



# Company Overview

April 2025

NASDAQ: JBIO

# Disclaimers

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Certain information contained in this presentation and statements made orally during this presentation relate to or are based on studies, publications and other data obtained from third-party sources as well as our own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, we have not independently verified, and make no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third party sources. Forecasts and other forward-looking information obtained from these sources are subject to the same qualifications and uncertainties as the other forward-looking statements in this presentation. Statements as to our market and competitive position data are based on market data currently available to us, as well as management's internal analyses and assumptions regarding the Company, which involve certain assumptions and estimates. These internal analyses have not been verified by any independent sources and there can be no assurance that the assumptions or estimates are accurate. While we are not aware of any misstatements regarding our industry data presented herein, our estimates involve risks and uncertainties and are subject to change based on various factors. As a result, we cannot guarantee the accuracy or completeness of such information contained in this presentation.

# Jade Biosciences is advancing potentially best-in-class therapies for autoimmune diseases

Current funding through 2027, well-beyond biomarker-rich JADE-001 healthy volunteer data

MOA	Program	Discovery	IND-enabling	Planned Clinical FIH	Planned Interim FIH Data	Potential Indications
anti-APRIL	JADE-001			2H25	1H26	IgAN
Undisclosed	JADE-002			1H26		Multiple systemic AI diseases
Undisclosed	JADE-003			1H27		Undisclosed

*Development candidates licensed from Paragon*

Assets designed to maximize clinical responses

Patient friendly, infrequent dosing



Notes: Jade has entered into an exclusive JADE-001 license agreement with Paragon Therapeutics. Jade holds an exclusive option to license JADE-002 and JADE-003 from Paragon. Jade has not yet entered into a license agreement with respect to JADE-002 or JADE-003.  
MOA – mechanism of action; FIH – First-In-Human; IgAN - IgA nephropathy; AI - autoimmune

# JADE-001: a potentially best-in-class anti-APRIL mAb for IgAN

# Jade is developing a potentially best-in-class anti-APRIL mAb



Estimated  
**\$10B+**  
branded  
market

Current treatments do not adequately address the need for **long-term disease-modifying therapy** in a typically young IgAN patient population



**Anti-APRIL**  
class poised to be  
frontline treatment  
for IgAN

Mechanism has potential to be disease modifying, **reducing pathogenic IgA** and proteinuria, **stabilizing kidney function**



Potentially  
**best-in-**  
**class**  
profile

JADE-001 is designed to have superior potency and an extended half-life for **maximal efficacy & convenient dosing**



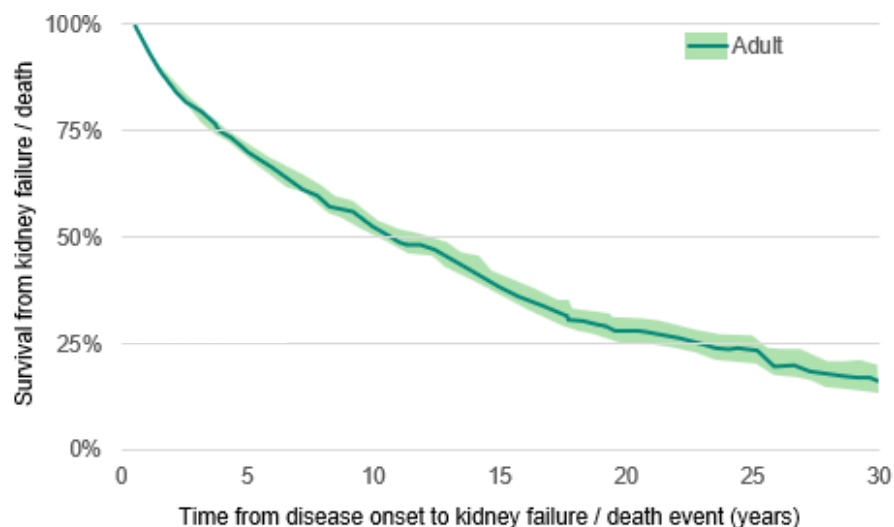
**Efficient**  
path to PoC  
and market

Biomarker-rich and highly translational HV data expected in 1H26; **surrogate endpoints expected to support potential IgAN approval**

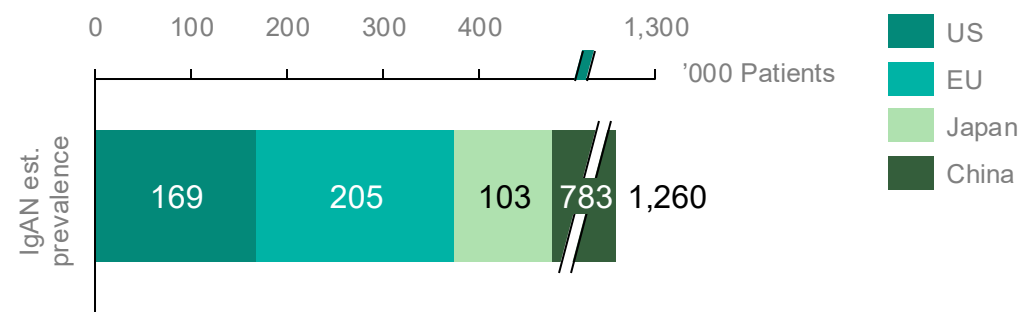
# ~169K+ IgAN patients in the U.S. with majority requiring treatment\*, representing potential \$10B+ market

IgAN patients with persistent proteinuria are **at risk of kidney failure**

IgAN is an **autoimmune kidney disease**, typically diagnosed in 20- to 30-year-olds, **requiring life-long therapy**.



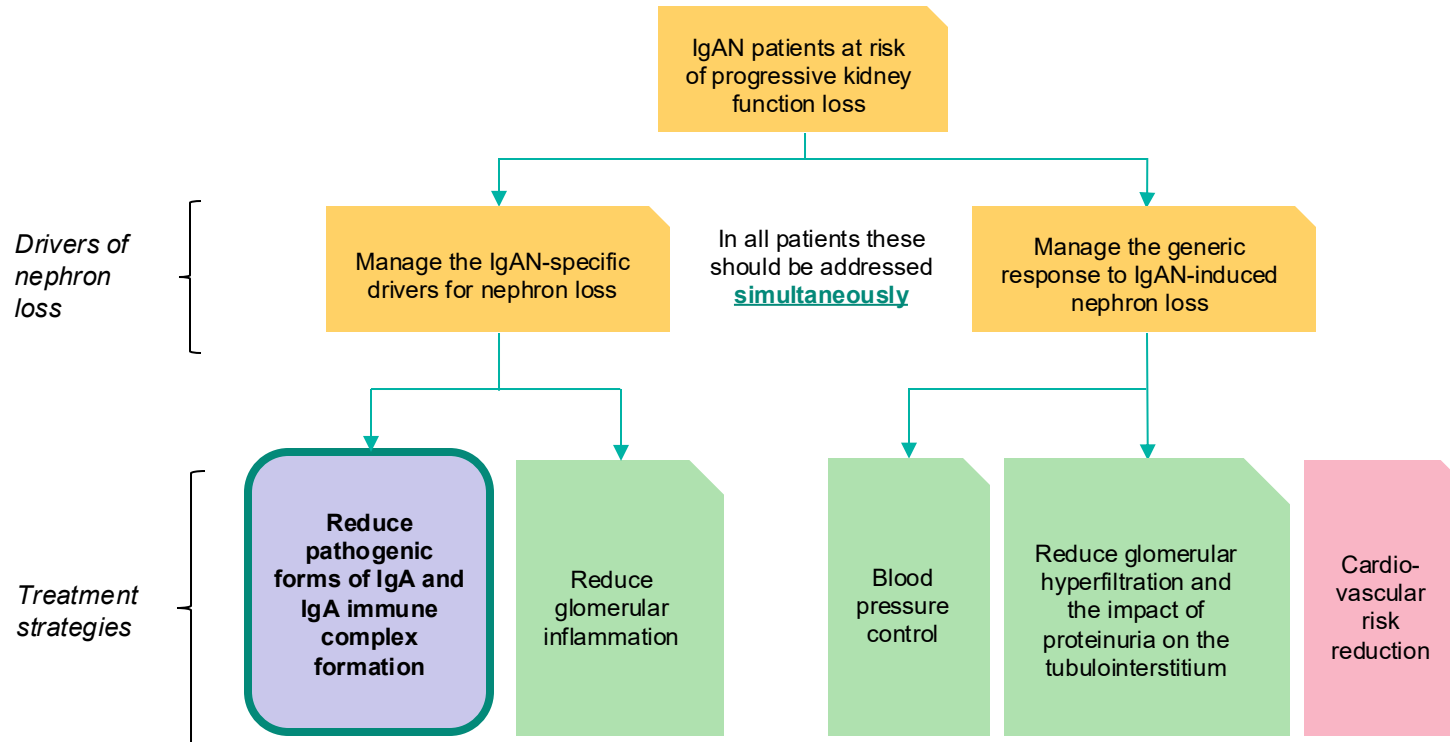
~1M+ global patients, **significant potential ex-U.S. market potential**



