



# Corporate Presentation

November 2024

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# Jade Biosciences is developing potentially transformative therapies for high-value Inflammation and Immunology indications

Jade's mission is to deliver best-in-class therapies for patients living with autoimmune diseases.

- Developing potential **best-in-class therapies for the treatment of autoimmune diseases**, including IgA nephropathy (IgAN).
- Fourth company launched to research and develop **antibody candidates licensed from Paragon Therapeutics**, an antibody discovery engine founded by Fairmount.
- **Following in the footsteps of Apogee, Spyre, and Oruka**, which have collectively raised ~\$1.8B and have generated clinical data utilizing Paragon's half-life extension technology.

MOA	Program	Discovery	IND-enabling	Planned Clinical FIH	Planned Healthy Volunteer Data
anti-APRIL	JADE-001			2H25	1H26
Undisclosed	JADE-002			1H26	
Undisclosed	JADE-003			1H27	

# Experienced management team with backing from Paragon

## Management



**Tom Frohlich**  
CEO



**Andrew King**  
CSO, Head of R&D



**Hetal Kocinsky**  
CMO



**Valerie Fauvelle**  
SVP, Regulatory & Quality



**Jason Wright**  
SVP, Chemistry, Manufacturing & Controls



**Jonathan Quick**  
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**Elizabeth Balta**  
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**Amy Sullivan**  
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**Sandy Lewis**  
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# JADE-001: a potential best-in-class anti-APRIL mAb for IgAN

# Jade is developing a potential best-in-class anti-APRIL mAb designed to have disease-modifying MoA in IgAN



Estimated \$10B+ newly branded market



*Current approved treatments don't adequately address young patient population with need for **long-term disease-modifying** therapy*



Anti-APRIL mechanism is potentially disease-modifying



*Shown to reduce **pathogenic IgA** and proteinuria, and **preserve kidney function***



JADE-001 has potential best-in-class profile



*Designed to have superior potency and half-life for **maximal efficacy** & **convenient dosing** in young patient population requiring life-long therapy*



Efficient development path to PoC and market

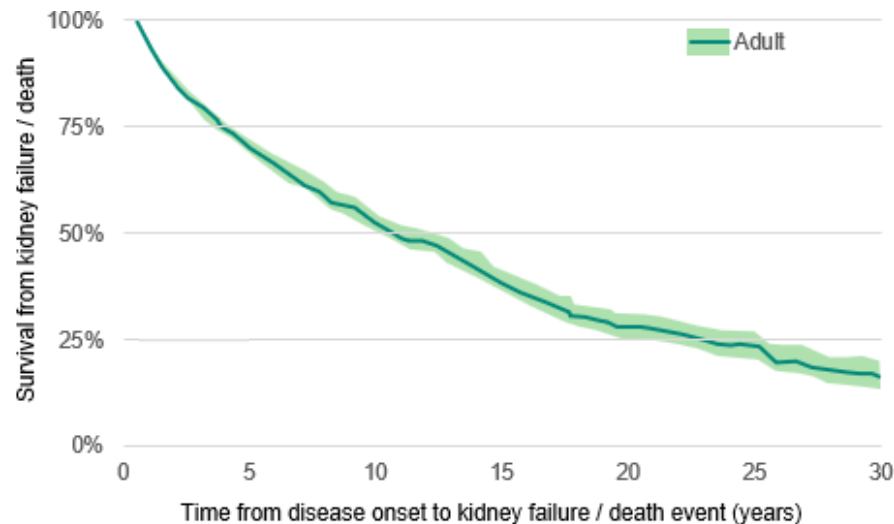


*HV IgA **biomarker closely correlated with efficacy in IgAN**; Potential **surrogate endpoints** support potential IgAN approval*

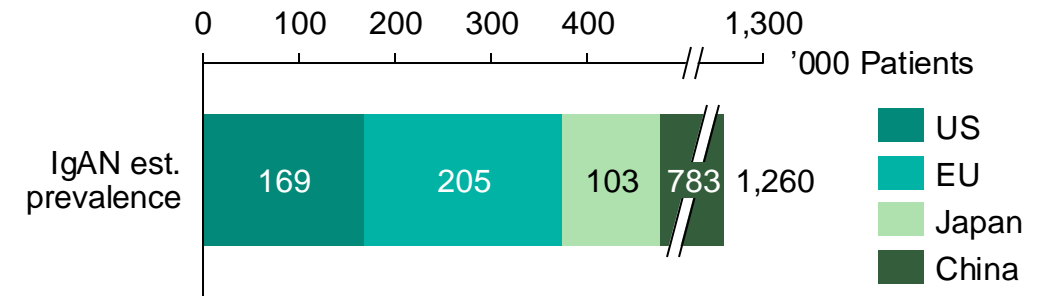
# ~169K+ IgAN patients in US, majority with persistent proteinuria, representing potential \$10B+ market

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-US market potential



- At a prevalence of ~169K in the US, with **~60-75% of patients with persistent proteinuria** requiring treatment per international guidelines, along with pricing of branded IgAN agents, the **US TAM is estimated to exceed \$10B annually**.

There is a high unmet need for **disease-modifying treatments that are safe, well-tolerated, and convenient for life-long therapy in a young patient population.**

# Current IgAN treatments leave significant unmet need, with no disease-modifying (i.e., long-term GFR-stabilizing) approved therapeutics

	ACEi / ARB	Systemic glucocorticoids	SGLT2i	Filpari	Tarpeyo	Fabhalta	Ideal IgAN therapy
MoA	Renin-angiotensin system inhibition	General immunosuppression	SGLT2 inhibition	Dual endothelin / angiotensin inhibition	GI-released systemic glucocorticoid	Complement Factor B inhibitor	
Status	Used off-label	Used off-label	Approved for CKD	Approved	Approved	Accelerated approval	
Therapeutic rationale	Supportive therapy (reduce glomerular pressure)	Immunosuppression	Supportive therapy	Supportive therapy	Immunosuppression	Reduce complement-driven pathology	Disease-modifying (depletes Gd-IgA1, stabilizes GFR)
Proteinuria reduction	~↓30-40%	~↓30-50% at 6M; none at 3Y	↓26% pbo-adj (UACR)	↓35% control-adj at 36W	↓32% pbo-adj at 36W	↓38% pbo-adj at 36W	60%+, ideally to < 0.3-0.5 g per day
GFR stabilization	X	X	X	X	X	No long-term data	✓
Safety	BBW (fetal tox), hyperkalemia, angioedema, AKI	Severe infections, edema, hypertension, bone density loss, etc.	UTIs, genital fungal infections, volume depletion	BBW + REMS (liver & pregnancy); hypotension, edema, AKI, hyperkalemia	Immunosuppression, edema, hypertension, weight increase, URTI	BBW + REMS (serious bacterial infections); URTI, abdominal pain	No notable safety issues, minimal immunosuppression
Annual dosing	365 x (or greater) 	180-270 x (6 to 9-month course) 	365 x 	365 x 	270 x (9-month course) 	730 x 	4-6 x (or fewer) 

