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Targeting the Source of IgA Nephropathy

November 28, 2022

Forward Looking Statements

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Agenda for Today's Meeting

Opening Remarks	Marshall Fordyce, MD	*Founder and Chief Executive Officer* Vera Therapeutics
Patient Story	Liz	*IgAN Patient*
Disease Burden and Pathogenesis of IgAN	Jonathan Barratt, PhD, FRCP	*Leads the Renal Research Group within the College of Life Sciences at the University of Leicester*
Ongoing Phase 2b ORIGIN Study of Atacicept in IgAN	Celia Lin, MD	*Chief Medical Officer* Vera Therapeutics
Summary and Q&A	Marshall Fordyce, MD	*Moderator*

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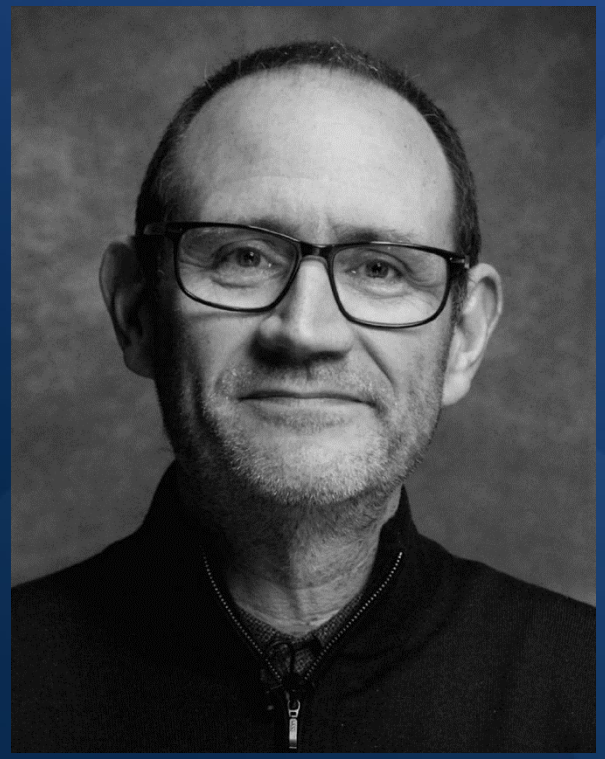
Jonathan Barratt, PhD, FRCP



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. Jonathan 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 FDA and American Society of Nephrology Kidney Health Initiative: Identifying Surrogate Endpoints for Clinical Trials in IgAN Workgroup.

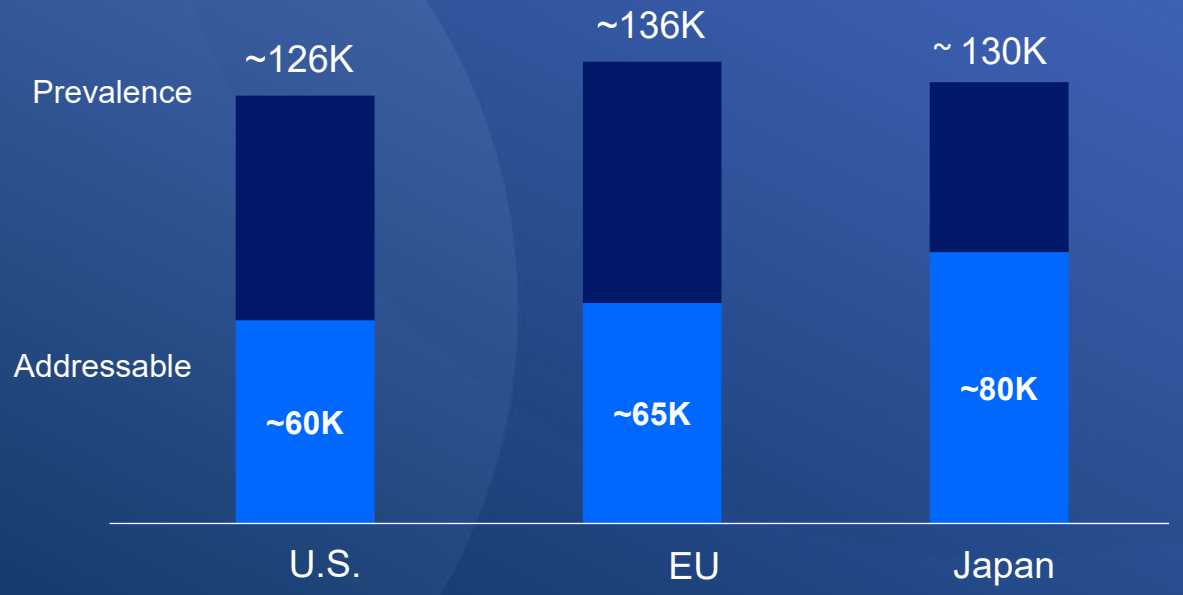
Disease Burden: IgA Nephropathy (IgAN) is the Most Common Primary Glomerulonephritis

Serious and progressive autoimmune disease of the kidney; average age of diagnosis 30 years old, severely impacting quality of life

Orphan Disease indication in the US and EU[1]

Up to 50% of IgAN patients progress to **end-stage renal disease**, resulting in need for **dialysis or transplant**

IgA Nephropathy Prevalence



- Significant need in the EU and Japan
- Higher incidence rates in Japan and other Asian countries

[1]Orphan Disease Designation not yet obtained for atacicept in IgAN. [2]Validation given recent FDA approval of TARPEYO; [3]ClearView Healthcare Partners Analysis 2021. All prevalence and addressable population were based on peak year forecast.

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IgAN Patient Characteristics:

Peak incidence in young adults ages 20-40

2:1 male predominance in the U.S. and Western Europe

Racial predilection for East Asians and white individuals

Presentation involves hematuria and proteinuria and can be asymptomatic

Screening has the potential for detection

Kidney biopsy is required for diagnosis

Effective Treatments are Lacking

Supportive Care

- Blood pressure optimization (systolic BP < 120 mmHg)
- Initial therapy with ACEi or ARB, irrespective of hypertension, if proteinuria is > 0.5 g/d

Corticosteroids

- Systemic steroids for severe cases and for limited timeframes
- Delayed-release budesonide approved for IgAN patients for a duration of 9 months

KDIGO 2021 Guidelines

- High-risk defined as UP > 0.75 – 1.0 gm/day
- Consider a 6-month course of glucocorticoid therapy: 2B (weak) recommendation
- Goal of therapy: UP < 1g/d

For patient at high risk of progression after adequate supportive care, KDIGO suggests enrollment in a clinical trial before all other consideration.

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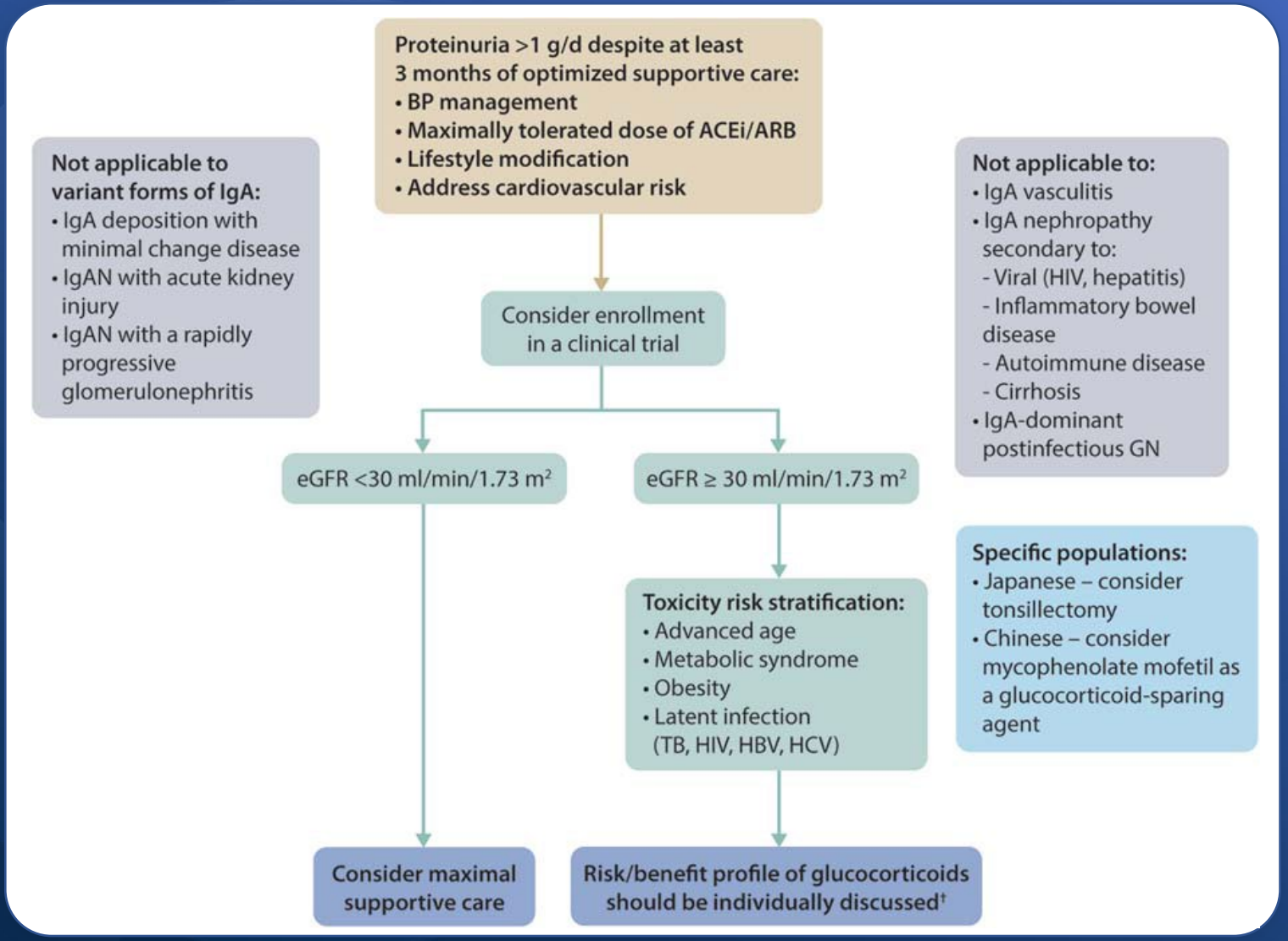
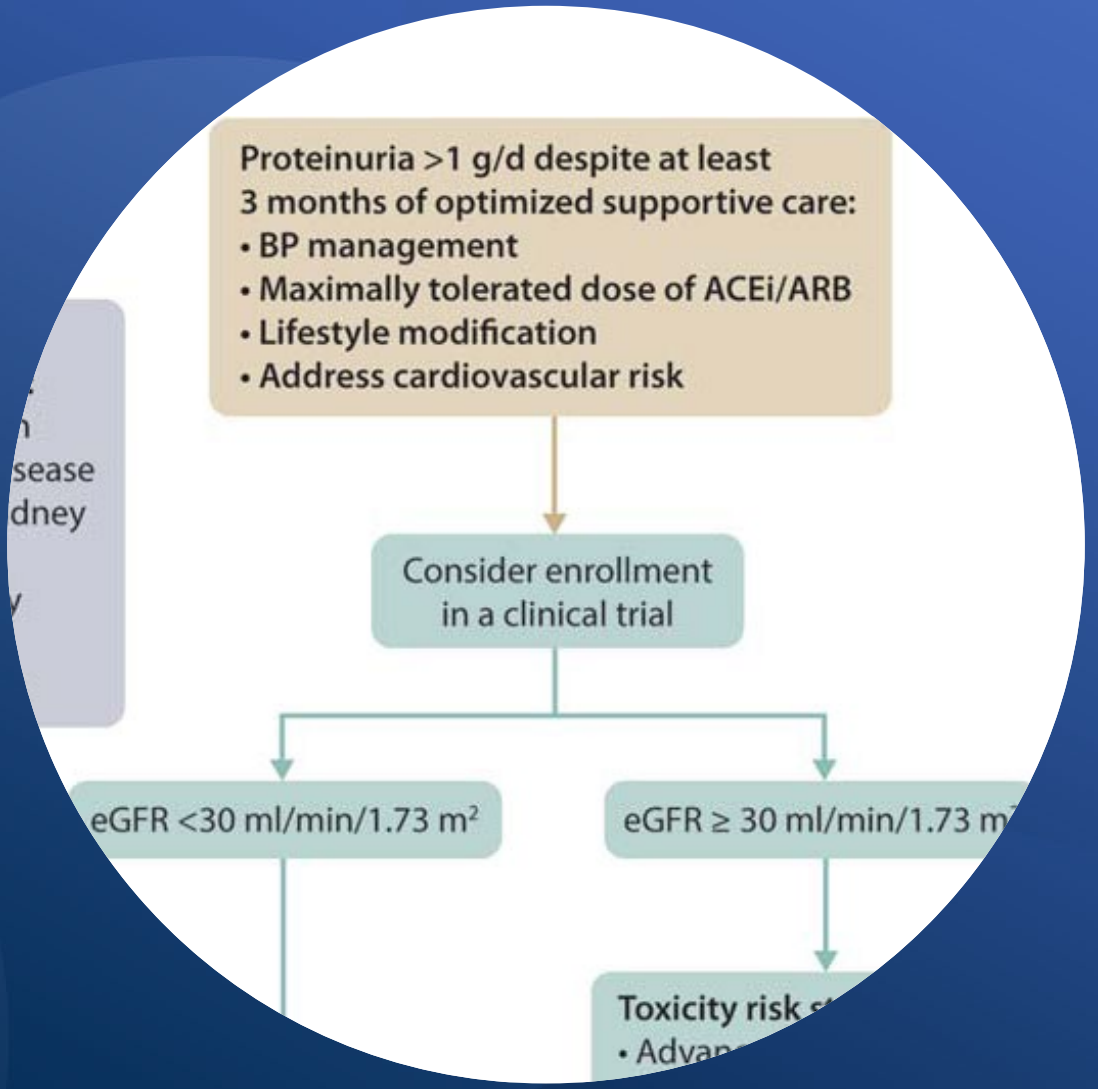
Risk Stratification and the Timing of Steroid Therapy

