



Setting the Standard
in IgA Nephropathy

Corporate Presentation

December 2024

Forward-looking statements

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Atacicept potentially *best* and *first-in-class* dual BAFF/APRIL B cell modulator in IgAN, with pipeline-in-a-product opportunity

IgAN Potential Best-in-Class



- eGFR normalization may suggest functional cure
- Only program with 2-yr data in Phase 2 → potential for commercial differentiation, if approved
- Only investigational drug with at home self administration of 1 mL QW and 90% patient retention at 2 yr
- Phase 3 read out on track for Q2 2025; if successful, anticipated PDUFA 2026

Indication Expansion



- B cell modulation represents a treatment paradigm shift for autoimmune diseases
- Atacicept clinical data to date supports potential for chronic administration
- Progressive expansion in addressable patients: initial autoimmune kidney disease opportunity >200K
- Additional potential upside in hematologic, rheumatologic, and other kidney indications

Resourced for Potential Launch



- Regulatory exclusivity expected through 2038 in US and 2037 in EU
- Currently ~\$677M *pro forma* cash, cash equivalents and marketable securities as of September 30, 2024¹
- Management focused on potential for successful commercial launch

BAFF = B cell activating factor; APRIL = A proliferation inducing ligand; eGFR = estimated glomerular filtration rate; IgAN = immunoglobulin A nephropathy; PDUFA = Prescription Drug User Fee Act; QW = once weekly; SC = subcutaneous.

1. Includes ~\$353M of cash, cash equivalents and marketable securities as of September 30, 2024 and additional ~\$324M in net proceeds from October 2024 follow-on equity offering. This estimate of the Company's cash, cash equivalents and marketable securities as of September 30, 2024 has not been audited.

Cumulative Atacicept data to date offers best-in-class potential



Atacicept



Sibeprenlimab¹



Povetacicept²



Zigakibart³

	Atacicept	Sibeprenlimab ¹	Povetacicept ²	Zigakibart ³
Mechanism	BAFF/APRIL inhibition	APRIL inhibition only	BAFF/APRIL inhibition	APRIL inhibition only
Dosing & Administration	25/75/150 mg SC QW (Ph2) 150 mg SC QW (Ph3) 1x1 mL self-administered	2/4/8 mg/kg IV (Ph2) 400 mg SC QM (Ph3) 1x2 mL in-clinic injection	80/240 mg SC QM (Ph1b) 1xTBD mL injection	450 mg IV Q2W (Ph2) 600 mg SC Q2W (Ph3) 2x2 mL in-clinic injection
Development Stage	Ph3	Ph3	Ph3	Ph3
Randomized Controlled Trial Data	✓	✓	✗	✗
Gd-IgA1 Reduction	64% at W36 vs 7% placebo	~60% at W52 vs ~+20% placebo	No placebo controlled data	No placebo controlled data
Hematuria	80% resolution at W36	Reductions at W36 (nonquantifiable)	Not reported	Not reported
UPCR Reduction vs Placebo	Δ 43% (p=0.003) at W36	Δ 43% at W36	No placebo controlled data	No placebo controlled data
eGFR Duration Data	24 months, n=102	12 months, n=145	12 months, n=1	12 months, n=35
Projected Commercial Launch	2026	2026	2027	2027

This data is based on a cross-trial comparison and not a head-to-head clinical trial; such data may not be directly comparable due to differences in study protocols, conditions and patient populations. Atacicept 150 mg data from Phase 2b ORIGIN trial shown for urine protein-creatinine ratio (UPCR), galactose-deficient immunoglobulin A1 (Gd-IgA1), and hematuria. 1. Ph2 4 mg/kg IV Gd-IgA1 data from Mathur M, et al. NEJM 2023, Ph2 4 mg/kg IV hematuria data from Barratt J, et al. WCN 2024, WCN24-AB-1799, Ph2 pooled sibeprenlimab UPCR data from Kooienga ASN 2022, TH-PO991, and estimated glomerular filtration rate (eGFR) data from Barratt J, et al. ASN 2023, abstr TH-PO1124; 2. Ph1b 80 mg data from Tumlin J, et al. ASN 2023, TH-PO1125, and Tumlin J, et al. WCN 2024, WCN24-AB-762. 3. Barratt J, et al. ERA 2024, late breaking abstract.



Strong financial position

~\$677M

Cash, cash equivalents,
and marketable securities
*(unaudited as of 9.30.24)*¹

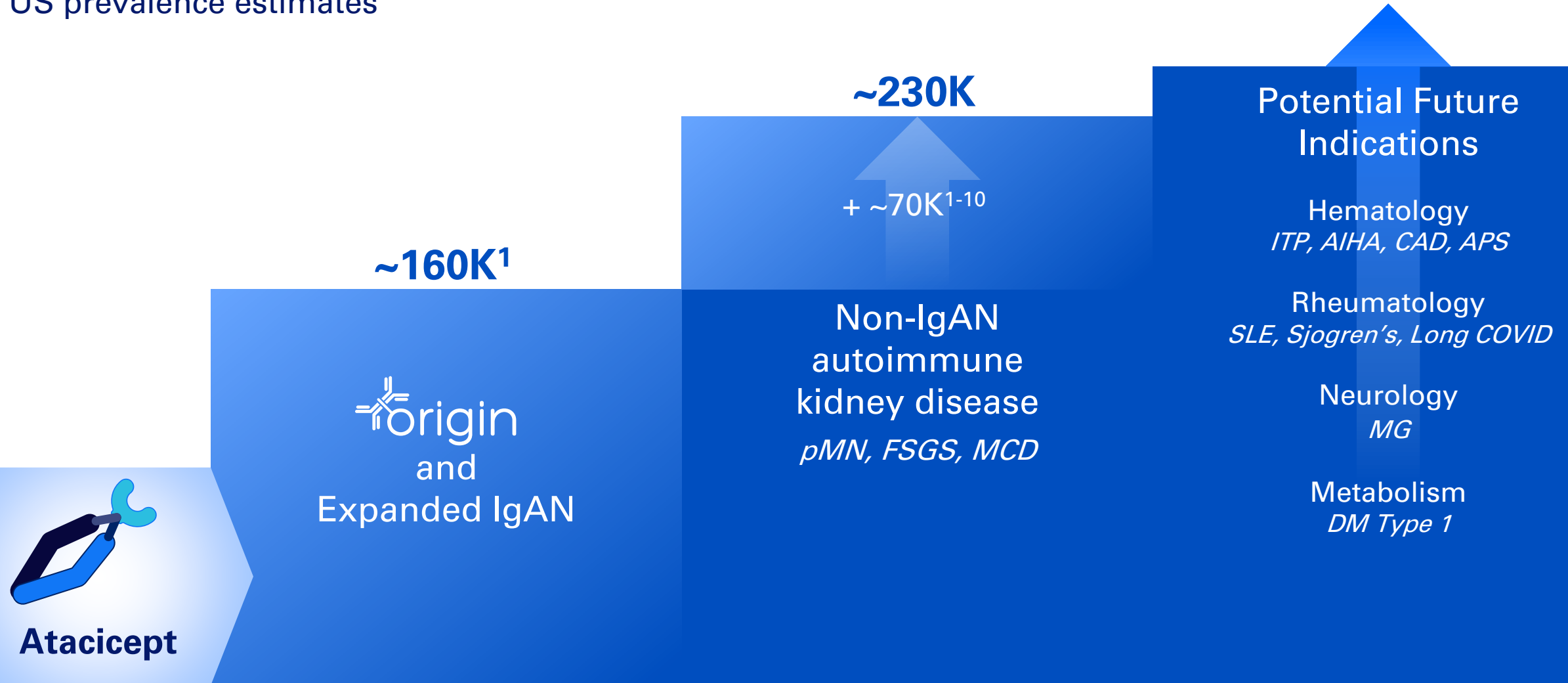
~63.4M

Shares outstanding
(as of 11.21.24)

1. Includes ~\$353M of cash, cash equivalents and marketable securities as of September 30, 2024 and additional ~\$324M in net proceeds from October 2024 follow-on equity offering. This estimate of the Company's cash, cash equivalents and marketable securities as of September 30, 2024 has not been audited.




Vera optionality to expand in autoimmune kidney disease & beyond

US prevalence estimates



Vera Therapeutics corporate estimates for peak year prevalence based on 1. ClearView Healthcare Partners Analysis; 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. pMN = primary membranous nephropathy; FSGS = focal segmental glomerulosclerosis; MCD = minimal change disease; ITP = immune thrombocytopenia; AIHA = autoimmune hemolytic anemia; CAD = cold agglutinin disease; APS = antiphospholipid syndrome; SLE = systemic lupus erythematosus; COVID = Coronavirus disease 2019; MG = myasthenia gravis; DM = diabetes mellitus.

Atacicept projected catalysts

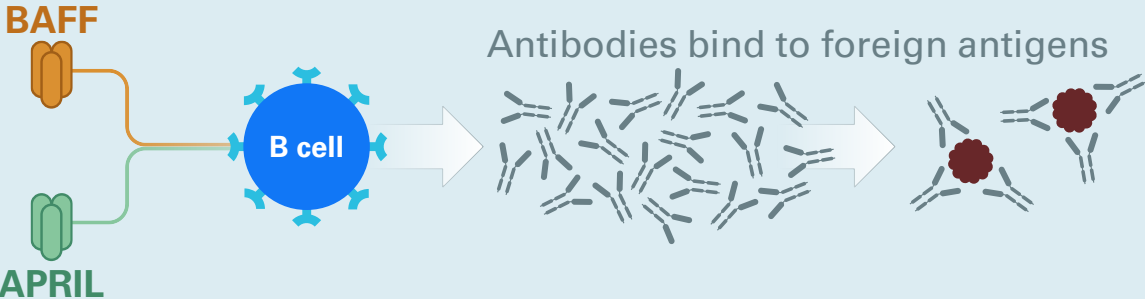
Catalyst	2024	2025	2026	
	Phase 3 primary endpoint cohort full enrollment	✓ 3Q		
	Phase 2b 96-week results	✓ 4Q		
	Phase 3 top-line results		● 2Q	
	BLA submission		● 2H	
	Projected US launch ¹			●
	Initiation	●		
	Initial data available		●	
	Initiation		●	
	Initial data available		●	

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

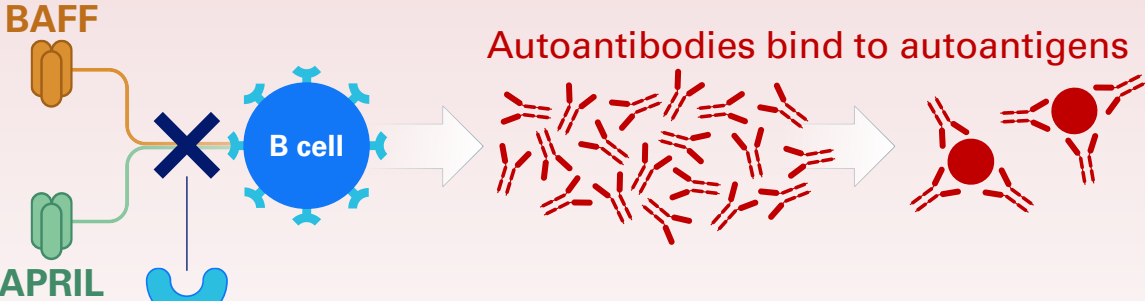
Based on management’s current assumptions. 1. Subject to US approval.

