

Phase 3 Trial Design

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



Key Inclusion Criteria

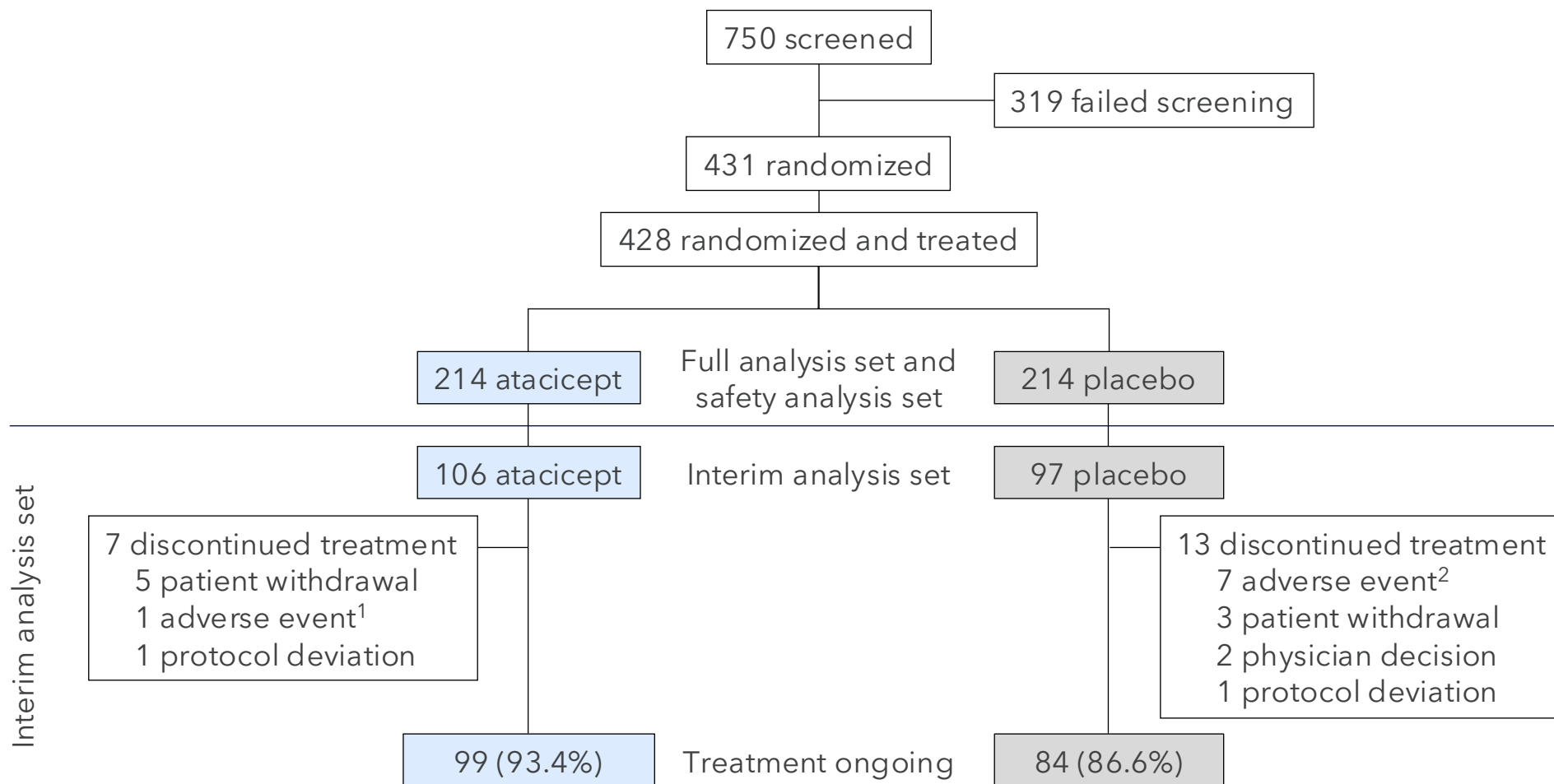
- Patients ≥ 18 years old with biopsy-proven IgAN and high risk of disease progression
- Stable and optimized RASi for ≥ 12 weeks, use of SGLT2i allowed
- UPCR-24h ≥ 1.0 g/g or UP ≥ 1.0 g per 24h
- eGFR ≥ 30 mL/min/1.73m²
- Blood pressure $\leq 150/90$ mmHg

Key Endpoints

- Primary efficacy: UPCR-24h % change at week 36
- Key secondary: eGFR change at week 104
- Gd-IgA1 change
- Hematuria resolution
- Safety

- Similar trial design, patient profile, and worldwide sites as ORIGIN 2b
- At home self-administered SC dose studied in ORIGIN 2b