Notes: Proteinuria reduction based on UPCR. Data from Praga & Nakamura trials (ACEi / ARB), STOP-IgAN & TESTING (glucocorticoids), DAPA-CKD (SGLT2i), PROTECT (Filpari), NeflgArd (Tarpeyo), APPLAUSE-IgAN (Fabhalta).  
 Sources: UpToDate; 2003 Praga (J Am Soc Nephrol); 2006 Li (Am J Kidney Dis); 2000 Nakamura (Am J Nephrol); 2022 Lv (JAMA); 2023 Campbell (Dove Press); Filpari Label; Tarpeyo Label; Fabhalta Label; KOL interviews. CKD – chronic kidney disease; UACR –urine albumin to creatinine ratio; BBW – black box warning; REMS – risk evaluation and mitigation strategy; AKI – acute kidney injury; URTI – upper respiratory tract infection

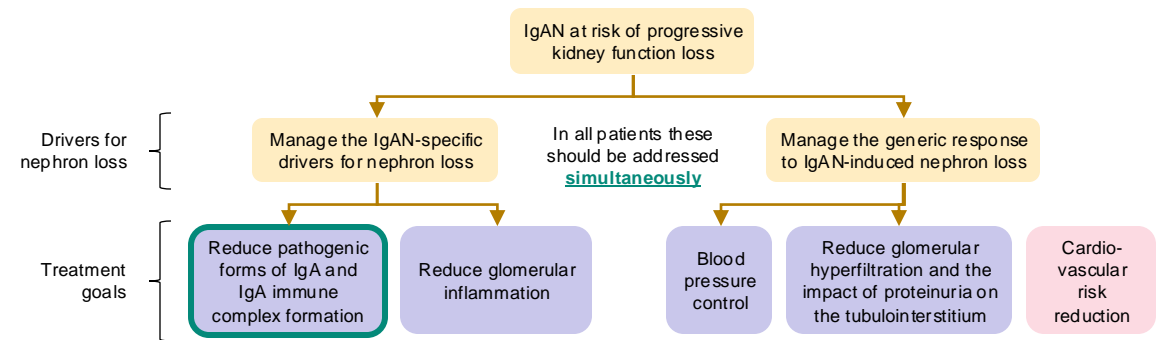


# Proposed updates to KDIGO guidelines highlight the need for therapies like JADE-001, which may reduce pathogenic IgA

Proposed guidelines expected to increase IgAN diagnosis and redefine treatment goals...

... and further underscore the importance of reducing pathogenic IgA in the treatment paradigm

- |                                   |  |
|-----------------------------------|--|
| <p><b>Patient population</b></p>  | <ul style="list-style-type: none"> <li>Recommends a <b>kidney biopsy in all adults with proteinuria <math>\geq 0.5</math> g/d</b> where IgAN is a possible diagnosis.</li> <li>Recommends all patients be <b>enrolled in an IgAN registry</b>.</li> </ul>  |
| <p><b>Risk of progression</b></p> | <ul style="list-style-type: none"> <li>Redefines risk of progressive loss of kidney function for <b>patients with <math>\geq 0.5</math> g/d of proteinuria</b> on or off treatment (previously <math>\geq 0.75</math>-1 g/d after maximal supportive care).</li> <li>Recommends <b>additional treatment should be initiated in all cases</b> where patients have proteinuria <math>\geq 0.5</math> g/d.</li> </ul> |
| <p><b>Proteinuria target</b></p>  | <ul style="list-style-type: none"> <li>Establishes a new, ideal treatment goal: proteinuria should be maintained at <b><math>&lt; 0.5</math> g/d, preferably <math>&lt; 0.3</math> g/d</b>.</li> <li><b>0.3 g/d</b> is the highly <b>stringent cutoff for clinical remission</b> used in the sibeprenlimab Phase 2.</li> </ul>   |



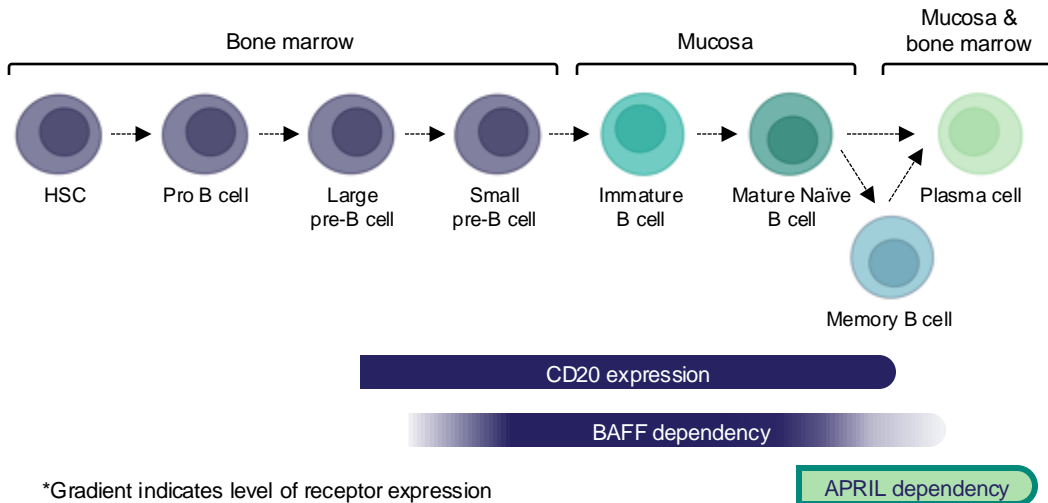
- Proposed guidelines state, “reduction or prevention of IgA immune complex formation should incorporate treatments that have been **proven to reduce pathogenic forms of IgA**”. Anti-APRILs and TACI-Fcs have **shown the best clinical data to date** for reducing pathogenic IgA.
- Guidelines also recommend therapies that prevent immune complex-mediated injury **should be used in combination with, and not as a replacement** for, therapies that reduce pathogenic IgA.

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

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

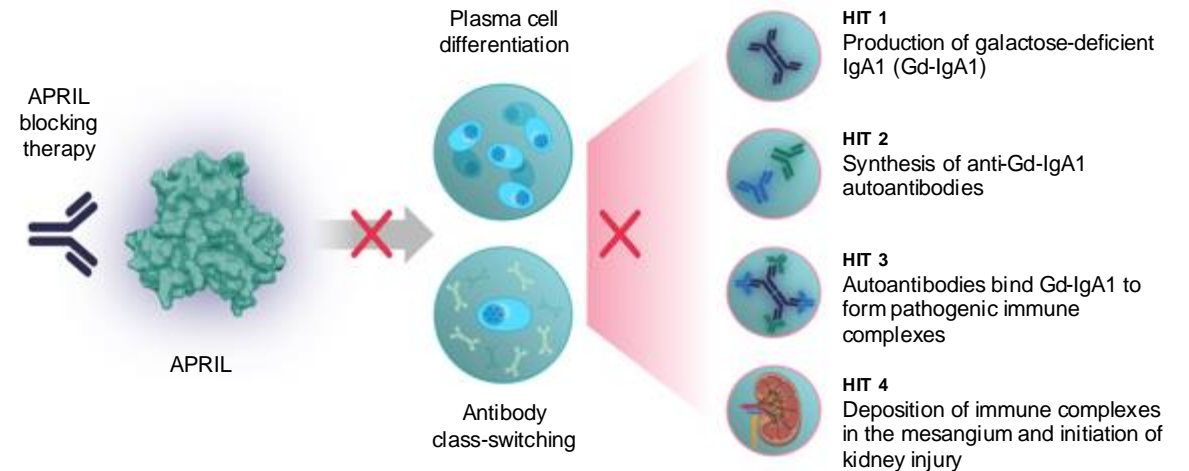
Broad B-cell depletion is ineffective in IgAN...