Atacicept has broad therapeutic potential to address multiple autoimmune diseases

Immunity in health



Autoimmune disease

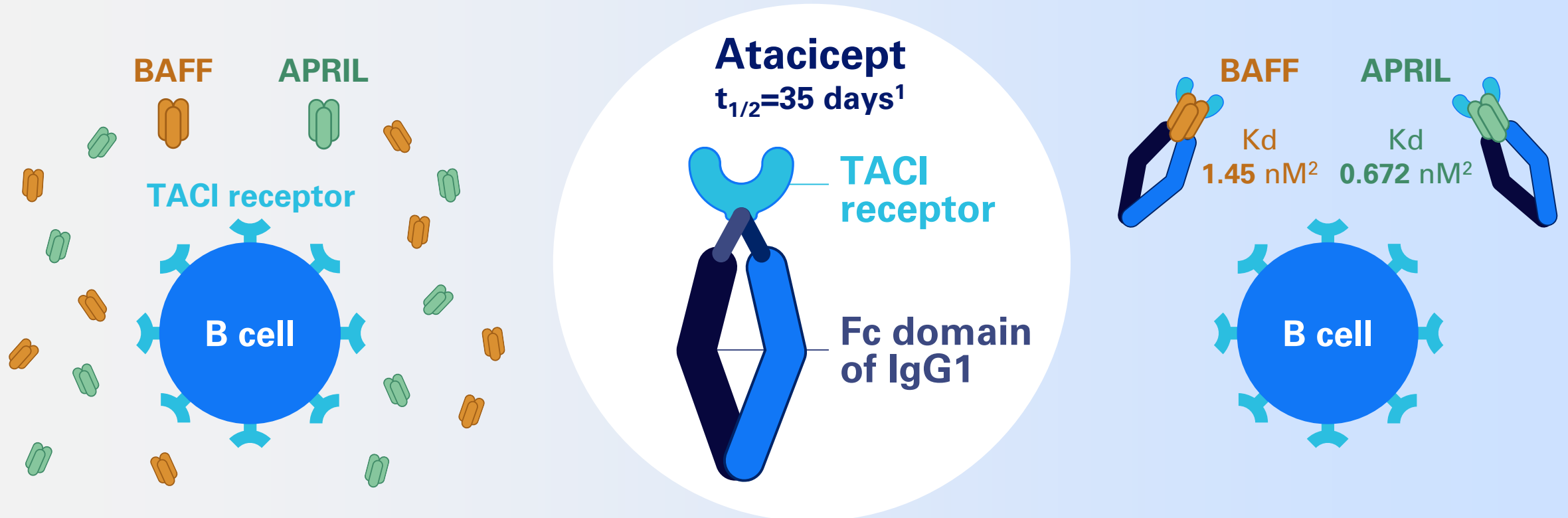


- Autoantigens and autoantibodies mediate autoimmune disease
- B cells are the source of autoantibodies → target cell of interest for therapeutic intervention
- B cells are fueled by two (and only two) cytokines, BAFF and APRIL

Atacicept: Rationally designed therapeutic of modern biotechnology that binds BAFF and APRIL with nanomolar potency offers the promise of precision modulation of B cells and autoantibodies

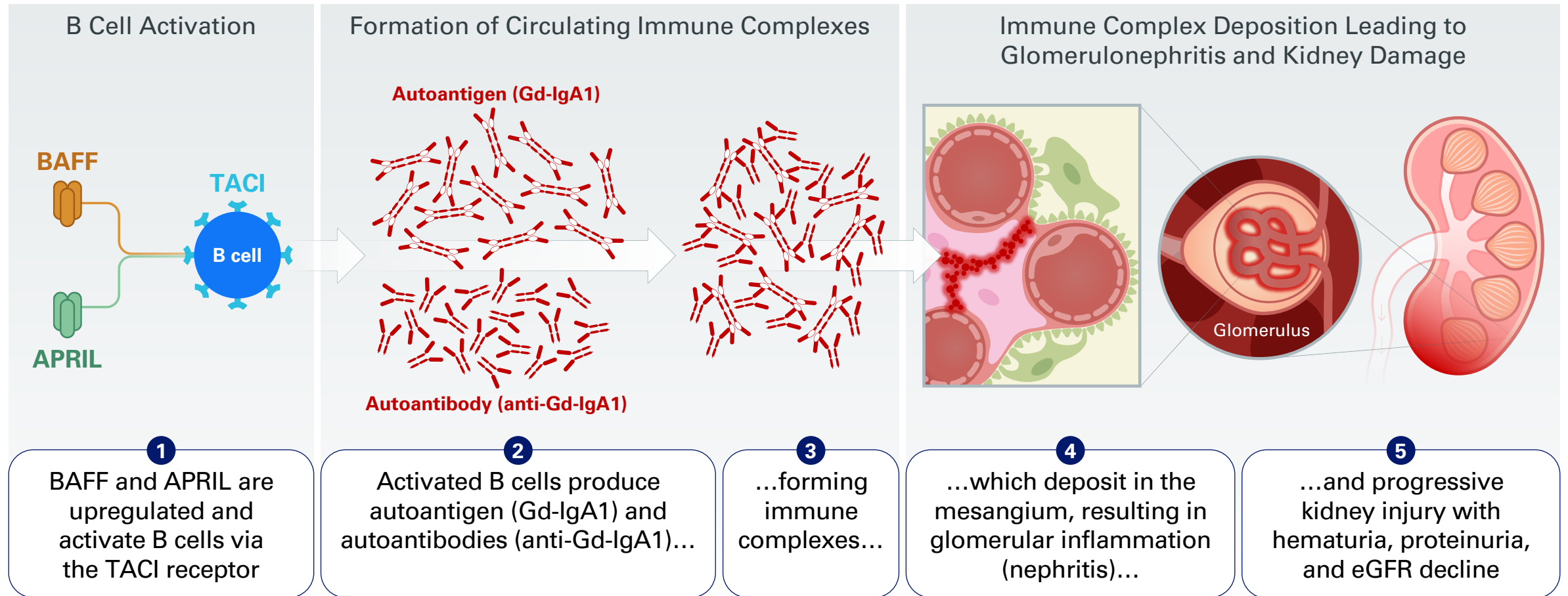
Atacicept is an example of rational drug design

Native TACI-Fc fusion: Soluble protein binds both BAFF and APRIL with nanomolar potency



Fc = fragment crystallizable; IgG1 = immunoglobulin G1; Kd = dissociation constant.
1. Willen D, et al. Eur J Drug Metab Pharmacokinet 2020;45(1):27-40; 2. Vera data on file.

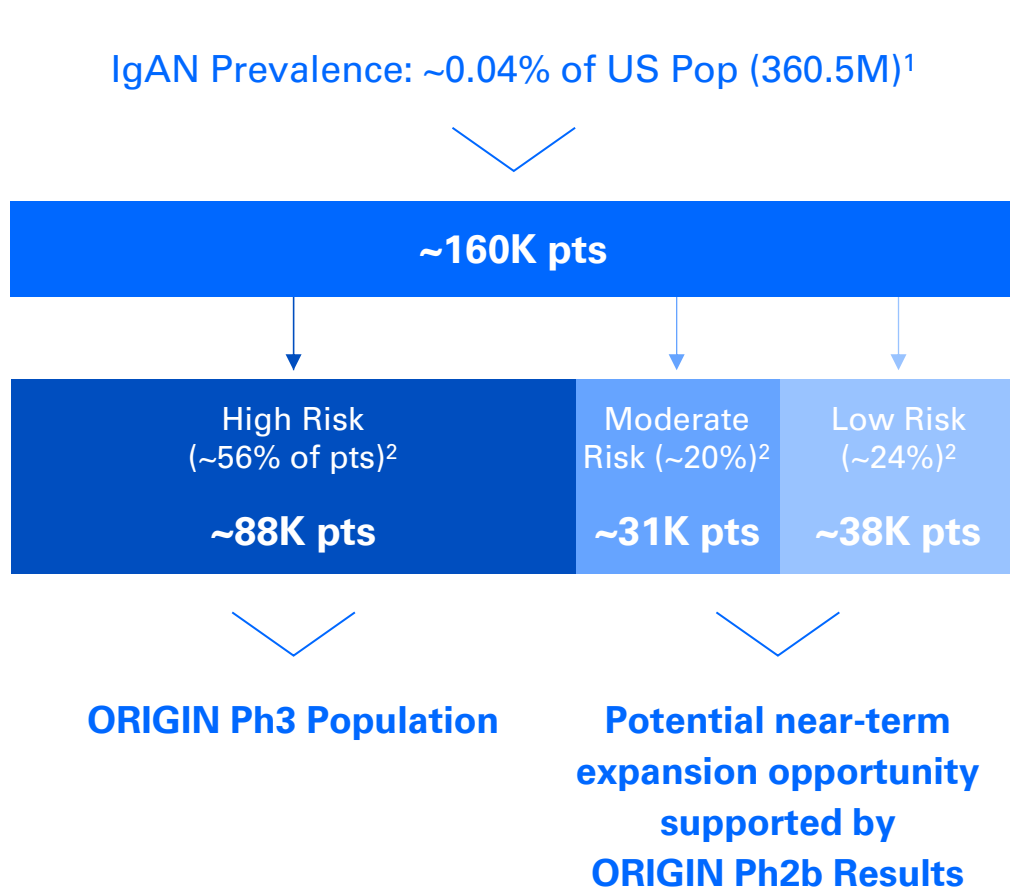
Lead indication: IgAN is a disease of B cell origin with kidney pathology



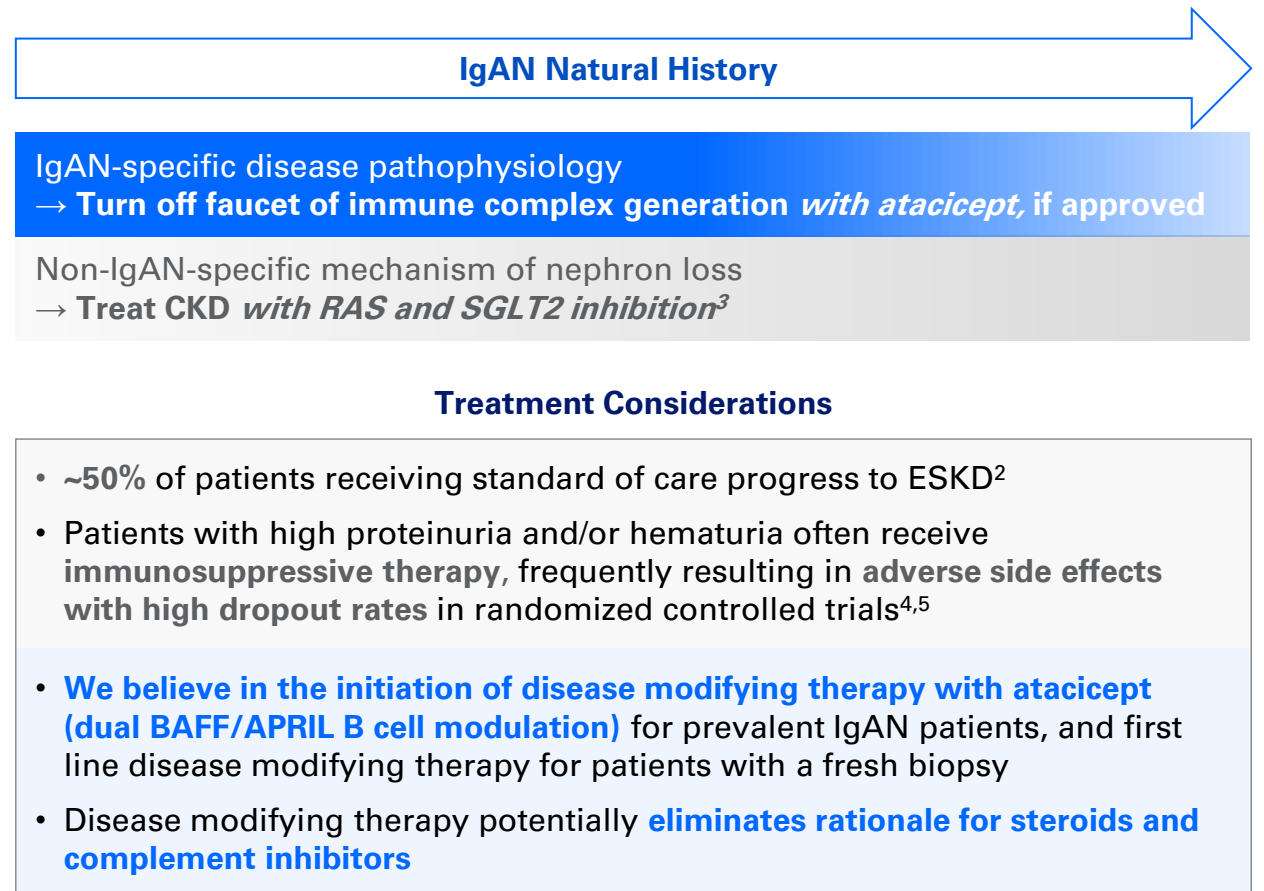
Gd-IgA1 = galactose-deficient immunoglobulin A1; TACI = transmembrane activator and calcium-modulator and cyclophilin ligand.

