



Harnessing the Power of Jasper's Differentiated c-Kit Therapeutic Approach

Nasdaq: JSPR

January 2023

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Briquilimab is an anti c-Kit antibody for acute and chronic therapy in multiple mast and stem cell diseases

Clinical validation in 5 for 5 transplant indications

Sickle cell disease (SCD) ✓

Severe combined immunodeficiency (SCID) ✓

Fanconi anemia (FA) ✓

Acute myeloid leukemia (AML) ✓

Myelodysplastic syndrome (MDS) ✓

Therapeutic development in chronic disease

Chronic Spontaneous Urticaria (CSU)
(Expected Start Q3 2023)

Low to Int. Risk MDS
(Expected Start March/ April 2023)

Expansion opportunities: chronic inducible urticaria, allergic asthma, prurigo nodularis, eosinophilic esophagitis

Efficient development with near-term catalysts

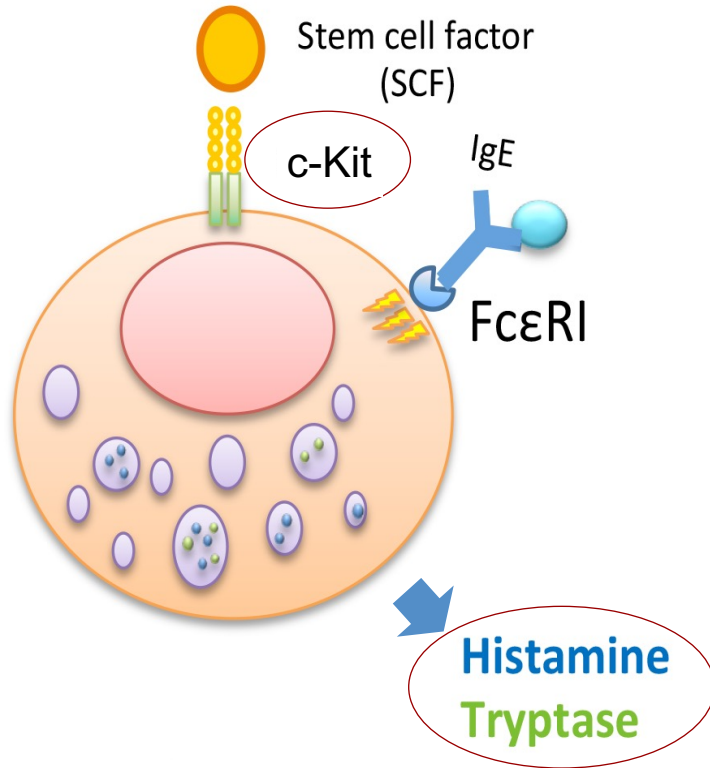
Initial data expected:
Late 2023: Lower to Int. Risk MDS
Mid 2024: CSU

SCID BLA submission targeted for 2024

Multiple transplant studies currently funded and run by partners

Tyrosine kinase KIT plays a central role in regulating mast and stem cell survival

Mast Cell



Blocking c-Kit signaling depletes mast cells

Mast Cells

Play a key role in immune system through the release of compounds that induce inflammation (degranulation)

Unregulated mast cell activation may cause episodes of hives and inflammation (i.e. Chronic Urticaria)

Briquilimab blocks c-Kit signaling and may lead to lasting depletion of mast cells in Chronic Urticaria and other mast cell diseases

Stem Cells

Hemopoietic stem cells are the pluripotent cells that drive ongoing production of red blood cells, platelets and immune cells

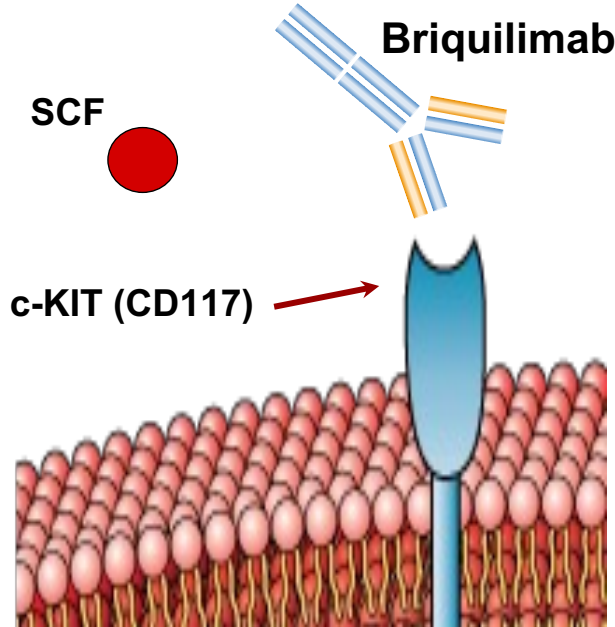
Many blood cancers and genetic diseases can be cured by depleting diseased stem cells and replacing with donor or gene modified stem cells

Briquilimab can be added to existing transplant regimens to block c-Kit signaling on stem cells and potentially deliver more effective transplants

Briquilimab is optimally designed to directly block c-Kit signaling

Briquilimab

Blocks SCF binding to c-Kit (CD117) to directly inhibit receptor signaling



Validated Mechanism of Action

Briquilimab is designed to directly block SCF from binding to c-Kit (CD117) with high affinity and avidity

- Aglycosylated IgG1 antibody directly inhibits stem cell factor from binding to the c-Kit receptor on mast and stem cells
- Inhibition of SCF signaling leads to depletion of mast cells in the skin and hematopoietic stem cells in the bone marrow
- Wide therapeutic potential across a range of mast and stem cell-mediated diseases

Favorable Drug Properties

- $K_d < 5\text{pM}$ affinity to human c-Kit with $\text{IC}_{50} \sim 70\text{pM}$
- Human mast cell survival bioassay $\text{IC}_{50} \sim 12.5\text{nM}$
- No Fc mediated ADCC or complement mediated cytotoxicity which reduces risk of adverse effects related to express CD117
- Human clinical data as IV or Sub-Q delivery

Encouraging Clinical Profile

- Predictable clearance from ages 3yrs to 79yrs, no need to monitor PK
- Demonstrated single agent lasting depletion of mast cells
- Demonstrated efficacy in 5 clinical transplant studies
- No briquilimab-related SAEs reported

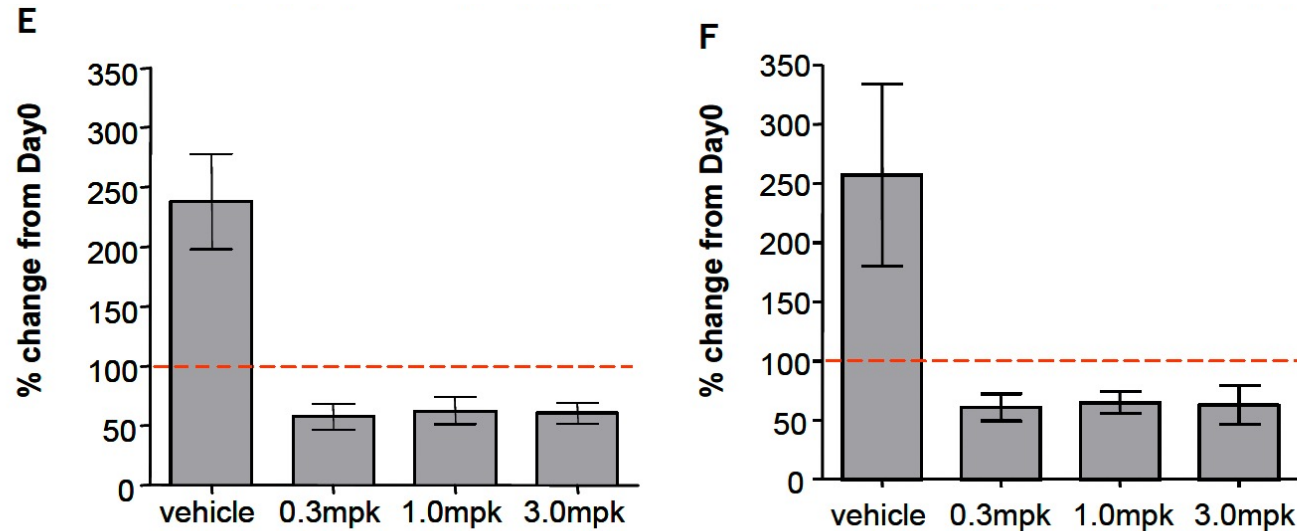
Briquilimab Therapeutic Development

Briquilimab for Chronic Urticaria

Briquilimab c-Kit and mast cell activity

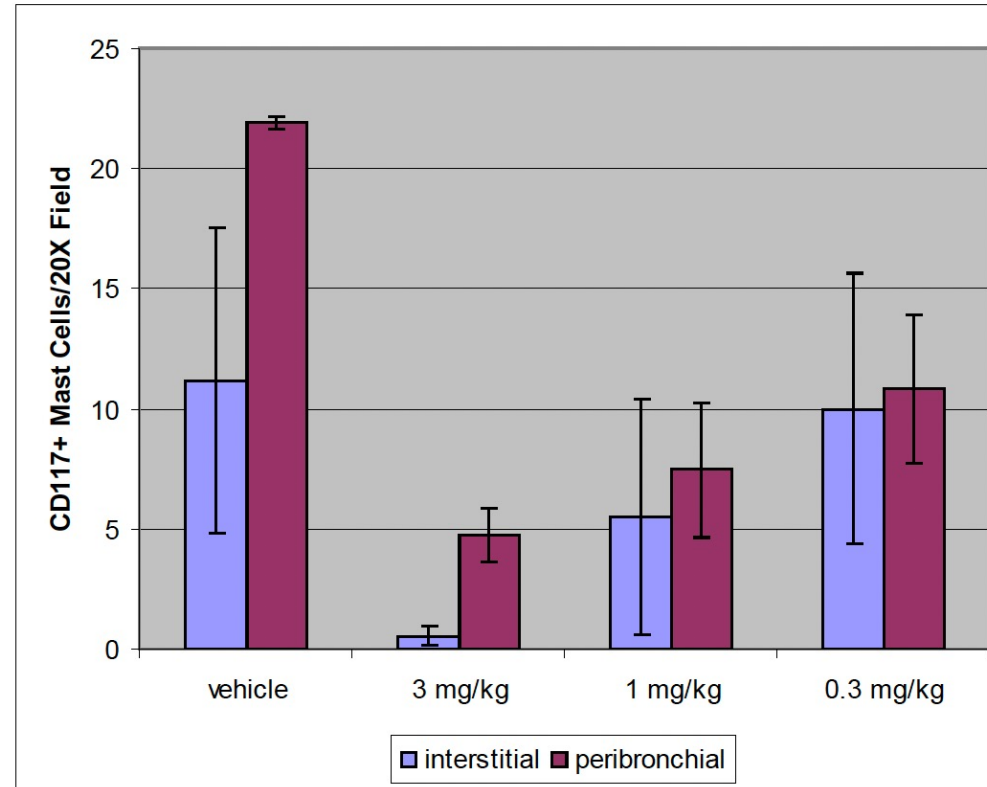
Activity Assay	Result
Binding affinity to human c-Kit Fc dimer	Kd < 5 pM
Biacore assessment of c-Kit signaling neutralization	Ki 70pM
Human mast cell c-Kit phosphorylation bioassay	IC ₅₀ 1.8nM
Human mast cell survival bioassay	IC ₅₀ 12.5nM
Monkey mast cell cutaneous wound proliferation assay	Vehicle: 150% increase in tryptase positive mast cells JSP191 (0.3, 1.0, 3.0 mg/kg): 50% reduction vs. baseline
Monkey basal mast cell depletion model	JSP191 (0.3, 1.0, 3.0 mg/kg): 16-21% depletion
Monkey lung mast cell depletion model	JSP191 (0.3, 1.0, 3.0 mg/kg): Dose dependent depletion

Briquilimab mast cell depletion in non-human primates following cutaneous wound injury



(A, B) Laser scanning cytometry was used to quantify MCs over the area of the entire skin biopsy. Two sections were averaged together from 2 independent biopsies from each of three animals. (C, D) The same samples were independently scored by a pathologist using light microscopy. (B, D) Biopsies from the same animal are used for the bar graphs of panels A and C. (E, F) Day 14 data was normalized to Day 0 for laser scanning cytometry (E) or pathologist scoring (F). v; vehicle.

