vera therapeutics™

Corporate Presentation

January 2025

Forward-looking statements

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Atacicept potentially *first* and *best-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; FDA Breakthrough Therapy Designation awarded 2024
- Only program with 2-year data in Phase 2 → potential for commercial differentiation, if approved
- Only investigational drug with at home self admin of 1 mL SC QW and 90% patient retention at 2 years

Potential Indication Expansion

- B cell modulation represents a treatment paradigm shift for autoimmune diseases
- Progressive expansion in addressable patients: initial autoimmune kidney disease opportunity >200K
- Strong clinical potential in hematologic, rheumatologic, and other indications

Resourced for Potential Launch

- Currently ~$677M *pro forma* cash, cash equivalents and marketable securities as of September 30, 2024[1]
- Management focused on potential for successful commercial launch
- Phase 3 read out on track for Q2 2025; if successful, anticipated PDUFA date 2026

APRIL = A proliferation inducing ligand; BAFF = B cell activating factor; 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 supports best-in-class potential

	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 (Phase 2) 150 mg SC QW (Phase 3) 1x1 mL self-administered	2/4/8 mg/kg IV (Phase 2) 400 mg SC QM (Phase 3) 1x2 mL in-clinic injection	80/240 mg SC QM (Phase 1b) 80 mg SC QM (Phase 3) 1xTBD mL in-clinic injection	450 mg IV Q2W (Phase 2) 600 mg SC Q2W (Phase 3) 2x2 mL in-clinic injection
Development Stage	Phase 3	Phase 3	Phase 3	Phase 3
Randomized Controlled Trial Data	✓	✓	X	X
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=8	19 months, n=33
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. Phase 2 4 mg/kg IV Gd-IgA1 data from Mathur M, et al. NEJM 2023, Phase 2 4 mg/kg IV hematuria data from Barratt J, et al. WCN 2024, WCN24-AB-1799, Phase 2 pooled sibeprenlimab UPCR data from Kooienga ASN 2022, TH-PO991, and eGFR data from Barratt J, et al. ASN 2023, abstr TH-PO1124; 2. Phase 1b 80 mg data from Madan A, et al. ASN 2024, FR-PO854; 3. Barratt J, et al. ASN 2024, FR-PO856.

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

BAFF/APRIL inhibition

~160K[1]

origin
and
Expanded IgAN

~230K

+ ~70K[1-10]

Non-IgAN autoimmune kidney disease

PMN, FSGS, MCD

Potential Future Indications

Hematology
ITP, AIHA, CAD, APS

Rheumatology
SLE, Sjogren's, Long COVID

Neurology
MG

Metabolism
DM Type 1

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

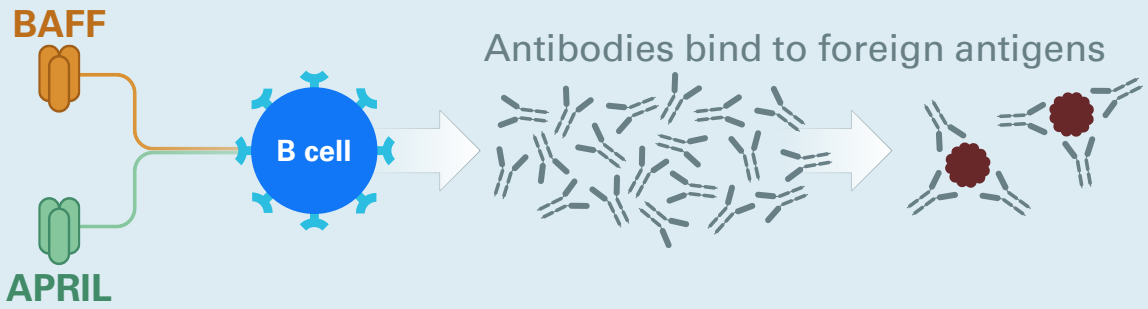
		Catalyst	2025	2026
origin	IgAN	Phase 3 full enrollment	2Q	
		Phase 3 primary endpoint	2Q	
		BLA submission	2H	
		Projected US launch[1]		
origin extend	IgAN	Initial data		
pioneer	IgAN, PMN, FSGS, MCD	Initiation		
		Initial data		

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

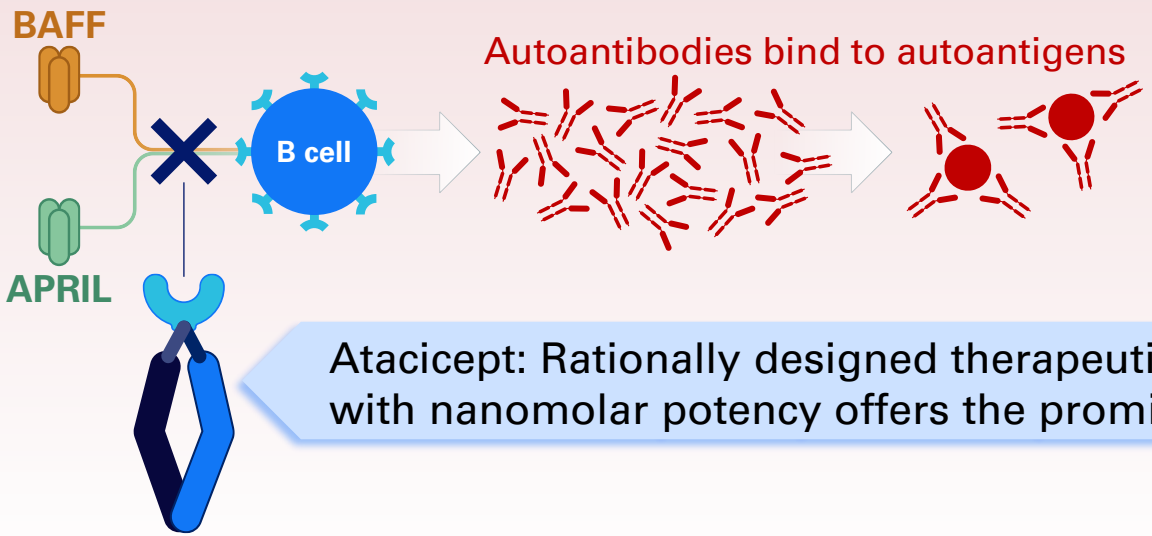
Based on management's current assumptions. 1. Subject to US approval.

Dual BAFF/APRIL inhibition has broad therapeutic potential to address multiple autoimmune diseases

Immunity in health



Autoimmune disease

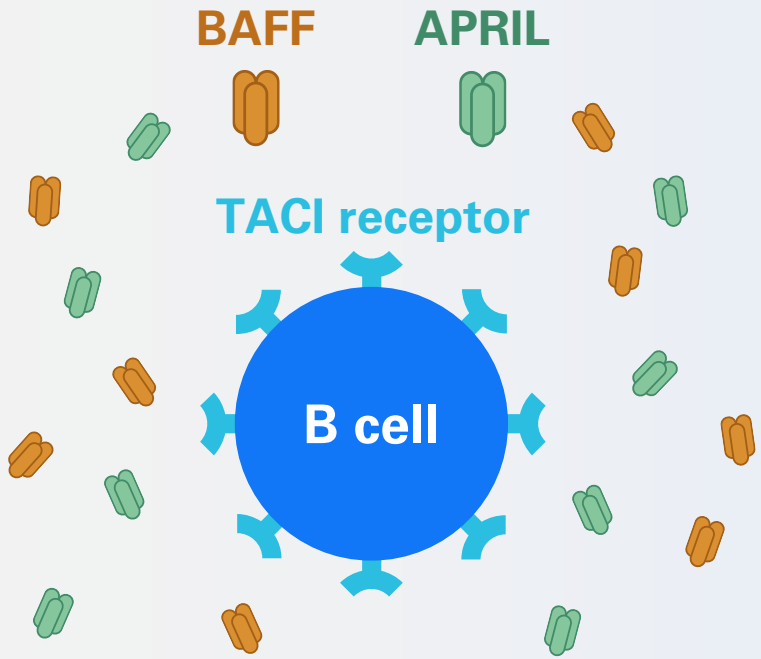


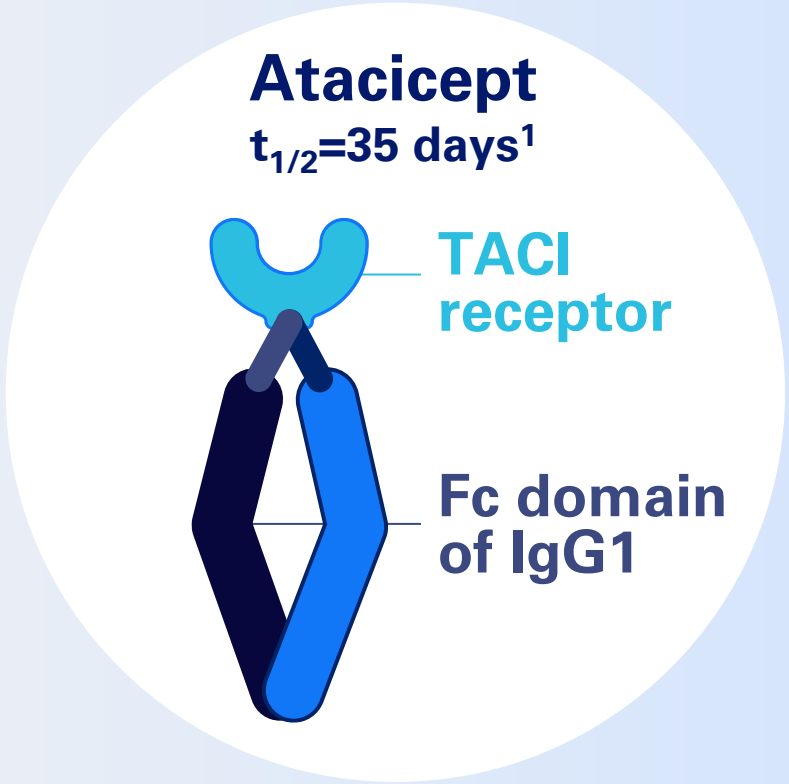
- Autoantigens and autoantibodies mediate autoimmune disease
- B cells source of autoantibodies → target cell of interest for therapeutic intervention
- B cells fueled by two cytokines, BAFF and APRIL