IgAN epidemiology considerations and treatment paradigm

Estimated IgAN Epidemiology in 2032E



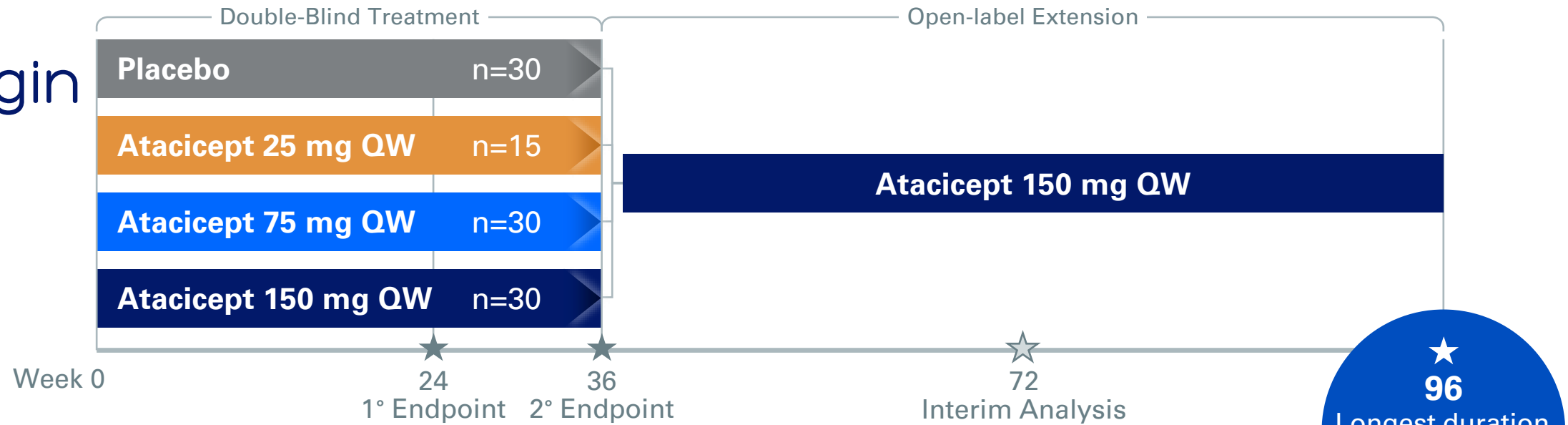
Diagnosis and Treatment Paradigm



Patient counts rounded to nearest 1,000. CKD = chronic kidney disease; ESKD = end stage kidney disease; RAS = renin-angiotensin system; SGLT2 = sodium-glucose cotransporter-2 inhibitor

1. ClearView Partners Analysis; 2. Pitcher D, et al. Clin J Am Soc Nephrol 2023. Low Risk assumed to be 0–0.44 g/g urine protein creatinine ratio (UPCR), Mod Risk assumed to be 0.44–0.88 g/g, High Risk assumed to be >0.88 g/g; percentage of patients per risk group in overall study population applied to estimated US IgAN prevalence; 3. Kidney Disease: Improving Global Outcomes 2021 Clinical Practice Guideline for the Management of Glomerular Diseases; 4. Rauen T, et al. N Engl J Med 2015; 5. Lv J, et al. JAMA 2022.

ORIGIN Phase 2b long-term data revealed in late breaking oral presentation during ASN Kidney Week



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 ≤150/90 mmHg

Endpoints

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

ET = end of treatment; RAASi = renin-angiotensin-aldosterone system inhibitor.

Demographics and baseline characteristics¹

	Atacept treated participants ² n=113
Age, median (range), y	37 (18, 67)
Male sex, n (%)	67 (59)
Race, n (%)	
White	59 (52)
Asian	51 (45)
Native Hawaiian or Other Pacific Islander	1 (1)
Other/not reported	2 (2)
eGFR, mean \pm SD, mL/min/1.73 m ²	62 \pm 28
UPCR by 24h urine, mean \pm SD, g/g	1.8 \pm 1.3

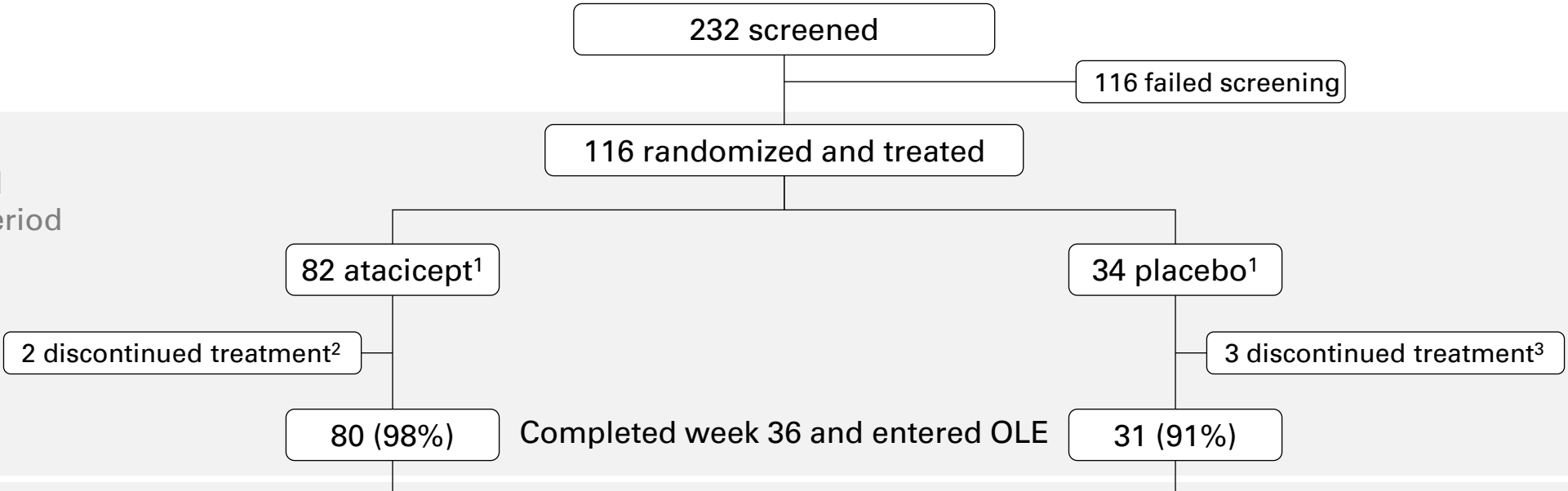
SD = standard deviation.

1. Baseline is defined as the last available measurement prior to the first dose of atacept.

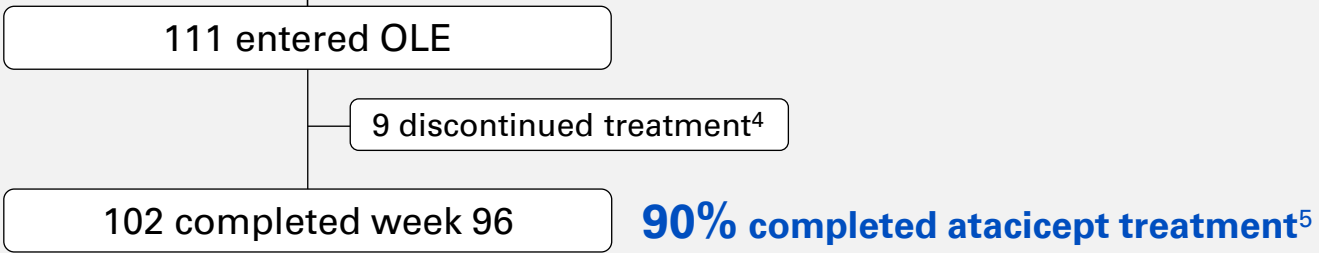
2. Atacept group includes all participants receiving any atacept dose at any timepoint.