ORIGIN 3 Interim Analysis Set Participant Disposition



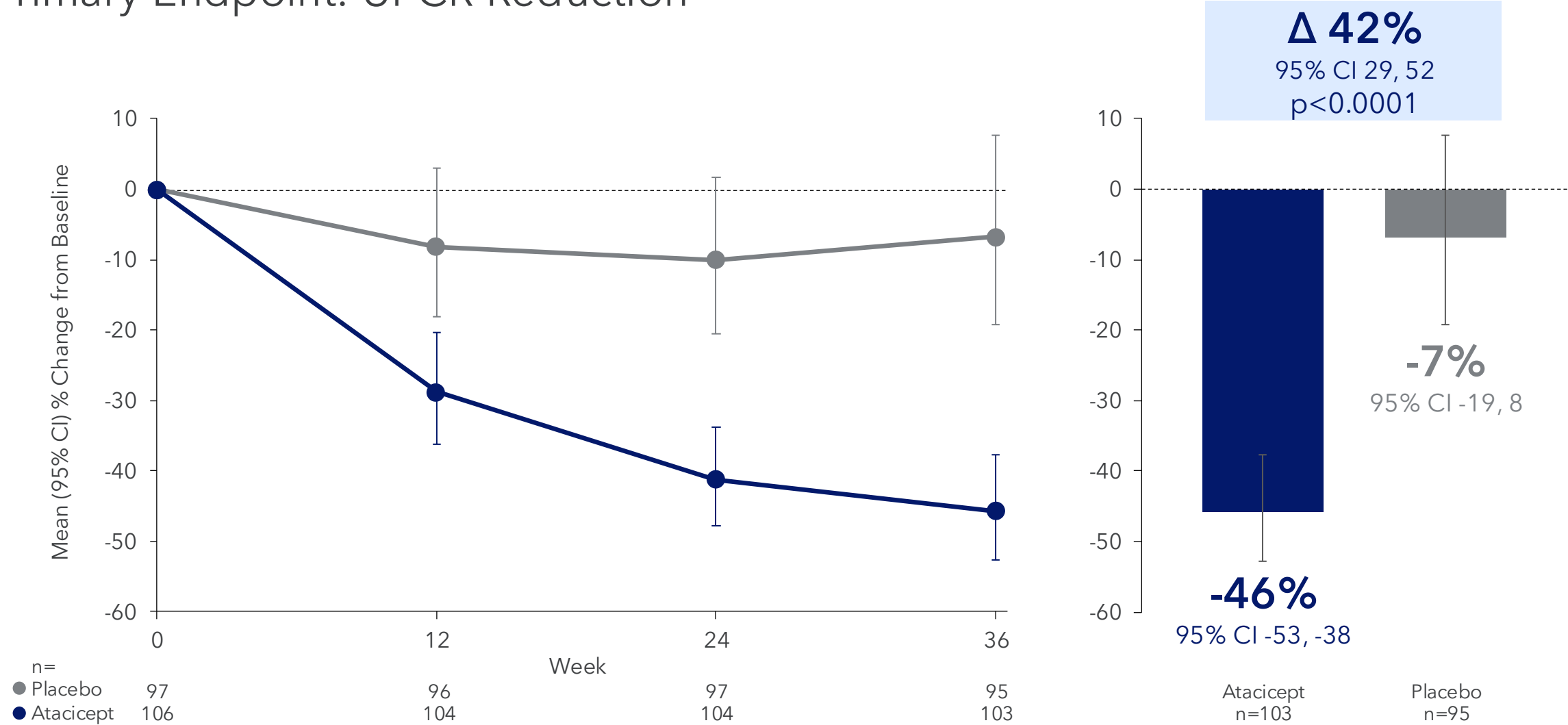
1. Rash.

2. Proteinuria, chronic kidney disease, IgAN, parophthalmia, pneumonia, pyelonephritis, osteonecrosis, carotid artery aneurysm.