- 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**.



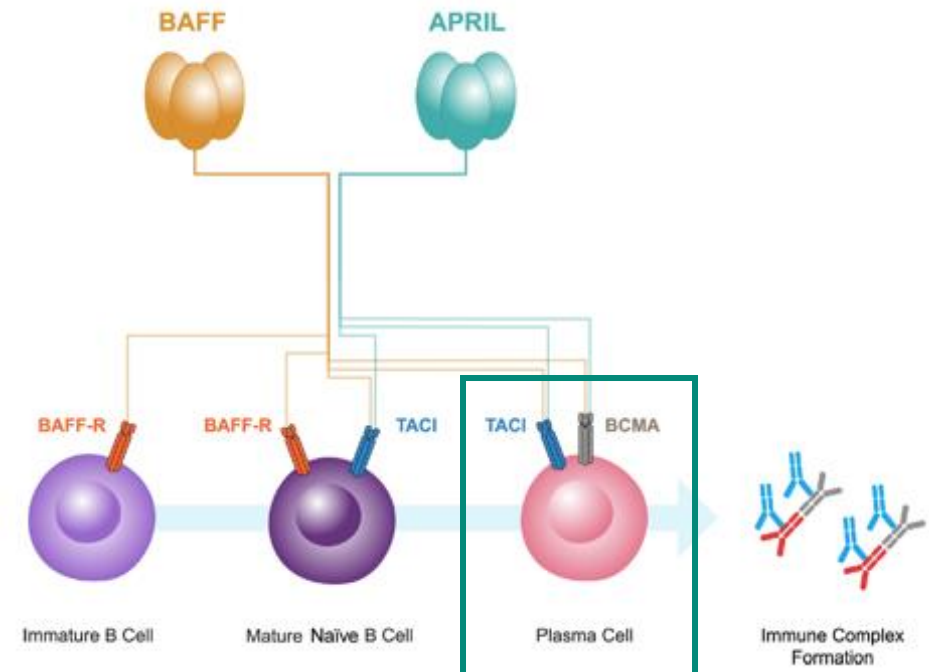
Neutralizing APRIL depletes Gd-IgA1, reduces proteinuria, and **preserves eGFR**, providing a **disease-modifying treatment** of IgAN without impacting B-cell development and maturation.

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

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

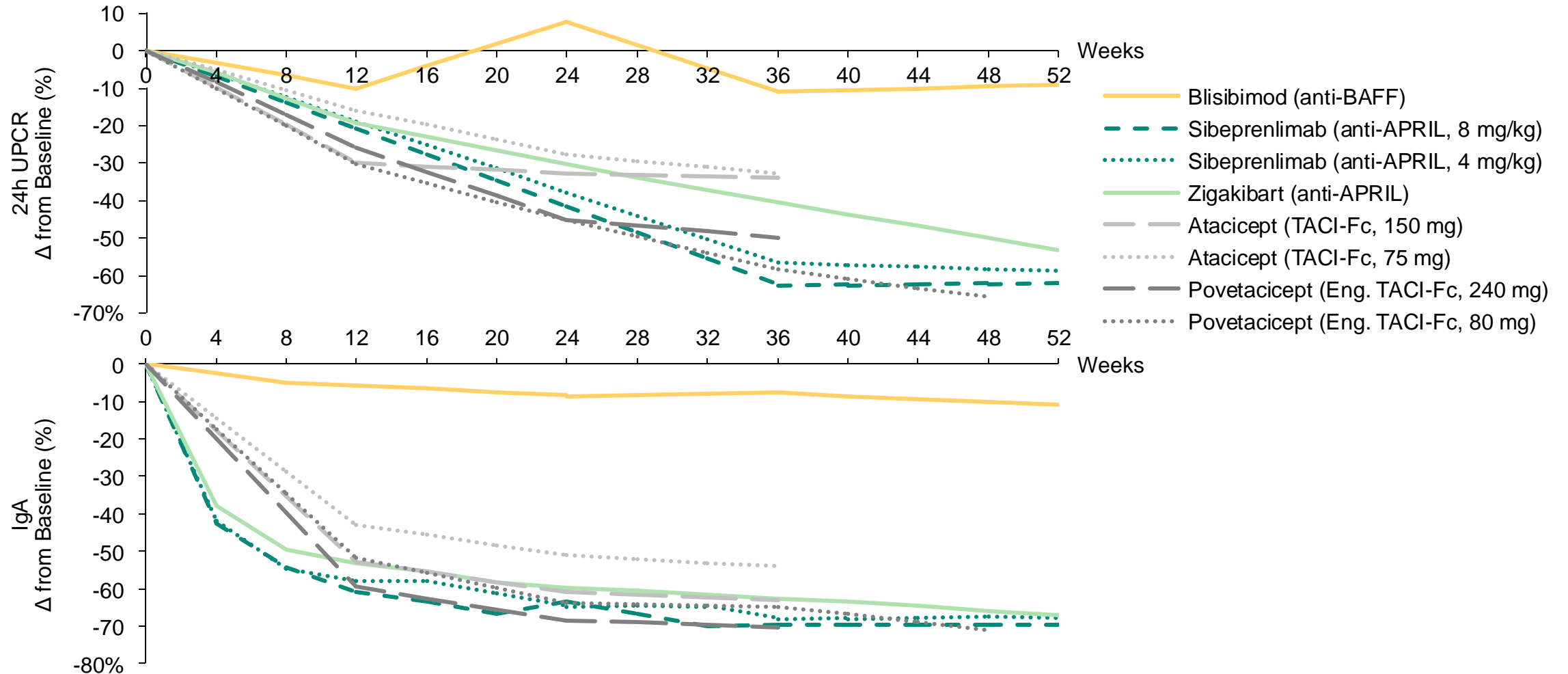
Targeting APRIL **selectively modulates plasma cells**, maintaining pool of mature B cells

	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



# Anti-APRILs have shown evidence of disease modification and clinical activity that matches or beats TACIs, with reduced immune suppression