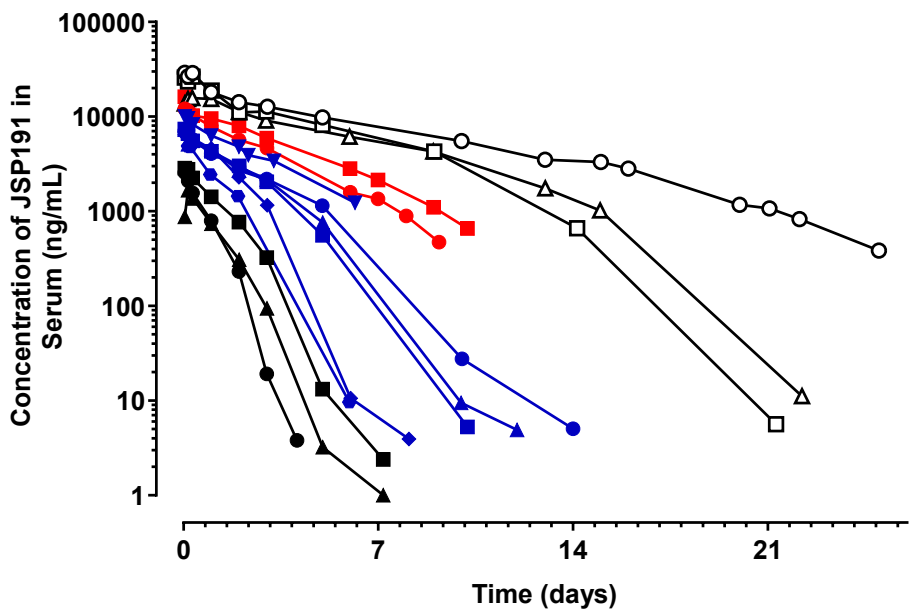
Briquilimab mast cell depletion in lungs of non-human primates



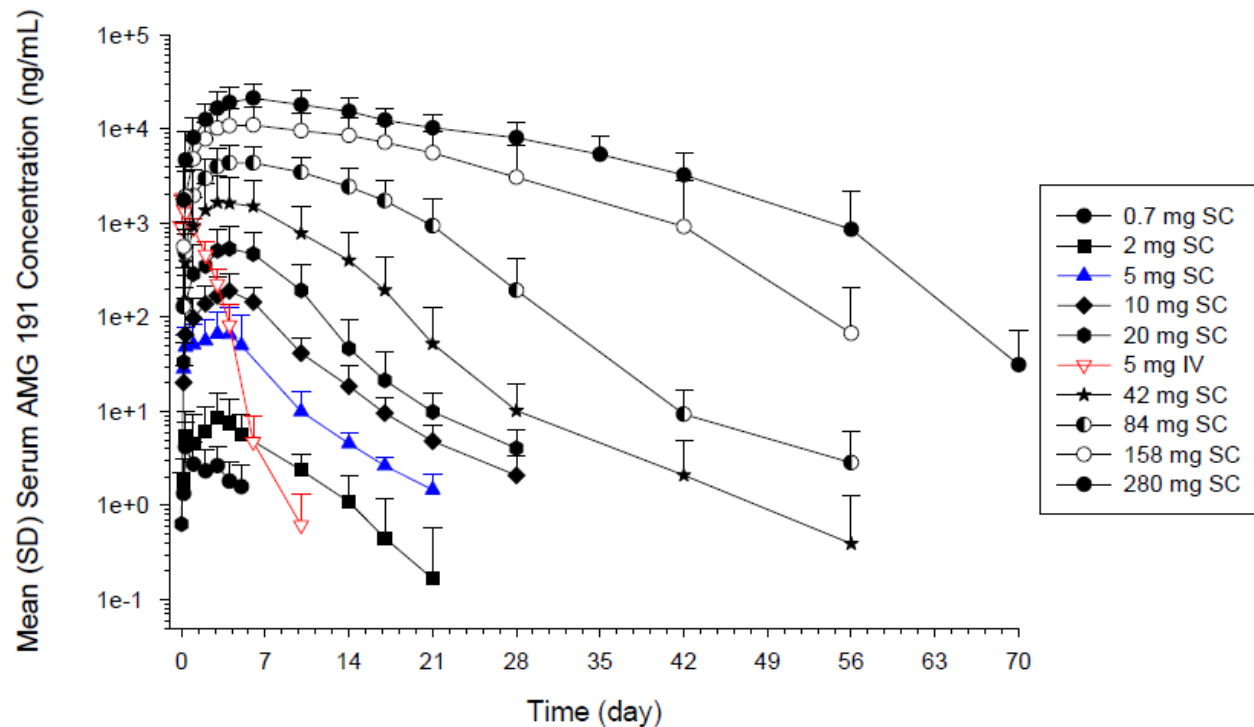
The effect of c-Kit inhibition on the mast cell population in the lung was evaluated in African green monkeys treated SC with 0 (vehicle control; n = 2), 0.3 (n = 4), 1.0 (n = 3), or 3.0 (n = 2) mg/kg AMG 191 once per week for 4 weeks. Mast cells were visualized by CD117 immunohistochemistry in lung samples collected at the study termination (day 28) and CD117-positive mast cells were counted manually in 10 20x parenchymal and 10 20x peribronchiolar lung fields per animal. The data is presented as the mean \pm SD for each treatment group.

Briquilimab can be dosed IV or subcutaneously (SQ)

Briquilimab IV serum concentrations

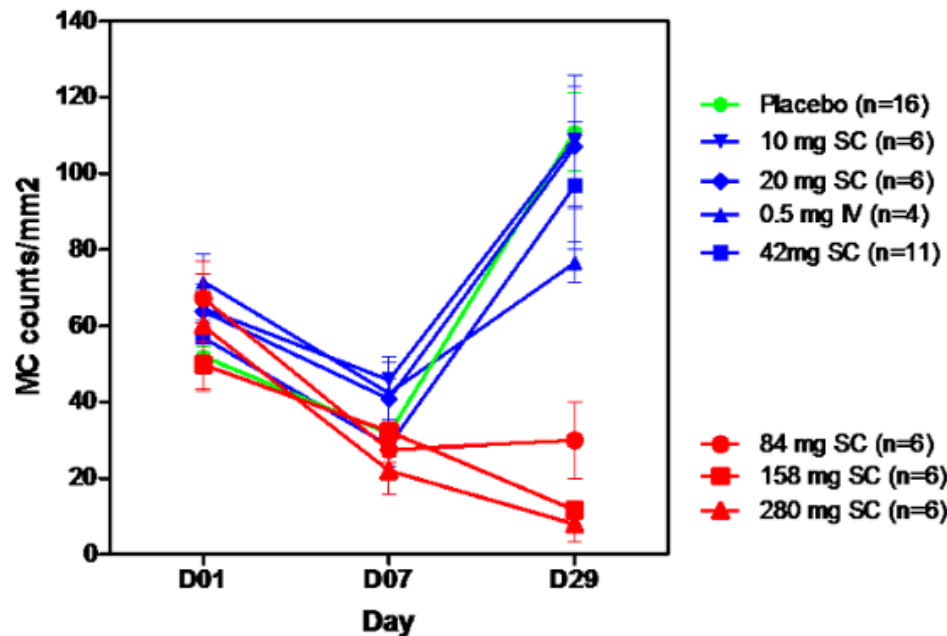


Briquilimab subcutaneous serum concentrations

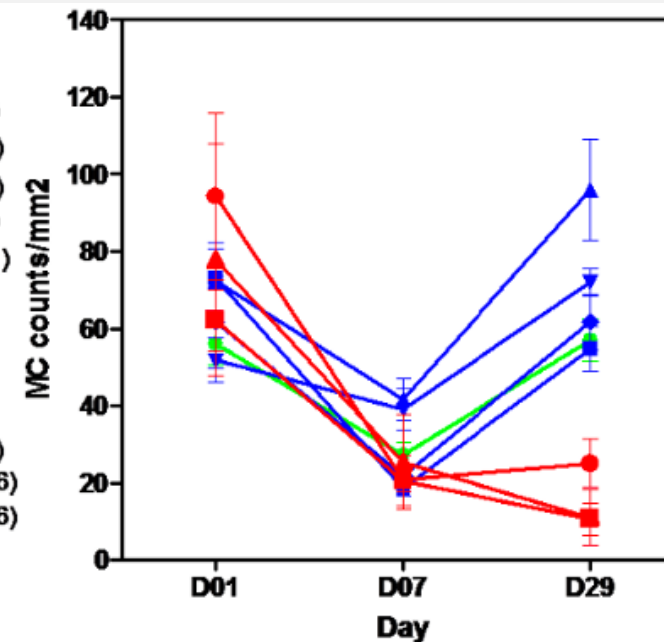


Phase I: Briquilimab robustly depletes tryptase and c-Kit positive skin mast cells

c-Kit + Mast Cells



Tryptase+ Mast Cells



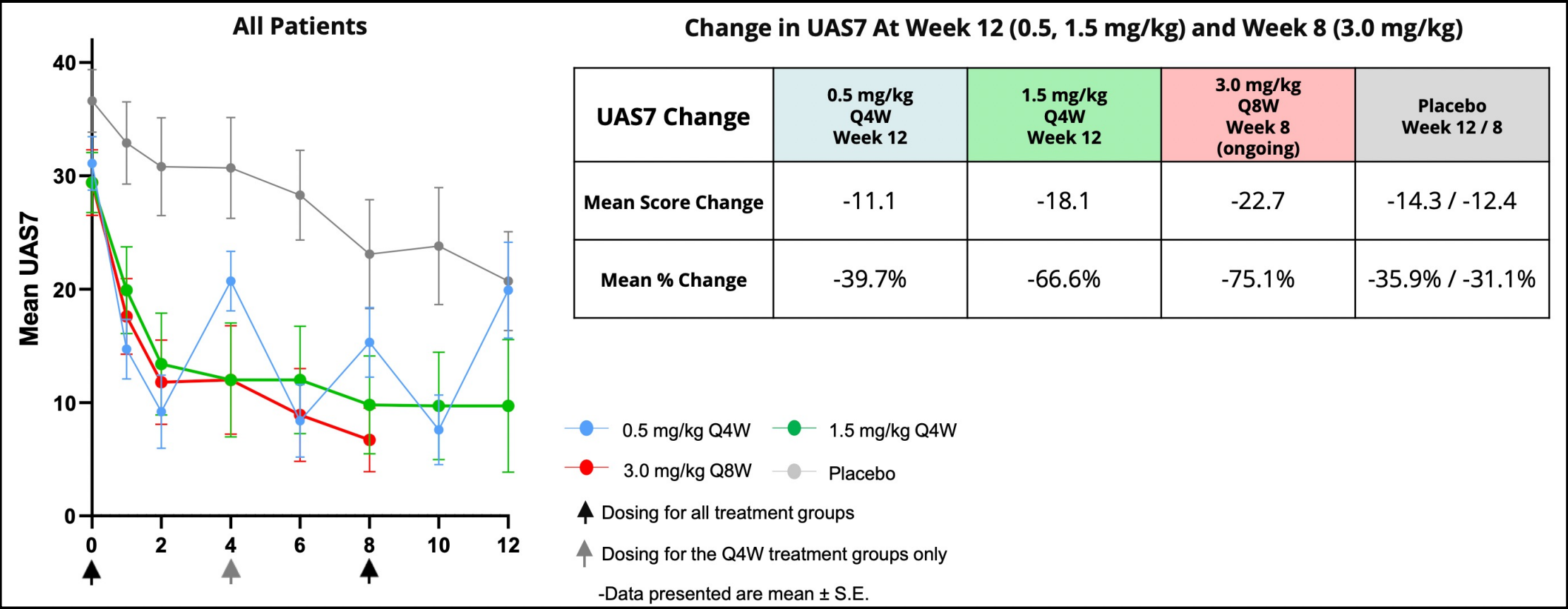
Briquilimab Phase 1a (N=71):