ORIGIN 3 and ORIGIN 2b Demographics and Baseline Characteristics

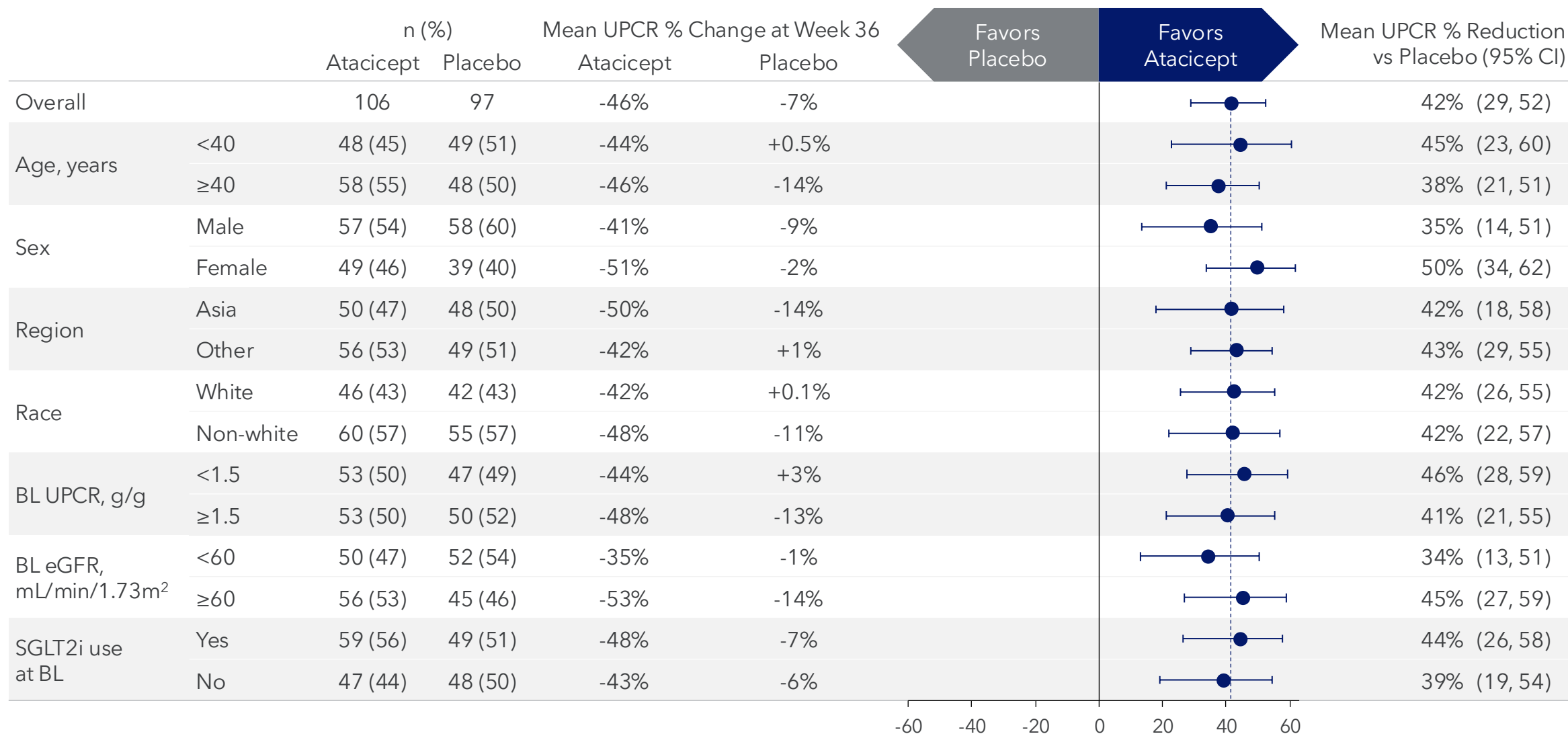
	ORIGIN 3 Interim Analysis Set			ORIGIN 2b
	Atacicept n=106	Placebo n=97	Total n=203	Total N=116
Age, median (range), years	40 (18, 72)	39 (19, 70)	40 (18, 72)	37 (18, 67)
Male sex, n (%)	57 (54)	58 (60)	115 (57)	69 (59)
Race, n (%)				
White	46 (43)	42 (43)	88 (43)	62 (53)
Asian	59 (56)	52 (54)	111 (55)	51 (44)
Black or African American	0	1 (1)	1 (0.5)	0
Native Hawaiian or other Pacific Islander	0	1 (1)	1 (0.5)	1 (1)
Other/not reported	1 (1)	1 (1)	2 (1)	2 (2)
Hispanic/Latino ethnicity, n (%)	14 (13)	6 (6)	20 (10)	4 (3)
eGFR, mean $\pm$ SD, mL/min/1.73 m ²	65 $\pm$ 28	65 $\pm$ 29	65 $\pm$ 28	63 $\pm$ 27
UPCR by 24h urine, mean $\pm$ SD, g/g	1.7 $\pm$ 0.9	1.8 $\pm$ 1.2	1.7 $\pm$ 1.0	1.6 $\pm$ 0.9
Time since biopsy, mean $\pm$ SD, years	2.5 $\pm$ 2.6	2.5 $\pm$ 2.4	2.5 $\pm$ 2.5	2.8 $\pm$ 2.8
SGLT2i use, n (%)	59 (56)	49 (51)	108 (53)	16 (14)