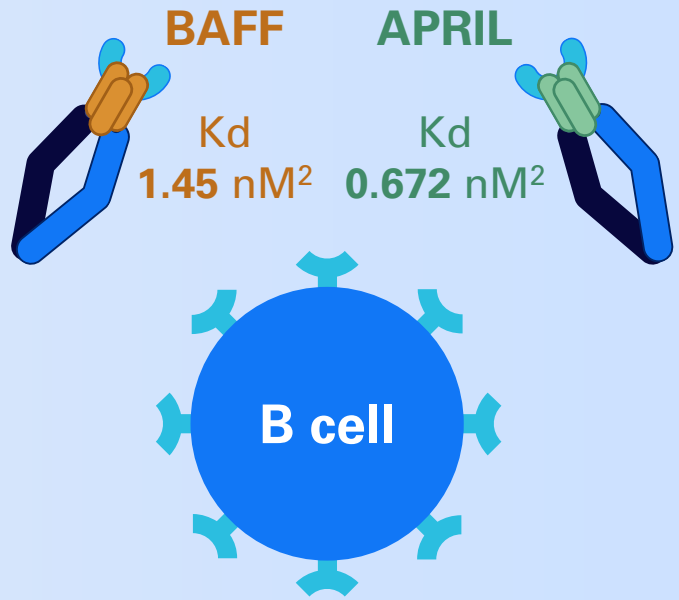
Atacicept: Rationally designed therapeutic of modern biotechnology that binds BAFF and APRIL with nanomolar potency offers the promise of precision modulation of B cells and autoantibodies

Atacicept is an example of rational drug design

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



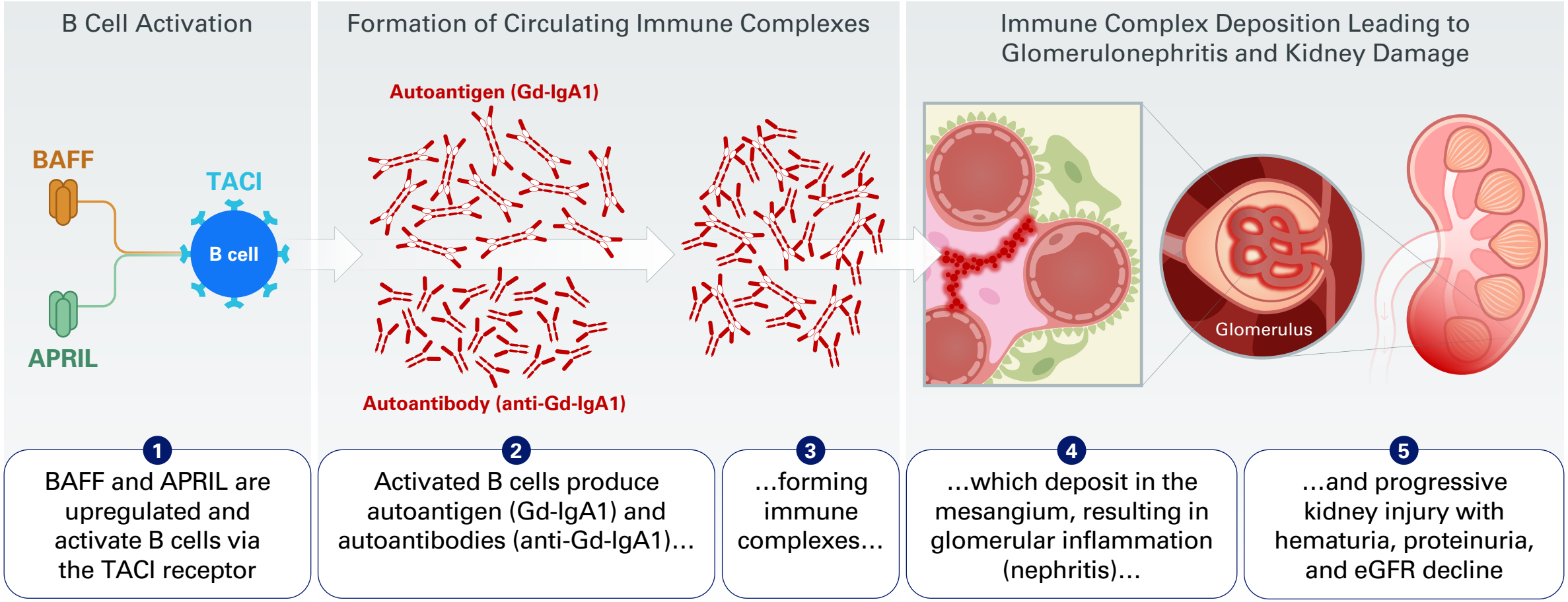




Fc = fragment crystallizable; IgG1 = immunoglobulin G1; Kd = dissociation constant; TACI = transmembrane activator and calcium-modulator and cyclophilin ligand.

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

IgAN Prevalence: ~0.04% of US Pop (360.5M)[1]

~160K pts

High Risk (~56% of pts)[2]	Moderate Risk (~20%)[2]	Low Risk (~24%)[2]
~88K pts	**~31K pts**	**~38K pts**

ORIGIN Ph3 Population

Potential near-term expansion opportunity supported by ORIGIN Ph2b Results

Diagnosis and Treatment Paradigm

IgAN Natural History

IgAN-specific disease pathophysiology
→ **Turn off faucet of immune complex generation *with atacicept,* if approved**

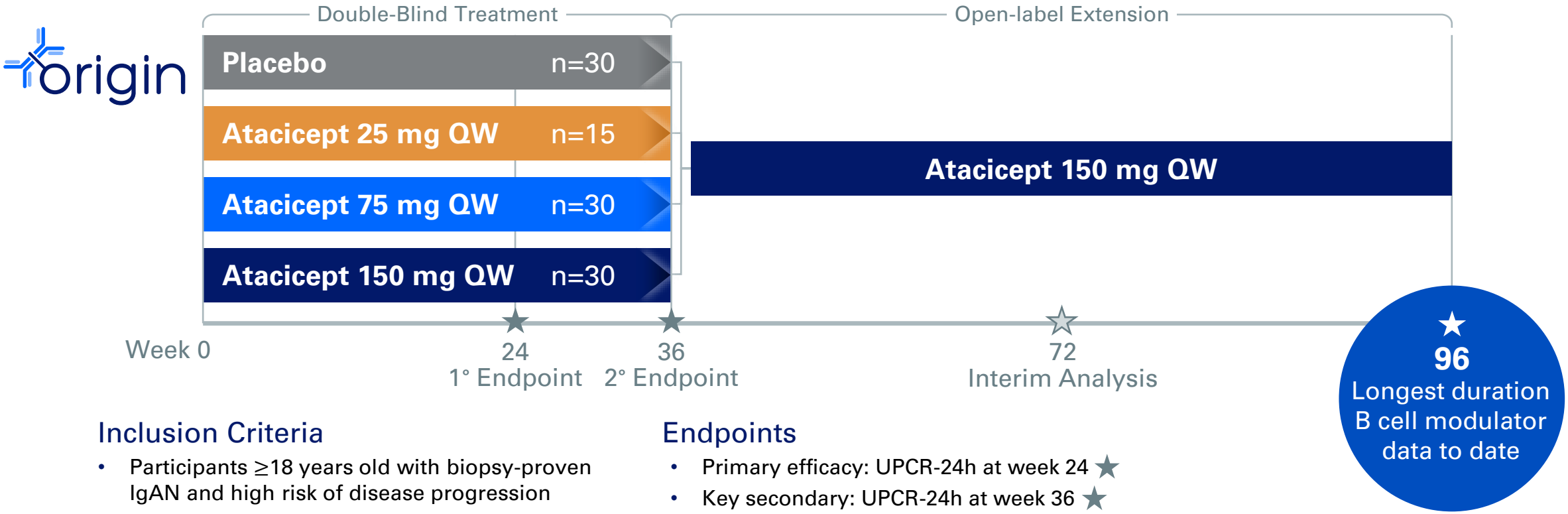
Non-IgAN-specific mechanism of nephron loss
→ **Treat CKD *with RAS and SGLT2 inhibition*[3]**

Treatment Considerations

- **~50%** of patients receiving standard of care progress to ESKD[2]
- Patients with high proteinuria and/or hematuria often receive **immunosuppressive therapy**, frequently resulting in **adverse side effects with high dropout rates** in randomized controlled trials[4,5]
- **We believe in the initiation of disease modifying therapy with atacicept (dual BAFF/APRIL B cell modulation)** for prevalent IgAN patients, and first line disease modifying therapy for patients with a fresh biopsy
- Disease modifying therapy potentially **eliminates rationale for steroids and complement inhibitors**

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 at ASN Kidney Week and JASN manuscript



