Introduction to Priovant

September 2023

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therapeutics

Anchor Asset: Brepocitinib Potential First-In-Class Dual Inhibitor of TYK2 and JAK1

Brepocitinib Highlights

Novel Mechanism: Dual inhibition of TYK2 and JAK1 is expected to provide greater efficacy than agents that inhibit either alone in multiple highly inflammatory autoimmune diseases

Robust Clinical Data: Statistically significant and clinically meaningful benefit in all six placebo-controlled studies completed to-date with oral formulation (once-daily administration)

Well-Characterized Safety Profile: Exposure in >1,400 subjects and patients suggests safety profile consistent with approved JAK inhibitors

Distinctive Strategy: Orphan and specialty autoimmune franchise across diseases with high morbidity and mortality, few approved therapies (including no JAKi), and pathobiologies for which both TYK2 and JAK1 inhibition are expected to contribute to efficacy

Two Ongoing Registrational Programs: Single registrational phase 3 study in dermatomyositis (DM) with data expected in 2025, potential product launch in 2026; large, global phase 2B study in systemic lupus erythematosus (SLE), designed to serve as one of two registrational studies reading out 4Q 2023

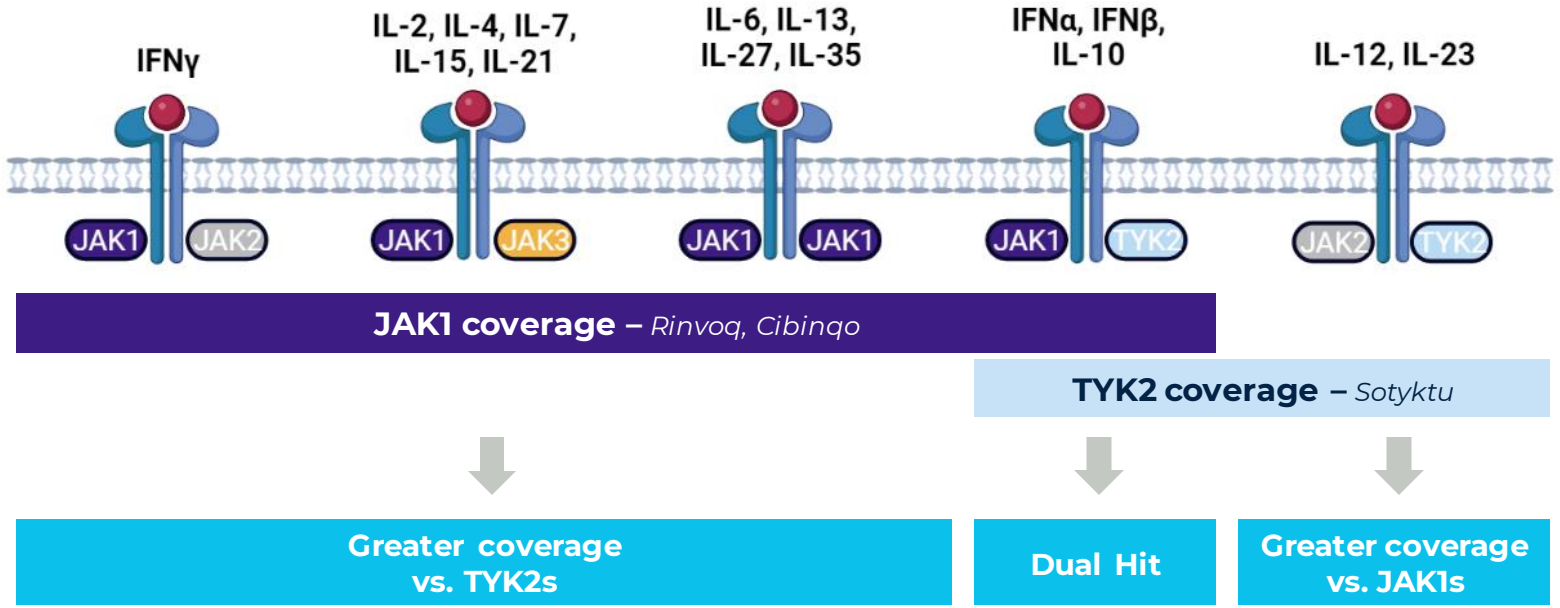
ORAL BREPOCITINIB

Optimized Pharmacology for Highly Inflammatory Diseases with Multiple Pathogenic Cytokines

Key insight: different JAKs for different diseases

Different diseases are driven by different combinations of cytokines, requiring inhibition of specific JAK isoforms to treat distinct indications most effectively

Field is currently focused on single isoform inhibitors (specifically TYK2 or JAK1)



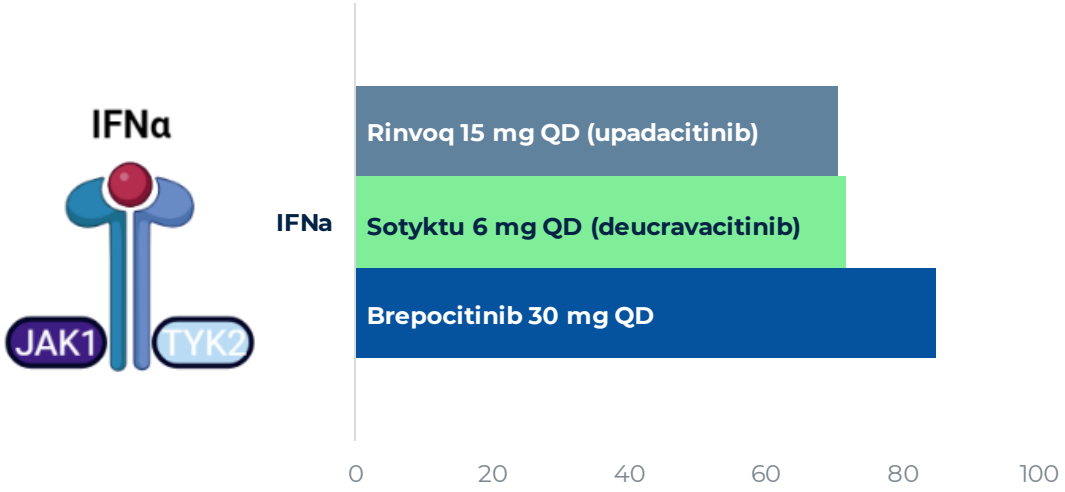
Brepocitinib was designed to target both TYK2 and JAK1