Primary Endpoint: UPCR Reduction



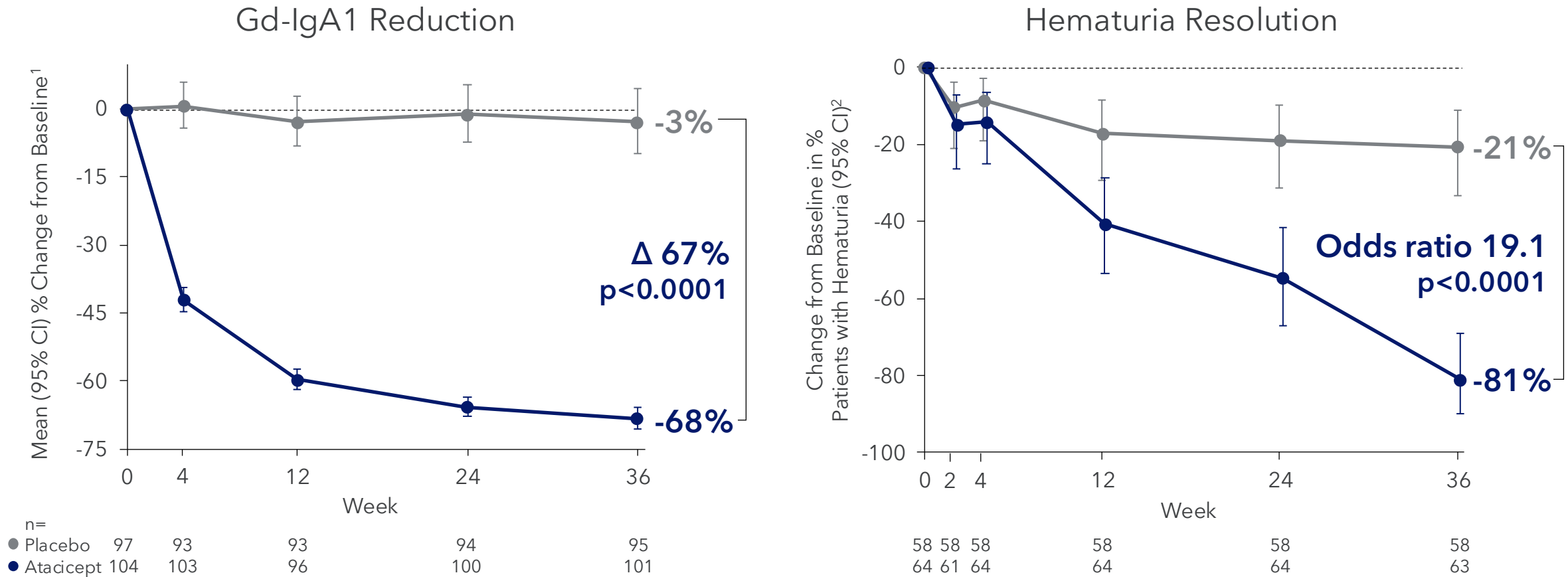
Interim Analysis Set (IAS) included the first 203 randomized participants who received ≥ 1 dose of trial drug. Change from baseline in natural-log transformed UPCR at Week 36 was analyzed using a mixed-effects model with repeated measurement (MMRM), including fixed effects for treatment group, visit as a categorical variable, treatment-by-visit interaction, baseline natural-log transformed UPCR, baseline eGFR category, SGLT2i use at baseline, and region, with participant as a random effect. log-transformed change from baseline in UPCR was estimated with use of least-squares means. To facilitate interpretation of the result, the least-squares means estimate was back-exponentiated to obtain the equivalent geometric mean percentage change. MMRM analysis included double-blind period data up to Week 36, regardless of treatment discontinuation or initiation of rescue treatment for IgAN or prohibited therapy; missing values after study withdrawal were imputed with jump to reference (placebo) approach for 100 times. The Rubin rule was used to combine results estimated from each of 100 imputation datasets by MMRM analysis.

UPCR Efficacy Consistent Across Prespecified Subgroups



Subgroup analyses were conducted using the same imputed data sets and the same MMRM model as for the primary analysis for each subgroup category, separately. If the subgroup was one of the covariates in the model, the covariate was removed from the model statement when the model was performed for the particular subgroup.

Gd-IgA1 Reduction and Hematuria Resolution



eGFR results not disclosed per FDA recommendation to sponsors of IgAN registrational trials

Analysis included all data up to Week 36 analyzed according to treatment policy strategy. Missing data were handled implicitly by statistical model. Nominal p-values are presented.
1. Change from baseline in natural log-transformed Gd-IgA1 was analyzed using MMRM similar to that for the primary endpoint. 2. Percentages represent change from baseline in number of participants with hematuria (urine dipstick blood ≥1+) at each visit divided by number of participants with baseline hematuria shown on the lower axis; resolution defined as urine dipstick blood of trace or negative. Odds ratio is calculated from a logistic regression model adjusted for covariates.

Adverse Events Generally Balanced Between Groups

Participants, n (%)	Atacicept n=214	Placebo n=214
Adverse events	127 (59)	107 (50)
Serious adverse events ¹	1 (0.5)	11 (5)
Adverse events leading to drug discontinuation ²	2 (1)	8 (4)
Adverse events of infections and infestations	68 (32)	60 (28)
Serious or severe infections and infestations	0	3 (1)
Opportunistic infections	0	0
Study drug related adverse events ³	63 (29)	22 (10)
Adverse events associated with injection site reactions ⁴	51 (24)	11 (5)
Hypersensitivity reactions	8 (4)	14 (7)
Adverse events leading to death	0	0

- Most adverse events were mild or moderate in severity
- Rate of serious adverse events was lower in the atacicept group compared with placebo
- No deaths occurred

Analysis of safety population (all participants randomized and treated) as of interim data cut on 15-May-2025.

1. Atacicept: 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 atacicept participants were due to eczema and erythema.

3. Majority were mild to moderate injection site reactions that did not lead to discontinuation.

4. Injection site reactions among atacicept recipients were largely characterized by injection site erythema, bruising, and pruritis.

No observed hypogammaglobulinemia (IgG <3 g/dL).