Reduction in SCF (c-Kit) positive and Tryptase positive mast cells*

No briquilimab-related SAEs in any clinical studies to date (>130 subjects treated)

Celldex's barzolvolimab has demonstrated proof-of-concept of c-Kit signal blockade for Chronic Spontaneous Urticaria patients

Barzolvolimab Phase 1b MAD (IV) Shows Efficacy in Chronic Spontaneous Urticaria Patients



Potential initial clinical study for Briquilimab in Chronic Spontaneous Urticaria

	Chronic Spontaneous Urticaria (CSU)
Patient Population	<ul style="list-style-type: none">• H1-antihistamines refractory• Xolair-naïve and/or Xolair-failed
Expected Enrollment	<ul style="list-style-type: none">• 20-40 patients
Study Duration	<ul style="list-style-type: none">• 12-18 months
Timing	<ul style="list-style-type: none">• Targeting Q2 2023 IND with Q3 2023 Study Start
Trial Design	<ul style="list-style-type: none">• Randomized, Placebo-Controlled• Test multiple ascending doses/frequencies• 12-week activity assessment with potential study expansion
Endpoints	<ul style="list-style-type: none">• PK/PD• Safety and tolerability• Urticaria Activity Score (UAS7)• Itch Severity Score (ISS7)• Hives Severity Score (HSS7)• Angioedema Activity Score (ASS7)

Key points of differentiation between Briquilimab and Barzolvolimab (CDX-0159)

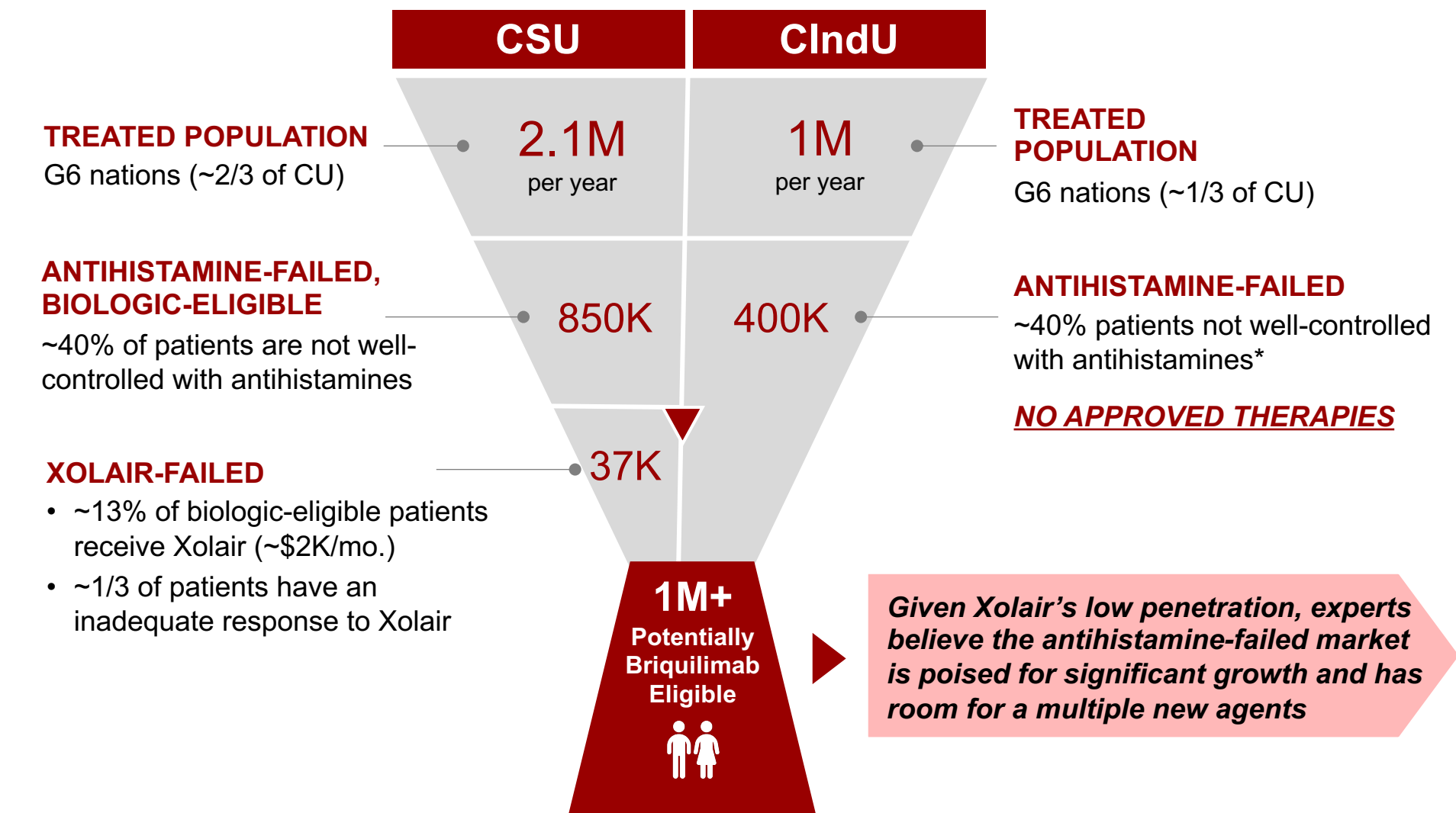
Key Similarities

- 1 c-Kit (CD117) monoclonal antibody**
 - Both antibodies are humanized, aglycosylated IgG1 inhibitors of c-Kit signaling on mast cells
- 2 On-target depletion of mast cells**
 - Phase 1a data suggests similar, dose-dependent inhibition of c-Kit positive and tryptase positive mast cells in the skin
- 3 Comparable PK/PD profile**
 - Phase 1a data (IV and SQ) suggests similar, predictable, PK profiles

Key Differences

- 1 Briquilimab directly blocks SCF binding**
 - Direct and complete blockage of ligand binding to the c-Kit receptor
- 2 Potential first to market anti c-Kit mAb**
 - Briquilimab SCID indication with potential BLA filing in 2024
- 3 Proven to work in multiple diseases**
 - Effective in transplant for AML, MDS, SCID, Fanconi Anemia and Sickle Cell Disease

Chronic urticaria represents a significant and expanding market, with estimated 1+ million potential patients



There is a significant unmet need for Chronic Urticaria patients who fail antihistamines – safer, more effective therapies are needed

Chronic Urticaria 1.5-3 million cases in the US ¹		
Population	Approved Therapies ²	Limitations
<div>H-1 Antihistamines</div> <div>↓</div> <div>Antihistamine-Failed</div>	<ul style="list-style-type: none">• Zyrtec• Claritin• Xolair (only approved agent in CSU; 2014)*• Non-approved agents: <i>cyclosporine, montelukast, dapsone*</i>	<ul style="list-style-type: none">• 30-50% of patients are still uncontrolled after FDA-approved doses²• 20-40% of patients have an inadequate response to Xolair³• Low penetration rate of Xolair (~13%) due to black box warning for anaphylaxis⁴

Significant unmet need in Chronic Urticaria patients who fail antihistamines
Safer, more effective therapies are needed to meet market potential

*Montelukast = Leukotriene receptor antagonist; cyclosporine = immunosuppressant; dapsone = sulphone
Sources: [1] GlobalData; [2] Zuberbier T, et al. (2021) *European Journal of Allergy and Clinical Immunology*; [3] Omalizumab prescribing information; [4] Market research conducted by Novartis (presentation in 2021).

Briquilimab can expand beyond Chronic Urticaria into other mast cell-mediated and inflammatory diseases

Mast Cell-Mediated Diseases (Addressed by c-Kit Inhibition)

Chronic Inducible Urticaria

Patients are inadequately controlled by antihistamines; only approved biologic therapy (Xolair) has significant safety concerns⁵

- **CIIndU:** 80K patients biologic-eligible (US)^{1,5}

Severe Asthma

Life-threatening disease with no approved biologics for ~50% of patients who lack Type 2-high disease⁵

- **Severe Asthma:** 500K patients biologic-eligible (US)^{2,5}

Other Mast Cell Diseases

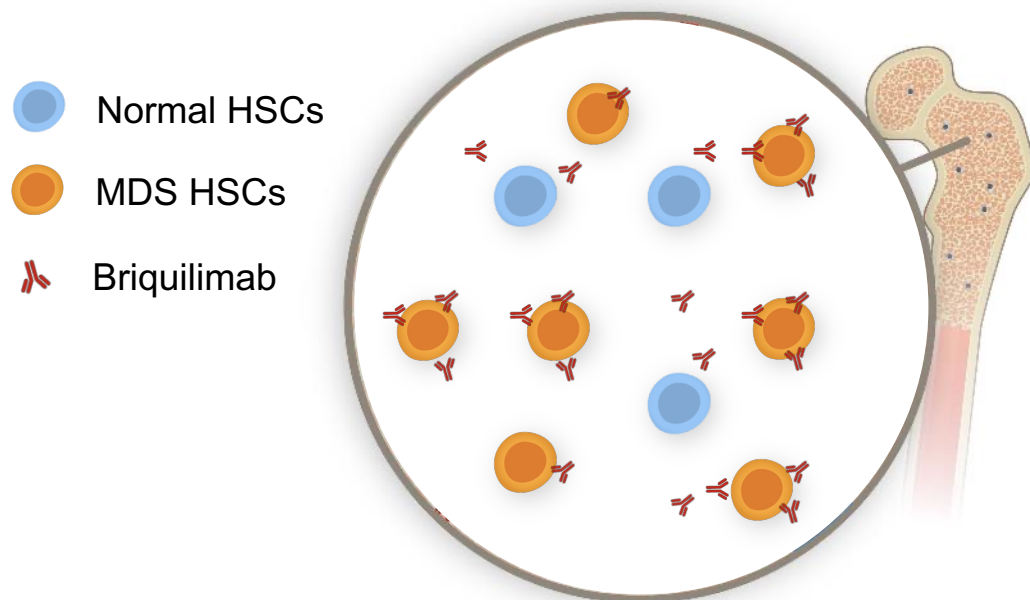
Numerous mast-cell mediated diseases are still inadequately controlled by current treatment options⁵

- **Prurigo Nodularis:** 75K patients biologic-eligible (US)^{3,5}
- **Eosinophilic Esophagitis:** 50K patients biologic-eligible (US)^{4,5}

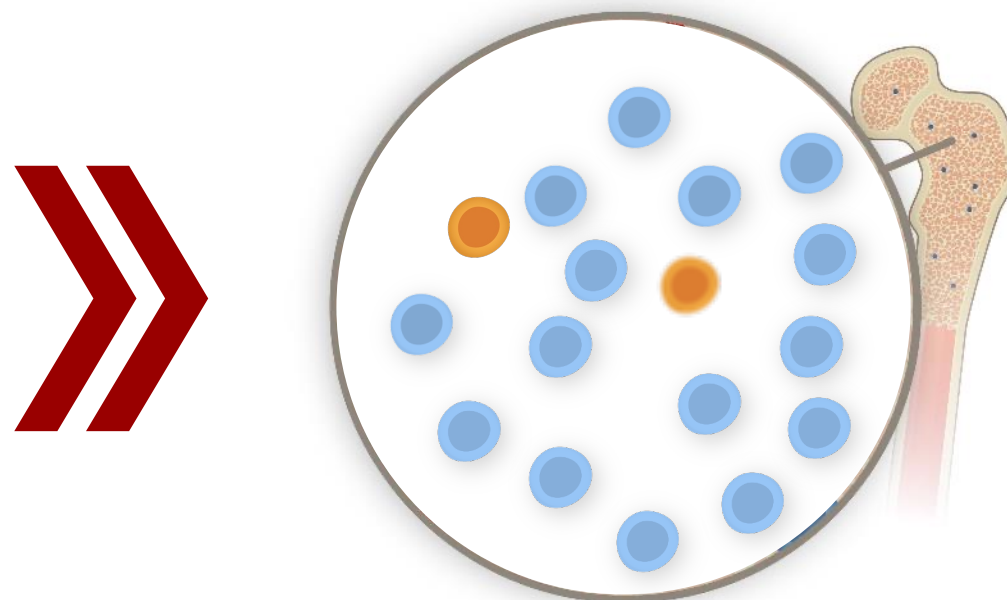
Briquilimab for Lower to Intermediate Risk MDS

