Corporate Presentation

December 2022

Forward Looking Statements

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Corporate Highlights

Our vision is to **change standard of care for patients with immunologic diseases**

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**

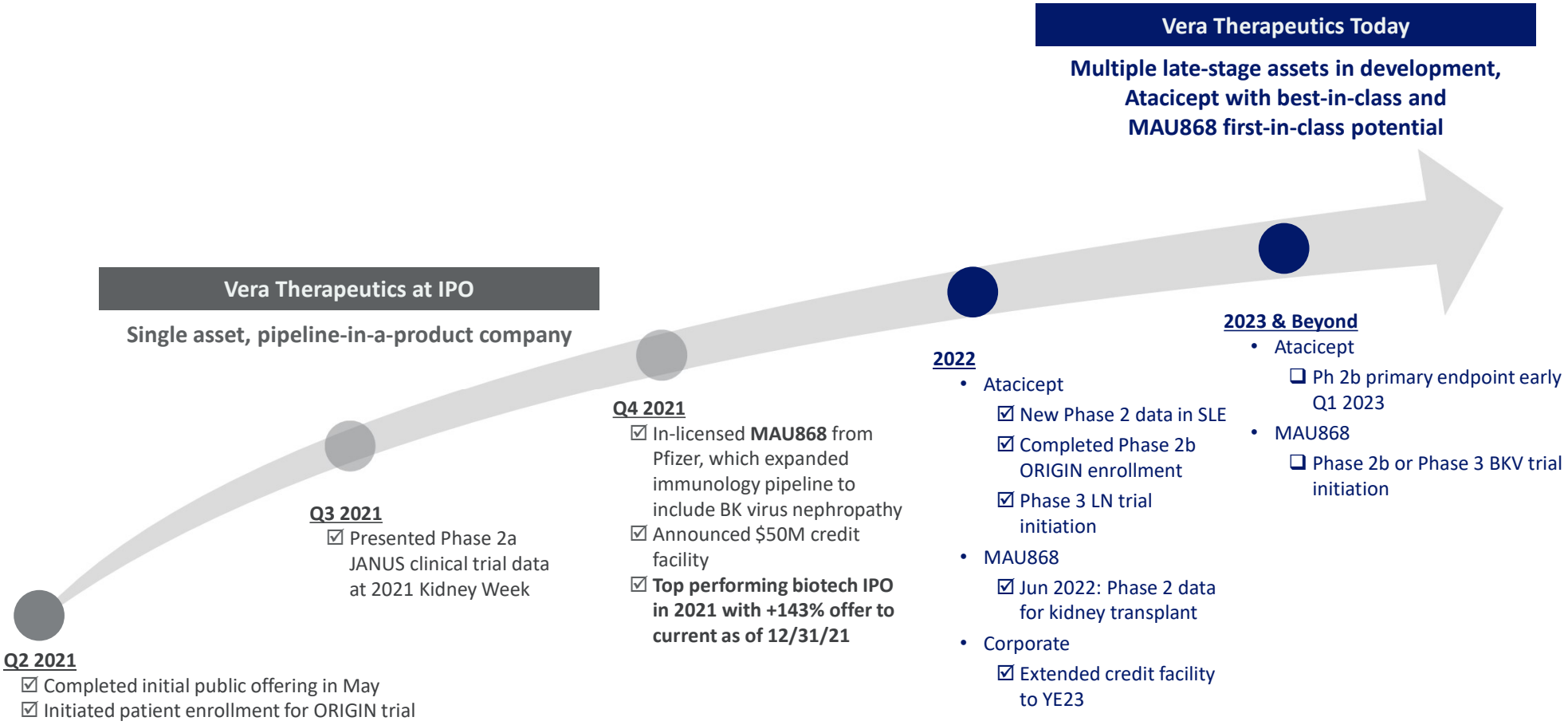
Phase 2b program in **IgA Nephropathy (IgAN),** clinical data in hand show **best-in-disease potential,** with **expected read-out early Q1 2023**

Initiated a Phase 3 program in Lupus Nephritis (LN), enabled by positive FDA feedback upon review of Phase 2 systemic lupus erythematosus (SLE) data and integrated safety data

Second late-stage asset, anti-BKV mAb, is a potential first-in-class agent targeting high unmet need condition with **encouraging proof-of-concept data** and expect to start a Phase 2b or 3 trial in 2023

Strong financial profile, **$134M cash and cash equivalents as of 9.30.22 (including Nov. debt drawdown)** and **access to a $25M credit facility** sufficient to fund operations to Q2 2024

Continued Momentum Into 2022 as the Top Performing Biotech IPO of 2021



Experienced Team with Successful Clinical and Commercial Track Record

Marshall Fordyce, M.D.
President and CEO

- >15 years drug development leadership
- Former Gilead, 7 new drug approvals, Project Lead for tenofovir alafenamide program

GILEAD

Celia Lin, M.D.
Chief Medical Officer

- >10 years drug development in Clinical Development and Medical Affairs at Genentech and Amgen
- Led Ph3 global trial execution in various therapeutics areas

Genentech AMGEN

Sean Grant, MBA
Chief Financial Officer

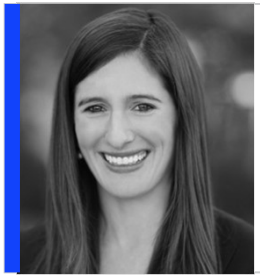
- >15 years in corporate strategy, finance, and capital raising
- Former healthcare banker with capital raising and M&A success

CareDx Your Partner in Transplant Care citi

Joanne Curley, PhD
Chief Development Officer

- >20 years drug development, former VP Gilead project and portfolio management

GILEAD



Lauren Frenz, MBA
Chief Business Officer

- >15 years industry experience, including global commercial planning and multiple blockbuster launches at Gilead
- Strategic advisory at SVB Leerink

GILEAD LEERINK

Joe Young, CPA, MBA
Chief Accounting Officer

- Leader of accounting & finance operations for public and private biotech companies, >20 years
- Big 4 audit background

Plexxikon EY cerus

Tom Doan
SVP, Clinical Operations

- >20 years of clinical operations experience
- Former Clinical Operations Therapeutic Area Head of Inflammation at Gilead

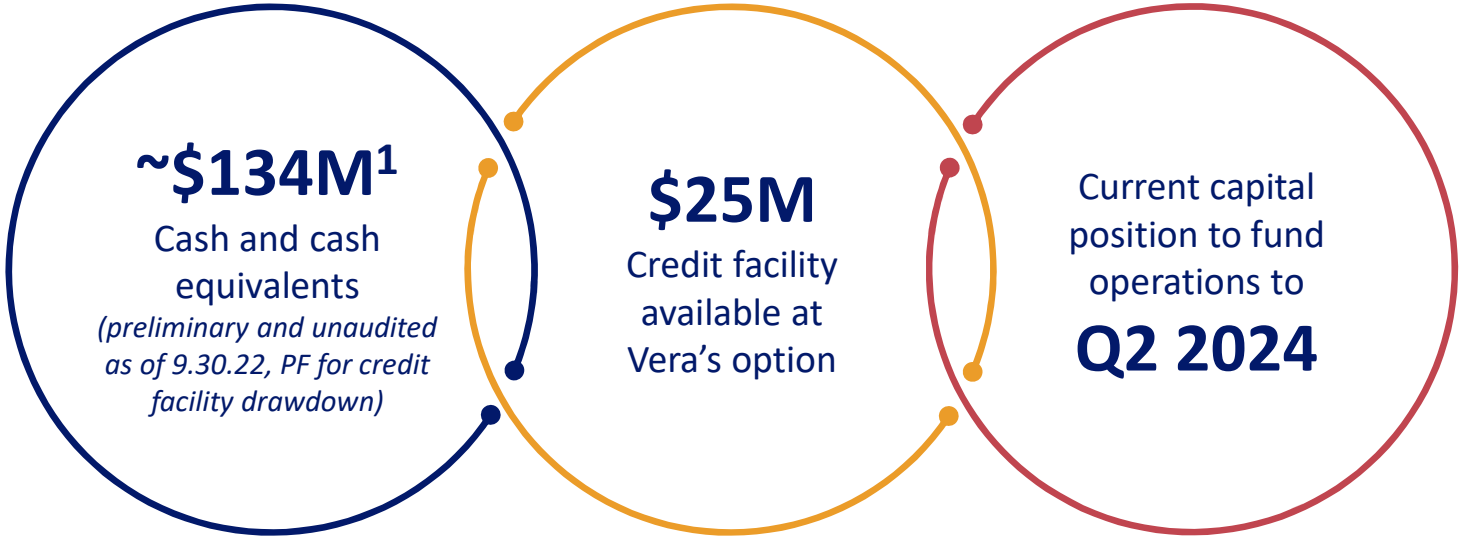
GILEAD Genentech

Board and Investors

ABINGWORTH Life Science Investing
RACapital
G/
MERCK
KLEINER PERKINS
Longitude CAPITAL
SOFINNOVA INVESTMENTS
VIVO CAPITAL
Fidelity INVESTMENTS
SURVEYOR
Octagon

vera therapeutics

Vera's Financial Position



[1] $114.4M of cash, cash equivalents, and marketable securities as of Sep 30, 2022. $134M is pro forma for $20M drawdown of Oxford debt facility in Nov 2022.