ORIGIN Program Summary

- IgAN is a B cell disorder with kidney pathology; BAFF and APRIL are the two key cytokines that drive B cell activity
- Atacicept is a rationally designed native human TACI-Fc fusion protein that binds BAFF and APRIL, with picomolar affinity, providing B cell modulation with antibody reduction
- ORIGIN Phase 2b 96-week results: reductions in Gd-IgA1, hematuria, and proteinuria, and stabilization of eGFR
- ORIGIN Phase 3 36-week results: statistically significant proteinuria reduction compared with placebo, with consistent efficacy across prespecified subgroups; also observed reduction in Gd-IgA1 and resolution of hematuria in patients with blood in their urine at baseline
- Safety profile of atacicept was favorable, and comparable to placebo, without evidence of immunosuppression (no serious, severe, or opportunistic infections)

A Phase 3 Trial of Atacicept in Patients with IgA Nephropathy

published in
The New England Journal of Medicine



Scan to visit NEJM.org



Matt Skelton

Executive Vice President, Commercial
Vera Therapeutics

Estimated IgAN epidemiology in 2032

US IgAN Prevalence: ~0.04% of US Population (360.5M)¹

~160K

+ ~40K
Low Risk
(~24% of patients)²

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



**BAFF/APRIL
inhibition**

~90K
High Risk
(~56% of patients)²

Phase 3
population

 **pioneer**

Phase 2 study ongoing

Patient counts rounded to nearest 1,000.

1. Clearview Healthcare Partners Analysis 2021; 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.

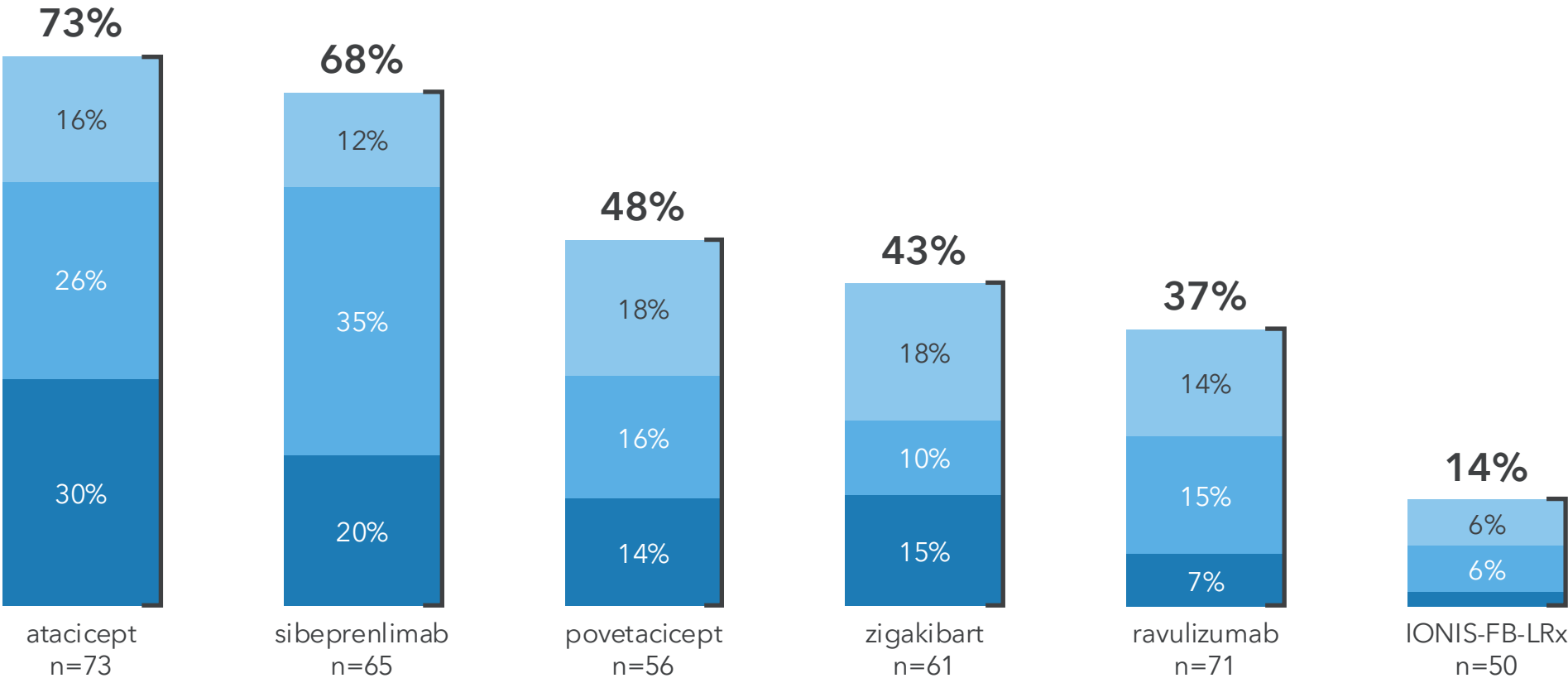
Nephrologists view atacicept as a desirable therapy for IgAN

Most Desired IgAN Pipeline Agent

% of familiar respondents (rated familiarity of product >3 on a 10-point scale)

Considering the pipeline agents listed, please rank order the TOP THREE that you would most like to see approved for use in IgAN.

