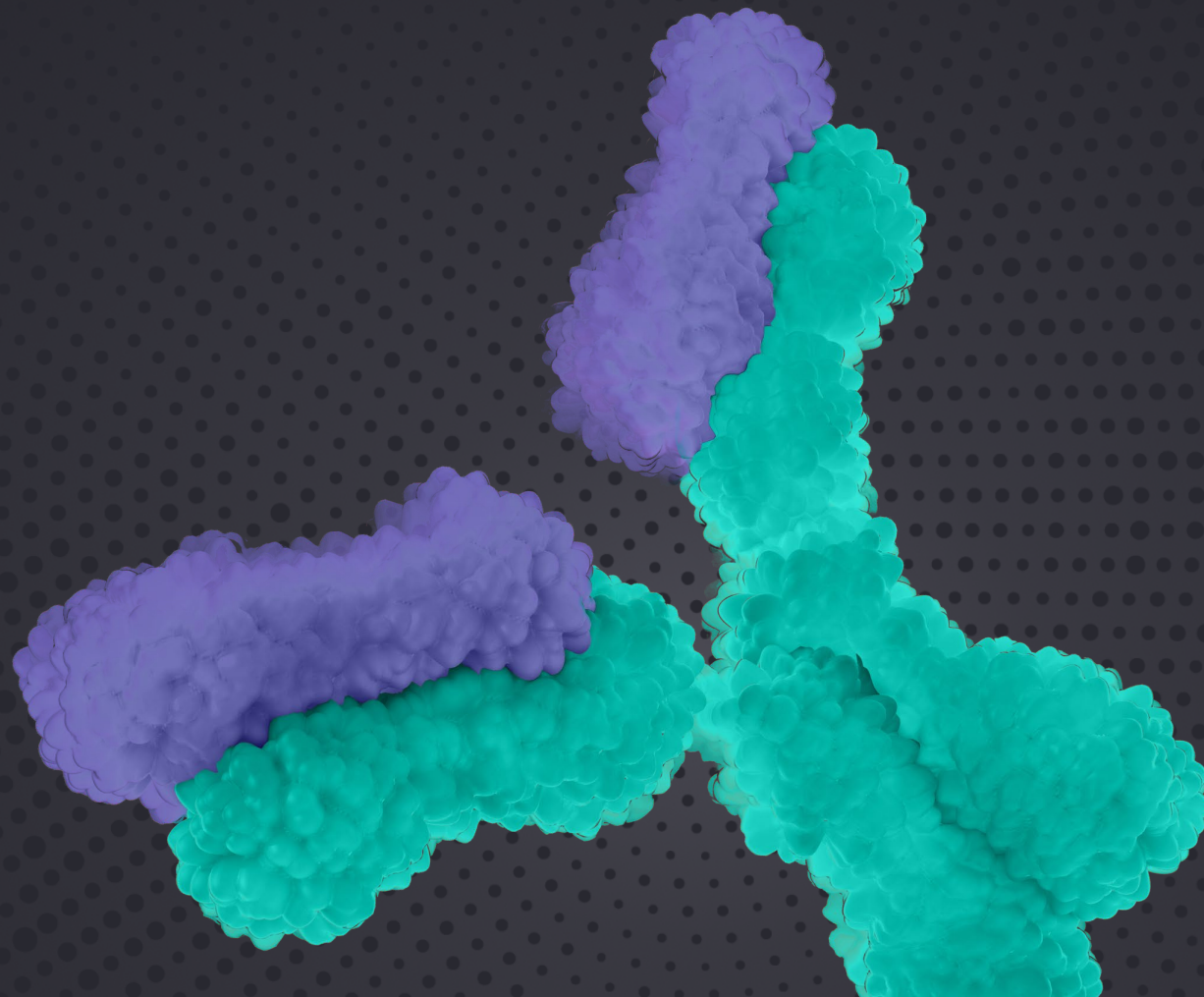


September 2026

Company Overview

NASDAQ: JBIO



Disclaimers

FORWARD LOOKING STATEMENTS

Certain statements contained in or made orally during this presentation, other than purely historical information, may constitute “forward-looking statements” within the meaning of the federal securities laws, including for purposes of the “safe harbor” provisions under the Private Securities Litigation Reform Act of 1995. These forward-looking statements include, but are not limited to, express or implied statements relating to the expectations, hopes, beliefs, intentions or strategies of Jade Biosciences, Inc. (“Jade”, the “Company”, “we”, “our” or “us”) regarding the future of its pipeline and business including, without limitation: Jade's cash runway; Jade's ability to achieve the expected benefits or opportunities with respect to JADE101, JADE201 and JADE301, including their best-in-class and best-in-disease potential; the expected timelines for the initiation of the Phase 3 clinical trial of JADE101 and the Phase 1 clinical trial of JADE301; the expected timelines for the availability of interim data from the Phase 2 clinical trial of JADE101 and the Phase 1 clinical trials of JADE201 and JADE301; the expected enrollment of the Phase 2 clinical trial of JADE101; Jade's plans to conduct a Phase 3 clinical trial of JADE101, the design and timing thereof and Jade's expectations that such trial will serve as a registrational study; Jade's proposed dosing strategy for JADE101 and its expected optimization of clinical activity and convenience; projected or simulated pharmacodynamic outcomes, including steady-state IgA reductions; the potential for the anti-APRIL class to become the foundational therapy for IgAN; the potential for JADE101 healthy volunteer data to be predictive of clinical activity; the potential of Jade's product candidates to enable clinical remission and their potential therapeutic uses, market opportunities, mechanisms of action, efficacy, durability, safety profiles, and dosing. The words “opportunity,” “potential,” “milestones,” “pipeline,” “can,” “goal,” “strategy,” “target,” “anticipate,” “achieve,” “believe,” “contemplate,” “continue,” “could,” “estimate,” “expect,” “intends,” “may,” “plan,” “possible,” “project,” “should,” “will,” “would” and similar expressions (including the negatives of these terms or variations of them) may identify forward-looking statements, but the absence of these words does not mean that a statement is not forward-looking. These forward-looking statements are based on current expectations and beliefs concerning future developments and their potential effects. There can be no assurance that future developments affecting Jade will be those that have been anticipated. These forward-looking statements involve a number of risks, uncertainties (some of which are beyond Jade's control) or other assumptions that may cause actual results or performance to be materially different from those expressed or implied by these forward-looking statements. These risks and uncertainties include, but are not limited to: interim results of a clinical trial are not necessarily indicative of final results and one or more of the outcomes may materially change following more comprehensive reviews of the data, as follow-up on the outcome of any particular participant continues and as more participant or final data becomes available; modeled and predicted data may not be realized in actual clinical studies and may not accurately represent performance of third party agents; the ongoing and planned clinical trials of JADE101, JADE201 and JADE301 and any other clinical trials may be delayed or may not demonstrate desirable efficacy or predicted performance; Jade's planned JADE101 Phase 3 clinical trial may be delayed based on FDA feedback or requirements, as the FDA retains broad discretion to require additional clinical data for any product candidate prior to the conduct of a Phase 3 clinical trial or submission for regulatory approval; even if such Phase 3 trial is successful, it may not support regulatory approval; adverse events and safety signals may occur; Jade may experience unanticipated costs, difficulties or delays in the product development process; Jade's product candidates may be delayed to a point where they are not commercially viable; clinical trial start up, enrollment or regulatory challenges may occur; challenges associated with Jade's dependence on third-party vendors for the development, manufacture and supply of its product candidates may occur; Jade may use its capital resources sooner than expected; and the other risks, uncertainties and factors more fully described in Jade's most recent filings with the Securities and Exchange Commission (including the Quarterly Report on Form 10-Q for the quarter ended June 30, 2026). Should one or more of these risks or uncertainties materialize, or should any of Jade's assumptions prove incorrect, actual results may vary in material respects from those projected in these forward-looking statements. You should not place undue reliance on forward-looking statements in this communication, which speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. Jade does not undertake or accept any duty to release publicly any updates or revisions to any forward-looking statements. This communication does not purport to summarize all of the conditions, risks and other attributes of an investment in Jade.

MARKET AND INDUSTRY DATA AND CROSS-STUDY COMPARISONS

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. This presentation contains comparisons of data across separate, non-head-to-head studies. No head-to-head clinical trial has been conducted comparing JADE101, JADE201 or JADE301 to other candidates or approved agents. Differences may exist between study designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable. In addition, data from third party products have been extracted via digitization and represent approximate values.

Advancing **potentially best-in-class therapies** for autoimmune diseases

Well-funded through multiple upcoming milestones; runway into the fourth quarter of 2028

Candidates designed to **maximize clinical activity** and allow **patient friendly, infrequent dosing**

PROGRAM	MOA	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	POTENTIAL INDICATIONS
JADE101	anti-APRIL					IgAN
JADE201	anti-BAFF-R					Multiple systemic AI diseases
JADE301	anti-IFN- β					Dermatomyositis

Development candidates from Paragon

Expected Milestones:

JADE101

- Interim Phase 2 Data: 2027
- Phase 3 Initiation: 1H 2027

JADE201

- Interim Phase 1 Data: 2027

JADE301

- Phase 1 Initiation: 4Q 2026
- Interim Phase 1 Data: 2H 2027

Notes: Jade has entered into exclusive license agreements with Paragon Therapeutics for JADE101, JADE201 and JADE301.

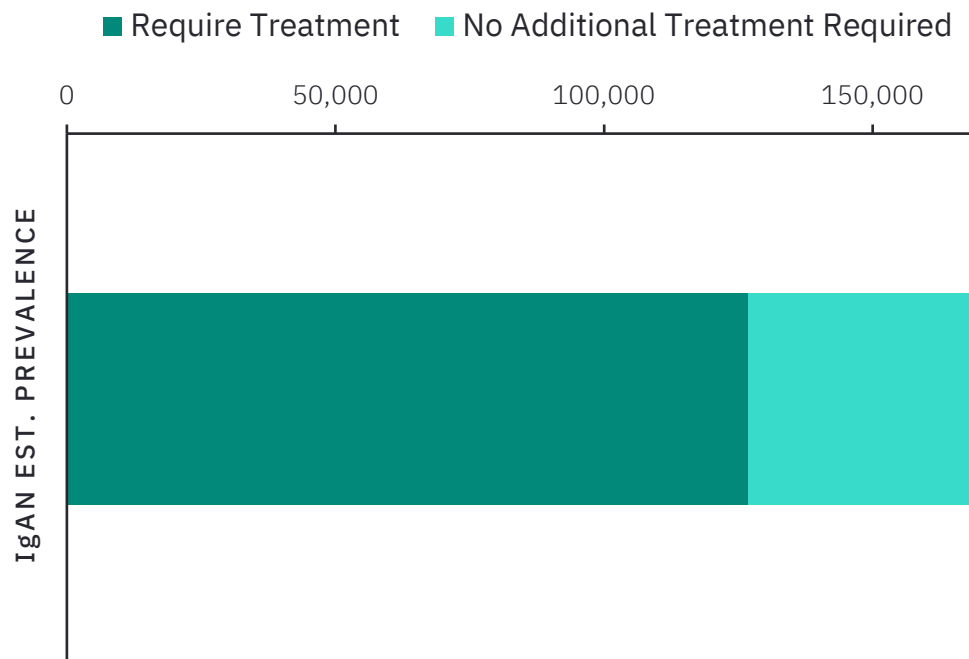
AI – Autoimmune; IgAN - IgA nephropathy; BAFF-R – B cell-Activating Factor Receptor; IFN-B – Interferon Beta; MoA – Mechanism of Action

JADE101

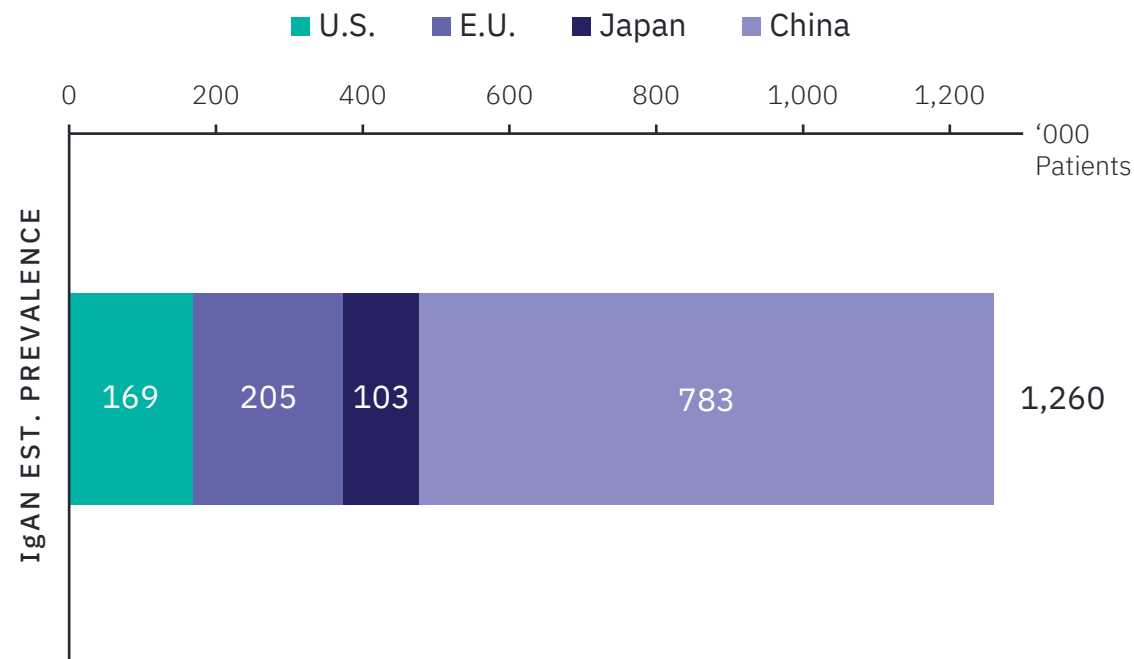
A potentially best-in-class anti-
APRIL mAb for IgAN

IgAN is a **\$20B+** potential market in the U.S. alone

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



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



Notes: Per KDIGO guidelines, treatment should be initiated in all cases where patients have proteinuria ≥ 0.5 g/day. U.S. prevalence estimate from Food and Drug Administration (FDA); EU prevalence estimate from European Medicines Agency (EMA); Japan / China prevalence estimates from a Novartis presentation. Estimated pricing of ~\$360K-\$390K per year based on

Voyxact.

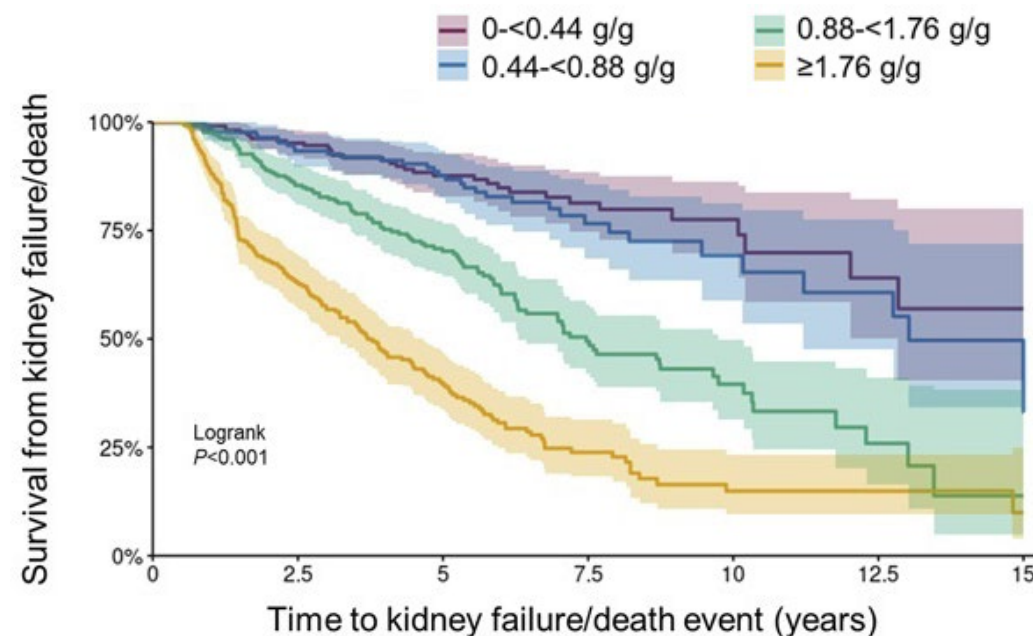
Sources: 2023 Pitcher (CJASN); FDA Reviews for Filispari / Tarpeyo; EMA; Novartis data publicly disclosed; 2018 Schena (Seminars in Nephrology); Reuters
U.S. – United States; E.U. – European Union

A need for **effective and convenient therapies** for lifelong treatment

IgAN is a **progressive autoimmune kidney disease** requiring **lifelong treatment**, with significant need for **well-tolerated, disease-modifying therapies** that offer **convenient dosing**.