~169K+ patients with IgAN in the U.S., with **60-75%** requiring treatment per international guidelines

There is a high unmet need for **disease-modifying treatments that are safe, well-tolerated, and convenient** particularly considering that IgAN is often **diagnosed in young adults** and requires **lifelong care**

# Proposed updates to KDIGO guidelines support the frontline therapeutic potential of the anti-APRIL class in IgAN



KDIGO updates anticipated to increase **IgAN diagnosis**, expand **at-risk patient population** requiring treatment, **lower proteinuria target** to clinical remission, and require **targeted therapies** that **reduce pathogenic IgA**.

## Expanding Patient Population

- Kidney biopsy recommended in all adults with proteinuria  $\geq 0.5$  g/d where IgAN is a possible diagnosis
- Recommends **additional treatment should be initiated in all cases** where patients have proteinuria  $\geq 0.5$  g/d

## Lower Proteinuria Targets

- Establishes new treatment goal: proteinuria maintained at  $< 0.5$  g/day, **preferably  $< 0.3$  g/day**

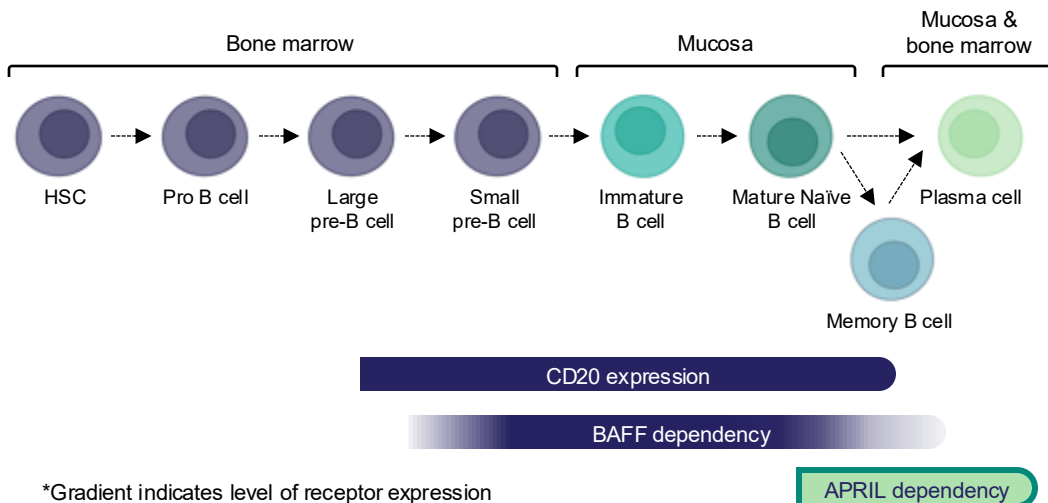
## Redefining Treatment Strategies

- New guidelines direct the use of treatments that have been **proven to reduce pathogenic forms of IgA**

# Reducing pathogenic IgA production by plasma cells is a potentially disease-modifying approach for IgAN

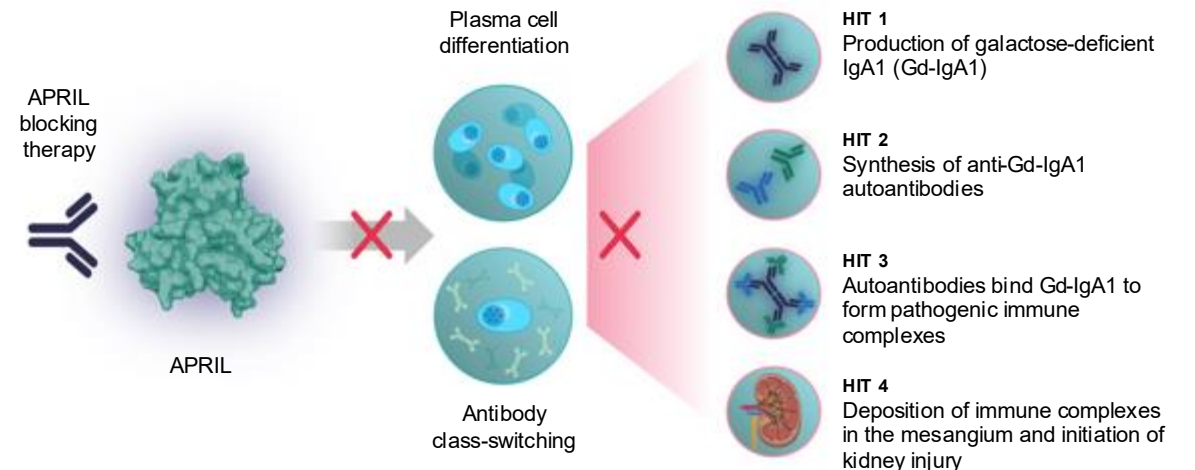
## Broad B-cell depletion is ineffective in IgAN...

- B-cell depletion with rituximab (anti-CD20) **failed to reduce Gd-IgA1, anti-Gd-IgA1 autoantibody, or proteinuria** and **did not impact eGFR**.
- BAFF neutralization (blisibimod) **did not reduce IgA or proteinuria**.



## ...while targeted plasma cell modulation is highly effective.

- APRIL and dual APRIL/BAFF neutralization **result in significant and sustained depletion of Gd-IgA1, reduction in proteinuria, and eGFR stabilization**.



Neutralizing APRIL depletes Gd-IgA1, reduces proteinuria, and preserves eGFR, providing a disease-modifying treatment of IgAN without impacting B-cell development and maturation.