Inclusion Criteria

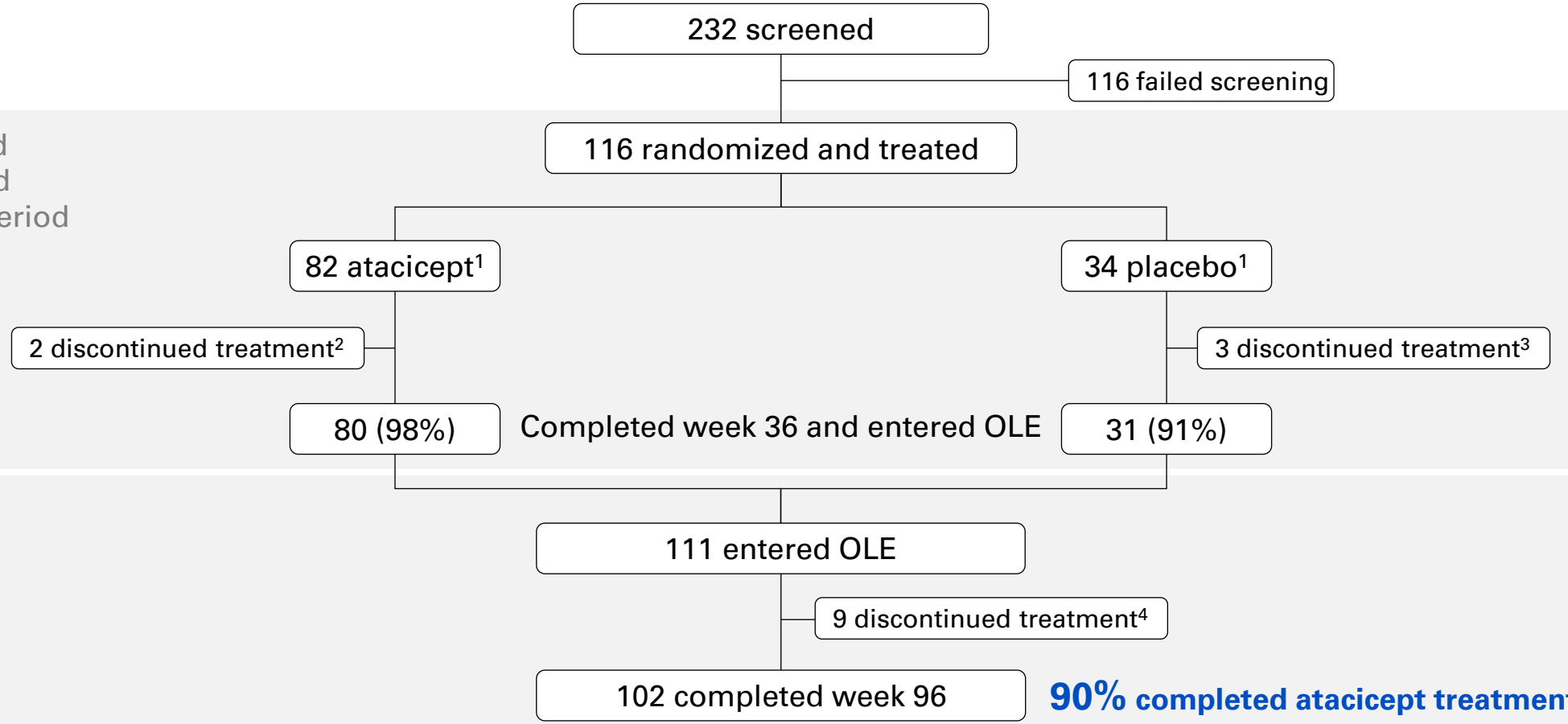
- Participants ≥18 years old with biopsy-proven IgAN and high risk of disease progression
- Stable and optimized RAASi for ≥12 weeks
- Use of SGLT2i allowed
- UPCR-24h >0.75 g/g or UP >0.75 g per 24h
- eGFR ≥30 mL/min/1.73 m^2
- Blood pressure ≤150/90 mmHg

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.

90% of participants completed atacicept treatment through 2 years



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…

Reduce Gd-IgA1

Resolve hematuria

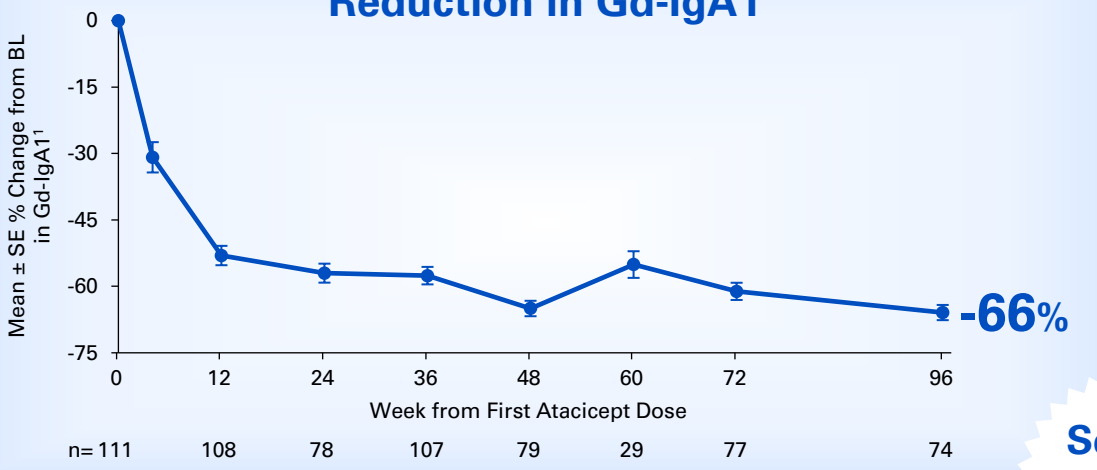
Reduce proteinuria

Stabilize eGFR

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

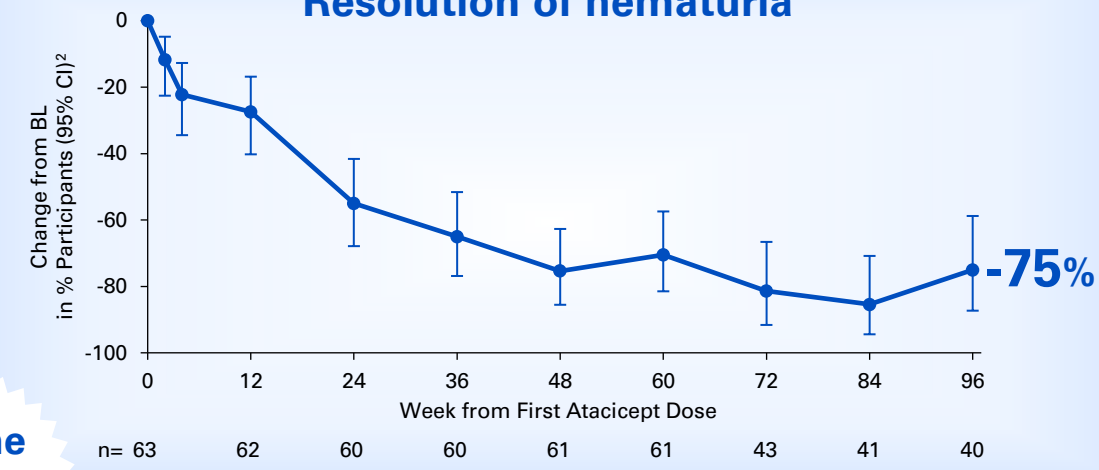
Including eGFR profile consistent with the general population of -1 mL/min/year