- IgAN is typically diagnosed in young adults between the ages of 16 and 35; **higher proteinuria** is associated with **greater risk of kidney failure**
 - Lifetime risk of progression to end-stage kidney disease begins at low proteinuria thresholds.

High lifetime risk of end-stage kidney disease



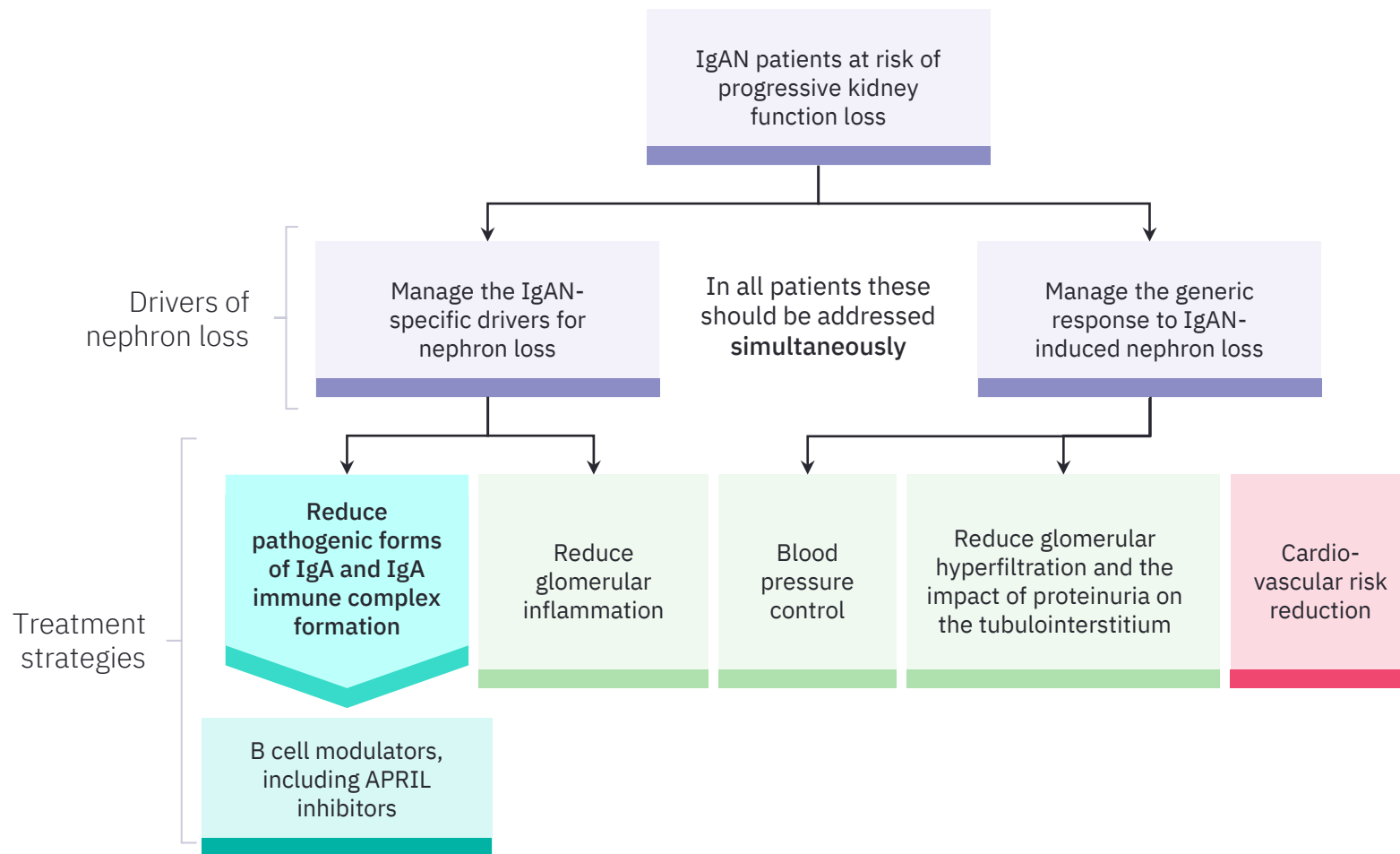
Notes: Per KDIGO guidelines, treatment should be initiated in all cases where patients have proteinuria ≥ 0.5 g/day.

Sources: 2023 Pitcher (CJASN); 2018 Schena (Seminars in Nephrology); Reuters; American Kidney Fund (Accessed May 2026)

Updated KDIGO guidelines position the anti-APRIL class as the **foundational therapy in IgAN**

KDIGO updates:

- Expected to increase IgAN diagnosis
- Expand at-risk patient population requiring treatment
- Lower proteinuria treatment target to <0.5 g/day, preferably <0.3 g/day
- Require targeted therapies that reduce pathogenic IgA



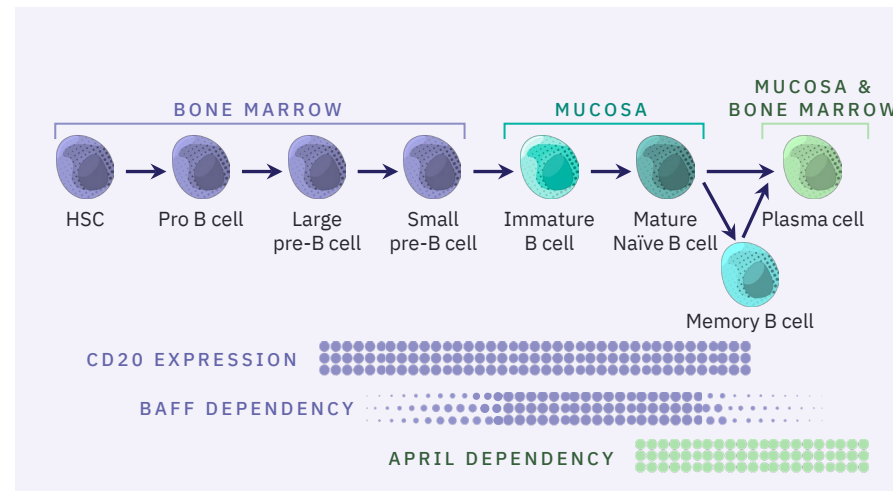
Reducing pathogenic IgA production by plasma cells

A potentially disease-modifying approach for IgAN

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

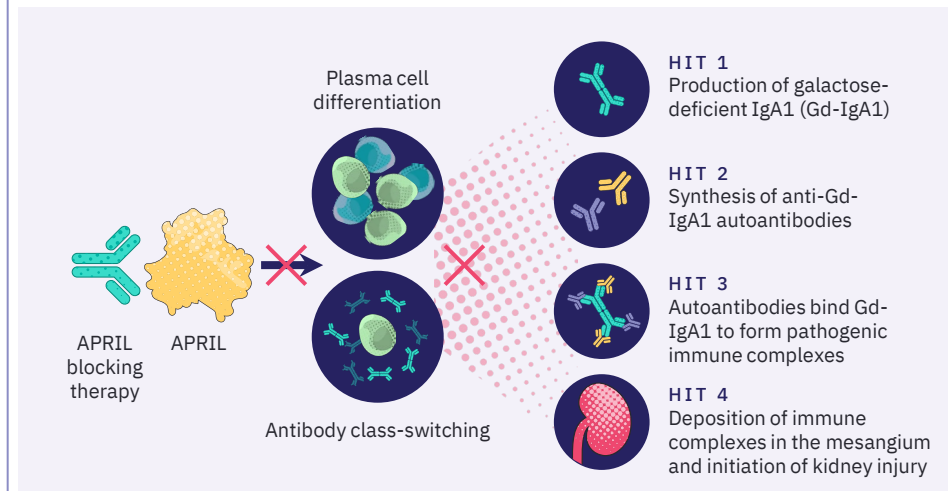
B cell depletion or reduction is ineffective in IgAN...

- B cell depletion with rituximab (anti-CD20) failed to reduce Gd-IgA1, anti-Gd-IgA1 autoantibody, or proteinuria and did not impact eGFR
- B cell suppression with 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



Phase 3 IgAN data have **not demonstrated additional patient benefit** from dual APRIL/BAFF vs selective APRIL inhibition

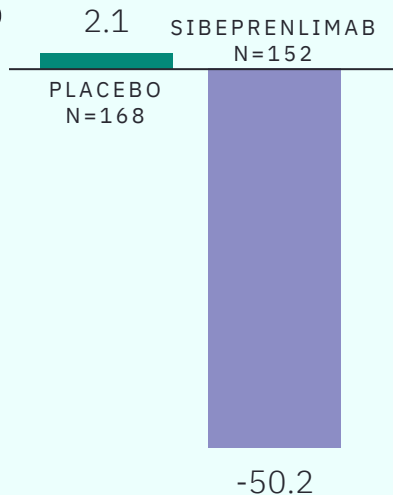
Study populations were representative of high-risk, global IgAN patients

Sibeprenlimab

UPCR Δ from baseline (% , 9 months)

Δ -51.2%

(p < 0.0001)



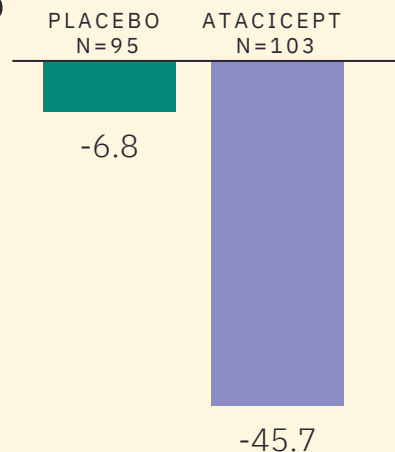
SELECTIVE ANTI-APRIL

Atacicept

UPCR Δ from baseline (% , 9 months)

Δ -41.8%

(p < 0.0001)



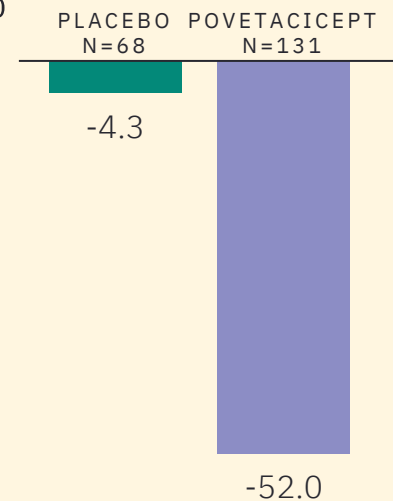
DUAL APRIL/BAFF

Povetacicept

UPCR Δ from baseline (% , 9 months)

Δ -49.8%

(p < 0.0001)



Notes: Information provided above is for illustrative purposes only and no head-to-head clinical trials have been conducted. Differences exist between study or trial designs and subject characteristics, and caution should be exercised when comparing data across trials. Data digitized from graphs where publications did not provide specific values.

Sources: Perkovic et al. ERA 2025 (sibeprenlimab), ORIGIN Phase 3 clinical trial (atacicept, NCT04716231), RAINIER Phase 3 (povetacicept, NCT06564142).
UPCR – Urine Protein-to-Creatinine Ratio; Δ – Delta

Potentially **best-in-class** properties of JADE101

Fully-human, ultra-high binding affinity, and extended half-life

Novel epitope discovered through *de novo* campaign to achieve first fully-human, potentially best-in-class anti-APRIL mAb

Half-life extension through validated YTE Fc modification

- Longer exposure intended to maximize clinical activity and reduce dosing interval to no more frequently than every 8 weeks

Ultra-high (fM) APRIL binding affinity

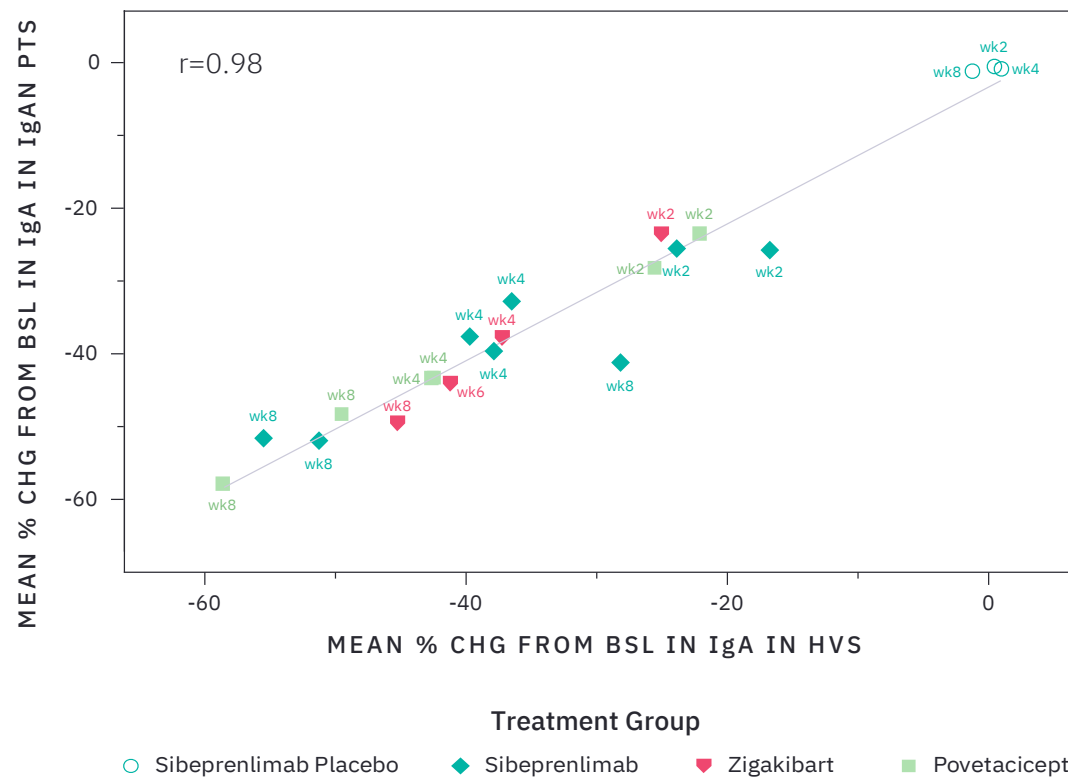
- Binds **APRIL** to neutralize activity
- **Selective to APRIL** to avoid unnecessary immune suppression
- **Greater APRIL binding affinity** than sibeprenlimab (~750x), zigakibart (~2,000x), and povetacicept (~20x)

Notes: Jade and its licensor, Paragon, have filed patent applications covering the subject matter of JADE101. No head-to-head clinical trials have been conducted between JADE101 and the referenced drug candidates. Cross-trial comparisons are inherently limited and presented for hypothesis-generating purposes only.