Hypothesis:
brepocitinib can provide best-in-class efficacy in indications mediated by the TYK2/JAK1 dimer and in diseases requiring broad cytokine coverage

In Vitro Data Support Mechanistic Benefits of Dual Inhibition of TYK2 and JAK1

Dual Hit

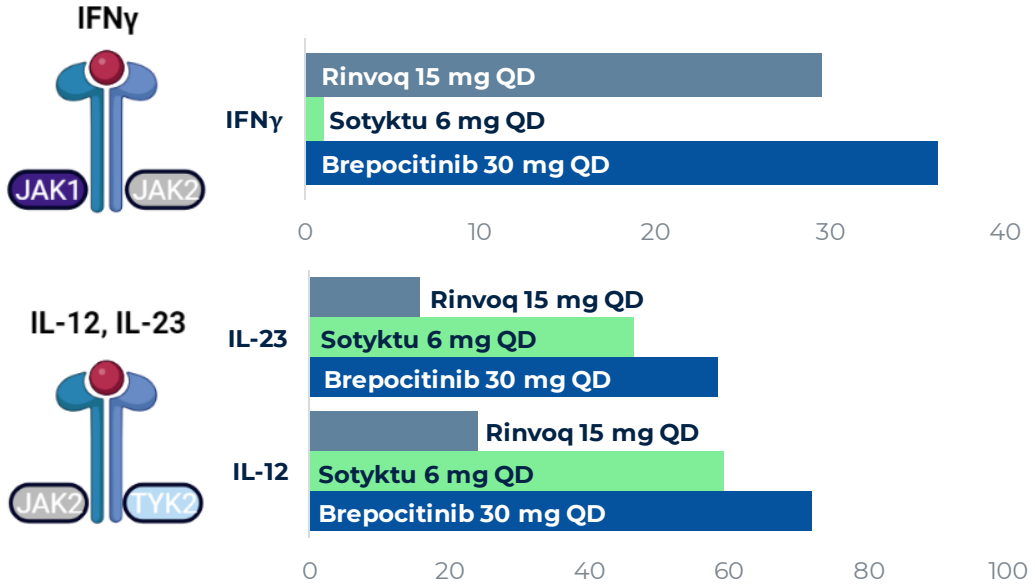
Percent Cytokine Inhibition at Modeled Exposures[1]



Brepocitinib may be able to achieve deeper Type I IFN suppression than is possible by targeting either TYK2 or JAK1 alone

Greater Coverage

Percent Cytokine Inhibition at Modeled Exposures[1]



Brepocitinib may recapitulate <u>in a single molecule</u> the cytokine suppression profiles of both the leading TYK2 and JAK1 agents

1) Figures reflect data generated from separate in vitro assays performed by Pfizer

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Statistically Significant and Clinically Meaningful Results Across Six Completed Phase 2 Studies

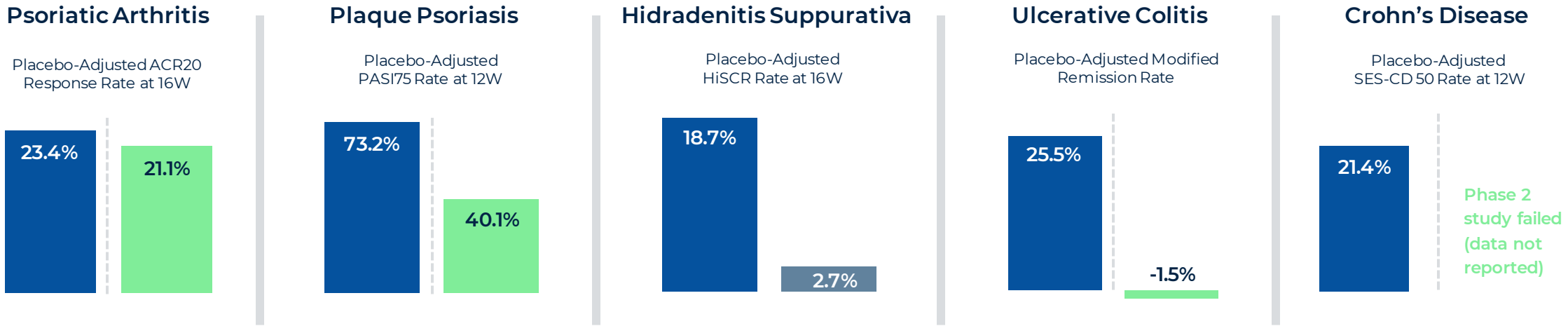
Study Population	N[1]	Brepocitinib Dose	Primary Endpoint Result	Statistical Significance
Psoriatic Arthritis Patients with active PsA	218	30 mg once daily	23.4% placebo-adjusted ACR20 RR at week 16	**P = 0.0197**
Plaque Psoriasis Patients with moderate-to-severe PsO	212	30 mg once daily	-10.1 placebo-adjusted CFB in PASI Score at week 12	**P < 0.0001**
Ulcerative Colitis Patients with moderate-to-severe UC	167	30 mg once daily	-2.28 placebo-adjusted CFB in Mayo Score at week 8	**P = 0.0005**
Alopecia Areata Patients with moderate-to-severe AA	94[2]	30 mg once daily[3]	49.18 placebo-adjusted CFB in SALT Score at week 24	**P < 0.0001[4]**
Hidradenitis Suppurativa Patients with moderate-to-severe HS	100	45 mg once daily[5]	18.7% placebo-adjusted HiSCR Rate at week 16	**P = 0.0298[4]**
Crohn's Disease Patients with moderate-to-severe CD	151	60 mg once daily[6]	21.4% placebo-adjusted SES-CD 50 Rate at week 12	**P = 0.0012[4]**

