



Company Overview

April 2025

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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 aims to develop transformative therapies for high-value inflammation and immunology indications

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

- Advancing potential **best-in-class therapies for autoimmune diseases**, including IgAN.
- Fourth company launched to research and develop **antibody candidates licensed from Paragon Therapeutics**, an antibody discovery engine founded by Fairmount.
- Building on success of **Apogee, Spyre, and Oruka**, which have generated clinical data using Paragon’s engineered antibody technology and **collectively raised ~\$2B.***

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 team with backing from Paragon

Company Leadership



Tom Frohlich
CEO



Andrew King
CSO, Head of R&D



Hetal Kocinsky
CMO



Valerie Fauvelle
SVP, Regulatory & Quality



Jason Wright
SVP, Chemistry,
Manufacturing & Controls



Amy Sullivan
SVP, Development
Operations



Jonathan Quick
SVP, Finance



Elizabeth Balta
GC & Corporate
Secretary



Sandy Lewis
SVP, Biometrics &
Clinical Strategy



Lori Stewart
SVP, People

Board of Directors



Eric Dobmeier
Board Chair



Erin Lavelle
Board of Directors



Lawrence Klein
Board of Directors



Tomas Kiselak
Board of Directors



Chris Cain
Board of Directors



Tom Frohlich
Board of Directors



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+ 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 the dominant treatment for IgAN

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



Potential best-in-class profile

*JADE-001 designed for superior potency and extended half-life for **maximal efficacy & convenient dosing** for life-long therapy*



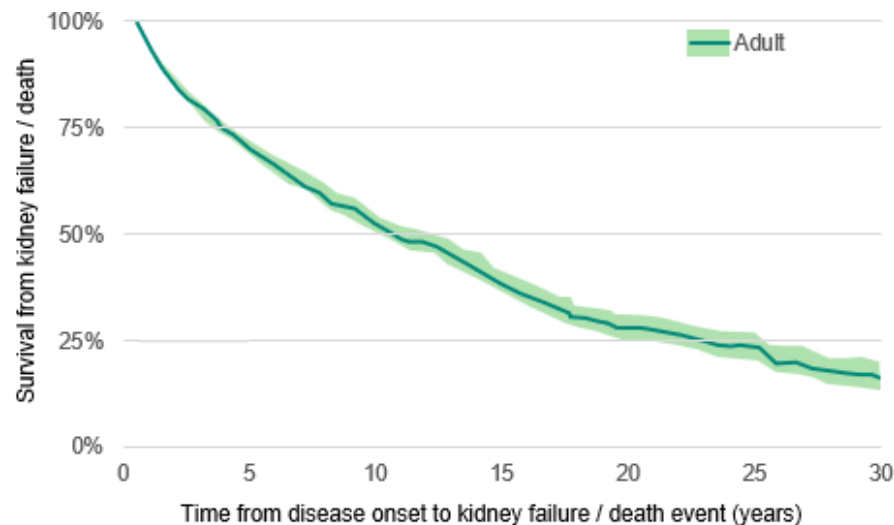
Efficient path to PoC and market

*HV IgA biomarker linked with efficacy in IgAN; **surrogate endpoints support potential IgAN approval***

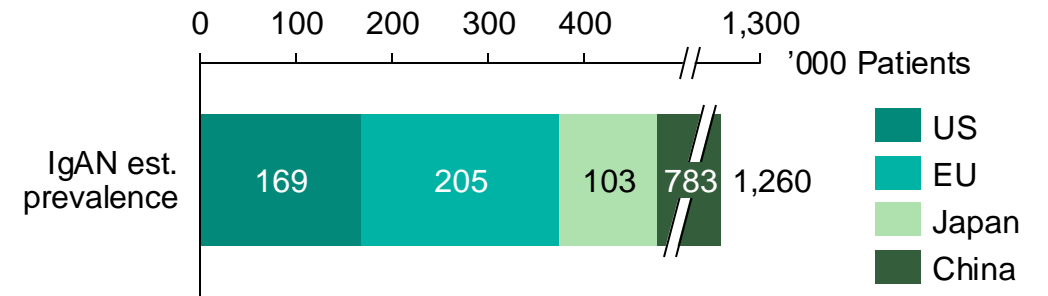
~169K+ IgAN patients in the U.S. with majority requiring treatment*, representing potential \$10B+ market

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-US 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 for life-long therapy in a young patient population.**










Notes: US prevalence estimate from FDA; EU prevalence estimate from EMA; Japan / China prevalence estimates from a Novartis presentation. Estimated pricing of ~\$120K-\$150K per year based on Filspari and Tarpeyo.

*Per KDIGO guidelines, treatment should be initiated in all cases where patients have proteinuria ≥ 0.5 g/day.

Sources: 2023 Pitcher (CJASN); FDA Reviews for Filspari / Tarpeyo; EMA; Novartis; 2018 Schena (Seminars in Nephrology); Reuters

Current IgAN treatments leave significant unmet need, with no disease-modifying, approved therapeutics