Strategic Vision: Develop Transformative Therapeutics for Immunologic Diseases

- Lead indications with **large markets and validating clinical data**
- Vera has **worldwide, exclusive licenses to develop and commercialize** atacicept from Merck KGaA and MAU868 from Pfizer/Novartis
- Experienced corporate development team with a **strategic focus to develop and commercialize novel therapies for immunologic diseases**

Atacicept

IgA Nephropathy (IgAN)

- Completed enrollment for Phase 2b trial, **primary endpoint data expected early Q1 2023**
- Tarpeyo approval sets precedent for UPCR endpoint

Lupus Nephritis (LN)

- Phase 2 results in SLE show **clinical efficacy in severe patients**
- Initiated Phase 3 trial

Additional Autoimmune Indications

- Dual inhibition of BLyS and APRIL **reduces disease-associated antibodies** with dose-dependence
- Potential for **best-in-class for B-cell targeting biologics**

MAU868

BK Virus Nephropathy

- Late-stage clinical asset for BK viral disease
- No currently FDA approved therapies
- Phase 2 **full results presented in Nov 2022**

Vera's Late-Stage Pipeline

Program	Indication	Phase 1	Phase 2	Phase 3	Next Milestone	Global Rights
Atacicept						
Fusion protein that blocks BLyS and APRIL	IgAN				Ph 2b primary endpoint early Q1 2023 ORIGIN	vera therapeutics
	Lupus Nephritis				Ph 3 primary endpoint 2026 COMPASS	vera therapeutics
MAU868						
Monoclonal antibody that neutralizes BK virus	BK Viremia in Renal Transplant				Ph 2b or Ph 3 trial initiation 2023	vera therapeutics
	BKV Cystitis in Hematopoietic Stem Cell Transplant (HSCT)	*Under Exploration*			Undisclosed	vera therapeutics

Atacicept for IgAN

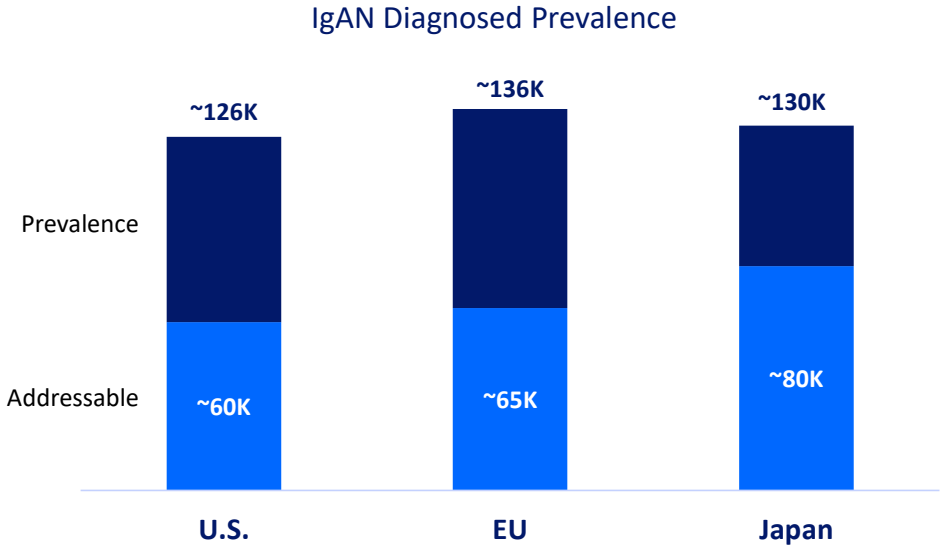
IgA Nephropathy (IgAN): Multi-Billion Dollar Market Opportunity

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**

~$6-10B Annual Market Opportunity Globally (US, EU, and Japan) for Novel IgAN Therapeutics[2]



- Higher incidence rates in Japan and other Asian countries

[1]Orphan Disease Designation not yet obtained for atacicept in IgAN. [2]ClearView Healthcare Partners Analysis. Prevalence and addressable population estimates based on peak year forecast.

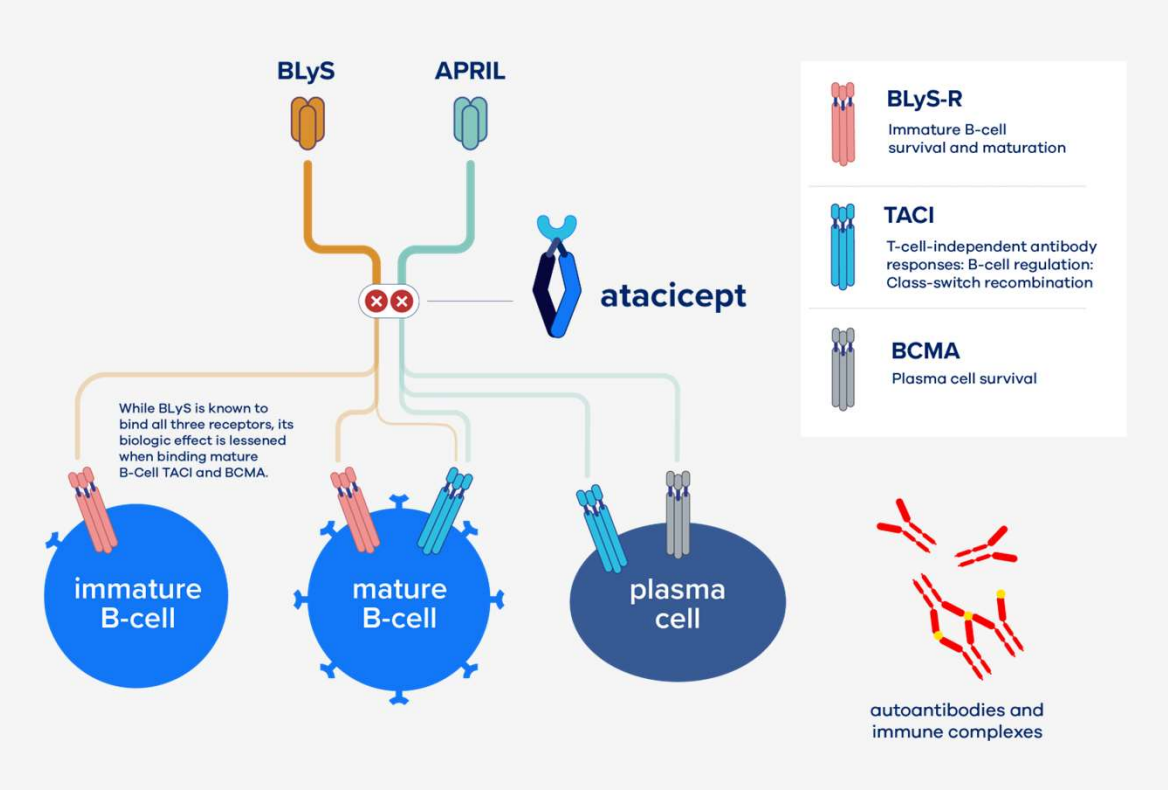
Current Standard of Care is Suboptimal

ACE inhibitors, ARBs	+/–	Corticosteroids (systemic)
• Blood pressure control • ACEi and ARB • **Limited clinical benefit**		• Immunosuppressive therapies used in severe cases • **Systemic steroids** used, but high dropout rate in randomized controlled trials due to well-known **acute + chronic steroid side effects**[1,2]

Standard of care poised for a disease-modifying biologic aiming to replace steroid use

[1,2] Lv J. et al. JAMA 2017. Manno C. et al. Nephrol Dial Transplant 2009.

Atacicept is a Dual Inhibitor (BLyS and APRIL) of Plasma Cells and B Cells with Potential to Address Multiple Autoimmune Diseases

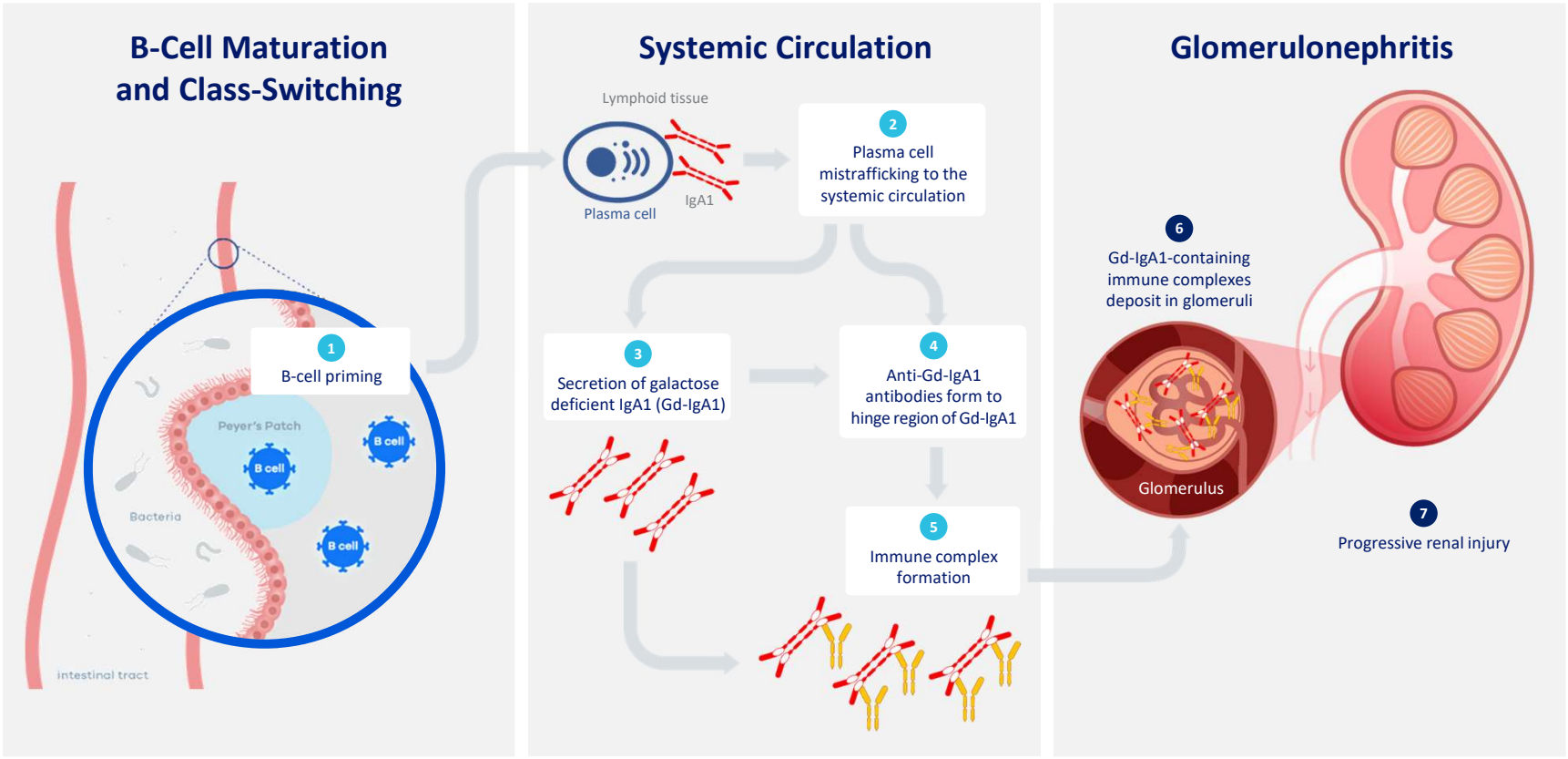


Key Considerations

- 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]
- Blocking BLyS alone works for SLE and LN, but may not be potent enough for IgAN