1) Overall study N represents patients randomized to all brepocitinib dose levels or placebo and excludes patients randomized to other agents
2) Includes patients from initial 24-week study period only
3) 60 mg QD for 4 weeks followed by 30 mg QD for 20 weeks
4) One-sided p-value (pre-specified statistical analysis)
5) Brepocitinib 45 mg once daily was the only brepocitinib dose evaluated in this study
6) Brepocitinib 60 mg once daily was the only brepocitinib dose evaluated in this study

CFB: change from baseline; RR: response rate

Clinical Experience Supports Value Of Dual TYK2/JAK1 Inhibition As Compared To Selective TYK2 Inhibition

In all indications where oral brepocitinib and a selective TYK2 inhibitor have been evaluated, brepocitinib's observed efficacy has been numerically greater



Psoriatic arthritis, plaque psoriasis, ulcerative colitis, and Crohn's disease: no direct head-to-head data available – cross-trial comparison of studies with different inclusion-exclusion criteria and design elements

Psoriatic Arthritis: brepocitinib 30 mg QD (Phase 2B – 16W; Data on file) vs. deucravacitinib 6 mg QD (Phase 2 – 16W, NCT03881059; Mease et al, Ann Rheum Dis 2022)
Plaque Psoriasis: brepocitinib 30 mg QD (Phase 2 – 12W; Data on file) vs. deucravacitinib 6 mg QD (Phase 3 POETYK-PSO-2 – 12W, NCT03611751; Armstrong et al, SDDS 2021 Poster 1042)
Hidradenitis Suppurativa: brepocitinib 45 mg QD vs ropsacitinib 400 mg QD (Phase 2 – 16W, NCT04092452; Data on file)
Ulcerative Colitis: brepocitinib 30 mg QD (Phase 2B – 8W; Data on file) vs. deucravacitinib 6 mg BID (Phase 2 LATTICE-UC – 12W, NCT03934216; Danese et al, ECCO 2022)
Crohn's Disease: brepocitinib 60 mg QD (Phase 2 – 12W, Data on file) vs. deucravacitinib (dose not specified, NCT03599622; BMY earnings deck Q2 2023)

Note: where more than one value is available for a competitor molecule (e.g., in the case of two Phase 3 studies), the higher value was selected for comparison to brepocitinib

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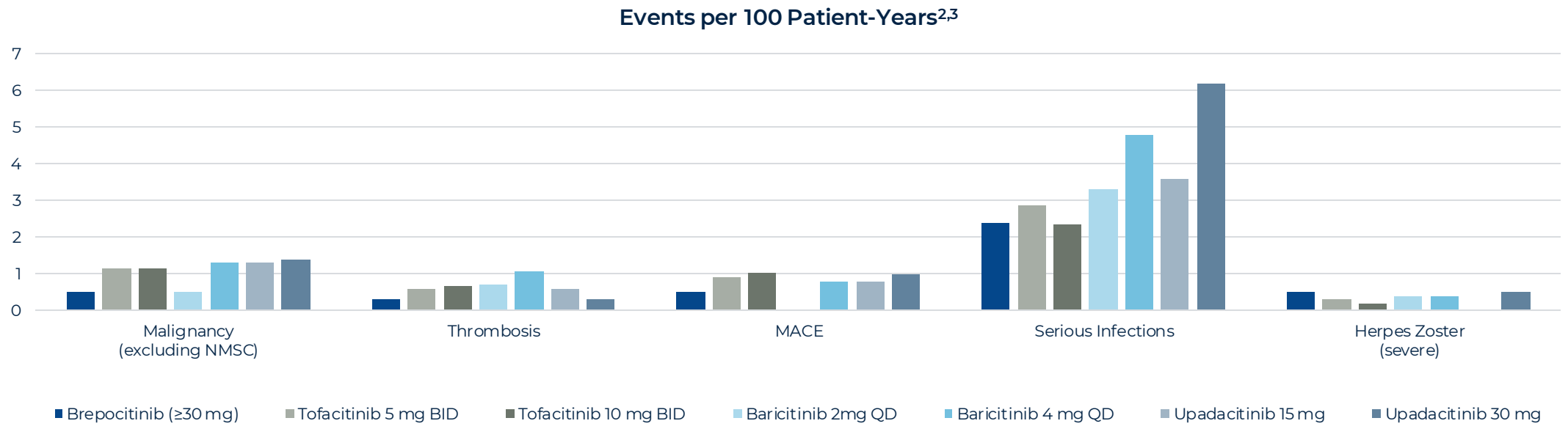
Safety Overview

Clinical experience in more than 1,400[1] exposed subjects and patients suggests a safety profile consistent with those of approved JAK inhibitors

