vera therapeutics™

R&D Day

October 2, 2024

Forward-looking statements

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Agenda

Opening Remarks	**Marshall Fordyce, MD** Founder and CEO, Vera Therapeutics
Vera Expansion Strategy	Robert Brenner, MD Chief Medical Officer, Vera Therapeutics
Q&A Panel	Jonathan Barratt, MD, PhD, FRCP Mayer Professor of Renal Medicine, University of Leicester Richard Lafayette, MD, FACP Professor of Medicine (Nephrology), Stanford University Medical Center Director, Stanford Glomerular Disease Center Brad Rovin, MD, FACP, FASN Lee A. Herbert Professor of Nephrology Ohio State University Wexner Medical Center
Closing Remarks	Marshall Fordyce, MD Founder and CEO, Vera Therapeutics

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 1.5 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 ~$384M cash, cash equivalents and marketable securities as of June 30, 2024
- Management focused on potential for successful commercial launch

BAFF = B cell activating factor; APRIL = A proliferation inducing ligand; eGFR = estimated glomerular filtration rate; IgAN = IgA nephropathy; SC = subcutaneous.

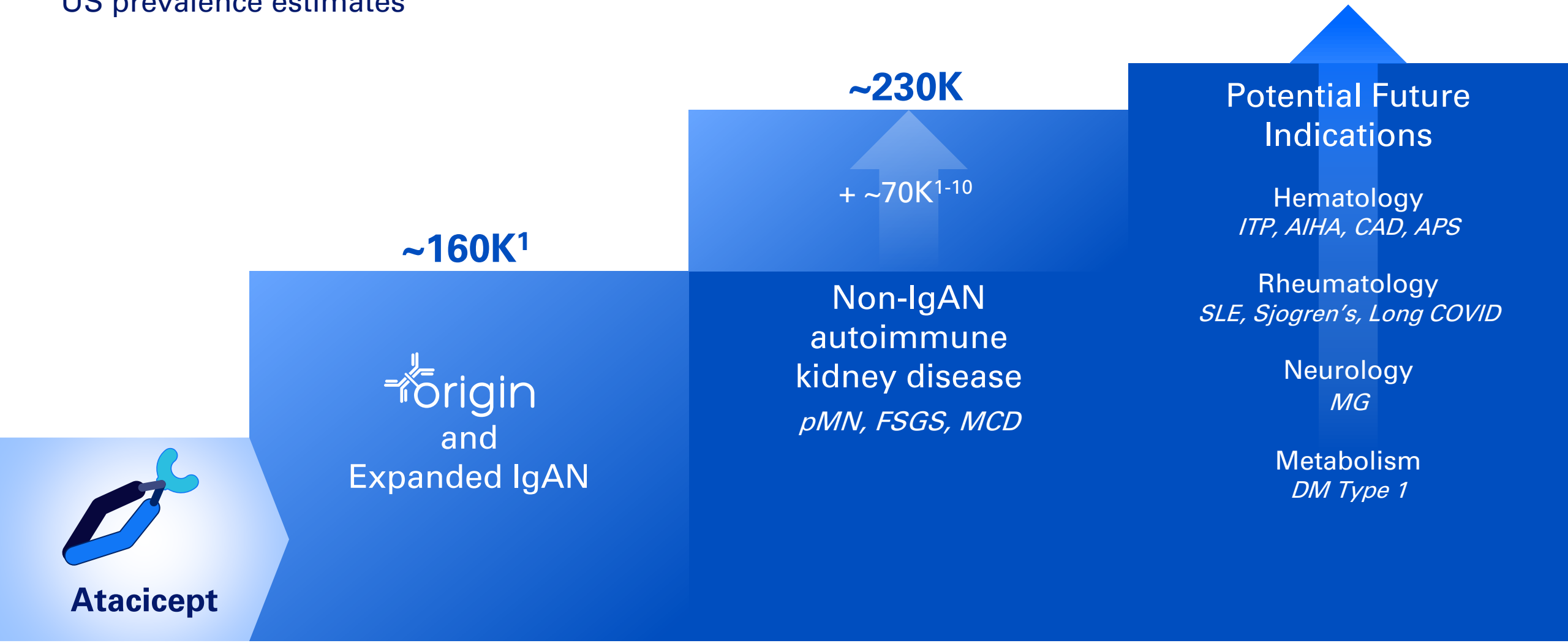
Cumulative Atacicept data offers best-in-class potential

	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 (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	✓	✓	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	18 months, n=109 24 months coming soon*	12 months, n=145	12 months, n=1	12 months, n=35
Projected Commercial Launch	2026	2026	2027	2027

*To be presented at ASN Kidney Week 2024.
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 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.

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; MG = myasthenia gravis; COVID = Coronavirus disease 2019; DM = diabetes mellitus.

Vera financial position is strong

~$384M

Cash, cash equivalents, and marketable securities
(as of 6.30.24)

~54.8M

Shares outstanding
(as of 8.5.24)

Atacicept: previously shared projected catalysts

origin
(IgAN)

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			

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

Based on management's current assumptions.

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Atacicept: additional projected catalysts

	Catalyst	2024	2025	2026
origin (IgAN)	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			
New clinical trial	Initiation			
	Initial data available			
New clinical trial	Initiation			
	Initial data available			

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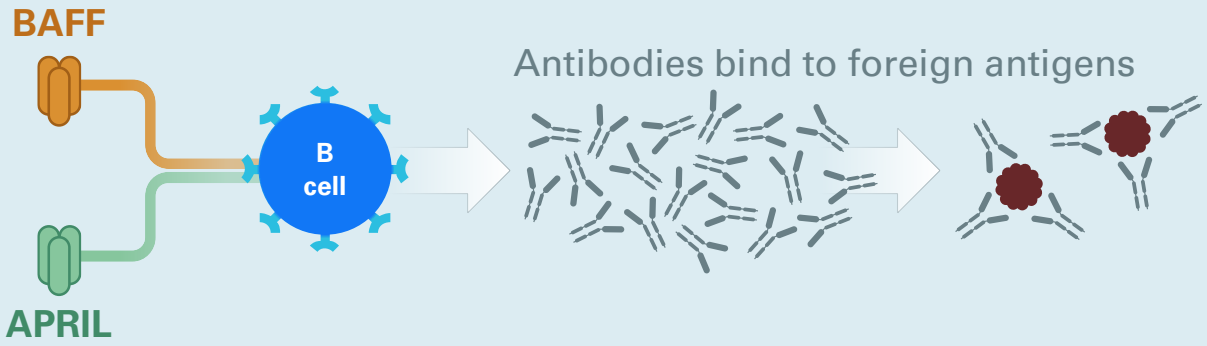
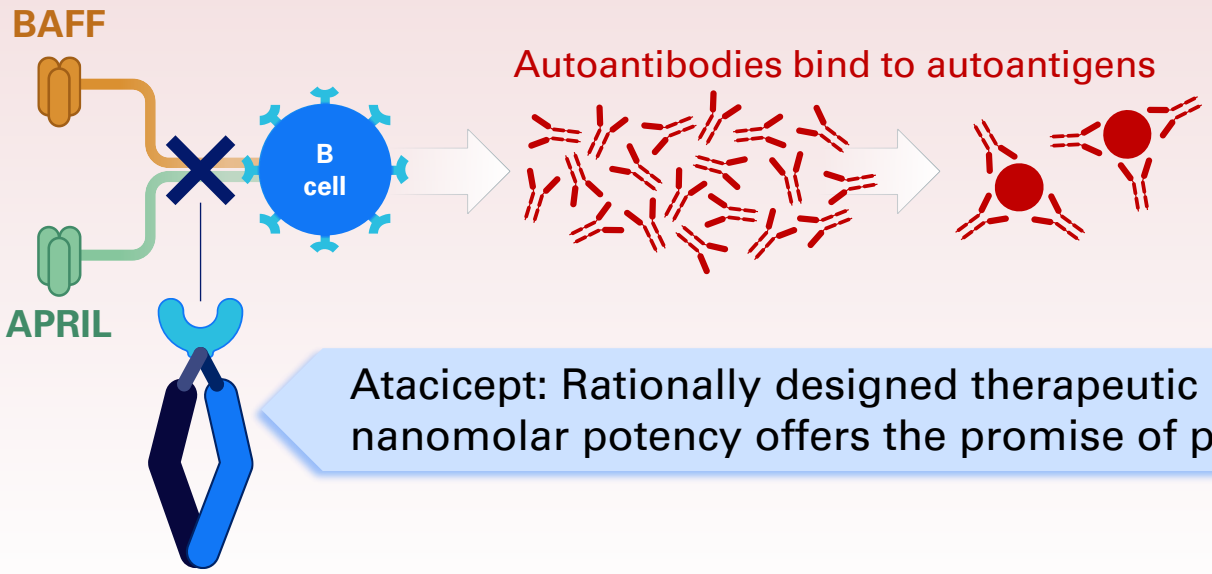
Ataicept has broad therapeutic potential in autoimmune disease