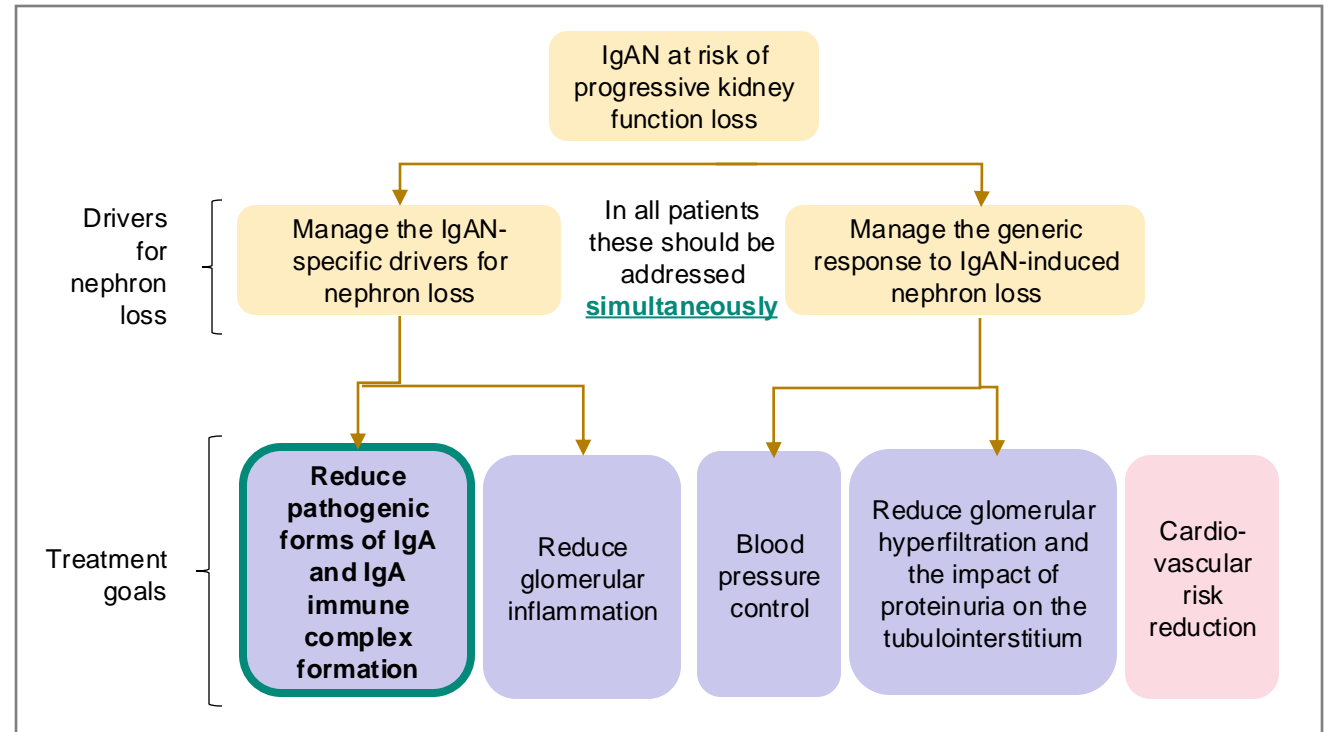
	ACEi / ARB	Systemic glucocorticoids	SGLT2i	Filspari	Tarpeyo	Fabhalta	Ideal IgAN therapy
MoA	Renin-angiotensin system inhibition	General immunosuppression	SGLT2 inhibition	Dual endothelin / angiotensin inhibition	GI-released systemic glucocorticoid	Complement Factor B inhibitor	
Status	Used off-label	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 (Filspari), 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); Filspari 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

- **Expanding Patient Population**
 - Kidney biopsy recommended in all adults with proteinuria ≥ 0.5 g/d where IgAN is a possible diagnosis
 - Recommends all patients enroll in an IgAN registry
- **Lower Proteinuria Targets**
 - Establishes new treatment goal: proteinuria maintained at < 0.5 g/day, **preferably < 0.3 g/day**
 - Recommends **additional treatment should be initiated in all cases** where patients have proteinuria ≥ 0.5 g/d
- **Redefining Treatment Goals**
 - New guidelines state clinicians should incorporate treatments that have been **proven to reduce pathogenic forms of IgA**

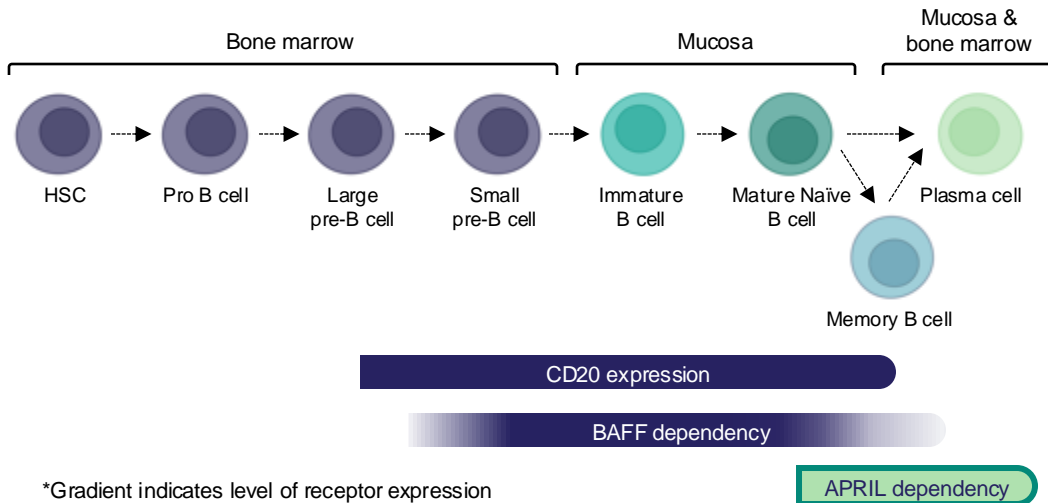


KDIGO updates anticipated to increase **IgAN diagnosis**, expand **at-risk patient population** requiring treatment, **lower proteinuria target** to clinical remission, and require **targeted therapies** that **reduce pathogenic IgA**.

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

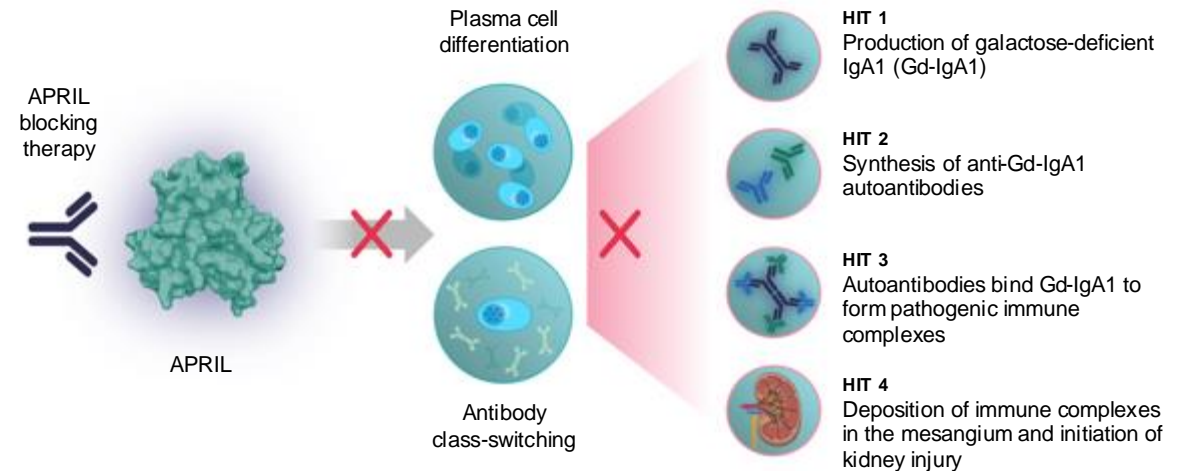
Broad B-cell depletion is ineffective in IgAN...

- 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**.