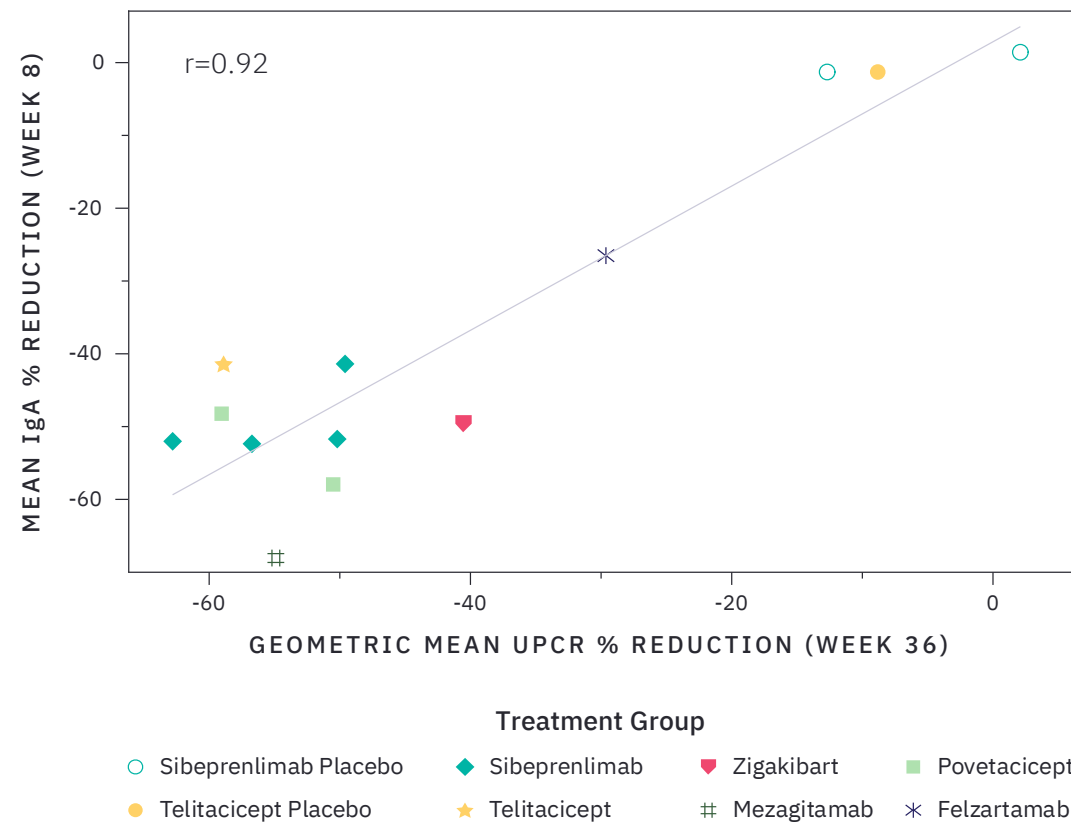
Sources: Internal SPR data; Evans, 2023 (Povetacicept); Benchmarks manufactured based on publicly available sequences.
Fc – Fragment crystallizable receptor; fM – Femtomolar; mAb – Monoclonal Antibody

IgA responses are consistent between HVs and IgAN patients and predictive of clinical activity

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



...and early IgA reduction further correlates with W36 UPCR reduction, in IgAN patients



Notes: Sibeprenlimab Phase 2 (intravenous, IV) and Phase 3 (subcutaneous) IgAN data are included. Phase 2 data are averages of the 4 mg/kg and 8 mg/kg cohorts (HV IV data is from the 6 mg/kg group); the two cohorts saw effectively equivalent IgA reduction at weeks 2, 4, and 8. Zigakibart UPCR data is at W52. Datapoints extracted via digitization and represent approximate values. The company does not possess the underlying raw third-party data. Trend lines are best linear fit. These data are derived from different trials at

different points in time, with differences in methodology, design and populations. Caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.

Sources: 2025 Gufford (ASN Presentation); Voyxact 2025

HV – Healthy Volunteer; W – Week; BSL – Baseline; CHG – Change; PTS – Patients

JADE101

Interim Phase 1 Data

JADE101 Phase 1 study met or exceeded objectives

Clinical data support potential for every-12-week subcutaneous dosing with best-in-class, disease-modifying potential

ENDPOINT	JADE101 OBJECTIVE	JADE101 DATA
Dosing	Single SC maintenance dose Q8W	✓ Potential for single SC maintenance dose Q12W
Safety	Favorable safety profile consistent with selective anti-APRIL mechanism	✓ Favorable safety profile; well-tolerated at all tested doses
Pharmacodynamics	≥55% IgA reduction sustained for ≥8 weeks - Rapid, complete and sustained suppression of fAPRIL	✓ ~70% IgA reduction sustained at 12 weeks at 700 mg; modeled to be maintained with single SC 350 mg Q12W maintenance dose ✓ IgA-lowering potency ~379-fold higher than sibeprenlimab; ~26-fold higher than povetacept ✓ Rapid, complete and sustained suppression of fAPRIL
Pharmacokinetics	- Extended half-life - TMDD mitigation - Minimal ADA impact on exposure	✓ ~8.7-fold longer half-life than povetacept; ~2.6-fold longer than sibeprenlimab ✓ TMDD threshold estimated ~2.5-fold lower than sibeprenlimab ✓ No apparent impact of ADA observed on PK or PD

Notes and Sources: Data cutoff – April 14, 2026. Data on sibeprenlimab (Sibe) and povetacept (Pove) from third-party data; Reported half-life of Sibe 9.3 days (Voyxact, 2025) and Pove 2.8 days (Davies, 2024). Sibe TMDD threshold based on a visual estimate of third-party data. No head-to-head study has been conducted comparing JADE101 to other candidates or approved agents. Differences exist between study designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from

a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.
 SC – Subcutaneous; TMDD – Target-Mediated Drug Disposition; ADA – Anti-Drug Antibodies; PK – Pharmacokinetics; PD – Pharmacodynamics; fAPRIL – Free A Proliferation-Inducing Ligand; Q8W – Every 8 Weeks; Q12W – Every 12 Weeks

JADE101 Phase 1 study ongoing

DESIGN

- Randomized, double-blind, placebo-controlled
- Single ascending dose
- Subcutaneous administration (175 mg/mL)

POPULATION

- 32 healthy adult volunteers
- N=8 per cohort (6:2 active:placebo)

OBJECTIVES

- Primary: Safety and tolerability
- Secondary: Pharmacokinetics
- Exploratory: Pharmacodynamics (APRIL, IgA, immunoglobulins); Immunogenicity

Dose levels and length of follow-up to date



Notes: ClinicalTrials.gov ID: NCT07059312. Numbers presented as subjects receiving JADE101 relative to placebo. Each cohort included a sentinel group, n = 2 (1 JADE101, 1 placebo); remainder dosed after safety clearance.

Baseline characteristics were typical of healthy volunteers

JADE101 AND PLACEBO	175 MG	350 MG	700 MG	1,400 MG	ALL COHORTS
N	8	8	8	8	32
Age, (yr), Mean (SD)	38.0 (7.86)	44.5 (9.87)	28.0 (9.04)	37.6 (13.70)	37.0 (11.51)
Female N (%)	2 (25)	6 (75)	3 (38)	5 (63)	16 (50)
White N (%)	5 (63)	6 (75)	4 (50)	3 (38)	18 (56)
Asian N (%)	2 (25)	1 (13)	3 (38)	1 (13)	7 (22)
BMI, (kg/m2) Mean (SD)	25.5 (2.70)	26.7 (2.88)	25.9 (3.05)	24.2 (3.25)	25.6 (2.97)

Notes: Data cutoff – April 14, 2026.
BMI – Body Mass Index; SD – Standard deviation

JADE101 demonstrated **favorable safety profile and was well tolerated** across all evaluated doses

- No severe AEs or deaths
- All TEAEs were mild/moderate in severity
- No clinically significant changes in ECGs or vitals
- No trends of signals in safety labs
 - No cases of IgG ≤ 3 g/L
- Well-tolerated locally by SC injection
 - 3/32 (9%) mild (2)/moderate (1) injection site erythema
 - 1/32 (3%) mild injection site pain
- No apparent impact of anti-drug antibodies was observed on PK or PD

Healthy Volunteer SAD Safety Summary

JADE101 AND PLACEBO	175 MG	350 MG	700 MG	1400 MG	ALL COHORTS
N	8	8	8	8	32
≥ 1 TEAE, n (%)	6 (75)	6 (75)	5 (63)	7 (88)	24 (75)
≥ 1 SAE, n	0	0	0	0	0
≥ 1 severe TEAE, n	0	0	0	0	0
Discontinued due to AE	0	0	0	0	0

TEAEs in the pooled safety analysis occurring in > 2 participants were headache (25%), upper respiratory tract infection (21.9%); injection site erythema, oropharyngeal pain, and pyrexia (9.4% each)

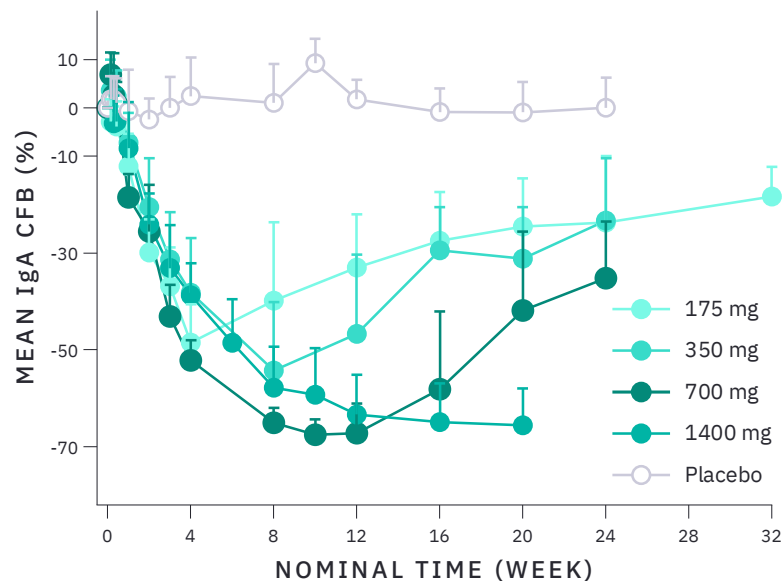
Notes: Data cutoff – April 14, 2026.

AE – Adverse event; ECG – Electrocardiogram; SAD – Single Ascending Dose; SAE – Serious Adverse Event; TEAEs – Treatment Emergent Adverse Events

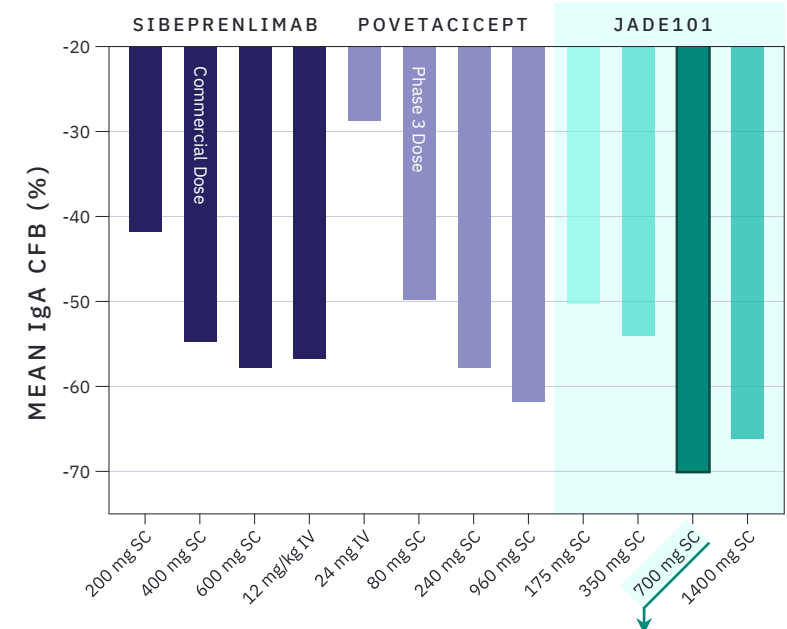
JADE101 single dose **IgA reductions exceeded** those reported with first-generation molecules

- Prolonged IgA reductions of ~70% following a single dose of JADE101
 - Reductions exceeded those reported for first-gen agents
 - Induction dose of 700 mg expected to drive ~70% IgA reductions, with 350 mg maintenance dose predicted to sustain this reduction
- Largest reported magnitude of IgA lowering effect with a single dose for an anti-APRIL or dual APRIL/BAFF
- IgA reductions sustained at 12 weeks at 700 mg

IgA Change from Baseline by Dose



IgA reduction in Healthy Volunteers



700 mg single dose predicted to reflect IgA responses with Q12W maintenance dosing (700 mg induction + 350 mg maintenance)

Notes and Sources: Data cutoff – April 14, 2026. Internal data; IgA responses estimated via noncompartmental analysis of individual JADE101 profiles and internal analyses of mean reported profiles of povetacicept (Davies, 2024) and sibeprenlimab (Mathur, 2022; Zhang, 2023). Povetacicept and sibeprenlimab data points extracted via digitization and represent approximate values. The company does not possess the underlying raw third-party data. No head-to-head study has been conducted comparing

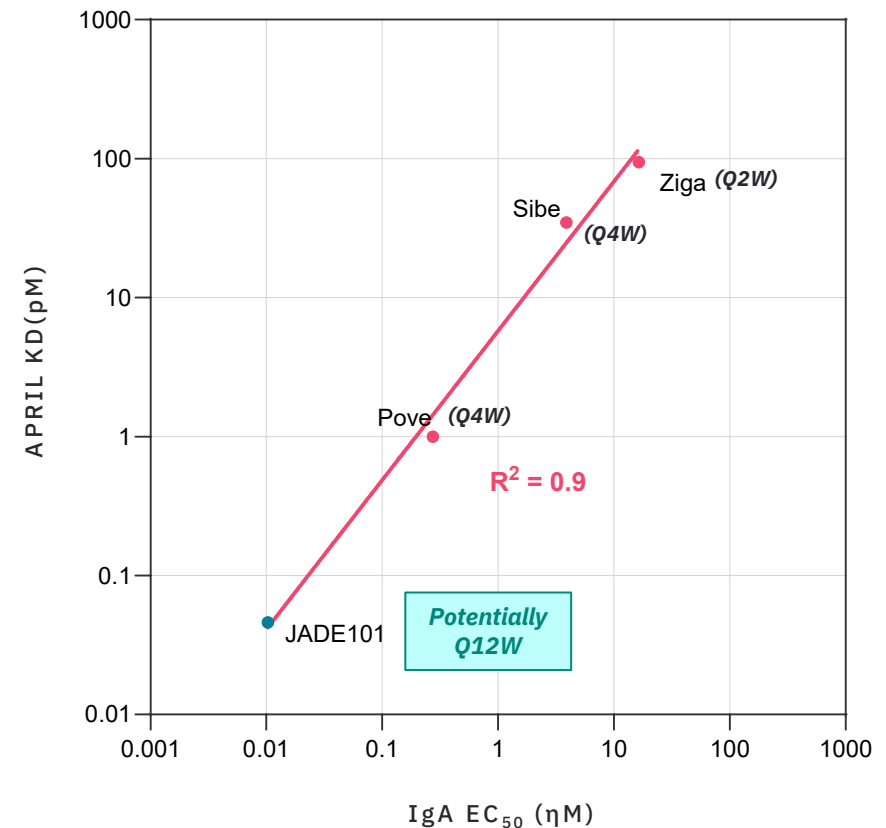
JADE101 to other candidates or approved agents. Differences exist between study designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.
First-gen – First Generation

JADE101 demonstrated **compelling *in vivo* potency** to lower IgA

- Ultra-high APRIL binding affinity predicted JADE101's enhanced *in vivo* potency to lower serum IgA in humans
 - JADE101 has shown rapid, deep and sustained IgA depletion

	JADE101	POVE	SIBE	ZIGA
IgA EC ₅₀ (nM)	0.010	0.27	3.9	16.4
IgA EC ₅₀ V JADE101	n/a	27x	379x	1,595x
Dosing Interval	Potentially Q12W	Q4W	Q4W	Q2W

APRIL Binding Affinity and IgA Lowering Potency



Notes and Sources: Data cutoff – April 14, 2026. Internal data; Evans, 2023 (Pove); Benchmarks manufactured based on publicly available sequences. IgA EC₅₀ estimates calculated using compartmental PK models linked to indirect response models to describe IgA kinetics built using JADE101-01 data or published PK and IgA concentration-time profiles in single dose studies and demonstrated maximum biological effects (Sibe: FDA Review 2025) Sibe (Mathur, 2022; Zhang, 2023); Ziga (Kooienga, 2025); Pove

(Davies, 2024); Data points extracted via digitization and represent approximate values. The company does not possess the underlying raw third-party data. These data are derived from different trials at different points in time, with differences in trial design and populations. No head-to-head clinical trials of JADE101 and other agents have been conducted.