	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>	<table border="1"> <tr> <th>IgA</th> <th>Gd-IgA1</th> <th>UPCR</th> </tr> <tr> <td>67%</td> <td>60%</td> <td>60%</td> </tr> </table> <p>N=79 (4/8 mg/kg pooled)</p>	IgA	Gd-IgA1	UPCR	67%	60%	60%	<table border="1"> <tr> <th>IgA</th> <th>Gd-IgA1</th> <th>UPCR</th> </tr> <tr> <td>64%</td> <td>69%</td> <td>53%</td> </tr> </table> <p>N=35 (600 mg)</p>	IgA	Gd-IgA1	UPCR	64%	69%	53%	<table border="1"> <tr> <th>IgA</th> <th>Gd-IgA1</th> <th>UPCR</th> </tr> <tr> <td>63%</td> <td>64%</td> <td>33%</td> </tr> </table> <p>N=32 (150 mg)</p>	IgA	Gd-IgA1	UPCR	63%	64%	33%	<table border="1"> <tr> <th>IgA</th> <th>Gd-IgA1</th> <th>UPCR</th> </tr> <tr> <td>65%</td> <td>69%</td> <td>59%</td> </tr> </table> <p>N=9 (80 mg)</p>	IgA	Gd-IgA1	UPCR	65%	69%	59%
IgA	Gd-IgA1	UPCR																										
67%	60%	60%																										
IgA	Gd-IgA1	UPCR																										
64%	69%	53%																										
IgA	Gd-IgA1	UPCR																										
63%	64%	33%																										
IgA	Gd-IgA1	UPCR																										
65%	69%	59%																										
<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																								

“The goal is to reduce pathogenic IgA and **get the disease under control right away**. The **APRIL class will be the backbone** [of therapy]. This class will **become first-line**.”

– European KOL

“These therapies **may change the thinking in IgAN**. Instead of first starting with a hemodynamic agent and then going to prednisone... **now we would start with [anti-APRIL and anti-APRIL/BAFF]**.”

– US KOL

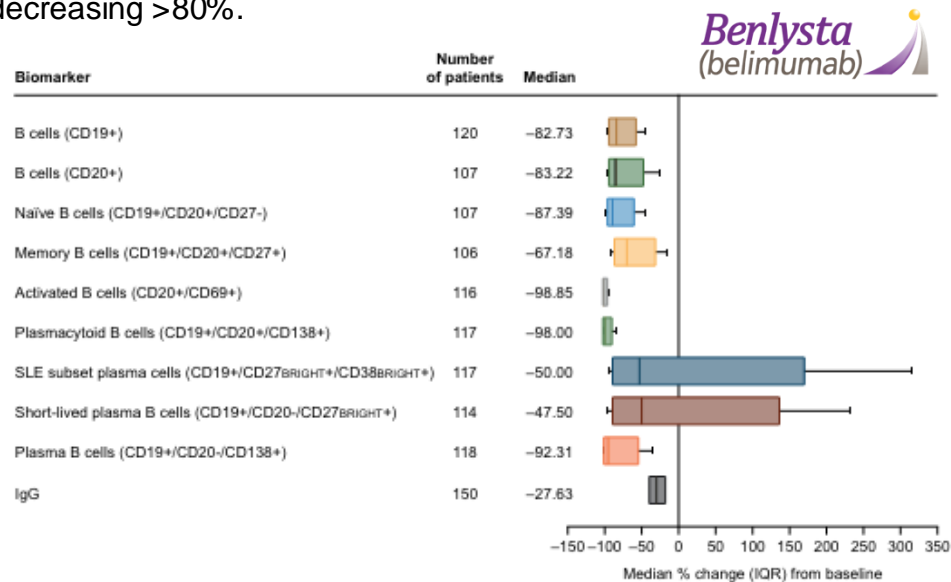
“If I biopsy a patient and they have clear inflammation, **if these were available, I would use them immediately** with ACEi / ARBs.”

– US KOL

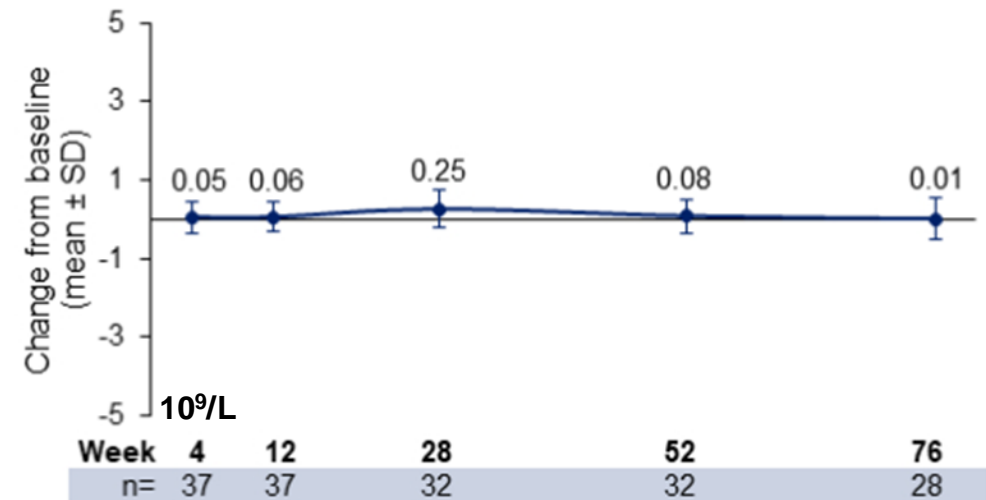
# BAFF inhibition is accompanied by the potential for significant long-term B cell depletion

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

- ~7-year data from belimumab 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.

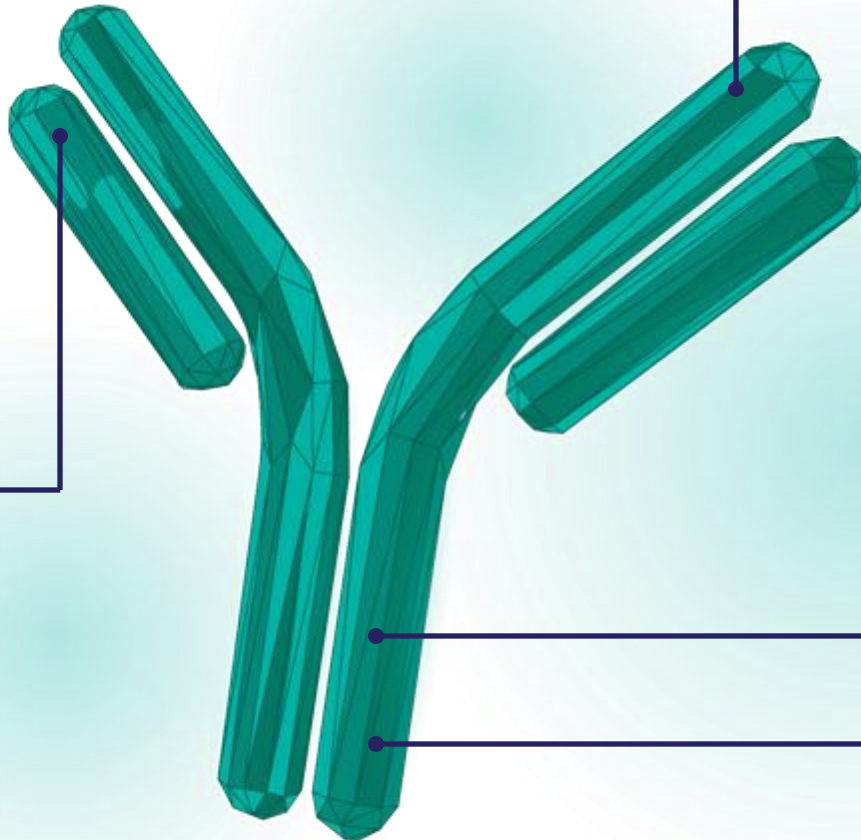
# JADE-001 is a potential best-in-class anti-APRIL