Guideline recommended management of patients with IgAN who remain at high risk for progression after maximal supportive care



Because no reliable disease modifying treatment exists, the KDIGO 2021 IgAN guidelines suggests referral to a clinical trial for patients at risk of progression **before consideration of glucocorticoids**



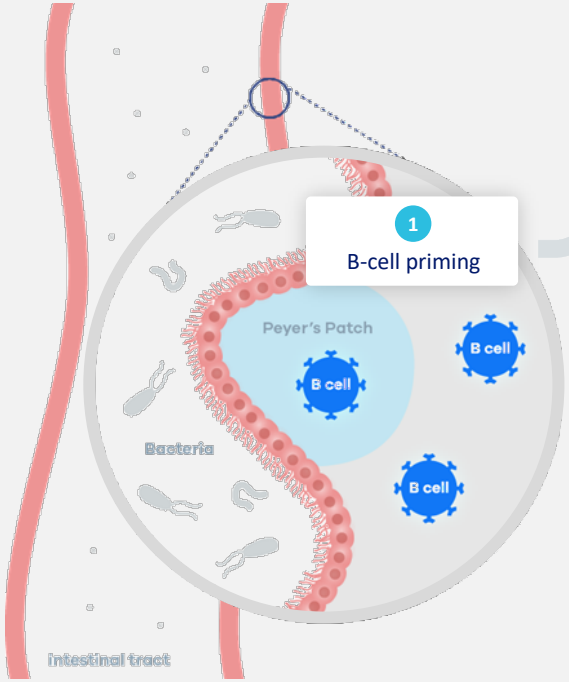
Unmet Medical Needs

Lack of disease modifying therapy	Avoidance or minimization of corticosteroids	Improve kidney function and reduce progression
• Supportive care such as RASi and blood pressure optimization does not target the source of the disease	• Steroid tolerability and complications • Limitation on long-term use	• Proteinuria is the premier surrogate marker for disease progression • Follow-on demonstration of eGFR improvement is important

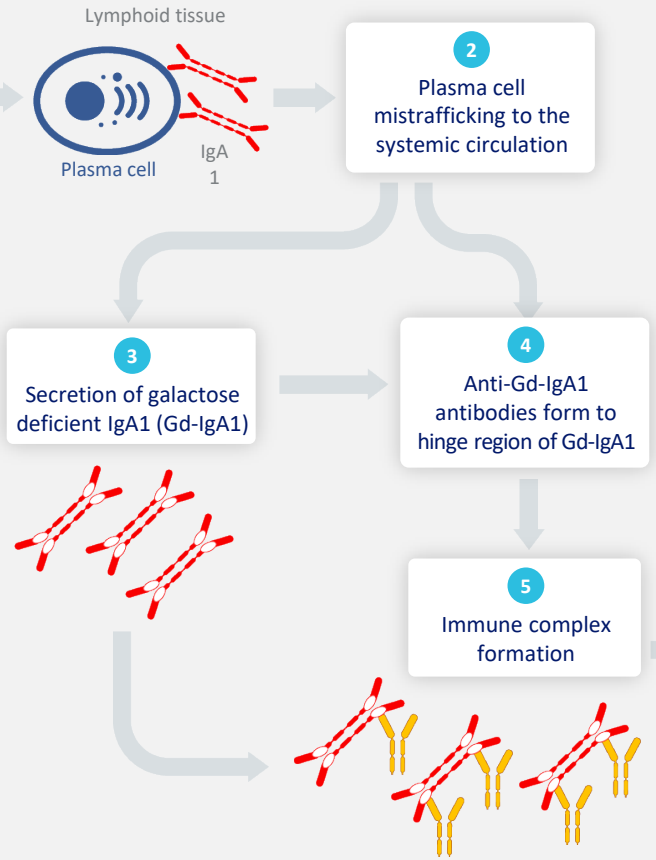
Overcoming the source pathogenesis of IgAN has the potential to address these needs.