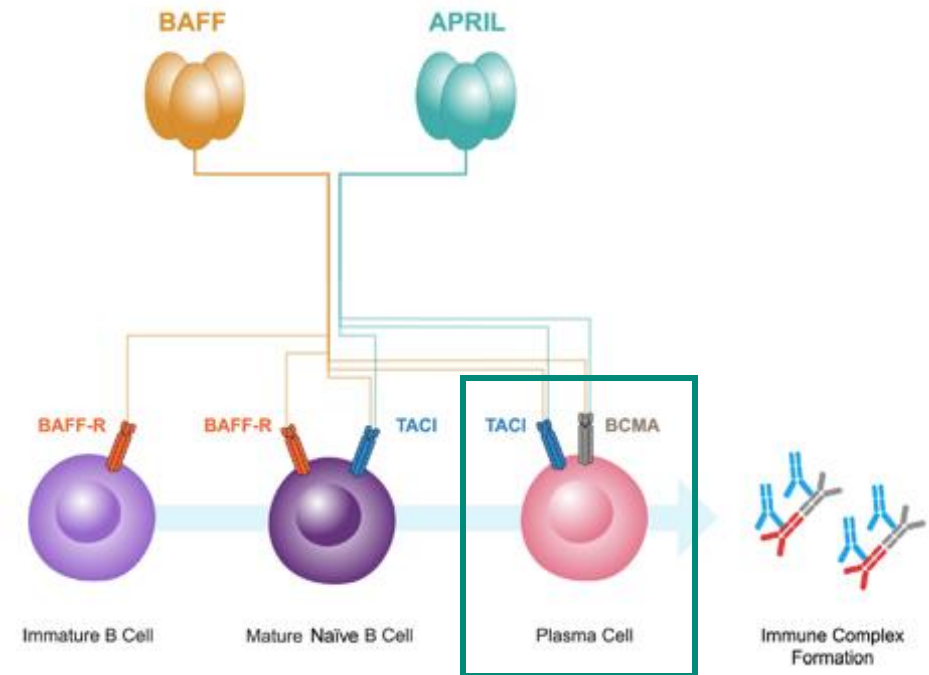


# Selectively targeting APRIL potentially provides disease modification without added immunosuppression of BAFF inhibition

APRIL is the plasma cell survival factor **critically linked to IgAN pathogenesis and disease activity**

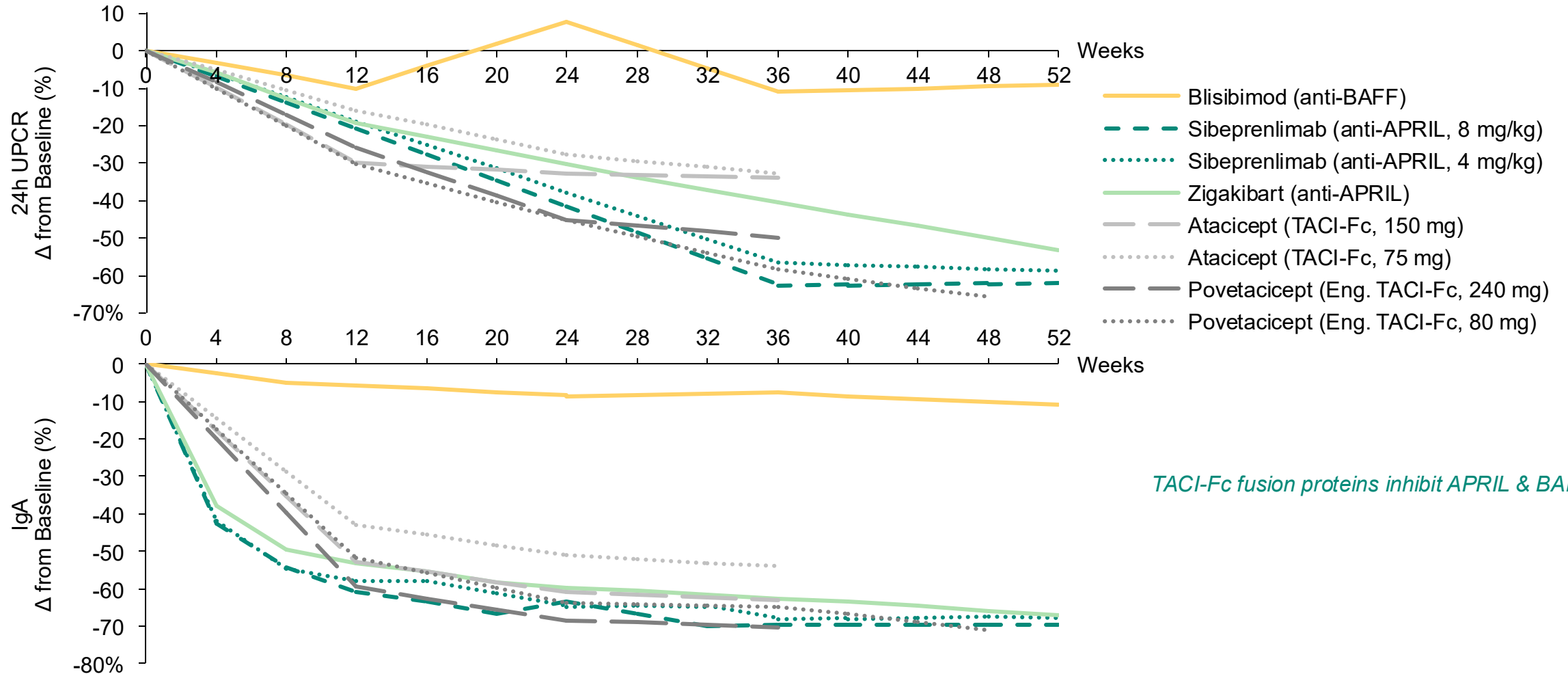
Targeting APRIL **selectively modulates plasma cells**, maintaining pool of mature B cells

	APRIL	BAFF
<b>Risk variant in IgAN GWAS</b>	✓	✗
<b>Elevated in IgAN patients and associated with disease severity</b>	✓	✓/✗
Promotes <b>excess secretion of Gd-IgA1</b> in IgAN patient lymphocytes <i>ex vivo</i>	✓	No data
Drives <b>IgA class switching</b> via TACI <i>in vivo</i>	✓	✗
<b>Overexpression in mouse model</b> leads to glomerular IgA deposition	✓	✓
KO mouse model <b>decreases IgA levels / IgA+ plasma cells</b> in small intestine	✓	✗
<b>Selective inhibition demonstrates preclinical / clinical efficacy</b> in IgAN	✓	✗



Existing genomic, mechanistic, IgAN model, and clinical data **support the importance of APRIL over BAFF** in IgAN, and APRIL-only blockade **avoids the potential for unnecessary immunosuppression**

# Reductions in proteinuria and IgA in IgAN clinical studies indicate APRIL inhibition is the driving force behind TACI-Fc efficacy



*TACI-Fc fusion proteins inhibit APRIL & BAFF*

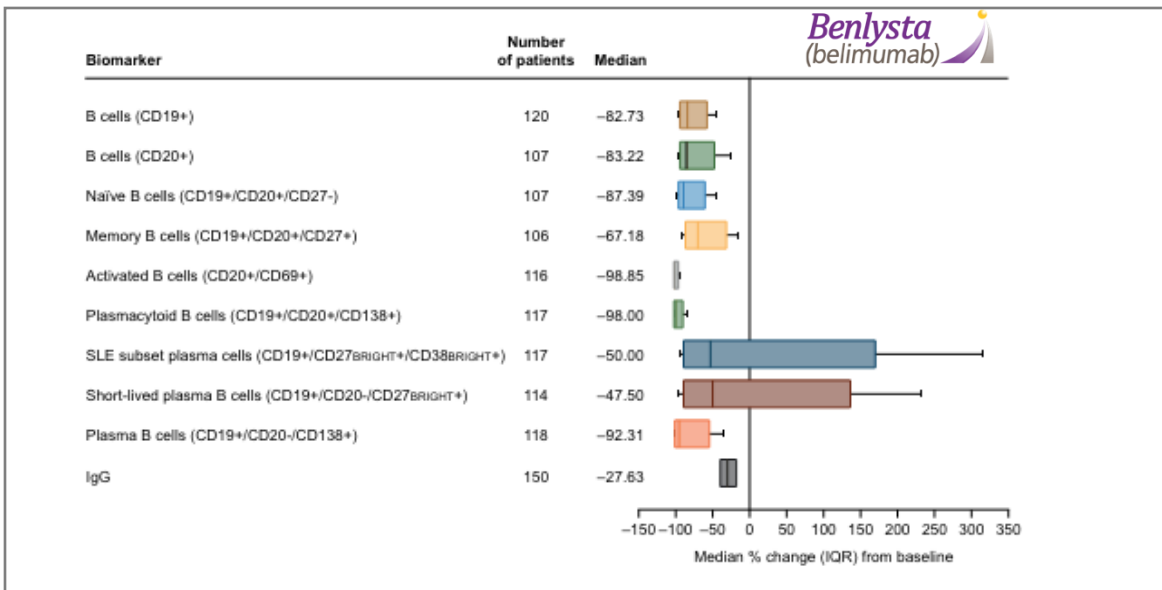
# No clinical evidence that inhibiting BAFF provides additional efficacy beyond APRIL alone in IgAN