Briquilimab's ability to directly deplete cancerous stem cells may be leveraged as a disease-modifying therapeutic in lower to intermediate risk MDS patients

Briquilimab administered in a LR-MDS patient



Shift of HSCs towards normal hematopoiesis



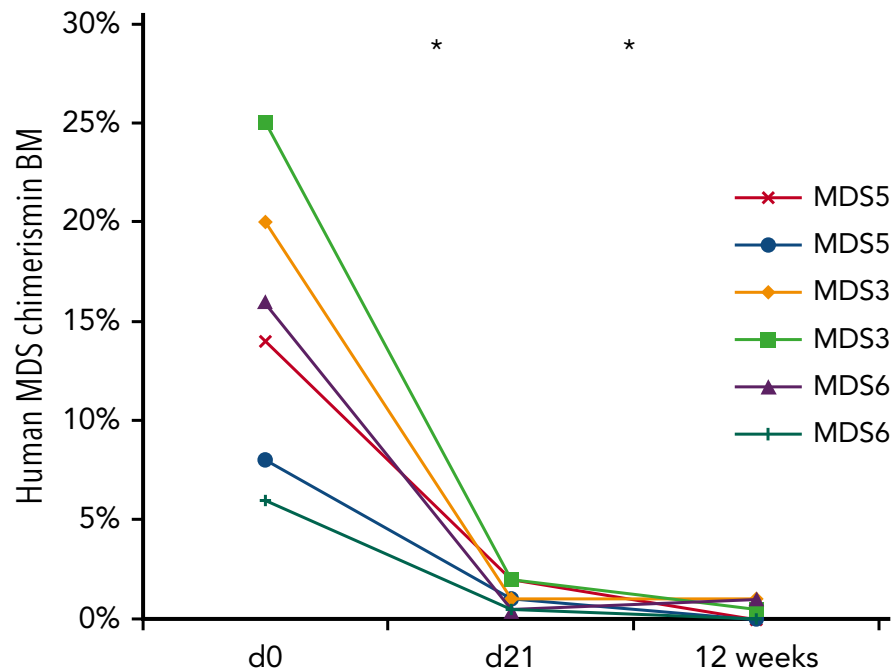
Patients are currently managed with supportive / stimulating therapies that do not target diseased cells

“ There is a tremendous unmet need for new treatments for MDS; rates of relapse are high, and many patients are not eligible for existing approved therapies...”

- Platzbecker et al., 2021 Leukemia

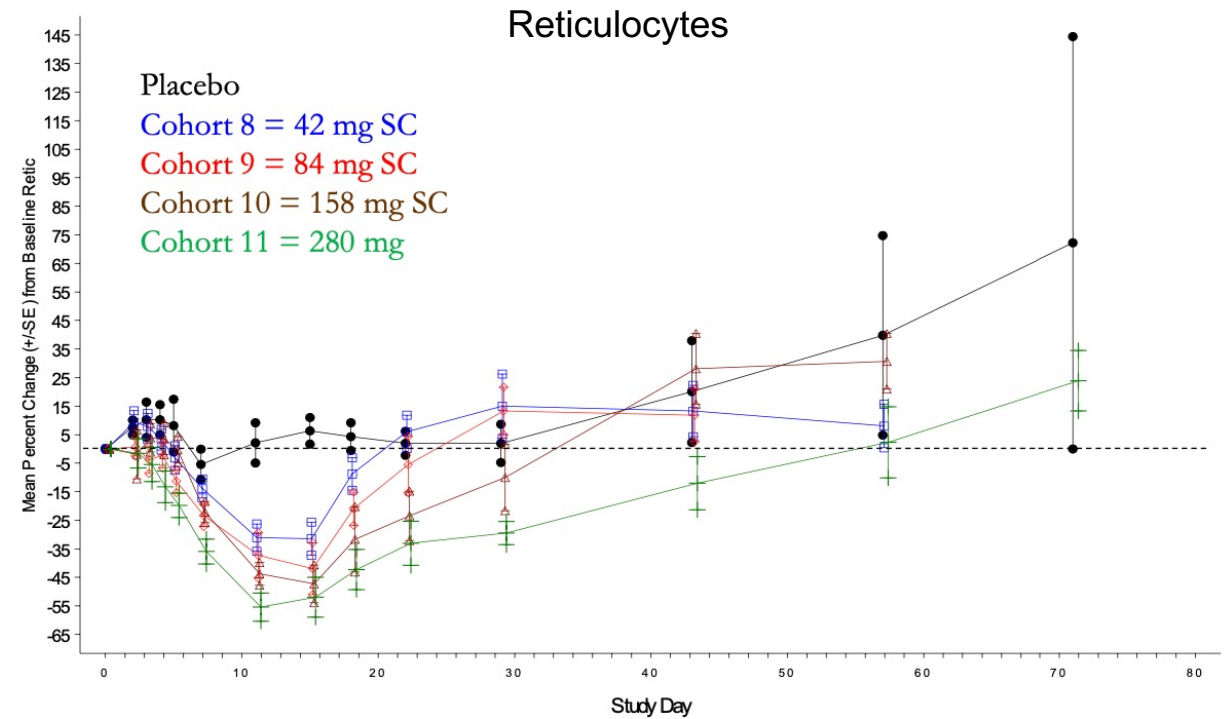
Briquilimab depletion may enable healthy HSCs to take over the bone marrow niche and restore functional hematopoiesis

Briquilimab lasting depletion of MDS cells in Xenograft model¹



MDS cells are depleted and stay depleted

Briquilimab depletion and rebound of healthy HSCs²



Healthy HSCs are depleted, recover and expand

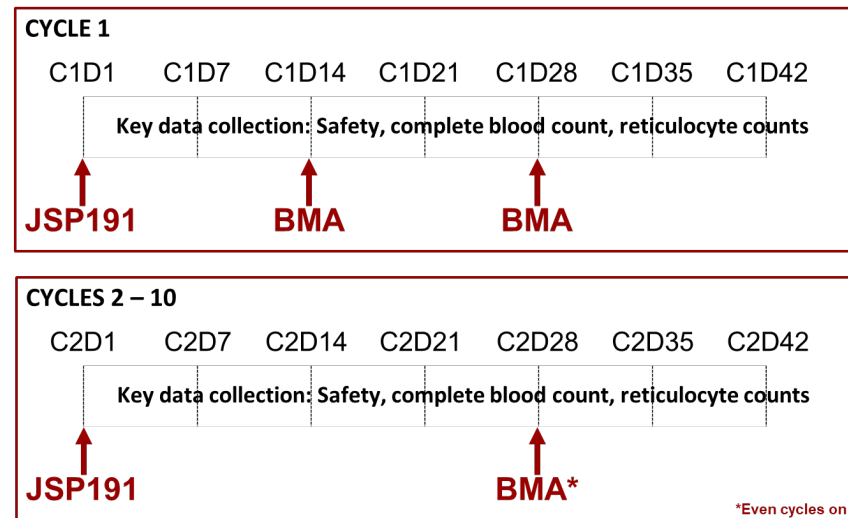
A Phase I open-label, dose-escalation, safety and tolerability study of briquilimab as a second line therapy in patients with lower-risk myelodysplastic syndrome

Population

- IPSS-R very low, low or intermediate risk MDS patients
- RBC transfusion dependence, thrombocytopenia or neutropenia

Single Arm Design

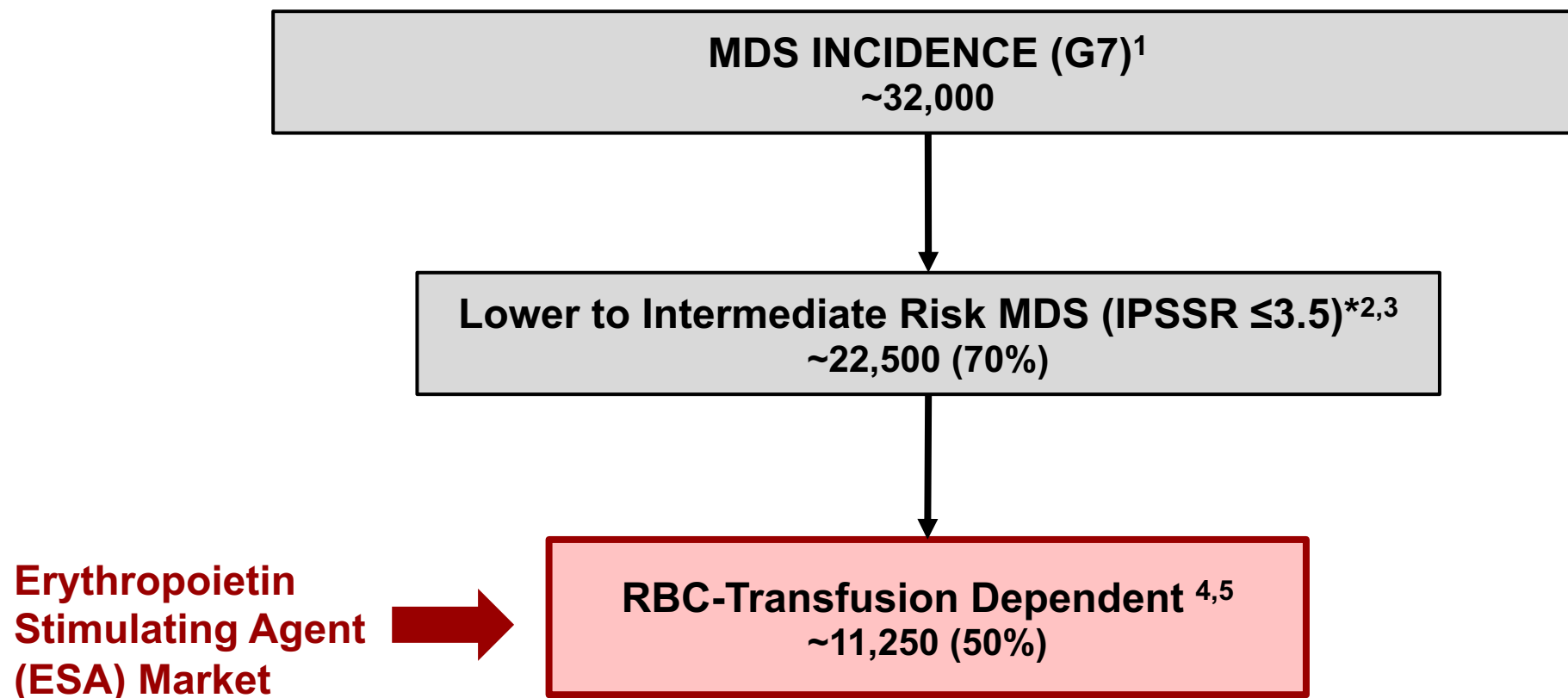
- **Dosing:** 4 cohorts – 0.3 mg/kg (w/ sentinel dose of 0.1 mg/kg), 0.6 mg/kg, 0.9 mg/kg, 1.2 mg/kg
- **Dose cycles:** Every 8 weeks
- **Size:** 3-6 per cohort



Key Assessments

- **Primary Objectives:** Safety, tolerability and DLT of MAD briquilimab
- **Secondary Objectives:** PK, Efficacy by HI-E/HI-P/HI-N and duration of response, reduction in RBC transfusions, ORR and duration of response by IWG 2006, progression free survival
- **Exploratory:** Depletion of leukemic stem & progenitor cells, hematopoietic stem & progenitor cells, molecular characteristics of LSCs/HSCs/HPCs, cytokine profile, briquilimab immunogenicity

Lower to intermediate risk MDS market opportunity



*Median survival for very low and low risk MDS is 8.8 years and 5.3 years, respectively.