Immunity in health



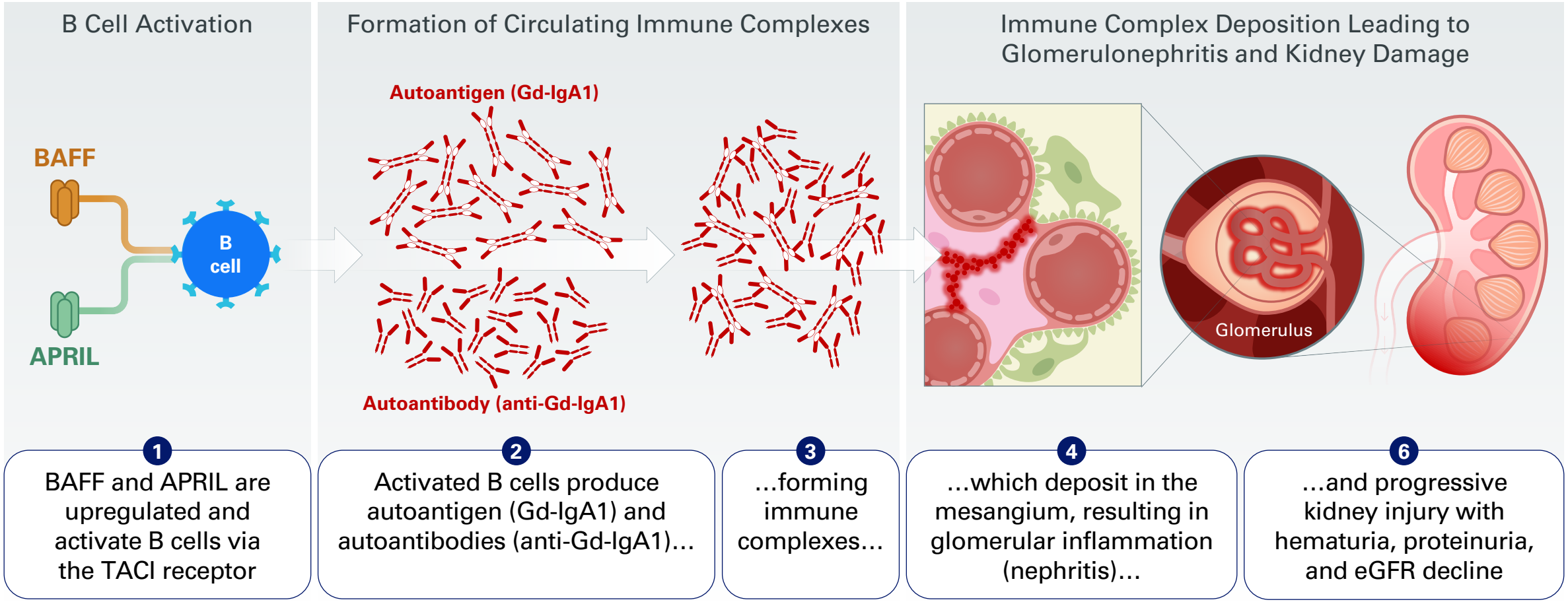
Autoimmune disease



- 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

Ataicept: 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

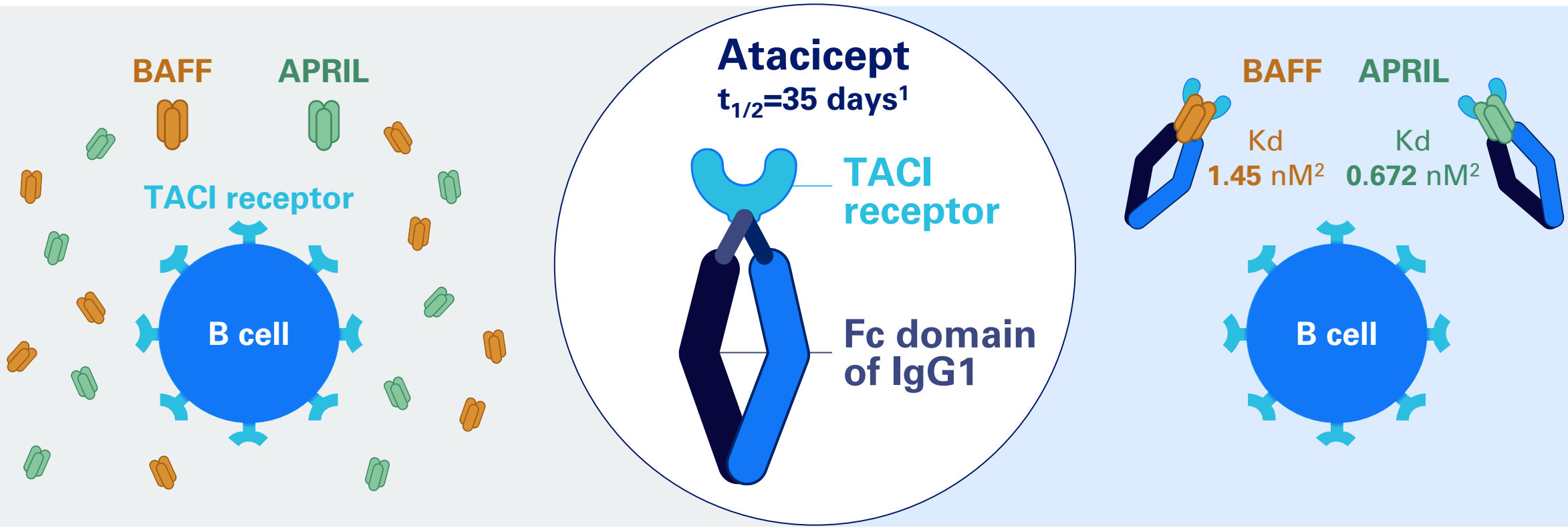
Lead indication: IgAN is a disease of B cell origin with kidney pathology



TACI = transmembrane activator and calcium-modulator and cyclophilin ligand.

Atacicept is an example of rational drug design

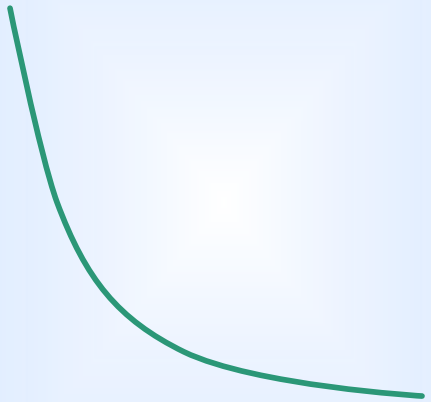
Native TACI-Fc fusion: Soluble protein binds both BAFF and APRIL with nanomolar potency



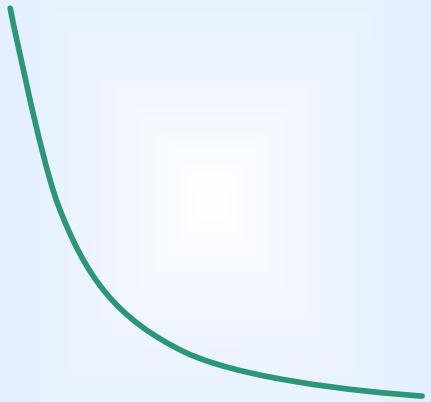
Fc = fragment crystallizable. 1. Willen D, et al. Eur J Drug Metab Pharmacokinet 2020;45(1):27-40; 2. Vera data on file.