	Sibeprenlimab	Zigakibart	Atacicept	Povetacicept
<b>MoA</b>	anti-APRIL	anti-APRIL	TACI-Fc	Engineered TACI-Fc
<b>Status</b>	P3	P3	P3	P3
<b>Δ from baseline in critical disease markers (W36 timepoint*)</b>	IgA	IgA	IgA	IgA
	Gd-IgA1	Gd-IgA1	Gd-IgA1	Gd-IgA1
	UPCR	UPCR	UPCR	UPCR
	67%	64%	63%	65%
	60%	69%	64%	69%
	60%	53%	33%	59%
	N=79 (4/8 mg/kg pooled)	N=35 (600 mg)	N=32 (150 mg)	N=9 (80 mg)
<b>GFR stabilization</b>	✓ (12 months)	✓ (18 months)	✓ (24 months)	✓ (12 months)
<b>Hematuria resolution</b>	✓	No data	✓	✓
<b>Safety</b>	✓ Well tolerated, no overall ↑ infections, slight ↑ in URTIs vs. pbo	✓ Well tolerated (no pbo), no drug discontinuations	✓ Well-tolerated, slight ↑ in infections (& URTIs) vs. pbo	✓ Well-tolerated (no pbo) 240 mg ↑ infections
<b>P3 Dosing</b>	400 mg SC, Q4W	600 mg SC, Q2W	150 mg SC, QW	80 mg SC, Q4W

Notes: Cross-trial comparisons are inherently limited and presented for hypothesis-generating purposes only. Zigakibart IgA / Gd-IgA data at W40; UPCR data at W52 (only timepoint available); change from baseline is not pbo-controlled; N represents patients on dose(s) for which data is shown. Atacicept infections/URTIs placebo - (32%/0%), 25 mg (38%/0%), 75 mg (49%/9%), 150 mg (39%/6%). Povetacicept infection rates: Grade 1/2/≥3 – 80 mg 10%/5%/0%, 240 mg 18%/27%/3%. Sibeprenlimab infections/URTIs placebo - (55%/0%), 2 mg/kg (39.5%/8%), 4 mg/kg (56%/12%), 8 mg /kg (53%/5%). Sources: 2023 Mathur (NEJM); 2024 Barratt (ERA Presentation); VERA January 2024 R&D Day; ALPN 2024 WCN Investor Update; 2024Madan (ASN Presentation)

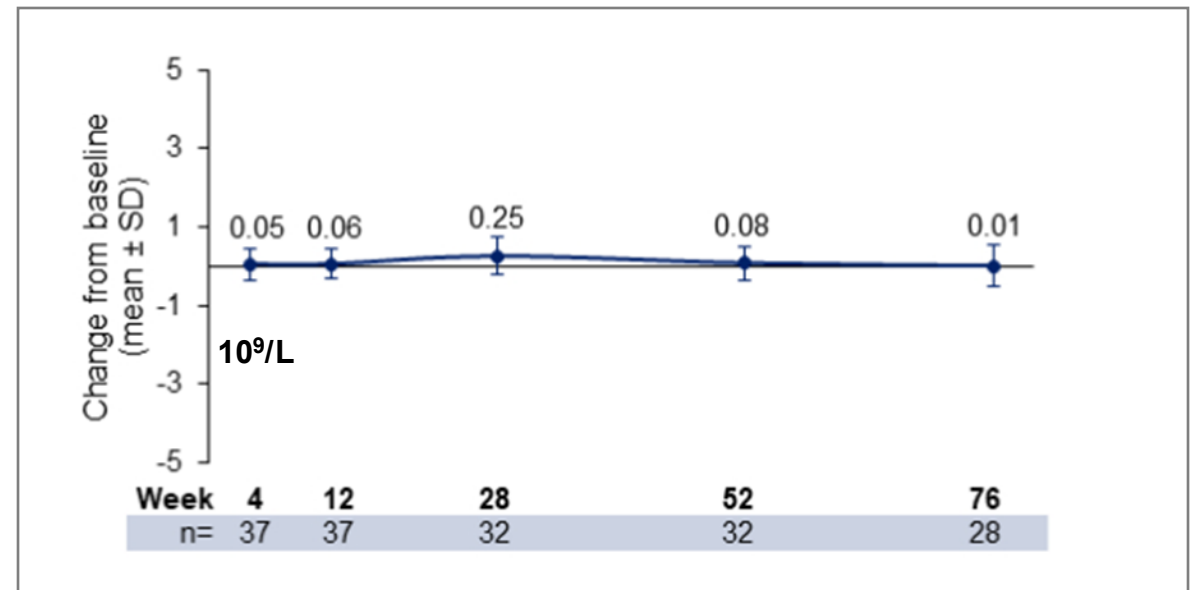
# BAFF inhibition is accompanied by the potential for significant long-term B cell depletion

Long-term BAFF inhibition significantly depletes all B cell populations...



~7-year belimumab data in SLE shows continuous BAFF inhibition lowers B cell populations from ~50% to ~99%, with most populations decreasing >80%.