Participant disposition through 96 weeks

Randomized double-blind treatment period



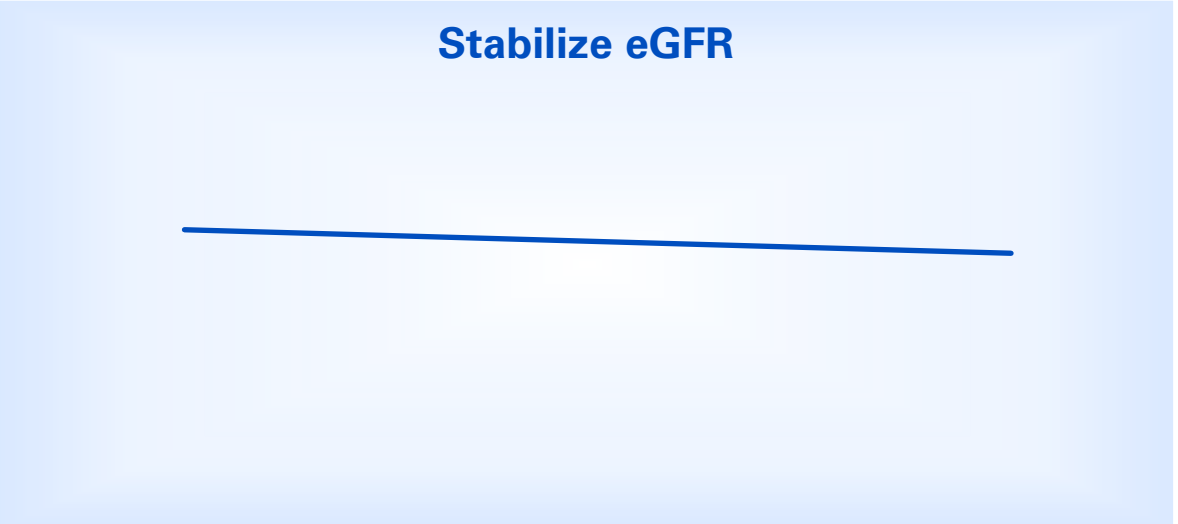
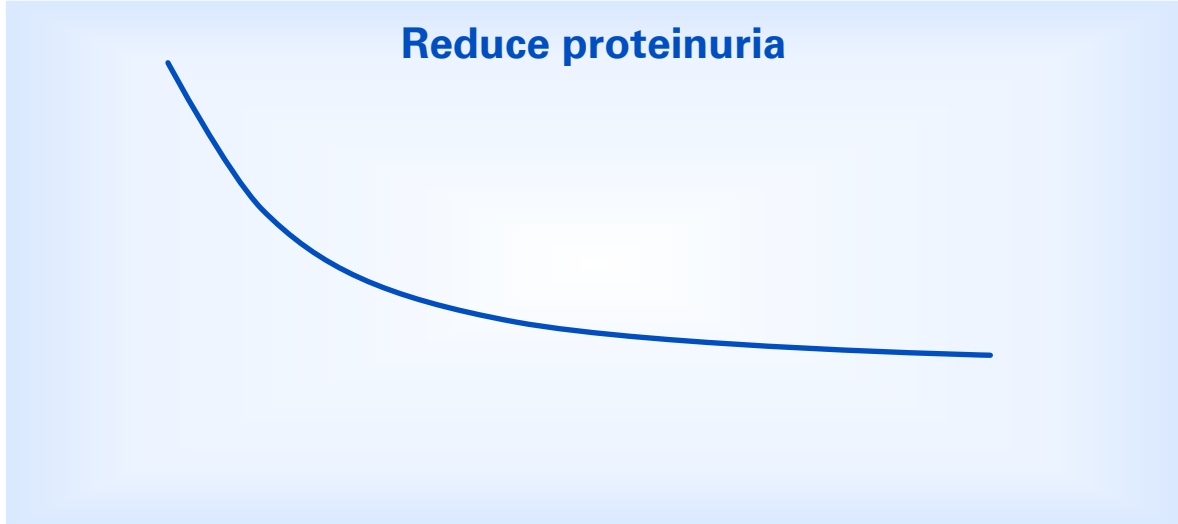
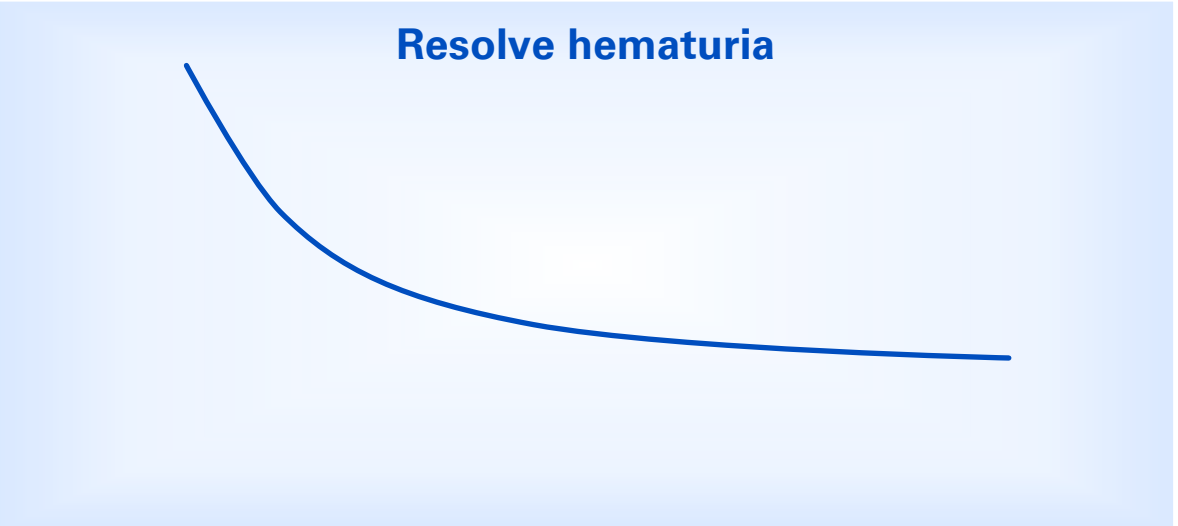
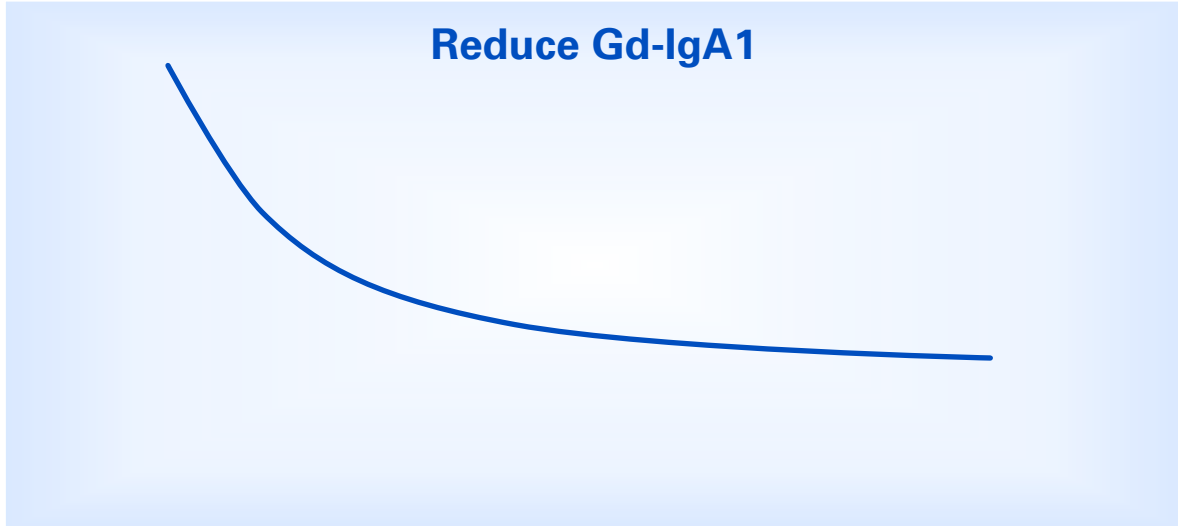
OLE period



OLE = open-label extension.
 1. Full analysis set and safety population.
 2. Discontinued to pursue elective surgery (n=1), and discontinued due to positive hepatitis B DNA and adverse event (n=1).
 3. Initiated prohibited medication for concomitant disease (n=1), discontinued due to plan to start prohibited medication for concomitant disease (n=1) and adverse event (n=1).
 4. Discontinued due to investigator decision (n=1), pregnancy (n=2), participant withdrawal (n=2), surgery (n=1), serious adverse event of pneumonia in a heavy smoker, resolved (n=1), adverse event of worsening alanine aminotransferase and aspartate aminotransferase (n=1), and medical monitor criteria (n=1).
 5. 90% = 102/113 (out of the 116 randomized and treated participants, 3 discontinued placebo prior to week 36).

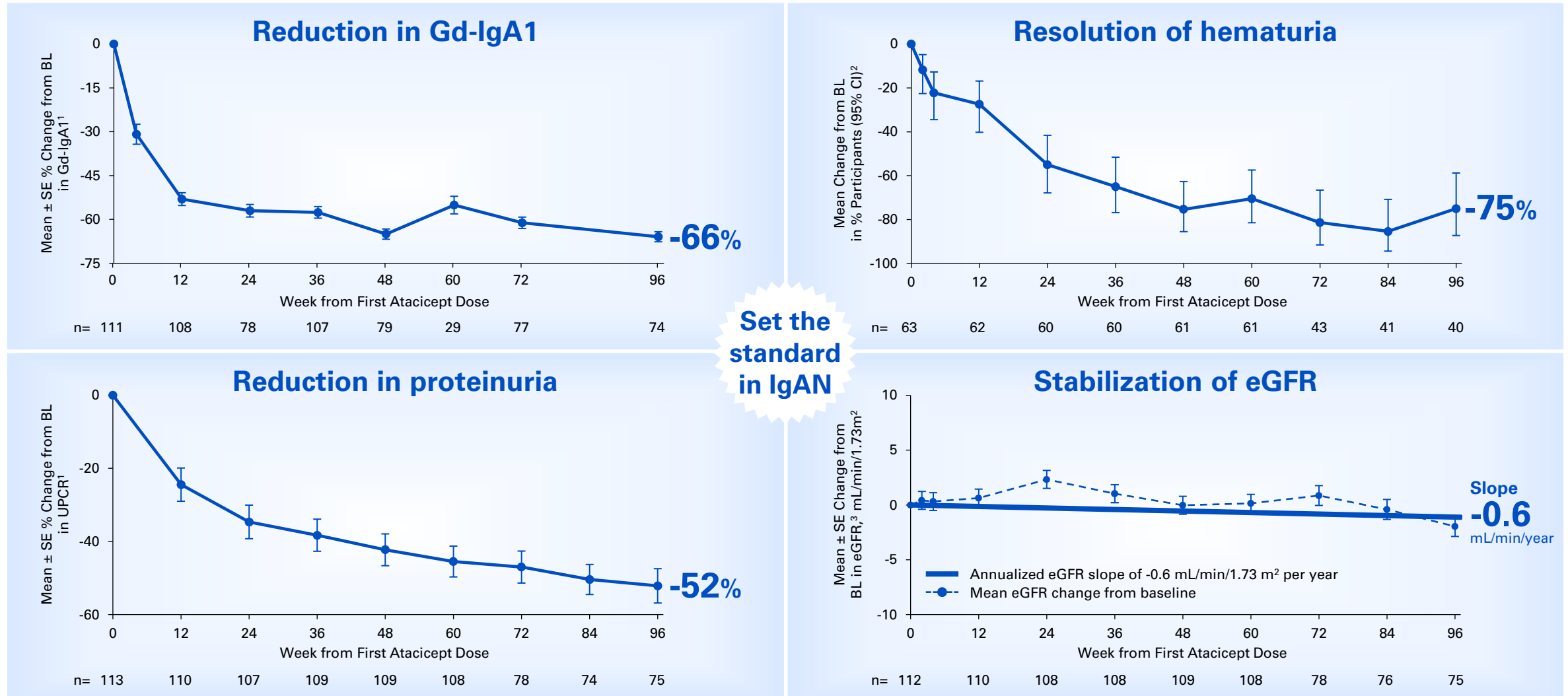


We believe an ideal IgAN disease modifying therapy would be expected to...



ORIGIN Phase 2b 96-week results consistent with IgAN disease modification

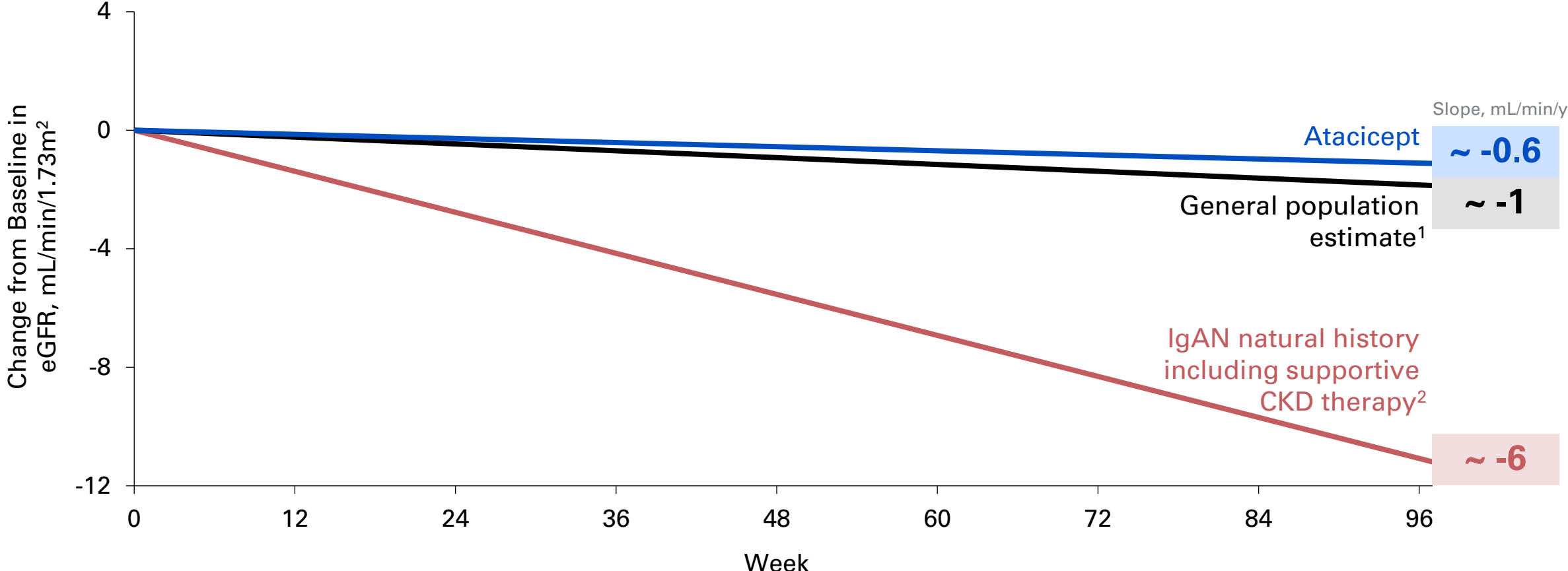
Including eGFR profile consistent with the general population of -1 mL/min/year



Set the standard in IgAN

Atacept group includes all participants receiving any atacept dose at each timepoint, with baseline (BL) defined as the last available measurement prior to the first dose of atacept. Data from weeks 0 to 60 includes participants who switched from placebo to atacept. 1. Percentage changes from BL computed using FDA-endorsed mixed-effects modeling; 2. Percentages represent change from BL in number of participants with hematuria at each visit divided by number with BL hematuria; 3. Changes from BL in eGFR were analyzed using MMRM analysis and LS estimation and SE were estimated from the model directly; eGFR slope was analyzed using mixed-effects model with random intercept and random slope and mean slope and SE were estimated from the model directly.