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

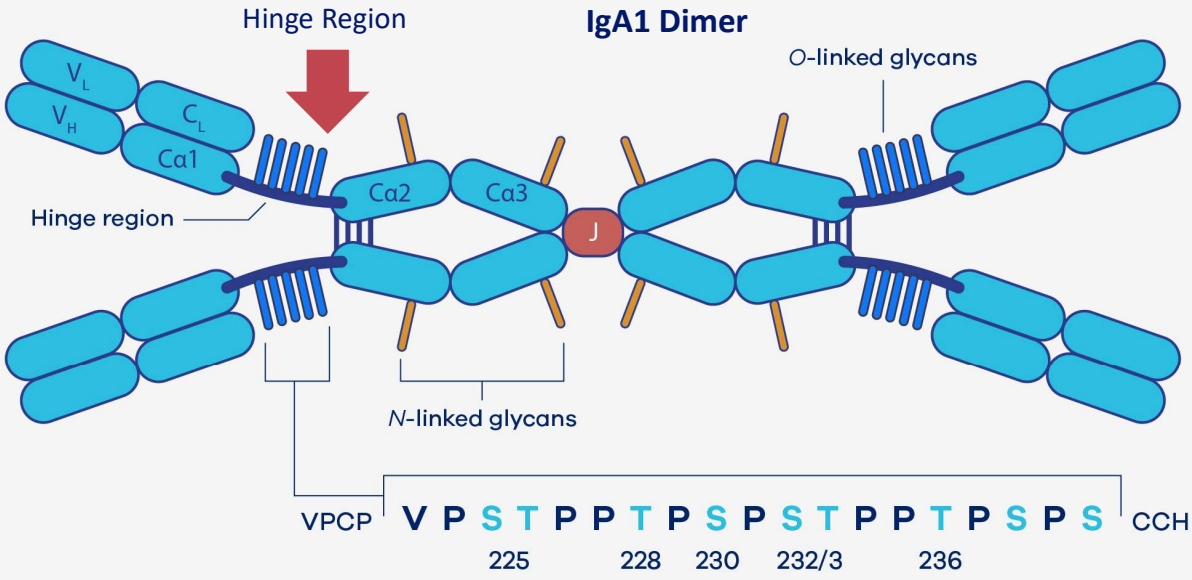
Atacicept Targets All Upstream Hits of IgAN Pathogenesis



= Steps that atacicept targets

Galactose-deficient IgA1 (Gd-IgA1) Plays a Central Role in IgAN Pathogenesis

*Source of autoantibodies is the **hinge region** of Gd-IgA1*



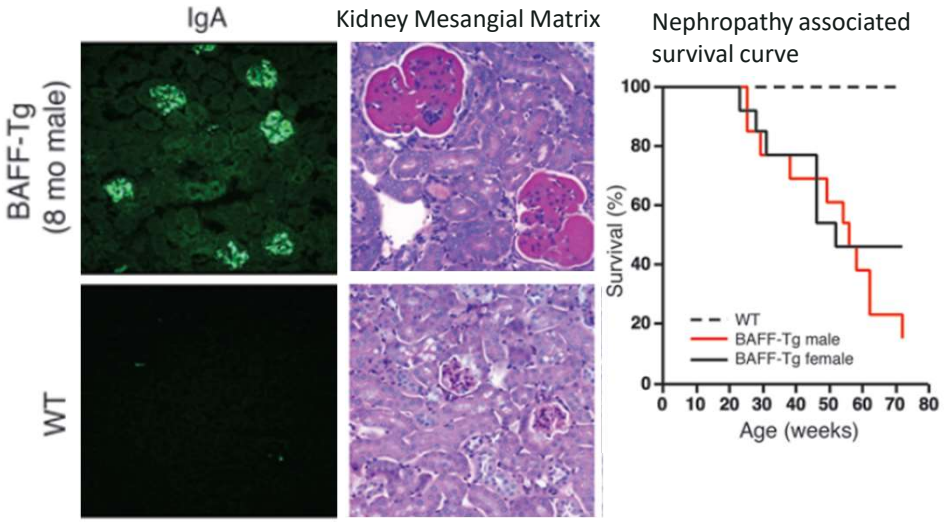
Gd-IgA1 and autoantibodies (IgA, IgG) represent disease-modifying targets for IgAN

Lai et al. Nature Reviews 2016.

BLyS Plays a Central Role in IgAN Pathogenesis and Key to Potent B-Cell Inhibition

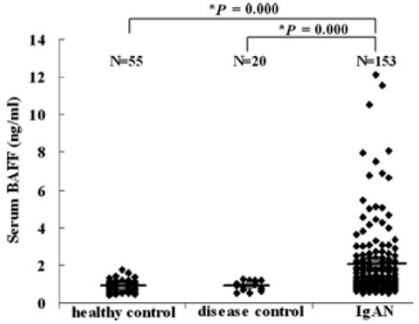
BLyS Overexpression Leads to Kidney IgA Deposits and Nephritis in Animal Model [1]

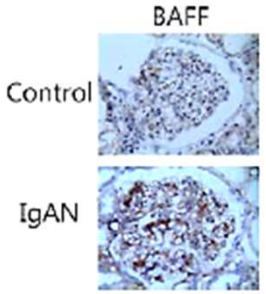
BLyS transgenic nephritic mice show mesangial IgA deposition, high circulating levels of aberrantly glycosylated polymeric IgA1 and nephropathy with age



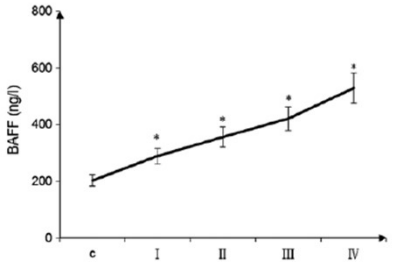
BLyS Levels are Elevated in IgAN Patients and are Associated with Disease Severity [2,3]

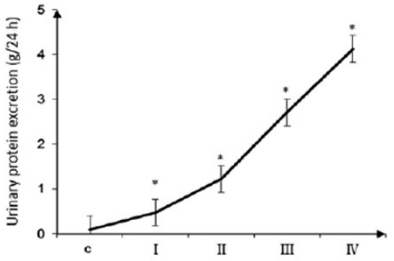
BLyS serum and renal levels are elevated in IgAN patients [2,3]





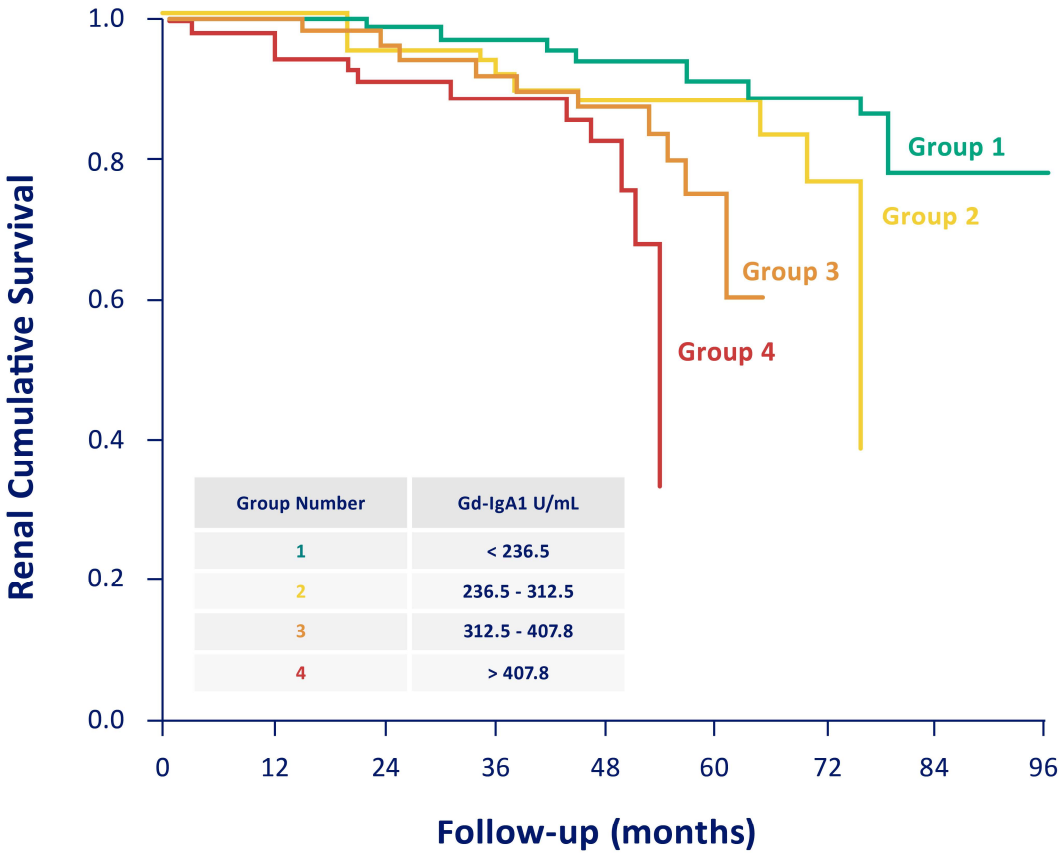
BLyS plasma levels are increased with increasing glomerular pathological score and are correlated with proteinuria [3]





[1]McCarthy D et al. JCI 2011. [2]Xin G et al. JNephrol 2013. [3]Cao Y et al. Molecular Medicine Reports 2020.