... whereas chronic APRIL inhibition does not impact circulating lymphocytes

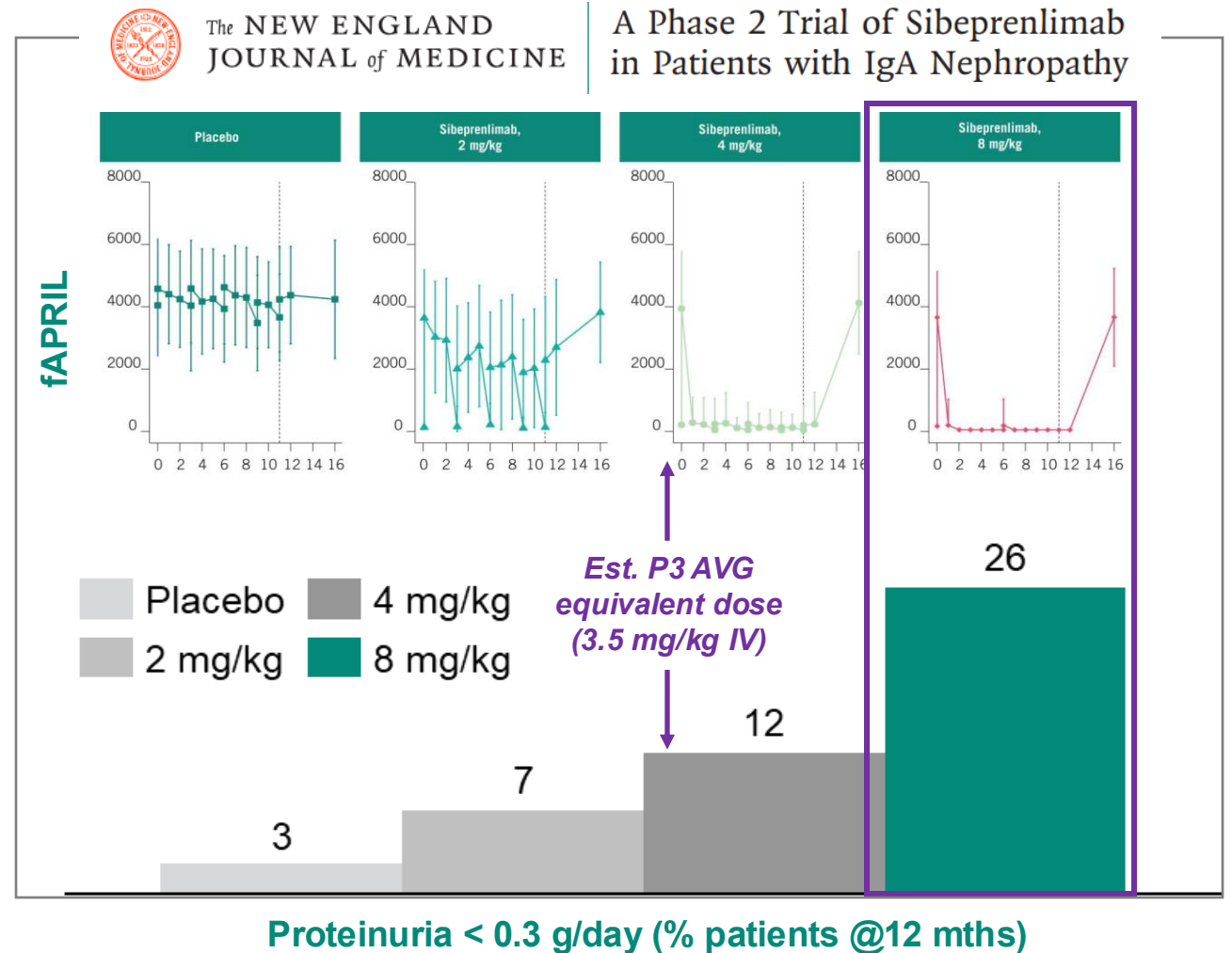


Long-term BAFF suppression, in an otherwise young and healthy patient population, is unnecessary given equivalent efficacy in IgAN from anti-APRILs and TACI-Fcs observed to date.

# Deeper APRIL suppression drives superior clinical efficacy

- Highest rates of **clinical remission** (proteinuria <0.3 g/day) for sibeprenlimab was accompanied by the **deepest levels of APRIL suppression**.
- **Safety profile consistent** across dose levels, with **no increase in overall infections**.
- Potential for anti-APRILs with **higher affinity** and **increased systemic exposure** to **provide more complete APRIL neutralization** throughout the dosing interval and **maximize clinical remission rates**.

JADE-001 has potential to **demonstrate superior clinical activity** by maximizing **remission rates** in **significantly more patients** than other anti-APRIL programs in development.

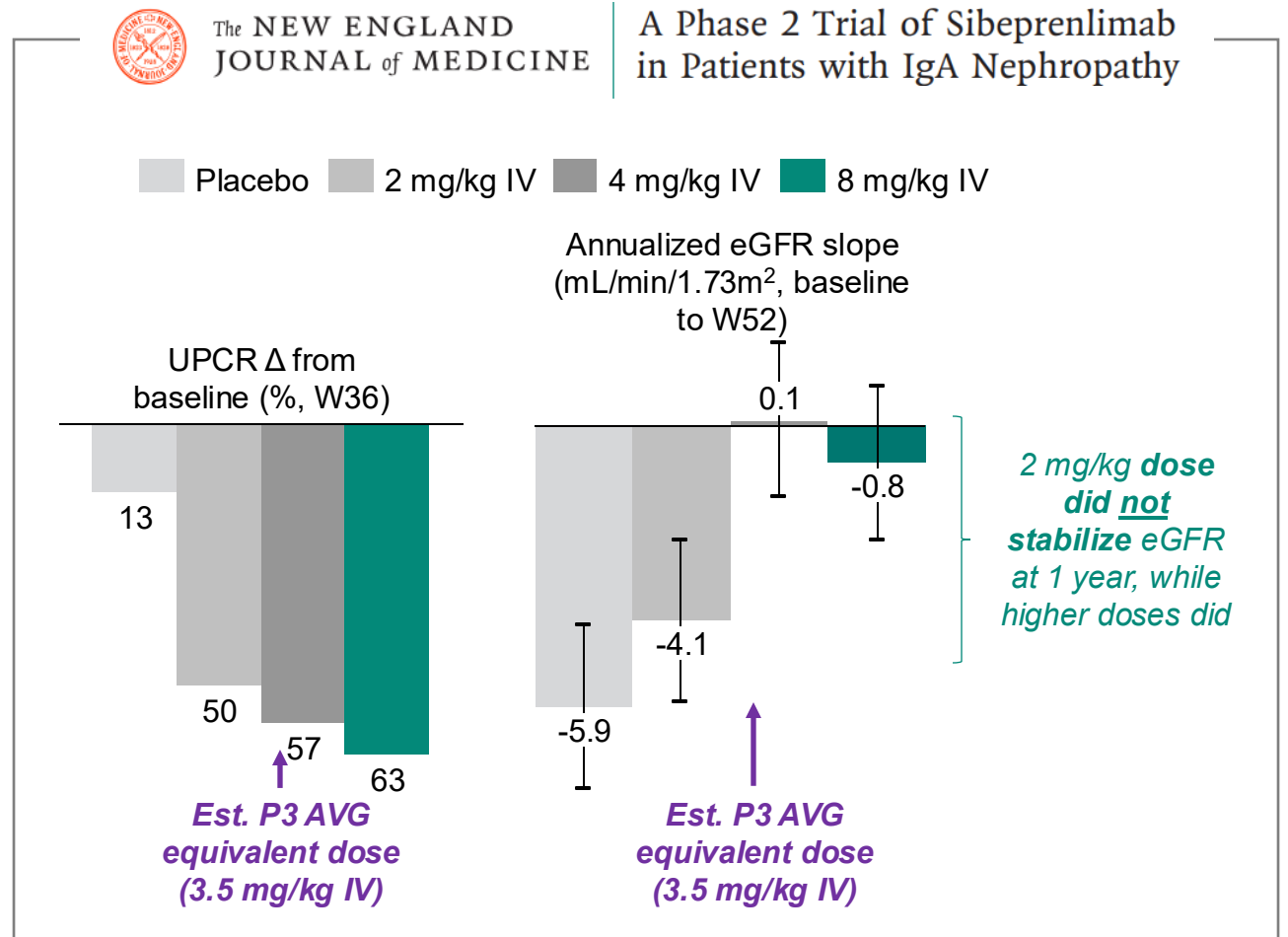


# Sibeprenlimab is potentially under-dosed in ongoing Phase 3 trial

Other anti-APRILs do not inhibit APRIL fully through the dosing interval

- **Sibeprenlimab** dosed as a single **400mg SC injection Q4W** in ongoing **global Phase 3 VISIONARY** trial.
- 400 mg SC Q4W **equates to ~3.5 mg/kg IV for average IgAN patient (2.5-6 mg/kg)**.
- Estimated Phase 3 equivalent dose range demonstrated **lower efficacy on key UPCR endpoints in Phase 2 ENVISION** trial.
- **~50%** of HV in P1 SAD showed positive antidrug antibody activity following single SC dose, which may further **impact PK, efficacy, and safety profile** in Phase 3.

Potential under-dosing of sibeprenlimab creates **additional opportunity for JADE-001** to demonstrate potentially best-in-class clinical activity for patients.