## Blocks APRIL with greater potency than clinical benchmarks

- Validated mechanism of action
- Binds **APRIL** to neutralize activity
- **Greater binding affinity** than sibeprenlimab ( $\geq 5x$ ) and zigakibart ( $\geq 14x$ )

Multiple antibody discovery strategies pursued to achieve potential best-in-class mAb

**Novel IP for composition of matter into 2040s**



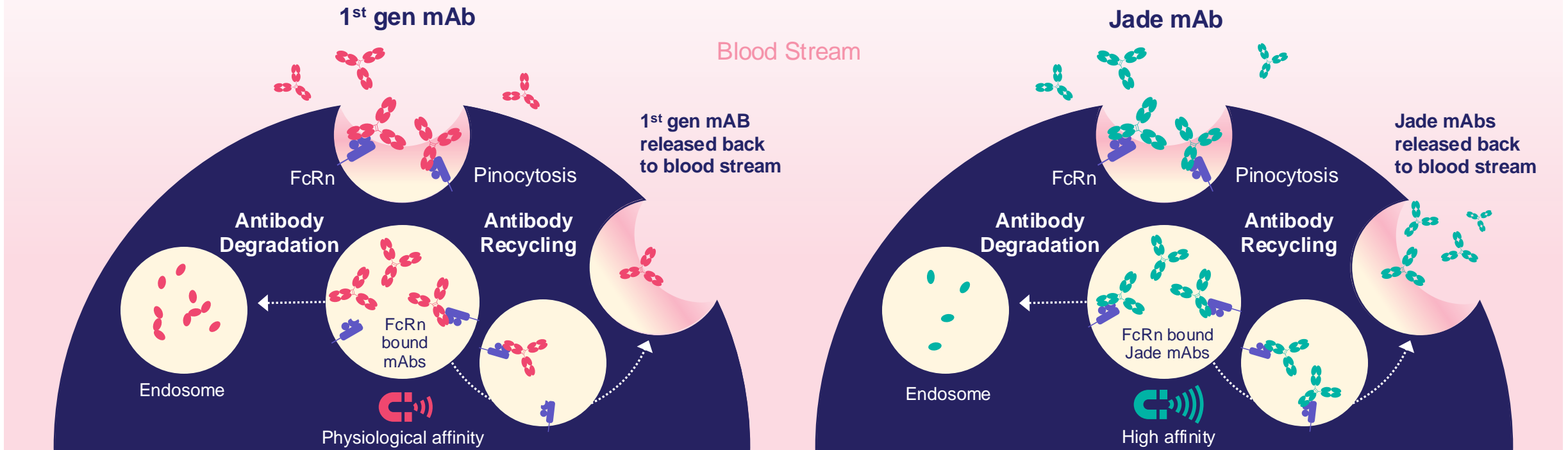
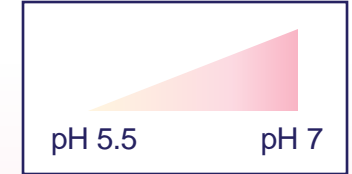
## Half-life extension through validated YTE Fc modification

- Longer exposure intended to reduce dosing frequency

Effector-null human IgG1 Fc

# Jade mAbs employ proven half-life extension (HLE) technology

- Jade mAbs designed to be recycled back into circulation more readily
- Drug exists at much higher levels for longer duration of effect
- Fewer injections decrease patient burden and can improve compliance and penetration





# JADE-001's goal is to introduce Q8W+ dosing for IgAN patients via HLE

Prior experience, including with Paragon-generated mAbs, indicates HLE could significantly improve dosing over anti-APRILs in development

- JADE-001 employs well-established HLE technology, with the potential for Q8W+ dosing.
- High potency can potentially further drive lower dosing frequency – which has already been demonstrated for APRIL by sibeprenlimab's Q4W dosing vs. zigakibart's Q2W dosing despite near-equivalent half-life.

	Human $t_{1/2}$ (days)	Est. Dosing Interval
<b>JADE-001 TPP</b> (HLE anti-APRIL mAb)	HV PK expected H1 2026 50+*	Targeting Q8W+
<b>Sibeprenlimab</b> (anti-APRIL mAb)	~23**	Q4W (400 mg)
<b>Zigakibart</b> (anti-APRIL mAb)	~20**	Q2W (600 mg)
<b>Atacicept</b> (TACI-Fc APRIL/BAFF)	6.7	QW (150 mg)
<b>Povetacicept</b> (TACI-Fc APRIL/BAFF)	3.7	Q4W (80 mg)

Sources: 2019 Myette (Kidney Intl); 2022 Mathur (KI Reports); 2018 Dulos (ASN Poster); 2020 Lo (ERA Poster); Apogee Corporate Presentation