- Ranked in top 3
- 3rd most desired
- 2nd most desired
- Most desired



Atacicept product profile is compelling to nephrologists

Perceived Impact	Factor	Rationale
	Atacicept eGFR stabilization (Ph2b data)	Key driver of preference / potential use
	Mechanism of action / disease-modifying therapy	HCPs find this attribute compelling; Gd-IgA1 reduction serves as evidence for academic clinicians
	Availability of long-term (96 week) data	HCPs note that atacicept can potentially reduce number of treatments needed to delay dialysis / kidney failure
	Appropriate for patients with >1 g/g UPCR, eGFR decline of 2.5 mL/min/y	This patient type identified as appropriate for treatment from patient case exercise
	Other biopsy findings, hematuria not required to prescribe atacicept	While these may increase motivation to prescribe, HCPs are willing to prescribe without these

Source: Primary Market Research, N=15 Nephrologists (11/2024).

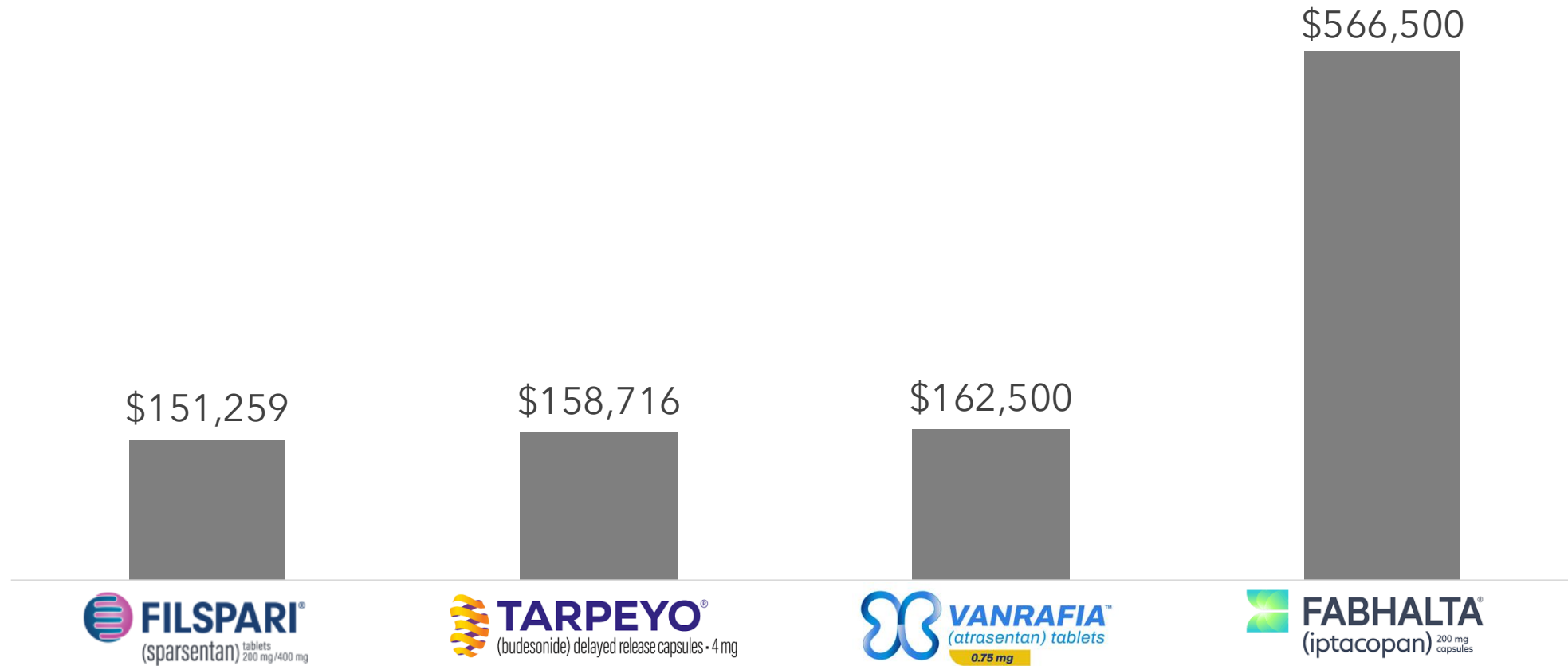
Atacicept could launch with characteristics similar to those of blockbuster drugs