# Potentially best-in-class profile of JADE-001



Femtomolar APRIL Affinity +  
Half Life Extension



## Potentially best-in-class efficacy

APRIL inhibitors demonstrate greater proteinuria reduction and increased clinical remission rates with higher exposures and more complete APRIL suppression



## Infrequent Q8W+ dosing

Minimizes burden in a typically young IgAN patient population potentially requiring life-long therapy ( $\leq 6$  injections/year)



## Avoids unnecessary immunosuppression

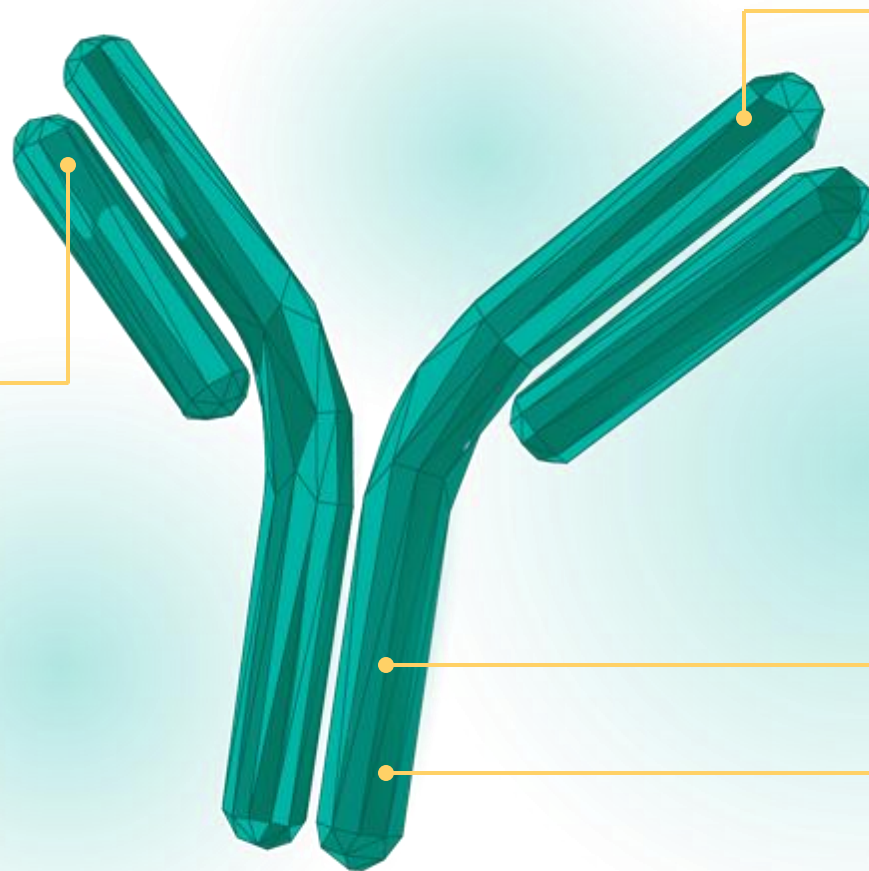
Selectively targeting APRIL provides disease modifying impact while avoiding B-cell depletion associated with BAFF inhibition



# Potentially best-in-class properties of JADE-001

Novel IP for composition of matter into mid-2040s

*De novo* antibody discovery campaign pursued to achieve fully-human, potentially best-in-class mAb



## Ultra-high (fM) APRIL binding affinity

- Binds **APRIL** to neutralize activity
- **Greater APRIL binding affinity** than sibeprenlimab, zigakibart, povetacicept and atacicept

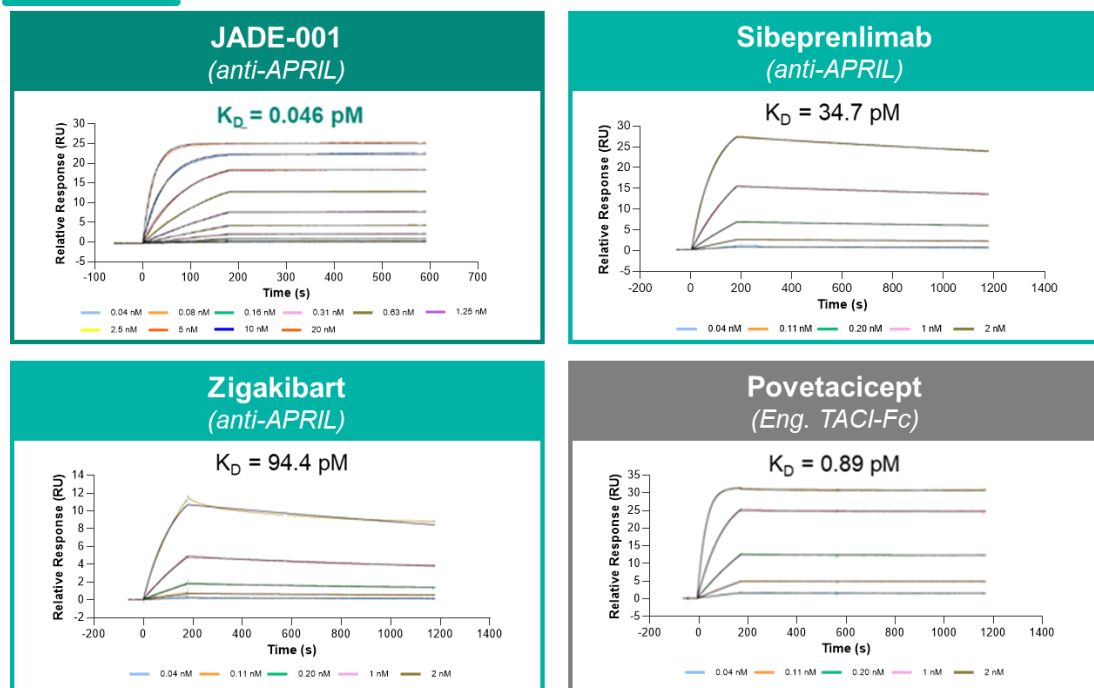
## Half-life extension through validated YTE Fc modification

- Longer exposure intended to maximize efficacy and reduce dosing frequency

## Effector-null human IgG1 Fc

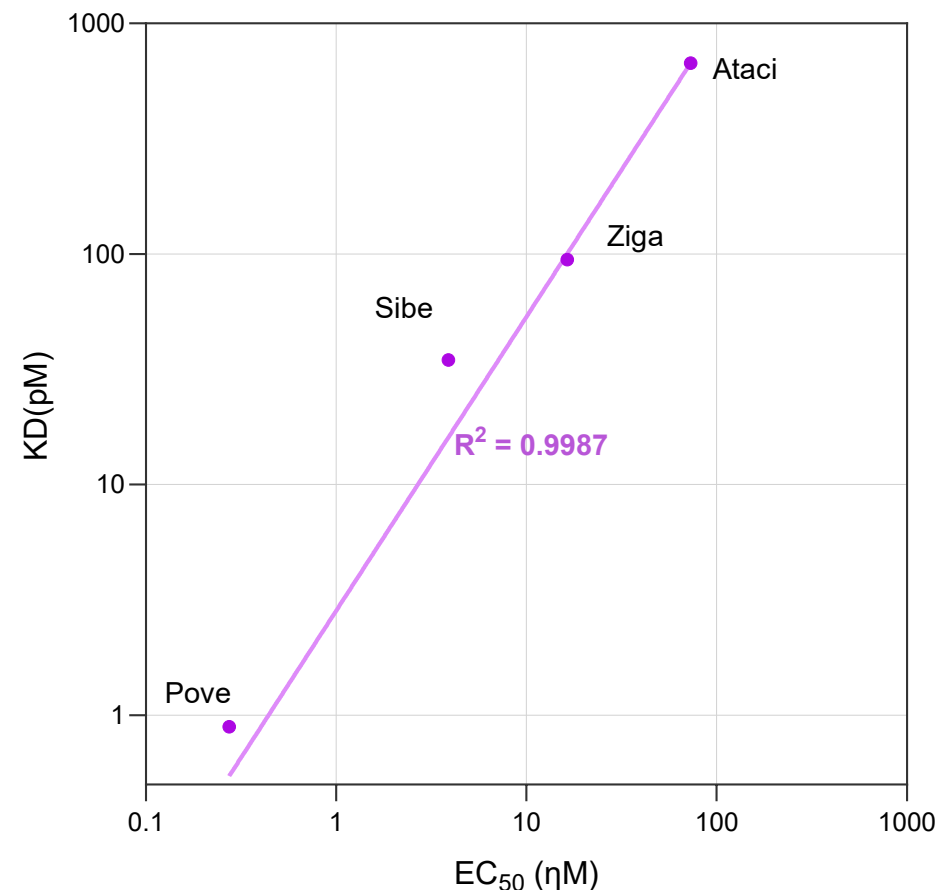


# JADE-001 has femtomolar affinity and a slow off-rate that is superior to other anti-APRILs currently in development



	$K_a$ (1/Ms)	$K_d$ (1/s)	$K_D$ (pM)	Affinity vs JADE-001
Sibeprenlimab	3.9E+06	1.4E-04	34.7	~755x↓
Zigakibart	2.5E+06	2.4E-04	94.4	~2,050x↓
Povetacicept	1.2E+07	1.1E-05	0.89	~20x↓
<b>JADE-001</b>	<b>2.3E+06</b>	<b>1.1E-07</b>	<b>0.046</b>	-