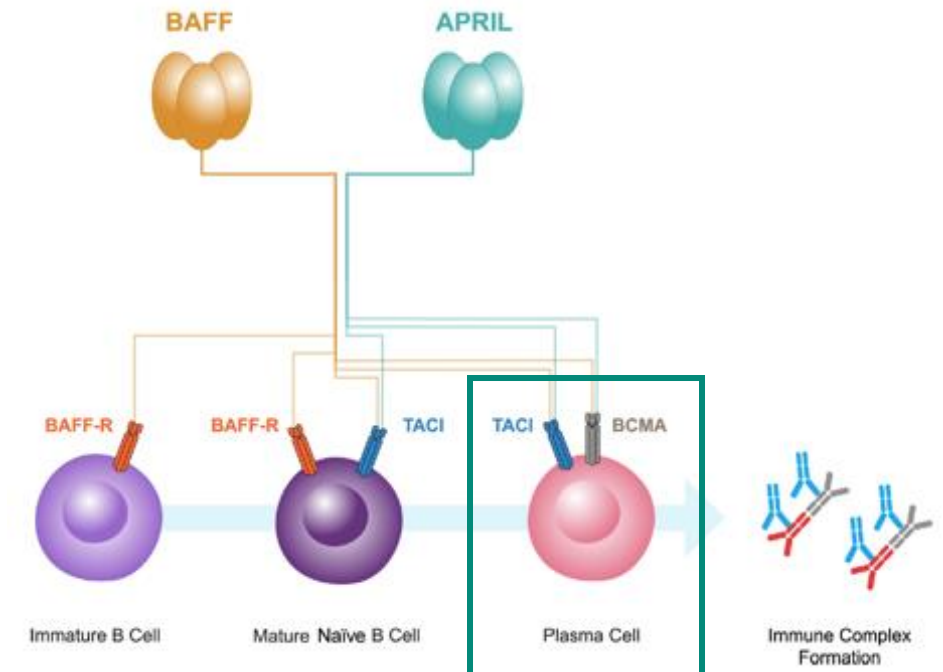
Neutralizing APRIL depletes Gd-IgA1, reduces proteinuria, and **preserves eGFR**, providing a **disease-modifying treatment** of IgAN without impacting B-cell development and maturation.

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**

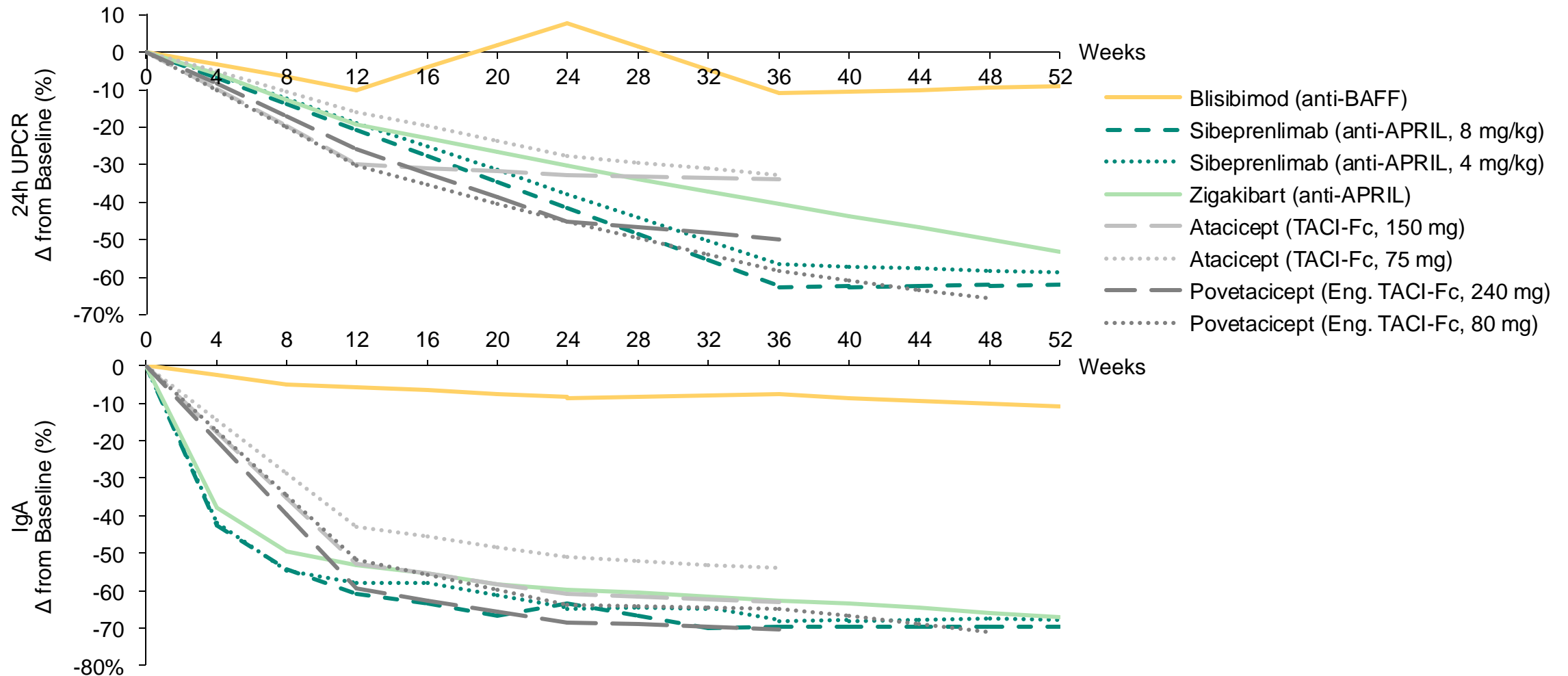
Targeting APRIL **selectively modulates plasma cells**, maintaining pool of mature B cells