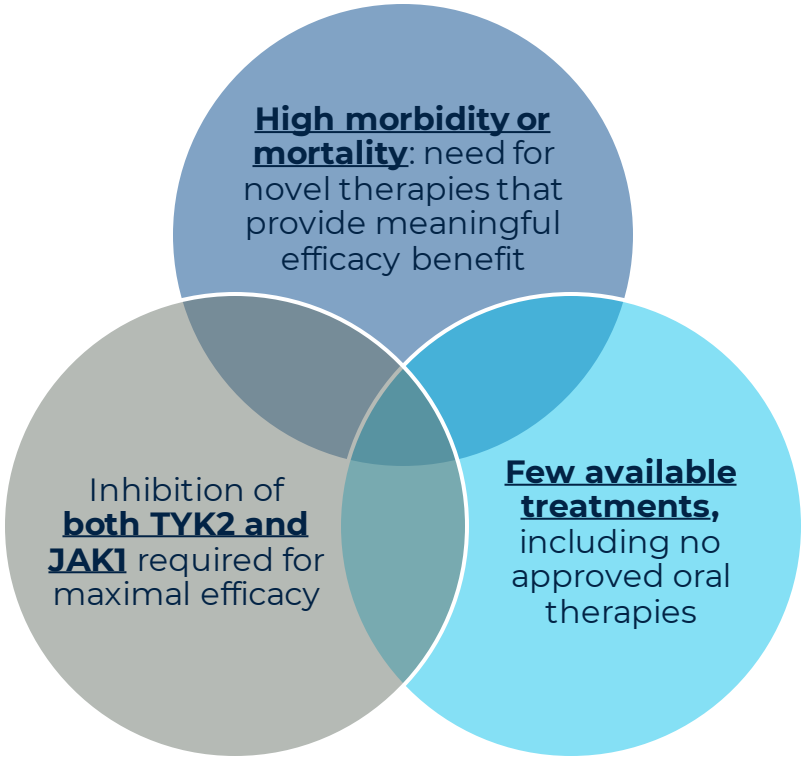
Comparable Incidence Rates of TEAEs of Interest vs. Approved JAKs

No direct head-to-head data available – cross-trial comparison of studies with different inclusion-exclusion criteria and design elements



1) Total exposure count includes 8 completed Phase 1 studies and 6 completed Phase 2 studies (PsA, PsO, AA, UC, Vitiligo, and HS) in addition to 2 ongoing Phase 2 studies (SLE and Crohn's).
2) Analysis includes only completed studies. Some patients received multiple dose levels. For any given dose level, patients are counted only once. Brepocitinib ≥30 mg comprises over 90% of aggregated patient-years of brepocitinib exposure in this analysis. Note that all data are shown as Events per 100-Patient Years except for tofacitinib, which is provided as Subjects with Event per 100 Patient-Years.
3) References for this chart provided in Appendix 1.

Priovant Focus: Indications with High Unmet Need and Tailored to Novel Mechanism of Dual TYK2/JAK1 Inhibition



Ongoing Registrational Programs

Dermatomyositis	• Large orphan indication with no NCEs approved in past 60 years and no other oral therapies in late-stage development • Registrational phase 3 study ongoing – TLR expected in 2025
Systemic Lupus Erythematosus (SLE)	• Potential to become leading oral therapy in SLE • Large phase 2B global study ongoing with data expected Q4 2023; designed as one of two registrational studies

Potential Expansion Opportunities

Hidradenitis Suppurativa	• Phase 2 results suggest potential for better efficacy than selective JAK1 inhibitors and comparable to leading biologics
Non-Infectious Uveitis	• PoC data expected Q1 2024

Dermatomyositis

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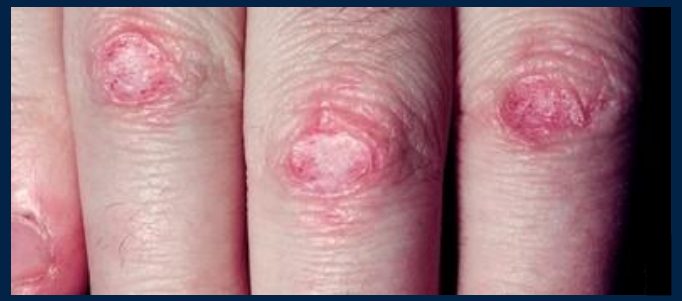
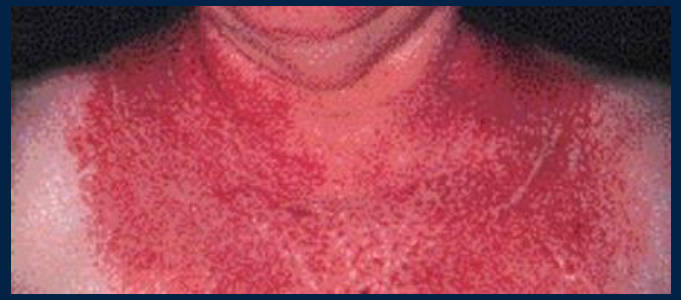


GOTTRON'S PAPULES

Red to violaceous papules overlying the knuckles



V-SIGN RASH

Irregular, patchy erythema on the chest

Dermatomyositis: A Debilitating Inflammatory Myopathy

Dermatomyositis is a rare, chronic, immune-mediated disease of the muscles and skin affecting approximately 37,000[1] adults in the United States

10-40%	Mortality at five years[2]
100%	Red, painful, itchy skin rash often disseminated across substantial body surface area
88%	Proximal muscle weakness[3], limiting activities of daily living (ADL)
42%	Interstitial lung disease[4], contributing to substantial morbidity
0	Other oral therapies in industry-sponsored late-stage development[5]
0	NCEs approved in last 60 years

1) PriovantTx estimates based on Reeder 2010, Smoyer-Tomic 2012, and claims analysis
2) Liu et al, Oncol Letters (2018)
3) Fardet et al, Medicine (2009)
4) Sun et al, Sem Arth Rheum (2021)
5) Phase 3 trials or adaptive Phase 2/3 trials

Dermatomyositis Prevalence

Increasing disease awareness and diagnosis has led to higher incidence and prevalence estimates over time; Priovant estimates there are approximately 37,000 adult DM patients in the United States

Ahlstrom et al (1993)
County-specific study in Sweden

Smoyer-Tomic et al (2012)
US healthcare claims data analysis from 2004-2008

Reeder et al (2010)
Medical record analysis of residents of Olmsted County, MN from 1976-2007