An ideal IgAN disease modifying therapy would be expected to…

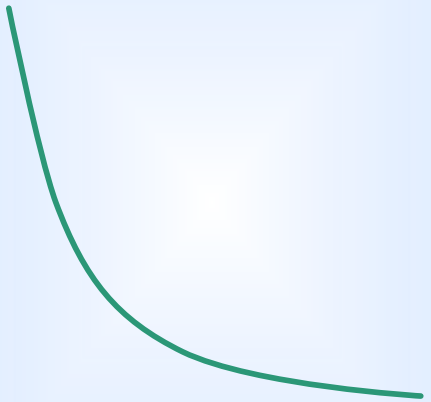
Reduce Gd-IgA1



Reduce hematuria



Reduce proteinuria



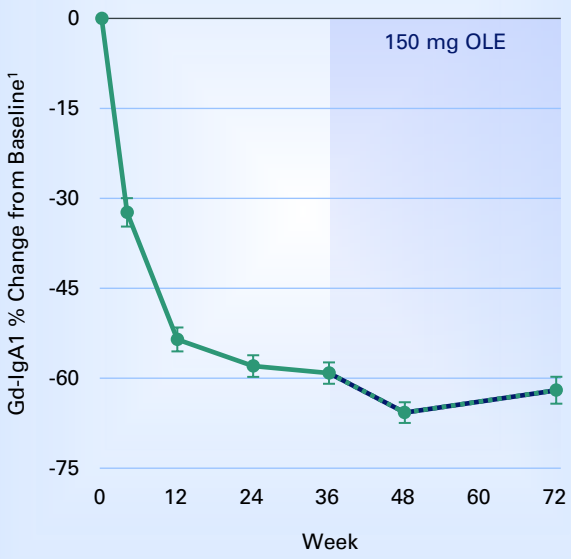
Convert eGFR profile to general population

ORIGIN Phase 2b 72-week results consistent with IgAN disease modification

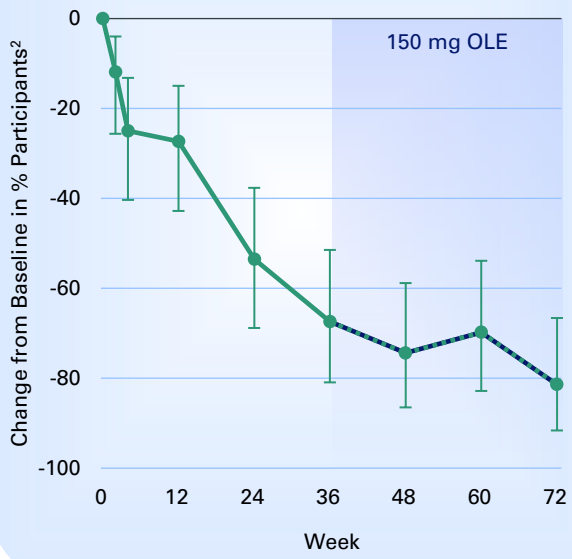
Including eGFR Profile consistent with the general population of -1 mL/min/year

FDA Breakthrough Therapy Designation

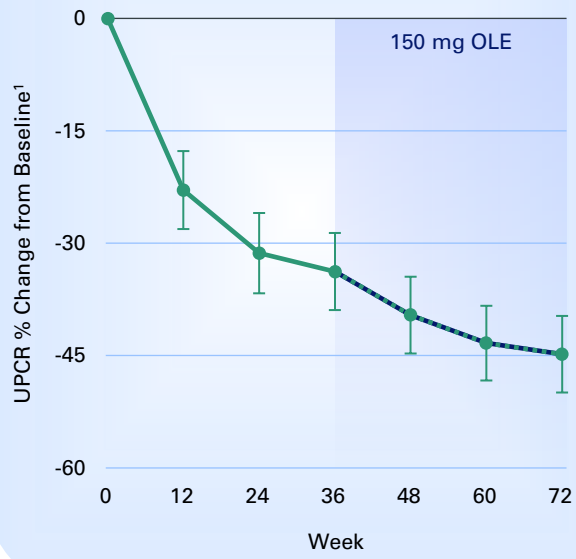
Reduction in Gd-IgA1



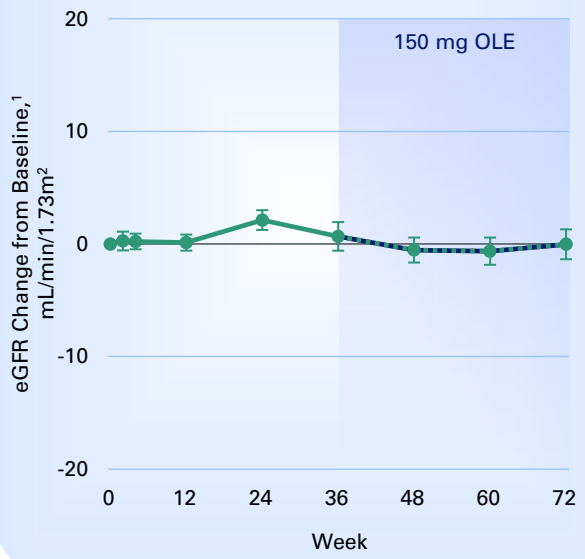
Reduction in hematuria



Reduction in proteinuria



eGFR profile consistent with general population



Set the standard in IgAN

Lafayette R, et al. ERA 2024, abstr 812.
1. Mean ± SE; 2. Change from baseline in percentage of participants with hematuria at each visit out of those with baseline hematuria.
Data from participants originally randomized to any atacicept group in the double-blind period in the intent-to-treat analysis for Gd-IgA1, hematuria, UPCR and eGFR. OLE = open-label extension.

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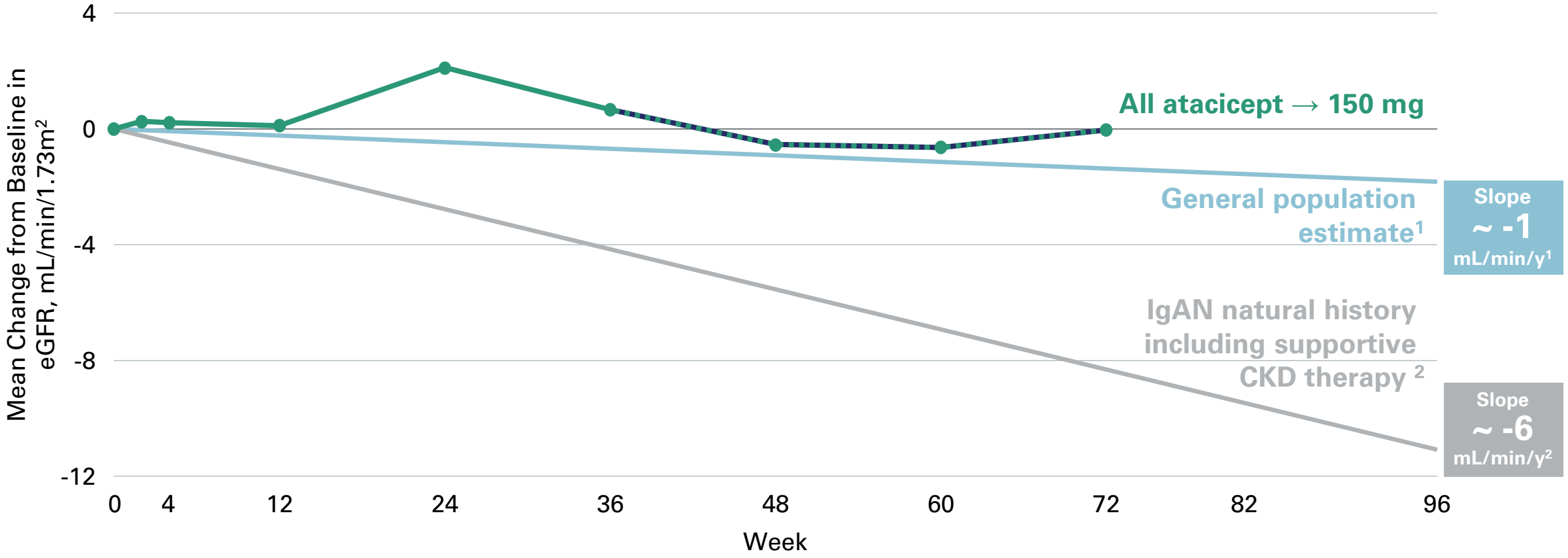
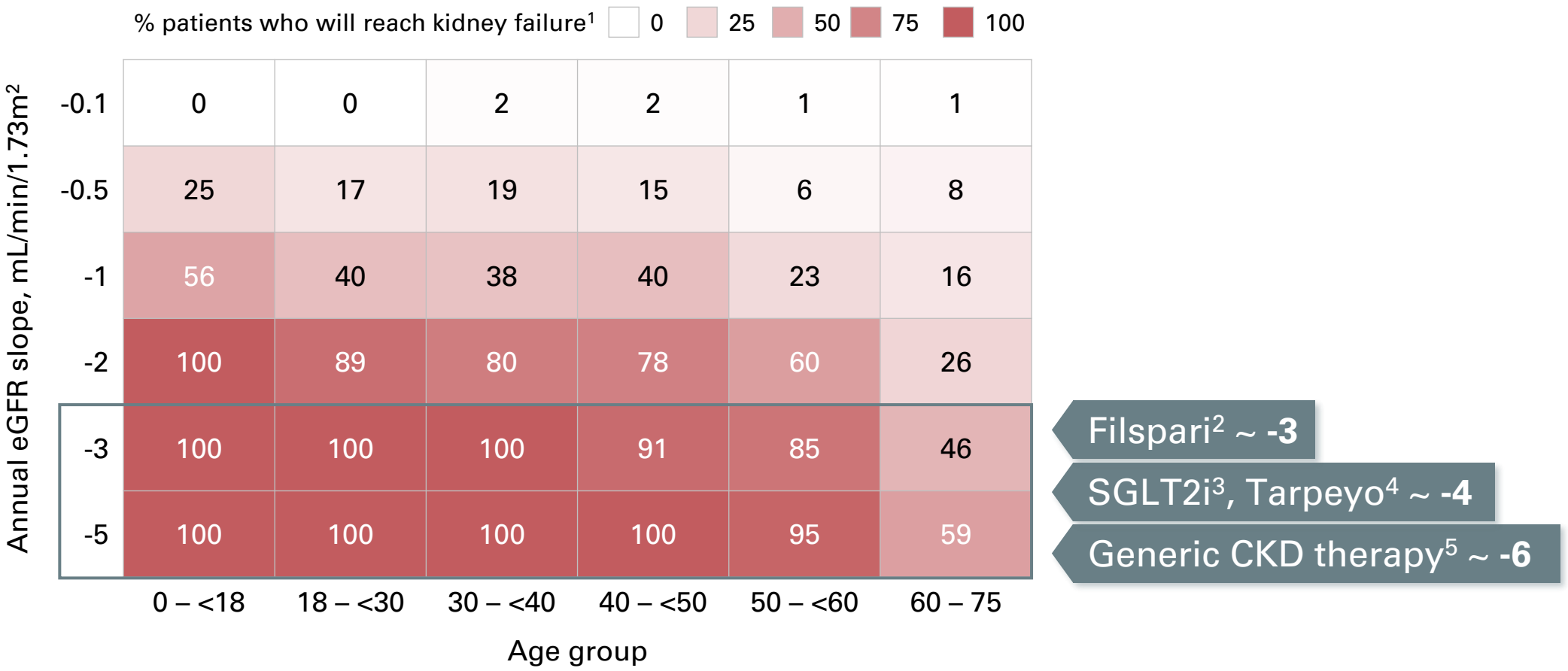
Atacicept treated participants have an eGFR slope profile consistent with *general population without kidney disease*