Sources: [1] Lubeck 2016, Blood; [2] GlobalData; [3] Greenberg 2012, Blood; [4] de Swart 2015, BJHaem; [5] de Swart 2020, Haematologica

Briquilimab Transplant Development

Sickle Cell & Beta Thalassemia

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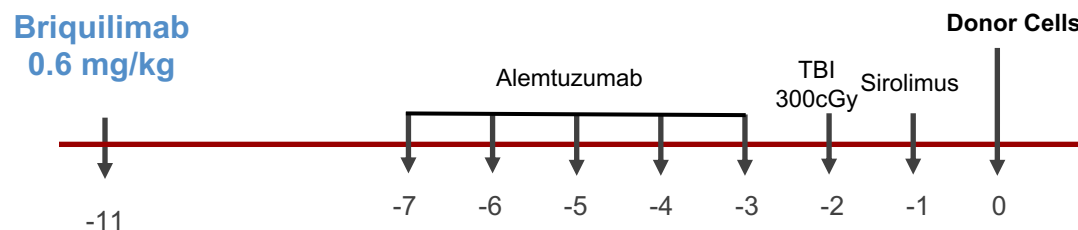
Phase I: Addition of briquilimab to non-myeloablative hematopoietic cell transplantation for Sickle Cell Disease and Beta-Thalassemia

Single Arm Trial Design

Study Endpoints

SCD patients

- High-risk SCD
- 6/6 HLA matched family donor available



- Percent myeloid (CD14/15) chimerism
- Donor stem cell engraftment
- Safety / GVHD

- Sickle cell disease (SCD) is a group of inherited red blood cell / hemoglobin disorders; patients with SCD have misshapen red blood cells, which prevents normal cell function / behavior
- Patients typically develop chronic problems such as renal disease, lung disease, and pulmonary hypertension, putting them at high risk for complications when treated with chemotherapy / radiation
- The NHLBI (NIH) is studying a briquilimab combination with alemtuzumab, low-dose irradiation, sirolimus + HSCT to potentially cure patients with sickle cell disease
- **First three briquilimab treated SCD patients have achieved successful transplant with first two patients at 100% donor myeloid chimerism at 60 days. First patient has achieved total hemoglobin of 13.3 g/dL at 5 months.**

SCID



Briquilimab conditioning for severe combined immunodeficiency (SCID)



Jasper SCID Strategy:

- Establish Single Agent Activity POC
- Focus on Re-transplant T-B- Subjects
- FDA Supportive of Ultra-Orphan Data Package
- Potential Priority Review Voucher

Briquilimab SCID phase I results: Safety and tolerability to date

Clinical Safety

- No briquilimab-related serious adverse events (SAEs)
- No myelosuppression
- No significant infusion reactions

Clinical Setting

- Protocol amended to allow for outpatient administration of briquilimab
- Based on safety and successful HSC engraftment in re-transplant SCID subjects, the study of briquilimab has been expanded to include newly diagnosed infants with SCID

Potential near-term biologics license application (BLA) and PRV opportunity for briquilimab in SCID

SCID is a lethal genetic immune disorder where HCT is the only proven cure

Potential FDA filing strategy

SCID re-transplant patients are ultra orphan, high unmet need population

Focus on current clinical data with briquilimab in 10 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

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

Fanconi Anemia



Phase I: Depleted donor stem cell transplant in children and adults with Fanconi Anemia after being conditioned with a regimen containing briquilimab

Single Arm Trial Design

Study Endpoints

- **Fanconi patients in bone marrow failure**
- $\geq 5 / 10$ HLA-matched related or unrelated donor



- Safety
- Donor stem cell engraftment
- Restoration of blood counts

- Fanconi is an inherited DNA repair deficiency that leads to progressive failure of hematopoietic stem and progenitor cells and transfusion dependency
- Allogeneic stem cell transplant is the only current cure, however Fanconi patients are highly susceptible to toxic side effects of conditioning with radiation or alkylating agents
- Successful allogeneic transplant will result in healthy bone marrow with normal DNA repair capabilities and normal blood cell counts
- Stanford is studying a briquilimab based conditioning regimen plus a TCR $\alpha\beta$ ⁺ T-cell/CD19⁺ B-cell Depleted Hematopoietic Graft to cure Fanconi patients with reduced risk of GVHD
- **The first two patients have achieved 100% donor myeloid chimerism along with recovery of normal blood counts. To date no GVHD or Veno-Occlusive Disease has been reported; grade 3 mucositis was observed.**

Potential Fanconi Anemia development path for briquilimab

Allogeneic stem cell transplant can restore bone marrow and blood formation in Fanconi Anemia patients

Development strategy

Continue to enroll under current protocol (n up to 12)

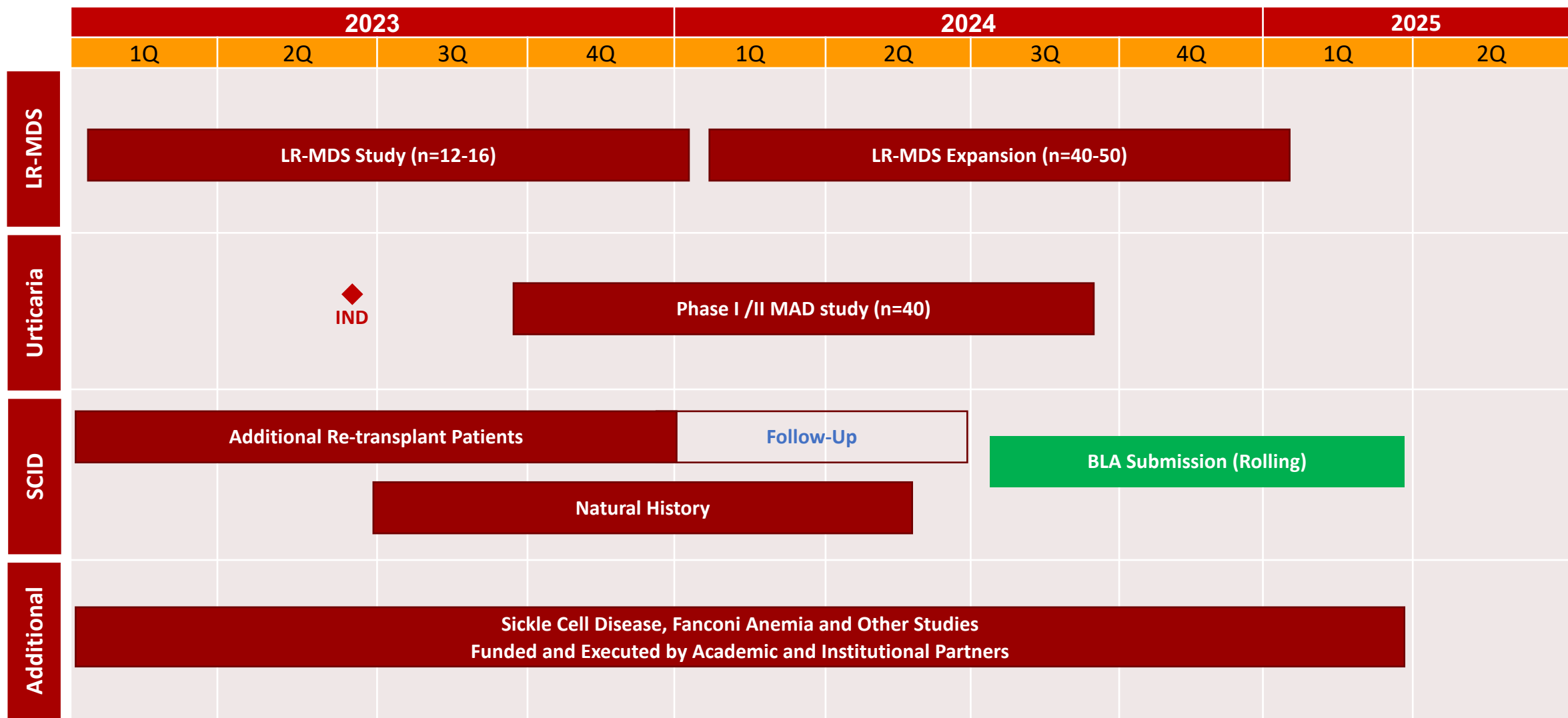
Discuss path to BLA with FDA with 6-month data in first three patients

- Example: Rocket Pharmaceuticals has communicated that FDA guidance of gene therapy efficacy in at least 5 of 12 patients may be sufficient to support a potential BLA filing¹

Consider expansion to additional clinical sites following FDA discussion

Successful development of briquilimab in Fanconi Anemia may lead to rare pediatric disease designation and a Priority Review Voucher

