Kira PHARMA

JASPER THERAPEUTICS

Corporate Presentation

September 2026

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Overview of the Merger Transaction and Financing

Merger creates a combined company with deep expertise in antibody drug development and immunologically-driven disorders

Merger

- On July 16, 2026, Jasper Therapeutics, Inc. (NASDAQ: JSPR) entered into a merger agreement with Kira Pharmaceuticals whereby Jasper acquired all outstanding shares of Kira in in an all-stock transaction

Concurrent $132M PIPE offering closed with use of proceeds focused on key value drivers

- Vensobafusp alfa - Stage 1 and Stage 2 data in rare renal disorders (IgAN, C3G, FSGS, others)
- Vensobafusp alfa - Ph. 3 initiation in PNH
- Vensobafusp alfa – Additional CMC and R&D
- Briquilimab pre-BLA meeting in SCID
- KP701 Ph. 1b data / Ph. 2 initiation in B-cell mediated diseases
- **Strong balance sheet expected to fund multiple clinical milestones and operating plan well into 2H28**

Mirador Out-License

- Kira out-licensed KP301, a preclinical long-acting anti-C5a monoclonal antibody, and KP402, a small molecule C5a receptor antagonist, to Mirador Therapeutics
- The out-licensing transaction provided an upfront payment of $12M, and potential development and sales milestone payments

The Combined Company Plans to Deliver Innovative Therapeutics in Multiple Immune-Driven Disorders

Significant Global Opportunity

- Large, addressable markets across complement, mast-cell and B-cell mediated disorders[1]

Vensobafusp alfa KP104 (C5 + Factor H)

- **Phase 2/3 ready in PNH, Phase 2 ongoing in renal disorders**
- Innovative, bifunctional biologic targeting both alternative & terminal pathways
- 2 year clinical data demonstrate rapid restoration of hemoglobin and 100% transfusion free outcomes
- Kira's China subsidiary fully supports the efficient and quality execution of ongoing Ph2 renal trial

Briquilimab (anti-KIT)

- **Potential BLA path in SCID conditioning and broad optionality in large mast cell / allergy markets**
- Orphan Drug & Fast Track Designation + Rare Pediatric Disease Designation in SCID
- Phase 1b/2a CSU study complete, opportunity in other mast-cell mediated disorders

Broad Portfolio

- **KP701** (long-acting CD79BxCD32B) – potential best-in-class, non-depleting B-cell targeting therapy for large chronic diseases, CTA/IND planned for 2027
- Multiple other programs targeting validated targets

Experienced Leadership Team

- The Combined Company is led by a core team of experienced biopharma industry veterans, seeking to address critical unmet need in immunologically-driven disorders

1) Evaluate Pharma annual worldwide sales estimates

Jasper Executive Team

Experienced drug developers focused on driving value

Jeet Mahal
Chief Executive Officer

Biotech executive and Board member with 30+ years of experience in development & commercialization

JASPER THERAPEUTICS · celito · PORTOLA · Johnson&Johnson

Herb Cross
Chief Financial Officer

Biotech executive with extensive experience in leadership roles at publicly traded biotech companies

JASPER THERAPEUTICS · ARMO BIOSCIENCES · ATRECA · AFFYMAX · KaloBios

Greg Keenan, MD
Chief Medical Officer

Rheumatologist, antibody drug developer & former CMO at publicly traded biotechs

Perelman School of Medicine University of Pennsylvania · MedImmune · Aurinia · AstraZeneca · VALENZABio

Matthew Ros
Chief Operating Officer

Former COO / CFO and Board member at publicly traded pharma companies

cogent BIOSCIENCES · VERASTEM ONCOLOGY · Fore · Epizyme · ARIAD · Bristol Myers Squibb · sanofi

Wenru Song, MD, PhD
Executive Vice President and Head of R&D

Ex-VP level clinical positions at large pharma companies

AstraZeneca · Takeda · Pfizer · Stanford MEDICINE

Innovative Pipeline Targeting High Value Immunology Targets

	Mechanism of Action	Indication	IND	Phase 1	Phase 2	Phase 3	Expected Upcoming Milestones
Vensobafusp alfa (KP104)	Anti-C5 mAb + Factor H Bifunctional Biologic	IgAN, C3G, FSGS	Phase 2 Basket Trial				**Q4 '26:** Interim data (Stage 1) **Q2 '27:** Updated data (Stage 1) **Q2 '27:** Interim data (Stage 2)
		PNH	Phase 3 Ready				**1H '27:** EOP2 FDA meeting
		New Indication	Phase 2 Ready				**Q4 '26:** Phase 2 plans
Briquilimab	Anti-KIT	SCID Conditioning	Pre-BLA Discussion				**Q4 '26:** Update on SCID program **Q1 '27:** Receive regulatory guidance
		Mast-Cell Mediated Diseases	Phase 2 Ready				**2H '26:** Update on next steps
KP701	Anti-CD79BxCD32B	Autoantibody-Mediated Disorders	Phase 1 Ready				**Q1 '27:** File Phase 1 CTA/IND **Q3 '27:** Phase 1a HV data
Discovery	Long-Acting Complement Targeted Biologics	Autoimmune Inflammatory Disorders					**Mid '27:** DC selection

All assets have global rights & extensive IP portfolios

IgAN; IgA Nephropathy. C3G; Complement 3 Glomerulopathy. FSGS; Focal Segmental Glomerulosclerosis. PNH; Paroxysmal nocturnal hemoglobinuria. SCID; Severe Combined Immunodeficiency.

Vensobafusp alfa (KP104)

Anti-C5 mAb + Factor H Bifunctional Biologic

Vensobafusp alfa: Phase 2/3 Ready, Potential Best-in-Class Complement Inhibitor

Vensobafusp Phase 2/3-ready

- Phase 2 PNH study complete with 2-year outcomes data
- 89% of patients with normal hemoglobin & 100% transfusion free
- PNH EoP2 planned 1H27
- Upcoming data from Renal Basket Study in Q4'26 and Q2'27

Dual Mechanism of Action:

- Simultaneous blockade of the alternative and terminal pathways – addressing both **upstream** activation and **downstream** lytic damage

Validated Rationale:

- Single alternative OR terminal pathway inhibitors are first line therapy across multiple hematological and renal disorders (PNH, IgAN, C3G, FSGS, aHUS)
- Significant efficacy or safety limitations with single pathway inhibitors

Translational Strategy:

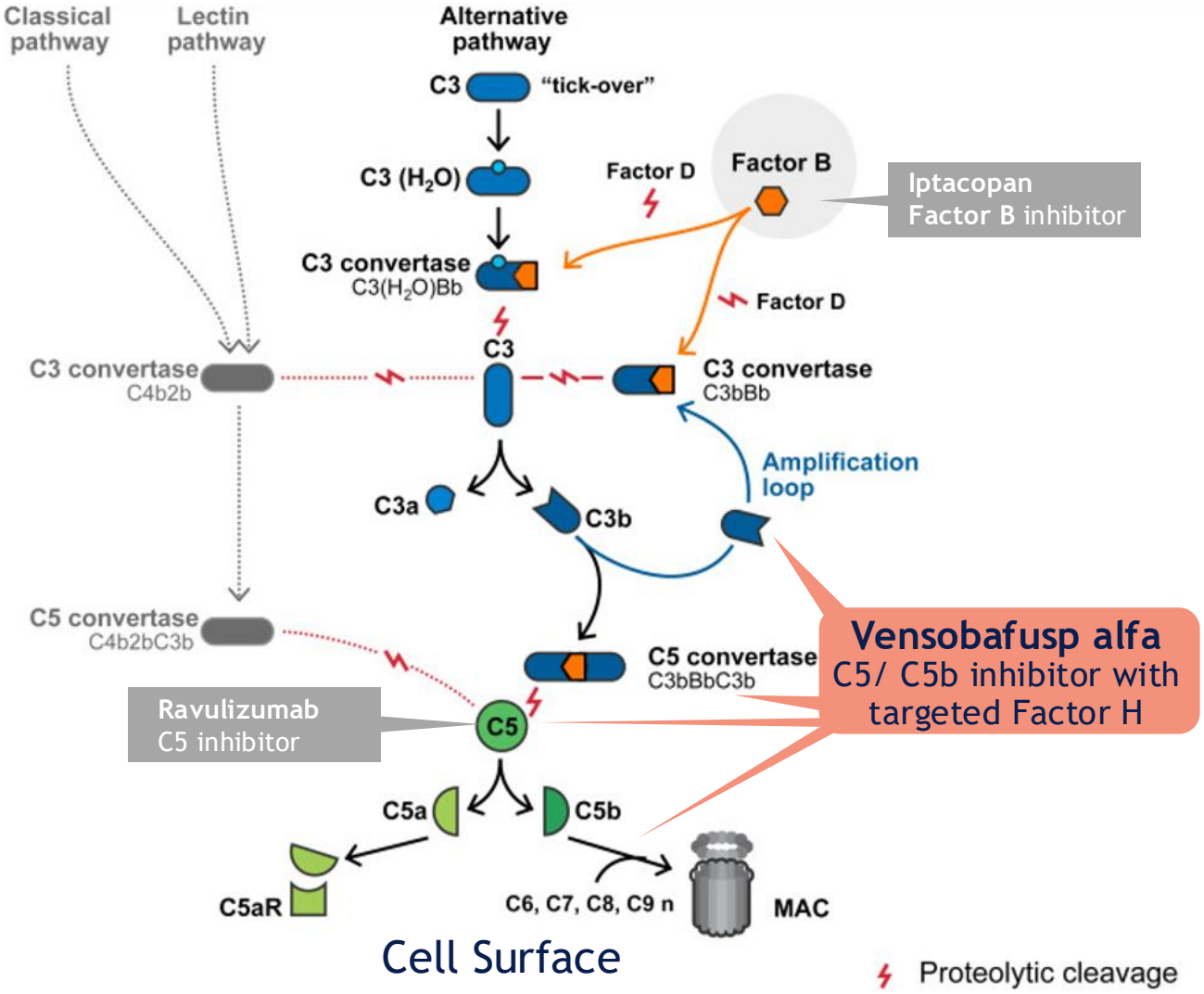
- Phase 2 PNH study complete
- Ongoing Phase 2 renal basket study will provide clinical data across multiple high-unmet-need nephrology indications

A novel monoclonal antibody with promising Phase 2 clinical data in PNH and broad potential in nephrology

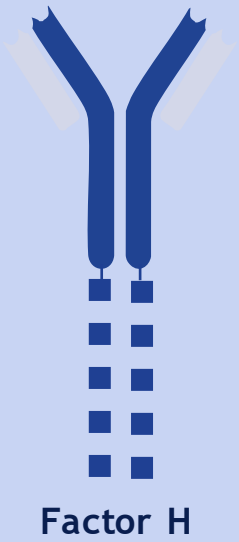
Vensobafusp alfa: Dual Mechanism of Action for Increased Potency and Rapid Onset

PROXIMAL (ALTERNATIVE)

TERMINAL



Vensobafusp alfa: Dual-targeting of Terminal (C5) and Alternative (Factor H) Pathways



- Unique **C5- and C5b-binding** epitope preventing MAC formation and concentrates drug to affected cell surfaces
- **Factor H** modulates alternative pathway amplification, rather than fully blocking (e.g., Factor B MOA)
- A more fine-tuned approach aims to reduce systemic infection liability and spare complement function needed to preserve host defense

Vensobafusp alfa: Favorable Efficacy Profile in PNH Patients at 48 Weeks

PNH: Phase 2 results (48 weeks)

100%	Hgb ≥2g/dl increase from baseline
6.6 g/dL	Hgb increase from baseline
89%	Hgb normalization (≥12 g/dL)
94%	LDH <1.5x ULN (with near normal LDH)
100%	Transfusion avoidance
100%	Clinically significant improvement in QoL

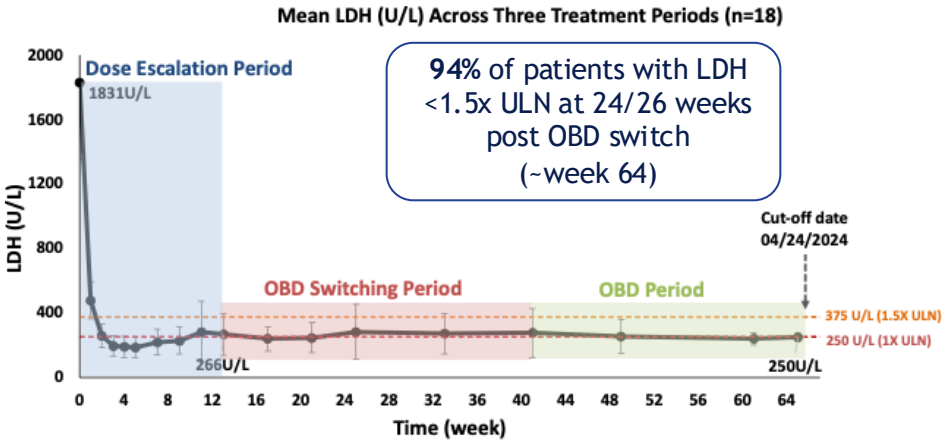
Confirming long-term effects in C5 naïve PNH patients

- Consistent and favorable safety profile
- Improved and sustained clinical responses at OBD treatment
- Demonstrated effectiveness in controlling IVH and EVH

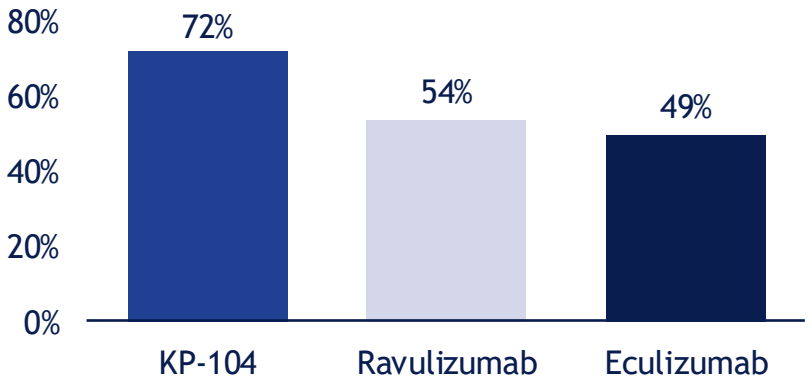
Strong clinical data supporting potential standard of care