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 7 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.

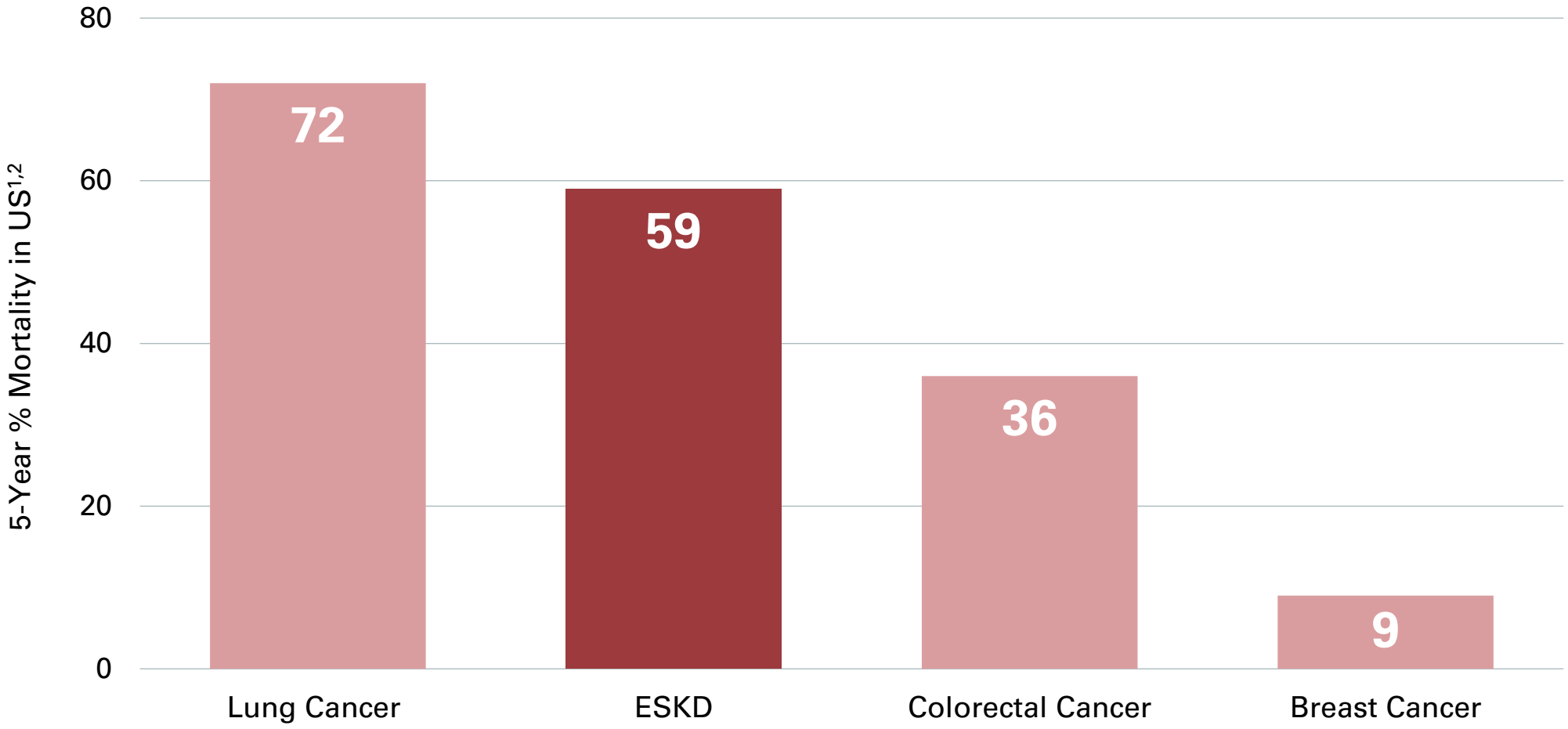
2024 Draft KDIGO IgAN guidelines call for target eGFR slope ≤ -1 mL/min/year



SGLT2i = sodium-glucose cotransporter-2 inhibitor.