Priovant Claims Analysis
Analysis of Komodo claims database from 2016-2020

A prevalence rate of **~14/100,000** individuals implies approximately **37,000** adult patients in the United States with dermatomyositis

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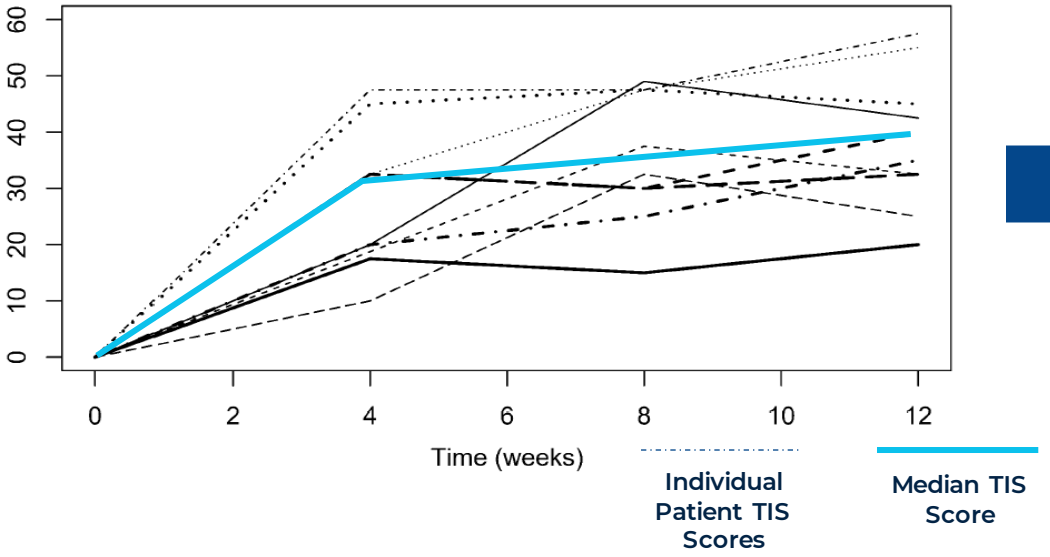
JAK INHIBITION IN DERMATOMYOSITIS

Clinical Proof-of-Concept with Tofacitinib

Investigator-initiated open-label study of tofacitinib in refractory dermatomyositis demonstrated activity comparable to that of IVIg, the only approved non-corticosteroid/corticotropin therapy for dermatomyositis

Study of Tofacitinib in Refractory Dermatomyositis (STIR)[1]
Total Improvement Scores



STIR Study
TIS Outcomes

Open-label, single-arm

100%
TIS20 Response Rate at Week 12

40
Median TIS Score at Week 12[3]

ProDERM Phase 3 Study (IVIg)[2]
TIS Outcomes

Double-blind, placebo-controlled

79%
TIS20 Response Rate at Week 16

43
Mean TIS Score at Week 12[3]

Cross-study comparison; no head-to-head data available

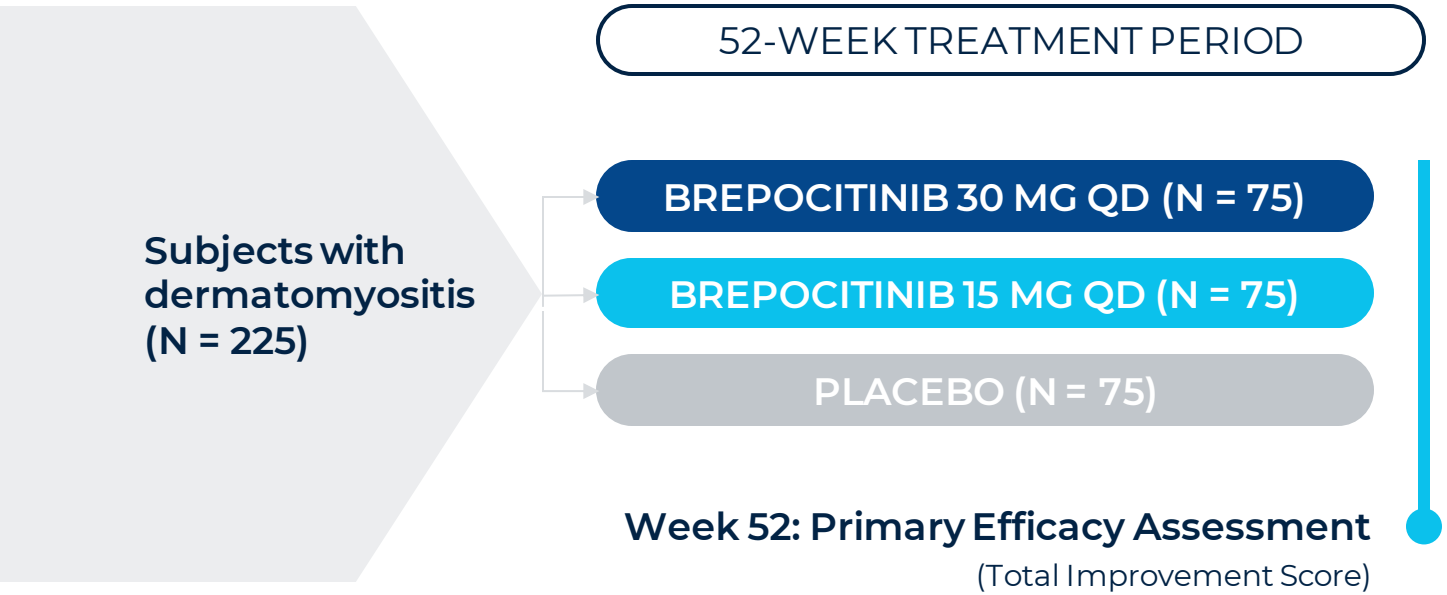
Clinical PoC further validated by extensive JAK case report literature[4]

1) Paik et al, Arth Rheum (2020)
2) Aggarwal et al, NEJM (2022)
3) STIR study: median is the only statistical analysis provided; ProDERM study: mean is the only statistical analysis provided
4) Paik et al, Clin Exp Rheum (2022)

DERMATOMYOSITIS

Single Phase 3 Study

Phase 3 program will use the total improvement score (TIS), a validated myositis improvement index that served as basis of approval for IVIg. Topline results expected in 2025 → potentially the next approved drug of any modality for dermatomyositis.