Pathogenesis of IgAN

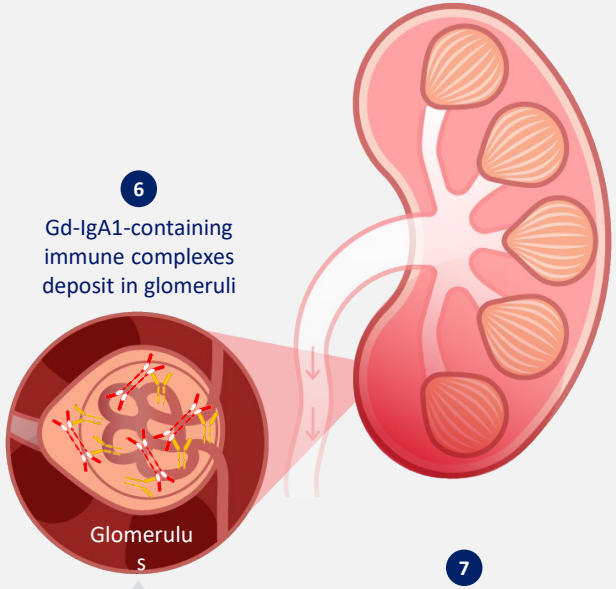
B-Cell Maturation and Class-Switching



Systemic Circulation



Glomerulonephritis



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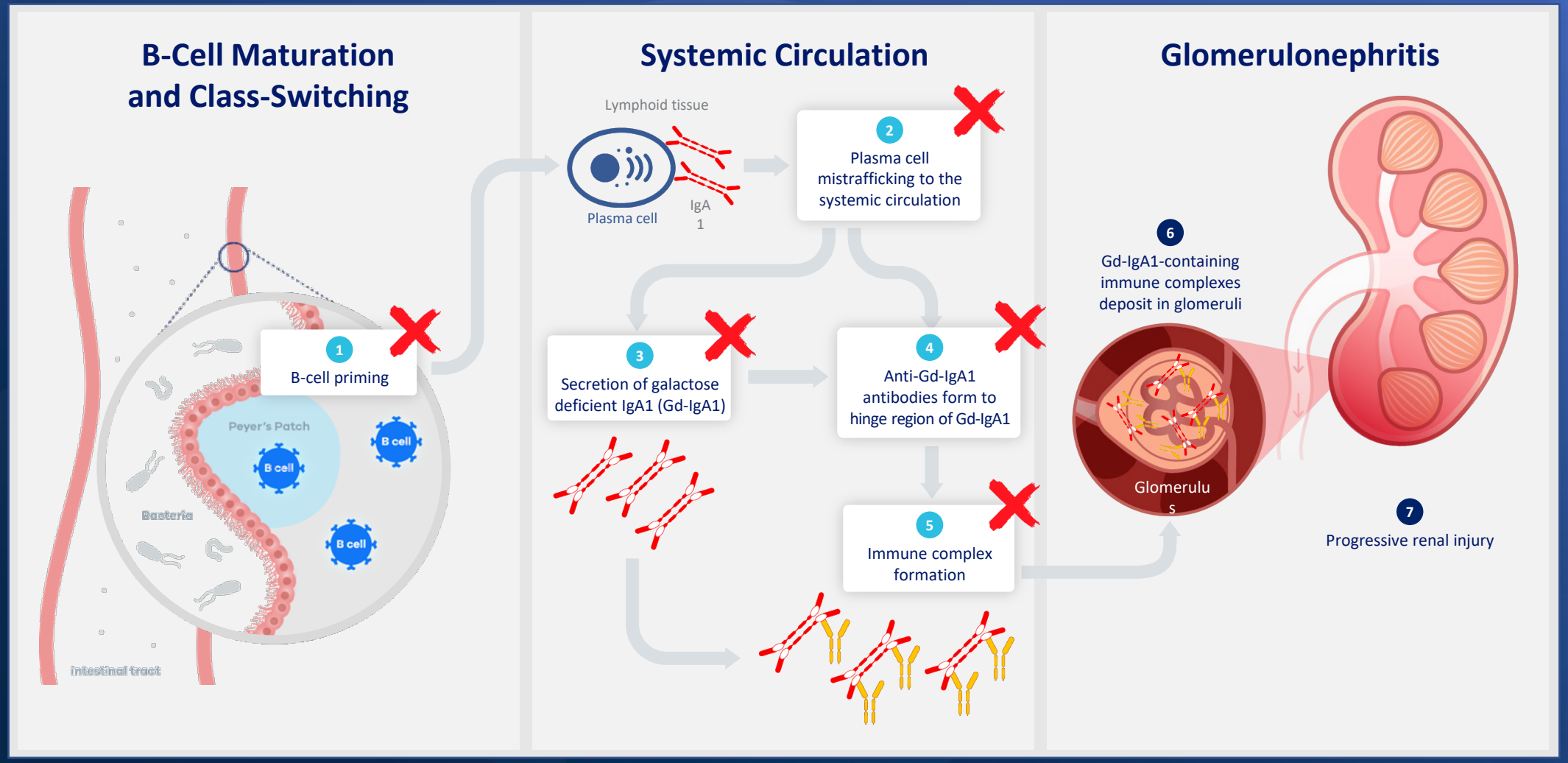
Atacicept Targets the Root of IgAN



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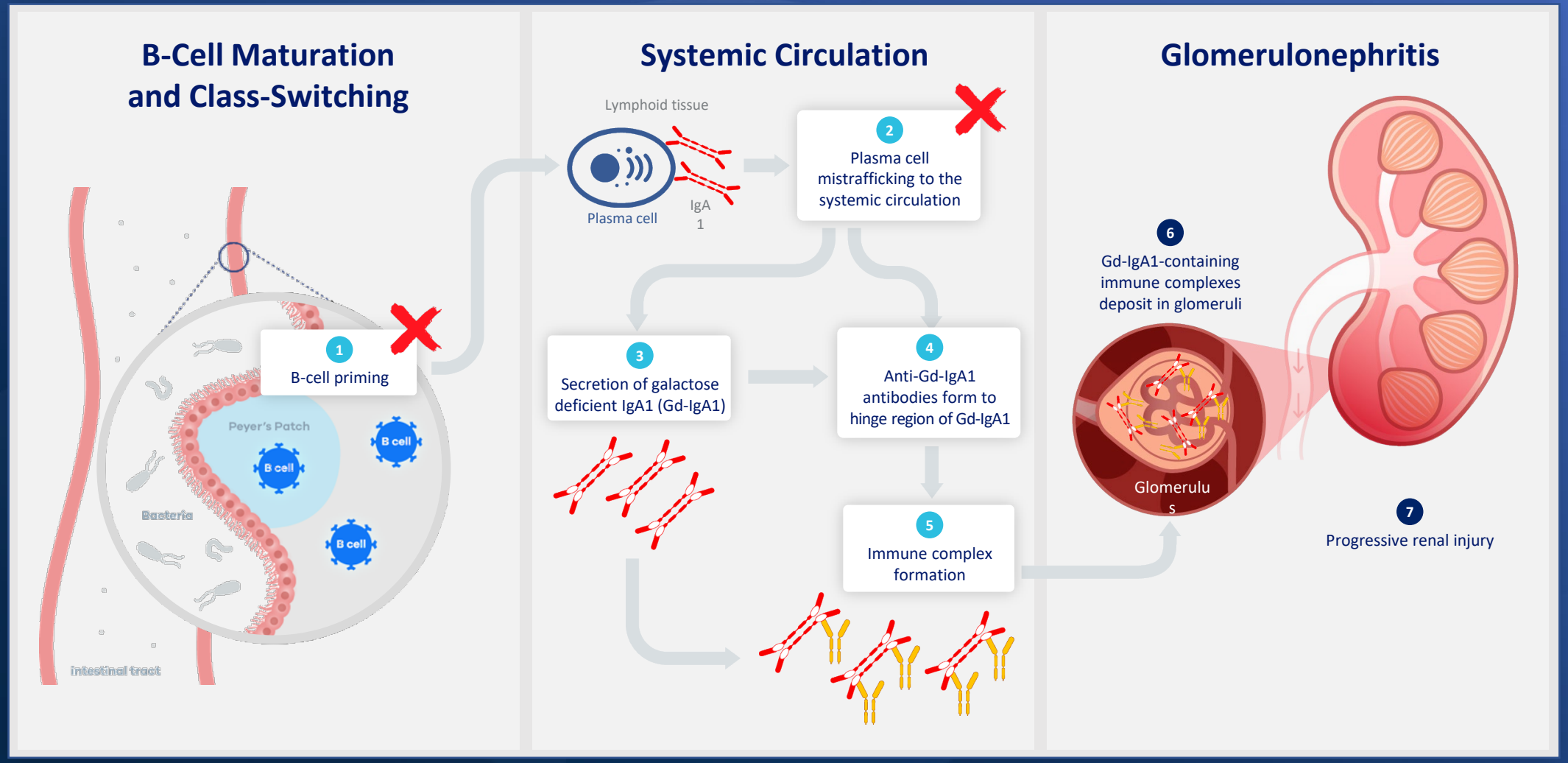
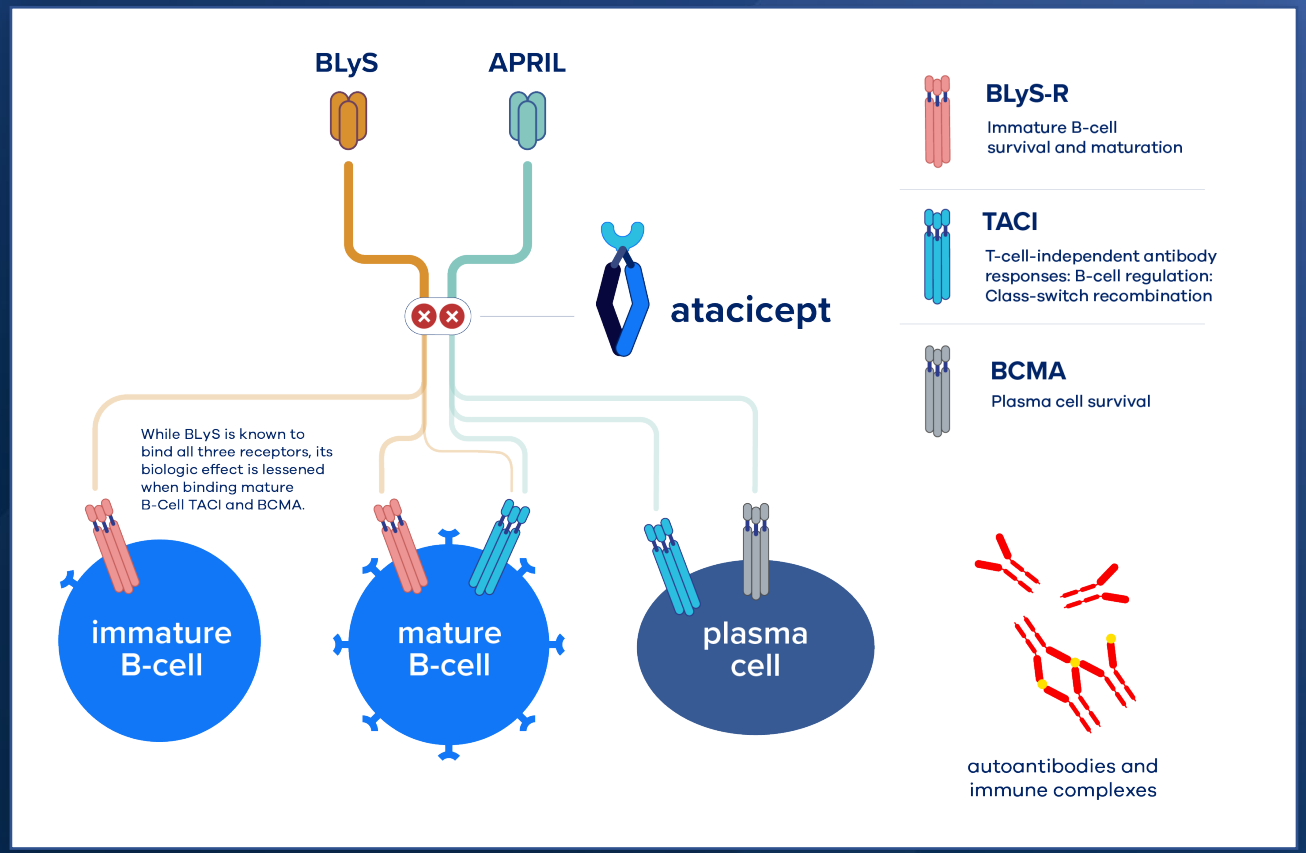
IgA Nephropathy Pathogenesis Begins with B Cells and Plasma Cells



= Steps that atacicept targets

Atacicept is a Dual Inhibitor (BLyS and APRIL) of B Cells and Plasma Cells



Key Considerations:

- Fully humanized fusion protein, subcutaneously administered
- Low nanomolar potency vs BLyS (Kd = 1.45 nM) and APRIL (Kd = 0.672 nM)
- Dual blockade by TACI-Ig shown to be more potent than blocking BLyS alone[1] and has benefit of targeting long-lived plasma cells[2], in addition to B cells, thus reducing autoantibody production[3]

1Haselmayer P et al. Eur J Immunol 2017;00:1–11. 2Hiepe F et al. Nat Rev Rheumatol 2011;3:170-178. 3Gordon et al. 2017 Arthritis & Rheumatology 69(1): 122-130.