Reduction in Gd-IgA1



-66%

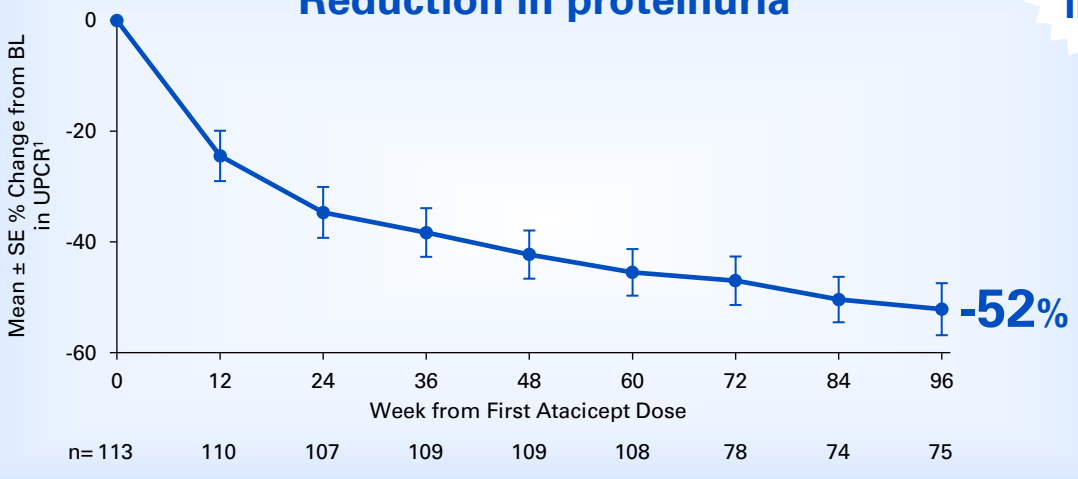
Resolution of hematuria



-75%

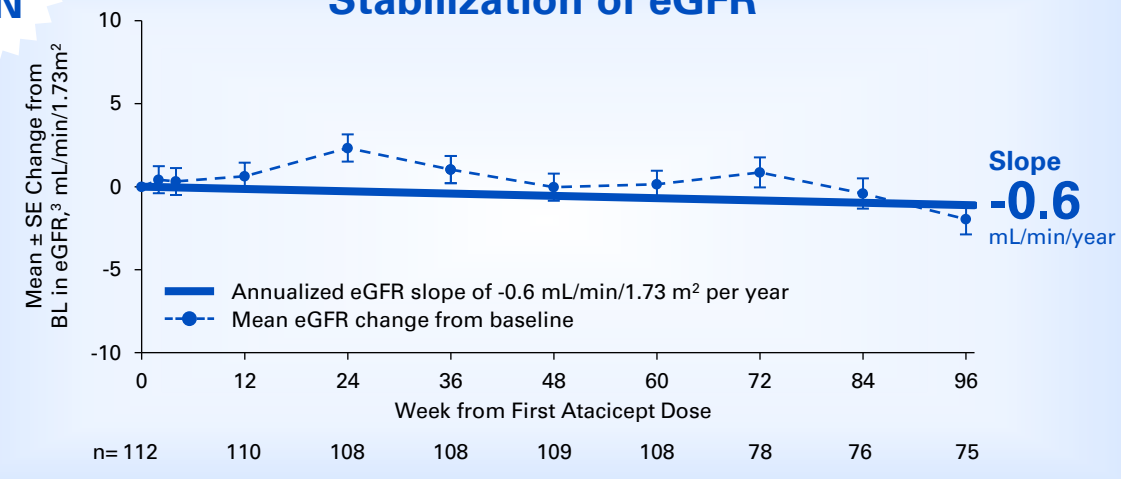
Set the standard in IgAN

Reduction in proteinuria



-52%

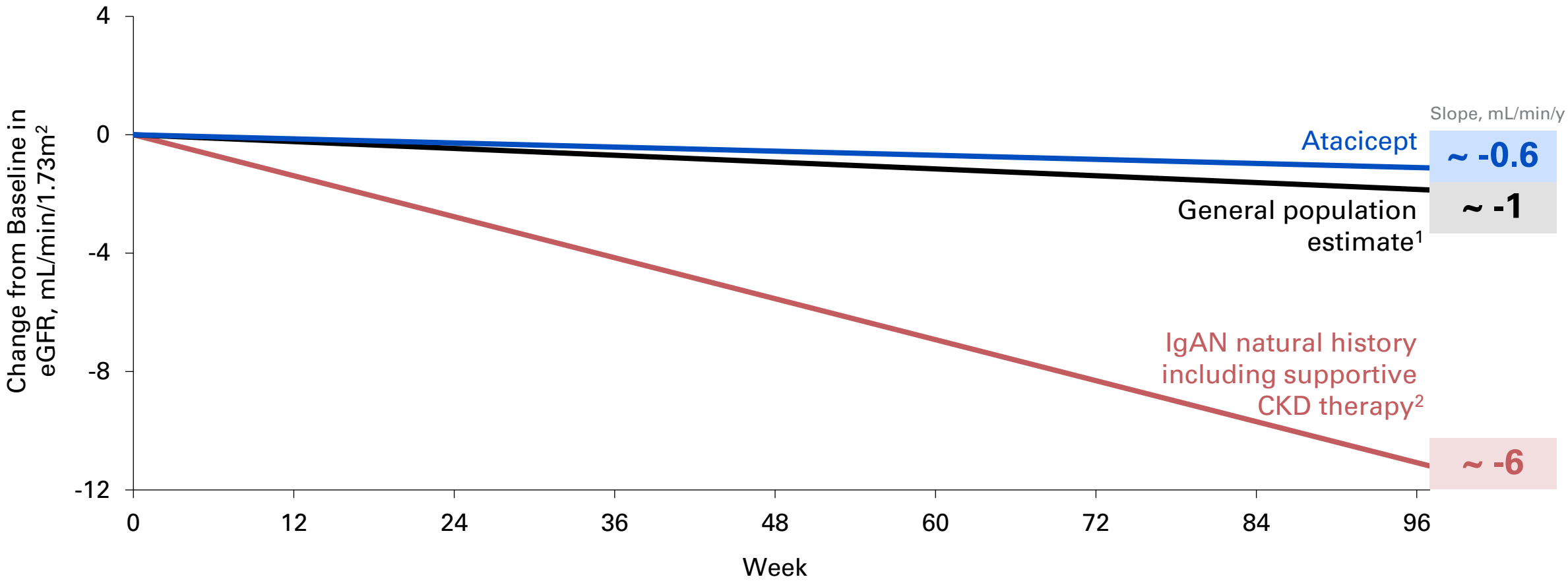
Stabilization of eGFR



Slope
-0.6
mL/min/year

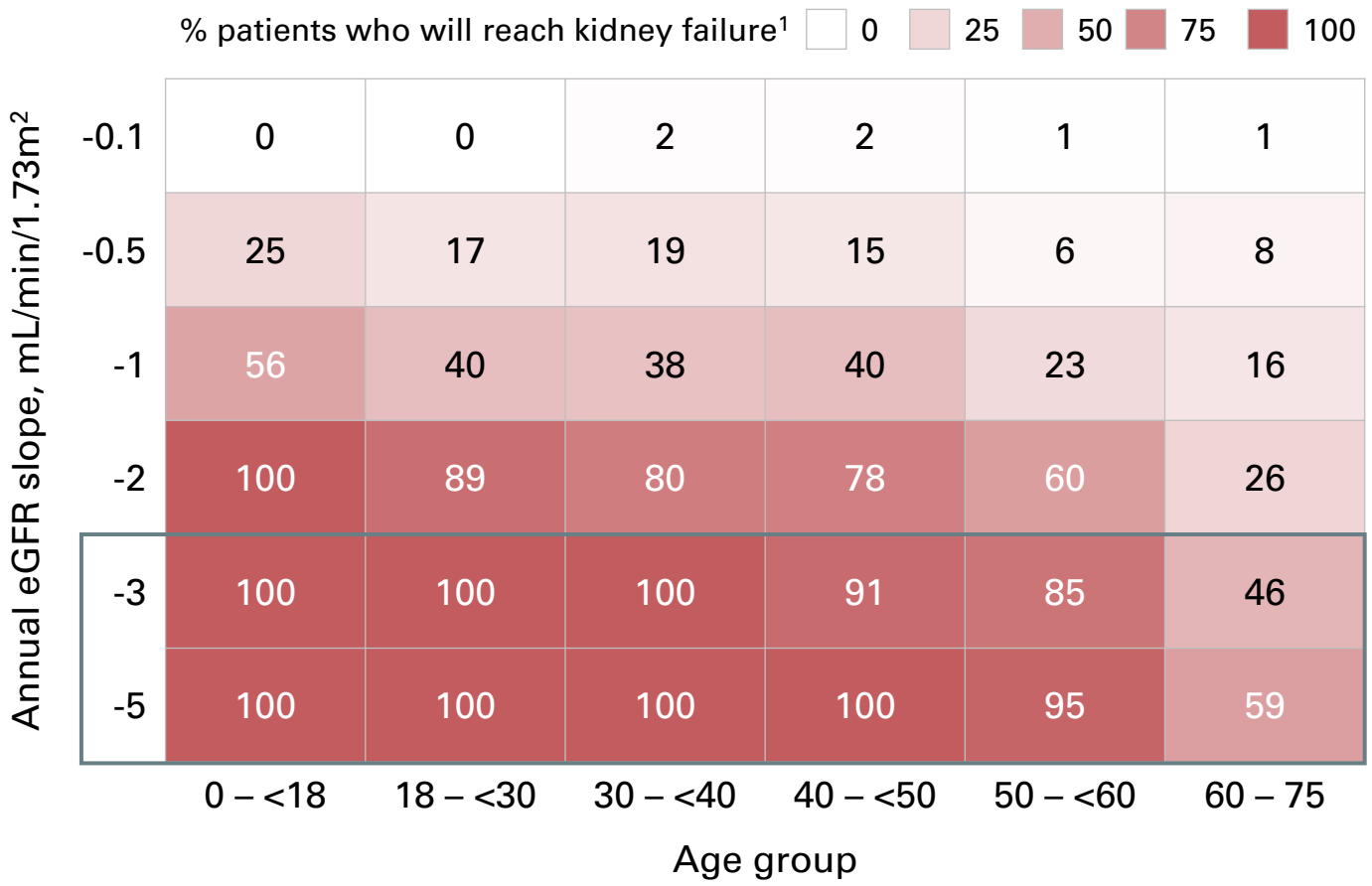
Atacicept group includes all participants receiving any atacicept dose at each timepoint, with baseline (BL) defined as the last available measurement prior to the first dose of atacicept. Data from weeks 0 to 60 includes participants who switched from placebo to atacicept.
1. Percentage changes from 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 trials[3-11]; 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/year