- Potent, potential first-in-class bifunctional complement inhibitor
- Potential new first-line monotherapy for PNH
- Favorable efficacy and safety profile

Rapid normalization of LDH support longer term clinical benefit correlation of thrombosis reduction



% Patients with LDH <1x ULN at 24/26 Weeks[1]

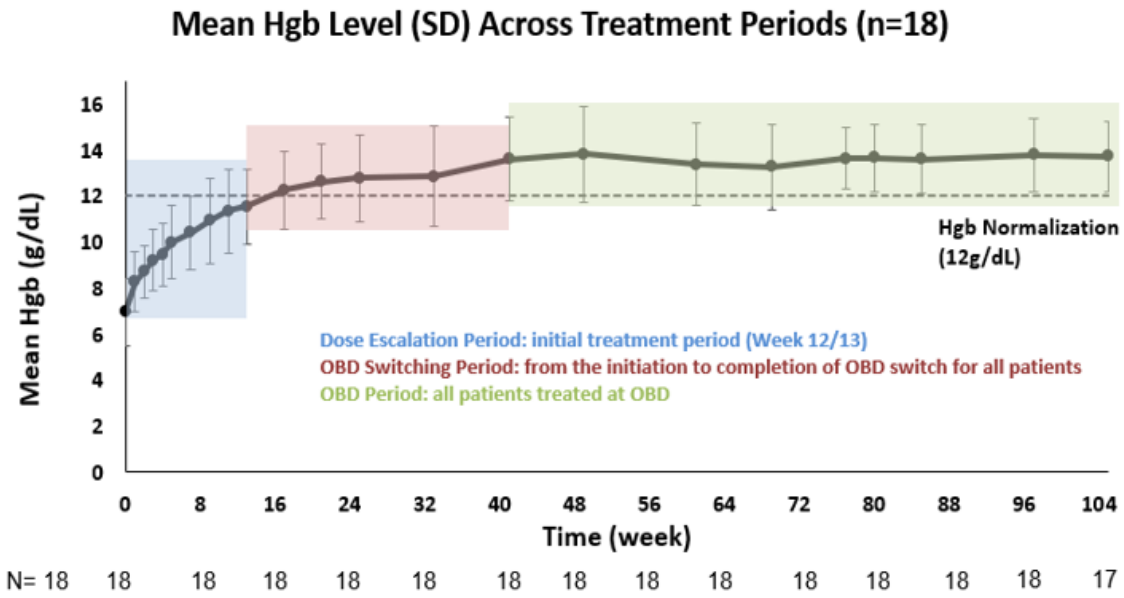


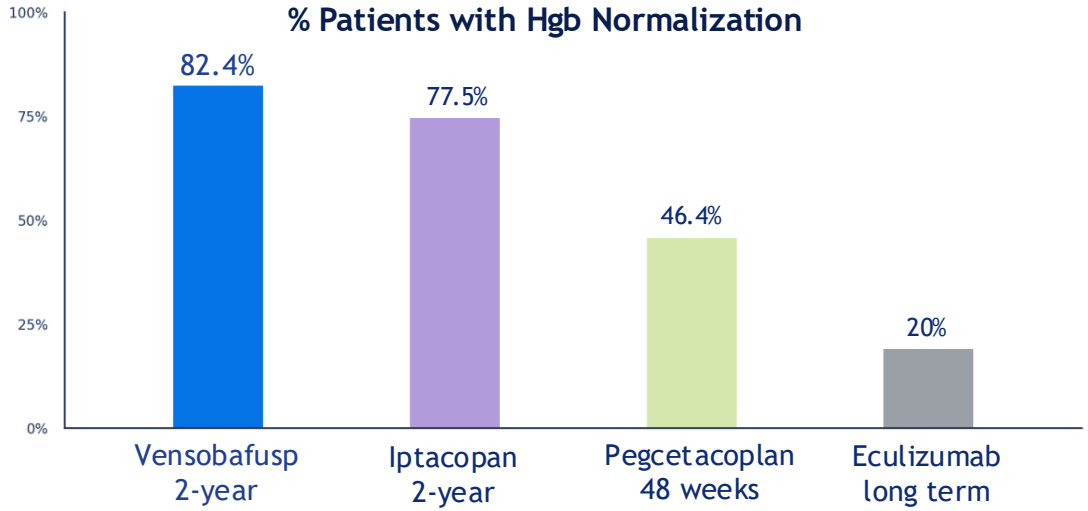
1) Ravulizumab and eculizumab data from Lee, J. et al. Blood (2019) 133 (6): 530-539, Doi.org/10.1182/blood-2018-09-876136
OBD: Optimal biologic dose

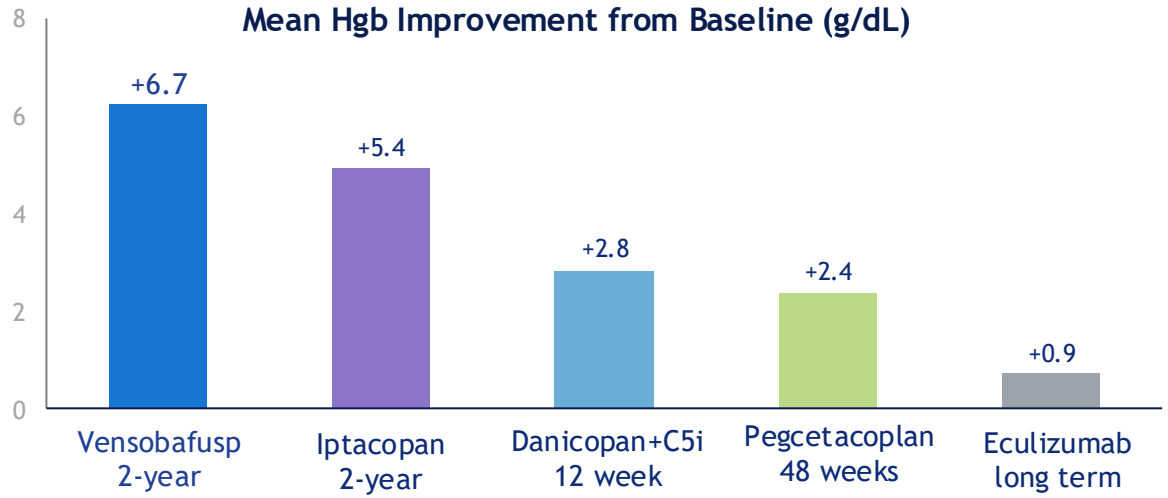
Vensobafusp alfa: Dual Inhibition Drives Durable Hgb Improvement