Rationale for Dual Inhibition of BLyS and APRIL for IgAN Patients

- BLyS and APRIL levels are elevated in IgAN Patients
- BLyS and APRIL levels associate with IgAN disease severity clinically and pathologically
  - Increased BLyS levels are associated with increased severity of pathologic damage and increased mesangial IgA deposition density[1,2]
  - Increased APRIL is associated with higher proteinuria, lower eGFR, and increased Gd-IgA1 level[3]

Inhibition of BLyS and APRIL levels may have higher potency and enable feasible, low-volume subcutaneous dosing

- Because APRIL is a binding competitor to BLyS, APRIL-only blockade may leave BLyS unopposed, the effects of which are not known
- Elevated BLyS levels after B-cell depletion is hypothesized to reduce response rates and increase recurrence rates[4,5]

1. Cao, et al, Molecular Medicine Reports 21: 795-805, 2020; 2. Li, et al, Molecular Medicine Reports 10: 1469-1474, 2014; 3. Zhai, et al, Medicine Volume 95, Number 11, March 2016. 4. Lee, et al, Nature Reviews | Drug Discovery volume 20 | March 2021 |. 5. Ehrenstein, et al, Nature Reviews Rheumatology, 2016.

Demonstrated Tolerability Profile In an Integrated Safety Analysis of Over 1,000 Patients on Atacicept

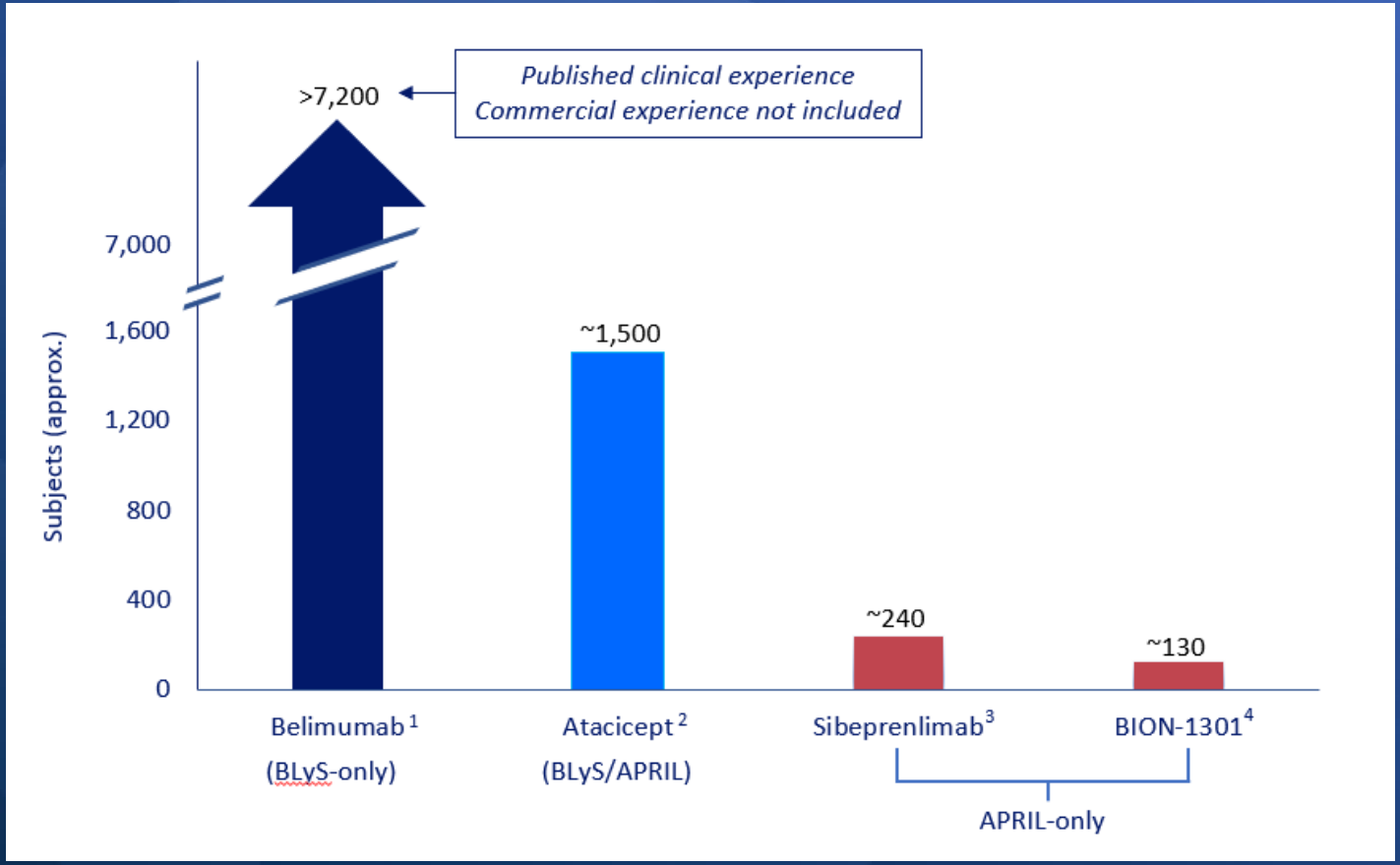
Infections rates comparable to placebo

	Placebo n=483	Atacicept 25 mg n=129	Atacicept 75 mg n=384	Atacicept 150 mg n=572	All subjects n=1568
Discontinuation due to AE	6%	11%	8%	8%	8%
Serious AE	11%	12%	13%	11%	11%
Severe AE	6%	8%	12%	10%	9%
Infections	44%	33%	47%	49%	46%
Serious infections	4%	1%	6%	4%	4%
Hypersensitivity	8%	6%	10%	10%	9%
Injection site reactions	11%	21%	28%	27%	22%
Cardiac arrhythmias	4%	9%	6%	4%	5%
Vestibular disorders	4%	4%	5%	5%	4%

Gordon, et al, Rheumatology Advances in Practice 2019;0:1–12
Studies included subjects with SLE/LN, rheumatoid arthritis, multiple sclerosis, and optic neuritis

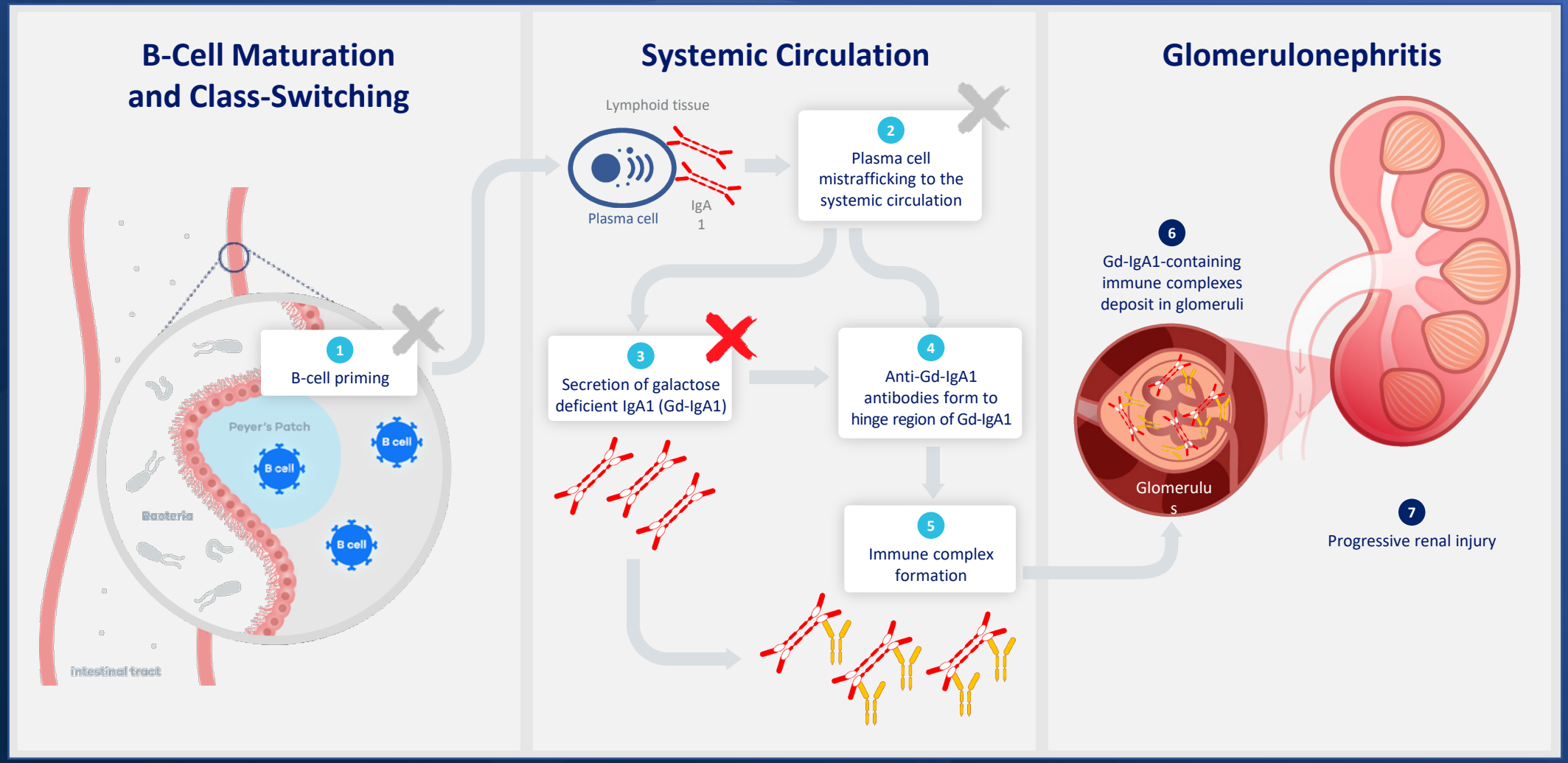
Well-Characterized Clinical Safety Database of Atacicept

Number of Subjects Administered at Least One Dose of Listed Medication as of November 2022 review of published literature