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

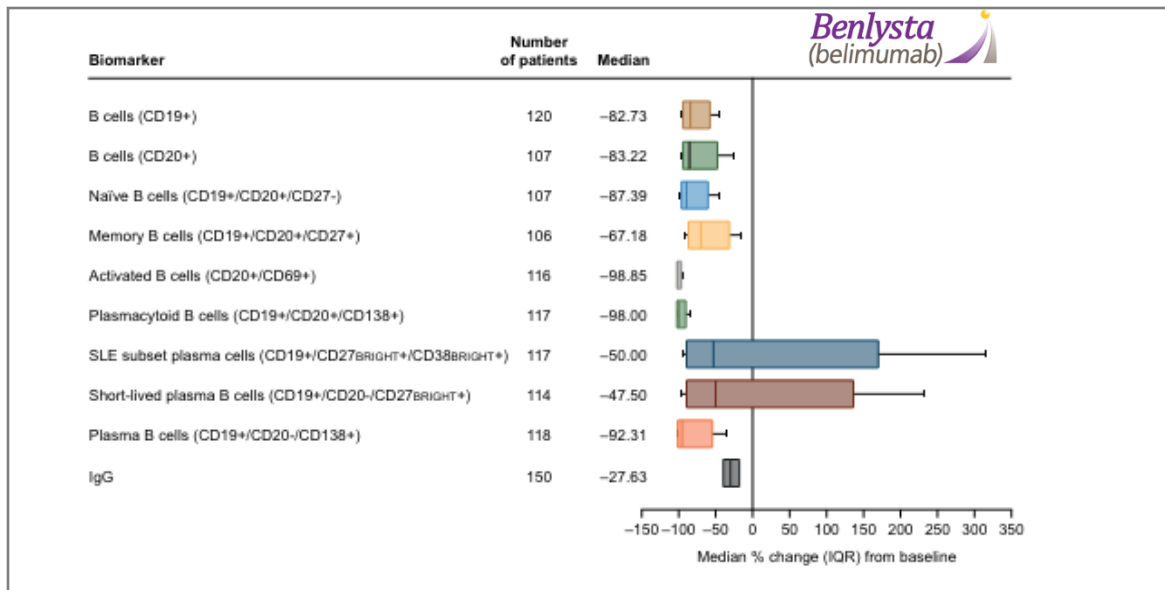
	Sibeprenlimab	Zigakibart	Atacicept	Povetacicept
MoA	anti-APRIL	anti-APRIL	TACI-Fc	Engineered TACI-Fc
Status	P3	P3	P3	P3
Δ from baseline in critical disease markers (W36 timepoint*)	IgA	IgA	IgA	IgA
	Gd-IgA1	Gd-IgA1	Gd-IgA1	Gd-IgA1
	UPCR	UPCR	UPCR	UPCR
	67%	64%	63%	65%
	60%	69%	64%	69%
	60%	53%	33%	59%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



Notes: *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%. Sibe 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; 2024 Madan (ASN Presentation)

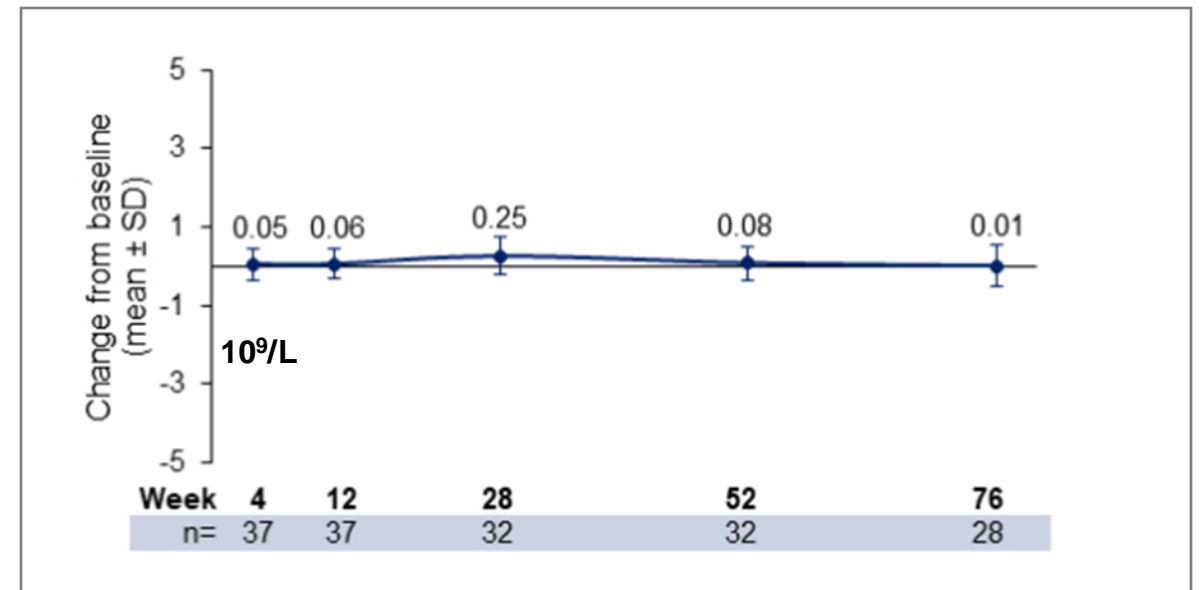
BAFF inhibition is accompanied by the potential for significant long-term B cell depletion

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.

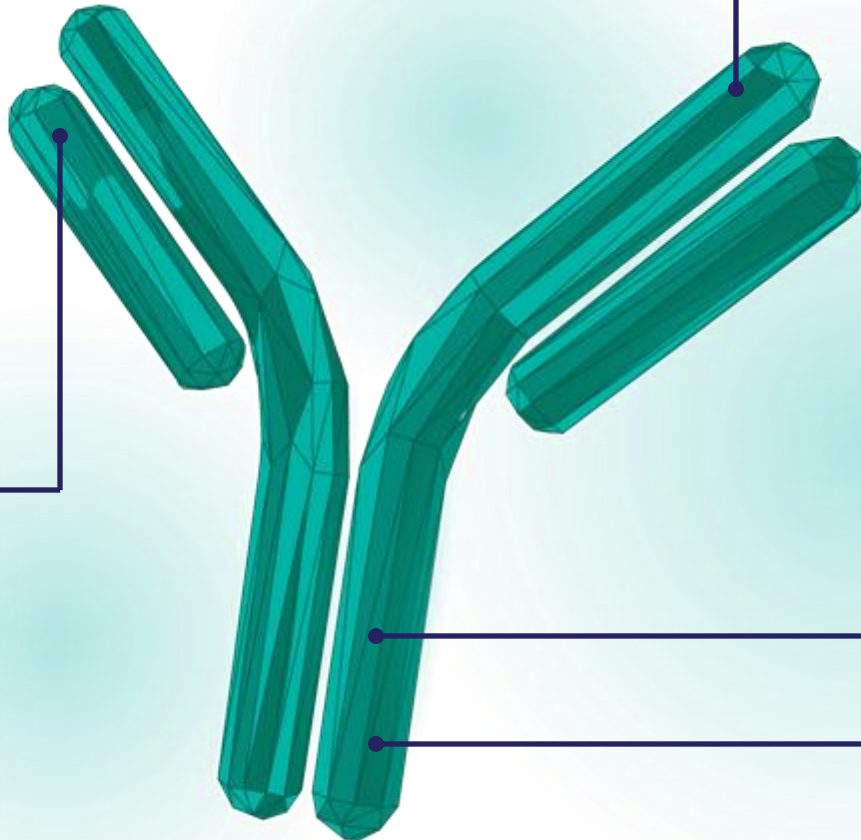
JADE-001 is a potential best-in-class anti-APRIL