High Gd-IgA1 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]

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

Atacicept 75mg Decreased Serum Gd-IgA1 Levels to the Lowest Risk Quartiles

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

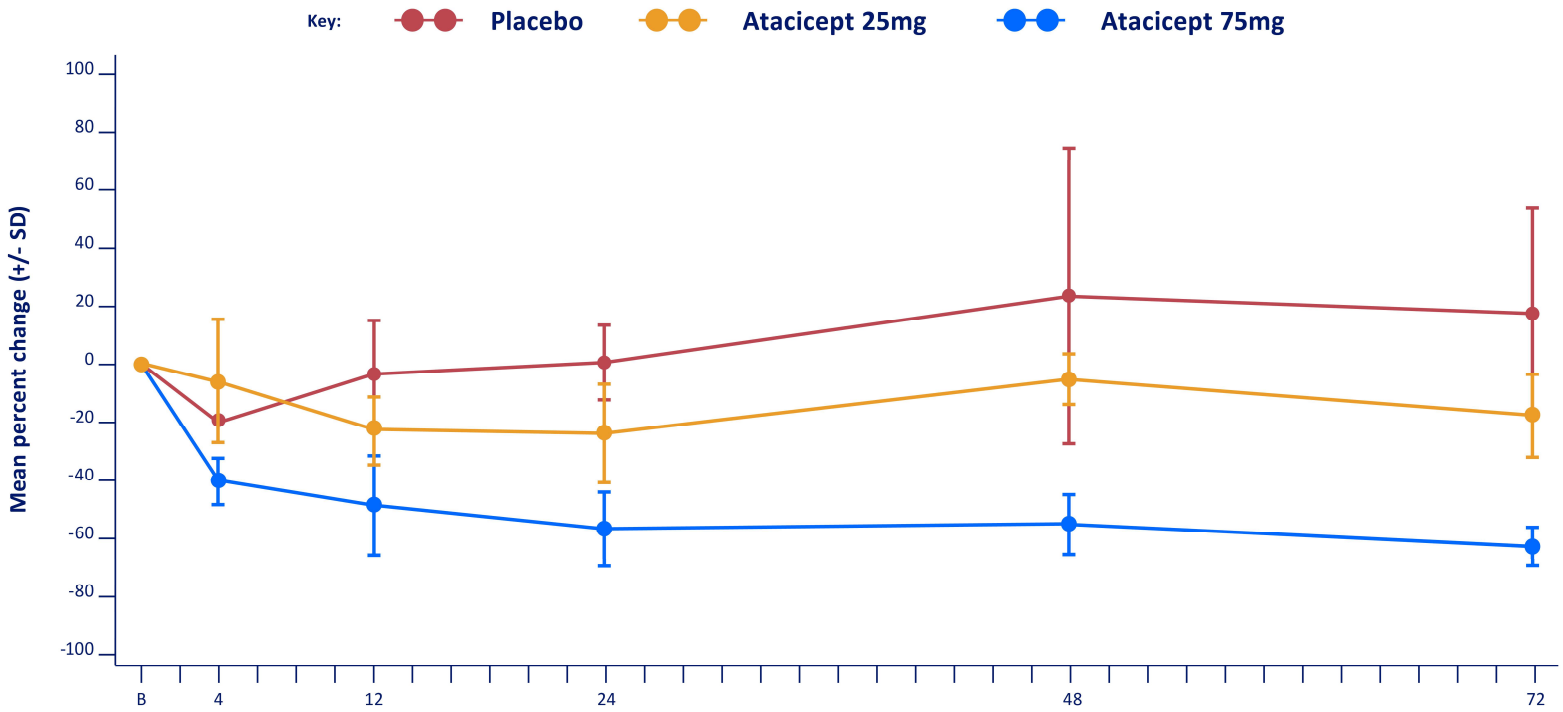
Quartiles determined by JANUS population

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

Barratt et al. American Society of Nephrology 2021.

Clear Dose-dependent Reductions on Serum Gd-IgA1 with Atacicept, and Atacicept 75 mg Reduces Gd-IgA Significantly (60%) and Durably

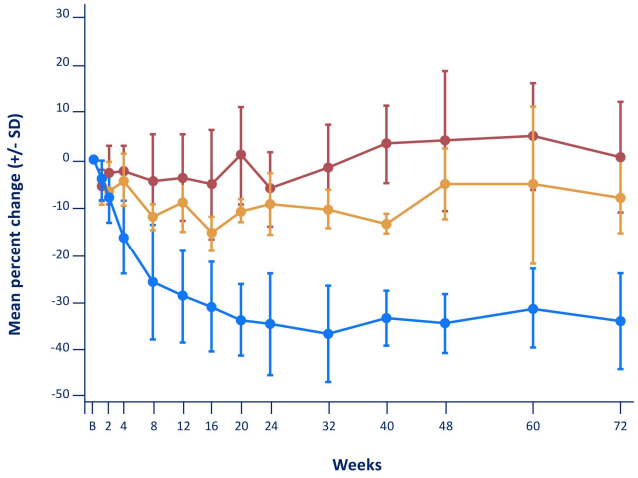


Placebo N=5	5	5	5	5	3
Atacicept 25mg N=6	6	5	5	3	3
Atacicept 75mg N=5	5	5	4	4	3

Barratt et al. KI Reports 2022.

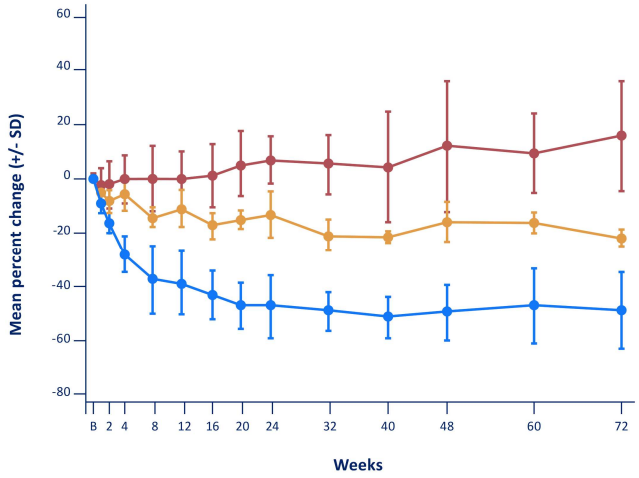
Clear Dose-dependent Reductions on Serum Immunoglobulins from Baseline with Atacicept

IgG



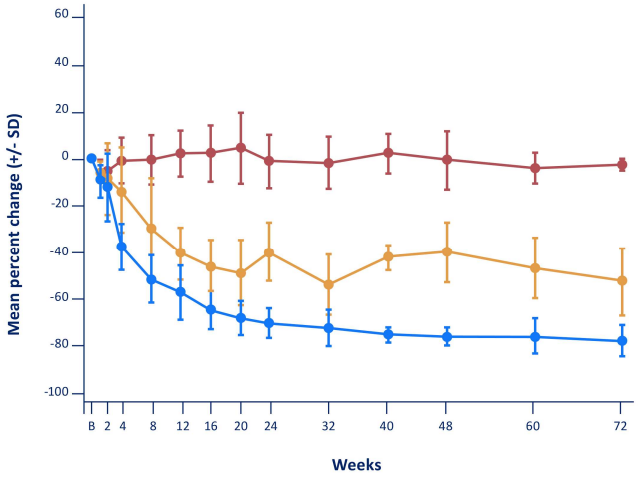
Placebo N=5	5	5	5	5	5	5	5	5	5	4	3
Atacicept 25mg N=6	6	6	5	5	5	5	4	3	3	3	3
Atacicept 75mg N=5	5	5	5	5	5	5	5	4	4	4	3

IgA



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

IgM

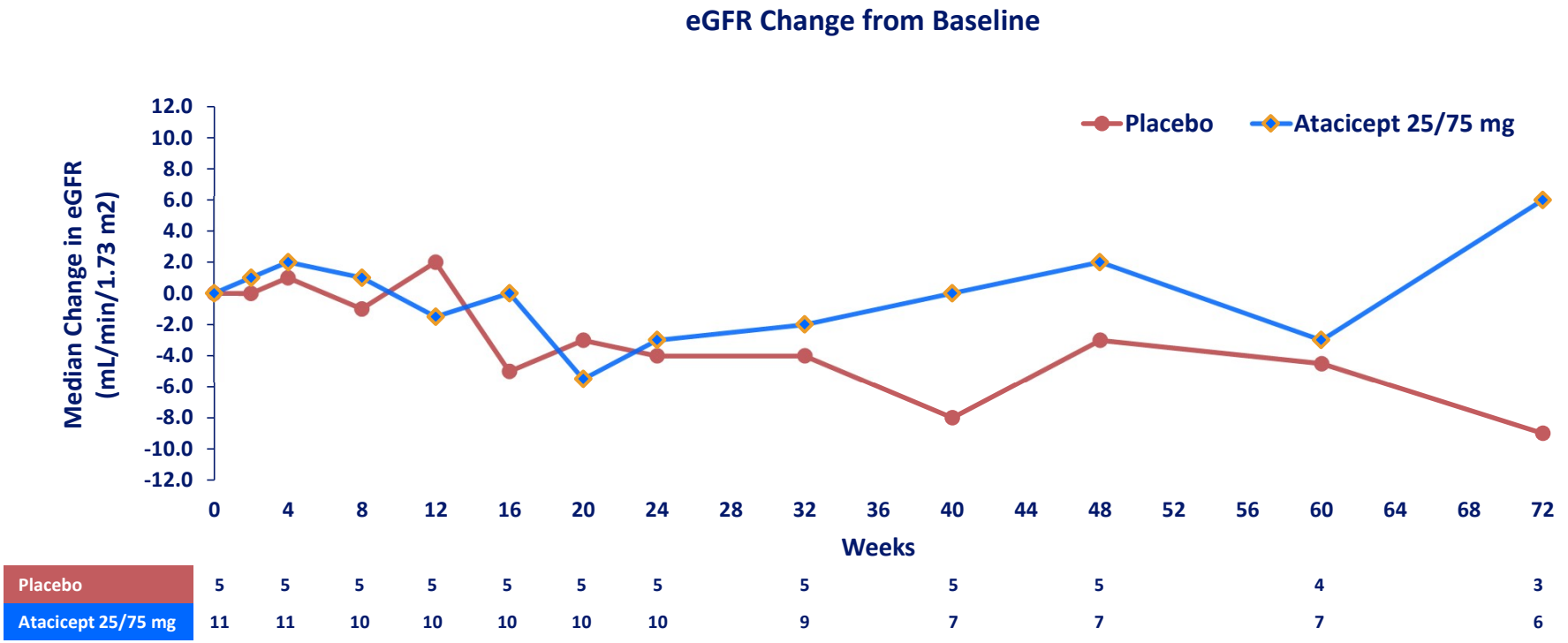


Placebo N=5	5	5	5	5	5	5	5	5	5	4	3
Atacicept 25mg N=6	6	6	5	5	5	5	4	3	3	3	3
Atacicept 75mg N=5	5	5	5	5	5	5	5	4	4	4	3

Key: Placebo Atacicept 25mg Atacicept 75mg

Barratt et al. KI Reports 2022.