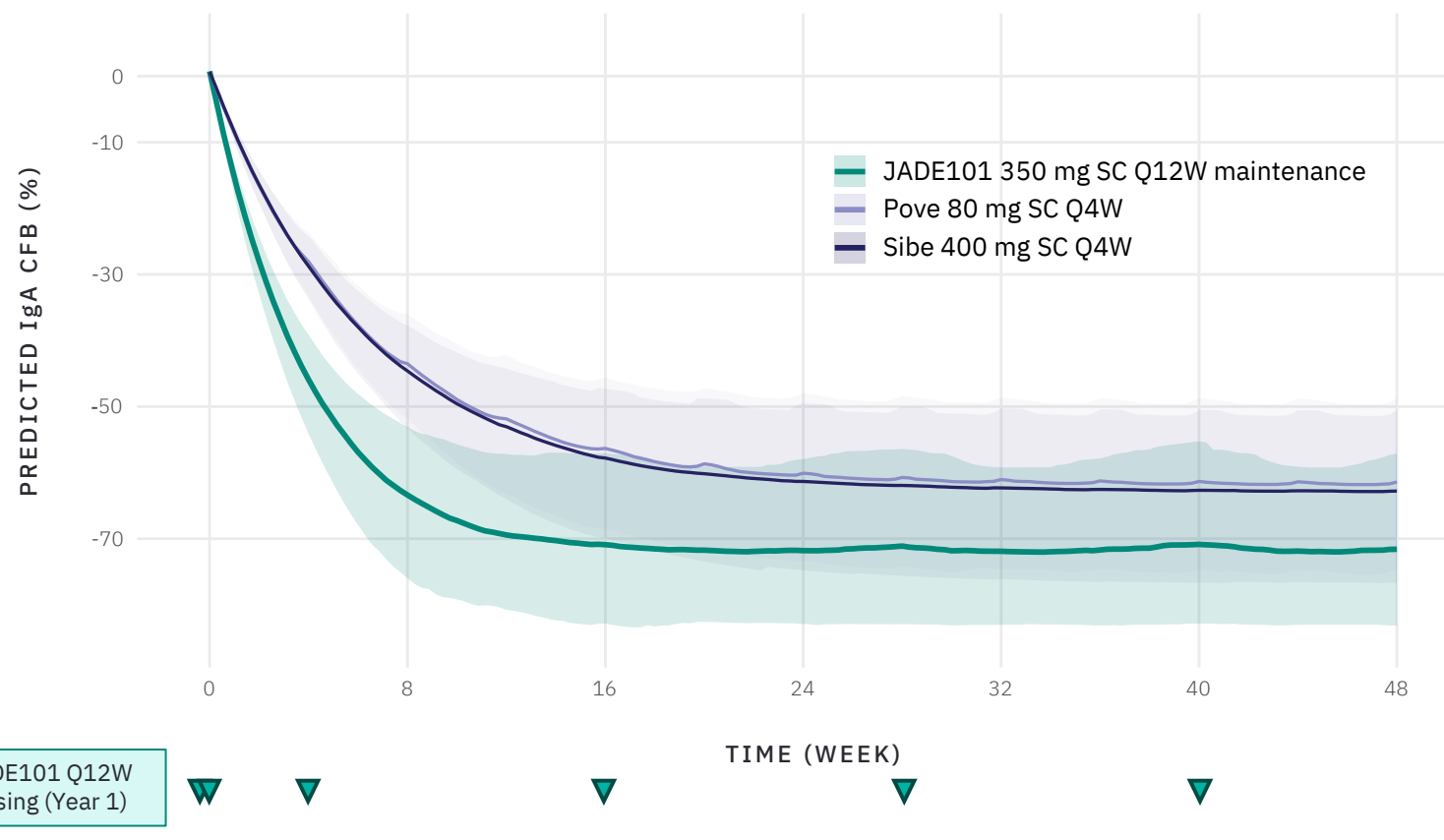
EC – Effective Concentration; V – Value; nM – Nanomolar

JADE101 PD modeling supports Q12W maintenance dosing

Deeper IgA reductions simulated over first-generation anti-APRILs

Simulated IgA vs Time – 48 weeks

- JADE101 induction dose (700 mg) predicted to rapidly maximize IgA depletion
- >70% IgA reductions projected at steady state with a single subcutaneous maintenance injection (350 mg) every 12 weeks
- IgA reductions modeled to be faster and deeper than first generation anti-APRIL or dual APRIL/BAFF



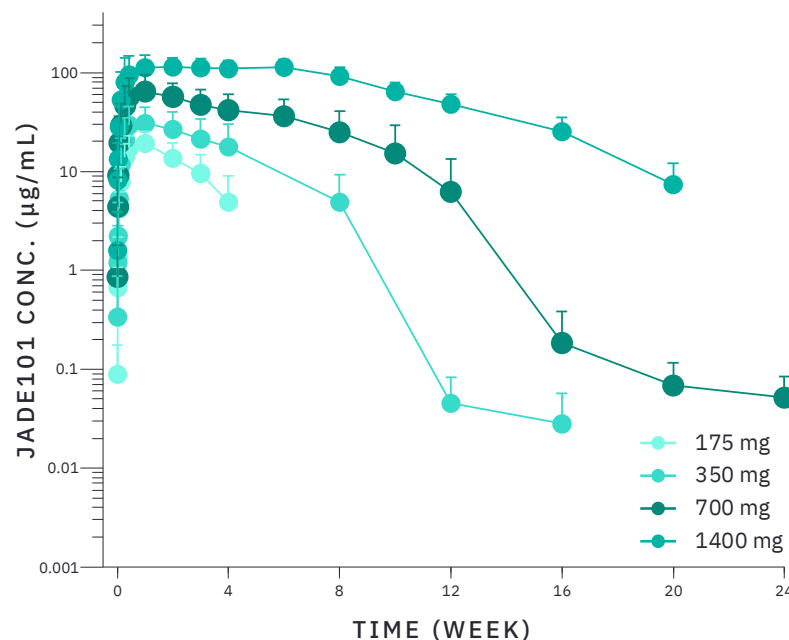
Notes and Sources: Data cutoff – April 14, 2026. Internal data; Solid lines represent the median and shaded area represents the 5th and 95th prediction intervals across 500 simulated trials. JADE101 population-based simulations informed by interim biomarker rich healthy volunteer data obtained in JADE101-01 integrated with data available in the public domain. Sibeprenlimab and povetacept simulations conducted via population-based approaches using internal models

informed by data available in the public domain (Pove: Davies 2024; Sibe: FDA Review 2025). Underlying povetacept and sibeprenlimab data points extracted via digitization and represent approximate values. The company does not possess the underlying raw third-party data. Modeled data may not be realized in actual clinical studies and may not accurately represent performance of third-party products.

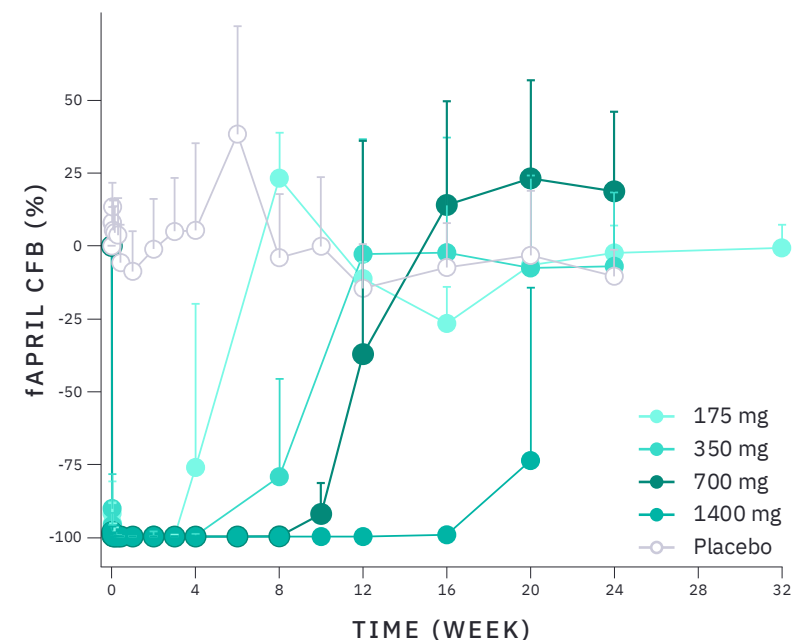
IgA reductions for 12 weeks enabled by dose-dependent PK and rapid, complete, and sustained fAPRIL suppression

- ~8.7 fold longer half-life than povetacicept; ~2.6 fold longer than sibeprenlimab
 - JADE101 half-life: 24.2 days
- JADE101 TMDD threshold estimated ~2.5-fold lower than sibeprenlimab
 - PK profiles of first-gen anti-APRIL mAbs strongly influenced by TMDD
- Deep serum fAPRIL suppression as rapidly as 2 hours post SC dose administration
 - >90% APRIL suppression sustained for a median of 85 days at 700 mg

Pharmacokinetics



fAPRIL

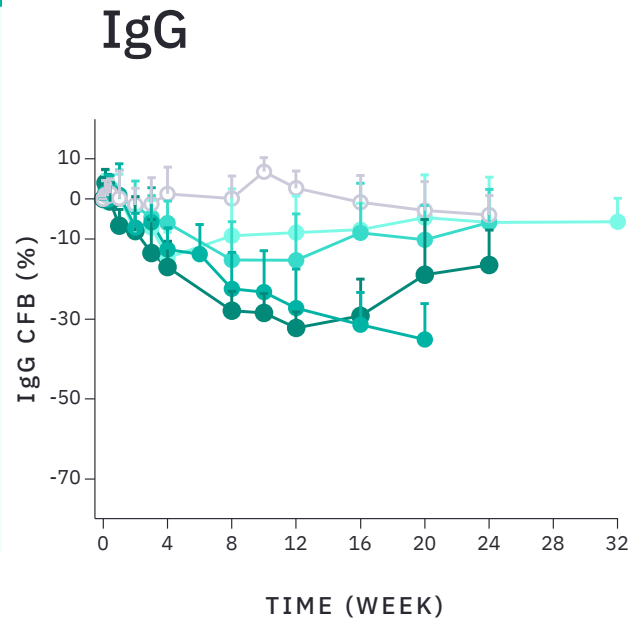


Notes and Sources: Data cutoff – April 14, 2026; Internal data. Half-life dependent on dose and dose interval; JADE101 half-life measured at 700 mg induction dose. Sibeprenlimab TMDD threshold based on visual estimate of third-party data. Duration of fAPRIL response (>90%) estimated via noncompartmental analysis of individual JADE101 participant profiles. No head-

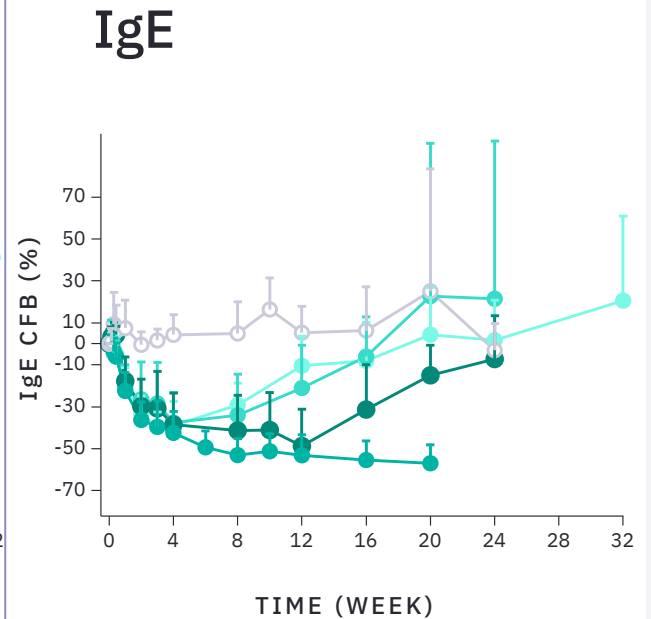
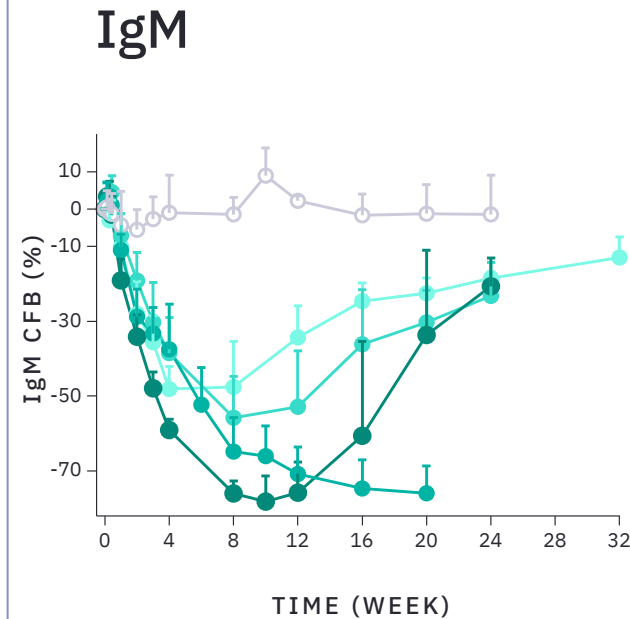
to-head study has been conducted comparing JADE101 to other candidates or approved agents. Differences exist between study designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.

Changes in IgG, IgM and IgE were consistent with the selective anti-APRIL MoA

- Magnitude of IgG-lowering consistent with relatively IgG-sparing selective-anti-APRIL MoA
- No cases of IgG ≤ 3 g/L
- Substantial IgM and IgE reductions



- 175 mg
- 350 mg
- 700 mg
- 1400 mg
- Placebo

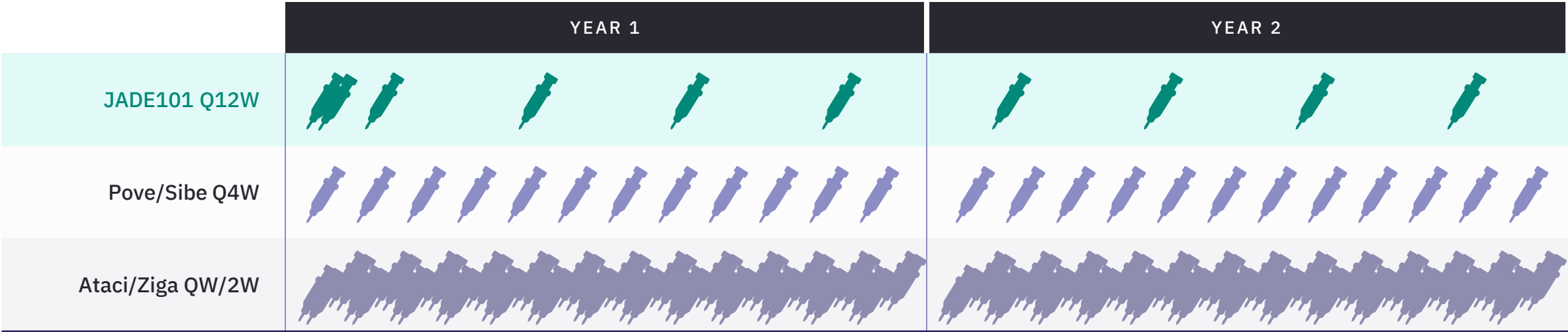


JADE101

Next steps

Q12W dosing expected to optimize **clinical activity and convenience for patients**

- Plan to evaluate two dosing regimens (Q12W and Q8W) in Phase 2 and Phase 3
 - Two dose Phase 3 may enable an accelerated Phase 3 initiation without awaiting Phase 2 data
 - Supports global regulatory expectations for dose-finding
- Initial induction dose intended to drive ~70% IgA reductions at earlier timepoints
 - Induction dose is 700 mg, followed by Q12W or Q8W maintenance dosing (350 mg) beginning at Week 4



Notes: JADE101 projected dosage frequency based on internal population pharmacodynamic modeling and dosing schedules remain subject to further clinical investigation. No head-to-head study has been conducted comparing JADE101 to other candidates or approved agents. Differences exist between study designs, patient characteristics and other factors, and caution

should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.