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



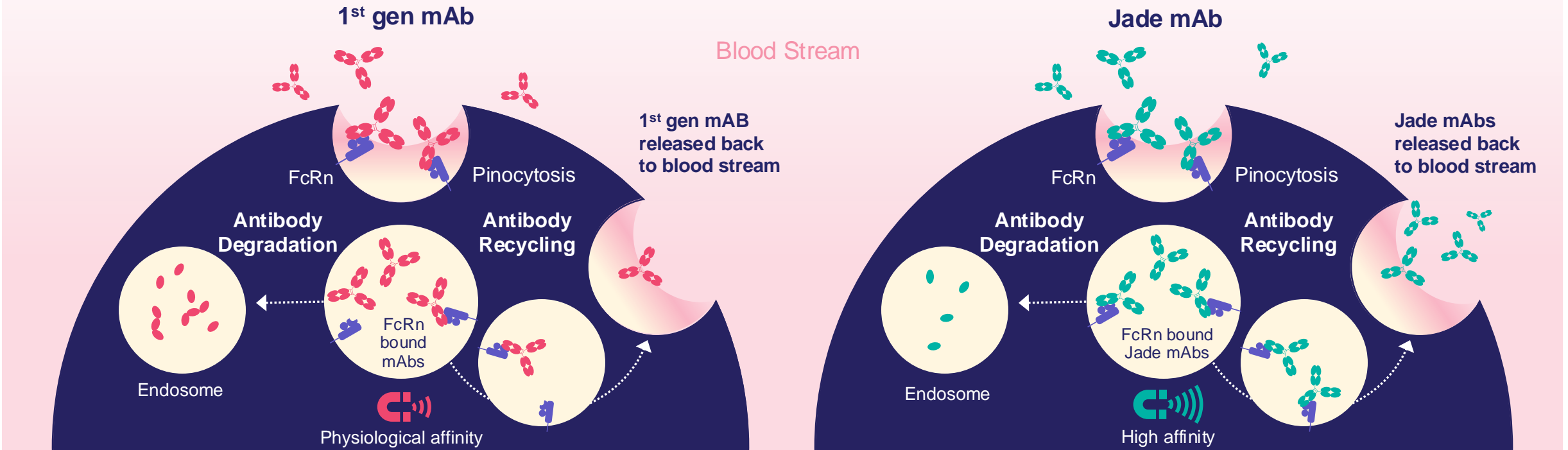
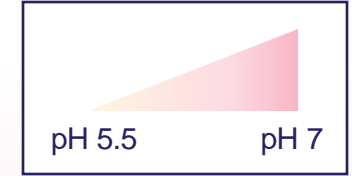
Half-life extension through validated YTE Fc modification

- Longer exposure intended to reduce dosing frequency

Effector-null human IgG1 Fc

Jade mAbs employ proven half-life extension technology

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



Goal of JADE-001 is to introduce Q8W+ dosing for patients with IgAN via well-established half-life extension technology

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

- **High potency** can potentially further drive lower dosing frequency
- Already 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
JADE-001 TPP (HLE anti-APRIL mAb)	HV PK expected H1 2026 50+*	Targeting Q8W+
Sibeprenlimab (anti-APRIL mAb)	~23**	Q4W (400 mg)
Zigakibart (anti-APRIL mAb)	~20**	Q2W (600 mg)
Atacicept (TACI-Fc APRIL/BAFF)	6.7	QW (150 mg)
Povetacicept (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

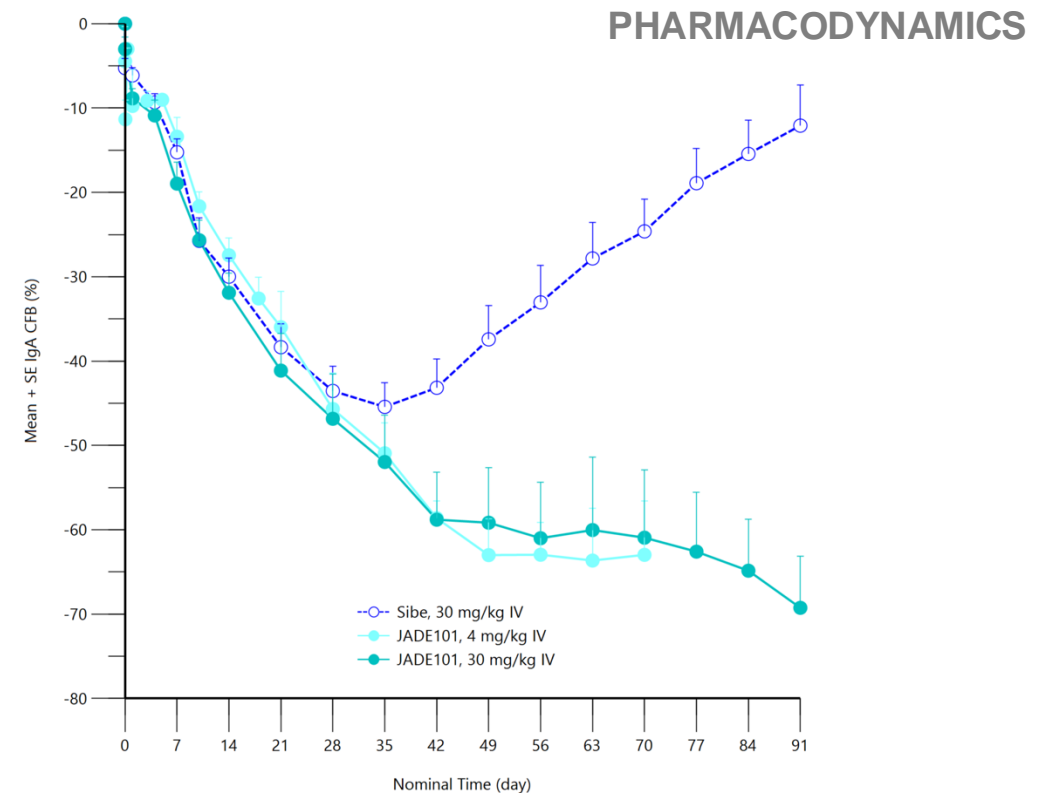
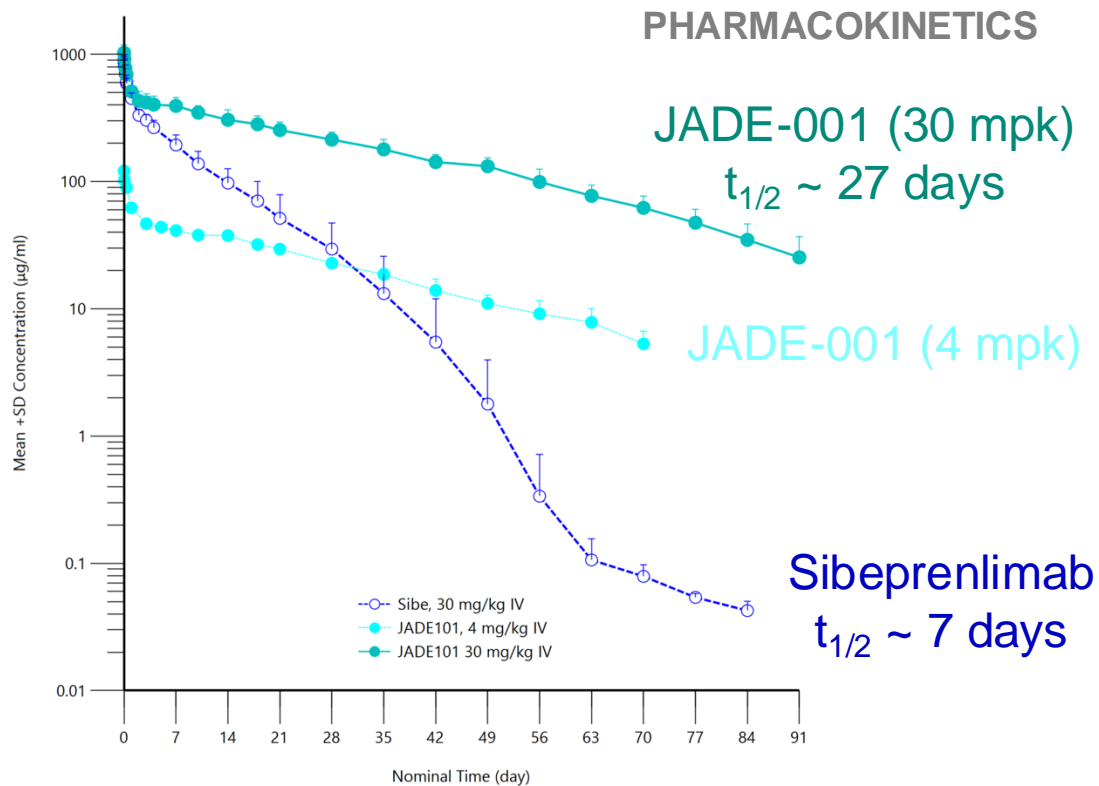
*Based on single dose studies in NHPs dosed with JADE-001 initial clone. A development candidate has been selected from a pool of profiled clones. We have entered into an exclusive JADE-001 license agreement with Paragon.