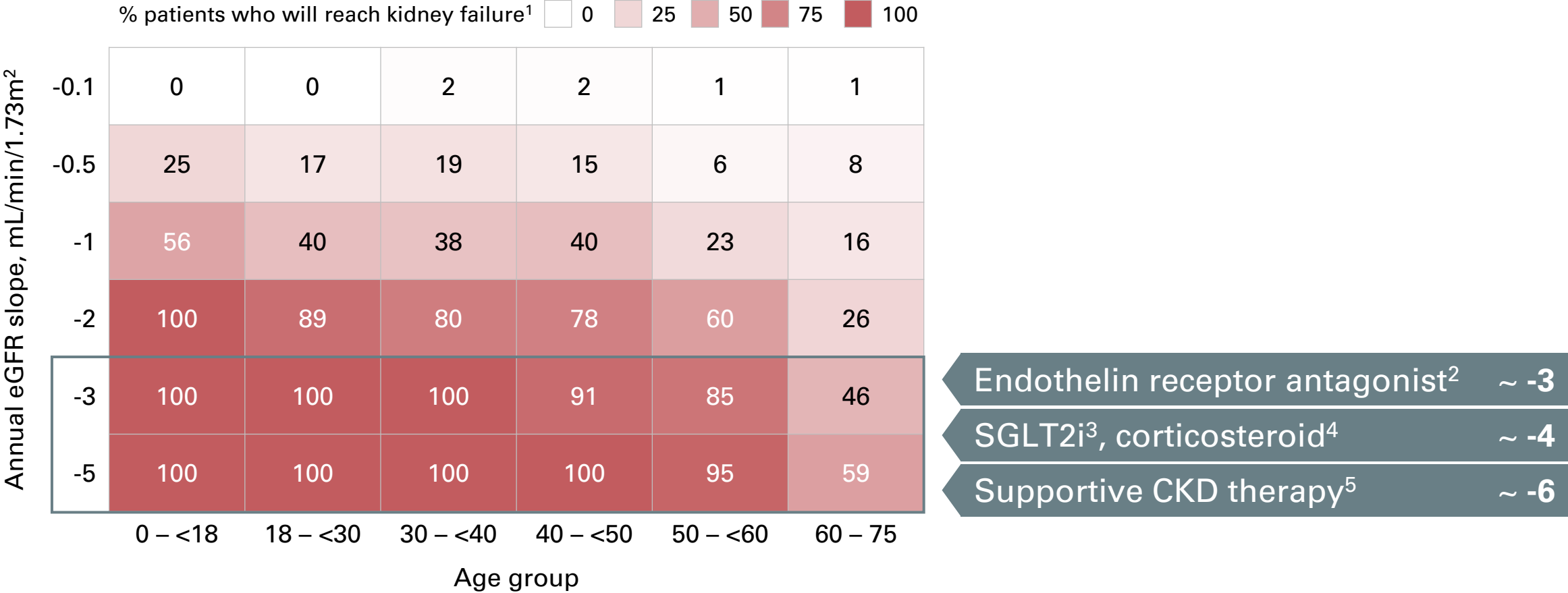
Atacicept treated participants have eGFR slope profile consistent with general population without kidney disease



This data is based on a cross-trial comparison and not a head-to-head clinical trial; such data may not be directly comparable due to differences in study protocols, conditions and patient populations. Projected eGFR trajectories for general population and IgAN natural history do not represent clinical data and assume a constant eGFR slope over time. CKD = chronic kidney disease.
1. Slope estimate from Baba M, et al. PLOS ONE 2015; 2. Average historical placebo slope from 9 clinical trials³⁻¹¹; 3. Lafayette R, et al. Lancet 2023; 4. Rovin BH, et al. Lancet 2023; 5. Li PK-T, et al. Am J Kidney Dis 2006; 6. Manno C, et al. Nephrol Dial Transplant 2009; 7. Lv J, et al. JAMA 2017; 8. Wheeler DC, et al. Kidney Int 2021; 9. Lv J, et al. JAMA 2022; 10. Zhang H, et al. ASN Kidney Week 2023, poster TH-PO1123; 11. Mathur M, et al. N Engl J Med 2023.

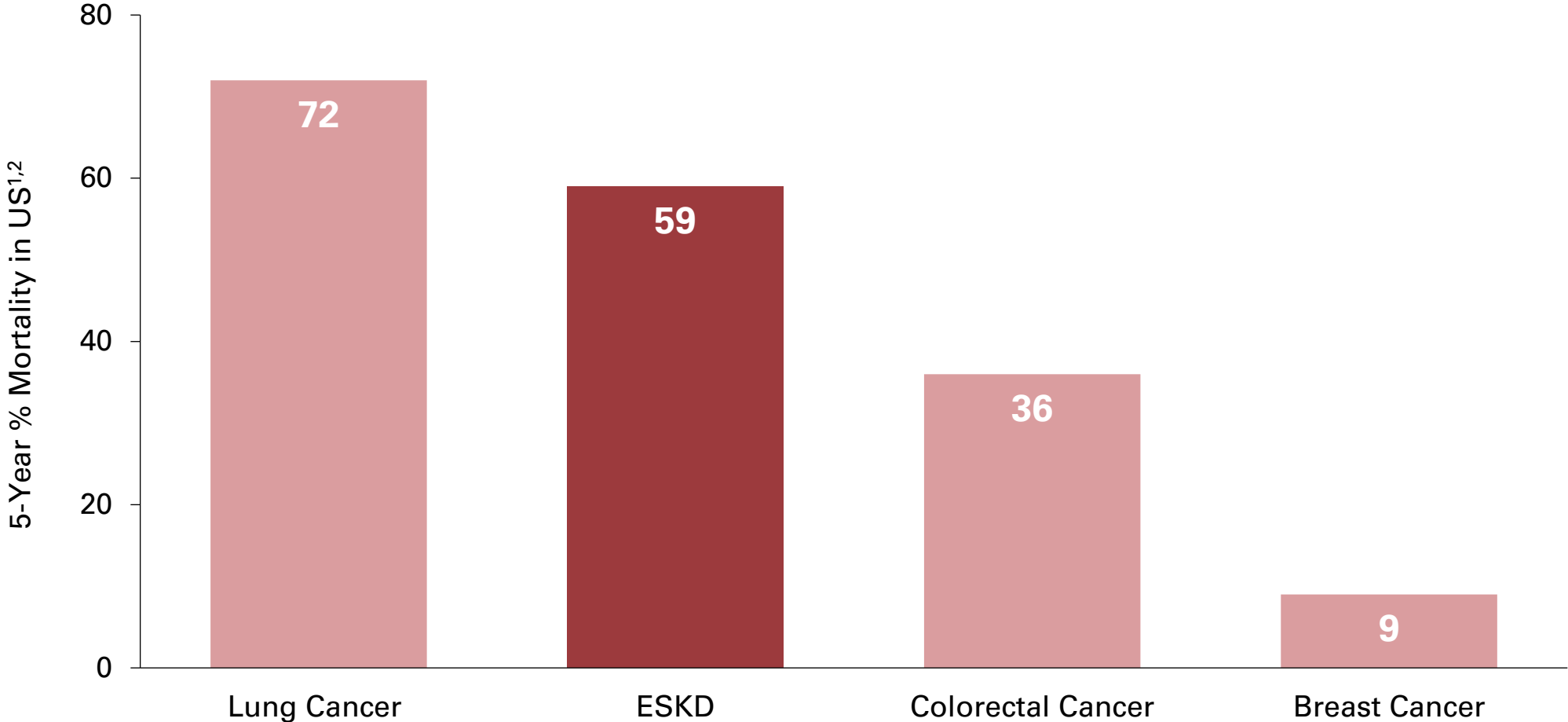


2024 Draft KDIGO IgAN guidelines call for target eGFR slope ≤ -1 mL/min/1.73m²



1. Adapted from Pitcher D, et al. CJASN 2023. eGFR slope data from approved therapies: 2. Rovin BH, et al. Lancet 2023; 3. Wheeler DC, et al. Kidney Int 2021; 4. Lafayette R, et al. Lancet 2023; 5. Average historical placebo (including chronic kidney disease standard of care) data from 9 clinical trials: Li PK-T, et al. Am J Kidney Dis 2006; Manno C, et al. Nephrol Dial Transplant 2009; Lv J, et al. JAMA 2017; Wheeler DC, et al. Kidney Int 2021; Lv J, et al. JAMA 2022; Zhang H, et al. ASN Kidney Week 2023, poster TH-PO1123; Mathur M, et al. N Engl J Med 2023; Lafayette R, et al. Lancet 2023, Rovin BH, et al. Lancet 2023.

5-year mean mortality in ESKD comparable to cancer in US



1. US CDC Cancer Statistics; 2. Thurlow JS, et al. Am J Nephrol 2021.

Atacicept generally well tolerated through 96 weeks: OLE adverse events profile consistent with randomized period

	Double-Blind Baseline to Week 36		Open-Label Extension Week 36 to 96 ¹
	Placebo n=34	All Atacicept n=82	Atacicept 150 mg n=111
Participants, n (%)			
TEAEs	28 (82)	60 (73)	85 (77)
Infections and infestations	11 (32)	35 (43)	43 (39)
Study drug-related TEAEs ²	14 (41)	42 (51)	52 (47)
Serious TEAEs ³	3 (9)	2 (2)	12 (11)
TEAEs leading to study drug discontinuation ⁴	1 (3)	1 (1)	2 (2)
Deaths	0	0	0

- Total participant exposure: median 96 weeks (range 3, 99); mean 91 weeks

TEAE = treatment-emergent adverse event.