JADE101 Phase 2 IgAN patient trial initiated; interim data anticipated in 2027



DESIGN

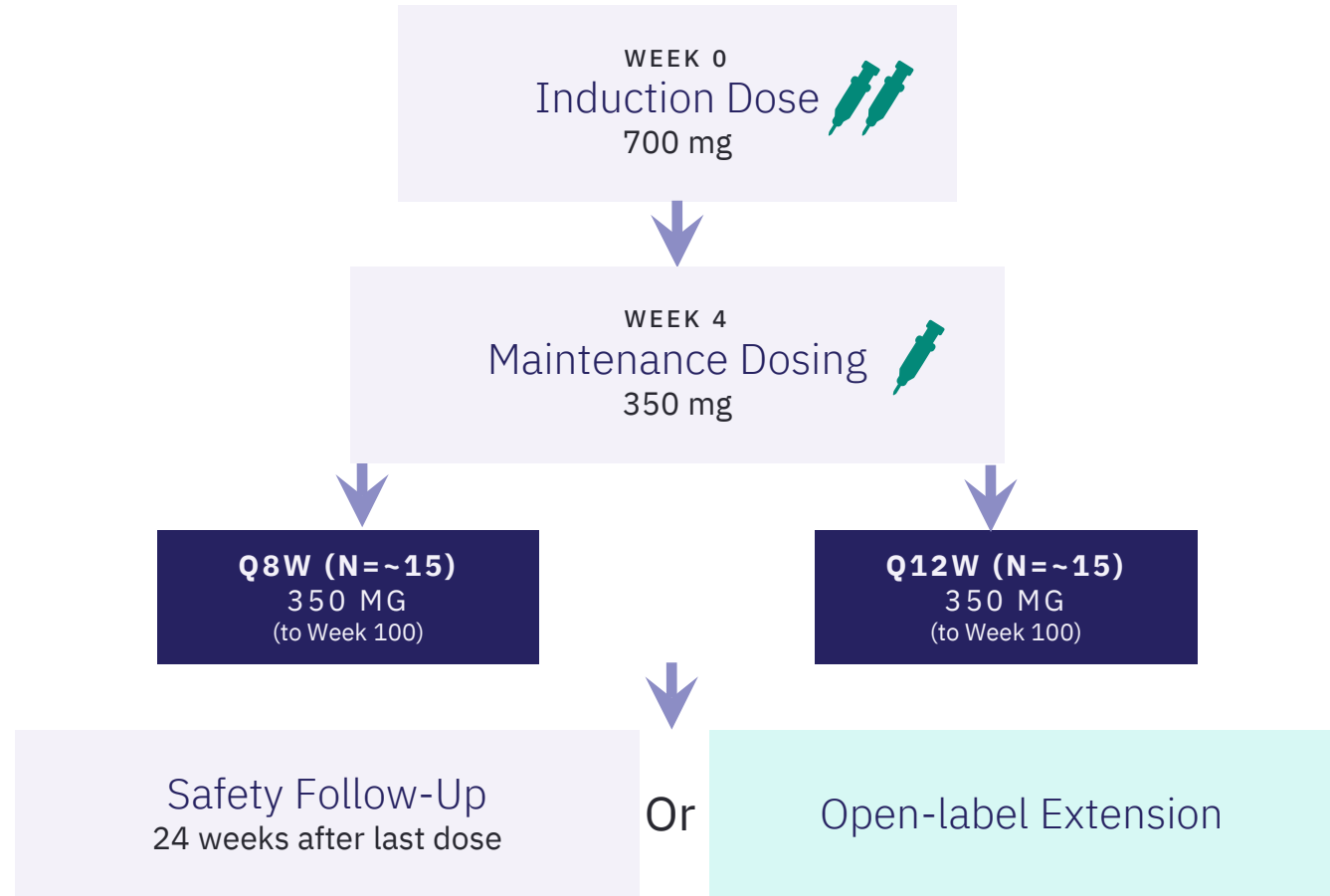
- Randomized, open-label
- Subcutaneous administration

POPULATION

- Adults with IgAN within 5 yrs
- Proteinuria ≥ 0.75 g/g
- eGFR ≥ 30 mL/min/m²
- Stable SOC ≥ 12 weeks

OBJECTIVES

- Safety and tolerability
- UPCR-24 over time (<0.5 g/day, <0.3 g/day)
- eGFR over time



JADE201

a potentially best-in-class
afucosylated anti-BAFF-R mAb

A potentially best-in-class afucosylated anti-BAFF-R mAb

With dual MOA B cell depletion to treat autoimmune diseases

JADE201 builds on ianalumab's proof-of-concept, adding HLE for expected **improved durability, less frequent dosing, and potentially best-in-class profile**

- B cell depletion has proven effective in autoimmune disease, but existing therapies like rituximab and anti-CD19 agents face limits:

Incomplete B cell depletion due to low target receptor expression on some B cell subsets or paucity of effector cells to mediate killing¹

Residual B cells in secondary lymphoid tissues and/or **ineffective depletion of B cells in ectopic lymphoid tissue** after treatment²

Sparing pathogenic autoantibody producing cells, including plasmablasts

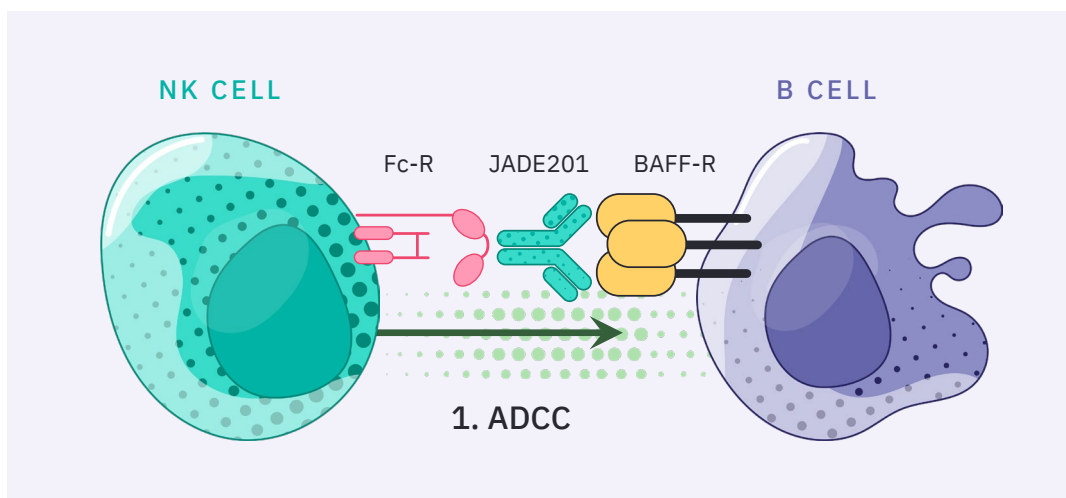
Resistance mechanisms, including increased BAFF expression following treatment with rituximab³

- Resistance mechanisms, particularly elevated BAFF after anti-CD20 therapy, enable autoreactive B cells to repopulate, undermining durability
- Ianalumab, an afucosylated anti-BAFF-R, provided proof-of-concept for overcoming these barriers, including clinical tissue B cell depletion⁴

JADE201's dual MOA expected to deliver deeper, **more durable B cell depletion**

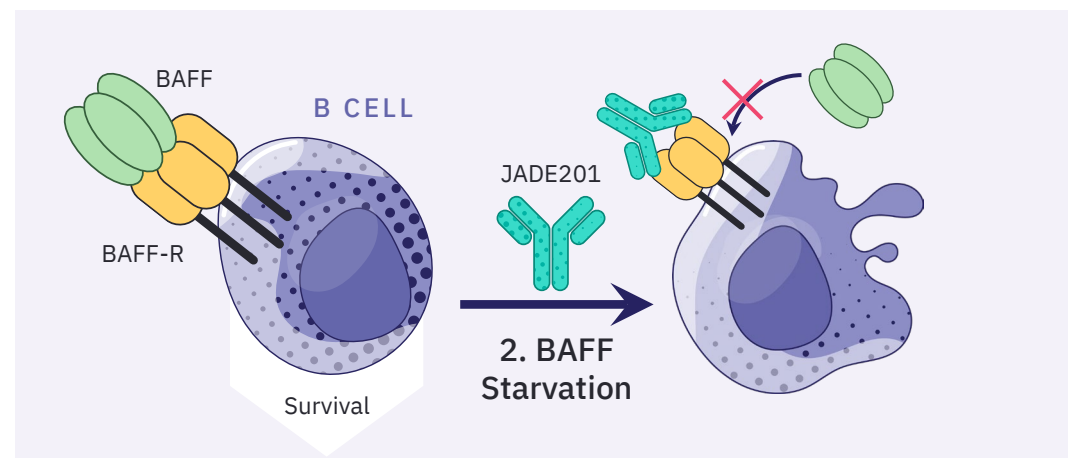
Direct Cytotoxicity via Enhanced Effector Function

- Validated mechanism that induces rapid B cell depletion
- Enhanced cytotoxicity by ADCC
- Potent depletion of circulating B cells

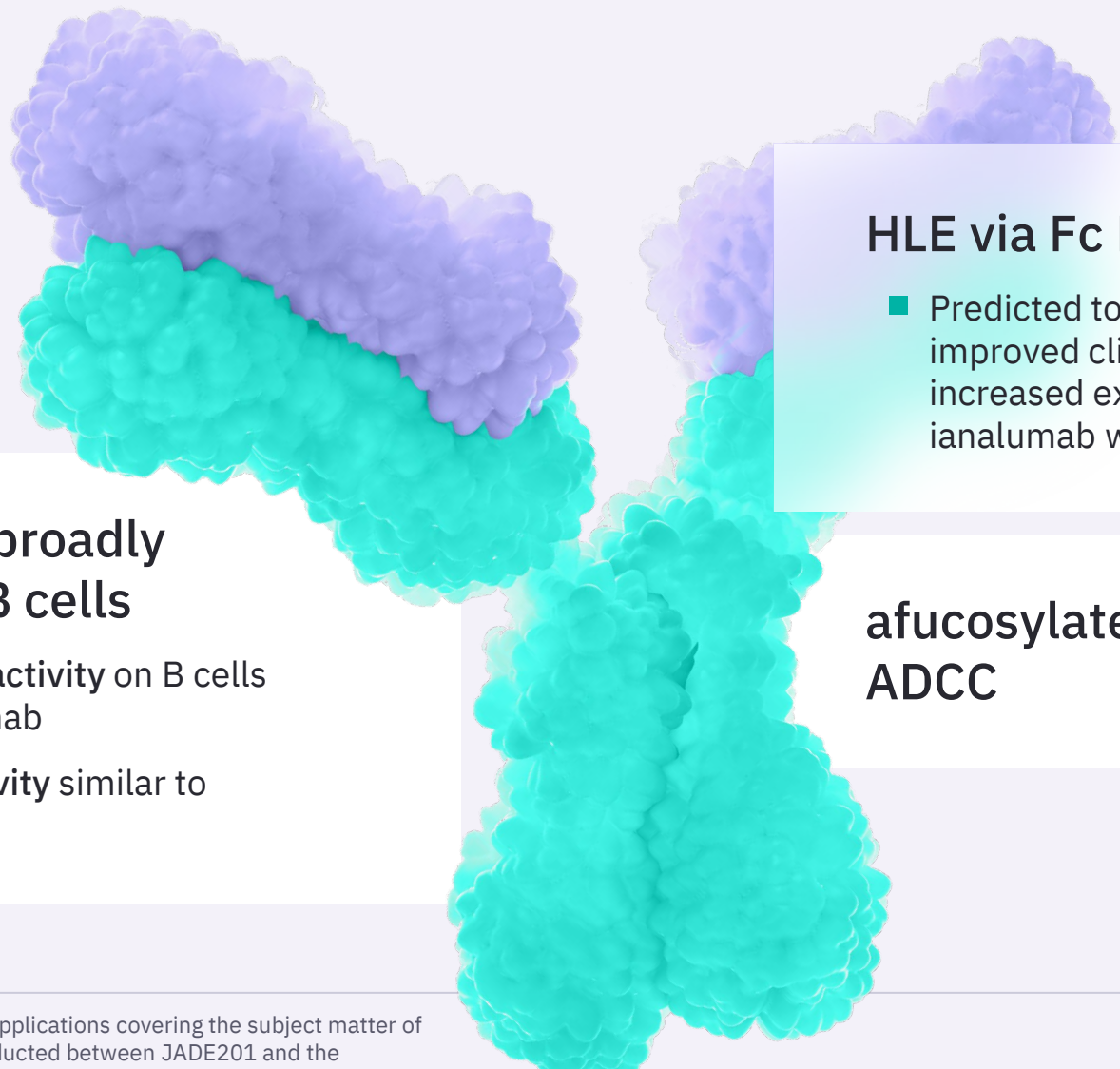


B Cell Inhibition and Depletion by BAFF Starvation

- Mechanism works in context of low receptor expression
- Relevant in secondary and ectopic lymphoid tissues where effector cells may be scarce
- Avoids B cell repopulation and resistance due to increased BAFF expression following B cell depletion with anti-CD20 agents



Potentially **best-in-class** properties of JADE201



Binds BAFF-R broadly expressed on B cells

- Enhanced ADCC activity on B cells similar to ionalumab
- Blocks BAFF activity similar to ionalumab

HLE via Fc LS mutation

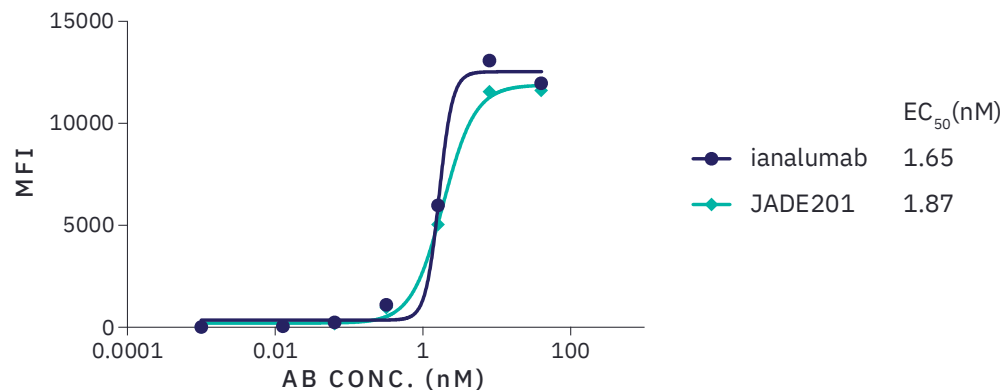
- Predicted to match, with potential for improved clinical activity due to increased exposure compared to ionalumab with less frequent dosing

afucosylated for enhanced ADCC

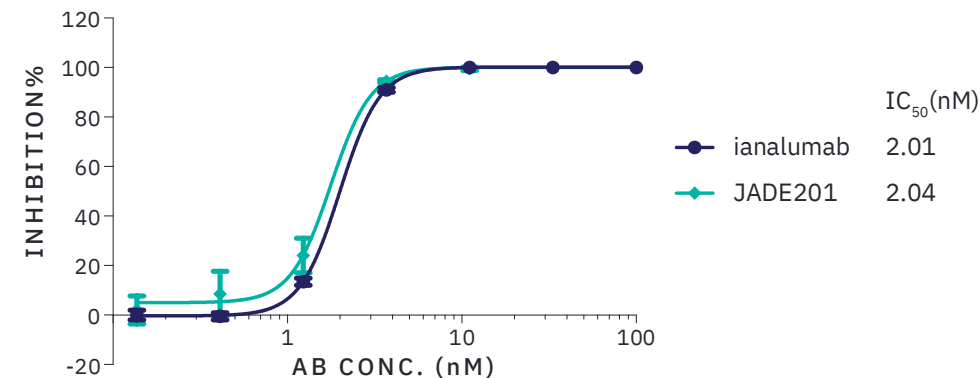
Notes: Jade and its licensor, Paragon, have filed patent applications covering the subject matter of JADE201. No head-to-head clinical trials have been conducted between JADE201 and the referenced agent. ADCC – antibody-dependent cellular cytotoxicity; HLE – half-life extension

JADE201 exhibits high BAFF-R binding affinity and functional activity in preclinical studies

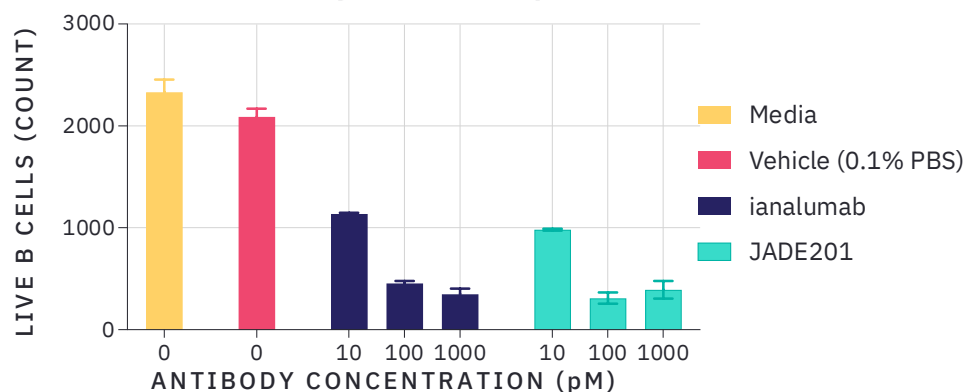
BAFF-R Binding (HEK Cells)



BAFF-R Blockade (Competition ELISA)



ADCC Activity Primary human CD19+ B Cells



Additional Attributes Similar Between Clones

- Affinity to human/cyno BAFF-R by SPR
- BAFF-R binding (Raji B cells)
- FcR binding (excluding FcRn*)
- C1q binding
- ADCC activity on Raji B cells

Note: No head-to-head clinical trials of JADE201 and the referenced agent have been conducted. Ianalumab manufactured from publicly available sequence.