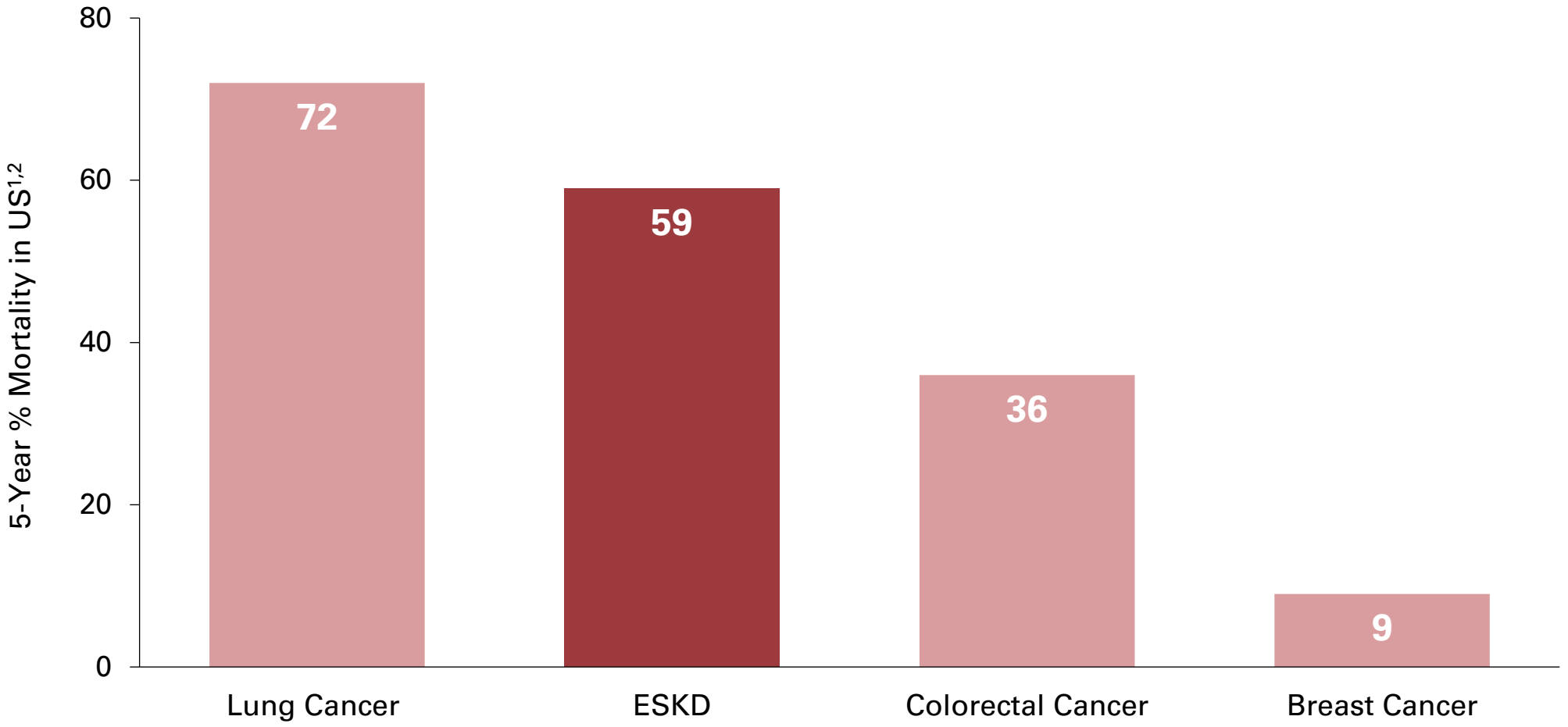
Endothelin receptor antagonist[2] ~ **-3**

SGLT2i[3], corticosteroid[4] ~ **-4**

Supportive CKD therapy[5] ~ **-6**

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]
Participants, n (%)	**Placebo n=34**	**All Atacicept n=82**	**Atacicept 150 mg n=111**
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

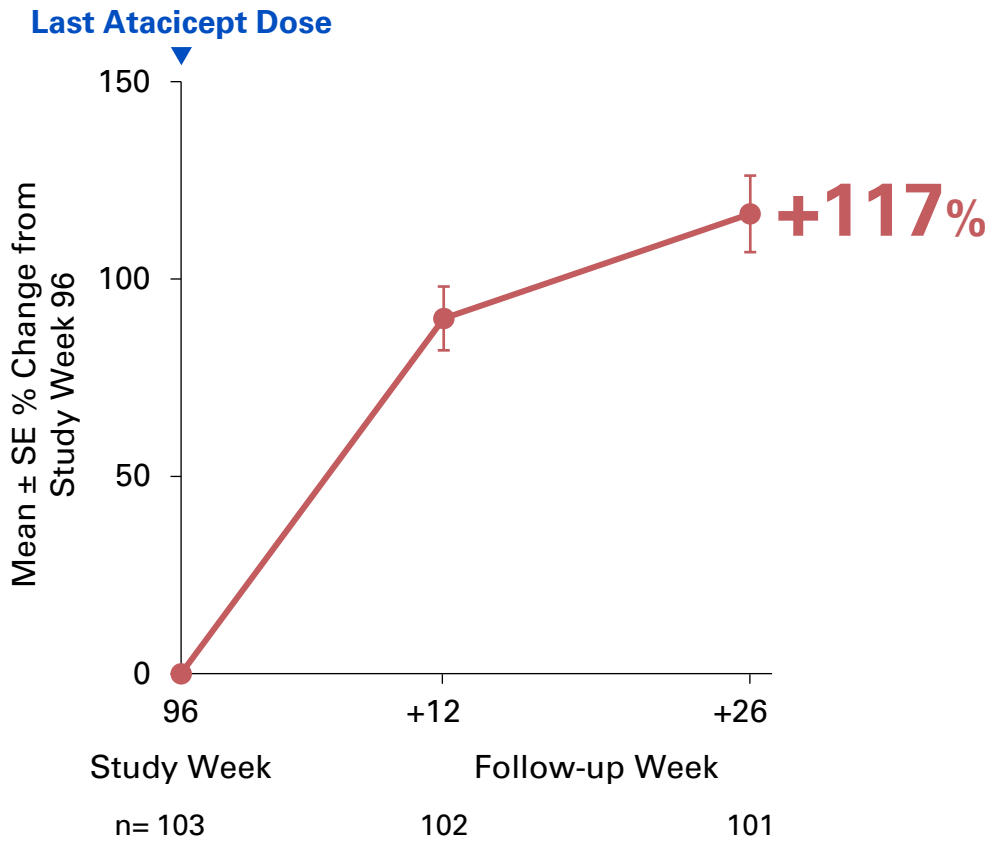
- Total participant exposure: median 96 weeks (range 3, 99); mean 91 weeks

TEAE = treatment-emergent adverse event.

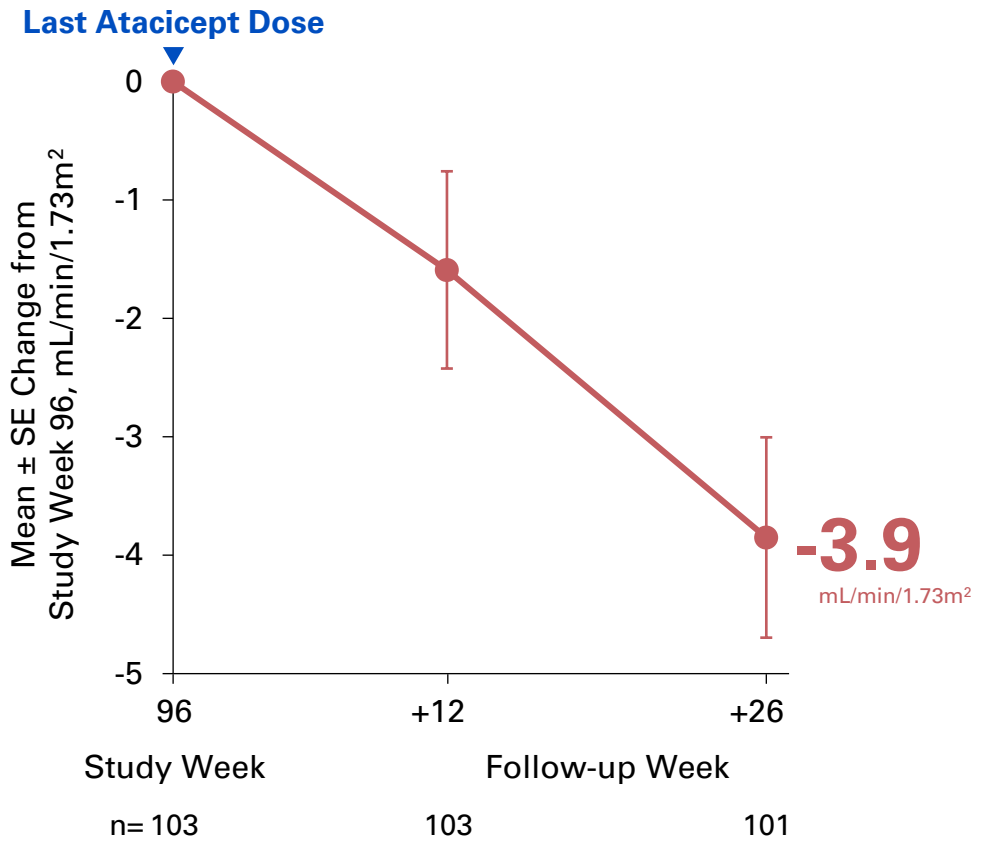
1. Week 96 cut-off includes all safety data as of June 03, 2024, including visits past Week 96. AEs were considered treatment-emergent during the open-label extension period if they started after the first dose of open-label atacicept 150 mg through the end of the trial. n=111 represents 80 atacicept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

Discontinuation of Atacicept resulted in pronounced increase in Gd-IgA1 and decrease in eGFR, potentially supporting a paradigm of chronic treatment

Gd-IgA1 % Change



eGFR Change



Analysis includes participants treated with atacicept who had a last on-treatment Gd-IgA1 or eGFR value in the study week 96 analysis window and at least one measure in the follow-up period, with the study week 96 values reset as the new baseline and analyzed along with the follow-up week 12 and 26 data. Gd-IgA1 % changes were computed using FDA-endorsed mixed-effects modeling. eGFR changes were analyzed using MMRM analysis and LS estimation and SE were estimated from the model directly.