Eligible Patients

Adult subjects with active dermatomyositis who are refractory or intolerant to at least one standard-of-care therapy

Primary Endpoint

Mean Total Improvement Score (TIS) at Week 52

Secondary Endpoints

- Proportion of subjects achieving TIS ≥ 40 points
- Manual Muscle Testing (MMT-8)

Safety Endpoints

Incidence of treatment-emergent AEs, SAEs, AEs of special interest, clinically significant vital signs or lab abnormalities

BREPOCITINIB

Only Late-Stage Oral Therapy in Industry-Sponsored Development for DM

Molecule/Program	Sponsor	Route of Administration	Development Stage
Global Development Programs			
Brepocitinib Dual TYK2/JAK1 inhibitor	priovant	Oral, once-daily	Phase 3
OCTAGAM 10% Human immunoglobulin (IVIg)	octapharma	IV Infusion	Approved
IgPro20 Human immunoglobulin	CSL Behring	Subcutaneous infusion	Phase 3
Dazukibart Anti-IFN-β mAb	Pfizer	IV infusion	Phase 3[1]
Efgartigimod Anti-FcRn antibody fragment	argenx	Subcutaneous injection	Phase 2/3[2]
ULTOMIRIS (ravulizumab) Anti-C5 mAb	ALEXION	IV Infusion	Phase 2/3
Ex-US Development Programs			
OLUMIANT (baricitinib) JAK1/2 inhibitor	Investigator-Initiated	Oral, once-daily	Phase 3[3] – France only

Includes ongoing and planned (announced) company-sponsored Phase 3 studies in dermatomyositis; excludes glucocorticoids and corticotropin injection

1) Basket study in PM & DM.
2) Basket study in multiple myositis subtypes (PM, DM, IMNM).
3) N = 62 patients

Systemic Lupus Erythematosus

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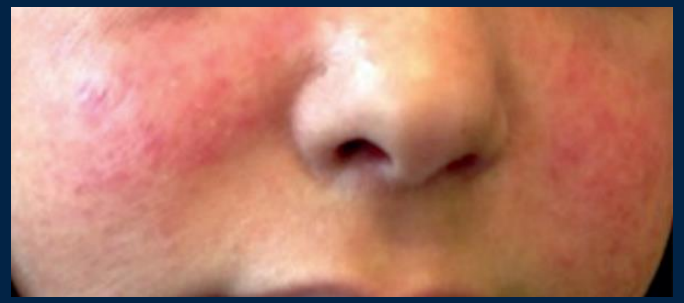
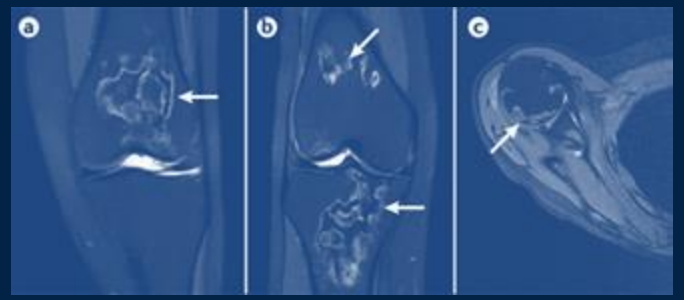


MALAR (BUTTERFLY) RASH

Typical skin complication found in up to 50% of patients with SLE



OSTEONECROSIS OF JOINTS

Complication of long-term OCS use in SLE

SLE: A Heterogeneous Connective Tissue Disease

SLE is a chronic autoimmune disease characterized by elevated levels of proinflammatory cytokines, autoantibodies, and autoreactive cell types that affects up to 300,000[1] people in the United States

50-60%	Patients with moderate or severe disease[2]
Most Common Symptoms	Rash, arthritis, fatigue, hematologic abnormalities, cardiorespiratory involvement[3]
2	New approved drugs in >20 years

Benlysta and Saphnelo have combined annual revenue >$1.5B despite modest efficacy (low teens pbo-adj delta on SRI-4)

Images adapted from Kaul et al (2016)
1) Centers for Disease Control
2) Priovant SLE claims analysis
3) Kaul et al, Nat Rev Dis Primers (2016)

Dual TYK2/JAK1 Inhibition May Overcome Single-Agent Limitations to Treating Lupus

Multiple interconnected pathways drive SLE biology: T-cells, B-cells, and IFN signaling

- Selective TYK2s and JAK1s address certain of these pathways, **but not all three**

Brepocitinib is **uniquely** suited to address all three axes simultaneously:

- Modulate T-cell activity via IL-12/IL-23 (**TYK2**)
- Modulate B-cell activity via IL-6, IL-7, and IL-21 (**JAK1**)
- Directly suppress type I IFN signaling (**TYK2** & **JAK1**)

Potential for brepocitinib superiority in lupus further supported by cross-trial comparisons vs. selective TYK2s and JAK1s in other indications