*LS mutation ~10x higher affinity to FcRn.

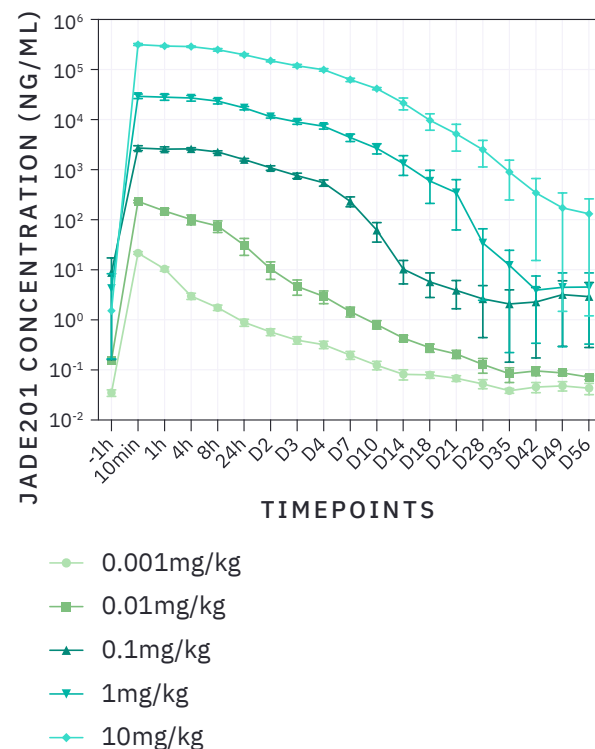
ELISA – Enzyme-linked immunosorbent assay; HEK – Human Embryonic Kidney cell; Cyno – Cynomolgus macaque; pM – Picomolar; SPR – Surface plasmon resonance; C1q – Complement component 1q

JADE201 demonstrates **deep B cell depletion** in NHPs

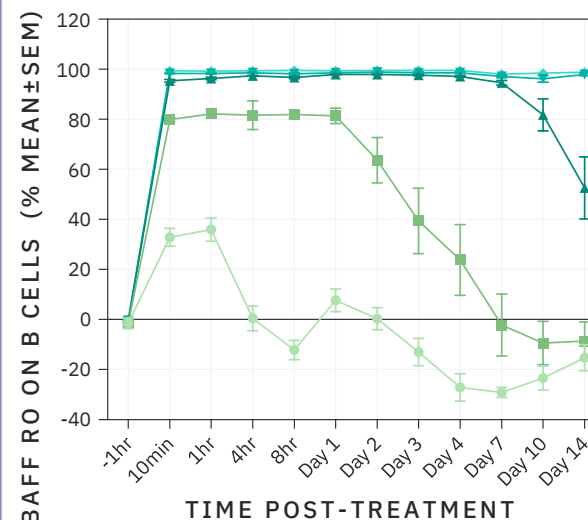
JADE201 demonstrates **dose-dependent PK**. Rapid RO observed with **complete RO achieved at doses above 1 mg/kg**

Deep and sustained B cell depletion achieved after single dose of JADE201 in NHPs

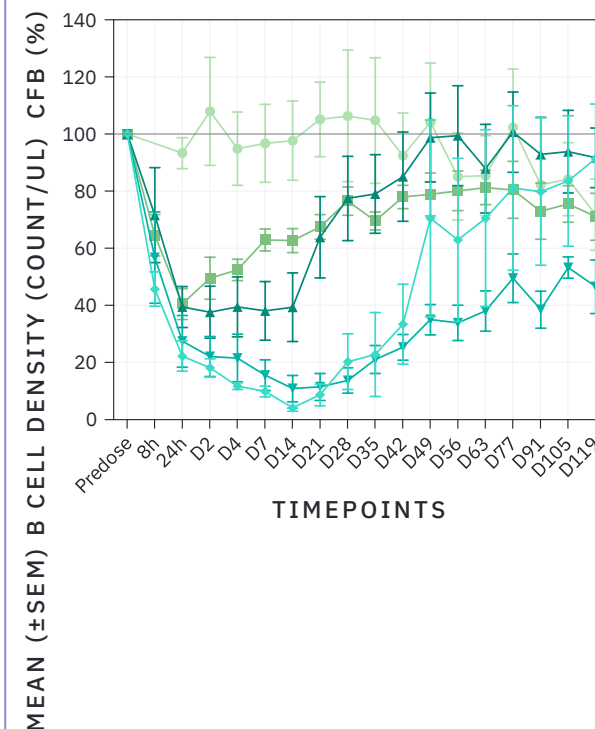
JADE201 PK



BAFF-Receptor Occupancy (RO)

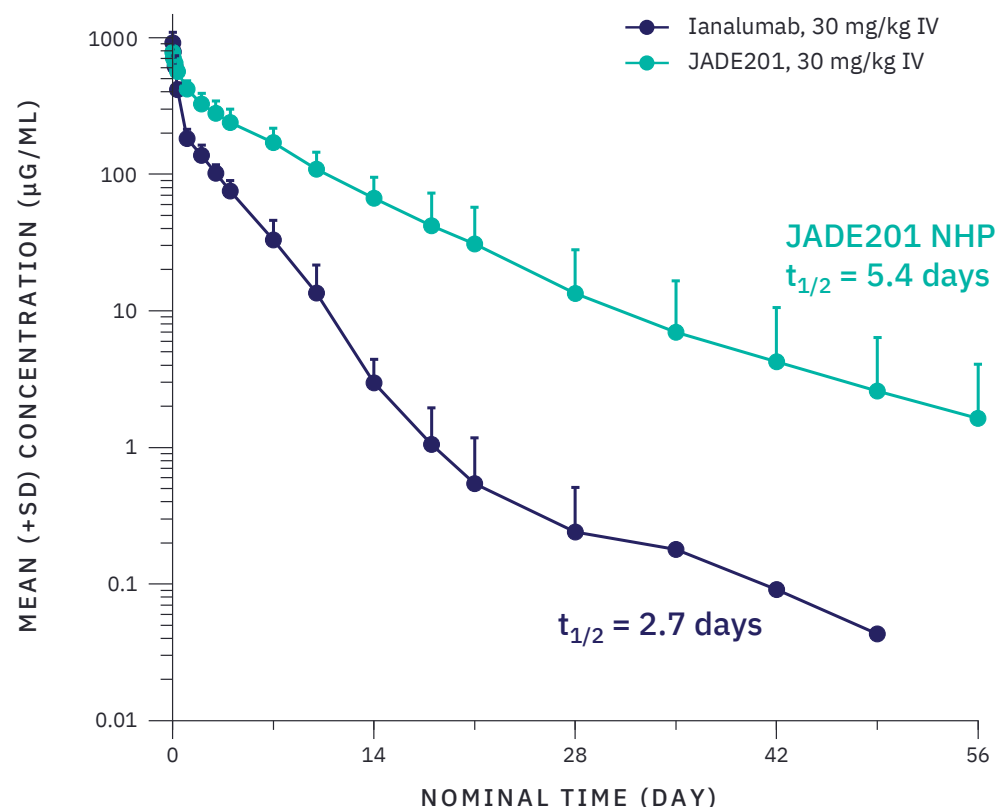


B Cell Depletion



JADE201 demonstrates a differentiated NHP PK profile from ianalumab

>2X HLE demonstrated in NHPs



HLE has potential to provide sustained BAFF receptor occupancy and improved clinical response

- Ianalumab has an observed human $T_{1/2} \sim 10$ days¹
- JADE201 with HLE has the potential to provide complete BAFF-R blockade for an extended duration
 - Potential for deeper, more durable clinical responses
 - Extended dosing interval providing a more convenient, infrequent SC dosing profile

Notes: Individual NHP time points that appear to be impacted by ADA excluded from half-life determinations and mean concentration-time plots. Information provided above is for illustrative purposes only and no head-to-head clinical trials have been conducted. Ianalumab manufactured from publicly available sequence.

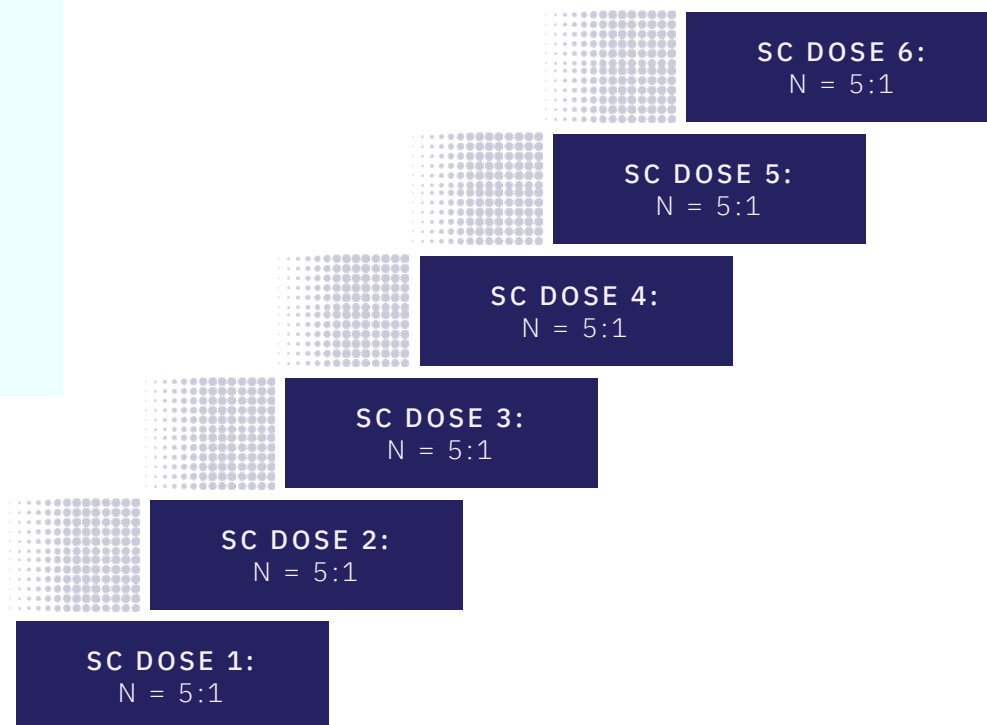
Sources: ¹2019 Dorner (Ann Rheum Dis)
NHP – Non-Human Primate; HLE – Half-Life Extension; SC – Subcutaneous

JADE201 **first-in-human trial** underway; interim data expected in 2027

JADE201 preclinical profile supports potential for **best-in-class clinical activity** with convenient, patient-friendly dosing

Phase 1 Study Design

Randomized, double-blind, placebo-controlled SAD study
SC administration in adults (n=36) with rheumatoid arthritis.



Objectives

PRIMARY

- Safety and tolerability

SECONDARY & EXPLORATORY

- Pharmacokinetics
- Pharmacodynamics
- Immunogenicity
- B cell depletion
- DAS28

Notes: Numbers presented as subjects receiving JADE201 relative to placebo. Each cohort to include a sentinel group, n = 2 (1 JADE201, 1 placebo); remainder dosed after safety clearance.
DAS – Disease Activity Score

JADE201 profile could enable **broad opportunity in multiple indications** where B cells are pathogenic

U.S. DISEASE PREVALENCE¹

Lupus nephritis ~85,000

Immune thrombocytopenia ~85,000

Sjögren's disease ~3.5M

Primary membranous nephropathy ~35,000

ANCA-associated vasculitis ~125,000

Rheumatoid arthritis ~2.0M

 **Best-in-class potential**  **First-in-class BAFF-R**

DISEASE SEVERITY AND KEY TX MODALITIES ACROSS INDICATIONS²



Moderate-to-severe

Refractory

CD20/CD19 B cell depleters e.g., rituximab, obinutuzumab

-  Proven efficacy and MoA target
-  Safety limitations, incl. high infection risk and black box
-  Limited durability, with relapse and re-treatment over time
-  Burdensome IV admin and dosing schedule

JADE201

anti-BAFF-R mAb

-  Targets MoA validated across multiple indications in late-stage trials
-  Potential for improved benefit / risk vs. B cell depleters
-  Potential for better durability with greater tissue depletion and blockade of resistance
-  Designed for improved dosing convenience

TCEs & Cell Therapies e.g., BiTEs, CAR-Ts, CAR-NKs

-  Addresses later-line and highly refractory disease
-  Cell therapy Tx could lead to full immune reset
-  Low applicability to majority of moderate-to-severe disease
-  High treatment burden, incl. IV admin and safety considerations

Notes: 1) Representative of potential best-in-class and first-in-class JADE201 indications; 2) Illustrative representation of proportion of moderate-to-severe disease vs. refractory patients across B cell implicated autoimmune diseases

Tx – Treatment; TCEs – T-cell Engagers; BiTEs – Bispecific T-cell Engagers; CAR-Ts – Chimeric Antigen Receptor T-cell Therapies; CAR-NKs – Chimeric Antigen Receptor Natural Killer; ANCA – Antineutrophil cytoplasmic antibodies; IV – Intravenous

JADE301:

IFN- β Neutralizing mAb for
Dermatomyositis (DM)

JADE301: building on **validated IFN- β biology** with **best-in-disease potential**; **rapid path** to human data

Dermatomyositis:

Large, underserved opportunity

- Debilitating autoimmune disease affecting skin and muscle
- ~50,000 adults in the U.S.; significant unmet need despite recent treatment advances

IFN- β :

Clinically validated mechanism

- IFN- β is a critical upstream driver of DM pathology
- Selective IFN- β inhibition has demonstrated clinical proof-of-concept in both muscle and skin components of DM

JADE301:

Disease-modifying approach

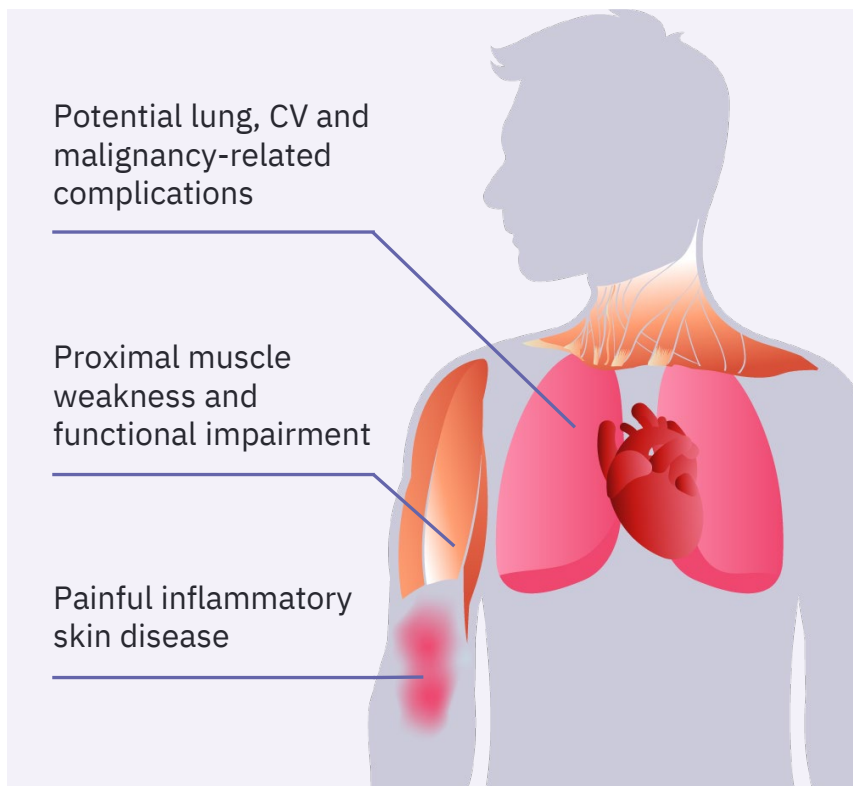
- High-affinity, potent and selective IFN- β neutralization in preclinical studies
 - 2.5x (pSTAT) to 8x (MX-1) higher potency than dazukibart*
- YTE modification for half-life extension
 - 3x longer NHP half-life than dazukibart*
- Preclinical profile supports planned SC administration

Phase 1 HV study expected to initiate in 4Q 2026; interim data anticipated in 2H 2027

Dermatomyositis is a debilitating autoimmune disease affecting ~50K U.S. adults

- Affects adults and children, with higher prevalence in women
- 60–80% of patients do not achieve sustained remission
- Mortality ~3X the general population