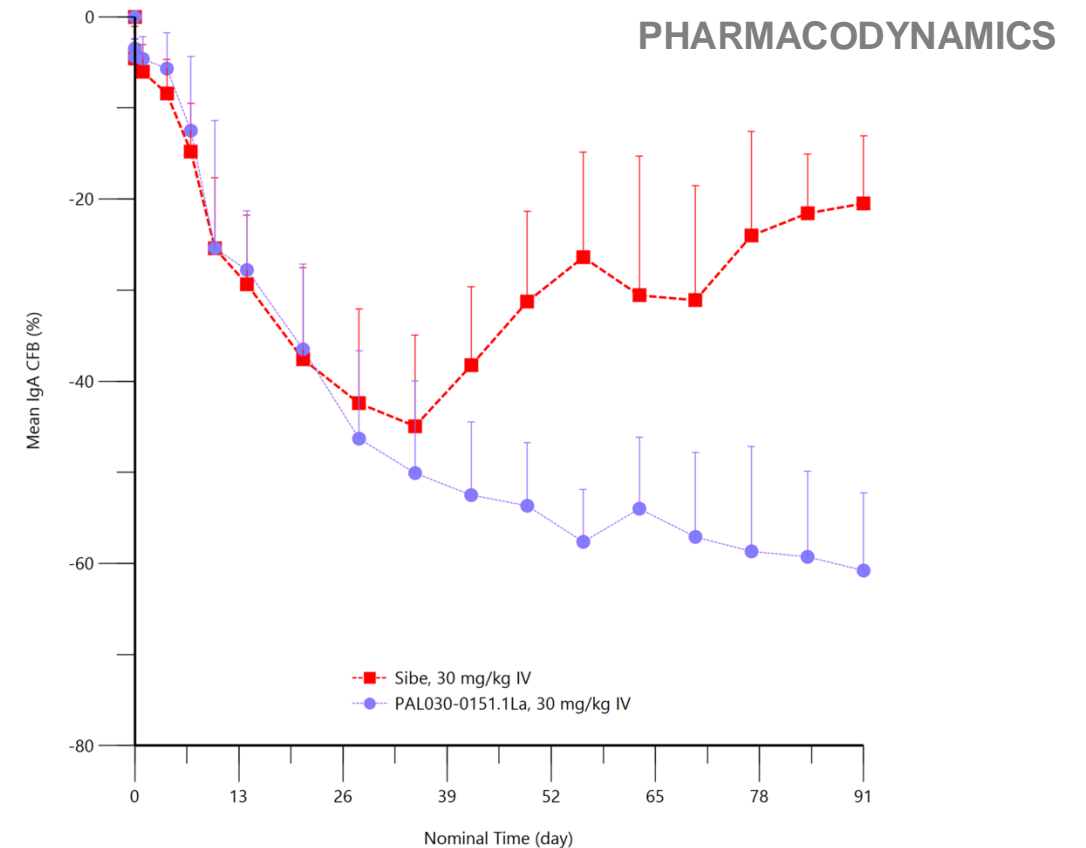
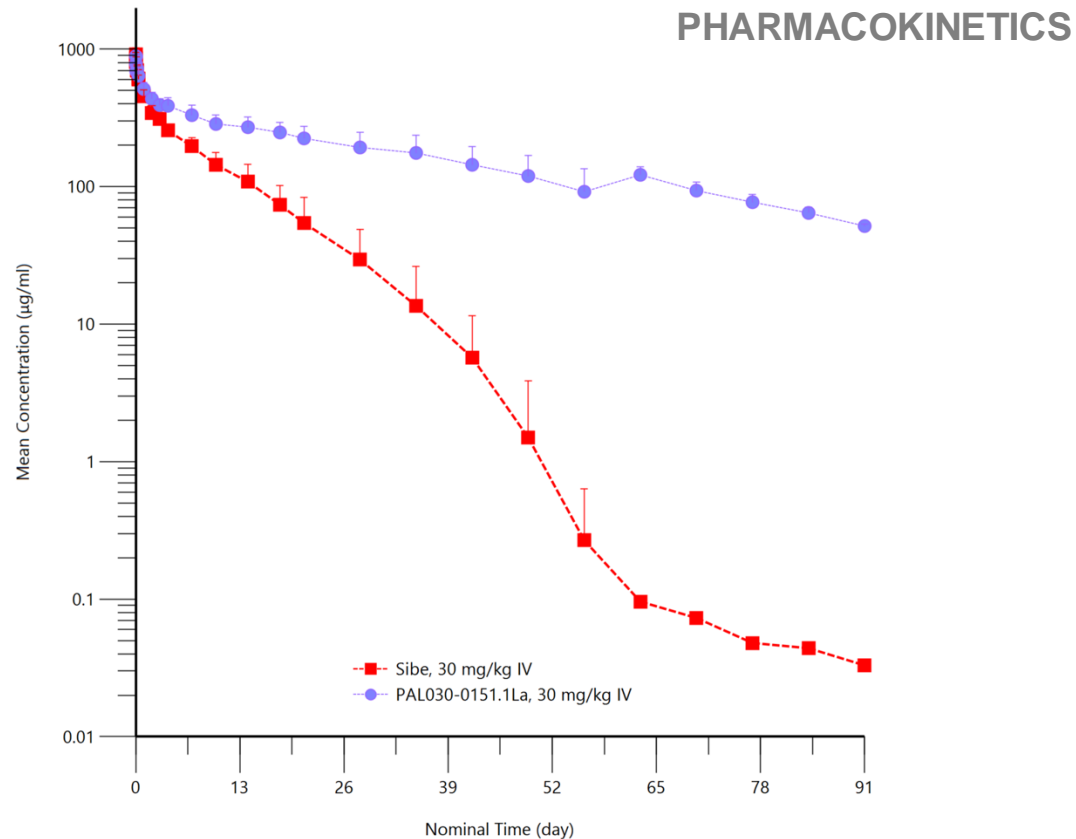
\*Based on single dose studies in NHPs dosed with JADE-001 initial clone. A development candidate has been selected from a pool of profiled clones. We have entered into an exclusive JADE-001 license agreement with Paragon.

\*\*Available anti-APRIL therapeutics demonstrate appreciable TMDD resulting in dose and dose frequency dependent  $t_{1/2}$ . Jade estimated  $t_{1/2}$  of benchmarks from publicly available data at the P3 dose and schedule via standard noncompartmental analysis of observed data bolstered with compartmental modelling approaches capturing clinically observed TMDD. Cross-trial comparisons are inherently limited and presented for hypothesis-generating purposes only.

# JADE-001 HLE strategy and profile in NHPs shows promise with early clone\*

~3X increased half-life over sibeprenlimab in NHPs...

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



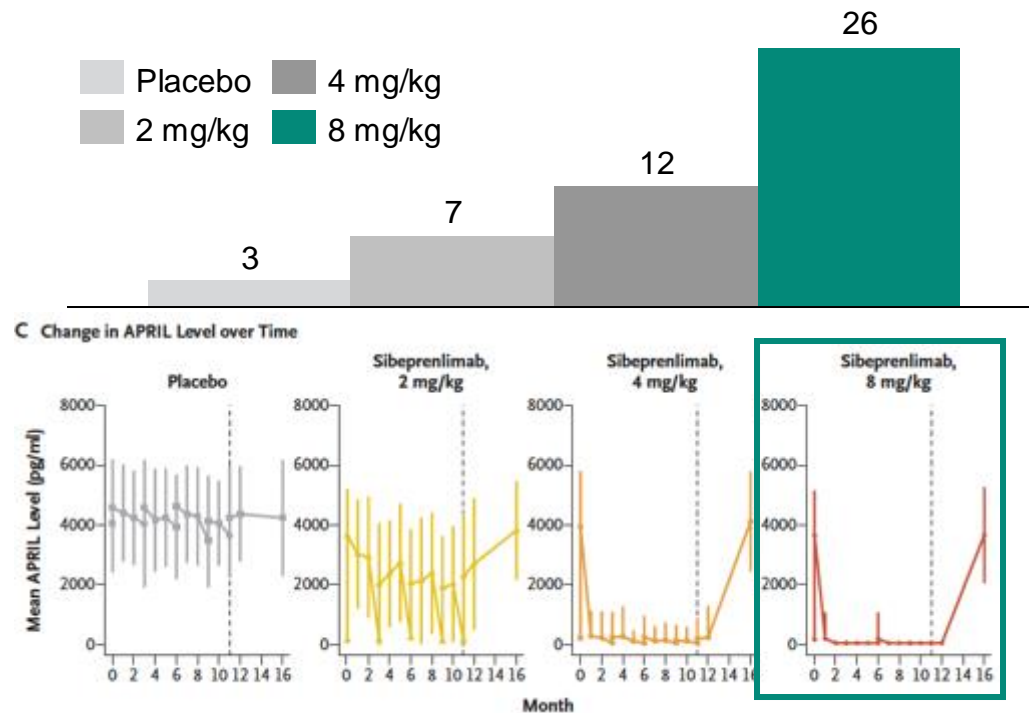
# Deeper APRIL suppression could drive superior efficacy

- The highest rates of **clinical remission** (<0.3 g/day urinary protein excretion) for sibeprenlimab were accompanied by the **deepest levels of APRIL suppression**.
- **Safety profile** was **consistent** across dose levels.
- Significant opportunity to drive **increased systemic exposure with HLE and maximize clinical remission**.
- JADE-001's **affinity** could further contribute to potential **best-in-class efficacy**.