Encouraging Trends of eGFR Stabilization from Baseline to Week 72 for IgAN Patients Treated with Atacicept

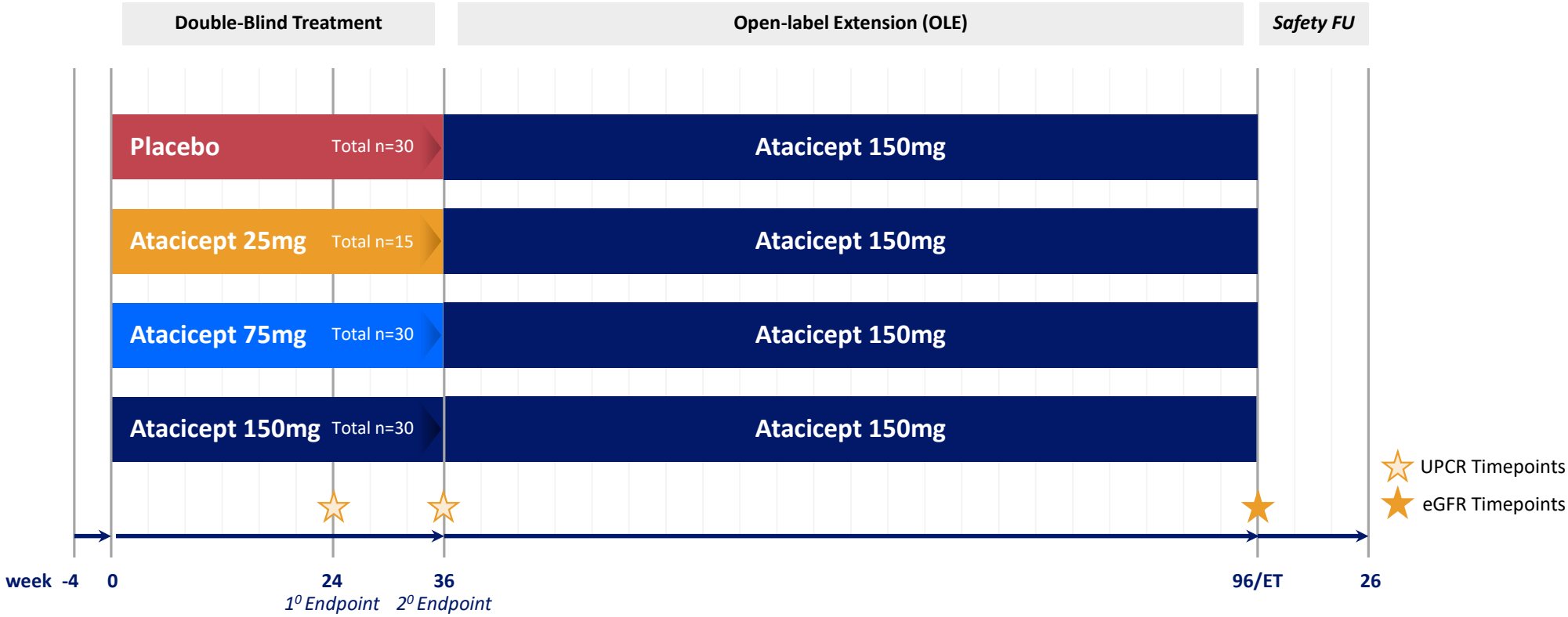


Atacicept Showed Stable eGFR for >1 Year vs 25% decline in Placebo

Barratt et al. KI Reports 2022.

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

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



Well Characterized Safety Profile from Clinical Experience

A total of 1,500+ subjects have received at least 1 dose of atacicept across different indications including two large SLE studies and long-term extension studies (as of Nov 2022)

Exposure-adjusted incidence rates (EAIRs) of serious infection and serious treatment emergent adverse event rates were **similar between atacicept and placebo**

No association between **risk of infection** and magnitude of pharmacodynamic effects with atacicept

Clinical trials require **standard risk mitigation** including up-to-date vaccinations, eligibility review by medical monitor, and education on early detection of signs/symptoms of infection

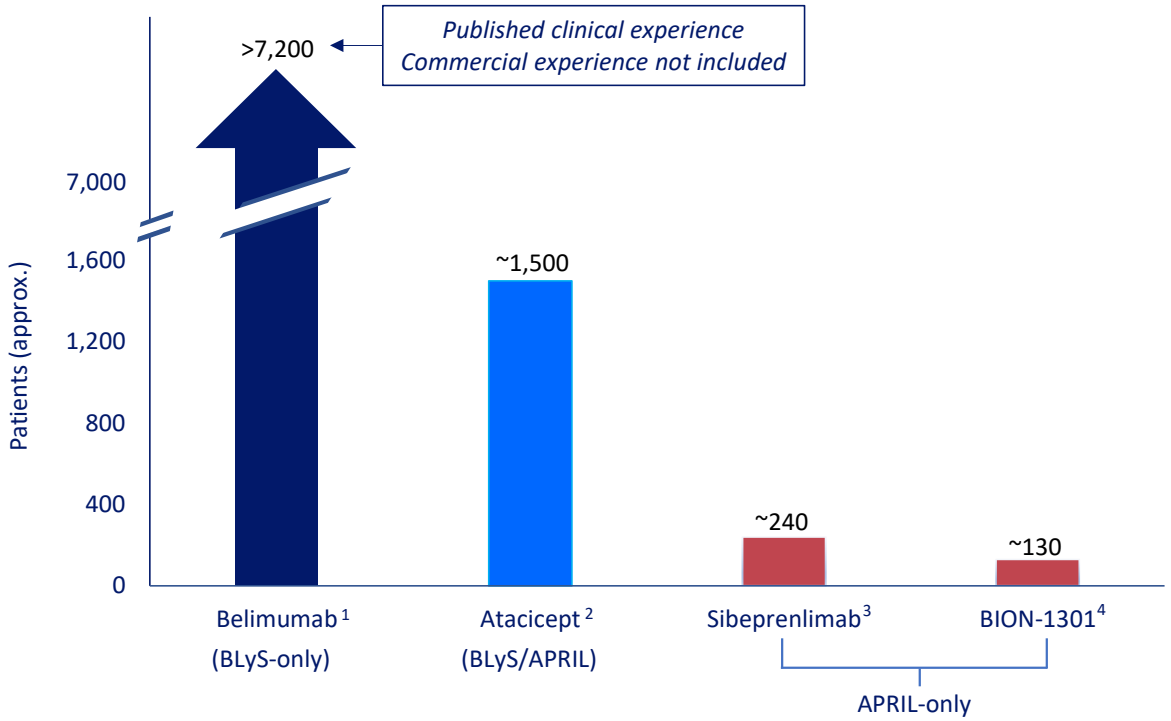
Demonstrated Tolerability Profile In an Integrated Safety Analysis of Over 1,000 Patients on Atacicept

Summary of adverse events (AEs) >5% in any arm, by dose in the double-blind placebo-controlled set

	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%

Adapted from Gordon et al. 2019. Integrated safety profile of atacicept: an analysis of pooled data from the atacicept clinical trial program. Rheumatology Advances in Practice; 0: 1-12.

Dual BlyS/APRIL and BlyS Alone Have Well Characterized Safety Databases Compared to APRIL-Only Approaches



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

[1] Belimumab data based on published results involving safety per Levy et al 2021; excludes other clinical studies and post-marketing / 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)

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

	B-Cell Modulators						
	vera therapeutics	RemeGen	CHINOOK THERAPEUTICS	Otsuka	calliditas THERAPEUTICS	TRAVERE THERAPEUTICS	CHINOOK THERAPEUTICS
Drug	**atacicept**	**telitacicept**	**BION-1301**	**sibeprenlimab**	**Tarpeyo**	**sparsentan**	**atrasentan**
Dose Regimen & Administration	75mg or 150mg Subcutaneous (1 x 1ml injection)	160mg or 240mg Subcutaneous (3 x 1ml injections)	450mg IV or 600mg Subcutaneous (2 x 2ml injections)	2-8mg/kg IV (Ph 2) 400mg SC (Ph 3) (1 x 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.

[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. [6] Molyneux et al ASN Kidney Week 2022. [7] Travere Press Release 8/16/2021. [8] Rastogi et al ASN Kidney Week 2022. [9] Kooienga 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

vera therapeutics

Lupus Nephritis Expansion Could Increase Atacicept's Blockbuster Potential

Lupus Nephritis: Multi-Billion Dollar Market Opportunity

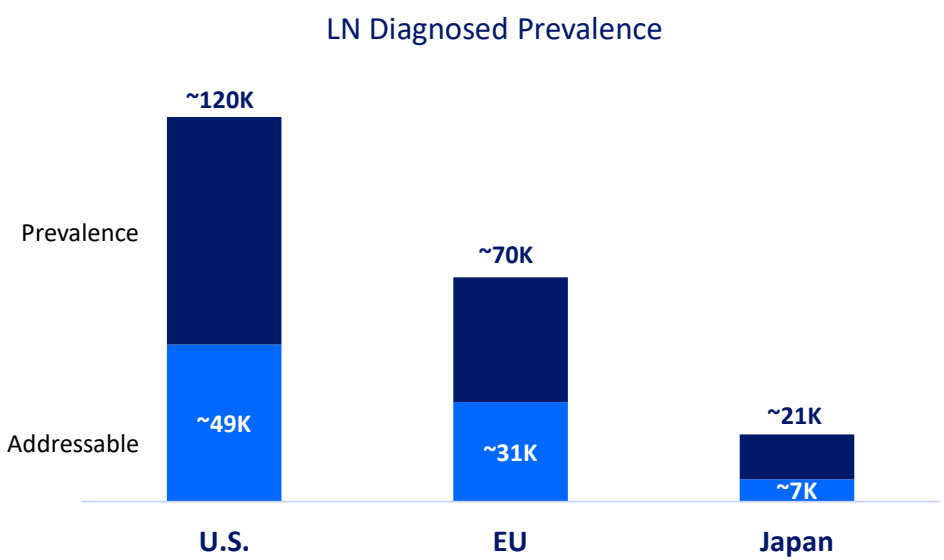
Severe renal manifestation of SLE, high morbidity and mortality, many patients progress to ESRD

Current treatment involves combination immunosuppressants and steroids

Two recently approved therapies, Benlysta (belimumab) and Lupkynis (voclosporin) leave room for improvement in risk/benefit for patients

- Benlysta Renal Response at 104 wks: 30% (active) vs 20% (placebo)[1]
- Lupkynis Renal Response at 52 wks: 41% (active) vs 23% (placebo)[2]

~$3-6B Annual Market Opportunity Globally (US, EU, and Japan) for Novel LN Therapeutics[3]



[1] Furie et al. NEJM 2020. [2] Arriens et al. Annals of the Rhematic Diseases 2021. [3]ClearView Healthcare Partners Analysis. Prevalence and addressable population estimates based on peak year forecast.