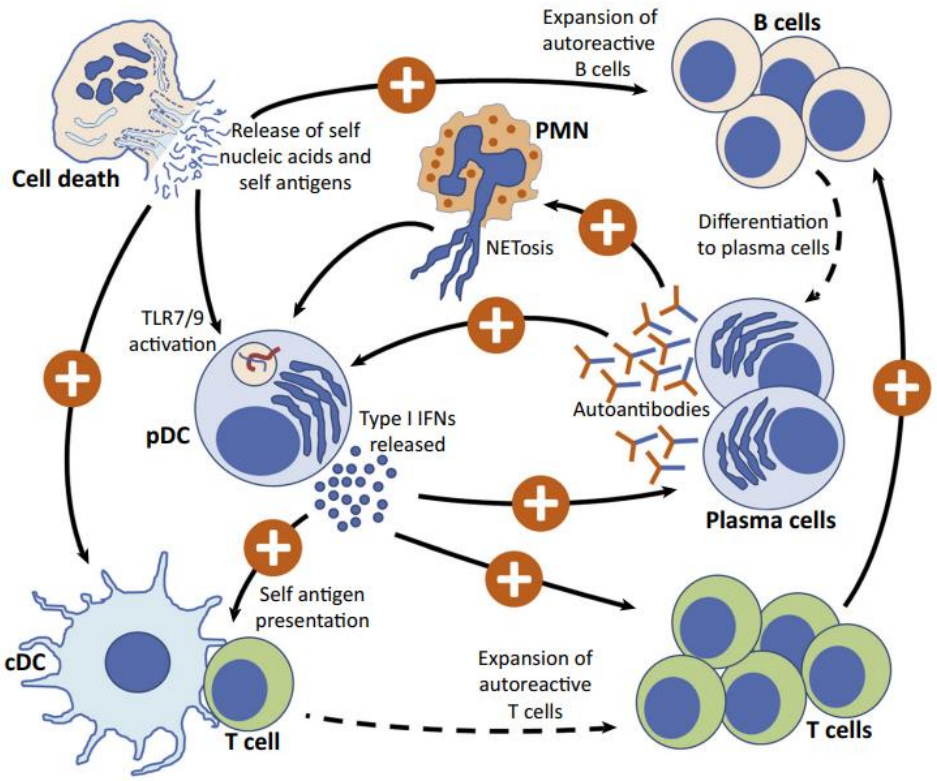
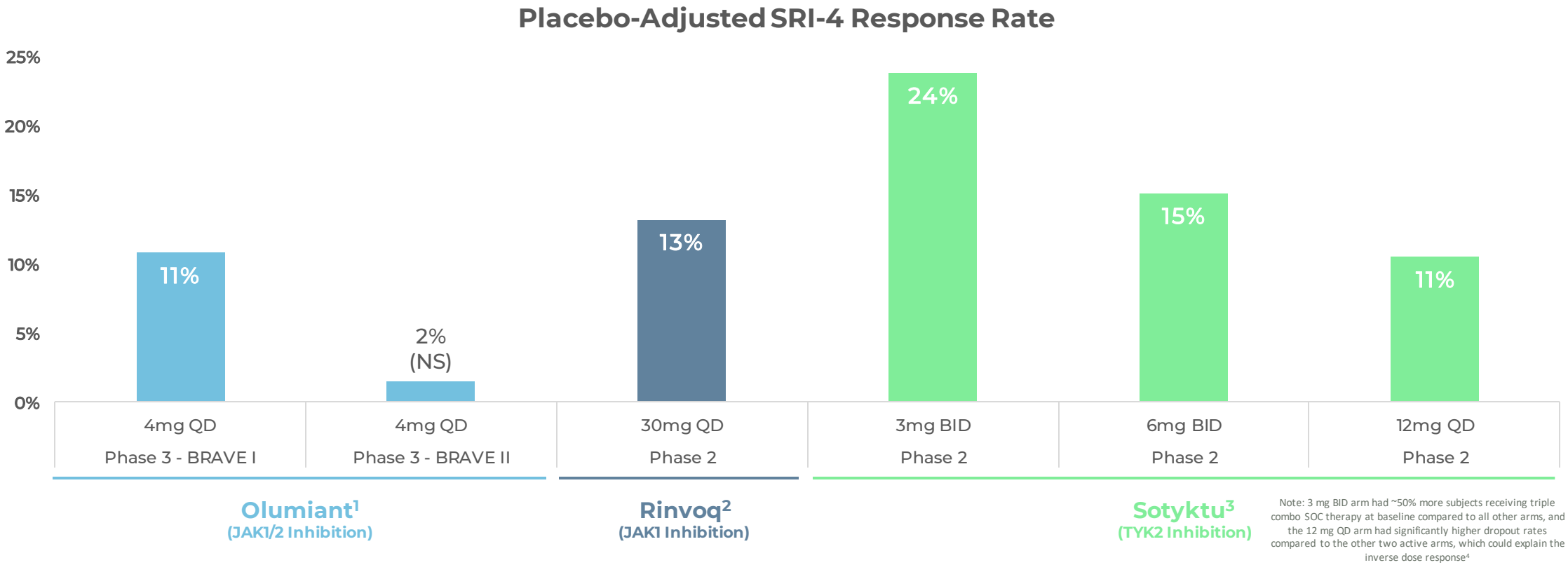


Figure adapted from Ganguly et al, Trends in Immunology (2017)

JAKS IN SLE

Clinical Proof-of-Concept

Through its novel dual TYK2/JAK1 mechanism of action, brepocitinib may be able to improve upon the efficacy shown by TYK2 or JAK1 inhibition alone, potentially stacking efficacy by combining independent axes of effect



Figures reflect cross-trial comparison and not results from a head-to-head study. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