Consistency with ORIGIN 2b instills confidence in ORIGIN 3

origin3

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 m^2
- 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

RASi = renin-angiotensin system inhibitor.

vera therapeutics

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
- 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 under way in 2025

- Biologic therapies utilizing at home, self-administered, SC 1 mL QW dosing have shown high compliance
- 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

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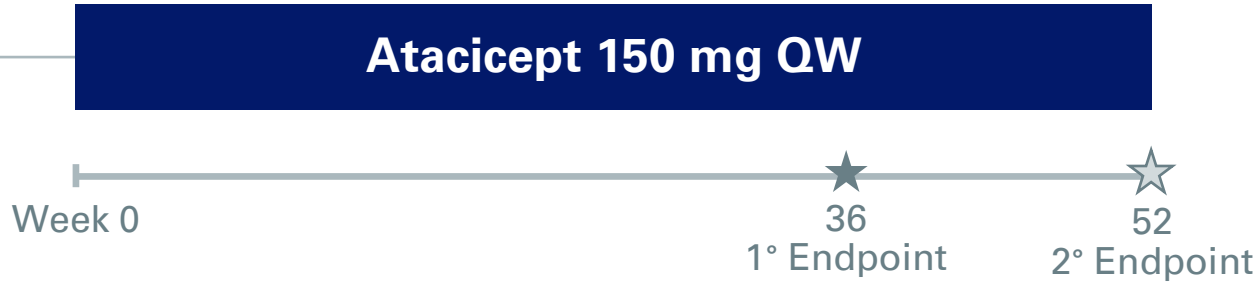
Expanded IgAN populations, n ≤120

1. Adult IgAN with low kidney function[1], n ≤20
2. Adult IgAN with low proteinuria[2], n ≤50
3. Adult IgAN with high proteinuria[3], n ≤20
4. Adolescent[4] IgAN at high risk of progression[5], n ≤10
5. Adult recurrent IgAN post kidney transplant, n ≤10
6. Adolescent[4] and adult IgAVN, n ≤10

[1]eGFR 20 to <30 mL/min/1.73 m^2
[2]UPCR <1.0 g/g
[3]UPCR ≥5.0 g/g
[4]Age ≥15 years
[5]UPCR ≥0.3 g/g



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).

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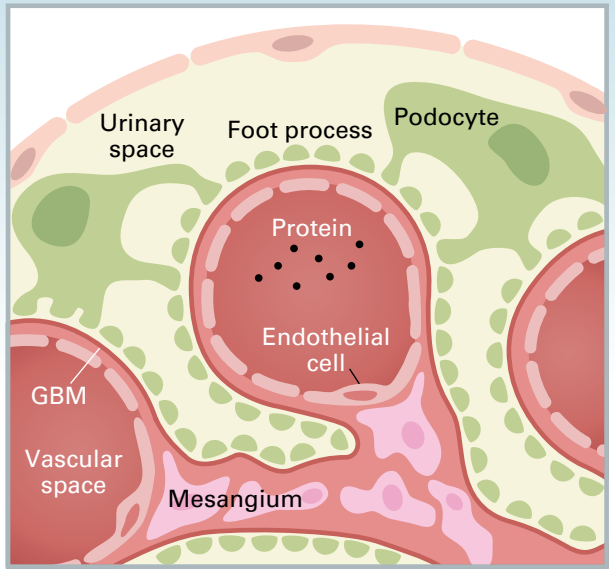
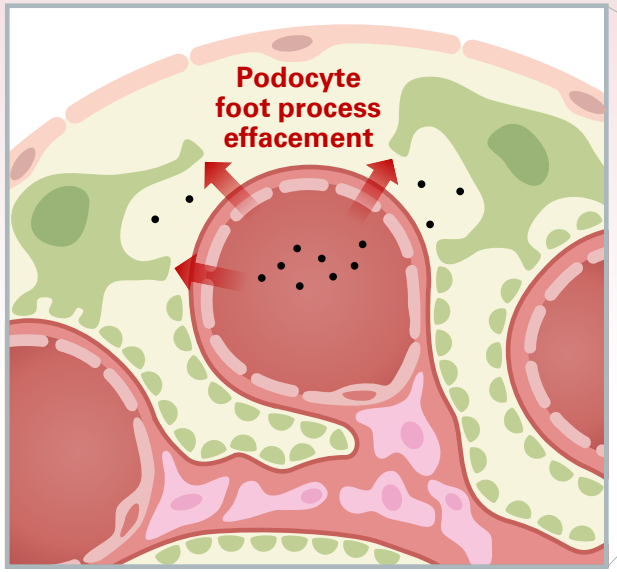
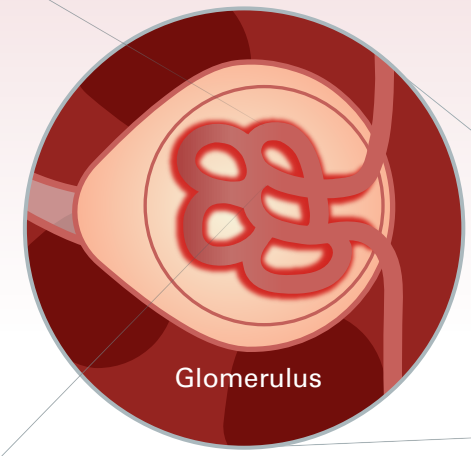
Autoimmune glomerular disease: podocyte injury and cytoskeletal derangement drives proteinuria and progressive disease

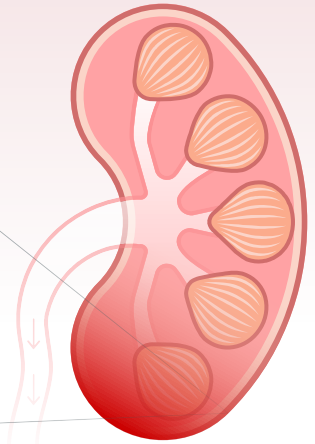
Healthy Podocyte Foot Processes



Disrupted Podocyte Foot Processes







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

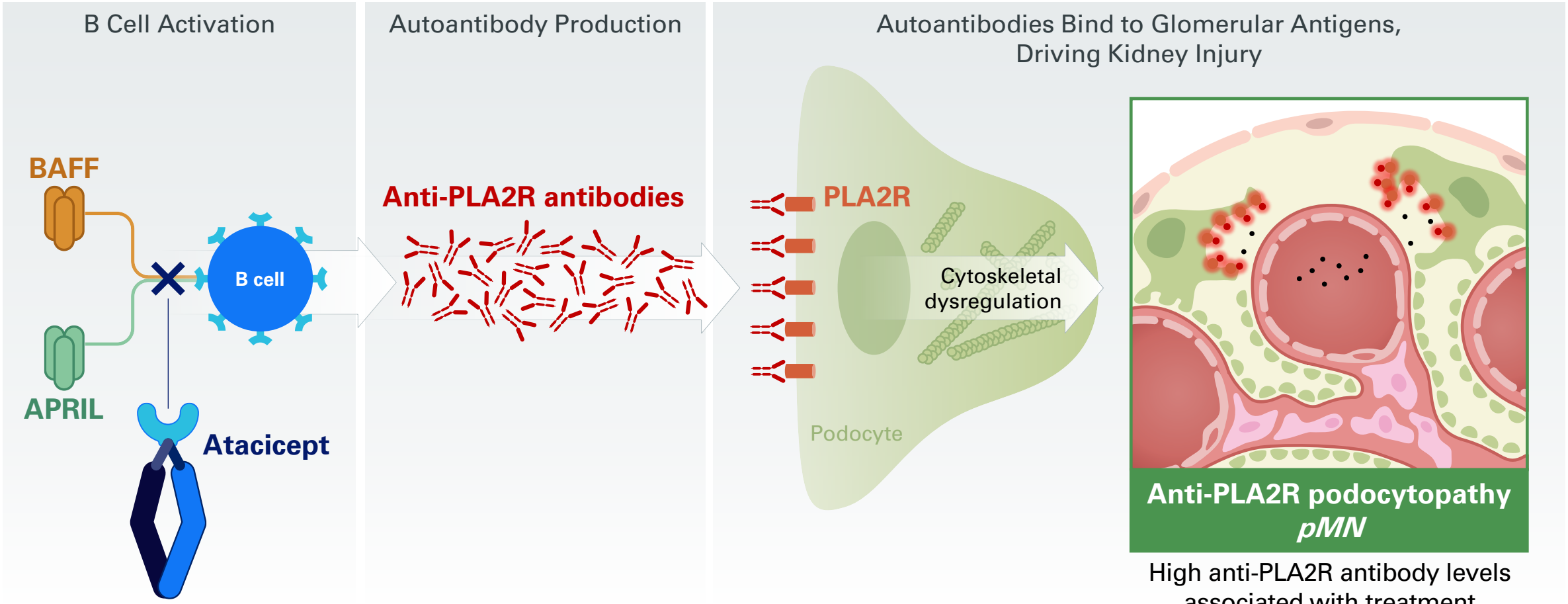
Causes of podocyte injury:
- Immune mediated injury
- Secondary causes
- Genetic predisposition
- Environmental factors

Resulting in clinically relevant alterations in the glomerular filtration barrier…

…leading to proteinuria including nephrotic syndrome and nephron loss

Kopp JB, et al. Nat Rev Dis Primer 2021. GBM = glomerular basement membrane.