[1] Belimumab 10 years experience from clinical trials by Levy et al 2021 (excluded commercial experience); [2] Atacicept Integrated Safety Analysis by Gordon et al 2019 plus IgAN JANUS and ORIGIN studies; [3] Sibeprelimab two P1 healthy volunteer studies (Mathur et al 2022, Zhang et al ASN 2021 poster), P2 ENVISION study in IgAN (Kooienga et al ASN 2022 poster); [4] BION-1301 two P1 healthy volunteer studies (Chinook 4th CKD Summit presentation), P1/2 IgAN study (Barratt et al ASN 2022 poster), P1 MM study (Bensinger et al ASCO 2019 abstract)

Atacicept was First to Show ≥60% Reduction of Gd-IgA1 in IgAN Patients

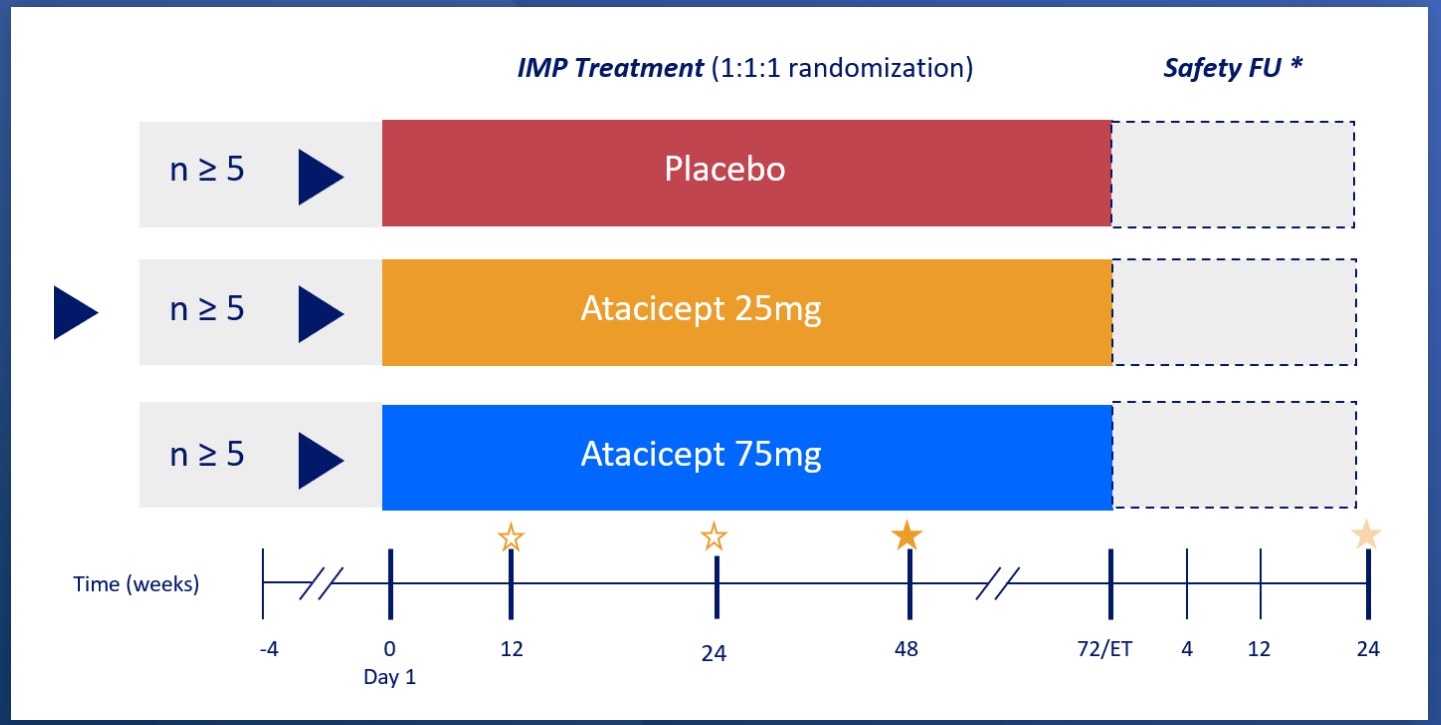


= Steps that atacicept targets

Phase 2a IgAN Trial (JANUS)

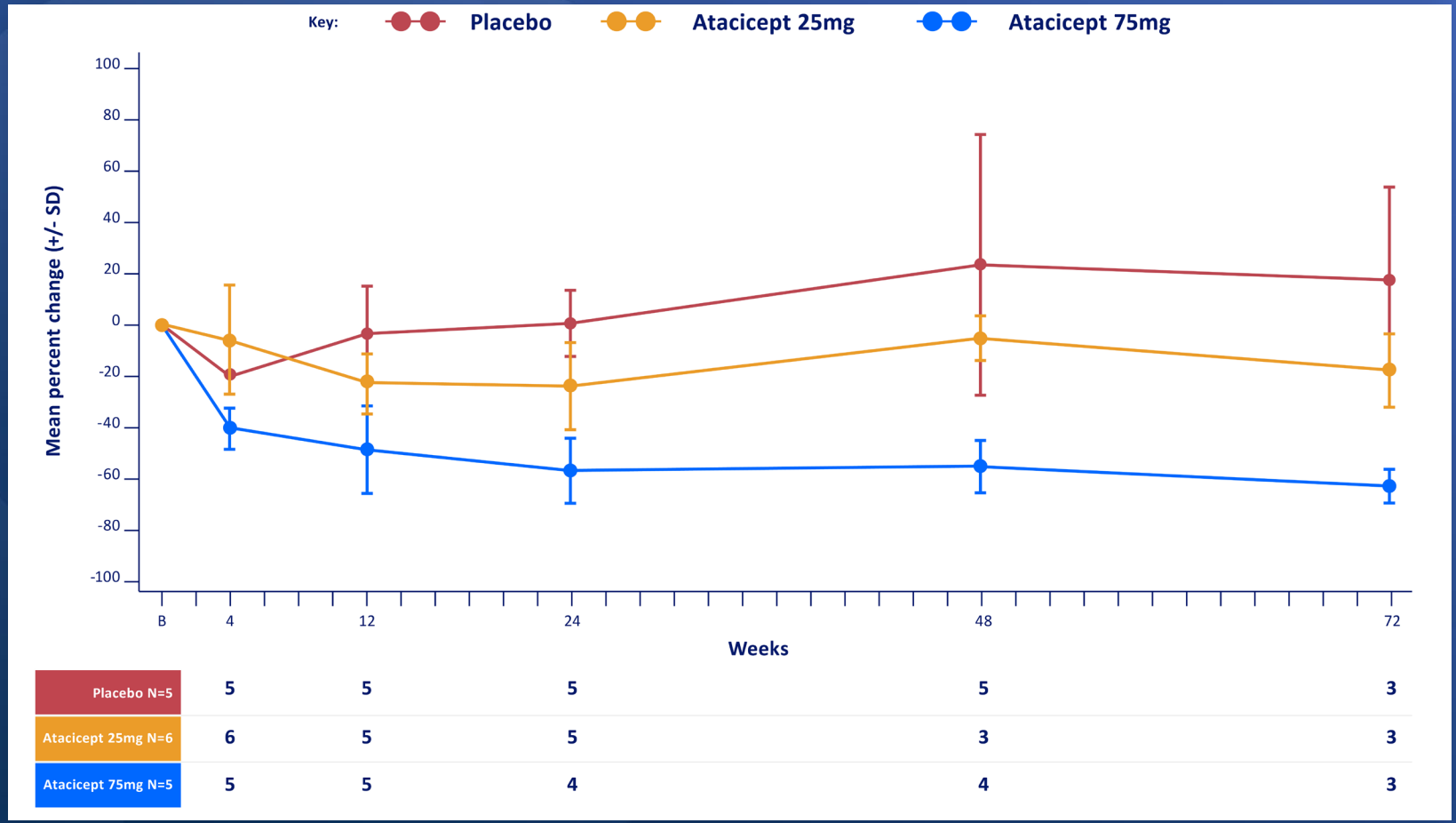
Study Design

- Patients ≥18 years with IgAN
- Proteinuria (UPCR) 0.75 to 6 mg/mg
- Stable ACE inhibitor and/or ARB ≥8 weeks



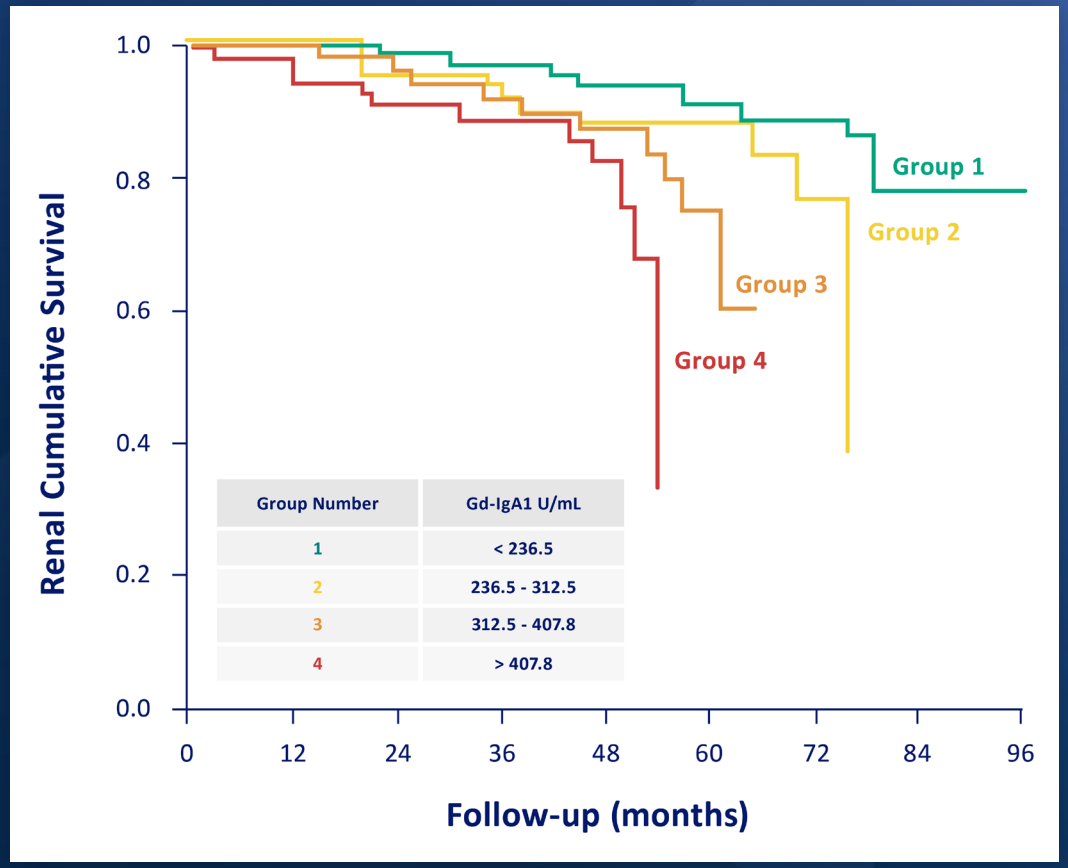
Atacicept Lowers Gd-IgA1 Antibody Levels

Clear dose-dependent reductions on serum Gd-IgA1 with atacicept, and atacicept 75 mg reduces Gd-IgA significantly (60%) and durably



[1] Barratt, J, et al, Kidney Int Rep (2022) 7, 1831–1841.