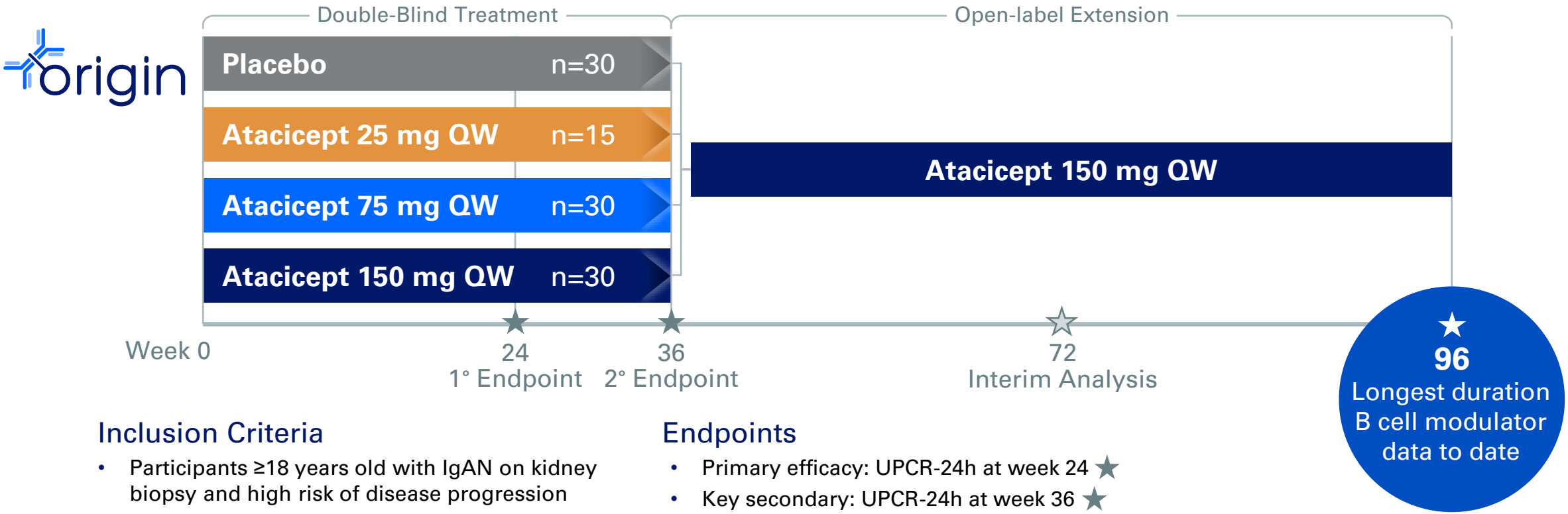
1. Adapted from Pitcher D, et al. CJASN 2023; 2. Rovin BH, et al. Lancet 2023; 3. Wheeler DC, et al. Kidney Int 2021; 4. Lafayette R, et al. Lancet 2023; 5. Average historical placebo (including chronic kidney disease standard of care) data from 7 clinical trials: Li PK-T, et al. Am J Kidney Dis 2006; 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



ESKD = end-stage kidney disease. 1. US CDC Cancer Statistics; 2. Thurlow JS, et al. Am J Nephrol 2021.

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



Inclusion Criteria

- Participants ≥18 years old with IgAN on kidney biopsy 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; UPCR = urine protein:creatinine ratio.

Consistency with ORIGIN 2b instills great confidence in ORIGIN 3

origin3

Inclusion Criteria

- Patients ≥18 years old with IgAN on kidney biopsy 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

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 study design and worldwide sites as ORIGIN 2b
- Same self-administered SC formulation and dose as used in ORIGIN 2b

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Atacicept expansion roadmap

US prevalence estimates

~160K[1]

origin
and
Expanded IgAN

Atacicept

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

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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. Document impact of withdrawal from therapy, followed by restart

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 half life also supports evaluation of extended dosing
- QM dose finding study in 2025

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

PIONEER: Phase 2 basket study in expanded IgAN cohorts

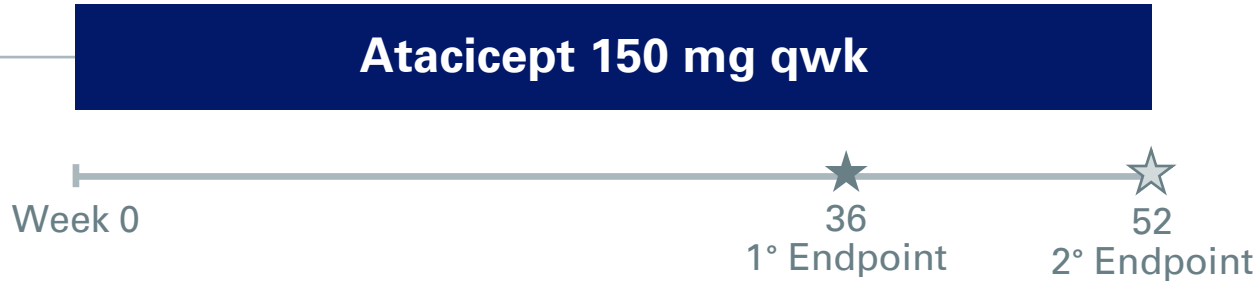
Patients ineligible for ORIGIN 3 will have an opportunity to enroll in PIONEER at same clinical sites

pioneer

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



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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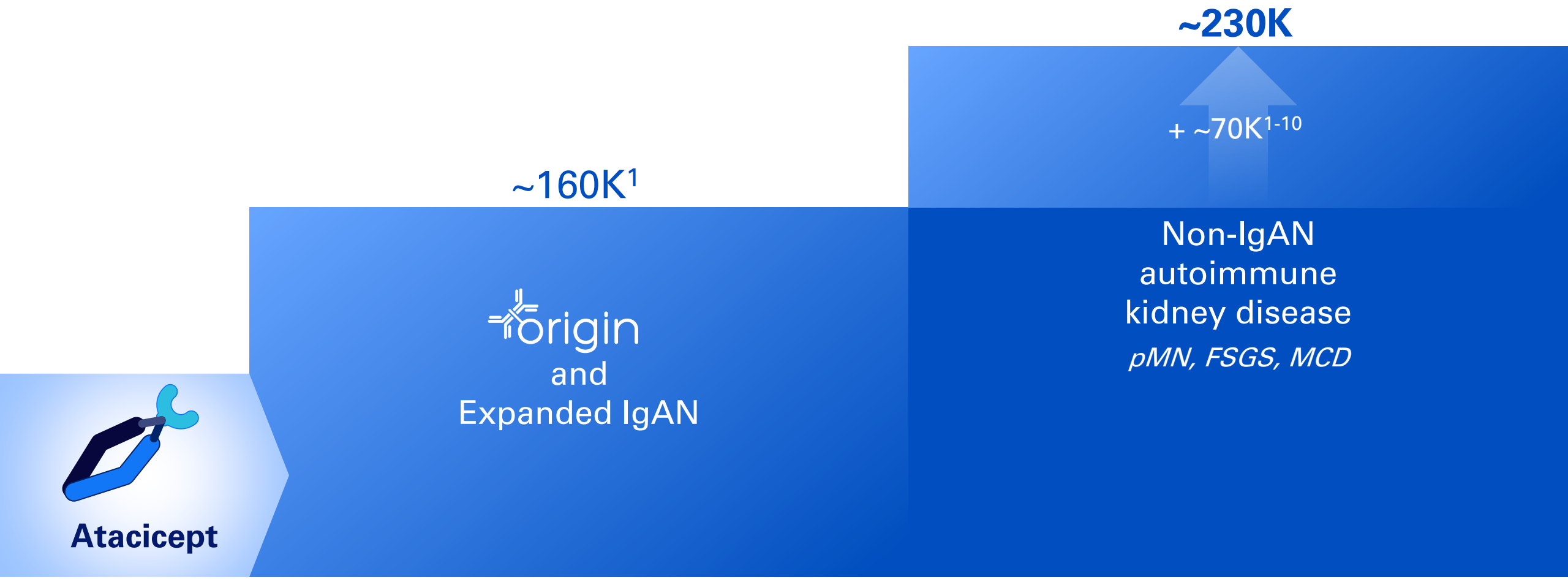
Atacicept expansion roadmap