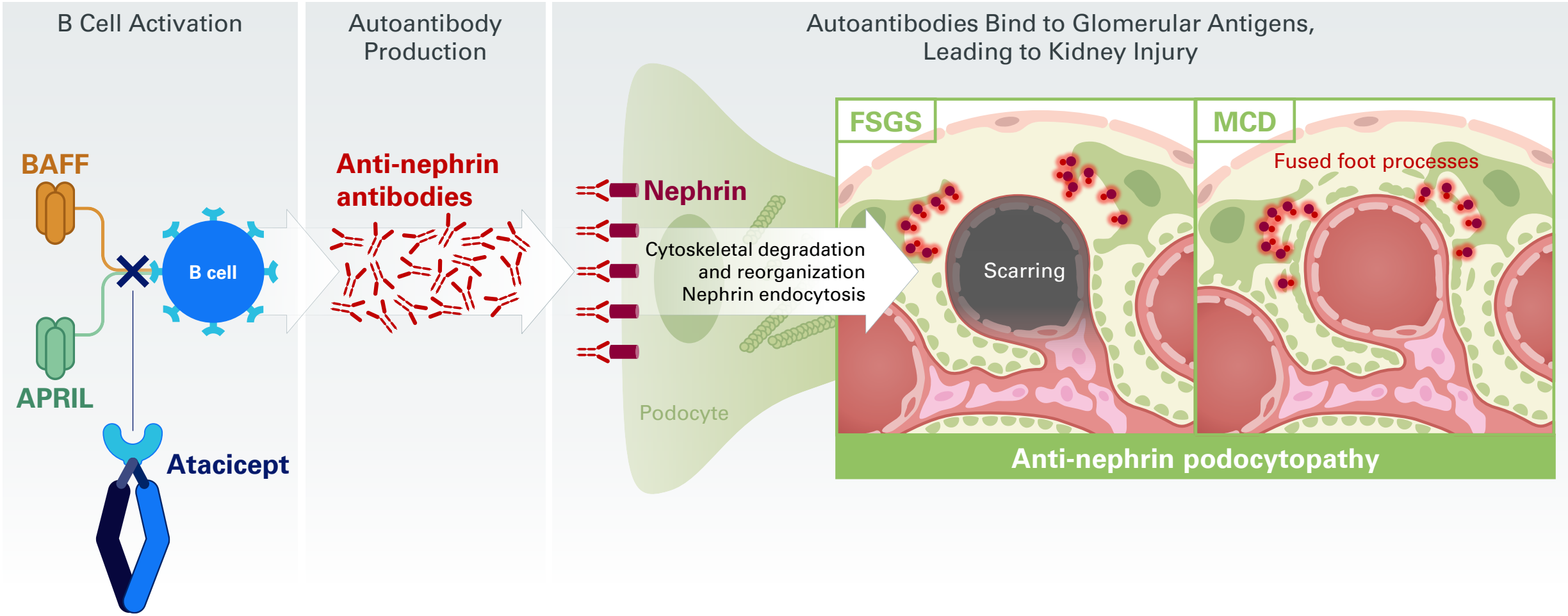
Atacicept mechanism of action has broad potential in autoimmune glomerular disease including membranous nephropathy



High anti-PLA2R antibody levels associated with treatment response in patients with MN[1]

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

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.

Atacicept expansion roadmap

US prevalence estimates

BAFF/APRIL inhibition

~160K[1]

origin

and

Expanded IgAN

~195K

+ ~35K[1-5]

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Anti-PLA2R podocytopathy

pMN

~230K

+ ~35K[2,3,6-10]

pioneer

Anti-nephrin podocytopathy

FSGS, MCD

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.

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

pioneer

Population 1, n ≤120
Expanded IgAN populations[1]

Population 2, n ≤20
Anti-PLA2R podocytopathy
(Membranous Nephropathy)

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

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
  - Change in anti-nephrin antibodies
- Safety

PLA2R = phospholipase A2 receptor.
1. 6 cohorts: adult IgAN with eGFR 20 to <30 mL/min/1.73 m^2, n ≤20; adult IgAN with UPCR <1.0 g/g, n ≤50; adult IgAN with UPCR ≥5.0 g/g, n ≤20; adolescent (≥15 y) IgAN with UPCR ≥0.3 g/g, n ≤10; Adult recurrent IgAN post kidney transplant, n ≤10; adolescent and adult IgA vasculitis nephritis, n ≤10.

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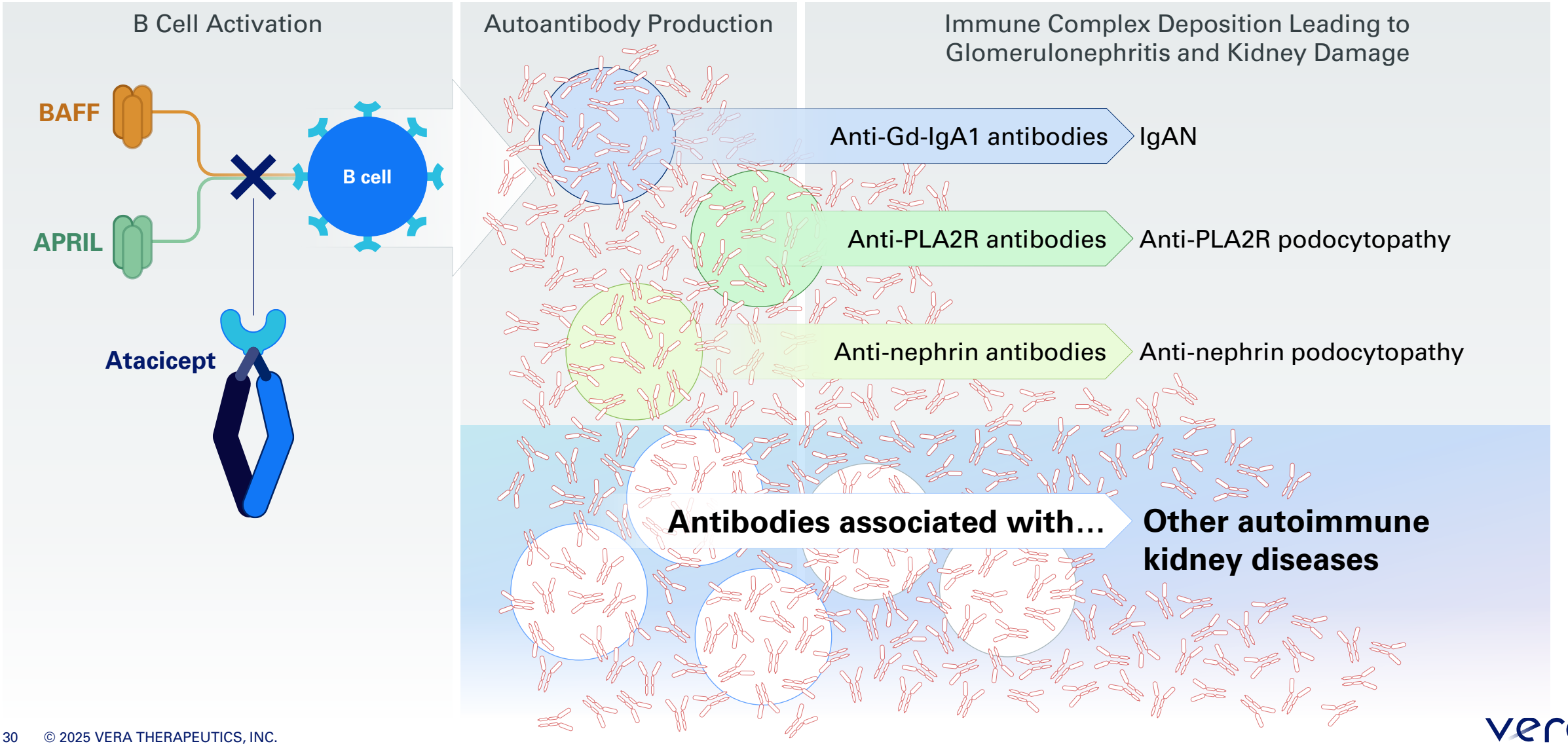
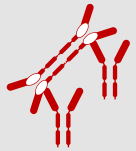
Targeting B cell production of autoantibodies against glomerular antigens offers the potential of additional kidney indications



Vision for an evolved approach to autoimmune glomerular disease

Identification of autoantigen/autoantibody constructs that drive autoimmune glomerular diseases

Gd-IgA1 & anti-Gd-IgA1



1999[1]

PLA2R & anti-PLA2R

2009[2]

Nephrin & anti-nephrin

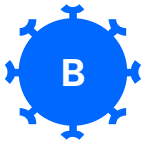
2022[3] **Today**

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.

Established leadership in B cell modulation and expanded breadth of expertise positions Vera for further innovation

R&D Accomplishments

- Jan 25 R&D Day: 72-week results
- ERA best abstract
- FDA BTD for atacicept in IgAN
- 96-week disease modification presented in ASN LB oral and JASN manuscript

ORIGIN 2b

- Primary endpoint cohort enrolled

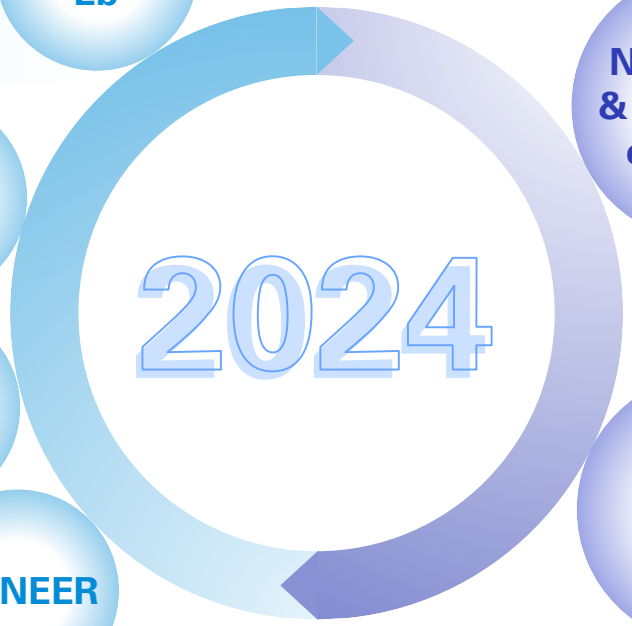
ORIGIN 3

- Study initiated

ORIGIN Extend

- Atacicept indication expansion to broader IgAN cohort, PMN, FSGS, MCD

PIONEER



Corporate Growth

New talent & functional expertise

- Research & discovery
- Bioassay and biomarkers
- Clinical pharmacology
- Translational medicine
- Preclinical development
- Pharmacovigilance
- Field Medical Directors
- Health economics & outcomes research
- Commercial