A chronic, multisystem disease



High impact on daily activities

58%

experience severe itch; results in sleep disruption for many patients



53%

unable to walk >one mile



50%

unable to bend, lift or stoop



37%

unable to climb one flight of stairs

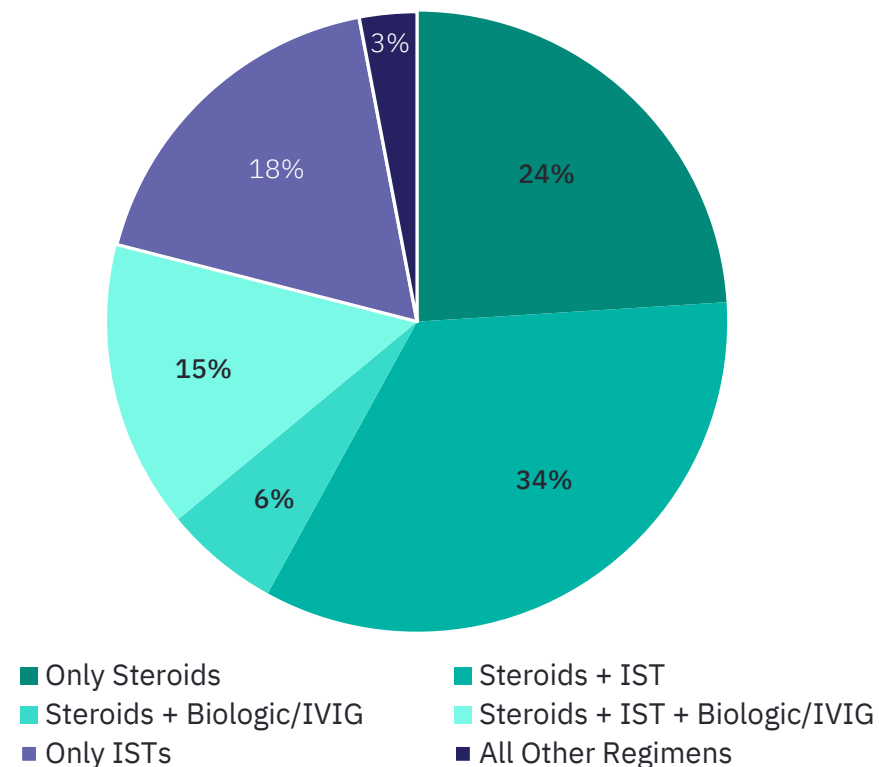


Despite treatment advances, **DM remains burdensome** with incomplete disease control

Opportunity for a targeted, disease-modifying and steroid-sparing therapy with durable control and convenient, infrequent SC administration

- Steroids are used in **~80% of DM patients and come with significant side effects**
- Treatment depends on **severity of clinical presentation, organ involvement, and autoantibody type**
- **High residual disease burden:** Nearly all patients experience pain, >50% require opioids
- Despite existing therapies, most adults with DM **experienced at least one disease flare in the past year**
- Even with a newly approved targeted therapy, **class-level safety considerations and residual disease** leave a continued need for better options

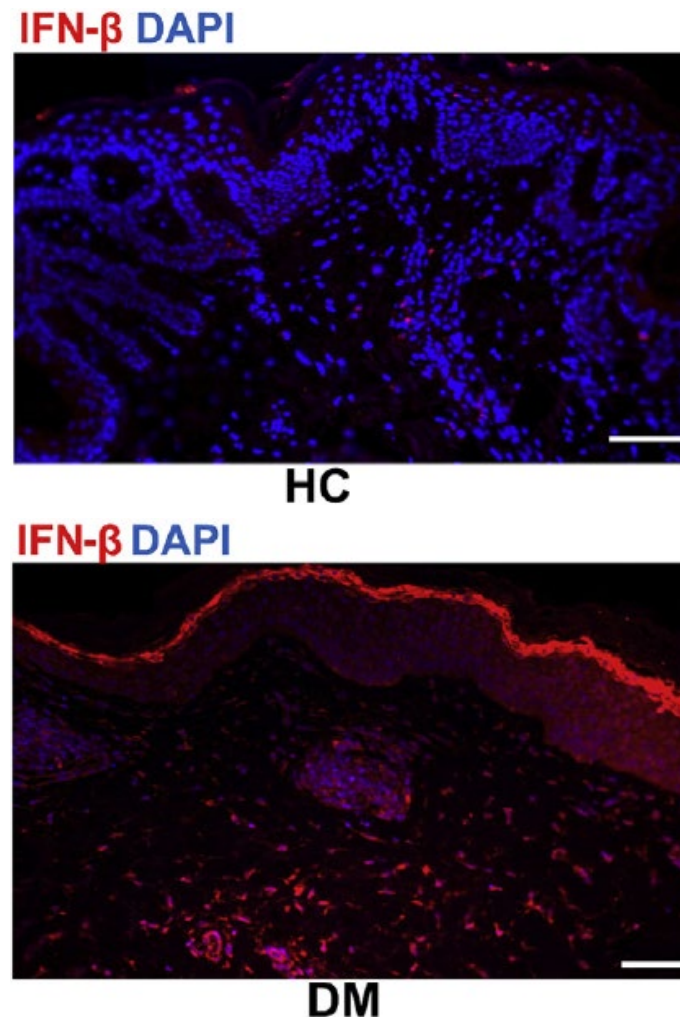
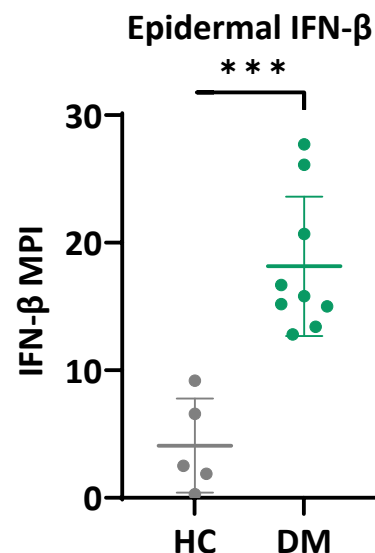
Therapies Received by ~34K Treated Dermatomyositis Patients



IFN- β is a **key driver** of skin and muscle disease in DM

Excess IFN- β can amplify inflammation, impair muscle repair and contribute to tissue damage

- Type I interferon signaling is markedly overactive in DM skin and muscle
- Excess signaling activates immune cells, impairs muscle repair and disrupts microvasculature
- IFN- β is the predominant type I interferon elevated in DM blood and skin
- Autoantibodies further drive expression by targeting regulators of IFN- β
- Serum IFN- β , but not IFN- α , correlates with skin disease activity
- Therapeutic IFN- β exposure has been linked to new-onset or worsening DM-like disease



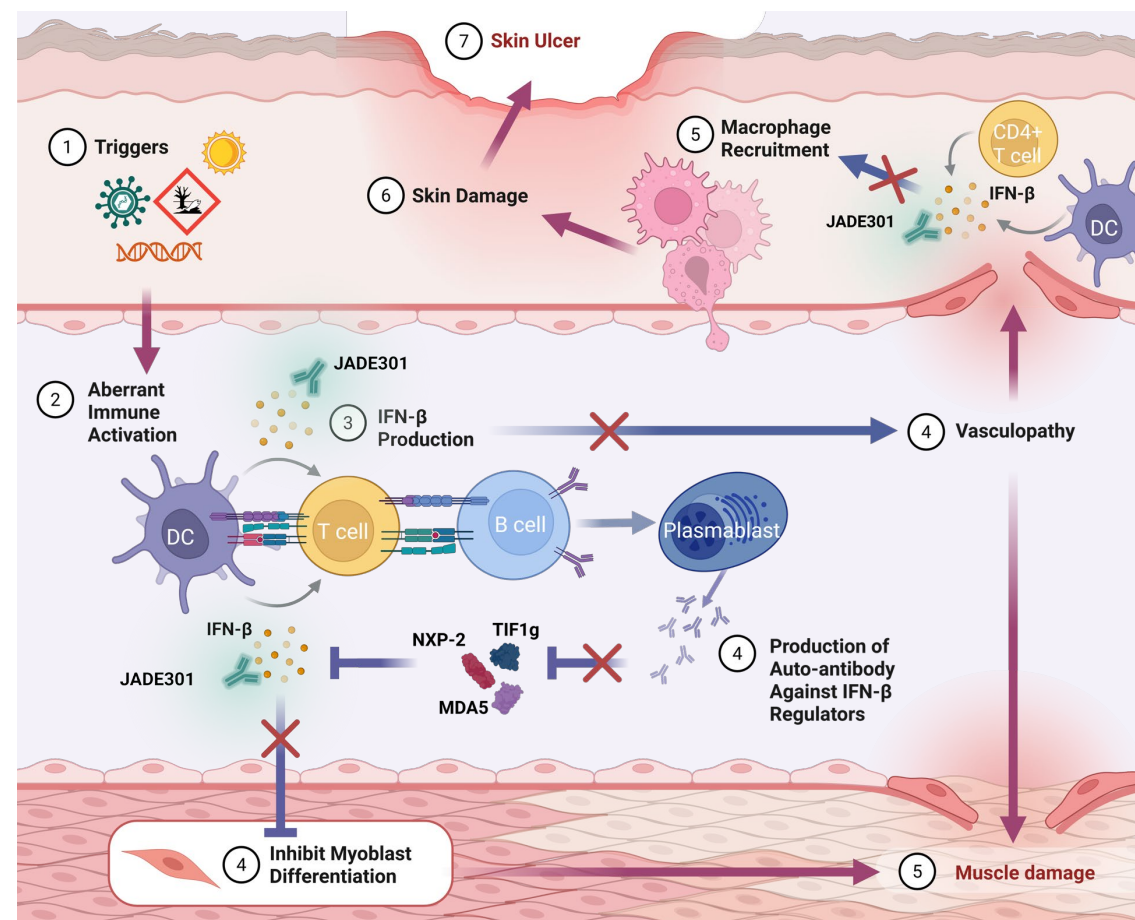
Sources: 2005 Greenberg (Ann Neurol); 2025 Bolko (Front in Immunol); 2017 Huard (Br J Dermatol); 2011 Liao (Ann Rheum Dis); 2024 Pinal-Fernandez (Ann Rheum Dis); 2021 Patel (J invest Derm); 2008 Somani (Arch Dermatol); 2015 Kolb-Maurer (Int J Mol Sci)

IFN- α – Interferon alfa; IFN- β – Interferon-beta; MPI – Mean Pixel Intensity; HC – Healthy Controls; DAPI – 4',6-diamidino-2-phenylindole (for nuclear staining)

JADE301 combines a **targeted mechanism** with a **differentiated molecular profile** in DM

JADE301 designed to selectively neutralize IFN- β and interrupt disease-driving signal

- Designed to directly inhibit activity of IFN- β , quieting immune activation, muscle and skin damage
 - Potential disease-modifying therapy in DM
- Designed to selectively neutralize IFN- β while sparing other cytokines, including IFN- α
 - Expected to have better safety profile versus IFNAR blockade, which increases susceptibility to viral infection and reactivation
 - More specific, with potential for enhanced clinical activity, while avoiding broad inhibition of multiple cytokine-signaling pathways, such as JAK/TYK2



Selective IFN- β blockade and SC dosing position JADE301 for best-in-disease potential

PRODUCT MECHANISM · ROUTE · STAGE	EFFICACY (PBO-ADJUSTED) WEEK 12 / FURTHEST REPORTED		SAFETY	DOSING CONVENIENCE
	TIS (MUSCLE)	CDASI (SKIN)		
IVIG (Octagam 10%) Immunoglobulin · IV · Approved	✓ 24.9 / 26.8 (W16)	○ nd / -8.0 (W16)	✗ Boxed warning: thrombosis, renal dysfunction/failure	✗ IV 2 g/kg over 2–5 days, every 4 weeks
Brepocitinib (Lisraya) TYK2/JAK1 inhibitor · Oral · Approved	○ ~10.0 / 15.3 (W52)	○ ~-4.5 / -4.6 (W52)	✗ Boxed warning: Infections, mortality, malignancy, MACE, thrombosis	✓ Oral, once daily
Anifrolumab Anti-IFNAR1 mAb · SC · Phase 3	nd	nd	○ Broad IFN blockade; increased infection risk (viral reactivation)	○ SC weekly
Efgartigimod FcRn antagonist · SC · Phase 3	○ ~13.0 (Ph 2) / 14.5 (W52, Ph 3, data ns)	nd	○ Increased ISRs, infections and Grade ≥ 3 AEs vs. PBO in Phase 3	○ SC weekly (5mL)
Dazukibart Anti-IFN- β mAb · IV · Phase 3	✓ 19.4 (Ph 2) / nd	✓ -16.3 (Ph 2) / nd	✓ Generally favorable Phase 2 safety profile	✗ IV over approx. 1 hour, every 4 weeks

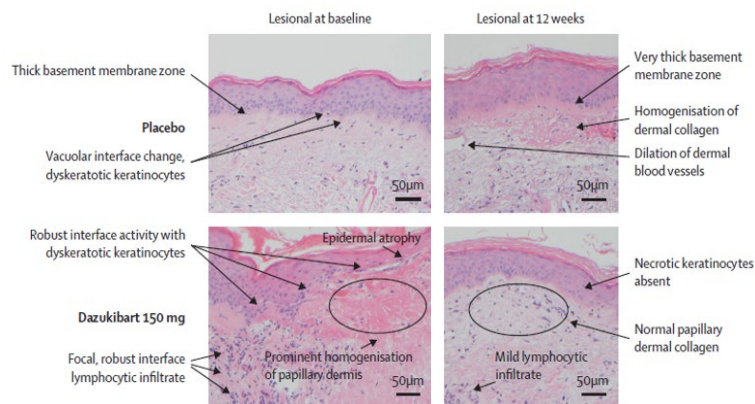
Notes: Available competitor Phase 2/3 and label data reflect reported or digitized placebo-adjusted mean or least-squares (LS) mean differences. Dazukibart TIS reflects prespecified sensitivity analysis in muscle full analysis set (FAS); CDASI reflects pooled skin FAS. Information provided above is for illustrative purposes only. Differences exist between study or trial designs, and caution should be exercised when comparing data across trials. No head-to-head clinical trials have been conducted between JADE301 and the referenced therapies.

Sources: 2025 Fiorentino (Lancet); Dazukibart Ph 3 (NCT05895786); 2026 Vleugels (NEJM); Lisraya Prescribing Info; 2022 Adas (Expert Rev Clin Immunol); Argenx R&D Spotlight June 2026; Argenx ALKIVIA topline Aug 2026 (DM cohort); AstraZeneca H1 & Q2 results July 2026; Saphnelo Prescribing Info; 2022 Aggarwal (NEJM); Octagam 10% Prescribing Info; TIS – Total Improvement Score; CDASI – Cutaneous Dermatomyositis Disease Area and Severity Index; AE – Adverse Event; FcRn – neonatal Fc receptor; MACE – Major Adverse Cardiovascular Event; ISR – Injection Site Reaction; PBO – Placebo; nd – no data; ns – not statistically significant; “~” – data digitized from graphs where publications did not provide specific values

IFN- β is a **clinically validated target for DM** based on clinical data of dazukibart

- Dazukibart Phase 2 data demonstrated best-in-disease improvement in cutaneous disease activity (CDASI), and was well-tolerated

- Dazukibart is dosed Q4W, IV
- 82% of patients achieved $\geq 40\%$ reduction in CDASI
- Clinically-meaningful TIS trend in muscle-predominant patients