	At-home, self-administered	1ml or less injection volume	Autoinjector option	27G or smaller needle	Once-weekly frequency
OZEMPIC (semaglutide)	✓	✓	X	✓	✓
mounjaro (tirzepatide)	✓	✓	✓	✓	✓
wegovy (semaglutide)	✓	✓	✓	✓	✓
trulicity (dulaglutide)	✓	✓	✓	✓	✓
HUMIRA (adalimumab)	✓	✓	✓	✓	X
DUPIXENT (dupilumab)	✓	X	✓	✓	X
Stelara (ustekinumab)	✓	✓	X	✓	X
Cosentyx (secukinumab)	✓	✓	✓	✓	X
Skyrizi (rizankinumab)	✓	✓	✓	✓	X
Enbrel (etanercept)	✓	✓	✓	✓	✓
Atacicept (est.)	✓	✓	✓	✓	✓

Patient retention in ORIGIN Ph 2b and Ph 3 trials have been consistently above 90%

Premium pricing in the IgAN space

Annual Prices for Approved IgAN Therapies¹



1. As of May 30, 2025. *Price for Tarpeyo reflects the 9-month recommended course of treatment. Full 12 month course of treatment would be \$213,010.

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

IgAN Market



- We believe the nephrology market is ripe for disruption due to low levels of approved product saturation
- Large market with unmet need
- Growing market with IgAN¹
- Favorable payer mix²

Atacicept



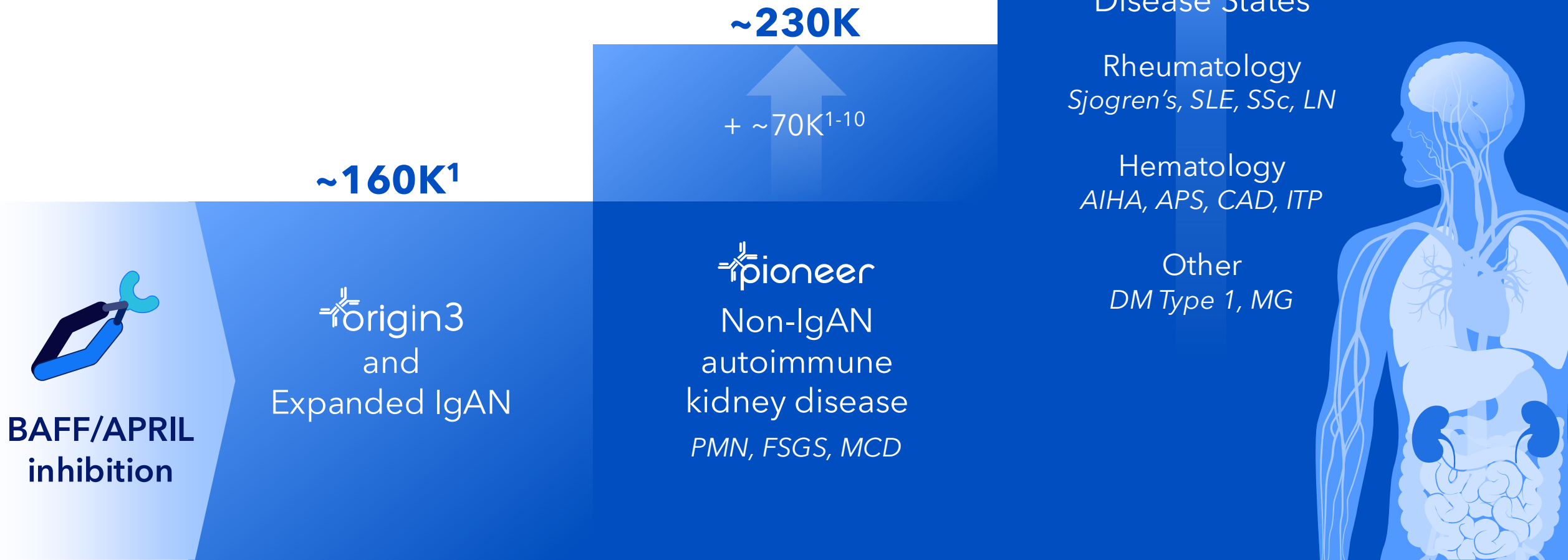
- Potentially differentiating product attributes
- eGFR data highly valued with enthusiastic support from therapeutic experts and insights captured at advisory boards³
- Experienced commercial team in place and ready to execute
- Exciting lifecycle opportunities

1. ClearView Healthcare Partners Analysis; 2. Bluepath Solutions research conducted in Q4 2024; 3. Spherix Realtime Dynamix US IgAN independent survey conducted in Q4 2024 and advisory boards conducted by Vera Therapeutics.

Robert M. Brenner, M.D.
Chief Medical Officer
Vera Therapeutics

Atacicept has potential in multiple large markets,
with an initial focus in autoimmune kidney disease

US prevalence estimates



AIHA, autoimmune hemolytic anemia; APS, antiphospholipid syndrome; CAD, cold agglutinin disease; DM, diabetes mellitus; ITP, immune thrombocytopenia; LN, lupus nephritis; MG, myasthenia gravis; SLE, systemic lupus erythematosus; SSc, systemic sclerosis.