Briquilimab development plan





Harnessing the Power of Jasper's Differentiated c-Kit Therapeutic Approach

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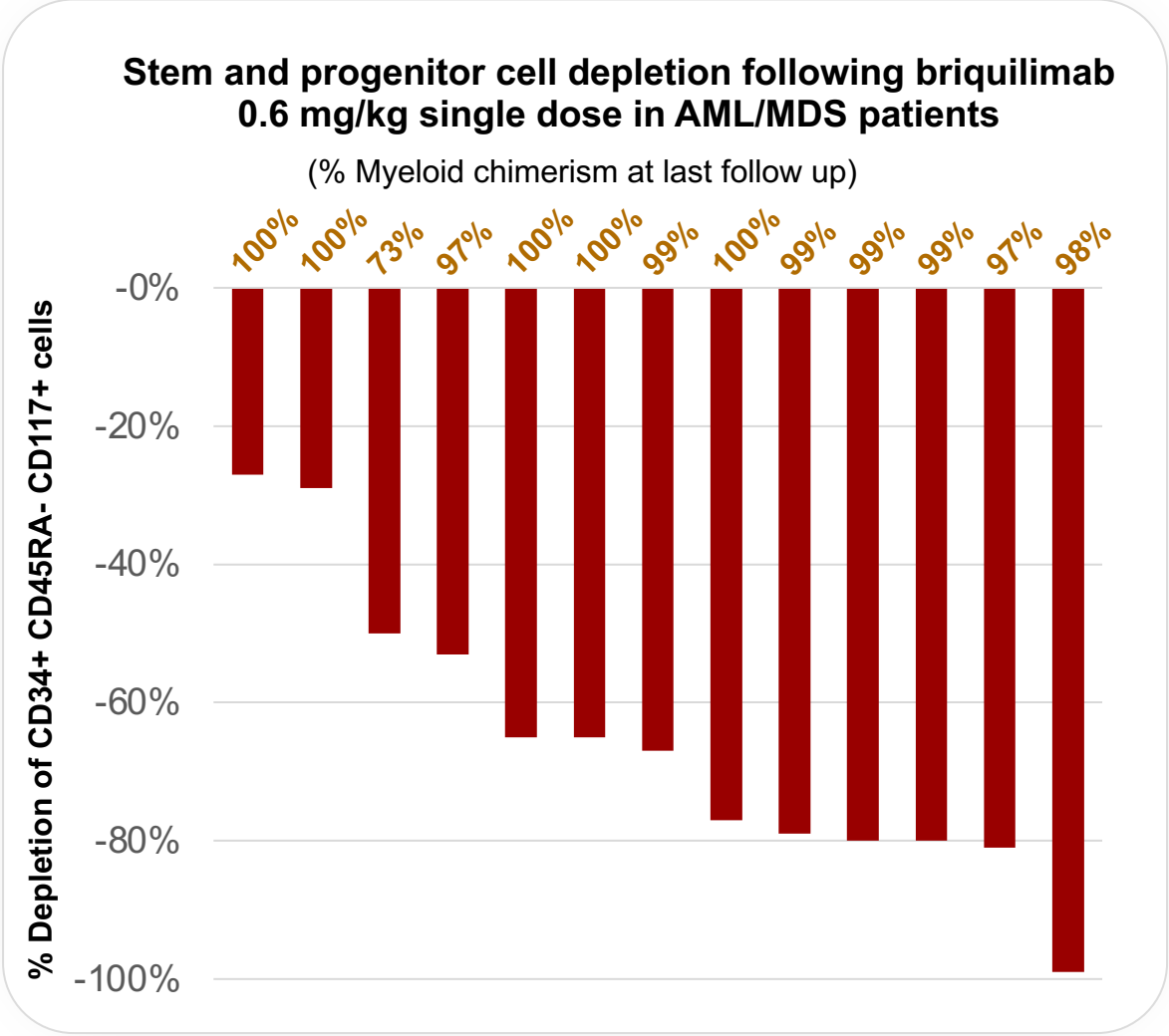
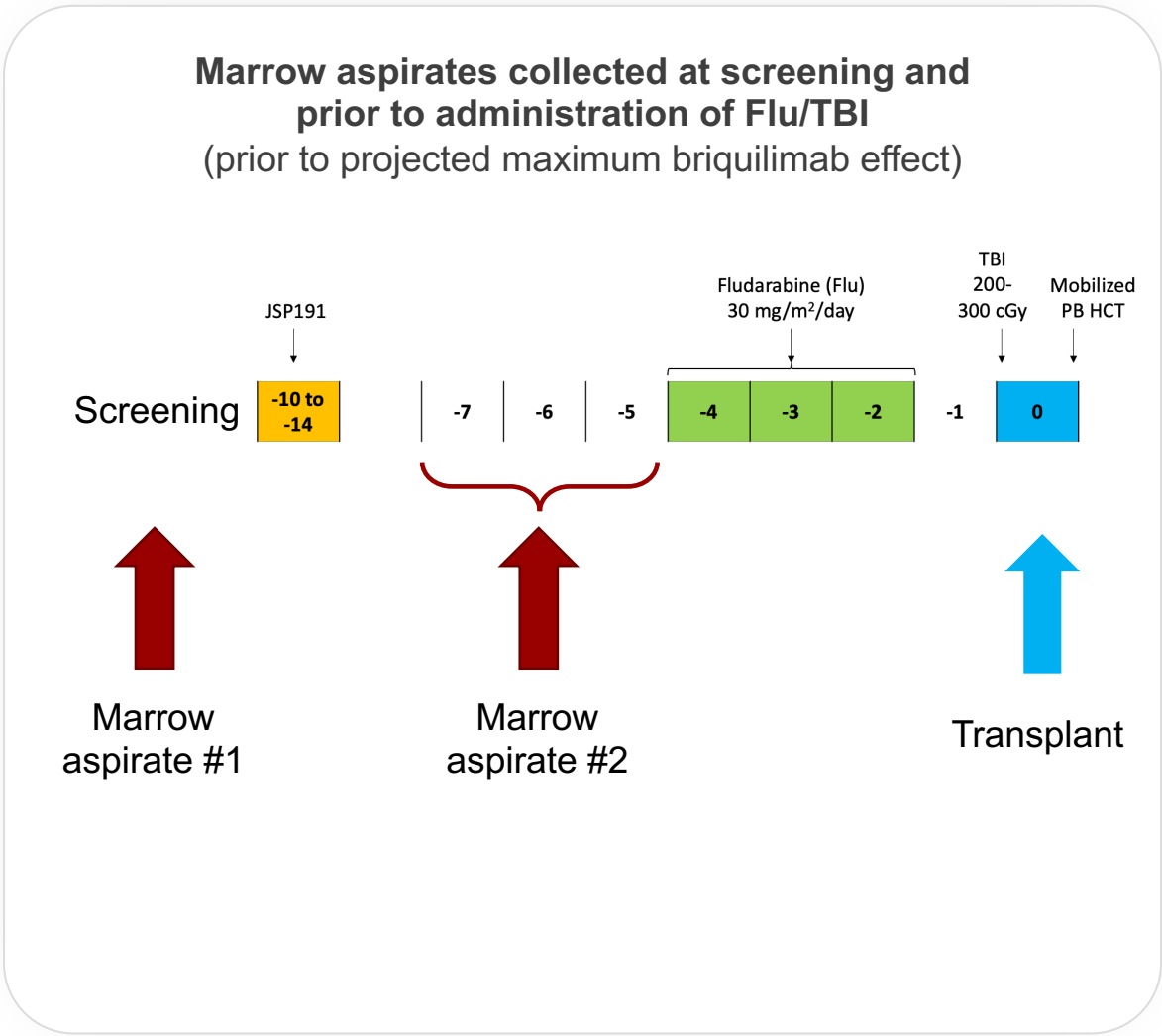
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Additional Slides

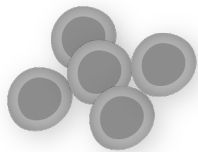
AML / MDS



AML/MDS Study: Single agent briquilimab depletes diseased stem & progenitor cells in bone marrow in 5 to 7 days

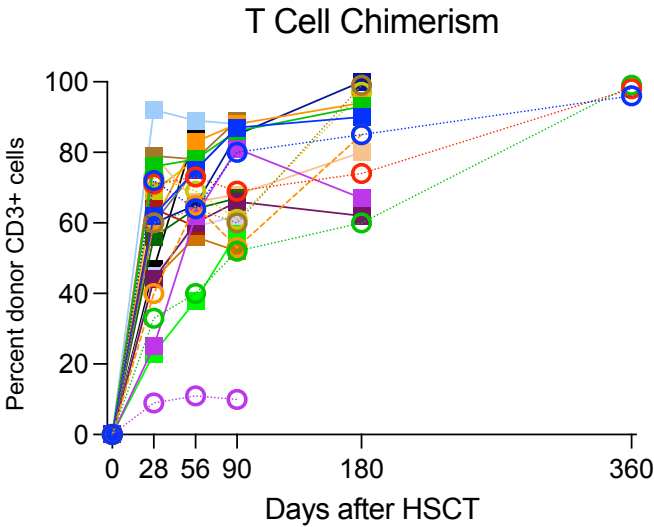
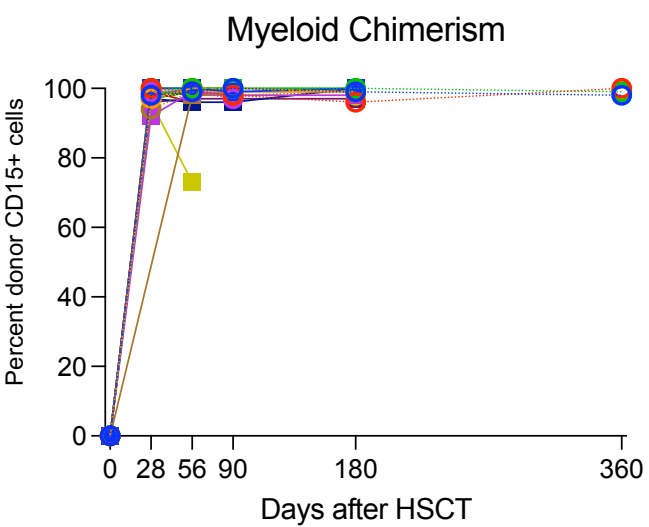


Briquilimab based conditioning demonstrates donor cell takeover of bone marrow stem cells and elimination of detectable cancer in older patients



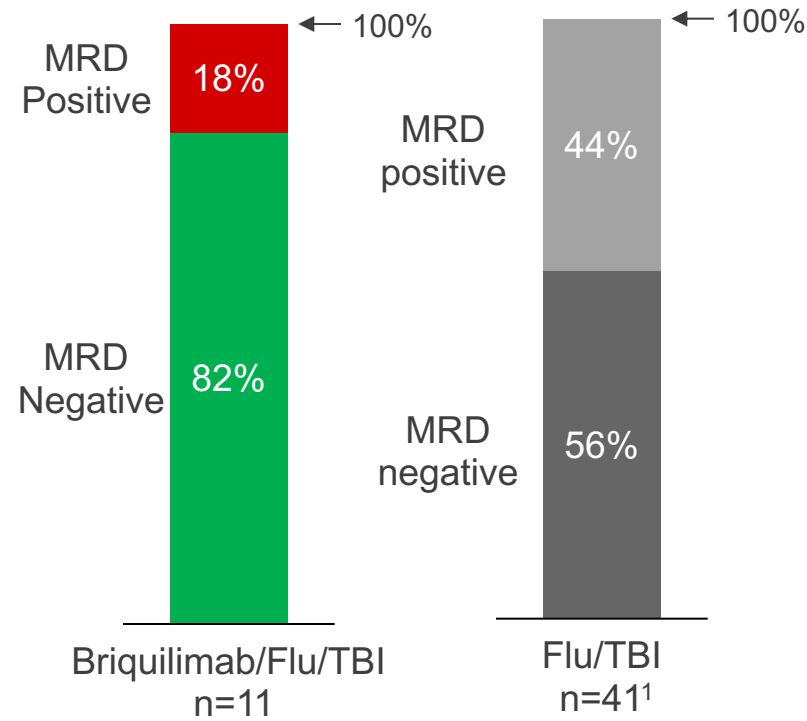
Donor cell takeover of the bone marrow

(Median donor chimerism)



Clearance of detectable cancer

(Measurable residual disease by flow cytometry)



No briquilimab-related SAEs, no 100-day transplant related mortality and proof of concept for outpatient stem cell transplant