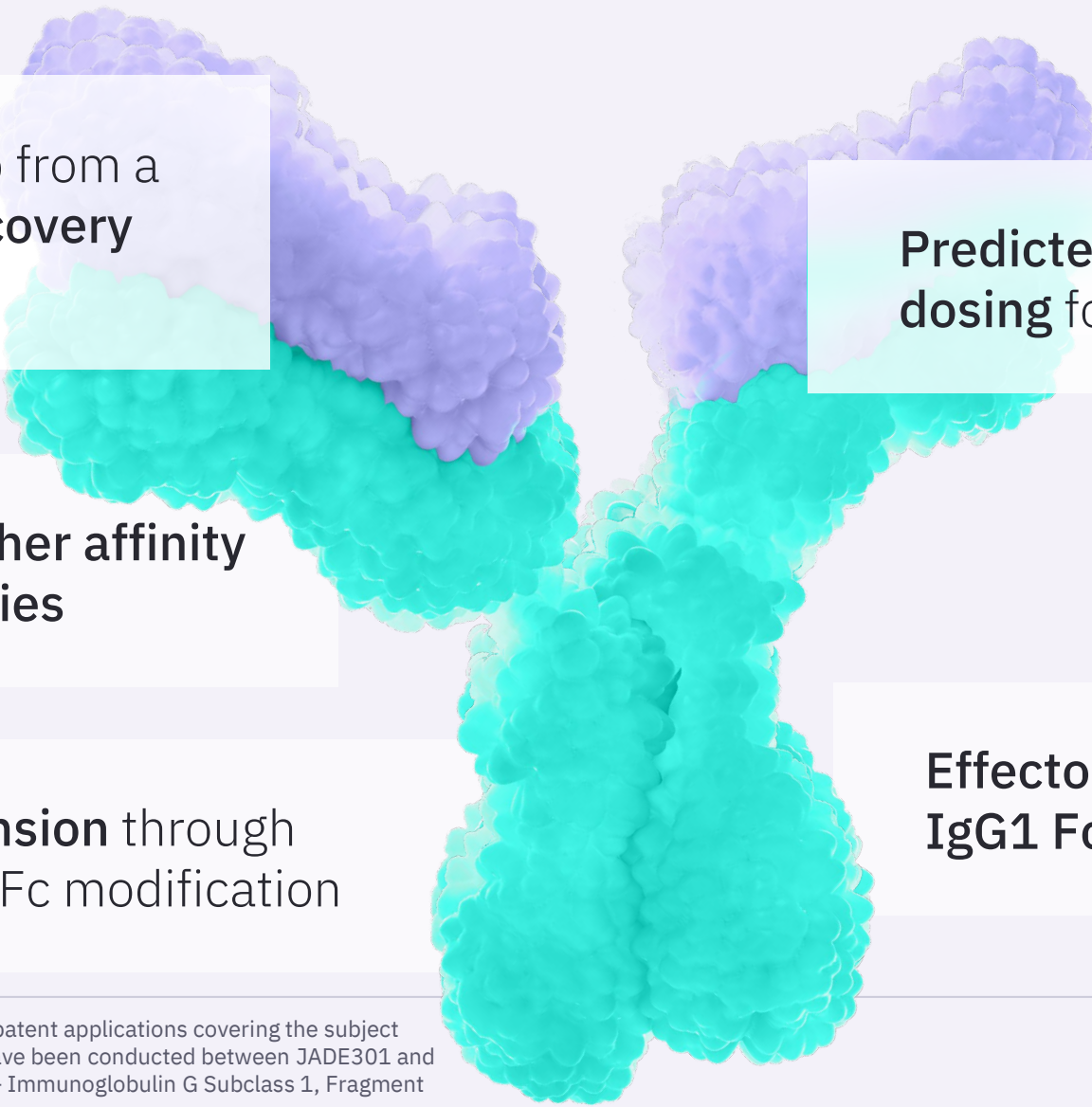
	DAZUKIBART PHASE 2 DATA												
MoA	anti-IFN-β												
Status	Phase 3 ongoing												
Absolute Δ in CDASI-A (cutaneous disease activity, W12)	PLACEBO 150 MG 600 MG <table><thead><tr><th>Group</th><th>Absolute Δ in CDASI-A</th><th>N</th></tr></thead><tbody><tr><td>PLACEBO</td><td>2.9</td><td>14</td></tr><tr><td>150 MG</td><td>16.6</td><td>15</td></tr><tr><td>600 MG</td><td>19.2</td><td>28</td></tr></tbody></table>	Group	Absolute Δ in CDASI-A	N	PLACEBO	2.9	14	150 MG	16.6	15	600 MG	19.2	28
Group	Absolute Δ in CDASI-A	N											
PLACEBO	2.9	14											
150 MG	16.6	15											
600 MG	19.2	28											
Observed Mean TIS (muscle disease improvement, W12)	PLACEBO 600 MG <table><thead><tr><th>Group</th><th>Observed Mean TIS</th><th>N</th></tr></thead><tbody><tr><td>PLACEBO</td><td>36.9</td><td>9</td></tr><tr><td>600 MG</td><td>56.4</td><td>9</td></tr></tbody></table>	Group	Observed Mean TIS	N	PLACEBO	36.9	9	600 MG	56.4	9			
Group	Observed Mean TIS	N											
PLACEBO	36.9	9											
600 MG	56.4	9											
Safety	Well-tolerated												
Dosing	600 mg IV, Q4W (~43% F)												

Sources: 2023 Vleugels (AAD Presentation); 2020 Neelakantan (Clin Pharm); 2022 Aggarwal (NEJM); 2020 Paik (A&R); 2025 Fiorentino D (Lancet); 2025 Fournier (EADV Journal)

MoA – Mechanism of Action; F – Subcutaneous bioavailability; CDASI – Cutaneous Dermatomyositis Disease Area and Severity Index; TIS – Total Improvement Score

Potentially **best-in-class** properties of JADE301

JADE301



Novel anti-**IFN- β** mAb from a *de novo* antibody discovery campaign

Predicted differentiated SC dosing for patient convenience

Binds IFN- β with **higher affinity** than existing therapies

Half-life extension through validated YTE Fc modification

Effector-null fully human IgG1 Fc

Notes: Jade and Jade's licensor, Paragon, have filed patent applications covering the subject matter of JADE301. No head-to-head clinical trials have been conducted between JADE301 and existing therapies. IFN- β – Interferon Beta; IgG1 Fc – Immunoglobulin G Subclass 1, Fragment Crystallizable; SC - Subcutaneous

JADE301 delivered **high-affinity, selective** and **potent IFN- β neutralization** in preclinical studies

SPR Binding to Recombinant Human IFN- β

JADE301
 $K_D = 11.3$ pM

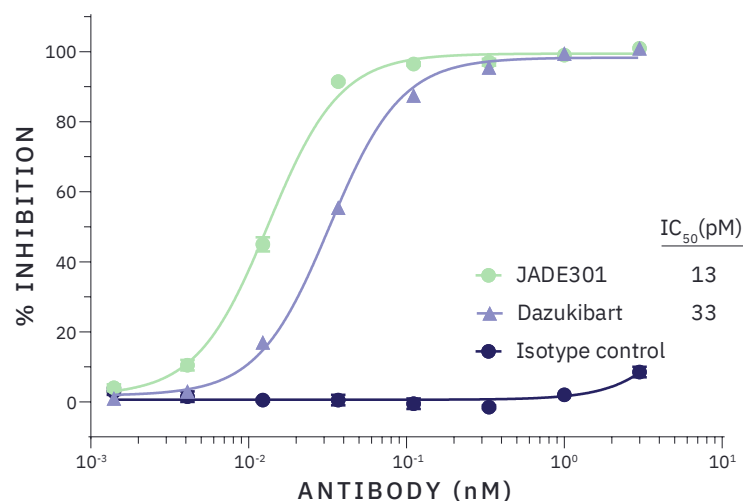
DAZUKIBART*
 $K_D = 28.1$ pM

BINDING SPECIFICITY (ELISA)

CYTOKINE	BINDING
IFN- α 1	Not detected
IFN- α 2a	Not detected
IFN- ω 1	Not detected
IFN- γ	Not detected

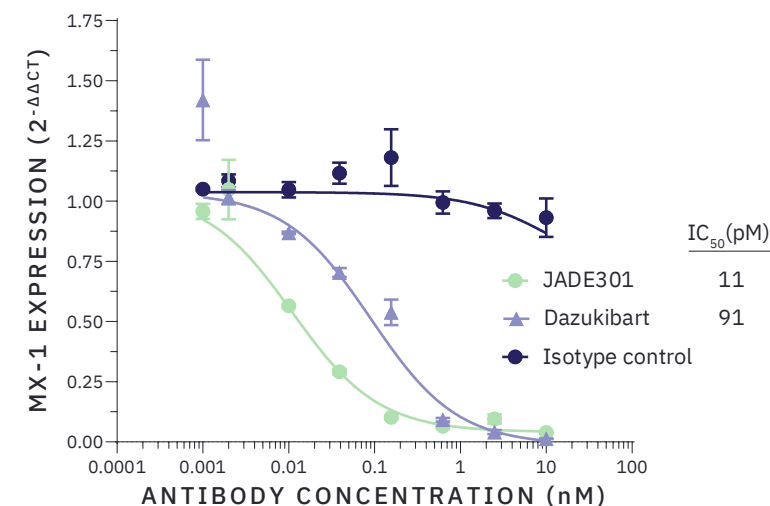
~2.5x higher binding affinity than dazukibart*, with no observed off-target IFN binding

Inhibition of IFN- β induced pSTAT



~2.5x higher potency than dazukibart*

Inhibition of IFN- β induced MX-1



~8x more potent IFN pathway suppression in disease-relevant skin cells than dazukibart*

Notes: Binding affinity measured by SPR; functional potency assessed by suppression of IFN- β induced pSTAT in a human immune cell line and MX-1 in human dermal fibroblasts.

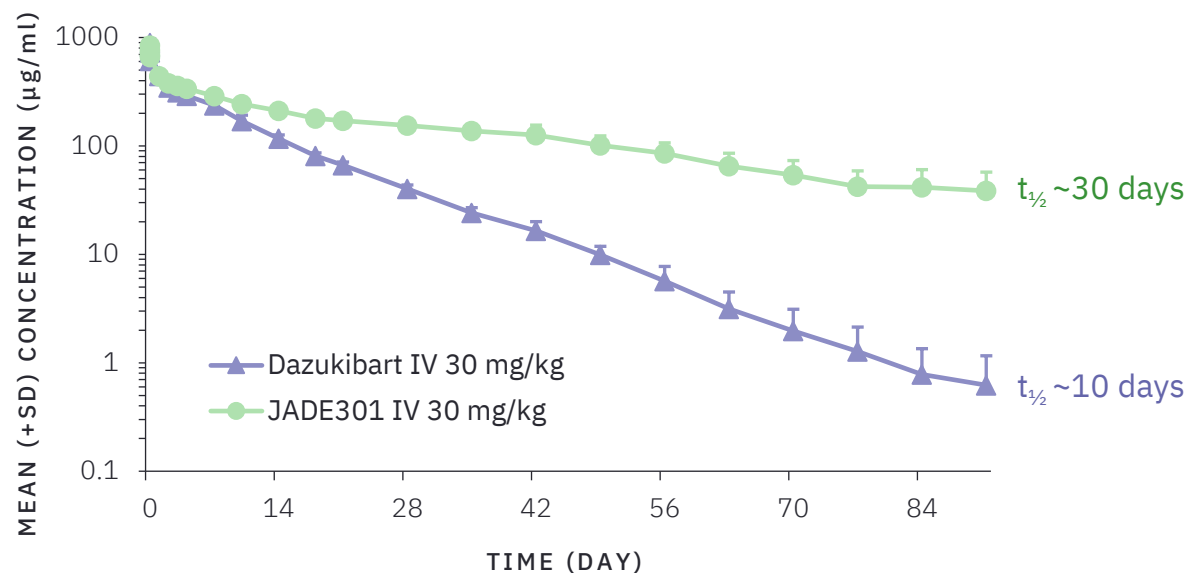
*Dazukibart comparator generated from publicly available sequence.

K_D - Dissociation Constant; pM – Picomolar; IFN- β – Interferon Beta; pSTAT - Phosphorylated Signal Transducer and Activator of Transcription; MX-1 - Myxovirus Resistance Protein 1

JADE301 showed extended half-life and favorable SC bioavailability in NHPs

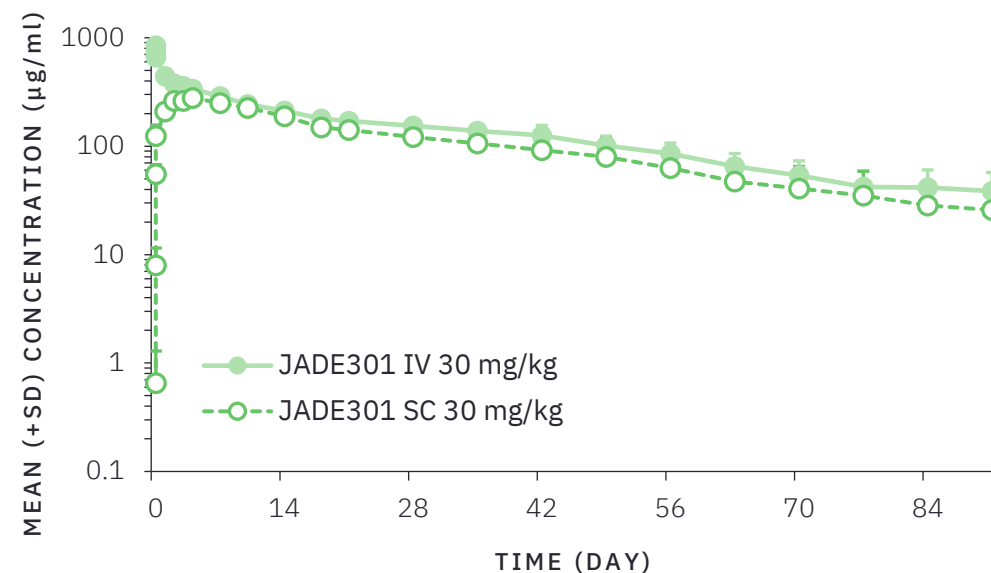
Preclinical data supports clinical evaluation of infrequent SC dosing

~3× longer IV half-life vs. dazukibart comparator



~30-day IV half-life in NHPs, driven by ~3-fold lower clearance

Favorable PK following SC administration



Enabled SC GLP NHP toxicology studies to support SC FIH

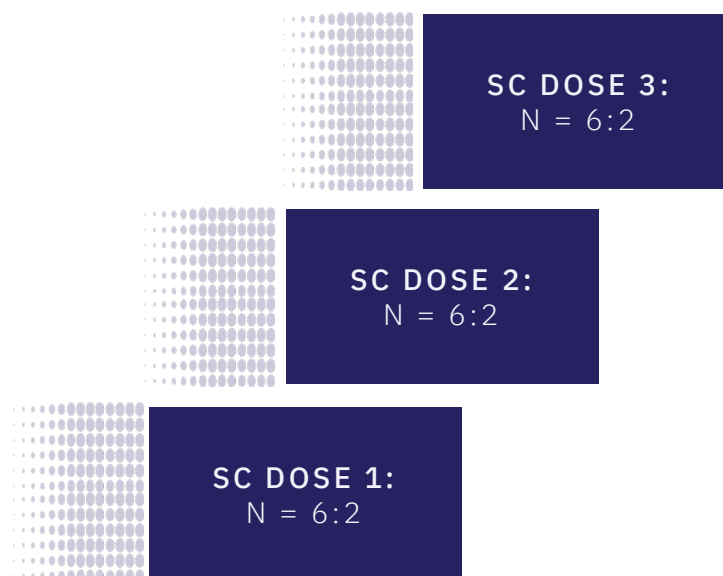
JADE301 **Phase 1 healthy volunteer study** expected to begin Q4 2026; interim data anticipated in 2H 2027

Phase 1 HV study designed to generate key data supporting JADE301's potential for **best-in-disease** clinical activity and convenient SC dosing

Phase 1 Study Design

Placebo-controlled, single ascending-dose study

SC administration in healthy volunteers (N=24)



Objectives

PRIMARY

- Safety and tolerability

SECONDARY & EXPLORATORY

- Pharmacokinetics
- Immunogenicity

Advancing **potentially best-in-class therapies** for autoimmune diseases

Well-funded through multiple upcoming milestones; runway into the fourth quarter of 2028

Candidates designed to **maximize clinical activity** and allow **patient friendly, infrequent dosing**

PROGRAM	MOA	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	POTENTIAL INDICATIONS
JADE101	anti-APRIL					IgAN
JADE201	anti-BAFF-R					Multiple systemic AI diseases
JADE301	anti-IFN- β					Dermatomyositis

Development candidates from Paragon

Expected Milestones:

JADE101

- Interim Phase 2 Data: 2027
- Phase 3 Initiation: 1H 2027

JADE201

- Interim Phase 1 Data: 2027

JADE301

- Phase 1 Initiation: 4Q 2026
- Interim Phase 1 Data: 2H 2027

Notes: Jade has entered into exclusive license agreements with Paragon Therapeutics for JADE101, JADE201 and JADE301.

AI – Autoimmune; IgAN - IgA nephropathy; BAFF-R – B cell-Activating Factor Receptor; IFN-B – Interferon Beta; MoA – Mechanism of Action

Current capitalization

Common stock	Shares outstanding	63,556,065
Common stock equivalents	Preferred stock (as converted to common stock)	12,622,000
	Pre-funded warrants	7,715,189
Common stock & common stock equivalents	Total outstanding	83,893,254

Thank you

JADEBIOSCIENCES.COM

INFO@JADEBIOSCIENCES.COM

NASDAQ: JBIO