High Gd-IgA1 is Associated with Reduced Time to Dialysis, Transplant, and Death



High Gd-IgA1 (Group 4) is associated with increased risk of ESRD and death[1]

Serum level of glycan-specific IgG antibodies is correlated with the level of urinary protein excretion[2] and the risk of progression to ESRD or death[3]

1Zhao N et al. Kidney Int 2012. 2Suzuki et al. JCI 2009. 3Berthoux F et al. J Am Soc Nephrol 2012.

Atacicept 75 mg Decreased Serum Gd-IgA1 Levels by up to Two Quartiles

Gd-IgA1 level (ng/ml)	Quartile
< 3.13	1ST
3.13-5.01	2ND
5.01-7.75	3RD
> 7.75	4TH

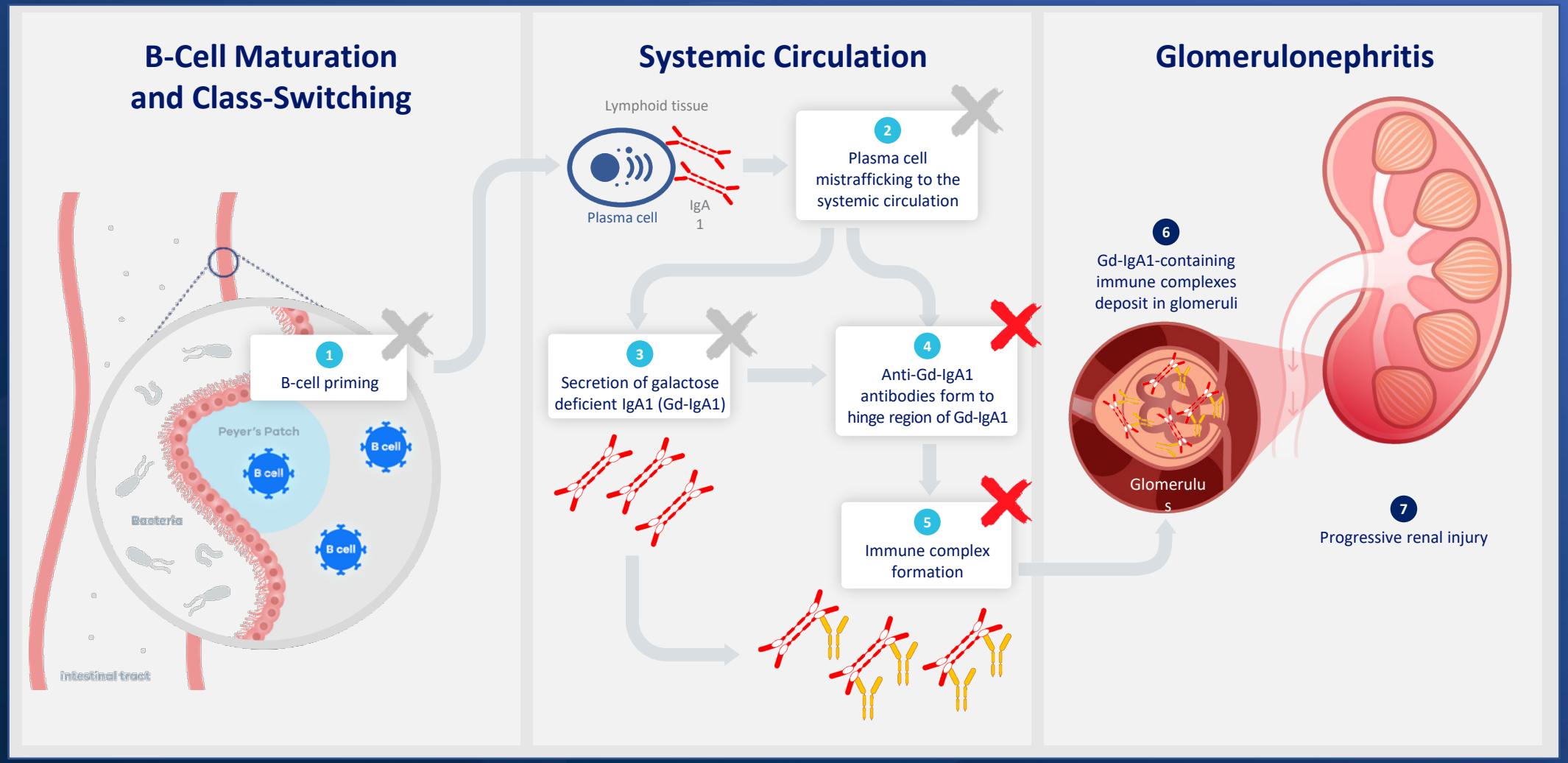
Quartiles determined by JANUS population

Subject	Allocation	Baseline	WEEK 4	WEEK 12	WEEK 24	WEEK 48	WEEK 72
1	Placebo	4TH	4TH	4TH	4TH	4TH	4TH
2	Placebo	4TH	3RD	4TH	4TH	4TH	4TH
3	Placebo	2ND	2ND	2ND	2ND	3RD	3RD
4	Placebo	2ND	1ST	2ND	2ND	2ND	
5	Placebo	4TH	3RD	4TH	4TH	4TH	
6	Atacicept 25mg	4TH	4TH	3RD	3RD	3RD	3RD
7	Atacicept 25mg	3RD	3RD	3RD	3RD	3RD	3RD
8	Atacicept 25mg	4TH	3RD	3RD	3RD		
9	Atacicept 25mg	2ND	2ND				
10	Atacicept 25mg	1ST	1ST	1ST	1ST		
11	Atacicept 25mg	2ND	2ND	1ST	2ND	2ND	2ND
12	Atacicept 75mg	3RD	1ST	1ST	2ND	1ST	
13	Atacicept 75mg	4TH	3RD	2ND	1ST	2ND	2ND
14	Atacicept 75mg	1ST	1ST	1ST	1ST	1ST	1ST
15	Atacicept 75mg	2ND	1ST	1ST		1ST	1ST
16	Atacicept 75mg	4TH	3RD	3RD	2ND		

After 24 Weeks, all subjects receiving atacicept 75mg had reductions in serum Gd-IgA1 to the lowest risk quartiles

[1] Barratt et al. American Society of Nephrology 2021

Atacicept Reduces Anti-Gd-IgA1 and Immune Complex Levels



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In JANUS, Atacicept Lowered anti-Gd-IgA1 and IgG-IgA Containing Circulating Immune Complexes

Anti-Gd-IgA1 in IgAN

Patients with elevated serum anti-Gd-IgA1 have a worse dialysis-free survival rate at 5 and 10 years post diagnosis[1]

Presented at ERA-EDTA 2022

Immune Complexes (ICs) in IgAN

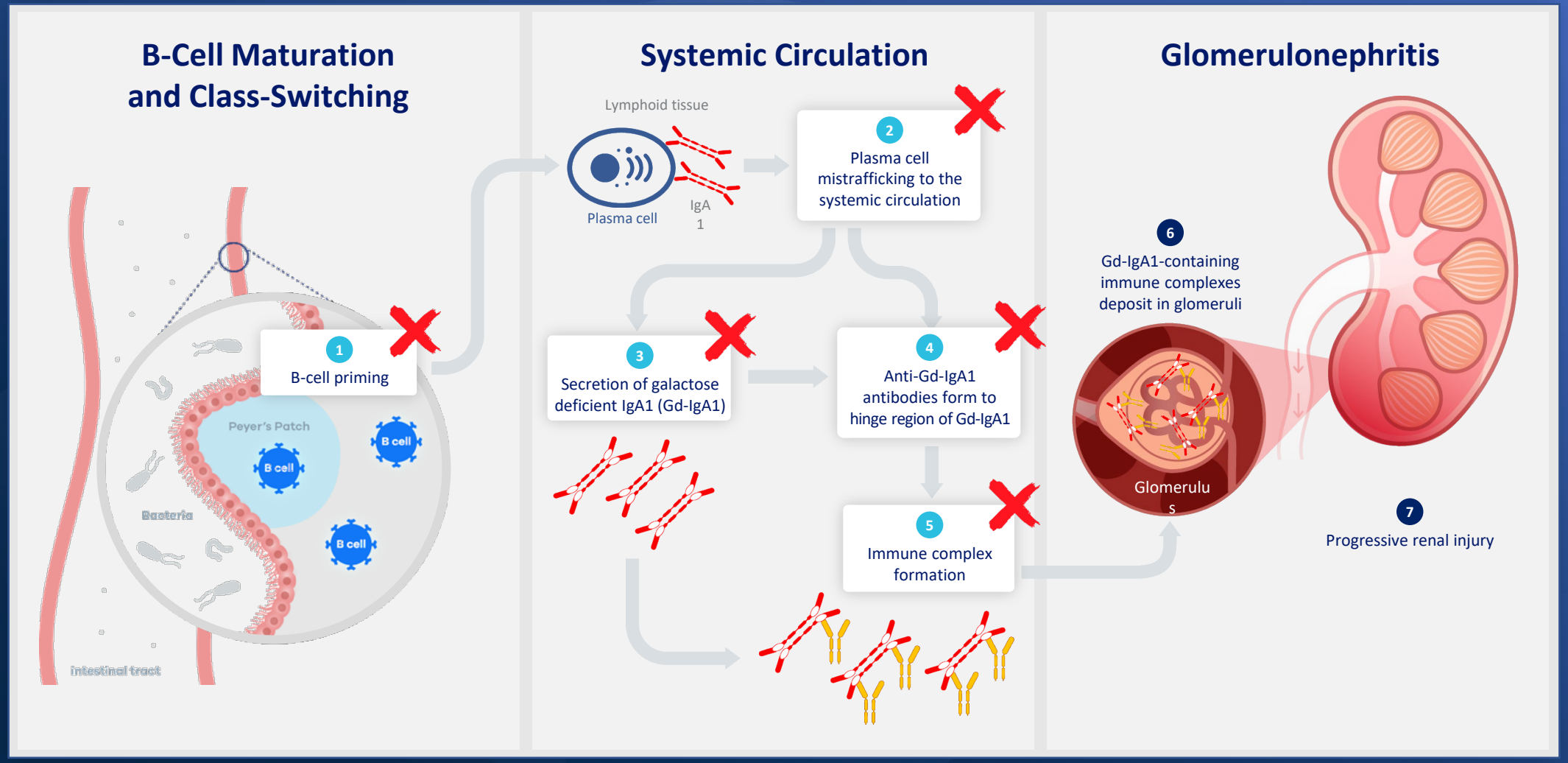
- Circulating ICs deposit in the glomerulus and lead to inflammation and disease[2]
- Clinical remission after therapy is accompanied by elimination of ICs from the kidney[3]
- IgAN recurs in kidney allograft recipients[3]