Hgb Normalization in 89% of Patients at 2 Years

Sustained Hgb Improvement Reflects Controls of Both IVH and EVH







Sources: KP104: ASH 2025 abstract, KP104-201 Phase 2 2-year update. Iptacopan: EHA 2025 poster PF660, APPOINT-PNH REP 2-year follow-up. Pegcetacoplan: 307 Open Label Extension Study 48 weeks

Vensobafusp alfa: Basket Study to Explore Rare Renal Indications

Multiple Opportunities to Achieve Rapid POC

Opportunity to demonstrate vensobafusp's best-in-class potential and differentiated profile in renal indications vs. other complement inhibitors

IgAN

- IgAN pathogenesis involves both alternative pathway and lectin pathway complement

C3G

- Vensobafusp demonstrated best-in-class potential, with improvements observed in renal function and pathology including C3 and C9 glomeruli deposition elimination compared to C5 mAb alone

FSGS and Other Renal Disorders

- Robust in vitro and in vivo data supporting multiple complement pathway in FSGS
- Opportunity to expand into other renal disorders

Vensobafusp alfa: Ongoing Phase 2 Renal Basket Study

Designed to Evaluate Safety, PK/PD and Efficacy in Multiple Renal Indications

Stage 1
- Assess the safety, tolerability and initial efficacy in IgAN
- 3 dose intervals / levels to assess safety and determine optimal dose
- Initial interim data in by YE ‘26

Stage 2
- Protocol designed to easily add new renal disease cohorts at existing clinical sites
- IgAN study expansion with optimal dose (n=30)
- Clinical data in multiple populations in 1H ’27

Stage 1 – IgAN PoC

- IV Load + Q2W (n=6)
- IV Load + Q4W (n=6)
- IV Load + Q8W (n=6)

Key Endpoints: UPCR, eGFR, PK, PD (multiple), TEAEs

Stage 2 - Expansion

- IgAN Expansion (n=30)
- C3G
- FSGS
- New Indication

Vensobafusp alfa: Multiple Indication Expansion Opportunities Across Hematology, Renal and Neurology Disease Areas

Indication Opportunities

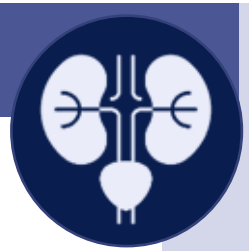
Hematology

- **Paroxysmal Nocturnal Hemoglobinuria (PNH) – Phase 2 Complete**
- Complement-Mediated Thrombotic Microangiopathy (CM-TMA)
- Atypical Hemolytic Uremic Syndrome (aHUS)

Estimated Market Opportunity:

PNH[1]: 28K patients
aHUS[1]: 9K patients
(US, EU5, Japan & China)

Renal



- **IgA Nephropathy (IgAN) – Ongoing Clinical Study**
- **C3 Glomerulopathy (C3G) – Potential Best-in-Class Profile**
- **Focal Segmental Glomerulosclerosis (FSGS)**
- Immune Complex-Mediated Membranoproliferative Glomerulonephritis (IC-MPGN)
- **Diabetic Kidney Disease (DKD)**
- **Other renal disorders under consideration**

IgAN[2]: 27k patients
C3G[2]: 4.3k patients
FSGS[2]: 6.5k patients
>$9B US market opportunity[2]

Other

- **Neurology: Neuromyelitis Optica Spectrum Disorder (NMOSD),**
- **Generalized (ACHR+) Myasthenia Gravis (gMG)**

gMG[1]: 260K patients
NMOSD[1]: 25K patients
(US, EU5, Japan & China)

1) Alexion R&D Day 2024
2) Novartis Renal Portfolio Investor Event and Target Patient Population 2023, Cantor KOL estimates, LifeSci Vera Therapeutics model

Briquilimab

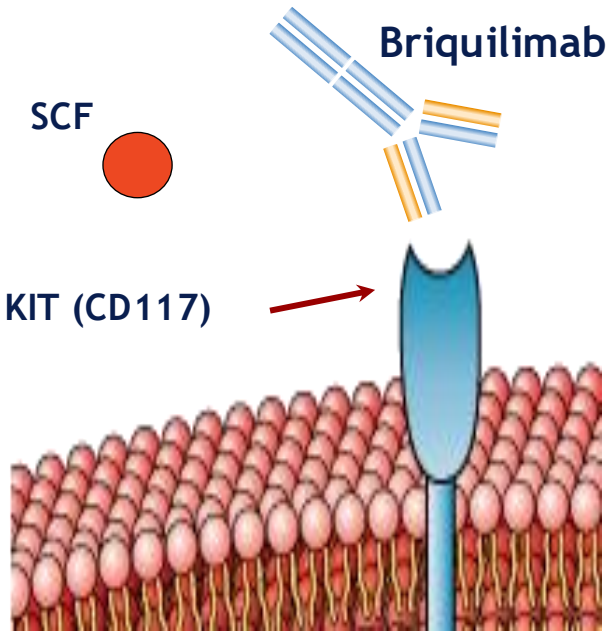
Potential Best-in-Class Anti-KIT Antibody

Briquilimab: Broad Therapeutic Potential Across Transplant & Immunology

Update on SCID program in Q4'26 & regulatory feedback expected in Q1'27

Briquilimab

Blocks SCF binding to KIT (CD117) to directly inhibit receptor signaling



Validated Mechanism of Action

- Briquilimab is designed to directly block SCF from binding to KIT (CD117) with high affinity and avidity
- Aglycoslyated IgG1 antibody directly inhibits stem cell factor from binding to the KIT receptor on mast and stem cells
- Inhibition of SCF signaling leads to depletion of mast cells in the skin and migration of hematopoietic stem cells from the bone marrow
- Wide therapeutic potential across a range of mast and stem cell-mediated diseases

Favorable Drug Properties

- Kd < 5pM affinity to human KIT with IC50 ~ 70pM
- Human mast cell survival bioassay IC50 ~12.5nM
- No Fc mediated ADCC or complement mediated cytotoxicity which reduces risk of adverse effects
- Human clinical data as IV or Sub-Q delivery

Encouraging Clinical Profile

- Predictable clearance from ages 3 to 79
- Demonstrated single agent lasting depletion of mast cells
- Demonstrated efficacy in multiple stem cell transplant and mast cell mediated disease studies
- Favorable safety profile demonstrated in over 200 clinical participants

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Briquilimab: A Differentiated Program in Transplant & Allergic Disorders

SCID, Fanconi, Sickle Cell and AML / MDS: N = ~40

- Positive, long-term data supporting use of Briquilimab for conditioning in HSCT
- **Results show successful donor chimerism and reconstitution of hematopoietic and immune systems**
- Favorable chronic safety profile with potential for differentiation
- Potential BLA filing in SCID
  - Rare Pediatric Disease Designation
  - Discuss path to BLA with FDA with data from first twelve patients