Significant Unmet Need Exists for Safe and Specific therapies That Have a Direct Impact on LN Disease Activity Without a High Risk of Infection

Increasing level of Need

Unmet Need	Description	KOL Perspective	Potentially Addressable by Atacicept
Disease Modifying Therapies	Physicians frequently expressed a need for **novel mechanisms of action that address the underlying pathophysiology** of LN specifically	*" I am treating patients in a non-specific manner with the current agents that are available to me. It can be very frustrating at times."* – US Nephrologist	✓
Improved Remission Rates	Although physicians find it **challenging to establish remission** in LN, it is viewed as a lower impact need given improved efficacy observed with newer agents	*" Remission rates are certainly not perfect, but they are better than they were when I first started treating LN."* – US Nephrologist	✓
Decreased Risk of Infection from IST	KOLs cited a **desire for safer, more tolerable agents** given risk of infection associated with use of current immunosuppressant agents	*" When we look at ISTs, we expect to see many infections, so a therapy lacking this side-effect would be incredibly beneficial."* – EU Nephrologist	✓

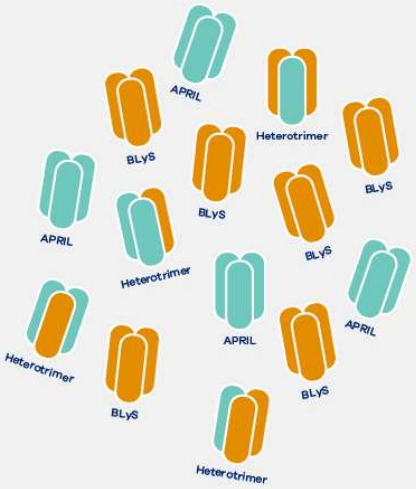
Key: ✓ Fully Addressed ✓ Partially Addressed

IST: Immunosuppressive Therapy; KOL: Key Opinion Leader. Source: Expert Interviews; ClearView Analysis.

Atacicept Blocks Elevated B Cell Cytokines Driving the Underlying Disease in Lupus Nephritis

Cytokine Abnormalities

1 Patients with Systemic Lupus Erythematosus may have higher levels of BLyS and APRIL, contributing to the pathogenesis of Lupus Nephritis



B-Cell Dysregulation

2 BLyS and APRIL promote the survival, maturation and differentiation of B-cells and plasma cells - that then produce disease-causing antinuclear and anti-DNA autoantibodies

2a B cell
Immature B-cells survive and mature

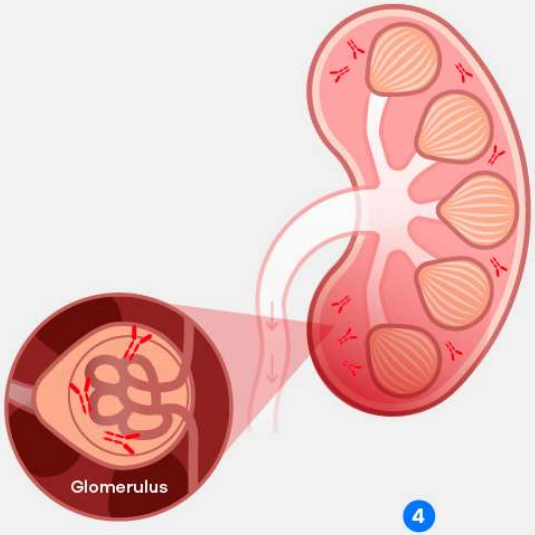
2b B cell
B-cells differentiate and class switch

2c
Plasma cells proliferate and create autoantibodies

2d
Autoantibodies circulate and form immune complexes

Glomerulonephritis

3 Autoantibodies and immune complexes deposit in glomeruli

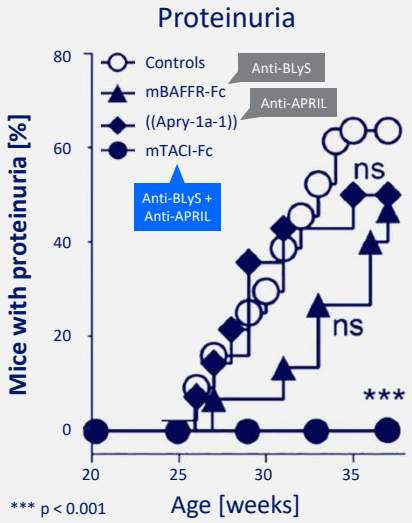


4 Progressive renal injury

Steps that Atacicept targets

Ataicept Has Potential to Outperform Approved BLyS-Only Drug

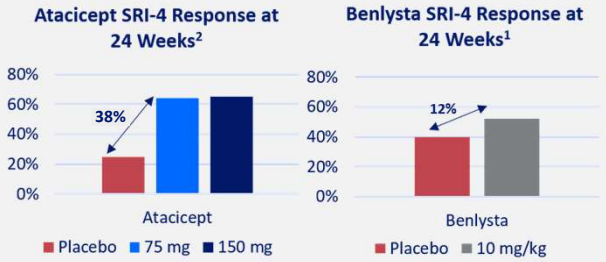
Pre-Clinical Evidence: BLyS-APRIL >> BLyS or APRIL alone



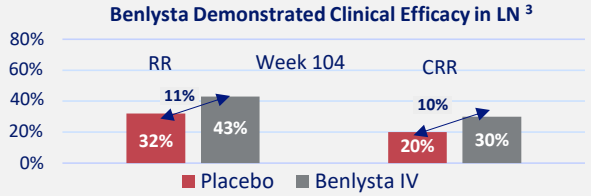
In mouse model of lupus nephritis, only atacicept was able to effectively prevent proteinuria compared to BLyS or APRIL alone

Haselmayer et al. Eur J. Immunol. 2017, Figure 2, page 1080.

Clinical Evidence: BLyS-APRIL >> BLyS or APRIL alone



*In similar serologically active SLE patients, BLyS-APRIL inhibition may provide better efficacy vs. BLyS alone**

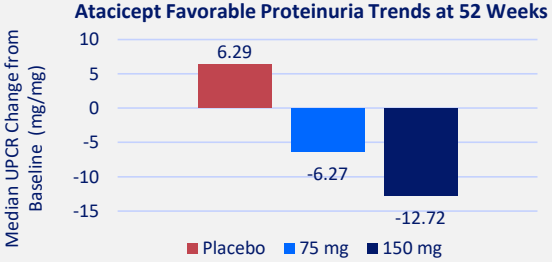


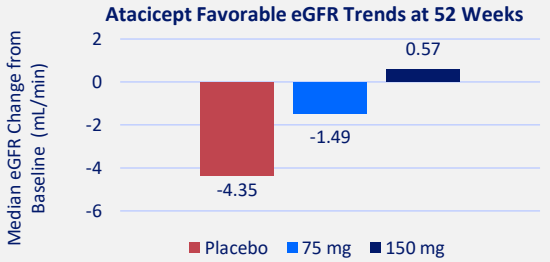
BENLYSTA approved in LN, but response rate (RR) still <50%; we believe there is room for improvement with dual blockade[3]

[1] van Vollenhoven et al. 2011. [2] Merrill et al. 2018. [3] Furie R et al., 2020 NEJM.

***This 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**

Clinical Evidence: Improved renal function in SLE patients

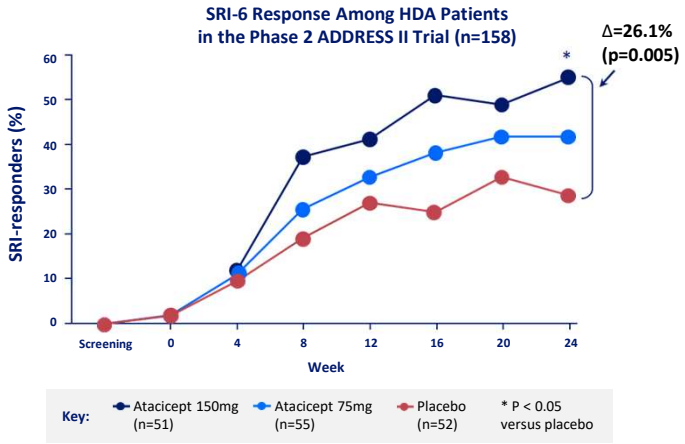


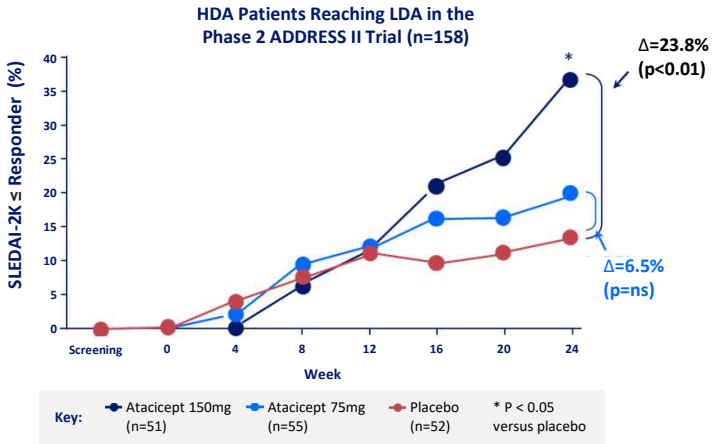


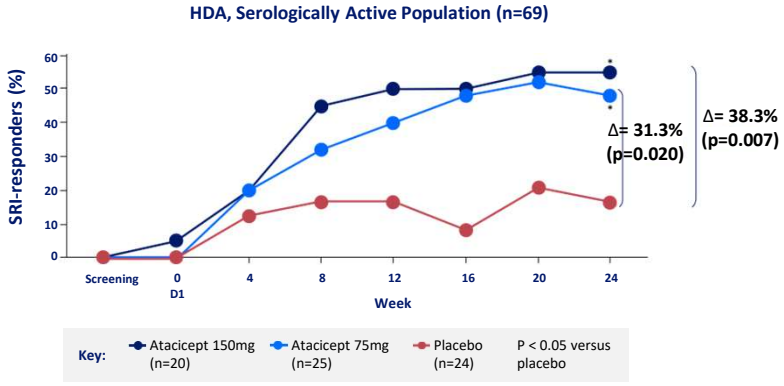
Phase 2 APRIL-SLE trial showed improved eGFR and proteinuria trends at 52 weeks in moderate to severe SLE patients