  Serum levels of Gd-IgA1, IgG autoantibodies, and IgA1–IgG ICs predict disease recurrence in renal allograft[4]

Presented at ASN 2022

1. Berthoux F et al. JASN 2012;1579-87. 2. Wyatt, et al, N Engl J Med 2013;368:2402-14. 3. Hotta, et al, American Journal of Kidney Diseases, Vol 39, No 3 (March), 2002: pp 493-502; 4. Berthelot, et al, Kidney International advance online publication, 10 June 2015

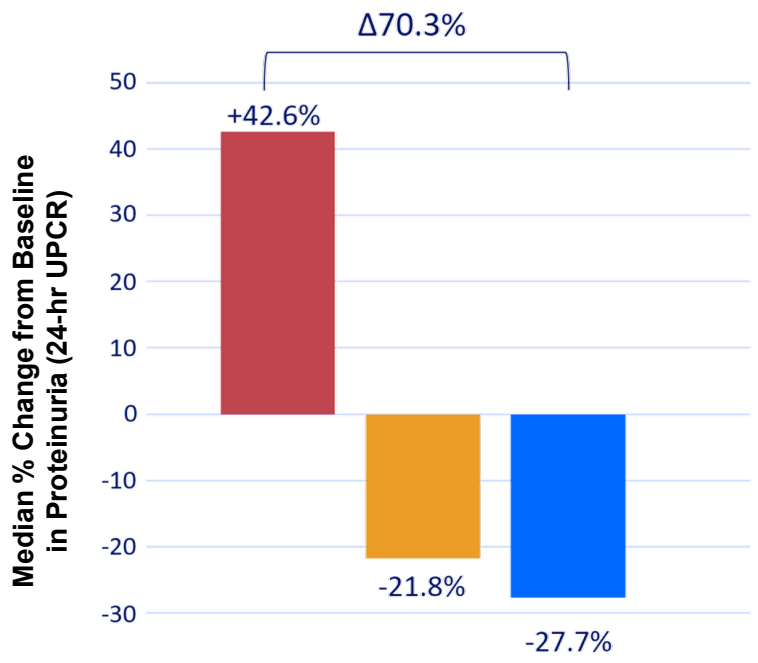
Atacicept Targets the Root of IgAN



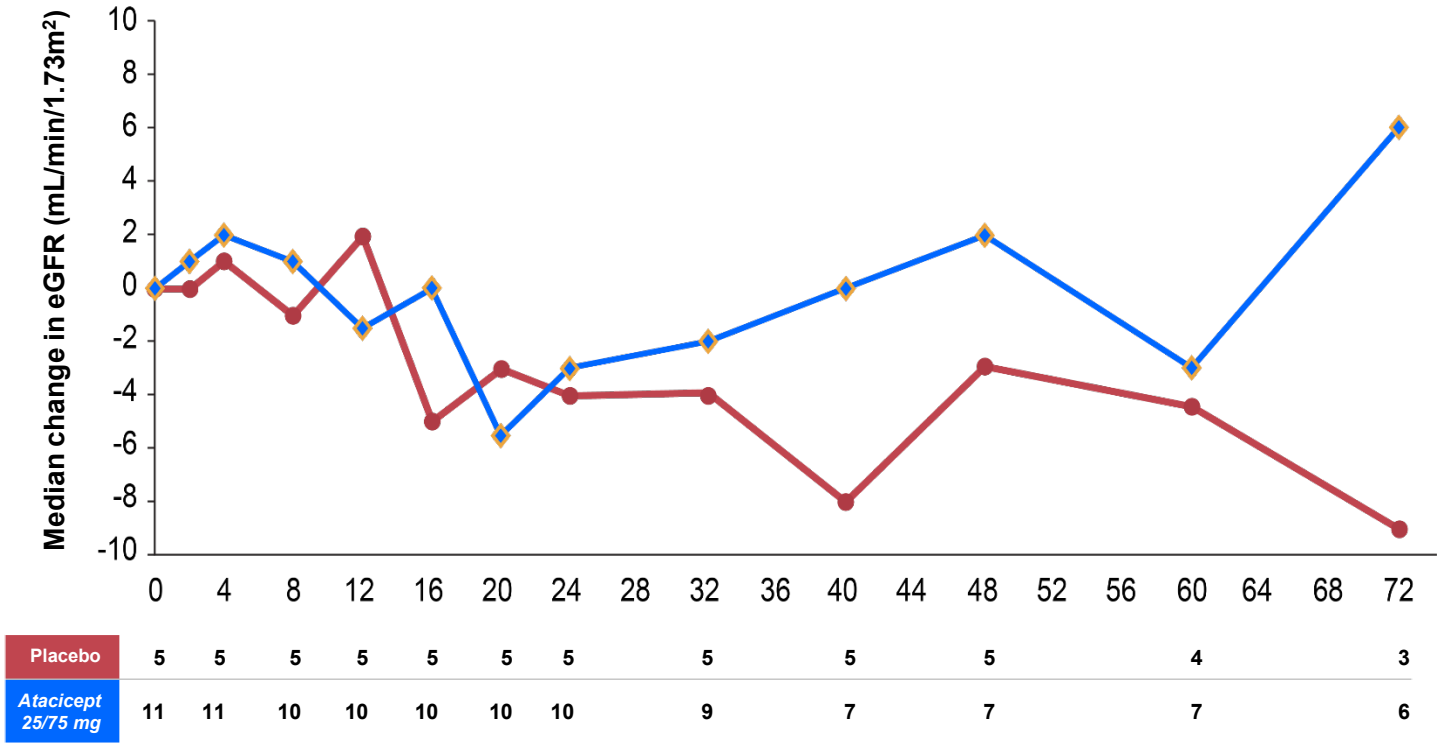
= Steps that atacicept targets

Targeting the Upstream Source Led to Clinical Results that Provide Proof-of-Concept for Atacicept

Change in Proteinuria by 24-hour UPCR at Week 24[1]



eGFR Change from Baseline[2]



Placebo	5	5	5	5	5	5	5		5		5		5			4		3
Atacicept 25/75 mg	11	11	10	10	10	10	10		9		7		7			7		6

Atacicept also Showed Stable eGFR for >1 Year vs 25% decline in Placebo

1 Barratt, J, et al, Kidney Int Rep (2022) 7, 1831–1841. 2 Internal analysis of JANUS CSR

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Summary

- Significant unmet need for disease-modifying therapies in IgAN
- BLyS and APRIL both have important signaling roles in IgAN
- Atacicept targets all upstream hits in IgAN pathophysiology, likely to improve renal function as measured by UPCR and eGFR (as shown in Ph 2a JANUS study)
- Atacicept has a well-characterized safety profile
- Atacicept showed trend of eGFR stabilization

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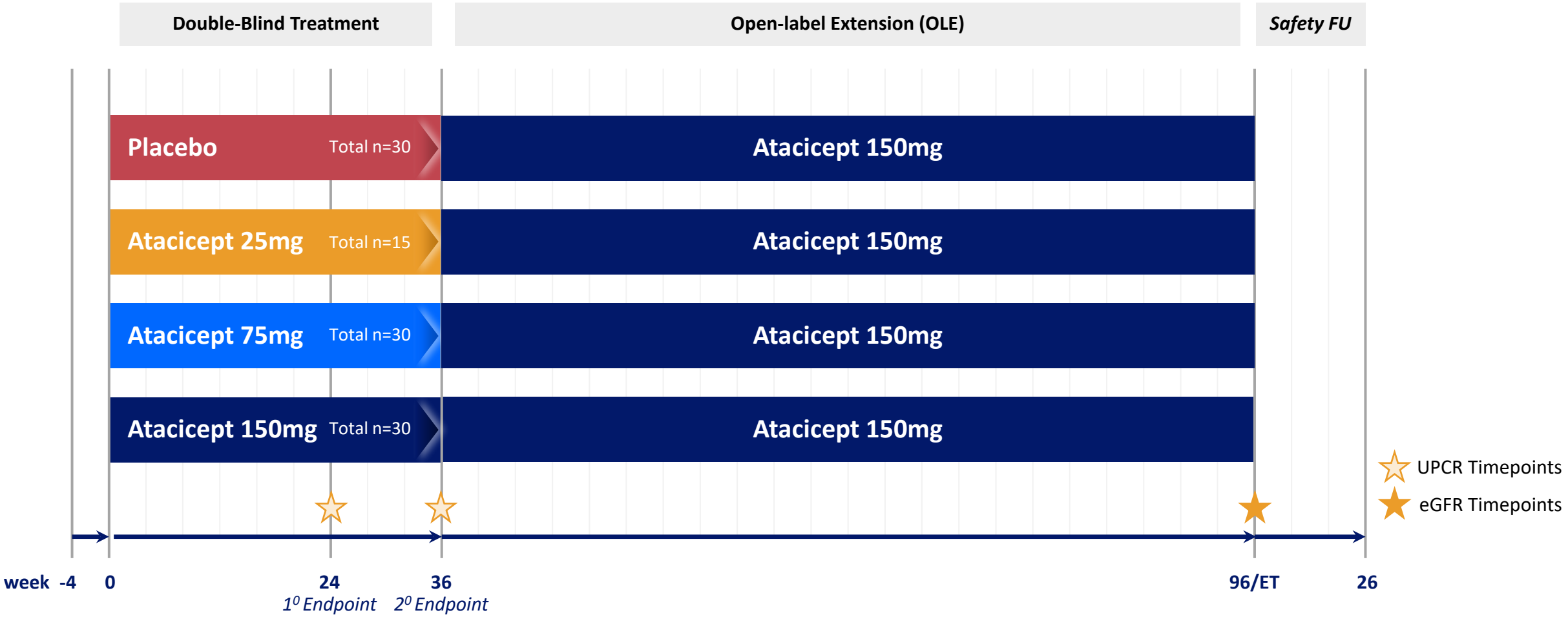
Ongoing ORIGIN Phase 2b Trial of Atacicept in IgAN