BEACON TRIAL + Open-Label Extension

CSU: N = 87
H1-AH[1] failed

- Deep disease control with ΔUAS7>25 in multiple cohorts
- Rapid onset with CRs observed as early as week 2
- >50% CR rate at 4 weeks post-dose in multiple dose cohorts[2]
- Favorable chronic safety profile with potential for differentiation
- Phase 2b protocol submitted to FDA

SPOTLIGHT TRIAL + Open-Label Extension

CIndU: N = 27
H1-AH failed (ColdU and SD)

- 92% CRs with 180mg single dose
- 67% CR by week 2 (180mg SD)
- 65% CR / WC maintained with 180mg Q8W in open-label extension
- Favorable safety profile observed

Kira PHARMA JASPER THERAPEUTICS

1) H1-AH: H1 receptor targeted antihistamine
2) Measured 4 week after initial dose for single dose patients and 4 weeks post-dose for patients dosed Q8W or Q12W

Briquilimab: Potential US Licensing Pathway & PRV Opportunity for SCID

SCID and Fanconi anemia are difficult to treat ultra-orphan disorders where HCT is the only proven cure, but challenging setting due to limited pre-conditioning options

Multiple Potential FDA Filing Strategies

SCID

- SCID re-transplant patients are ultra orphan, high unmet need population
- Focused on data with Briquilimab in 11 SCID re-transplant T-B- patients
- Immune reconstitution (chimerism, naïve T-cells)
- Function immunity (reduction of IVIG, infections, response to vaccination)
- Additional longitudinal data in existing patients and separate natural history data, 5 years follow up data
- Potential approval path could be similar to Rocket Pharmaceuticals recent approval of KRESLADI

Fanconi Anemia

- Allogeneic stem cell transplant can restore bone marrow and blood formation in Fanconi anemia patients
- Development strategy:
- Discuss path to BLA with FDA with 2 yr-data in first three patients
- Consider expansion to additional clinical sites following FDA discussion
- Successful development of Briquilimab in Fanconi anemia could lead to rare pediatric disease designation and a Priority Review Voucher

Briquilimab granted Rare Pediatric Disease Designation in SCID & may be eligible for Priority Review Voucher (PRV) on approval

Briquilimab: Rapid and Deep Control of Chronic Spontaneous Urticaria

Target Product Profile

Potency

- Direct blockade of SCF binding site
- High affinity c-Kit (K_d <5pM) and potency (IC50 70pM)

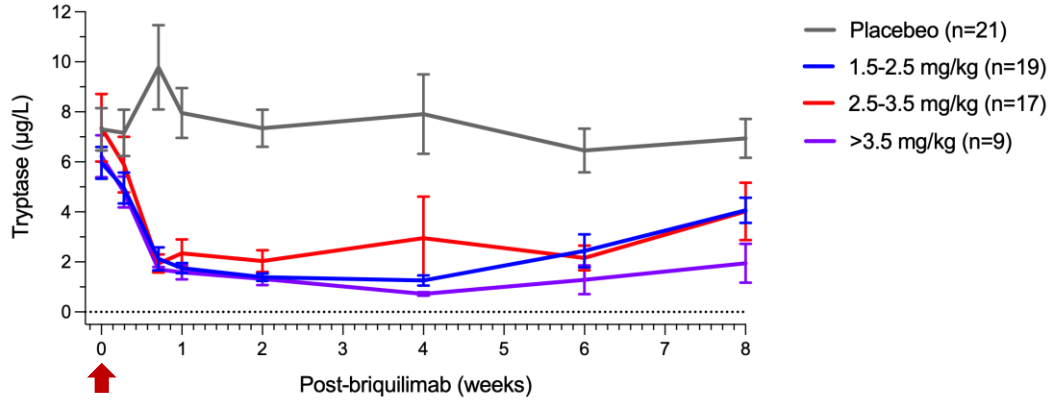
Speed

- Rapid T_{max} and high C_{max}
- Over 50% Complete Responses (CRs) and >25pt UAS7 reduction by week 4 in multiple cohorts

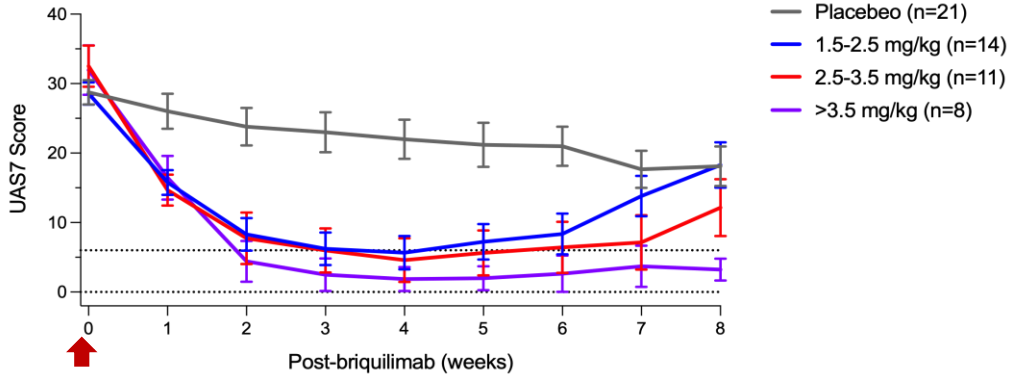
Clearance

- Nine-day subcutaneous half life
- Allows restoration of KIT signaling between doses to minimize unwanted hair & skin effects

Briquilimab rapidly suppresses serum tryptase by week 1 and maintains suppression to week 8 with single dose (BEACON)



Briquilimab rapidly leads to complete response that is maintained through 8 weeks with single dose (BEACON)

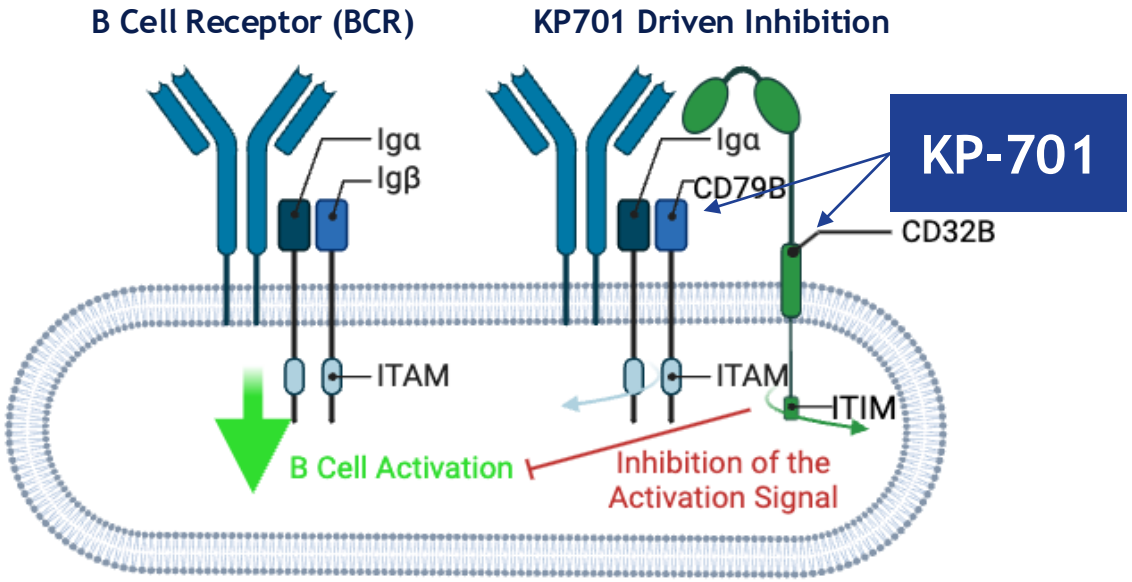


KP701

Potentially Best-in-Class Anti-CD79BxCD32B

KP701: Dual Engagement of CD79B and CD32B to Drive B-cell Tolerance

KP701 Mimics Endogenous B Cell Inhibition Pathway by Cross Linking CD79B (BCR) and CD32B