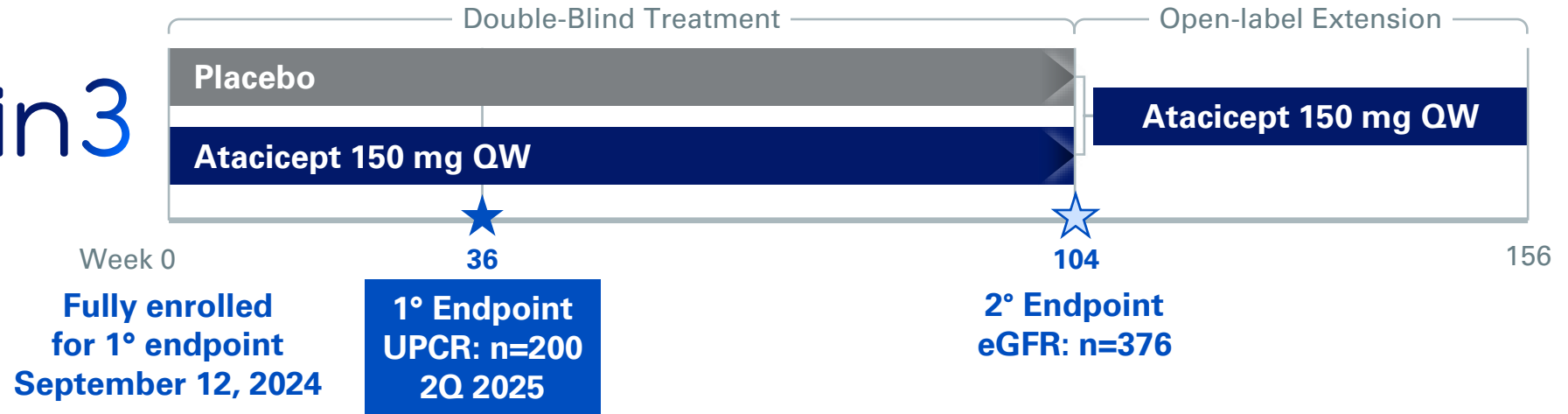
1. Week 96 cut-off includes all safety data as of June 03, 2024, including visits past Week 96. AEs were considered treatment-emergent during the open-label extension period if they started after the first dose of open-label atacicept 150 mg through the end of the trial. n=111 represents 80 atacicept and 31 placebo who entered the open-label extension.

2. Mostly injection site reactions.

3. Serious TEAEs during double-blind period were previously reported (Lafayette R, et al. *Kidney Int.* 2024;S0085-2538(24)00236-9); serious TEAEs during the OLE: excess abdominal fat and left basal bronchopneumonia (n=1), acute kidney injury (n=1), angioedema (n=1), termination of pregnancy (n=1), post cricoid ulcer (n=1), pancreatitis, passed out common bile duct stone, and acute cholecystitis (n=1), tonsillitis (n=1), pneumonia (n=1), acute coronary syndrome required hospitalization (n=1), left 5th metatarsophalangeal joint gout (n=1), mild flare of IgA nephropathy (n=1), and urethral stricture worsening (n=1).

4. Reasons for discontinuation during double-blind period were previously reported; discontinuations during the OLE were due to: pneumonia in a heavy smoker, resolved (n=1); and worsening alanine aminotransferase and aspartate aminotransferase, resolved and unrelated to study treatment (n=1).

Consistency with ORIGIN 2b instills confidence in ORIGIN 3



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/1.73 m²
- Blood pressure ≤150/90 mmHg

Key Endpoints

- Primary efficacy: UPCR-24h at week 36 ★
to support potential accelerated approval
 - >90% power at week 36
- Key secondary: eGFR change up to week 104 ★
 - 90% power for eGFR Δ 4 mL/min at week 104
- Safety

- Operational efficiency leveraging similar trial design and worldwide sites as ORIGIN 2b
- Same self-administered SC formulation and dose as used in ORIGIN 2b

Atacicept expansion roadmap

US prevalence estimates



Atacicept

~160K¹

**origin
and
Expanded IgAN**

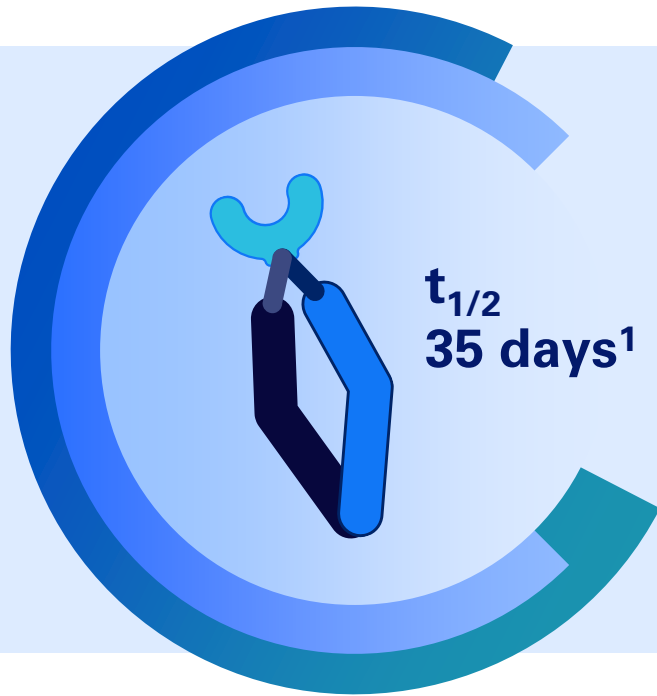
1. Vera Therapeutics corporate estimates for peak year prevalence based on ClearView Healthcare Partners Analysis.

ORIGIN Extend: Commitment to providing long-term access to atacicept for all ORIGIN participants



- Phase 2 extension study in participants who complete ORIGIN 2b/3; initiating Q4 2024
- Objectives:
 1. Provide patients with extended access to atacicept prior to commercial availability in their country/region
 2. Capture longer-term data for research purposes
 3. Generate data from reinitiation of atacicept treatment following off-treatment period

Atacicept at home, self-administered QW dosing highly attractive; QM program begins in 2025



- Biologic therapies utilizing at home, self-administered, SC 1 mL QW dosing widely used and accepted
- This dosing paradigm has the potential to support atacicept as a foundational therapy for IgAN
- Atacicept's half life also supports evaluation of extended dosing
- QM dose finding study planned in 2025

1. Willen D, et al. Eur J Drug Metab Pharmacokinet 2020;45(1):27-40.

PIONEER: Phase 2 basket trial in expanded IgAN cohorts

Patients ineligible for ORIGIN 3 will have an opportunity to enroll in PIONEER at same clinical sites



Expanded IgAN populations, n ≤120

- 1 Adult IgAN with low kidney function¹, n ≤20
- 2 Adult IgAN with low proteinuria², n ≤50
- 3 Adult IgAN with high proteinuria³, n ≤20
- 4 Adolescent⁴ IgAN at high risk of progression⁵, n ≤10
- 5 Adult recurrent IgAN post kidney transplant, n ≤10
- 6 Adolescent⁴ and adult IgAVN, n ≤10

¹eGFR 20 to <30 mL/min/1.73 m²

²UPCR <1.0 g/g

³UPCR ≥5.0 g/g

⁴Age ≥15 years

⁵UPCR ≥0.3 g/g

Atacicept 150 mg QW



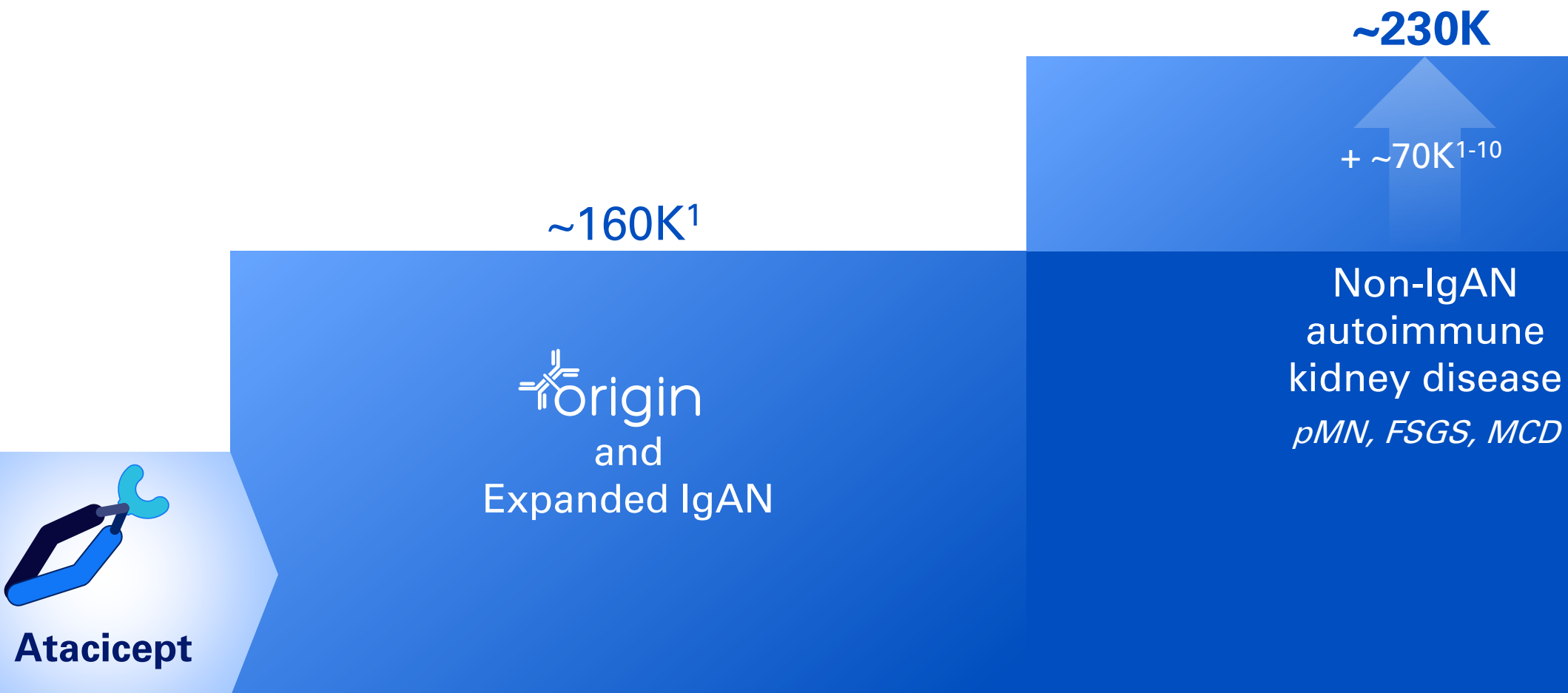
Key Endpoints

- Primary: UPCR change at week 36
- Key secondary: eGFR change at weeks 36, 52
- Gd-IgA1 change at weeks 36, 52
- Change in percentage of participants with hematuria at weeks 36, 52
- Safety

IgAVN = immunoglobulin A vasculitis nephritis (purpura nephritis).

Atacicept expansion roadmap

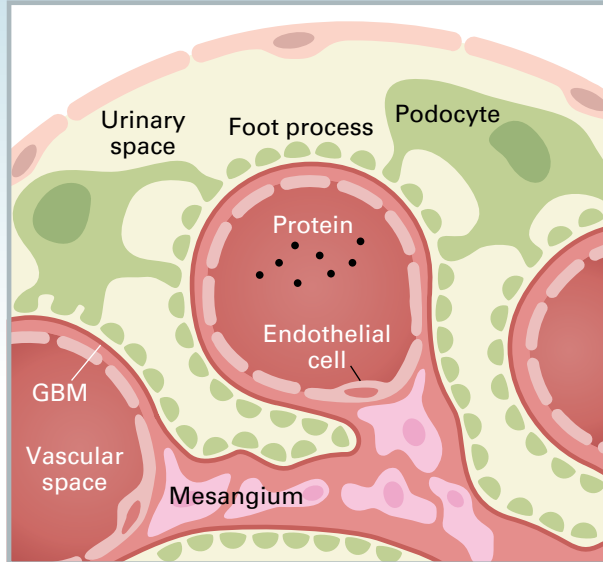
US prevalence estimates



Vera Therapeutics corporate estimates for peak year prevalence based on 1. ClearView Healthcare Partners Analysis; 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.