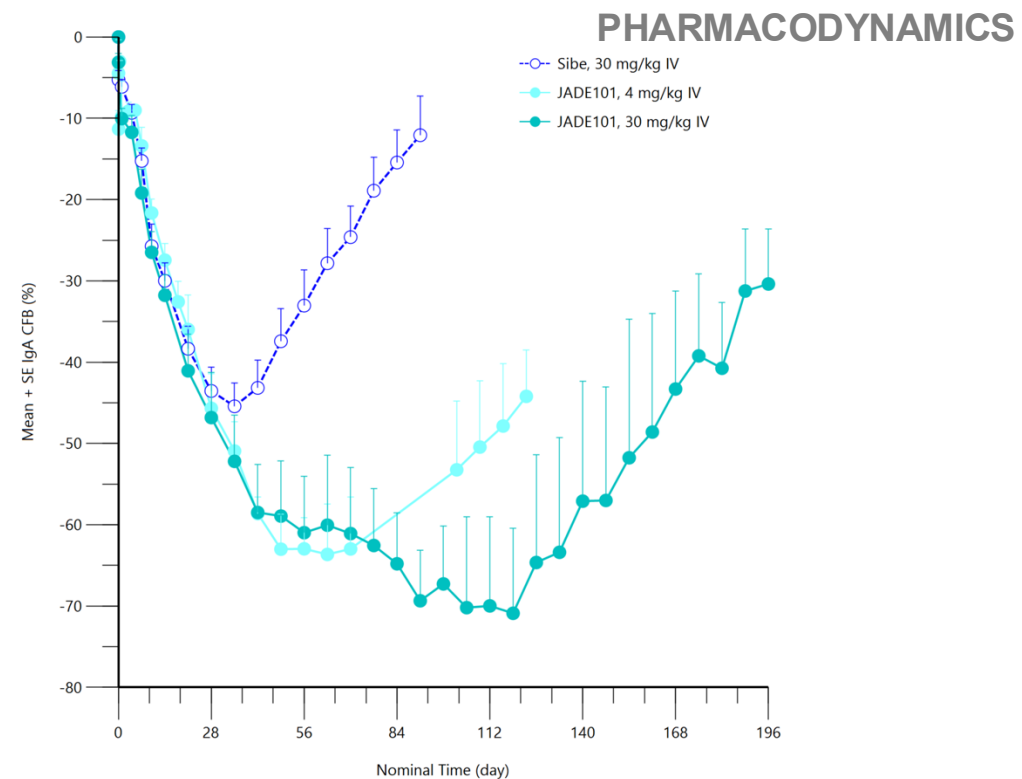
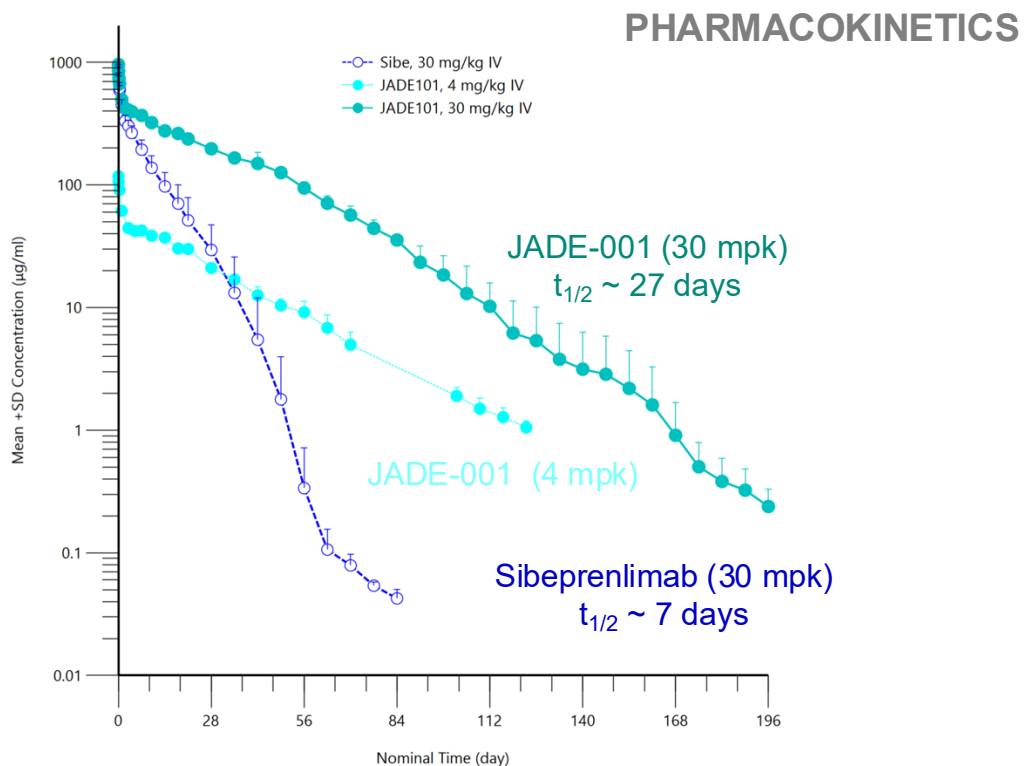
APRIL affinity by SPR is highly predictive of *in vivo* potency to lower serum IgA in humans



# JADE-001 exhibits a highly differentiated NHP PK/PD profile from sibeprenlimab

>3X increased half-life compared to sibeprenlimab in NHPs coupled with successful mitigation of TMDD ...

... which is accompanied by deep and prolonged IgA reduction in NHPs following a single, saturating dose



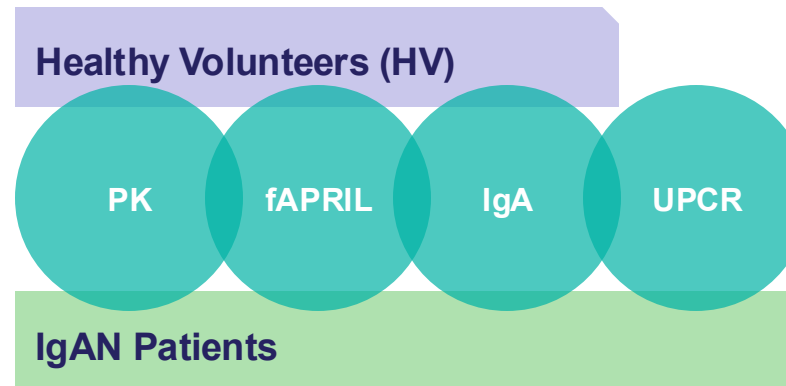
**JADE-001 has the potential to extend dosing interval through low clearance via half-life extension, target-mediated drug disposition mitigation & ultra-high (fM) human affinity.**

# Anticipated 1H26 HV data potentially positions JADE-001 for accelerated development

PK, fAPRIL and IgA HV data expected in 1H 2026 and will define the dose and schedule designed to fully suppress fAPRIL throughout the dosing interval in IgAN patients.