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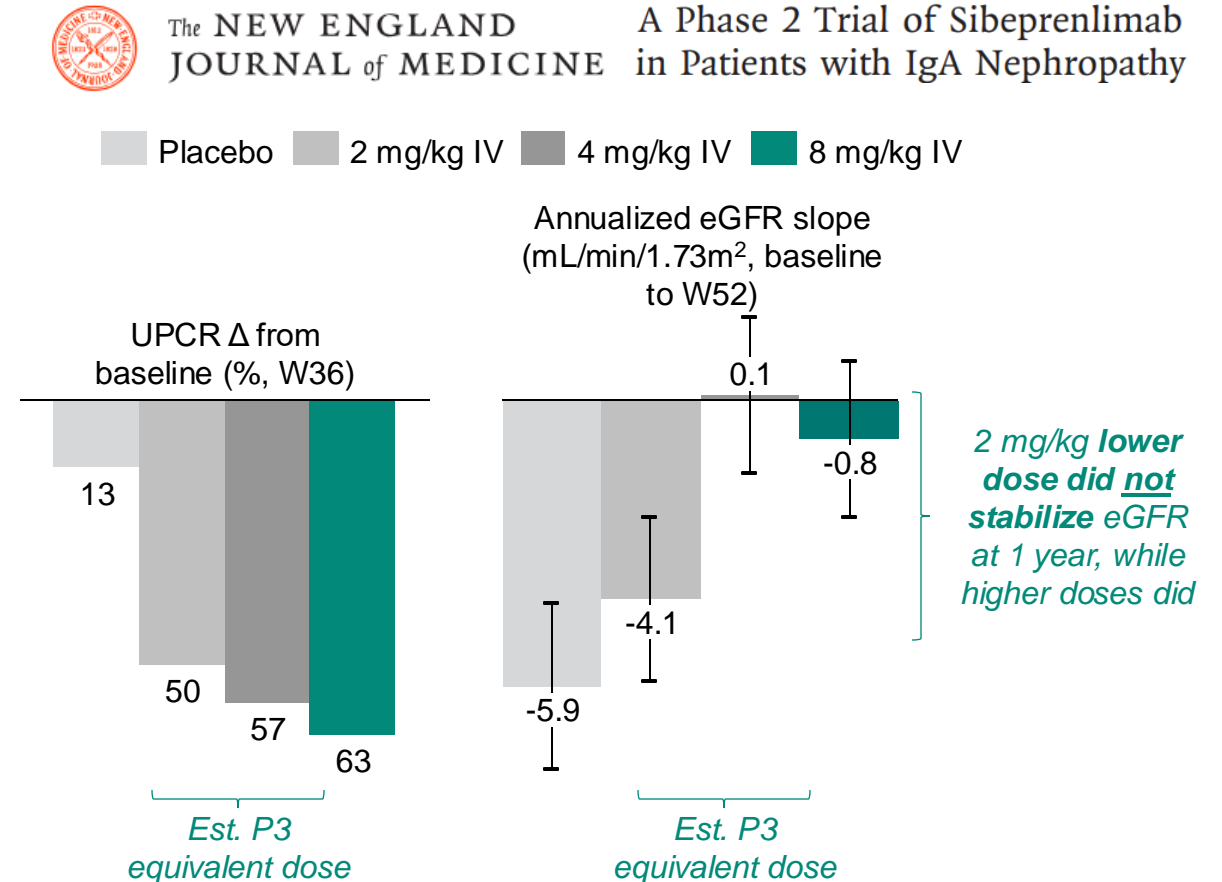
A Phase 2 Trial of Sibeprenlimab  
in Patients with IgA Nephropathy



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

- **Sibeprenlimab** is being dosed as a single **400mg SC injection Q4W** in ongoing **global Phase 3 VISIONARY** trial.
- 400 mg SC Q4W is **equivalent to ~3.5 mg/kg IV for average IgAN patient (range 2.5-6 mg/kg)**.
- The estimated Phase 3 equivalent dose range **demonstrated lower efficacy on key endpoints in Phase 2 ENVISION** trial (as seen on right).
- **~50%** of healthy volunteers in P1 SAD demonstrated positive antidrug antibody activity following a 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 potential best-in-class clinical activity for patients.

# Potential path to early clinical proof-of-concept and accelerated approval

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MOA	Program	Discovery	Phase 1 Initiation	Potential Healthy Volunteer Data	Potential Indications
anti-APRIL	JADE-001	Ongoing	2H25	1H26	IgAN

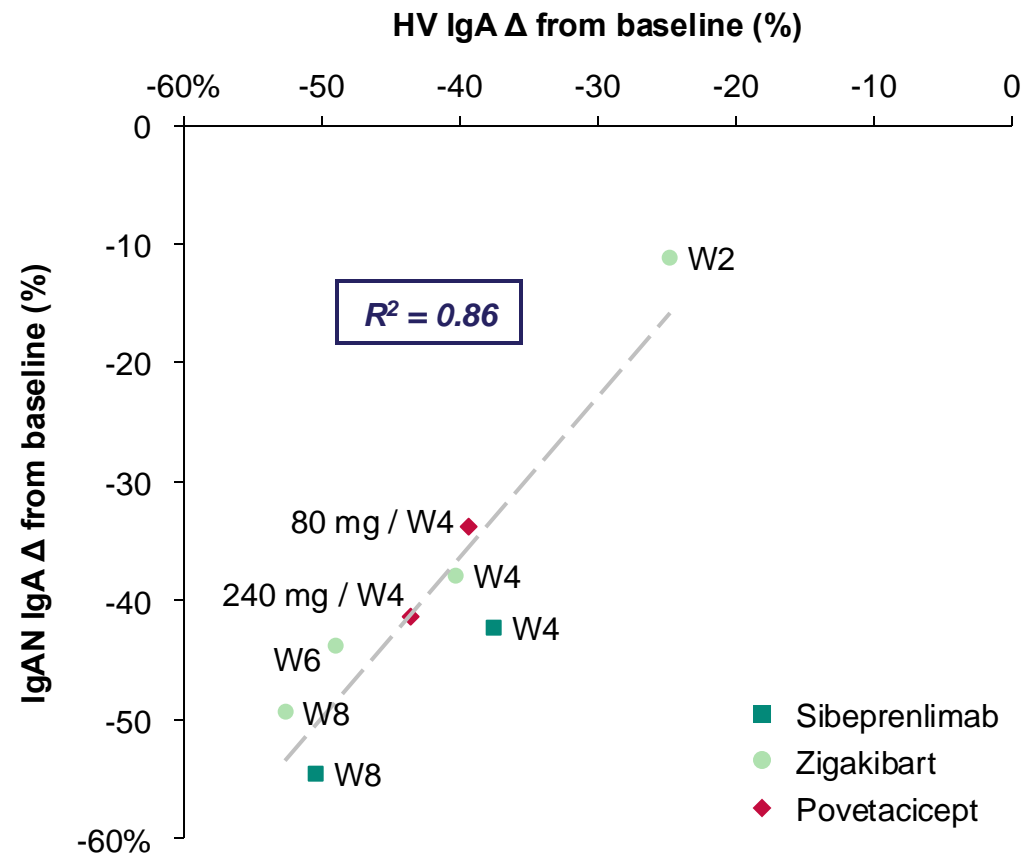
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- **NHP and Phase 1 PK/PD** could provide early signals of clinical activity; **IgA reduction** in HVs has been observed to be **highly correlated** with **clinical activity**.
- 9-month proteinuria data, which we believe is highly **predictive of kidney function preservation**, provides support for US submission for **accelerated approval and potentially offers a faster path** to market prior to eGFR confirmatory data.