Phase 2b IgAN Trial (ORIGIN): Powered for Proteinuria 1^{0} Endpoint

Patients ≥18 years with IgA nephropathy and high risk of disease progression



ORIGIN Phase 2b Population and Endpoints

ORIGIN data-readout expected early Q1 2023

Inclusion Criteria

- ≥ 18 years old
- IgAN on renal biopsy
- UPCR-24h > 0.75 mg/mg or UP-24h > 0.75 g per 24h
- eGFR ≥ 30
- Stable and optimized RAASi for 12 weeks
- BP ≤ 150/90

Endpoints

- Primary efficacy
  - UPCR-24h at week 24
  - 80% power to detect a 28% difference between the combined 75mg and 150mg arm vs PBO
- Key Secondary
  - UPCR-24h at 36 weeks
- Exploratory
  - Rate of change eGFR through 36 weeks
  - Change in Gd-IgA1
- Safety

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We Believe Atacicept Has Best-in-Disease Potential in IgAN

B-Cell Modulators

Drug	atacicept	telitacicept	BION-1301	sibeprenlimab	Tarpeyo	sparsentan	atrasentan
Dose Regimen & Administration	75mg or 150mg Subcutaneous (One 1ml injection)	160mg or 240mg Subcutaneous (Three 1ml injections)	450mg IV or 600mg Subcutaneous (Two 2ml injections)	2-8mg/kg IV (Ph 2) 400mg SC (Ph 3) (One 2ml injection)	16mg Oral	200mg or 400mg Oral	0.75mg Oral
Mechanism	Dual BLyS/APRIL inhibition	Dual BLyS/APRIL inhibition	APRIL inhibition only	APRIL inhibition only	Corticosteroid (reformulated budesonide)	ETaR/AT1R antagonism	ETaR antagonism
Current Stage of Development	Phase 2b	Phase 2a (China only data)[4]	Phase 1/2	Phase 3	Marketed	Phase 3	Phase 3
Proteinuria Reduction vs Control	***(150mg data to come)***[1] 28% delta (75mg, week 24)[2]	49% delta (240mg, week 24)[4] 25% delta (160mg, week 24)[4]	N/A (open label only)	43% delta (week 36)[8]	29% delta (week 36)[5]	35% delta (week 36)[7]	N/A (open label only)
Gd-IgA1 Reduction vs Baseline	***(150mg data to come)***[1] 60% reduction (75mg, week 24)[2]	N/A	~65% reduction (n=5, 450mg IV, n=2, 600mg SC, week 24)[3]	N/A	~34% reduction (week 36)[6]	N/A	N/A
Safety	Well tolerated, comparable to placebo	Injection site reactions (~70%); No drop-outs[4]	1 pt had drug withheld due to IgG drop[3]	17% related to study drug; 7% drug interruption[9]	~20% drop-out[5]	N/A	~5-10% drop-out[3,8]

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.

*Approximate FD Market Cap as of 11/7/2022 FactSet. 1 150mg dose studied in Ph2b ORIGIN trial. 2 Barratt et al Kidney Int Rep. 2022. 3 Barratt et al ERA-EDTA 2022. Barratt et al ASN Kidney Week 2022. 4 Lv et al. ASN Kidney Week 2021. 5 TARPEYO Package Insert. 6Molyneux et al ASN Kidney Week 2022. 7Travere Press Release 8/16/2021. 8Rastogi et al ASN Kidney Week 2022. 9Kooienga et al ASN Kidney Week 2022. Data presented at "month 9" assumed to be at week 36. "N/A" indicates that either the drug was not evaluated in IgAN through a clinical trial, or it was evaluated in IgAN but this data point was not reported

Potential Value Creation Over Next 18 Months

Program	Indication	Catalyst	2022	2023	2024+
Atacicept	IgA Nephropathy	*Presented data on Gd-IgA1, anti-Gd-IgA1, and immune complexes from Phase 2a JANUS trial*	☑		
		Completed enrollment in Phase 2b ORIGIN trial	☑		
		Present 24-week data from ORIGIN trial		☐	
		Initiate Phase 3 trial		☐	
		Present topline Phase 3			☐
	Lupus Nephritis	*Initiated Phase 3 COMPASS trial*	☑		
		Present topline COMPASS data			☐
MAU868	BK Viremia in Renal Transplant	*Presented full results from Phase 2 trial*	☑		
		Initiate Phase 2b or Phase 3 trial		☐	

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

Conclusions

Lead clinical-stage asset, atacicept, is a potential disease-modifying agent with well-characterized clinical safety; MOA targets B-cells and plasma cells with **pipeline-in-a-drug potential**

BLyS and APRIL each play central roles in **B-cell and plasma cell modulation**

Atacicept targets all upstream hits of IgAN pathogenesis

Demonstrated tolerability profile in an integrated safety analysis of **over 1,000 patients on atacicept**

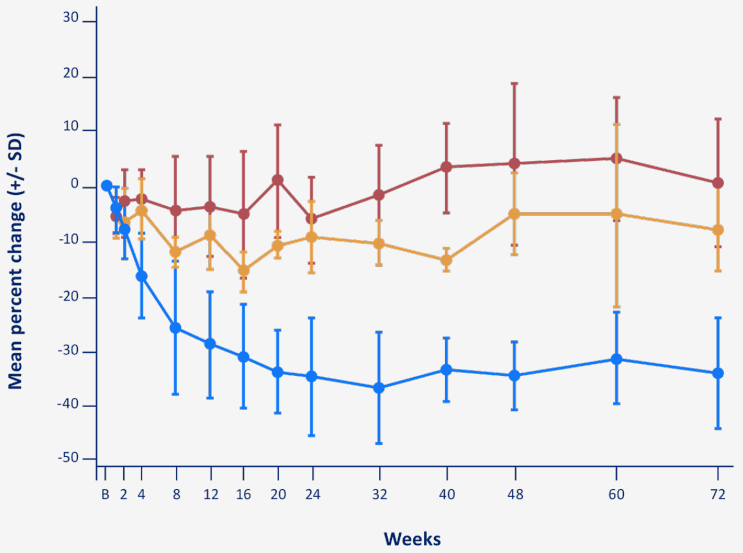
Phase 2b program in IgAN expected to **read-out early Q1 2023**

vera therapeutics

Appendix

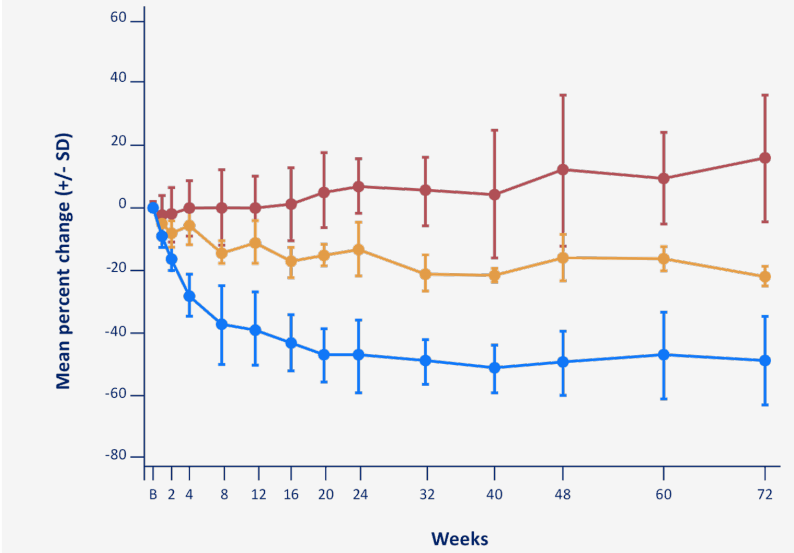
Phase 2a IgAN Trial (JANUS): Clear Dose-Dependent Reductions on Serum Igs

IgG



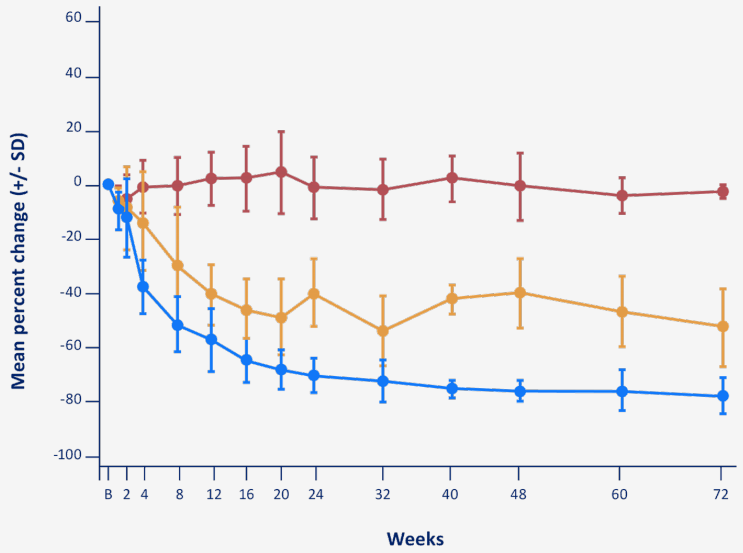
Placebo N=5	5	5	5	5	5	5	5	5	5	5	4	3
Atacicept 25mg N=6	6	6	5	5	5	5	5	4	3	3	3	3
Atacicept 75mg N=5	5	5	5	5	5	5	5	5	4	4	4	3

IgA



Placebo N=5	5	5	5	5	5	5	5	5	5	5	4	3
Atacicept 25mg N=6	6	6	5	5	5	5	5	4	3	3	3	3
Atacicept 75mg N=5	5	5	5	5	5	5	5	5	4	4	4	3

IgM



Placebo N=5	5	5	5	5	5	5	5	5	5	5	4	3
Atacicept 25mg N=6	6	6	5	5	5	5	5	4	3	3	3	3
Atacicept 75mg N=5	5	5	5	5	5	5	5	5	4	4	4	3

Key: Placebo Atacicept 25mg Atacicept 75mg