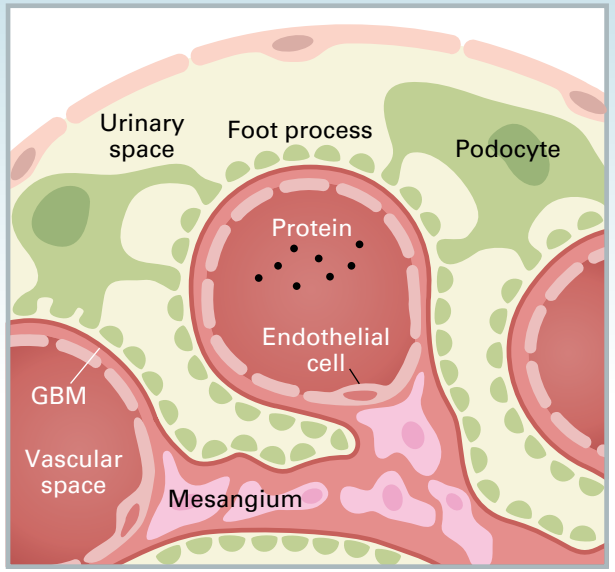
US prevalence estimates



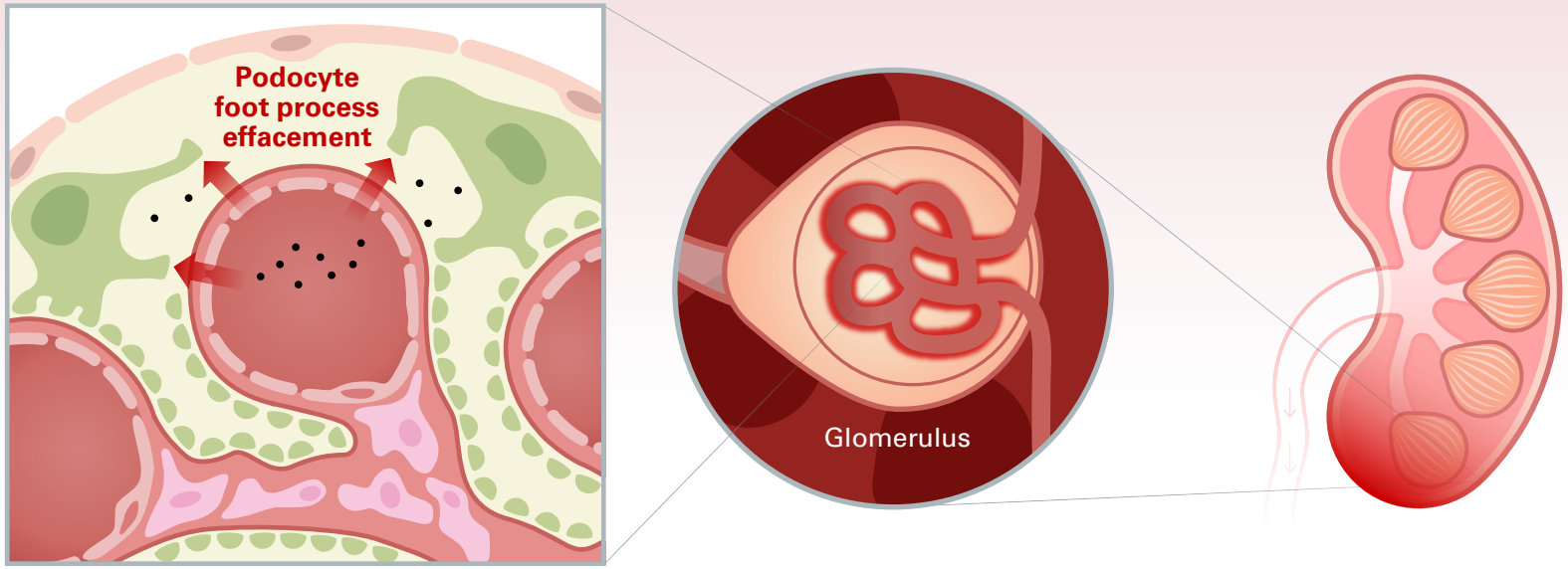
Vera Therapeutics corporate estimates for peak year prevalence based on 1. ClearView Healthcare Partners Analysis; 2. US Census 2023; 3. McGrogan A. Nephrol Dial Transplant 2011; 4. Couser ASN 2017; 5. Beck LH. N Engl J Med 2009; 6. Filler G. Am J Kidney Dis 2003; 7. Troyanov S. J Am Soc Nephrol 2005. 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



Disrupted Podocyte Foot Processes



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

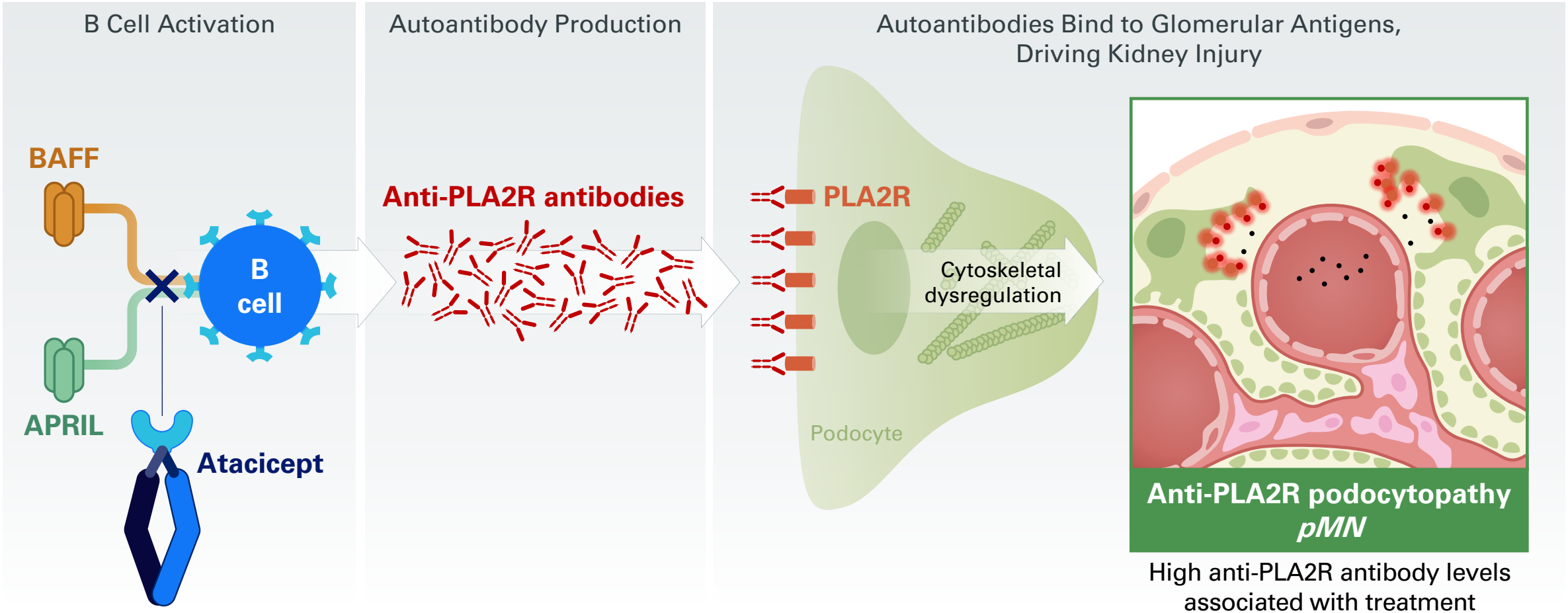
Causes of podocyte injury:
- Immune mediated injury
- Secondary causes
- Genetic predisposition
- Environmental factors

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.

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.

Atacicept expansion roadmap

US prevalence estimates

~195K

+ ~35K[1-5]

~160K[1]

origin

and

Expanded IgAN

pioneer

Anti-PLA2R podocytopathy

pMN

Atacicept

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.