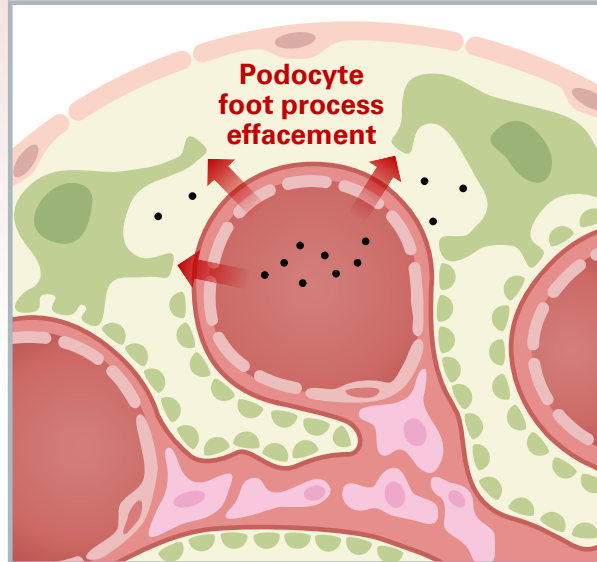
Autoimmune glomerular disease: podocyte injury and cytoskeletal derangement drives proteinuria and progressive disease

Healthy Podocyte Foot Processes



Podocytes play a key role in preventing large molecules (proteins) from being filtered into urine

Disrupted Podocyte Foot Processes

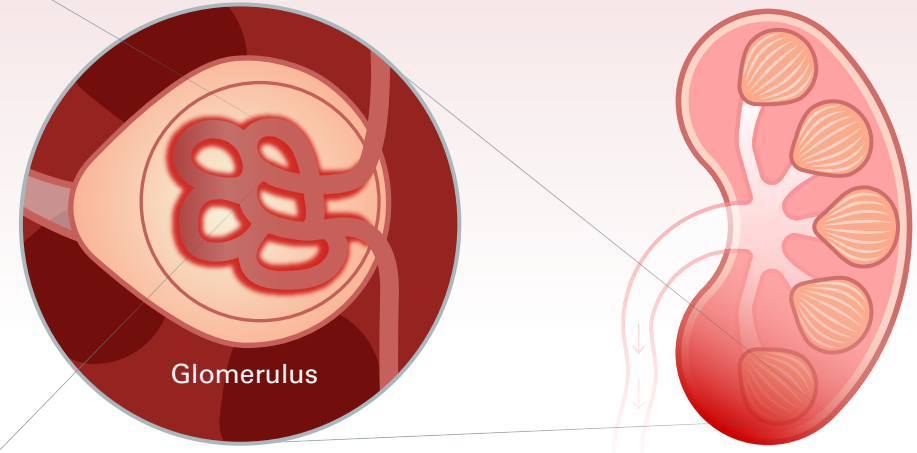


Causes of podocyte injury:

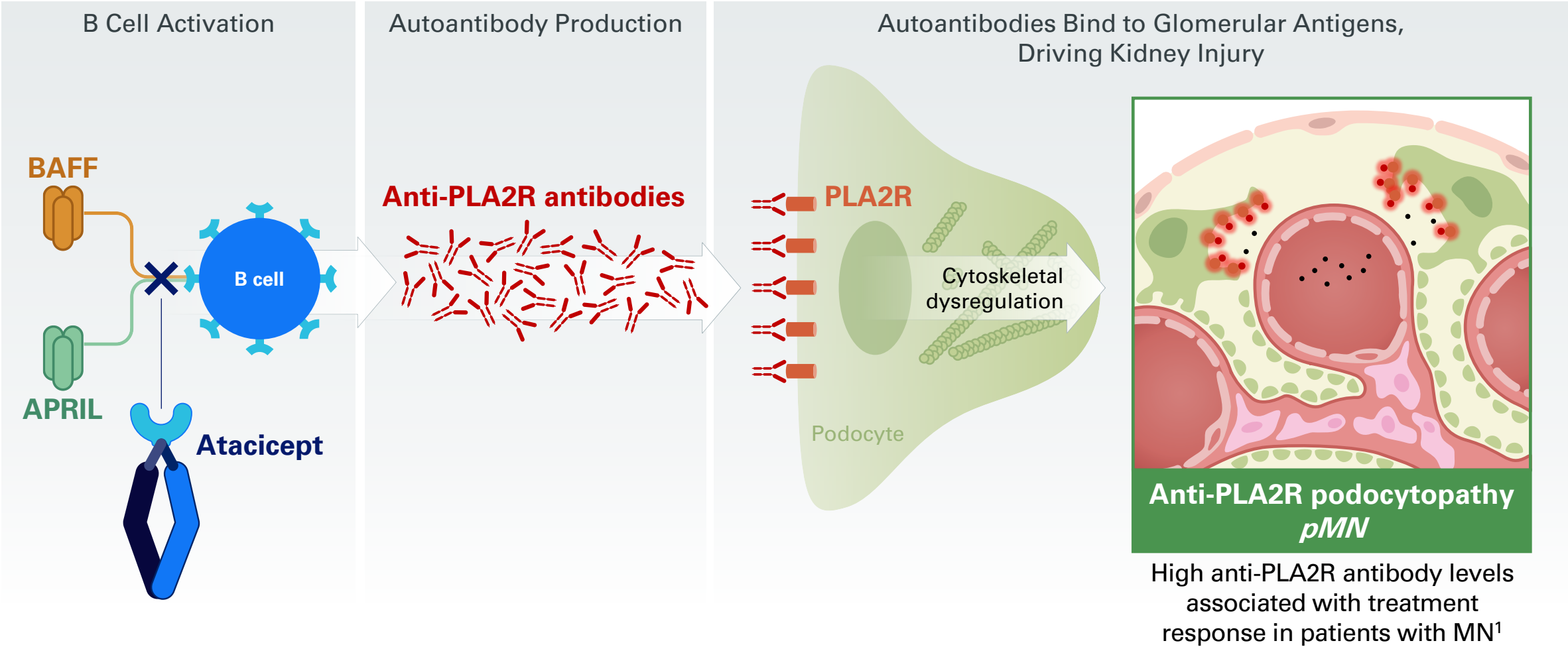
- Immune mediated injury
- Secondary causes
- Genetic predisposition
- Environmental factors

Resulting in clinically relevant alterations in the glomerular filtration barrier...

...leading to proteinuria including nephrotic syndrome and nephron loss



Atacicept mechanism of action has broad potential in autoimmune glomerular disease including membranous nephropathy



PLA2R = phospholipase A2 receptor.
1. Barbour SJ, et al. CJASN 2023.

Atacicept expansion roadmap

US prevalence estimates



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PIONEER: Operationally efficient Phase 2 basket trial in expanded IgAN and anti-PLA2R podocytopathy (PMN)



Population 1, $n \leq 120$
Expanded IgAN populations

Population 2, $n \leq 20$
Anti-PLA2R podocytopathy
(Membranous Nephropathy)

Atacicept 150 mg QW

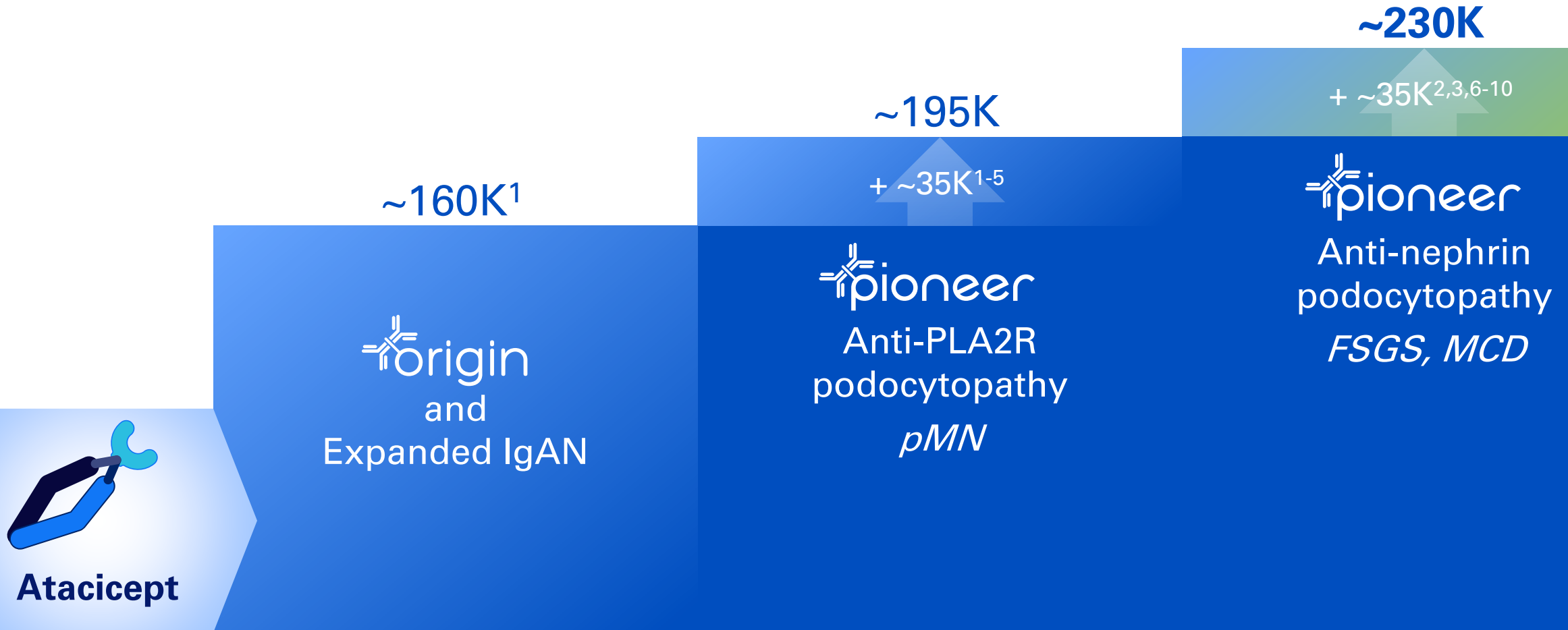


Key Endpoints

- Primary: UPCR change at week 36
- Key secondary: eGFR change at weeks 36, 52
- Exploratory:
 - Gd-IgA1 change at weeks 36, 52
 - Change in percentage of participants with hematuria at weeks 36, 52
 - Change in anti-PLA2R antibodies
- Safety

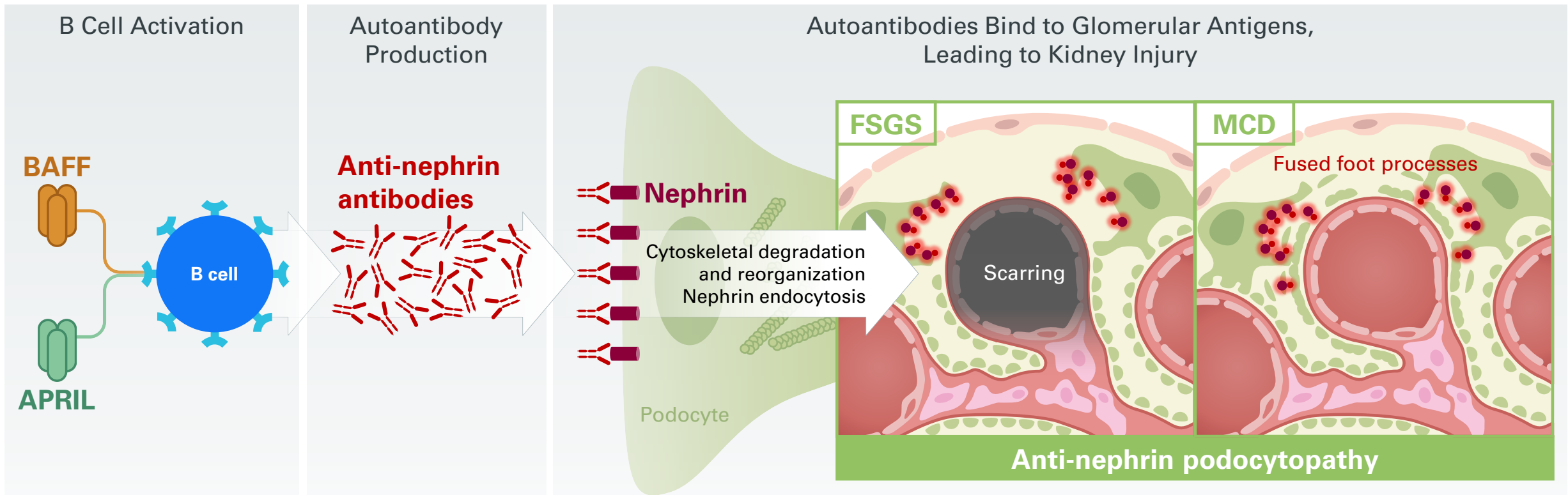
Atacicept expansion roadmap

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FSGS and MCD are histologic diagnoses with heterogeneous etiology; Autoimmunity, including anti-nephrin antibodies, is one driver of disease



FSGS = focal segmental glomerulosclerosis; MCD = minimal change disease. Kopp JB, et al. Nat Rev Dis Primer 2021; Fogo AB. Nat Rev Nephrol 2015.