Vera Therapeutics corporate estimates for peak year prevalence based on 1. ClearView Healthcare Partners Analysis 2021; 2. US Census 2023; 3. McGrogan A. Nephrol Dial Transplant 2011; 4. Couser ASN 2017; 5. Beck LH. N Engl J Med 2009; 6. Filler G. Am J Kidney Dis 2003; 7. Troyanov S. J Am Soc Nephrol 2005. 8. Hommos MS. Mayo Clin Proc 2017; 9. Hengel FE. N Engl J Med 2024; 10. Vivarelli M. Clin J Am Soc Nephrol 2017.

PIONEER: Operationally efficient Phase 2 basket trial in expanded IgAN and anti-PLA2R & anti-nephrin podocytopathies



Population 1
Expanded IgAN populations¹

Population 2
Anti-PLA2R podocytopathy
(PMN)

Population 3
Anti-nephrin podocytopathy
(MCD/FSGS)

Atacicept 150 mg SC QW

Week 0

52

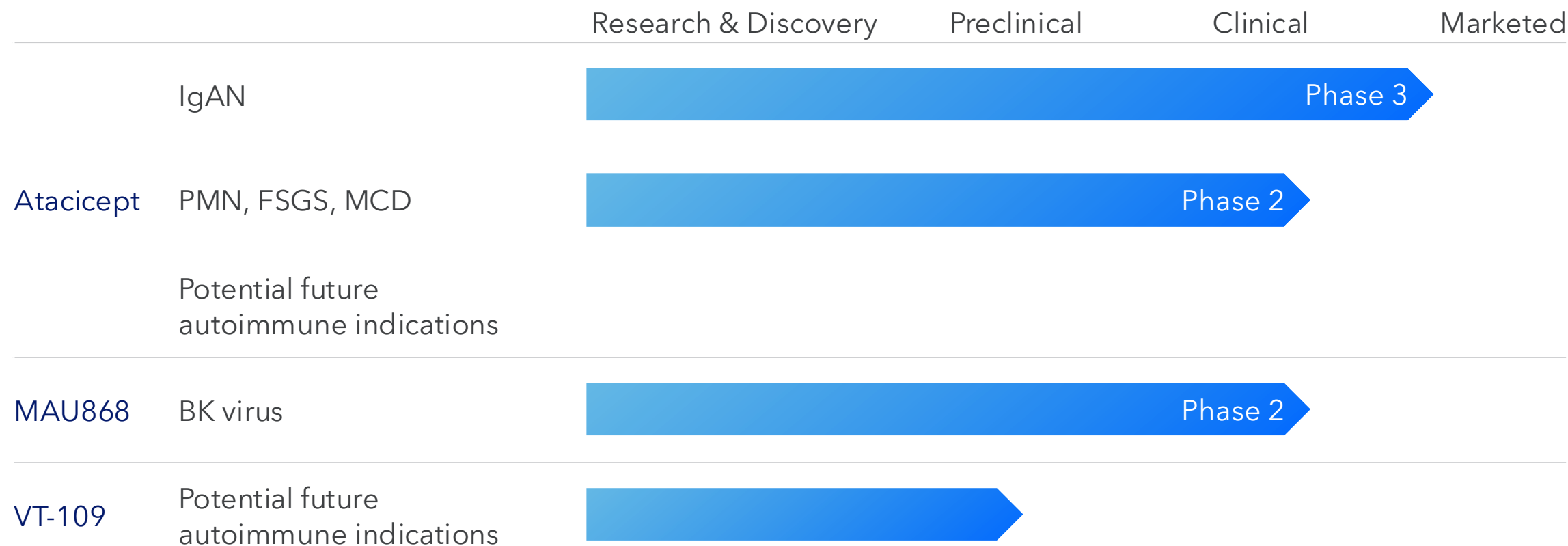
Key Endpoints

- Primary: UPCR change
- Key secondary: eGFR change
- Exploratory:
 - Gd-IgA1 change
 - Change in percentage of participants with hematuria
 - Change in anti-PLA2R antibodies
 - Change in anti-nephrin antibodies
- Safety

PLA2R, phospholipase A2 receptor.

1. Includes cohorts with age ≥ 10 y, UPCR ≥ 0.5 g/g, eGFR ≥ 20 mL/min/1.73m², recurrent IgAN post kidney transplant and/or IgA vasculitis nephritis (varies by cohort). ClinicalTrials.gov NCT06983028.

Vera Pipeline



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

Q&A

Marshall Fordyce, M.D.	Founder and Chief Executive Officer	Vera Therapeutics
Robert M. Brenner, M.D.	Chief Medical Officer	
Sean Grant	Chief Financial Officer	
Matt Skelton	Executive Vice President, Commercial	
Jonathan Barratt, Ph.D., F.R.C.P.	Mayer Professor of Renal Medicine	University of Leicester
Richard Lafayette, M.D., F.A.C.P.	Professor of Medicine (Nephrology)	Stanford University Medical Center

The logo for Vera Therapeutics features the word "vera" in a large, white, lowercase sans-serif font. A thin white line extends from the bottom of the 'v' and curves around the 'e'. Below "vera" is the word "therapeutics" in a smaller, white, lowercase sans-serif font, followed by a trademark symbol (TM). The background is a solid dark blue with a pattern of lighter blue hexagons on the left side.

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therapeutics™