**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 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



Deeper APRIL suppression drives superior clinical efficacy

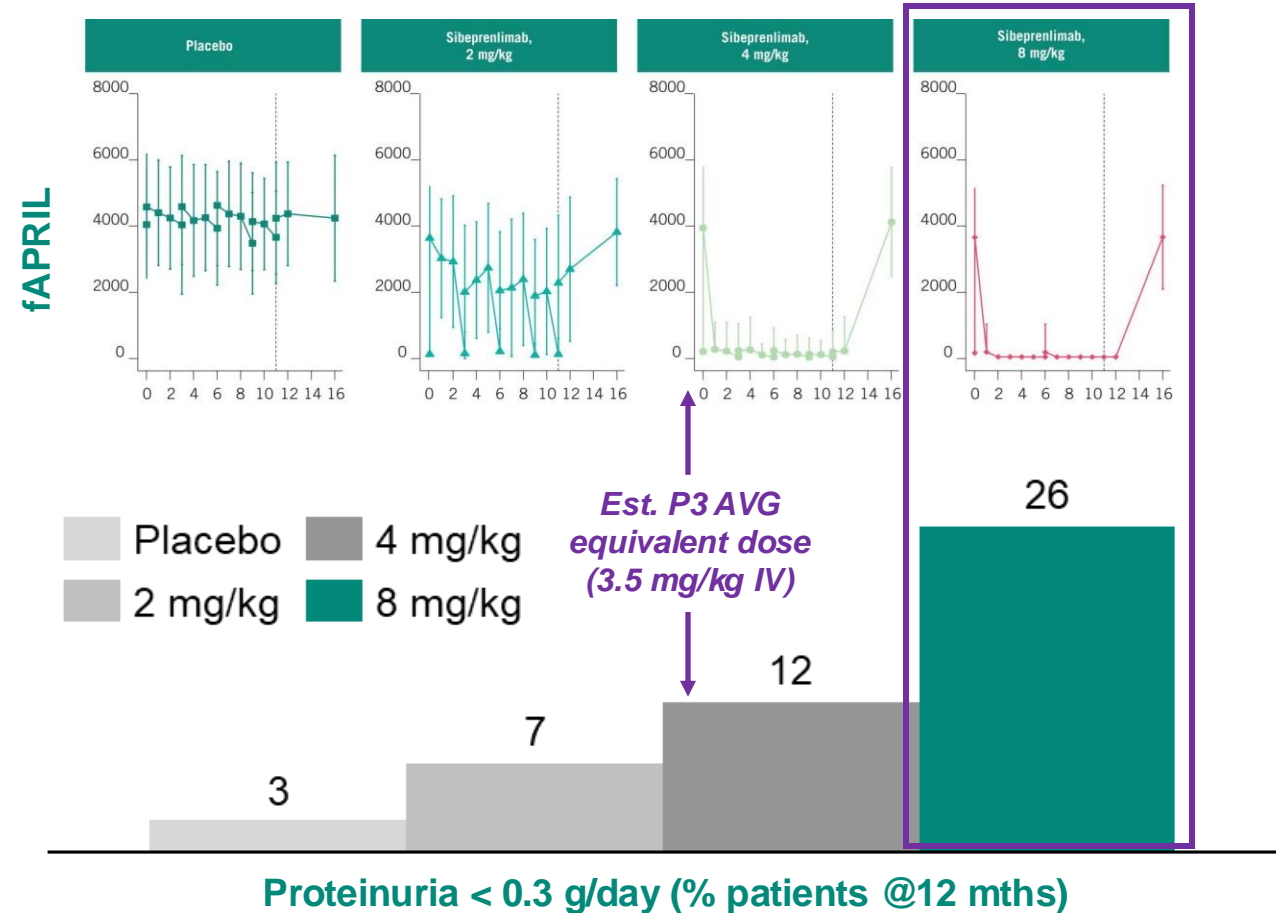
- Highest rates of **clinical remission** (proteinuria <0.3 g/day) for sibeprenlimab were accompanied by the **deepest levels of APRIL suppression**.
- **Safety profile consistent** across dose levels.
- Potential for anti-APRILs with **higher affinity** and **increased systemic exposure** to **provide 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.