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



Population 1, $n \leq 120$
Expanded IgAN populations

Population 2, $n \leq 20$
Anti-PLA2R podocytopathy
(Membranous Nephropathy)

Population 3, $n \leq 20$
Anti-nephrin podocytopathy
(Minimal Change Disease/FSGS)

Atacicept 150 mg QW

Week 0

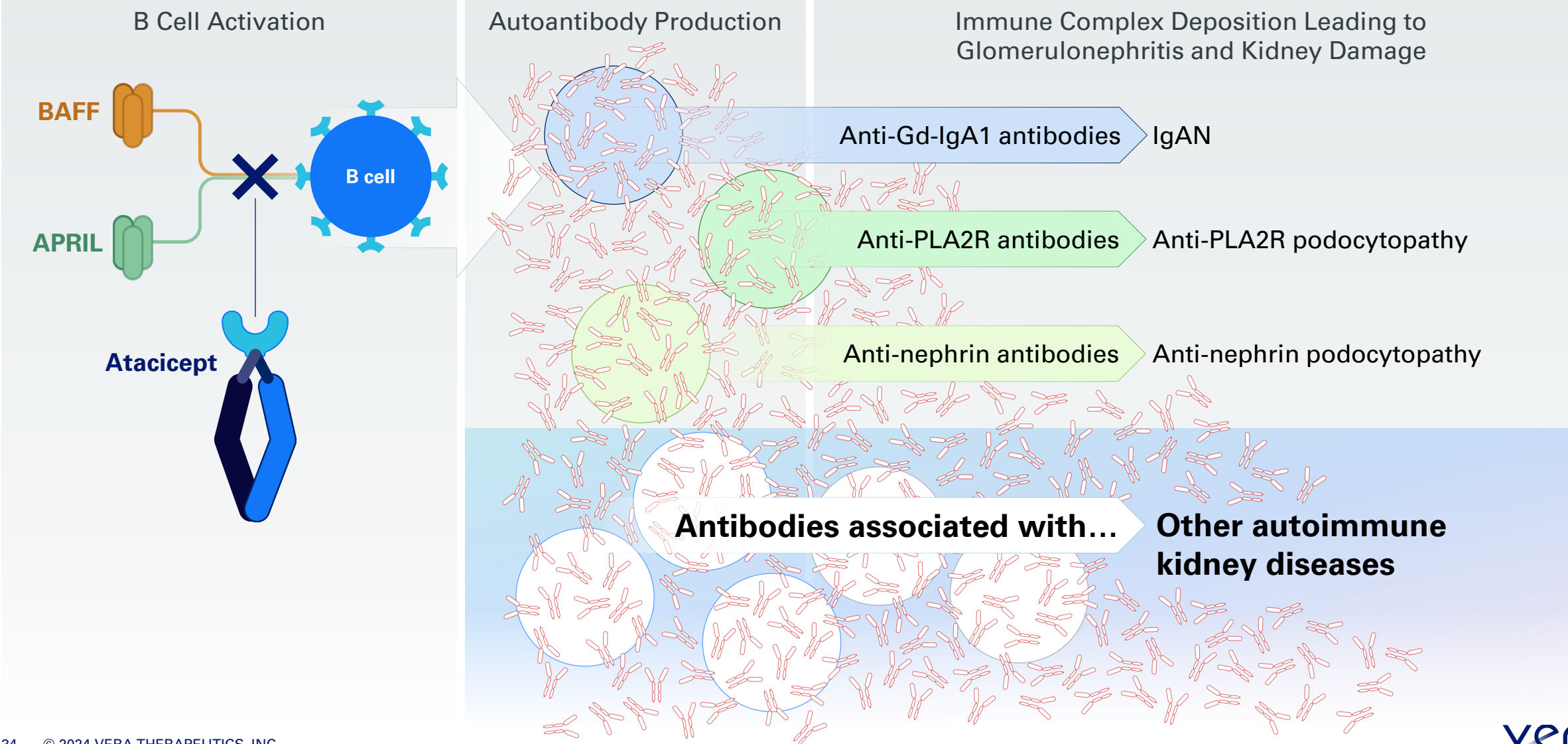
36
1° Endpoint

52
2° Endpoint

Key Endpoints

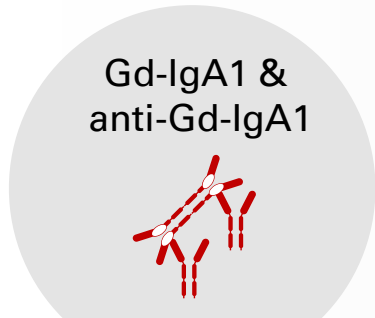
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 - Change in anti-nephrin antibodies
- Safety

Targeting B cell production of autoantibodies against glomerular antigens offers the potential of additional kidney indications



Vision for an evolved approach to autoimmune glomerular disease

Identification of autoantigen/autoantibody constructs that drive autoimmune glomerular diseases



Gd-IgA1 &
anti-Gd-IgA1

1999¹



PLA2R &
anti-PLA2R

2009²



Nephrin &
anti-nephrin

2022³

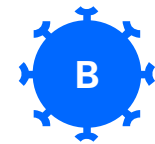
Today

**Future
autoantigen/autoantibody
constructs identified**

Importantly, atacicept represents both a **potential therapeutic agent** and also a **diagnostic tool**:

Characterize patients with proteinuric and nephritic conditions based on responsiveness to a diagnostic trial of atacicept

Patients demonstrating a response have a **B-cell modulatory responsive autoimmune glomerular disease**



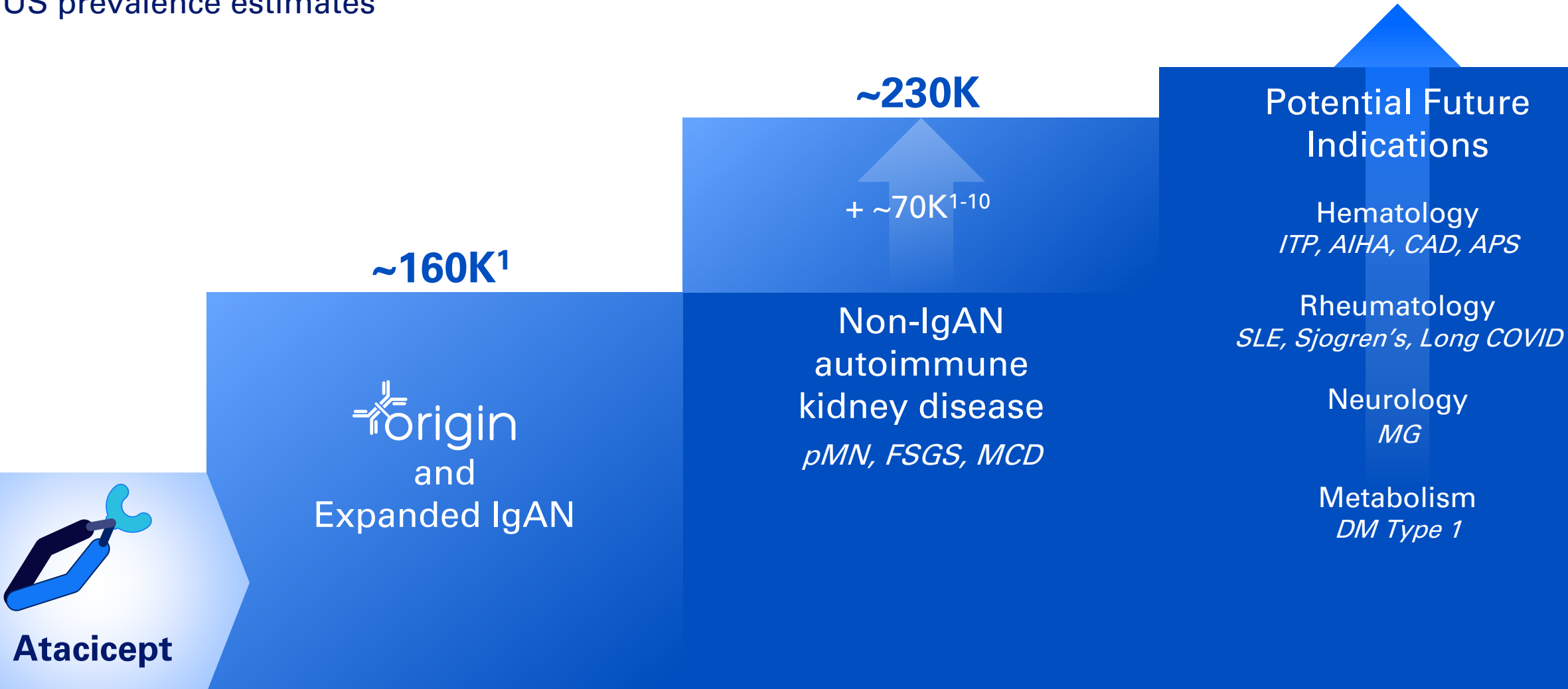
Does not require pre-existing elucidation of all autoantigen/autoantibody constructs

Provides an opportunity for both future clinical investigation and long-term treatment

1. Tomana M, et al. J Clin Invest 1999. 2. Beck LH, et al. N Engl J Med 2009. 3. Watts AJ, et al. JASN 2022.

Vera optionality to expand in autoimmune kidney disease & beyond




US prevalence estimates



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 pMN = primary membranous nephropathy; FSGS = focal segmental glomerulosclerosis; MCD = minimal change disease; ITP = immune thrombocytopenia; AIHA = autoimmune hemolytic anemia; CAD = cold agglutinin disease; APS = antiphospholipid syndrome; SLE = systemic lupus erythematosus; MG = myasthenia gravis; COVID = Coronavirus disease 2019; DM = diabetes mellitus.



Atacicept projected catalysts

Catalyst	2024	2025	2026	
	Phase 3 primary endpoint cohort full enrollment	✓ 3Q		
	Phase 2b 96-week results	✓ 4Q		
	Phase 3 top-line results		● 2Q	
	BLA submission		● 2H	
	Projected US launch ¹			●
	Initiation	●		
	Initial data available		●	
	Initiation		●	
	Initial data available		●	

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

Based on management’s current assumptions. 1. Subject to US approval.



vera

therapeutics™