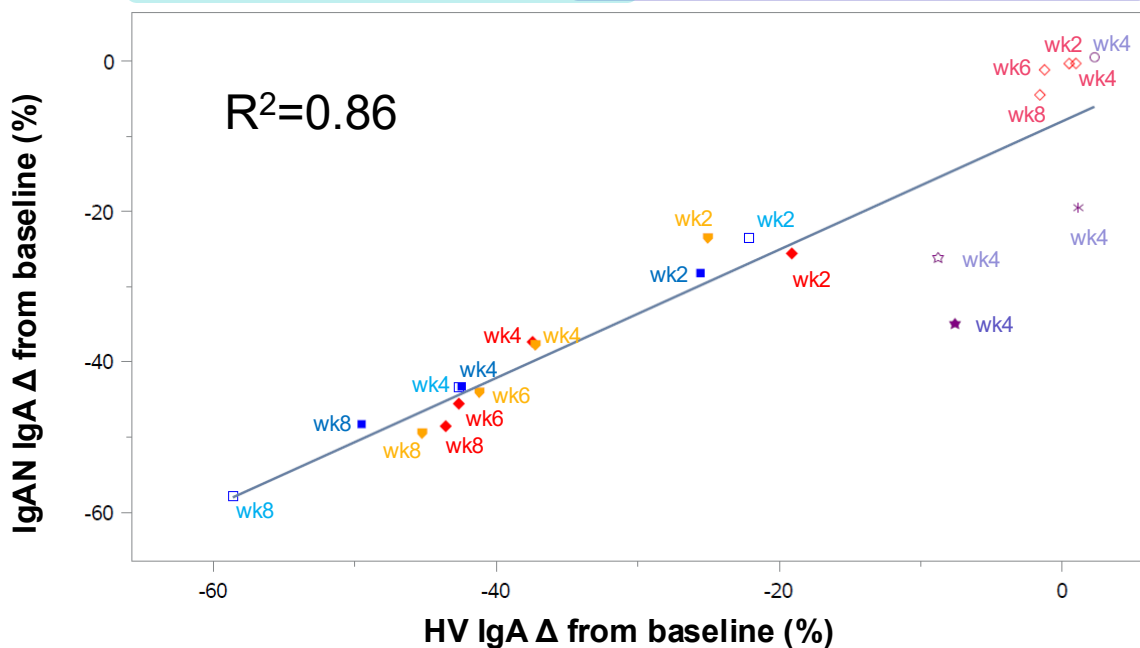
- Anti-APRIL MOA provides **biomarker rich data predictive of clinical efficacy**
- **Consistent PK/PD** relationships in **HV and IgAN patients**
  - HV PK highly predictive of IgAN PK and directly linked to fAPRIL suppression
  - HV IgA reduction expected to highly correlate with IgAN IgA reduction
  - Early IgA response expected to highly correlate with future UPCR reduction in IgAN



# Biomarker-rich, directly translational HV data supports the potential to accelerate clinical development

IgA reduction in HVs is **highly correlated** with IgA reduction in IgAN patients at **multiple time points**...

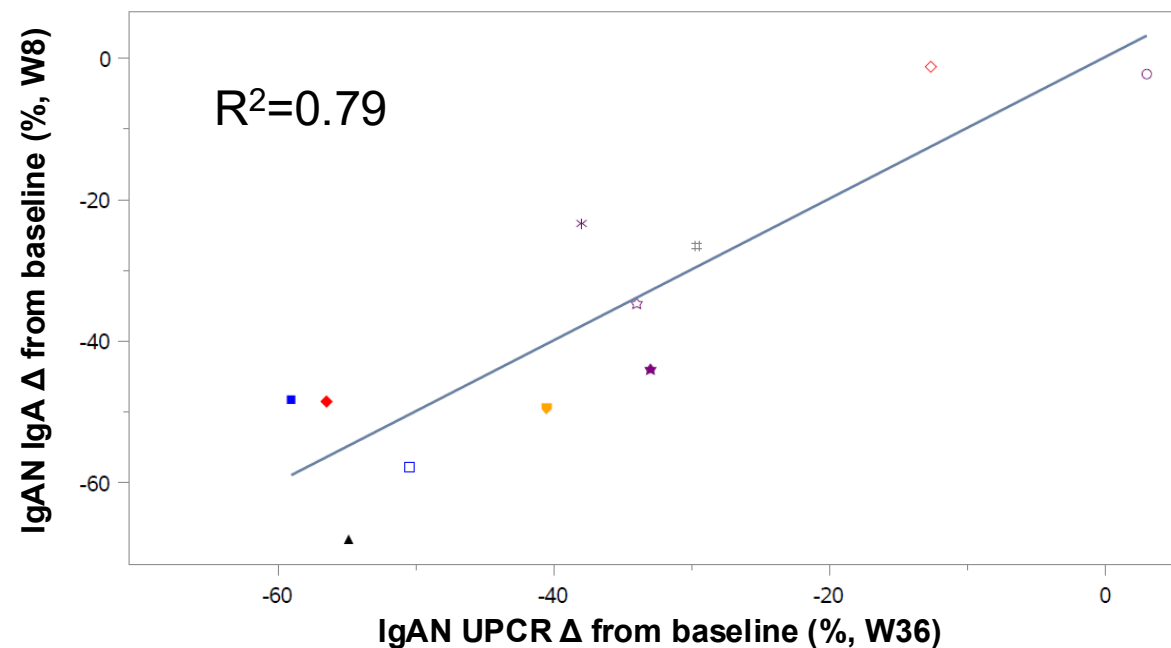
**IgAN Patients** VS **Healthy Volunteers**



- ◆ Sibeprenlimab Pooled
- Povetacicept 80 mg
- Atacicept Placebo
- ☆ Atacicept 75 mg
- ◆ Zigakibart 600 mg
- ◇ Sibeprenlimab Placebo
- Povetacicept 240 mg
- Atacicept 25 mg
- ★ Atacicept 150 mg
- ⊞ Felzartamab 16 mg/kg 9 doses
- ▲ Mezagitamab 600 mg 16 doses

...and **early IgA reduction** further correlates with **W36 UPCR reduction**, the endpoint for accelerated approval

**IgAN Patients**



# Pipeline beyond JADE-001

## Additional Jade programs expected to focus on best-in-class product profiles in high-value autoimmune indications

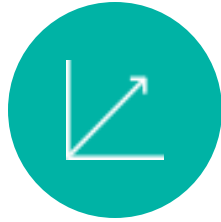
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Autoimmune indications with **significant market opportunity**



Potentially **best-in-class** and **best-in-indication** product profile



Potential **rapid path** to clinical PoC



**Limited competition** expected



**Jade team expertise**

Evaluating additional opportunities to **build pipeline of potentially best-in-class** autoimmune therapies.

# Jade Biosciences is advancing potentially best-in-class therapies for autoimmune diseases

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Patient friendly, infrequent dosing

## Capitalization following close of merger with Aerovate

**\$49.9M** cash as of March 31, 2025

**+\$192.7M** net proceeds from PIPE on April 28, 2025

	Number of Shares
Common stock	
Common stock equivalents	
Common stock & common stock equivalents	
Shares outstanding	32,235,927
Preferred stock (as converted to common stock)	12,622,000
Pre-funded warrants	7,766,247
<b>Total outstanding</b>	<b>52,624,174</b>

Notes: The cash balance figure is preliminary and is subject to change pending completion of our unaudited financial statements for the quarter ended 3/31/25. Additional information and disclosures would be required for a more complete understanding of our financial position and results of operations as of 3/31/25. Our independent registered public accounting firm has not audited, reviewed or performed any procedures with respect to this figure and, accordingly, does not express an opinion or any other form of assurance about them. Number of shares are on an as-converted basis and following the 1:35 reverse stock split effected in connection with the merger. The pro forma, post-split fully diluted share count, which includes equity incentives such as employee stock options, is approximately 60.6 million shares. Refer to AVTE and JBIO SEC filings for additional information.





# Thank you

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