- New approach to silence pathogenic B-cells/ plasmablasts without deep depletion which leads to significant increased risk of infections (e.g., Rituxan)
- Positive PoC with Obexelimab (CD19xCD32B) in Ph3 study of IgG4-RD with once weekly dosing
- KP701 potentially more potent (CD79BxCD32B)MOA suppresses B-cell function, lowers cytokine and autoantibody production in preclinical and NHP models
- CTA ready with efficient path to proof-of-concept in HV and autoimmune disease patients
- Once-monthly dosing potential

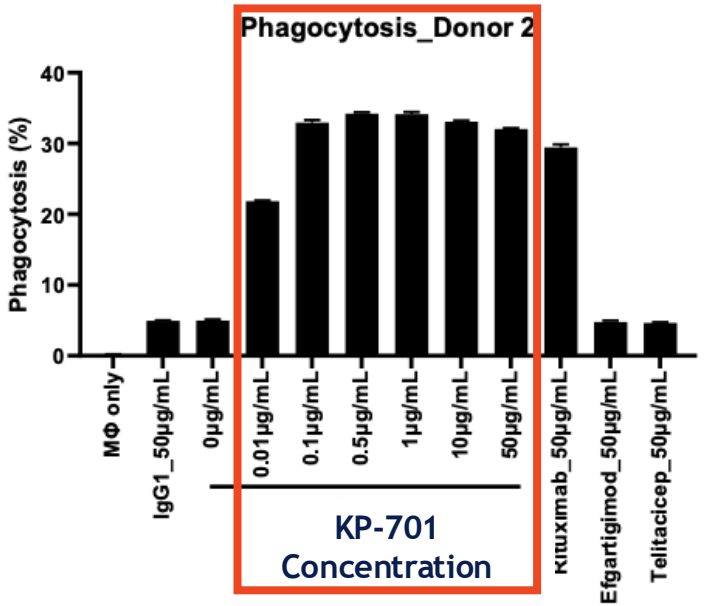
KP701 has shown a strong inhibitory profile against B cells, including antibody production and proliferation without ADCC / CDC or TDCC

Source: O'Neil et al. 2011. Monophosphorylation of CD79a and CD79b ITAM motifs initiates a SHIP-1 phosphatase-mediated inhibitory signaling cascade required for B-cell anergy

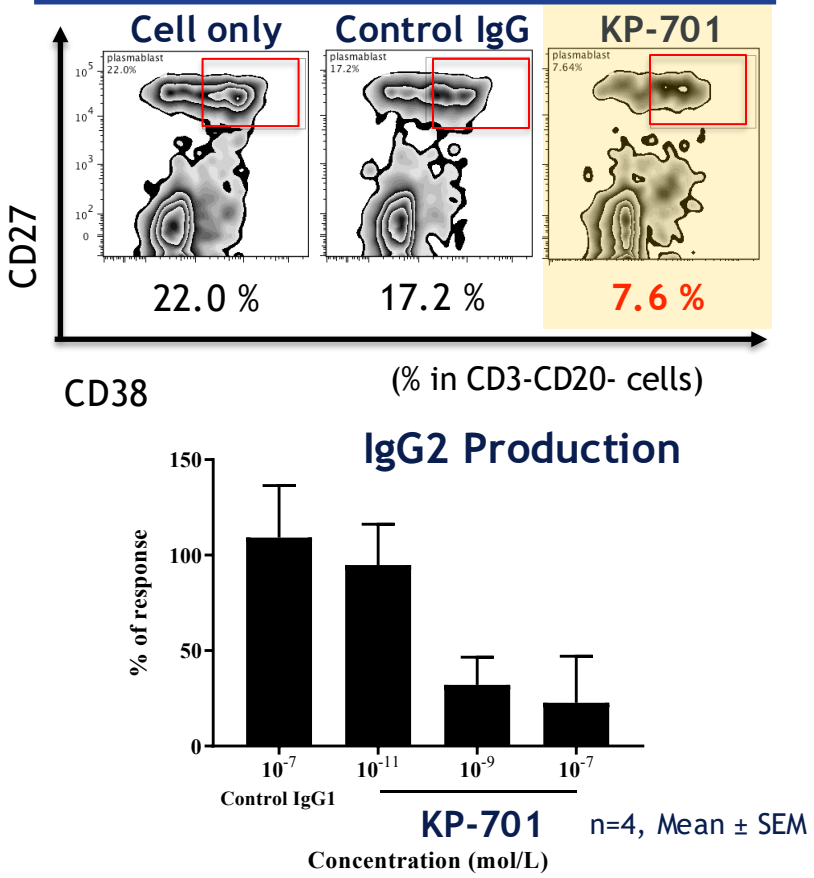
KP-701: Potent Effects on B-cell Proliferation & Phagocytosis vs. Existing Programs

Superior potency observed compared to Obexilimiab, PRV3279 and Rituximab

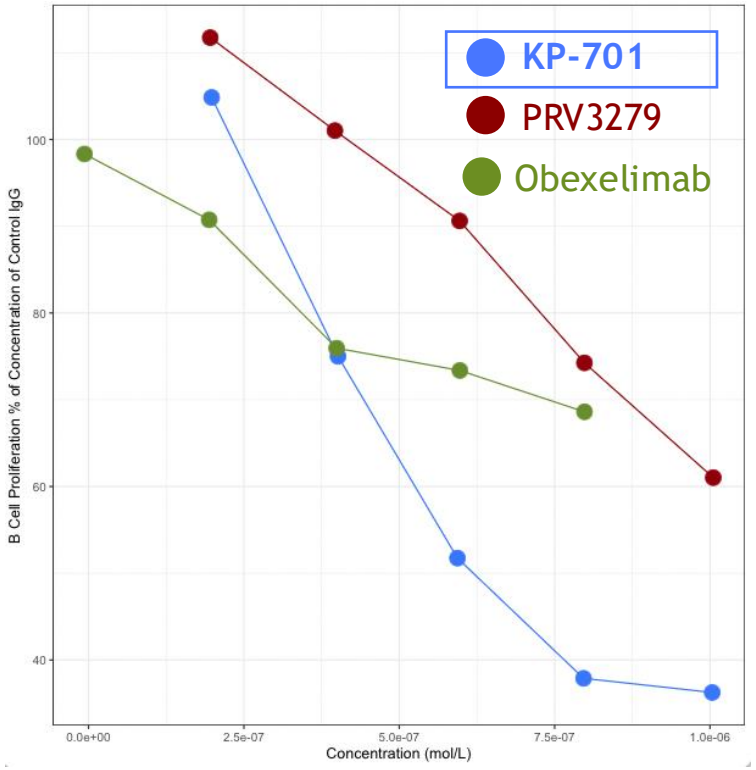
Rapid Phagocytosis of B cells at Low Concentrations vs. Existing B-cell Agents