Strong financial position

- Two transformative financings leading to ~$677M *pro forma* cash position[1]

1. Unaudited as of September 30, 2024. 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.

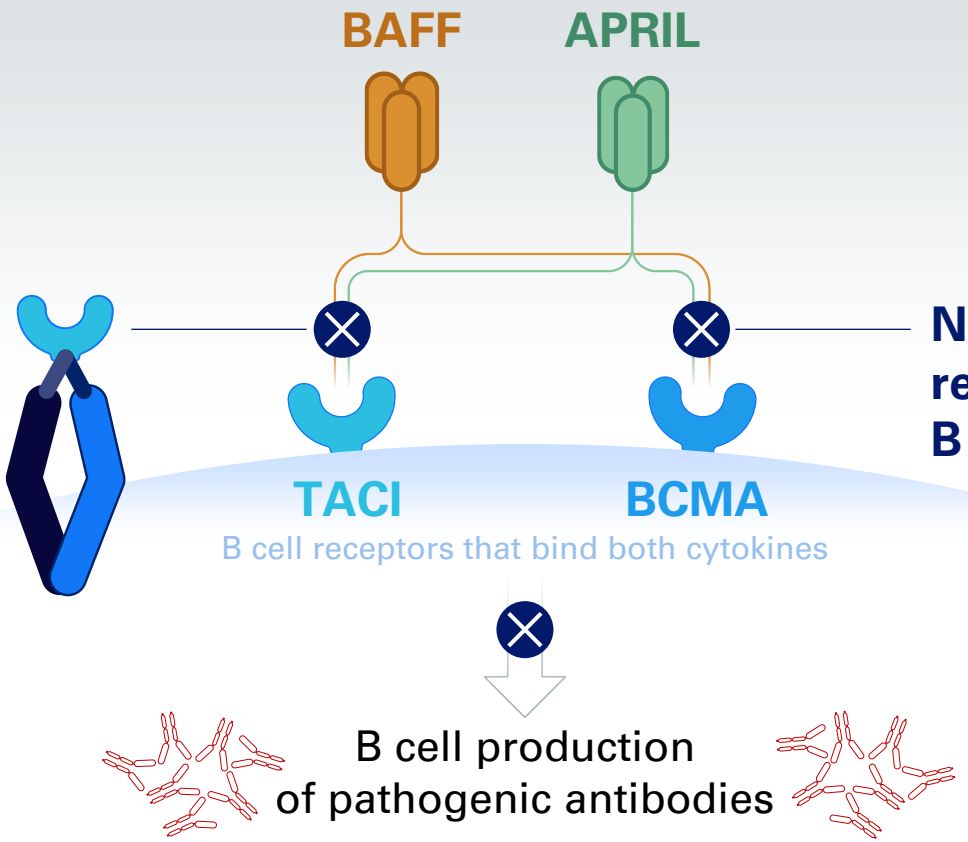
Opportunity to innovate and extend leadership in B cell modulation

Current landscape of B cell modulators

Monoclonal antibodies binding either BAFF or APRIL alone

Fc fusion proteins containing TACI or TACI variants

Atacicept
Longest duration of B cell modulator data to date showing eGFR stabilization

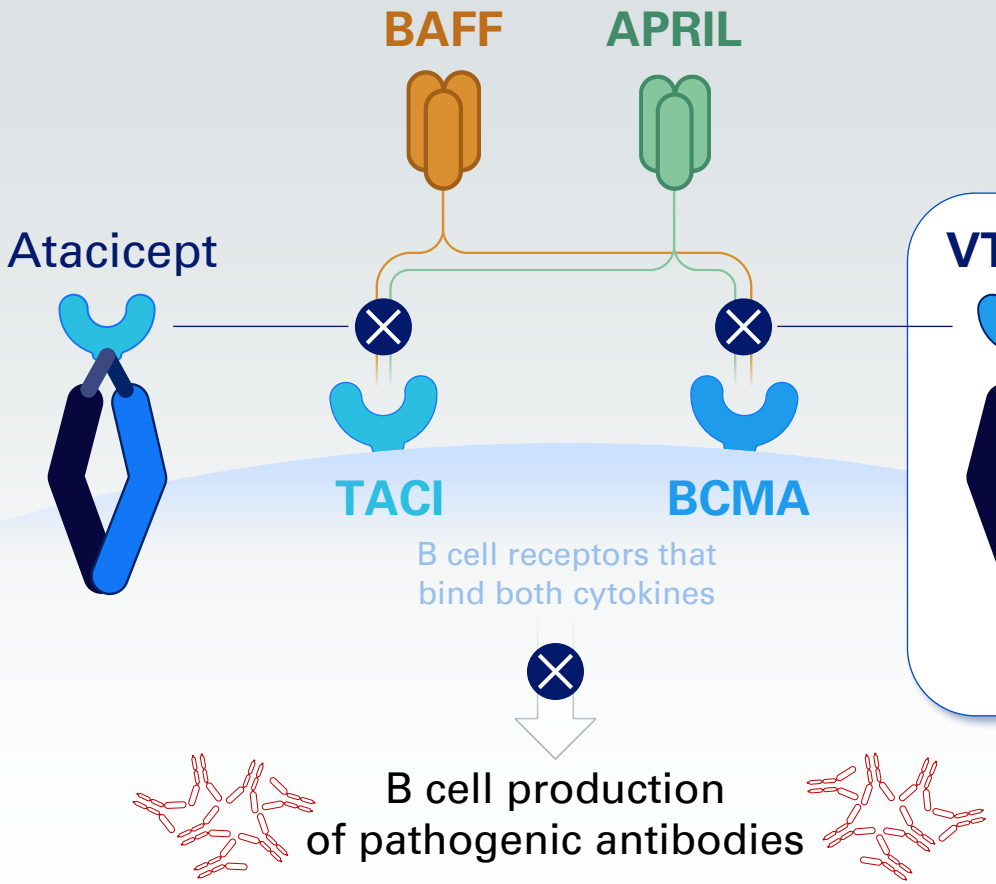


Novel BCMA constructs represent an innovative B cell modulatory unlock

BCMA = B cell maturation antigen.

Novel, next-generation dual BAFF/APRIL inhibitor

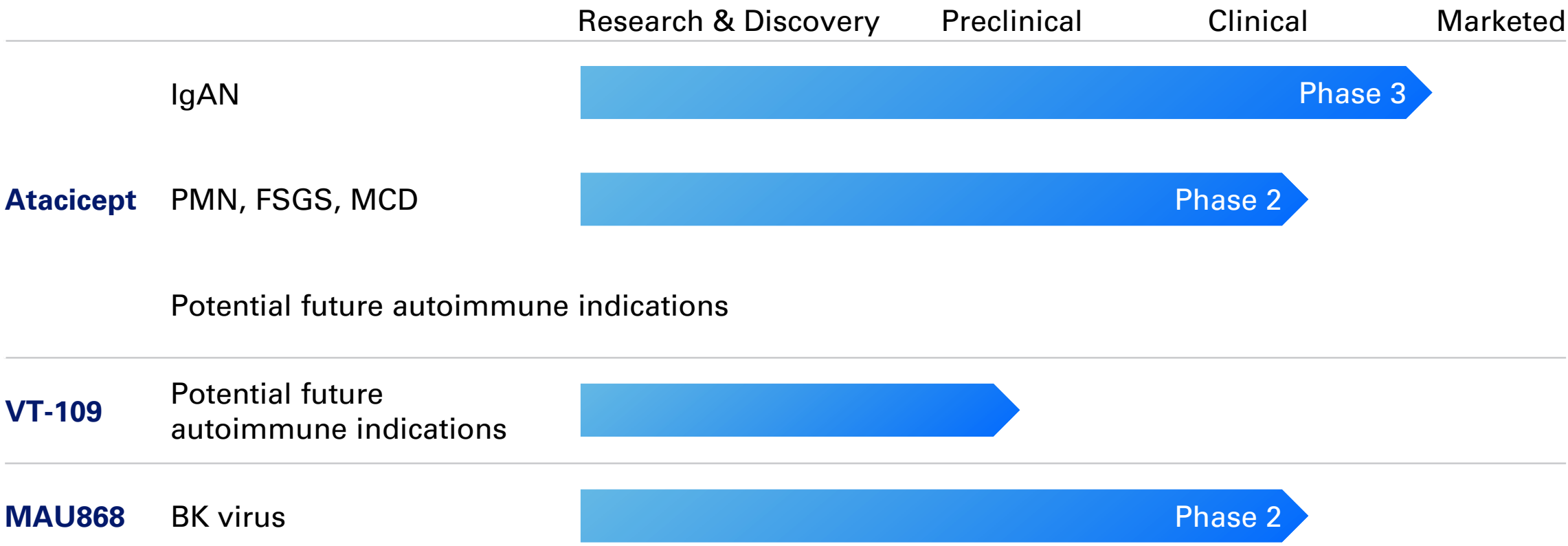
Potential for additional patient benefit across diseases and populations



VT-109

- Novel molecule engineered by team at Stanford University
- Picomolar binding affinity for BAFF and APRIL, attractive PK and half-life
- Novel composition may offer differentiation on
  - Frequency of administration
  - Route of administration
  - Other characteristics of molecule

Vera Pipeline



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

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therapeutics™