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

pioneer

Population 1, n ≤120
Expanded IgAN populations

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

Atacicept 150 mg qwk

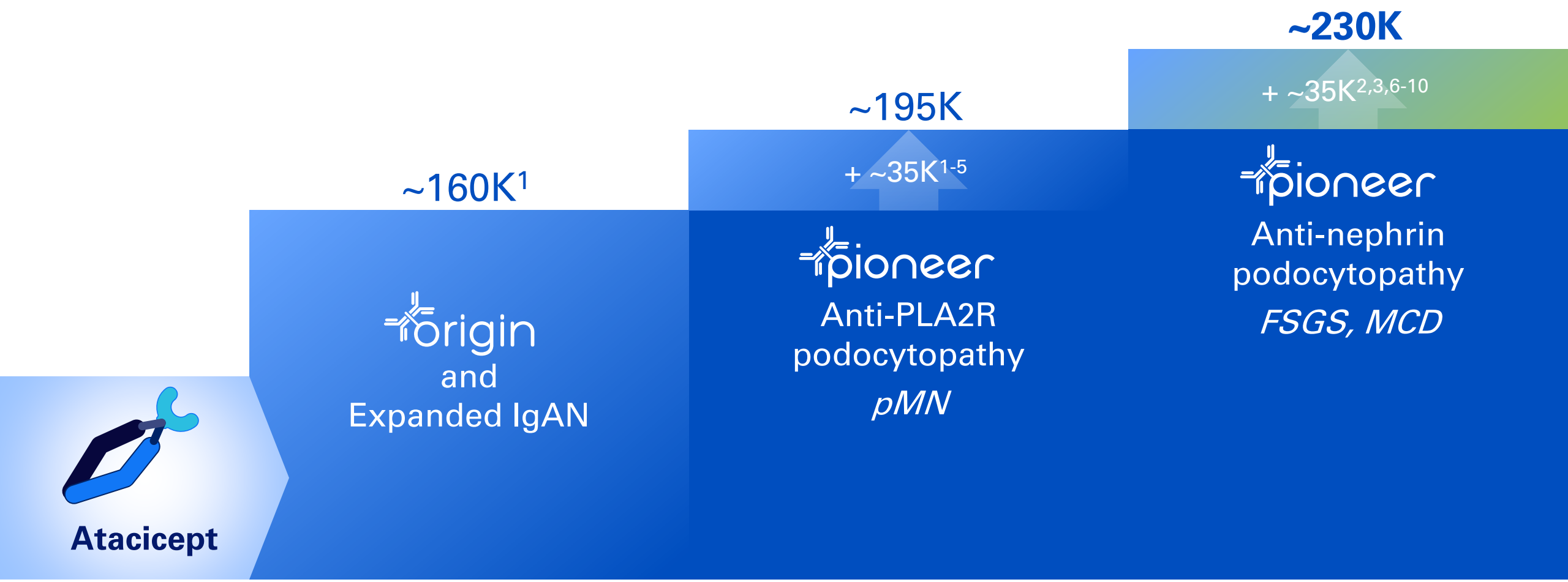
Endpoints

- 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

vera therapeutics

Atacicept expansion roadmap

US prevalence estimates



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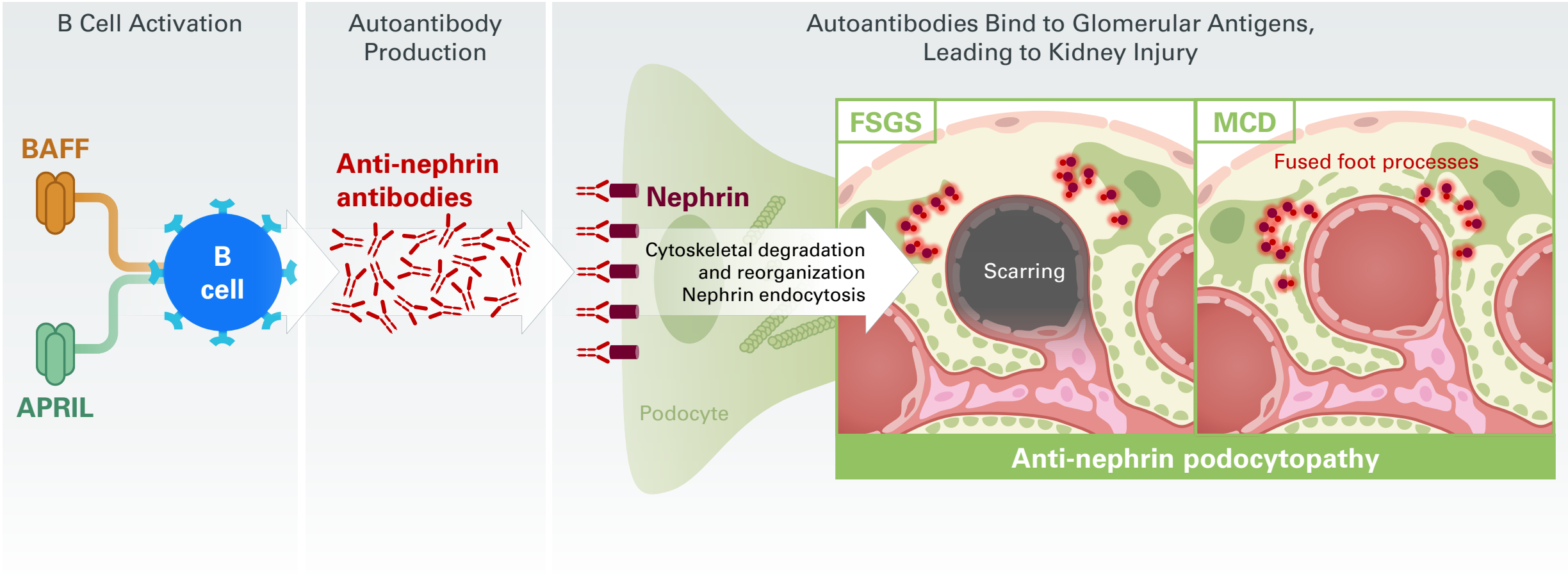
FSGS and MCD are histologic diagnoses with heterogeneous etiology; Autoimmunity, including anti-nephrin antibodies, is one driver of disease



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 study in expanded IgAN and anti-PLA2R & anti-nephrin podocytopathies

pioneer

Population 1, n ≤120
Expanded IgAN populations

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

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

Atacicept 150 mg qwk

Endpoints

- Primary: UPCR change at week 36
- Key secondary: eGFR change at weeks 36, 52
- Exploratory:
  - Gd-IgA1 change at weeks 36, 52
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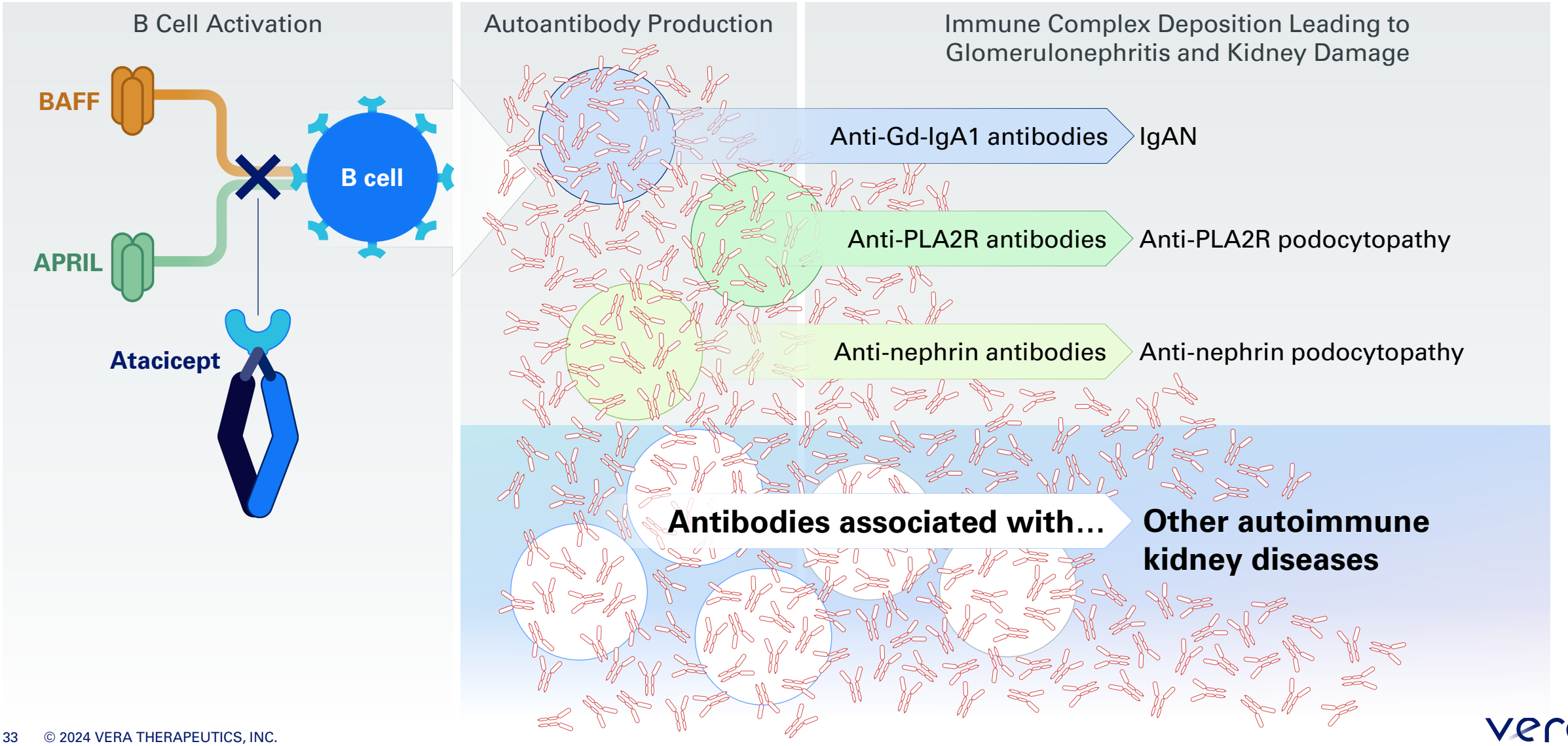
  - Change in anti-PLA2R antibodies
  - Change in anti-nephrin antibodies
- Safety