KP-701 Activity on SLE Plasmablasts & IgG2



KP-701 Superior Activity in B-cell Proliferation Assay*

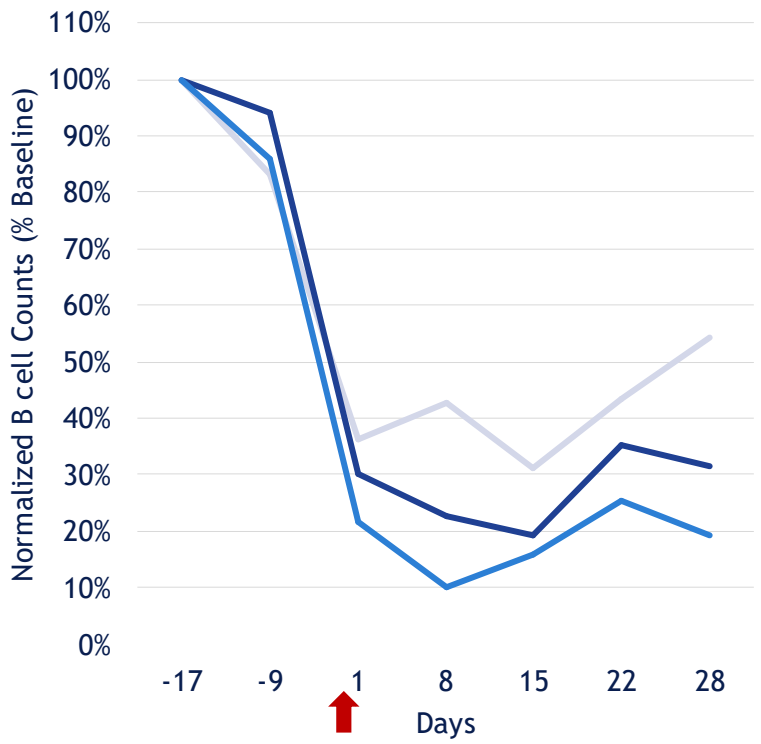


Source: Internal data, Xencor and Macrogenics publications
*Head-to-Head Study

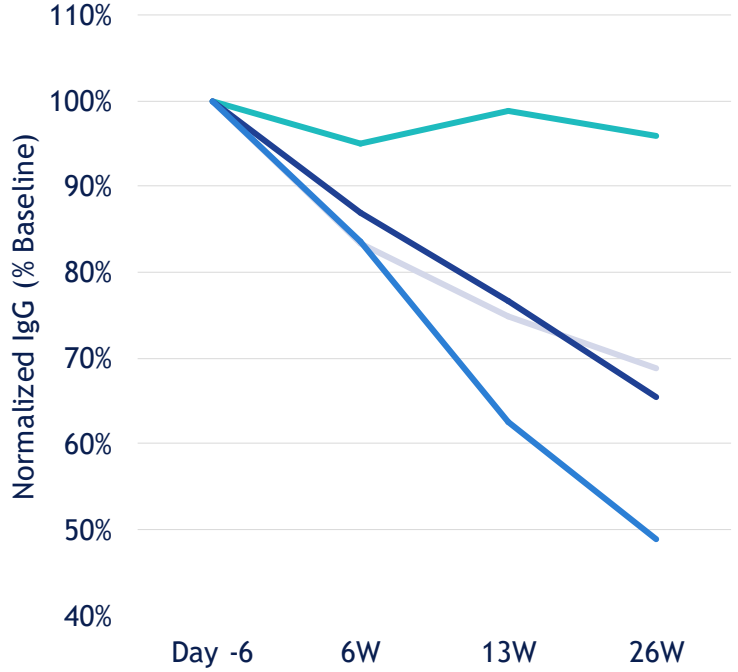
KP-701: Unique Reductions in B Cells & Immunoglobulins (IgG & IgA) in NHPs

Strong reductions in IgG, IgA and B cells

Reduction of CD3-CD20+ in Peripheral Blood

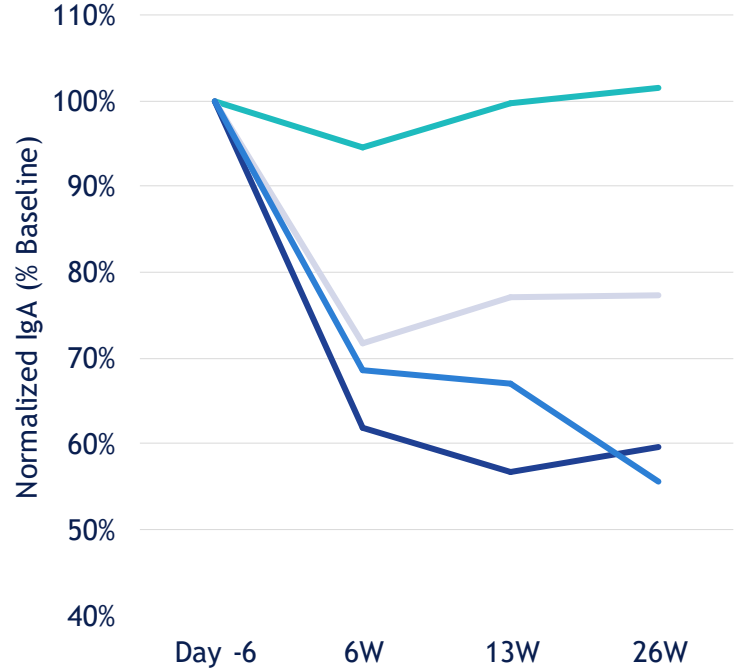


IgG



R_x= Q1W for 26 Wks

IgA



R_x= Q1W for 26 Wks

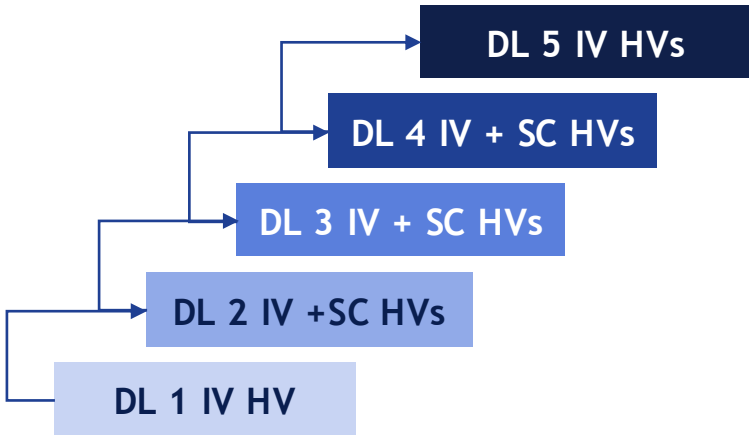
Control 1 mpk 3 mpk 30 mpk

Source: Internal data

KP 701: Streamlined Phase 1a/b Strategy to Deliver Rapid Proof-of-Concept

Opportunity to Evaluate Safety, PK / PD & Preliminary Activity in Autoimmune Disease (AID)