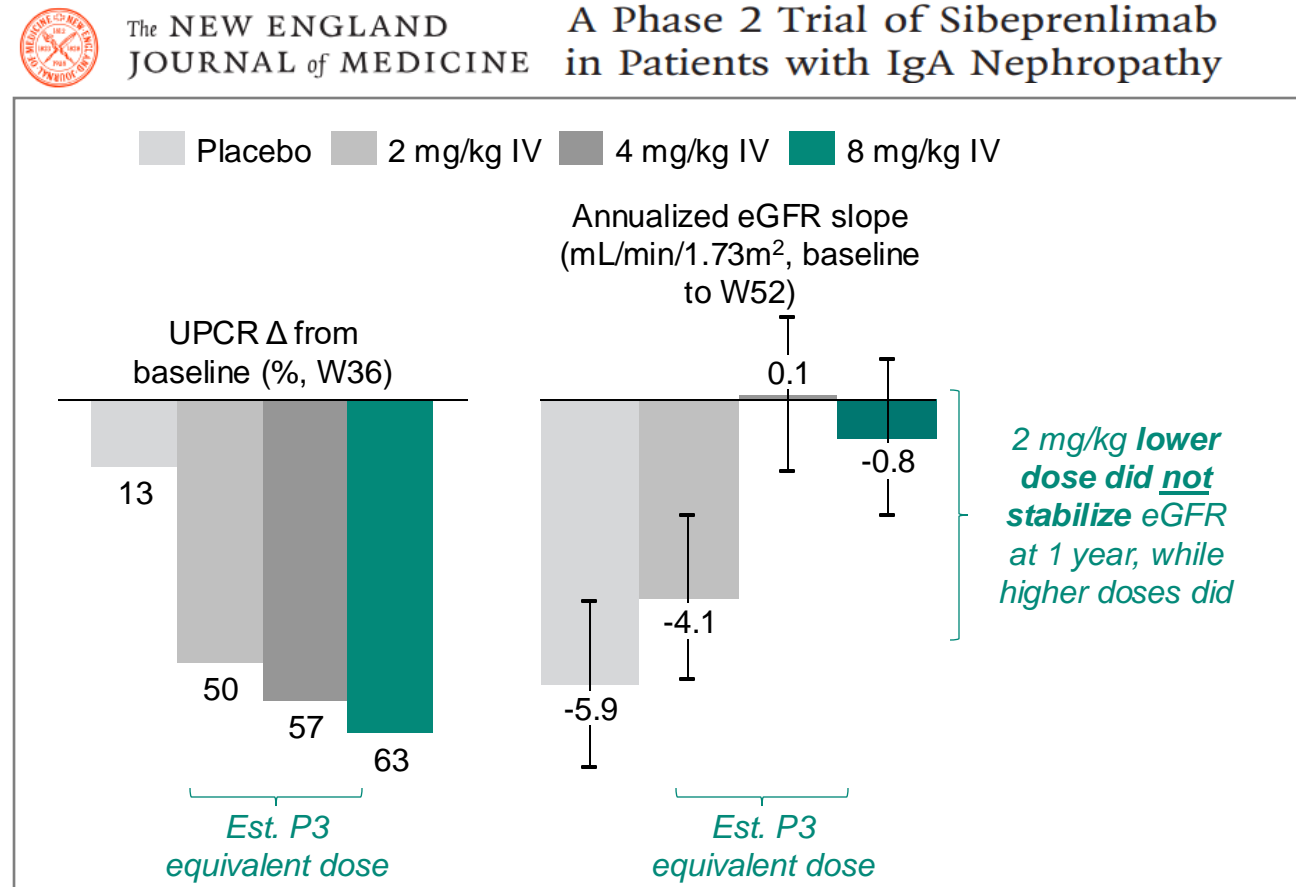
The NEW ENGLAND JOURNAL of MEDICINE

A Phase 2 Trial of Sibeprenlimab in Patients with IgA Nephropathy



Sibeprenlimab is potentially under-dosed in ongoing Phase 3 trial

- **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 endpoints in Phase 2 ENVISION** trial (as seen on right).
- **~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 potential best-in-class clinical activity for patients.

Potential path to early clinical proof-of-concept and accelerated approval in the US

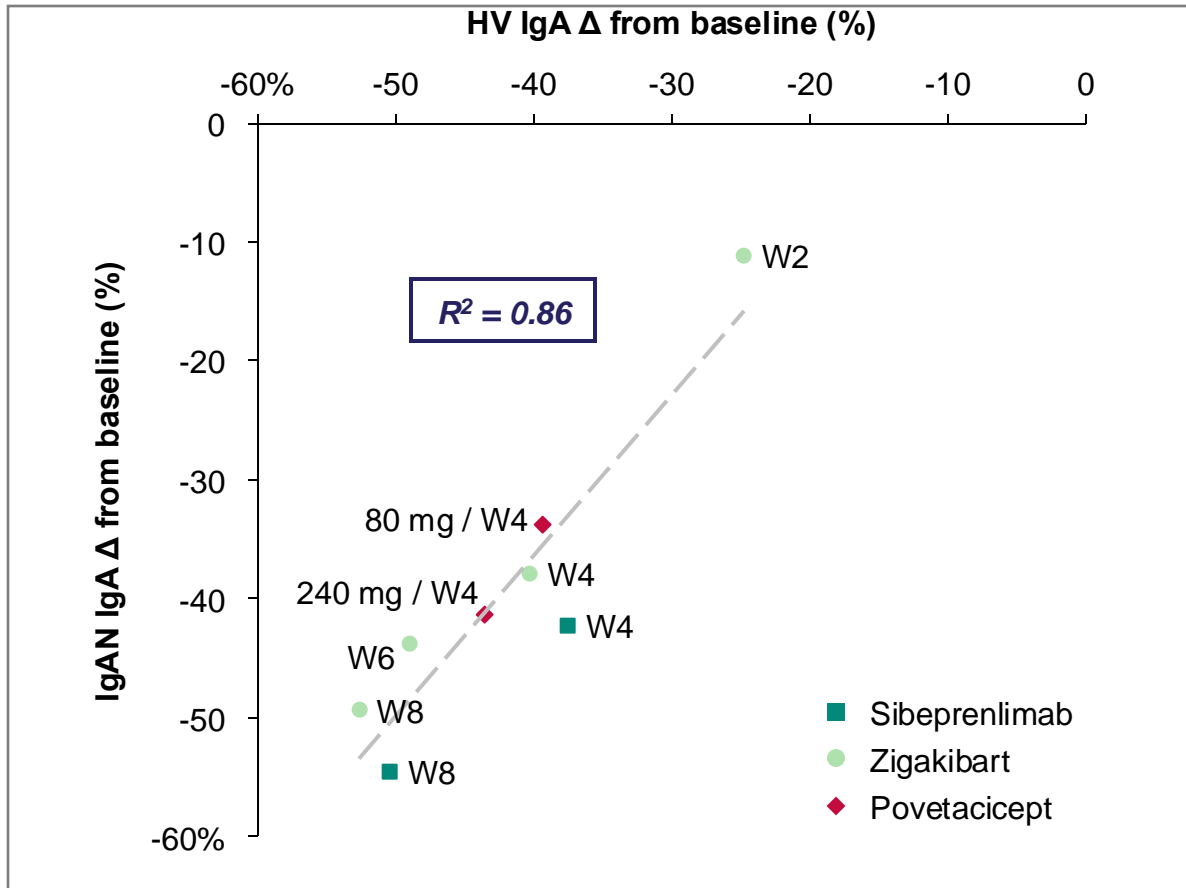
MOA	Program	Discovery	Phase 1 Initiation	Potential Healthy Volunteer Data	Potential Indications
anti-APRIL	JADE-001	Ongoing	2H 2025	1H 2026	IgAN