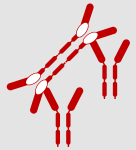
vera therapeutics

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



Vision for an evolved approach to autoimmune glomerular disease

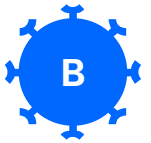
Identification of autoantigen/autoantibody constructs that drive autoimmune glomerular diseases

Gd-IgA1 & anti-Gd-IgA1	PLA2R & anti-PLA2R	Nephrin & anti-nephrin	**Future autoantigen/autoantibody constructs identified**
			
1999[1]	2009[2]	2022[3] **Today**	

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**



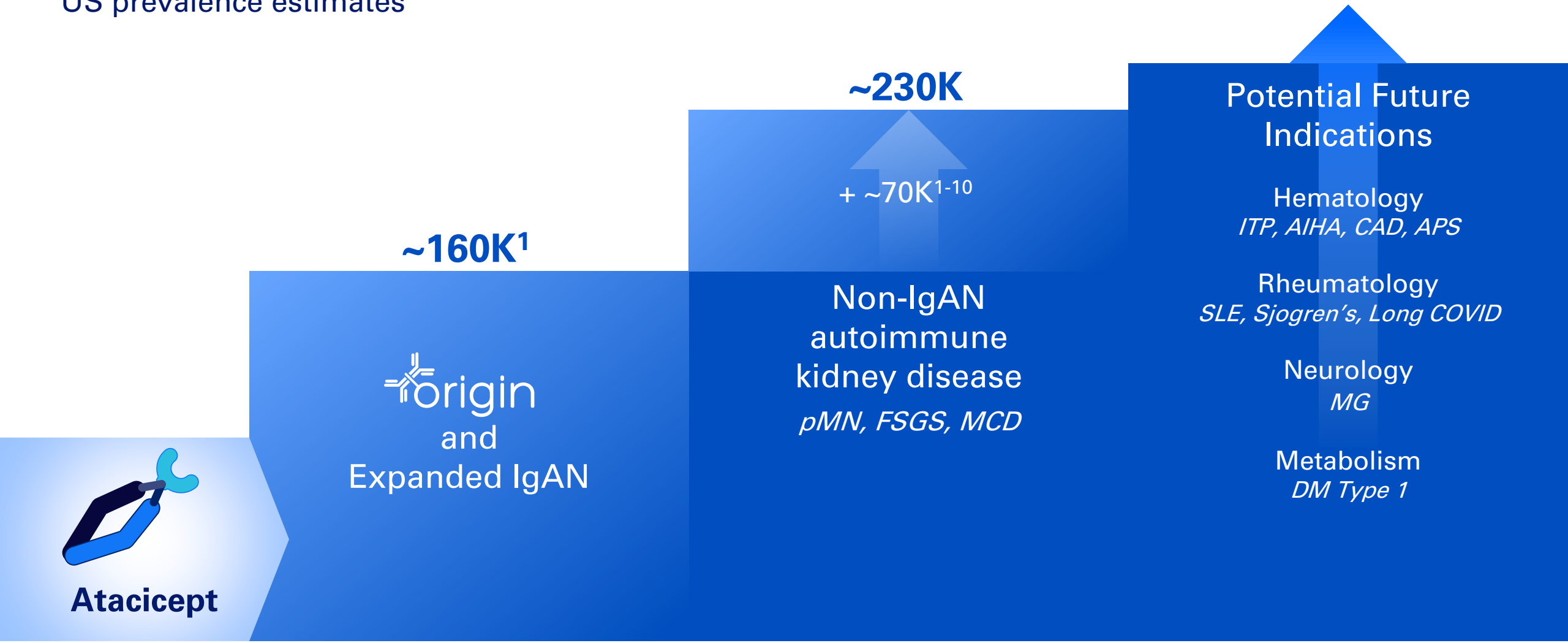
Does not require pre-existing elucidation of all autoantigen/autoantibody constructs

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
origin (IgAN)	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			
origin extend (IgAN)	Initiation			
	Initial data available			
pioneer (IgAN, PMN, FSGS, MCD)	Initiation			
	Initial data available			

Vera holds worldwide, exclusive rights to develop and commercialize atacicept

Based on management's current assumptions.

Agenda

Opening Remarks	Marshall Fordyce, MD Founder and CEO, Vera Therapeutics
Vera Expansion Strategy	Robert Brenner, MD Chief Medical Officer, Vera Therapeutics
Q&A Panel	**Jonathan Barratt, MD, PhD, FRCP** Mayer Professor of Renal Medicine, University of Leicester **Richard Lafayette, MD, FACP** Professor of Medicine (Nephrology), Stanford University Medical Center Director, Stanford Glomerular Disease Center **Brad Rovin, MD, FACP, FASN** Lee A. Herbert Professor of Nephrology Ohio State University Wexner Medical Center
Closing Remarks	Marshall Fordyce, MD Founder and CEO, Vera Therapeutics

Q&A Panel

Jonathan Barratt
MD, PhD, FRCP
UNIVERSITY OF LEICESTER

Dr. Barratt leads the Renal Research Group within the College of Life Sciences, University of Leicester. His research is focused on a bench-to-bedside approach to improving our understanding of the pathogenesis of IgAN, a common global cause of kidney failure. Dr. Barratt is the IgAN Rare Disease Group lead for the UK National Registry of Rare Kidney Diseases (RaDaR), and a member of the steering committee for the International IgAN Network. He works closely with pharmaceutical companies interested in new treatments for IgAN, is Chief Investigator for a number of international randomized controlled Phase 2 and 3 clinical trials in IgAN, and was a member of the U.S. Food and Drug Administration and American Society of Nephrology Kidney Health Initiative: Identifying Surrogate Endpoints for Clinical Trials in IgAN Workgroup.

Richard Lafayette
MD, FACP
Stanford MEDICINE

Dr. Lafayette is a Professor of Medicine (Nephrology) and Director of the Stanford Glomerular Disease Center at Stanford University Medical Center. Dr. Lafayette completed his medical education at New York Medical College and went on to complete his residency at the Long Island Jewish Medical Center, and his fellowship at Stanford University School of Medicine. Dr. Lafayette is board-certified in Internal Medicine and Nephrology.
Dr. Lafayette served as the Associate Chair of the Stanford University Department of Medicine from 2002–2007, the Clinical Chief of Nephrology at Stanford University from 1999–2012, and currently serves as the Director of the Stanford Glomerular Disease Center since 2010. Dr. Lafayette was honored in America's Top Doctors, Best Doctors from 2004–2018, and received America's Top Doctors Award, Castle Connolly Medical Ltd. from 2014–2022. Dr. Lafayette has been part of the following boards and professional organizations: Editorial Board, Kidney News, American Society of Nephrology (2010–2021) Member, Glomerular Disease Advisory Committee, American Society of Nephrology (2013–2017) Member (ex-officio), Communications Committee, American Society of Nephrology (2015–Present).

Brad Rovin
MD, FACP, FASN
THE OHIO STATE UNIVERSITY
WEXNER MEDICAL CENTER

Dr. Brad H. Rovin is the Lee A. Hebert Professor of Nephrology. Dr. Rovin received his BS in Chemical Engineering from Northwestern University and his MD from the University of Illinois Medical School. He completed a residency in Internal Medicine at Barnes Hospital in St. Louis, Missouri, and a Fellowship in Nephrology at Washington University School of Medicine, St. Louis. He joined the Ohio State University College of Medicine Faculty in 1990, became Director of the Division of Nephrology in 2004, and served as Vice Chairman of Medicine for Research from 2009-2019. In 2019 he became the Medical Director of the Ohio State University Center for Clinical Research Management.
Dr. Rovin has had several leadership roles in the American Society of Nephrology, including running the Glomerular Diseases Pre-Course and Co-Editing NephSAP-Glomerular Diseases. Most recently, he was appointed Deputy Editor of *Kidney International*,. He also is Co-Chair for glomerular disease guideline development for the Kidney Disease Improving Global Outcomes effort.
Dr. Rovin's laboratory studies the immunopathogenesis of glomerular and autoimmune diseases. He is heavily involved in clinical trial development and design for investigator-initiated and industry-sponsored trials. He is a founding member of NephroNet, a grass-roots nephrology clinical trial organization, and the Lupus Nephritis Clinical Trials Network. He is and has been the Principal Investigator on several trials of novel therapeutics for glomerular diseases.

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