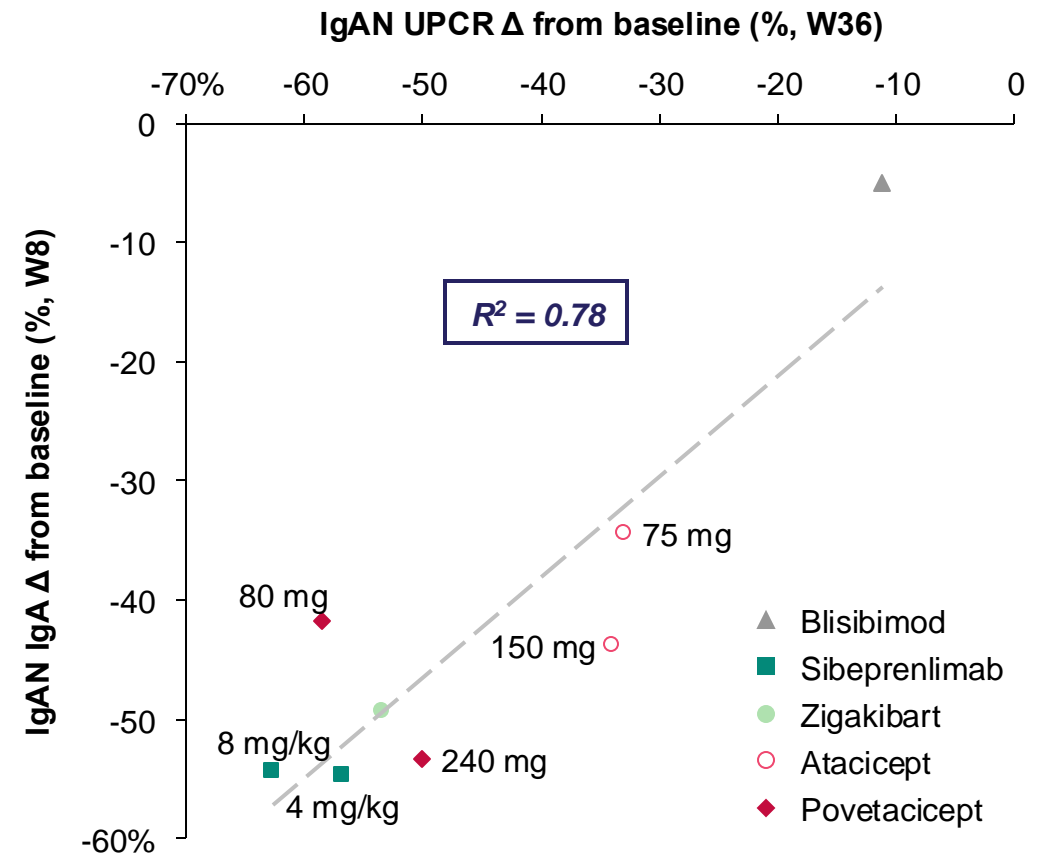
Proof-of-concept **IgA healthy volunteer data expected in 1H 2026**

# IgA reduction in healthy volunteers is the critical inflection point for clinical development in IgAN

IgA reduction in HVs has been observed to be **highly correlated** with IgA reduction in IgAN patients



...and IgA reduction was observed to correlate with W36 UPCR reduction, the **endpoint for accelerated approval**



# Potential of JADE-001 in IgAN

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**Potential Disease-modifying MoA**

Potential to deplete pathogenic IgA and avoids broad B-cell inhibition



**More convenient dosing**

Enabled by half-life extension technology



**Potential best-in-class clinical activity**

Designed for superior potency and half-life with potential to maximize clinical remission

# Pipeline opportunities beyond IgAN



# Additional Jade pipeline programs are expected to focus on best-in-class product profiles in high-value I&I indications



I&I indications with **significant market opportunity**



Potential **Best-in-class** and **best-in-indication** product profile



Potential **Rapid path** to clinical PoC



Expected minimal **competition**



Jade team **expertise**

Team is evaluating additional opportunities to **build pipeline of potentially best-in-class I&I therapies.**

# Jade Biosciences is developing transformative therapies for high-value I&I indications

- Approximately \$300 million raised to date, including anticipated proceeds from an oversubscribed pre-closing private financing, from syndicate of top tier healthcare investors, including:



MOA	Program	Discovery	IND-enabling	Planned Clinical FIH	Planned Healthy Volunteer Data
anti-APRIL	JADE-001			2H25	1H26
Undisclosed	JADE-002			1H26	
Undisclosed	JADE-003			1H27	

# Estimated capitalization following close of transactions with Aerovate and pre-closing private placement

		Shares on an as-converted basis	Expected ownership of the combined company	Estimated dividend per share
<b>Aerovate</b> <ul style="list-style-type: none"> <li>Shares of common stock outstanding</li> </ul>		28,867,711	1.6%	\$2.25*
<b>Jade Biosciences</b> <ul style="list-style-type: none"> <li>Shares of common stock outstanding (including shares underlying option grants)</li> <li>Series A shares</li> </ul>		202,760,666	98.4%	N/A
		428,776,000		
<b>Pre-closing financing</b> <ul style="list-style-type: none"> <li>Shares of common stock</li> <li>Pre-funded warrants</li> </ul>		932,531,887		
		262,898,748		
<b>Estimated total shares of common stock of the combined company post-closing**</b>		1,855,835,012		

\*Prior to closing, Aerovate expects to declare a cash dividend to pre-merger Aerovate stockholders, distributing excess net cash estimated to be approximately \$65 million.

\*\*Please refer to AVTE's SEC filings for additional information, including the Registration Statement on Form S-4 that AVTE intends to file in connection with the transaction.

# Thank you

# JADE-001 HLE strategy and profile in NHPs shows promise\*

~3X increased half-life over sibeprenlimab in NHPs...

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