- **NHP and Phase 1 PK/PD** could provide early signals of clinical activity; **IgA reduction** in health volunteers has been observed to be **highly correlated** with **clinical activity**.
- 9-month proteinuria data **predictive of kidney function preservation**, supports potential for **accelerated approval and faster path** to market prior to eGFR confirmatory data.

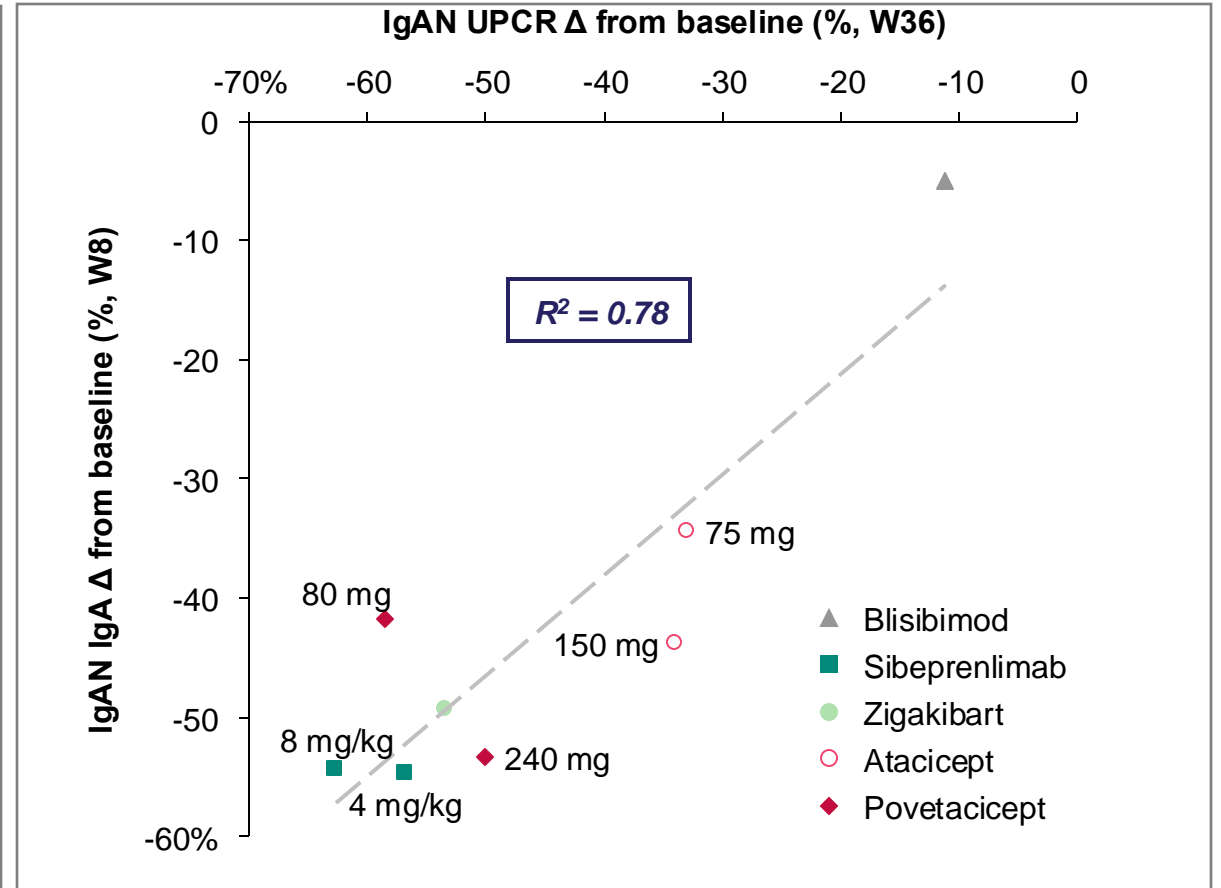
Proof-of-concept **IgA healthy volunteer data** expected in 1H 2026

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**



Pipeline opportunities beyond IgAN

Additional Jade programs expected to focus on best-in-class product profiles in high-value inflammation and immunology 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**

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

Jade aims to develop transformative therapies for high-value inflammation and immunology indications

Jade is well capitalized to advance programs with ~\$300M* of committed funding from a syndicate of top healthcare investors



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 of Jade following pre-closing private placement and subsequent closing of merger with Aerovate

		Shares on an as-converted basis	Expected ownership of the combined company	Estimated per share dividend based on \$69.6m dividend
Aerovate <ul style="list-style-type: none"> Shares of common stock outstanding 		28,985,019*	1.4%	\$2.40**
Jade Biosciences <ul style="list-style-type: none"> Shares of common stock outstanding (including shares underlying option grants) Series A shares 		407,057,032	98.6%	N/A
		441,688,000		
Pre-closing financing <ul style="list-style-type: none"> Shares of common stock Pre-funded warrants 		967,999,030		
		274,315,274		
Estimated total as-converted shares of common stock of the combined company post-closing***		2,120,044,355		

*Including in the money options, this number equals 29,743,481.

**Aerovate's Board has declared a special cash dividend in the aggregate amount of \$69.6 million payable in cash to Aerovate stockholders of record as of April 25, 2025 and scheduled to be paid on April 29, 2025.

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

Note: The estimates reflected on this slide are based on the best available information as of April 9, 2025 and are subject to change as the transaction advances toward closing.



Jade
BIOSCIENCES

www.JadeBiosciences.com

info@jadebiosciences.com