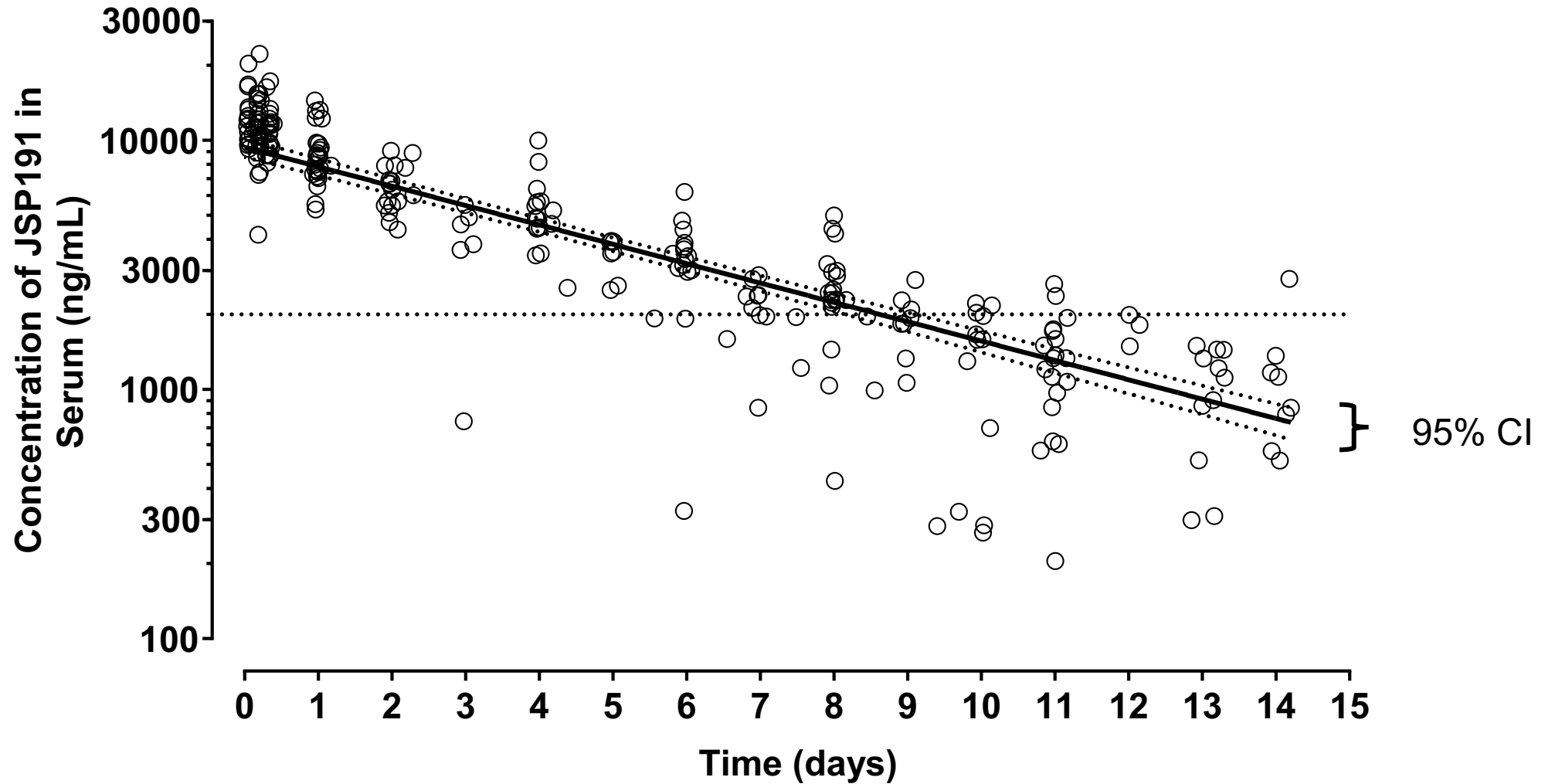
Clinical Safety

- No briquilimab-related SAEs
- No significant briquilimab infusion reactions
- One subject with refractory late onset grade 3 acute GI GVHD
- One subject with secondary graft failure
- No 100-day transplant-related mortality
- No significant mucositis, hypersensitivity, hepatotoxicity, or other organ damage

Clinical Setting

- Protocol allows for outpatient conditioning
- 14 subjects given outpatient briquilimab along with outpatient transplant

0.6 mg/kg IV Briquilimab Pharmacokinetics: consistent and predictable clearance



Preclinical

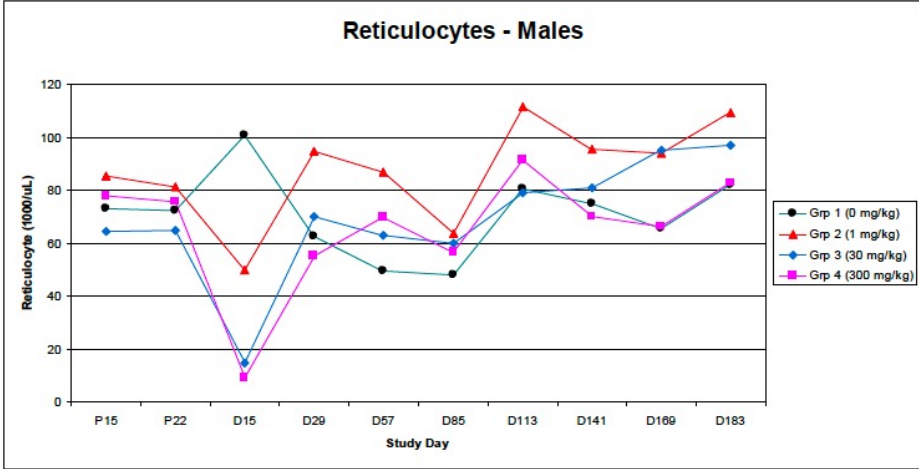


Briquilimab 6-month non-human primate (NHP) toxicology study

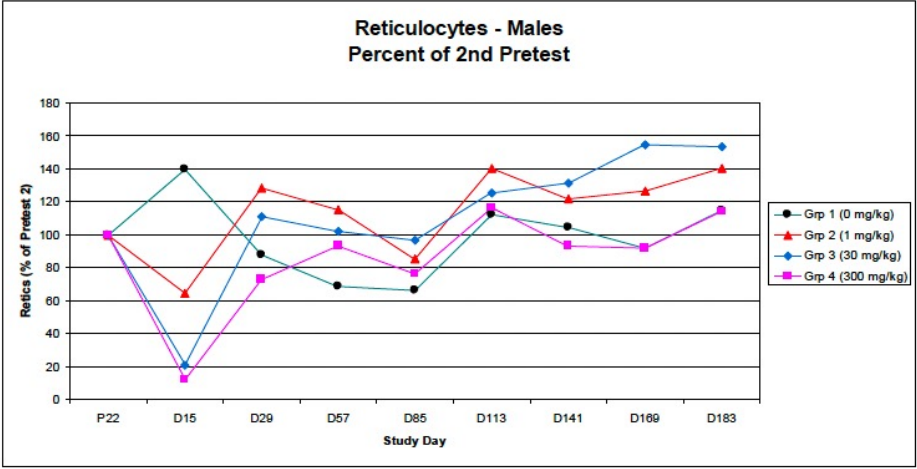
- 4 groups (control, 1, 30, 300 mg/kg weekly subcutaneous), n=8 to 12 per group, 50% female
- Dosing for 26 weeks followed by 32-week treatment-free phase
- Test article-related clinical signs: Paleness of skin and fur in all males and females given 30 or 300mg/kg
- Test related clinical pathology effects:
 - Adverse hematology change due to decrease in absolute reticulocyte counts and red cell mass at 30 and 300 mg/kg weekly
 - Adverse effect on sperm motility, density, morphology; decreased weight of epididymis and testes; germ cell loss in epididymis and testes
 - Increased platelet counts in 30 and 300 mg/kg group
 - Decreased number of colonic mast cells
 - All clinical pathology resolved by end of 32-week treatment-free phase
- Anti-drug antibodies: Binding antibodies detected in 13/28 (46%) dosed animals of which 6 also tested positive for neutralizing antibodies

Reticulocytes and Hemoglobin Levels – 6mo NHP toxicology study

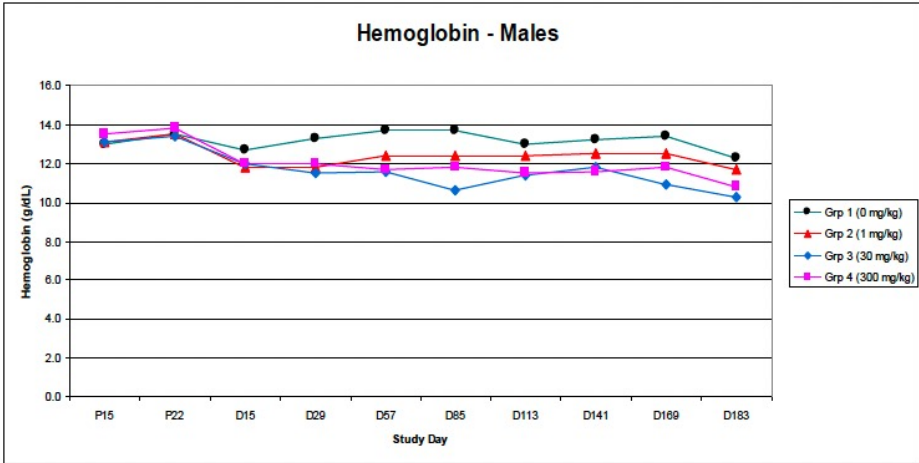
Absolute



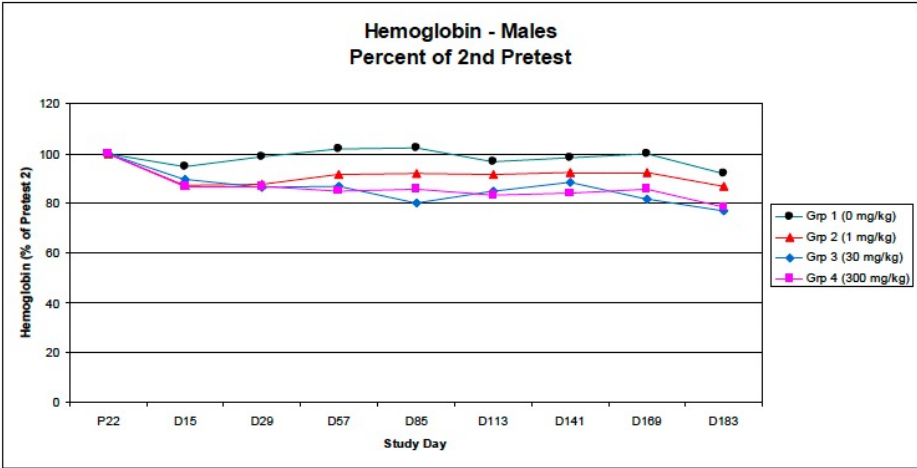
Percent of 2nd Pretest



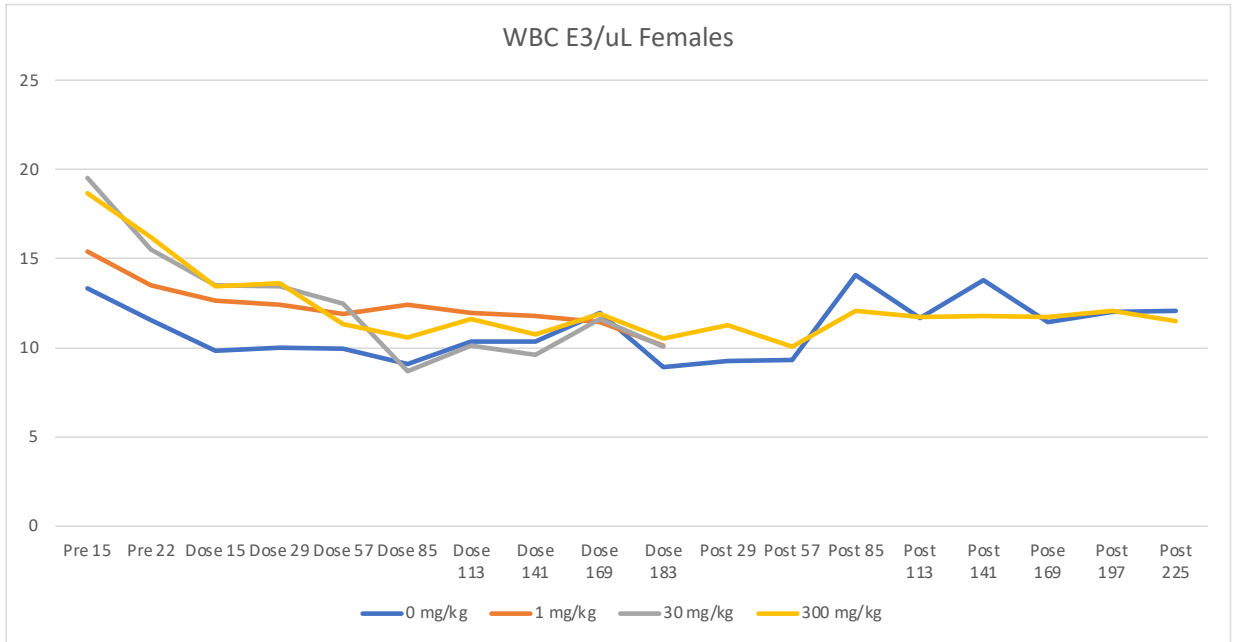
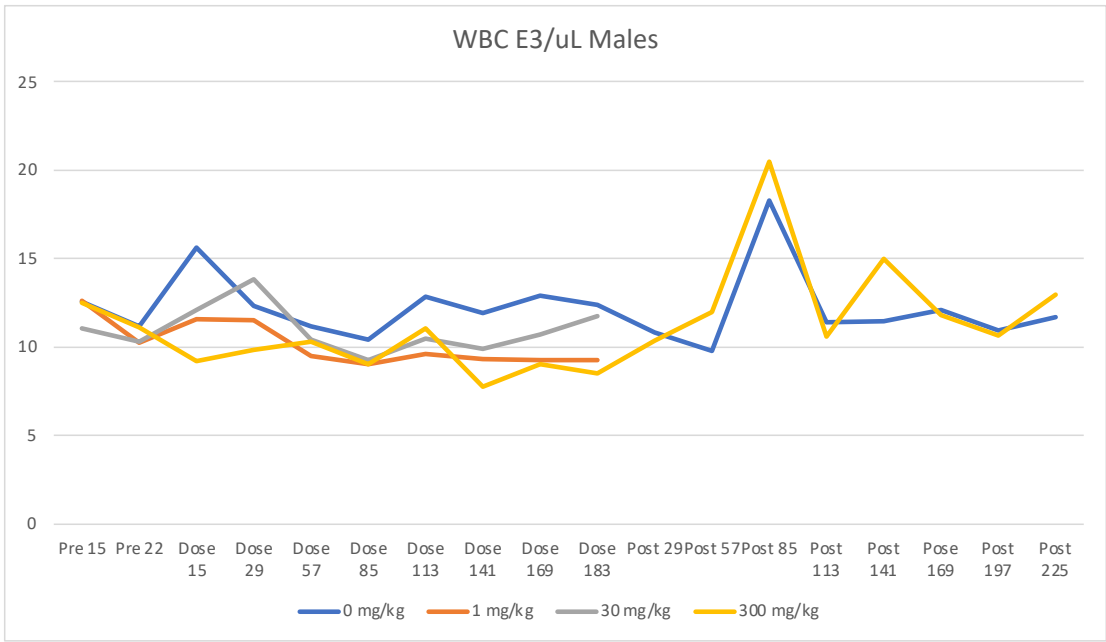
Hemoglobin - Males