Phase 1a - SAD



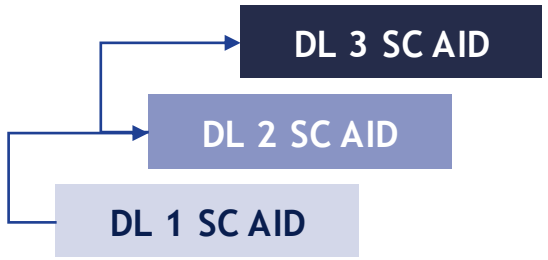
PK Lead-In:

- Patients randomized; 4 patients in 3:1 ratio of drug to placebo

Primary Endpoints: TEAEs, PK, PD (B-cell depletion / recovery), Ig's

Exploratory Endpoints: TBD

Phase 1b - MAD



SC PKPD & POC:

- Patients randomized patients in 3:1 ratio of drug to placebo

Primary Endpoints: TEAEs, PK, PD (B-cell depletion / recovery), Ig's

Exploratory Endpoints: B cell & disease relevant markers

Designed to provide PK / PD, safety data & early signals on durability of KP-701 in AID

Phase 1a:

- Assess the safety, tolerability, and PK of KP-701 in patients with HVs and autoimmune disease
- B cell and serum Ig PD to help enable the starting dose for 1B

Phase 1b:

- Evaluate the dose-response and further assess the safety and PK of KP-701 in patients with AID

KP 701: Broad Therapeutic Potential for Large Patient Populations

Rapid proof-of-concept in rheumatology with potential expansion into other therapeutic areas

Rheumatology

- Rheumatoid Arthritis
- Lupus (SLE)
- Primary Sjögren's Syndrome
- Inflammatory Myositis

Endocrinology

- Graves' Disease
- Thyroid Eye Disease
- Hashimoto's
- Obesity

Neurology

- Multiple Sclerosis
- Myasthenia Gravis
- NMOSD

Dermatology

- Immune Thrombocytopenia (ITP)
- Autoimmune Hemolytic Anemia (AIHA)
- Inflammatory Myositis

Summary

$132M PIPE Expected to Fund the Combined Company Through Multiple Anticipated Clinical Catalysts, Well Capitalized into 2H 2028

Multiple anticipated Phase 2 data readouts, two Phase 1 updates and potential Phase 3 start planned

	2026E	2027E	2028E
Vensobafusp (KP104)	**Q4** – Phase 2, Stage 1 interim data (rare renal) **Q4** – New indication Phase 2 plans	**Q2** – Phase 2, Stage 1 updated data **Q2** - Phase 2, Stage 2 interim data (rare renal) **1H** – EOP2 meeting (PNH) **Q4** – New indication interim data	**Q1** – Potential Phase 3 initiation (PNH)
Briquilimab	**2H** – Update on next steps for non-transplant indication **YE -** Statistical analysis for SCID	**Q1** – Regulatory guidance on SCID **EOY** – Potential BLA submission*	**EOY** – Potential US BLA approval & PRV issuance*
KP701		**Q1** – File Phase 1 CTA/IND **Q3** – Phase 1a HV data **Q4** – Phase 1b initiation	**Q2** – Phase 1b data **Mid-Year** – Phase 2 initiation
Early-Stage Programs		**Mid-Year** – DC Selection	

*Subject to FDA alignment, additional funding

Kira PHARMA

JASPER THERAPEUTICS

Corporate Presentation

September 2026

Vensobafusp alfa: Potential Best-in-Class Profile Across Multiple Indications

Target Product Profile

Dual Complement Blockade

- Simultaneously inhibits C5 cleavage and enhances Factor H activity, uniquely addressing both effector and amplification loops

Mechanistic Rationale

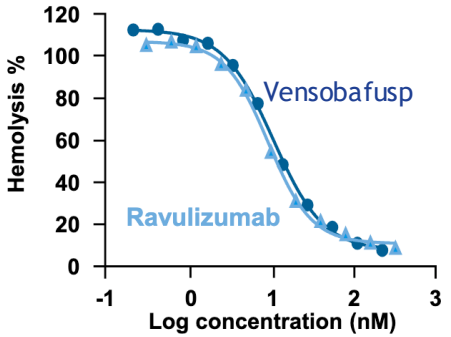
- **C5 Inhibition:** Prevent MAC cytotoxicity and C5a-mediated inflammation
- **Factor H Modulation:** Restores the regulation of the alternative pathway, preventing continuous C3b amplification

Goal

- Durable control of complement activation upstream and downstream – "reset" rather than single-node blockade

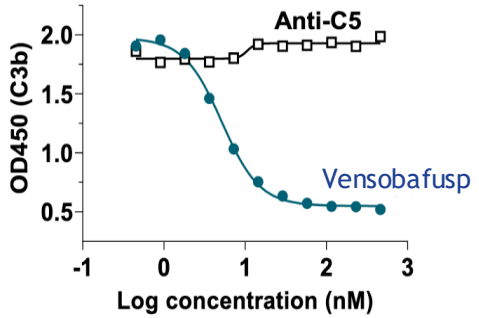
KP104 as potent as Ravulizumab in inhibiting CP-triggered terminal pathway complement activation*

C5 Inhibition Assay



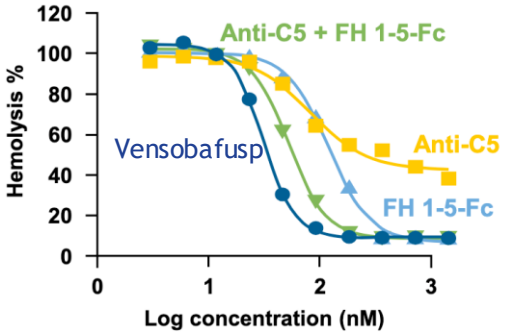
Unlike anti-C5 mABs, vensobafusp also inhibits AP complement*

LPS-Based ELISA assay



Vensobafusp is **more potent** than anti-C5 mAb or Factor H SCR1-5-Fc (alone or combined) in inhibiting AP-triggered terminal pathway complement activation*

Rabbit RBCs Lysis Assay



Vensobafusp more potent than:

- Anti-C5 mAb alone
- Alternative pathway (AP) regulatory (Factor H1-5-Fc) alone
- Anti-C5 mAb and AP regulator (FH1-5-Fc) together

Source: Internal data
*Head-to-Head Study