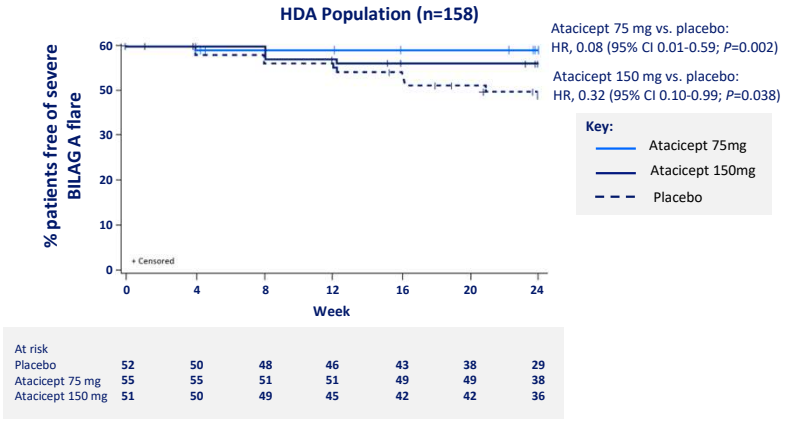
Isenberg et al. ERA-EDTA 2022 Presentation

Atacicept Phase 2 Trial Shows Evidence of Clinical Efficacy In SLE







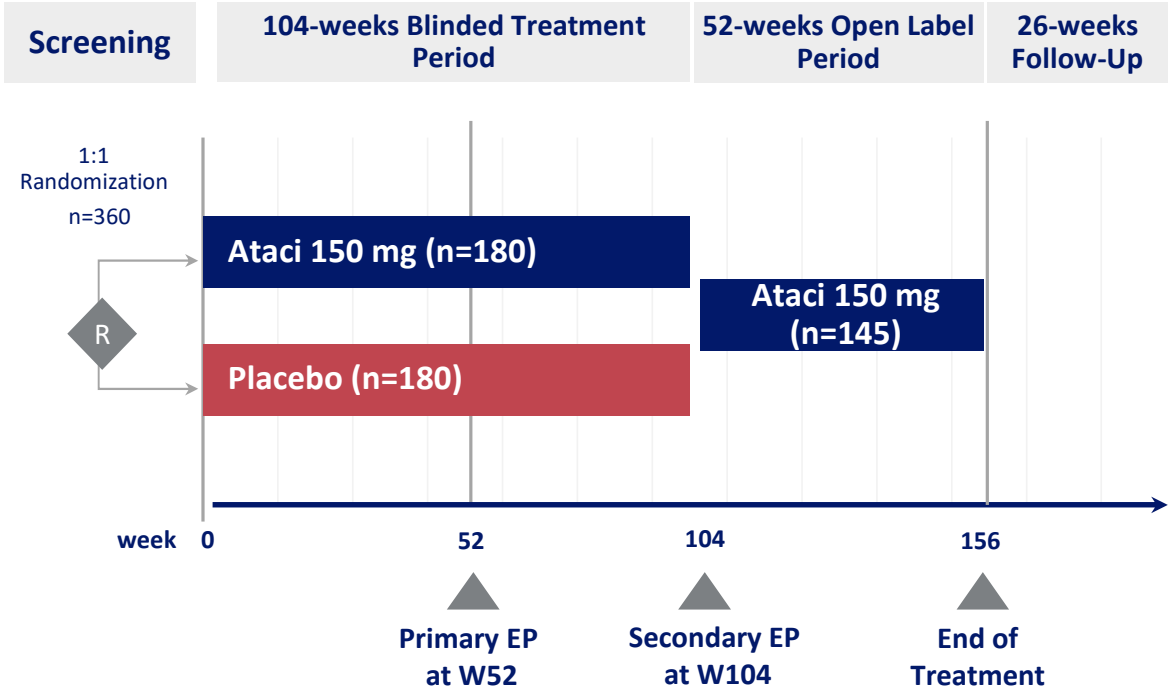


At risk							
Placebo	52	50	48	46	43	38	29
Atacicept 75 mg	55	55	51	51	49	49	38
Atacicept 150 mg	51	50	49	45	42	42	36

[1]SRI-6 response is defined as ≥**6-point reduction in the SELENA-SLEDAI score**, and no new British Isles Lupus Assessment Group (BILAG) A organ domain score or 2 new BILAG B organ domain scores, and no worsening (<0.30-point increase) in Physician's Global Assessment (PGA) score. HDA: High Disease Activity (SLEDAI≥10). LDA: Low Disease Activity (SLEDAI≤2). Merrill et al (2018).; Morand et al (2020).

Phase 3 Trial of Atacicept for Lupus Nephritis (COMPASS): Study Design

A multi-national, randomized, placebo-controlled pivotal trial



Study Endpoints

Primary

- Complete renal response at Week 52

Secondary

- Alternate definition of renal response at Week 52
- Complete renal response at Week 104
- Time to death or renal-related event
- Time to UPCR of ≤0.5 mg/mg

Phase 3 Trial of Atacicept for Lupus Nephritis (COMPASS): Study Population

Study Population

- Patients with active lupus nephritis, adding atacicept to standard-of-care
- Initial or maintenance therapy with MMF
- All patients will receive initial high-dose corticosteroid (CS) treatment

Key Differences vs Prior LN study (APRIL-LN)

- Atacicept 150 mg SC QW, vs loading-dose (atacicept 150mg twice weekly x 4 weeks)
- MMF target dose of 2g/day, vs higher dose 3g/day
- Lower dose of corticosteroid regimen

Key Inclusion Criteria

- Male/female subjects ≥18 years of age
- Biopsy-proven active LN with 24-hour UPCR >=1.0 mg/mg

Key Exclusion Criteria

- eGFR <=30 mL/min/1.73 m^2
- Sclerosis in 50% of glomeruli on renal biopsy
- Evidence of rapidly progressive glomerulonephritis
- Concomitant significant renal disease
- Serum IgG < 7 g/L
- Active infection or high infectious risk

We Believe Atacicept Has Best-in-Disease Potential In Lupus Nephritis

Drug	atacicept	Benlysta	Ianalumab	Gazyva	Lupkynis	Cosentyx	Saphnelo
Administration	Subcutaneous	Intravenous or Subcutaneous	Subcutaneous	Intravenous	Oral	Subcutaneous	Intravenous
Mechanism	Dual BLyS/APRIL inhibition	Anti-BLyS	Anti-BLyS-R	Anti-CD20	Calcineurin inhibitor	Anti-IL-17A	Anti-IFNAR1
Current Stage of Development	Phase 3	Marketed	Phase 3	Phase 3	Marketed	Phase 3	Phase 3
% of Patients Achieving CRR vs Control	To come	10% delta (week 104)[2]	N/A[5]	12% delta (week 52)[4]	18% delta (week 52)[3]	N/A[5]	14.4% delta (week 52)[6,7]
Safety	Integrated safety analysis >1,400 patients	7.2% discontinued due to AEs[2]	N/A[5]	Serious AEs and serious infections not increased[4]	14% discontinued (at 23.7 mg BID) due to eGFR decrease[3]	N/A[5]	11.8% discontinued due to AEs[7]

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.

[2]Furie et al. NEJM 2020. [3]Rovin et al. The Lancet 2021. [4]Rovin et al. Presented at ASN Kidney Week, November 2019, Abs FR-OR136. [5]"N/A" indicates that these drugs were not evaluated in LN through a clinical trial. [6]900 mg for first 3 doses, then 300 mg thereafter. [7]Jayne et al. Ann Rhem Dis. 2021.

New Clinical-Stage Asset: MAU868, A Novel Monoclonal Antibody Against BK Virus

BK Virus Infection: Potential for a Blockbuster Market Opportunity

BK Virus (BKV) leads to significant morbidity and mortality in transplant patients

80-90% of healthy adults have been infected with BKV and the virus remains latent in healthy adults

BKV can be reactivated when a patient is immunocompromised

BKV impacts two immunocompromised populations including **kidney transplant patients** and **hematopoietic stem cell transplant (HSCT) recipients**

No approved anti-BKV treatments in the United States

Unserved Market ~$1B+ Commercial Opportunity WW in 2036[1]

Kidney Transplants: ~80,000 RTx per year WW

Viruria (30-50%)	**40,000 pts –** measurable BKV
Viremia (10-20%)	**15,000 pts** – kidney at risk
Nephropathy (3-4%)	**3,200 pts** – irreversible damage
Rejection (1-2%)	**1,500 pts** – *kidney loss*

HSCT Procedures: ~100,000 HSCT per year WW

Allogeneic (50%)	**50,000 pts –** higher risk of BKV
Viremia (10-35%)	**22,500 pts** – risk of cystitis
Cystitis (6-16%)	**10,500 pts** – hemorrhagic cystitis

- BKV Nephropathy is the **leading cause of allograft loss**
- BKV in HSCT patients have **increased risk of severe hemorrhagic cystitis**

[1] Back Bay Analysis.