Hemoglobin - Males
Percent of 2nd Pretest



White Blood Cells – 6mo NHP toxicology study



Phase I Healthy Volunteers

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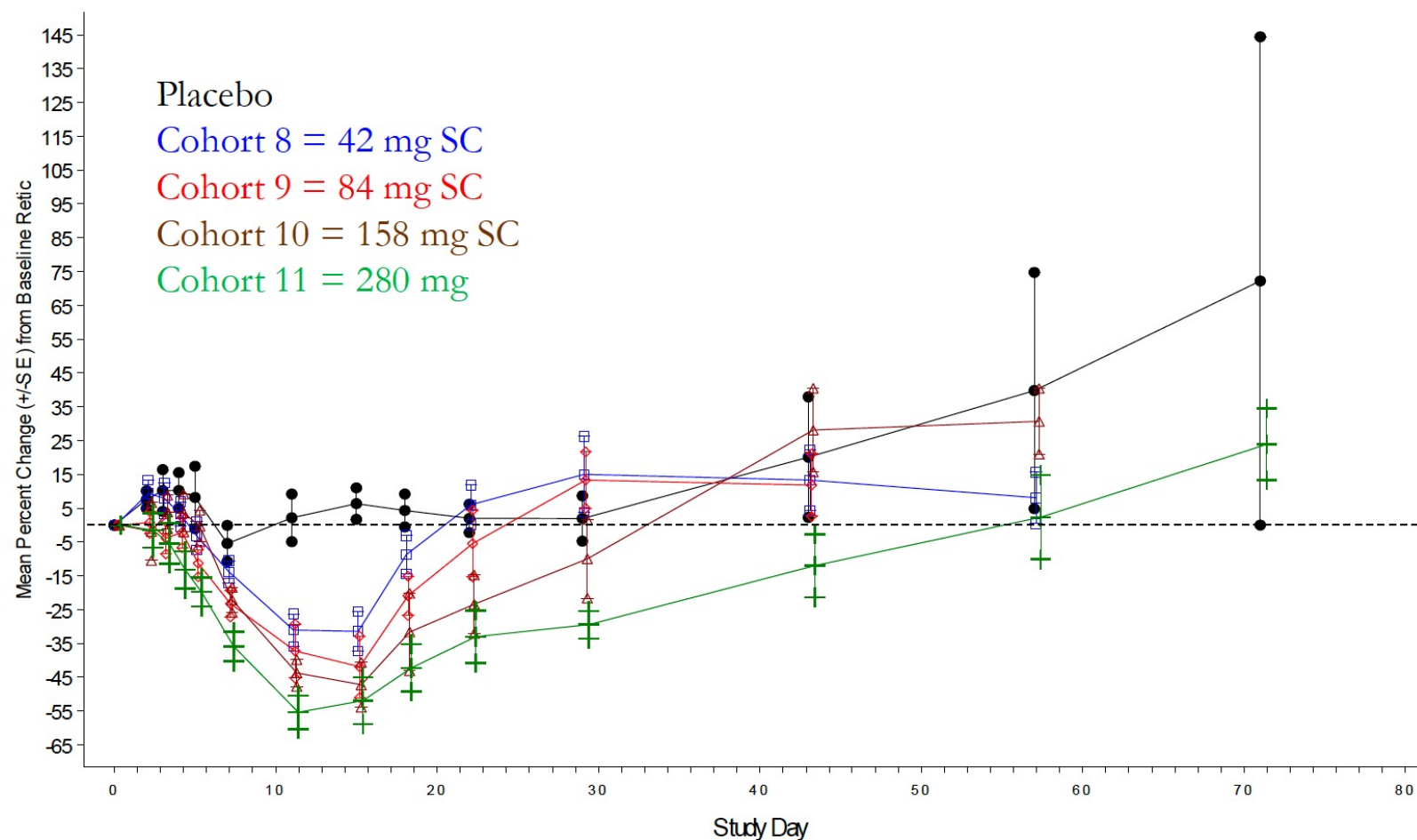
Briquilimab (JSP191/AMG191) Phase I SAD Study: Adverse events of interest

Adverse Event Description	Grade	Number of Subjects	Cohort	Comment
Dysgeusia	1/2	5	11 (280 mg SC)	All recovered over approximately 4-8 weeks
Upper Respiratory Tract Infection	1*	14	All	All 14 subjects on AMG 191, compared to 0 placebo subjects
Headache	2	4	4 (5 mg SC)	4 additional subjects dosed at 3:1 (AMG 191:placebo)
Acute Infusion Reaction (urticaria, facial erythema, transient decrease in blood pressure)	2	1	7 (5 mg IV)	Subject treated with promethazine 25 mg p.o., hydrocortisone 100 mg i.v., and 250 mL normal saline i.v. Adverse event duration = 6 days
Decreased neutrophil count	2	3	8 (42 mg SC)	8 additional subjects dosed at 3:1 (AMG 191:placebo)
Injection site reaction	1	Sentinel pair (1 active; 1 placebo)	9 (84 mg SC)	Acute injection site reaction subsided w/o treatment. Urticaria developed approx. 9 hrs post-dose and lasted >24hrs. Subject treated with oral doses of: prednisone 40 mg, promethazine 25 mg, cetirizine 10 mg, and paracetamol 1g.
Urticaria	2			

* All mild (Grade 1) AEs except for two Grade 2 events

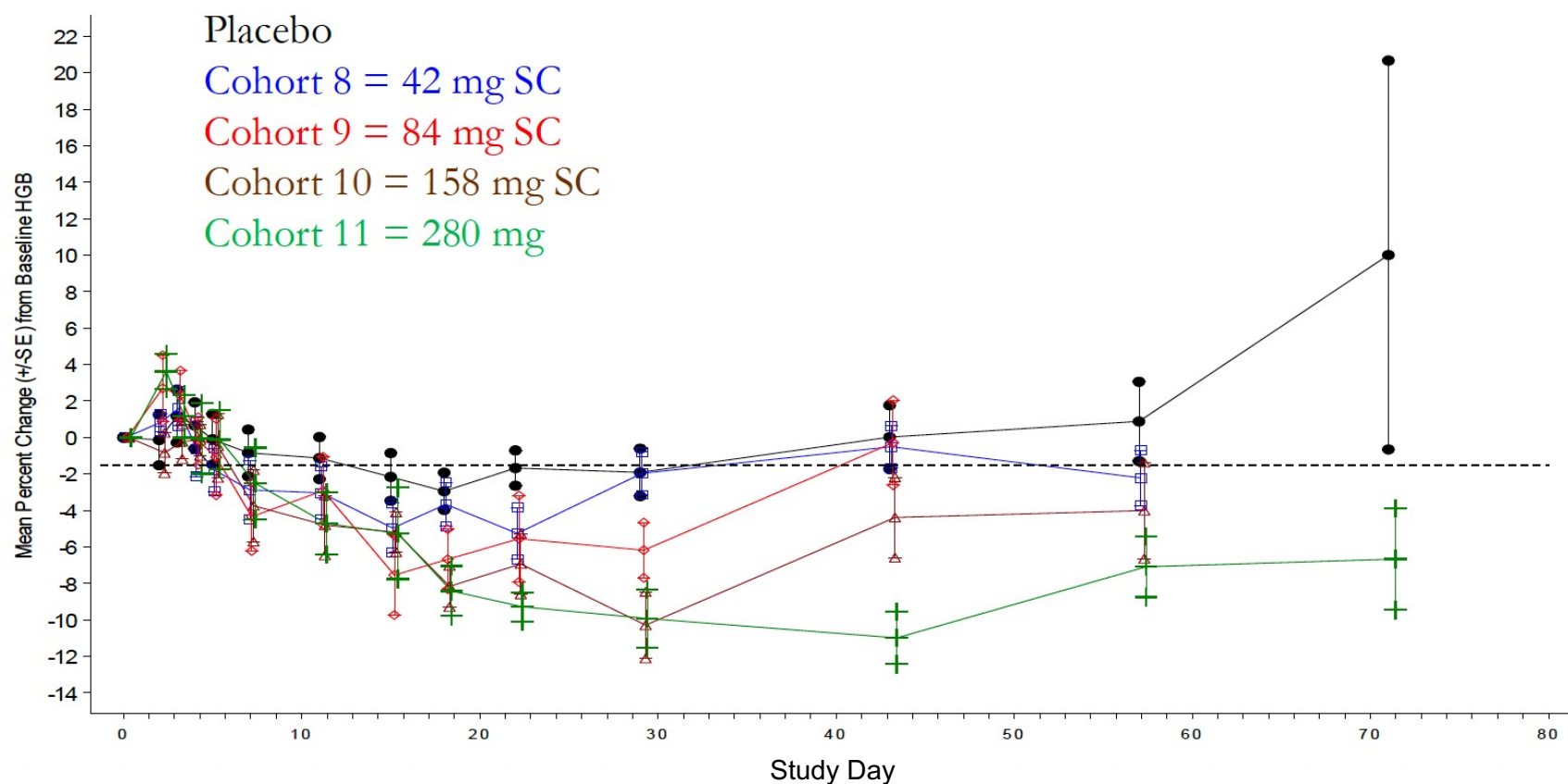
Phase I – Human SAD

Reticulocytes – Percent Change (Mean \pm SD) from Baseline: Cohorts 8, 9, 10 and 11 (Draft)



Phase I – Human SAD

Hemoglobin – Percent Change (Mean \pm SD) from Baseline: Cohorts 8, 9, 10, and 11 (Draft)



MAD – Grade 3 adverse event

One subject reported an allergic reaction 36 hours after 2nd dose in cohort 1 (14 mg q2w)

- Urticaria, facial angioedema, no respiratory symptoms, no clinically relevant changes in vital signs
- No previous report of urticaria or angioedema
- Treatment with promethazine 25 mg and prednisone 40 mg, cetirizine 10 mg
- Within 15 hours, urticaria symptoms resolved and angioedema improved (mild periorbital and upper lip swelling)
- Urticaria returned the following day despite continued cetirizine and resolved following additional treatment with prednisone
- Subject received no further briquilimab/placebo

Phase I: Healthy volunteer immunogenicity summary

- Pre-existing ADAs: 2/77 (Briquilimab (AMG191) treated subjects)
- Developing ADAs: 13/77
- Neutralizing ADAs: 8/77
- Post-dose ADAs seen in subjects given 20mg SC or greater, but no clear relationship to dose or results of punch biopsy wound model