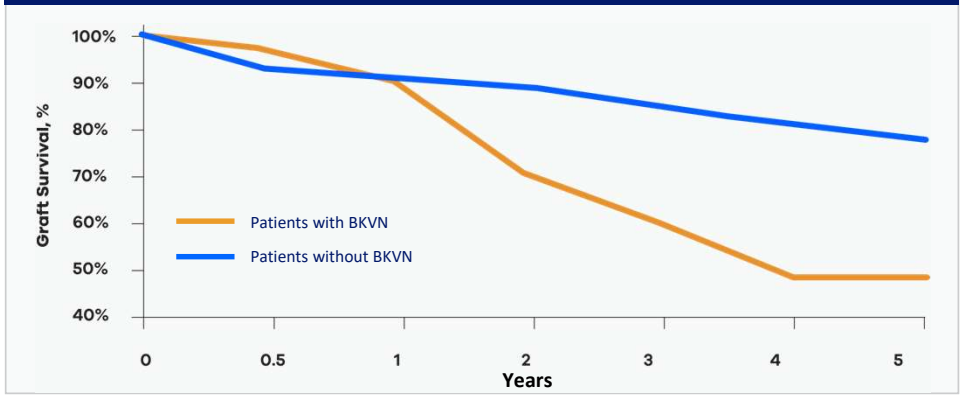
Kidney Transplants: BKV Nephropathy is a Leading Cause of Allograft Loss

There is a need in renal transplants for a BKV treatment option that could address escalating BKV infections early without risking immune system allograft rejection.

Kidney Transplants: ~80,000 RTx per year WW

Viruria (30-50%)	**40,000 pts – measurable BKV**
Viremia (10-20%)	**15,000 pts** – kidney at risk
Nephropathy (3-4%)	**3,200 pts** – irreversible damage
Rejection (1-2%)	**1,500 pts** – *kidney loss*

Graft Survival in Kidney Transplant Patients is Worse with BKV Nephropathy



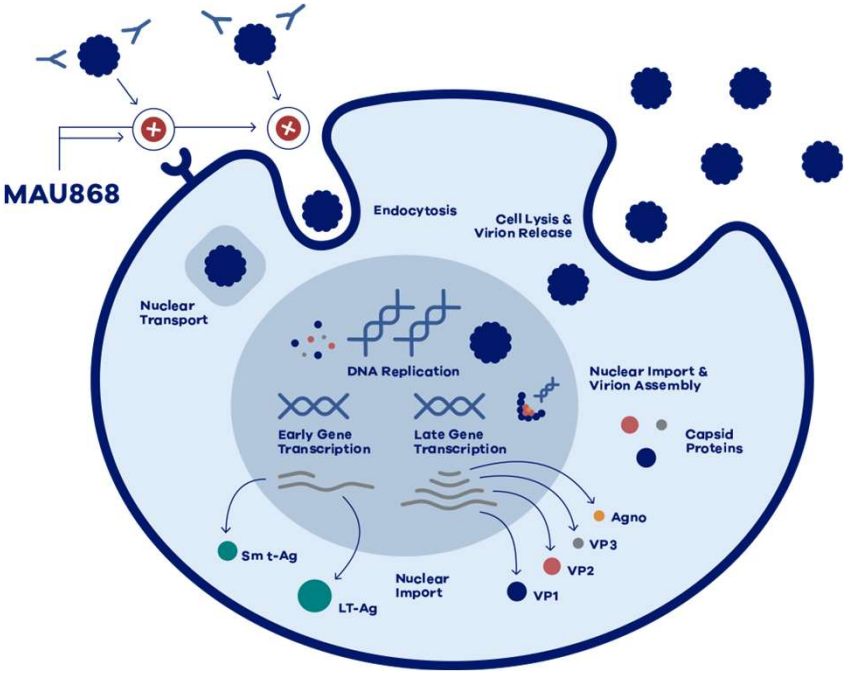
Vasudev, B et al. Kidney International 2005

- Poor Transplant Outcomes with BKV Reactivation
  - BKV viremia is associated with reduction in renal function and allograft survival
  - BKV nephropathy is associated with allograft loss
- Current Treatment for BKV in Renal Transplant: Reduce Immunosuppression Measures
  - In response to BKV reactivation, physician will lower immunosuppression, with risk of allograft rejection

MAU868: Potential First-in-Class Neutralizing Antibody Targeting BK Virus (BKV)

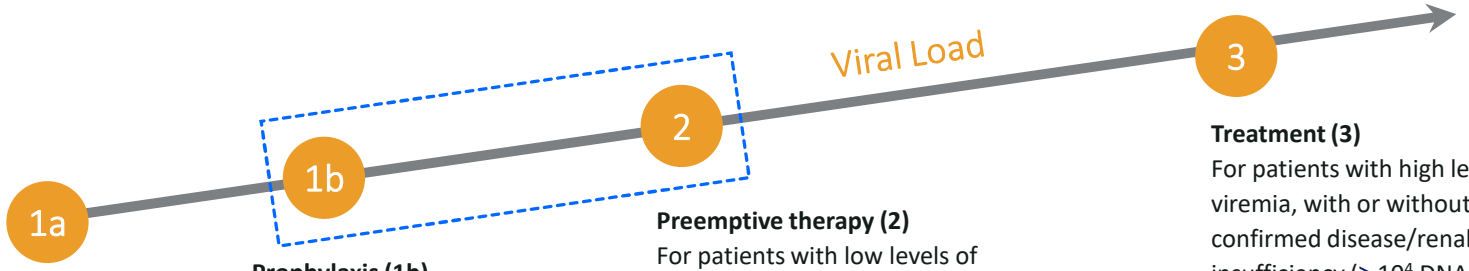
Blocks BKV Virion Binding
Designed to disrupt cell surface binding and to prevent cell entry and spread of infection

- **Novel Target:** mAb that neutralizes infection by blocking BKV virion binding to host cells
- **Active Against All Genotypes:** Sub-nanomolar potency against all major genotypes
- **Proven Mechanism:** Neutralization of virus infection effective in other approved mAb therapies
- **More Potent than IVIG:** ~10,000 fold more potent *in vitro*



BKV Treatment Paradigms

Goal of therapy: prevent clinically significant viremia/disease



Prophylaxis (1a):
For all patients

Prophylaxis (1b)
For high-risk patients (e.g., viruria ≥ 10^7 DNA copies/mL; cord blood transplant; haploidentical, others)

Preemptive therapy (2)
For patients with low levels of viremia (<10^4 DNA copies/mL)

Treatment (3)
For patients with high levels of viremia, with or without biopsy confirmed disease/renal insufficiency (≥ 10^4 DNA copies/mL)

Optimal Positioning for MAU868 value proposition

Intervention	Definition
Prophylaxis (a)	Administration of MAU868 to all patients before any evidence of BKV replication in plasma
Prophylaxis (b)	Administration of MAU868 to high-risk patients before any evidence of BKV replication in plasma
Preemptive	Administration of MAU868 at an early stage of BKV infection or replication (VL <10^4) in plasma
Treatment	Administration of MAU868 given at/after disease diagnosis (i.e. at VL ≥ 10^4 in plasma)

DNA copies/mL ≥ 10^4 has been correlated in two studies with BKV nephropathy (Hirsch et al; Limaye et al)
Clinically significant viremia is BKV mL ≥ 10^4 DNA copies/mL

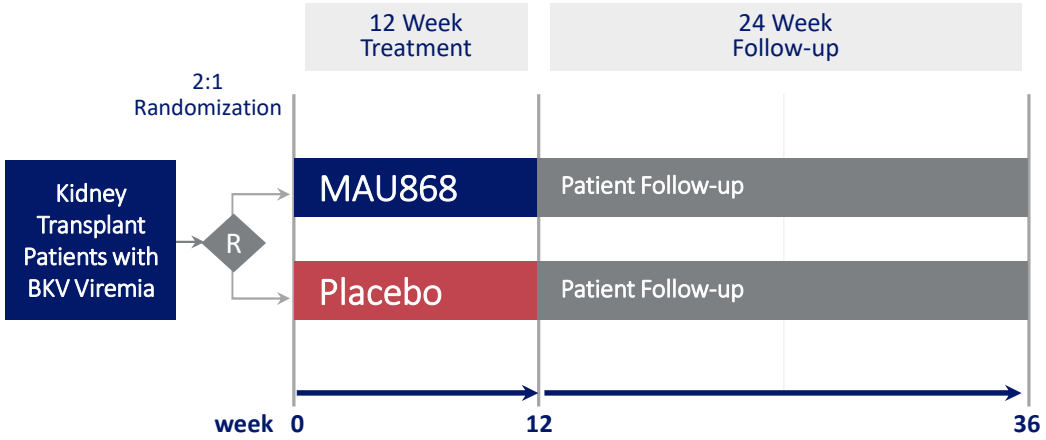
Phase 2 Trial of MAU868 in Kidney Transplant Patients with Active BKV

MAU868-201 Trial Design

Study Population

- Kidney transplant within one year of enrollment in the trial
- Documented BKV viremia within 10 days prior to enrollment in the trial
- Viral load ≥ 10^4 log10 copies/ml, but no more than ≤ 10^7 log10 copies/ml, or consecutive positive VLs if most recent is ≥ 10^3 log10 copies/ml

Randomized, Double-blind, Placebo-controlled Phase 2 Study



Dose Cohorts (n=12 each) 4 IV doses over 12-wks

Cohort 1 1350 mg x 4
Cohort 2 6750 mg x 1 followed by 1350 mg x3

Study Endpoints

Primary

- Safety, tolerability

Secondary

- BKV-related outcomes including viremia, nephropathy, graft function and rejection, PK

Clinicaltrials.gov Identifier: NCT04294472

Antiviral Effect and Renal Effect of MAU868 vs Placebo at Week 12

	MAU868 (n=20)	Placebo (N=8)	P-value
Log reduction in BK viremia- median (interquartile range [IQR]) DNA copies/ml	-1.14 (-1.88,-0.50)	0.37 (-0.72,0.04)	0.051
Proportion of patients with a reduction of BK plasma viral load- n (%)			
by ≥ 1 log	11 (55%)	1 (13%)	0.040
to < lower limit of quantification (LLOQ)	4 (20%)	0	0.172
to < 10^4 DNA copies/ml	15 (75%)	3 (38%)	0.061
Change in estimated glomerular filtration rate [eGFR (CK-EPI)]- median (IQR) mL/min/1.73m^2	2.0 (-5.0,4.0)	-6.0 (-8.5,-0.5)	0.217

Source: Jordan S et al. A Randomized Phase 2 Study of Mau868 Vs Placebo To Treat Bk Viremia In Kidney Transplant Recipients [abstract]. *Am J Transplant*. 2022; 22 (suppl 3).

Summary

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

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