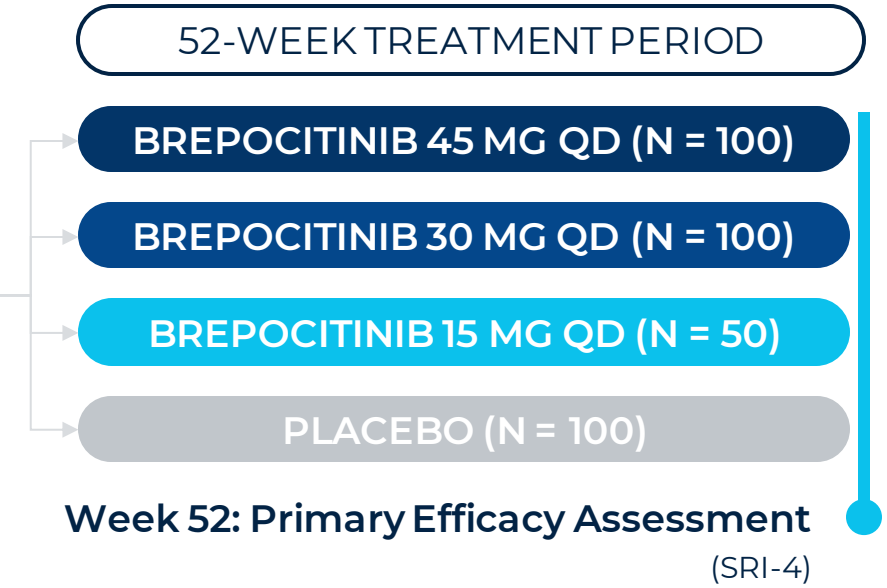
NS: not significant
1) Olumiant P3 data at Week 52 (Morand et al, Lancet 2023). Results for BRAVE-II P3 study were not statistically significant
2) Rinvoq P2 data at Week 48: Merrill et al, EULAR Abstract OP0139 (2023)
3) Sotyktu P2 data at Week 32: EULAR 2022 Abstract LB0004
4) EULAR 2022 Presentation LB0004

ONGOING

Phase 2B Study in SLE

Large phase 2B study was designed to serve as 1 of 2 potential registration-enabling studies; primary endpoint is SRI-4, a validated SLE improvement index; top-line data anticipated in Q4 2023

Subjects with active, moderate/ severe SLE (N = 350)



Eligible Patients

Adult subjects with moderate to severe active lupus who are receiving a stable regimen of SOC immunosuppressive agents

Primary Endpoint

Systemic Lupus Responder Index change of 4 (SRI-4) at Week 52

Secondary Endpoints

- Time to first severe flare
- Lupus Low Disease Activity State (LLDAS)
- Reduction in steroid usage
- Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI-A) response

Safety Endpoints

Incidence of treatment-emergent AEs, SAEs, AEs of special interest, clinically significant vital signs or lab abnormalities

As study was ongoing at time of Priovant-Pfizer transaction, Pfizer remains study sponsor

Key Competitive Landscape in SLE

Molecule/Program	Sponsor	Route of Administration	Development Stage
Orals			
Brepocitinib TYK2/JAK1 inhibitor	priovant	Oral, once-daily	Phase 2B
Deucravacitinib Allosteric TYK2 inhibitor	Bristol Myers Squibb	Oral, once/twice-daily	Phase 3
Cenerimod S1P1 Receptor Modulator	idorsia	Oral, once-daily	Phase 3
Upadacitinib JAK1 inhibitor	abbvie	Oral, once-daily	Phase 3
Biologics			
Obinutuzumab Anti-CD20 mAb	Roche	IV Infusion	Phase 3
Litifilimab Anti-BDCA2 mAb	Biogen	SC injection	Phase 3
Dapirolizumab pegol Anti-CD40L mAb	UCB Pharma	IV Infusion	Phase 3
Telitacicept Anti-BLyS/APRIL mAb	RemeGen	SC injection	Phase 3
Ianalumab Anti-BAFF-R mAb	NOVARTIS	SC injection	Phase 3

Source: Global Data; list of competitive programs is not exhaustive

Other Indication Overviews: HS & NIU

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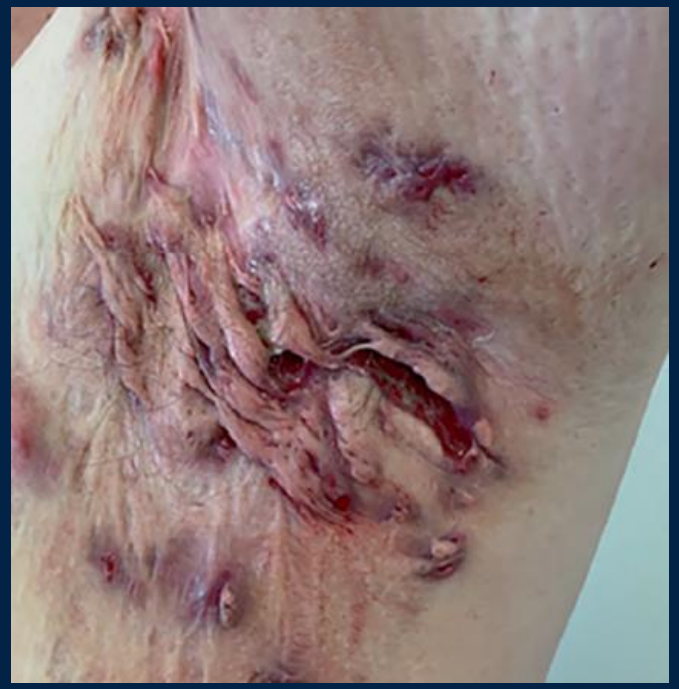


HIDRADENITIS SUPPURATIVA

Extensive skin involvement with fistula (tunnel) formation and scarring in a Hurley Stage III patient

HIDRADENITIS SUPPURATIVA (HS)

A Severe, Disfiguring Inflammatory Skin Disease

HS is an inflammatory skin disease characterized by the formation of painful nodules and abscesses in intertriginous zones (skin folds) that affects approximately 170,000 individuals in the United States[1]

Key Symptoms	Nodule, abscess, and tunnel formation in skin folds
Comorbidities	Metabolic syndrome[2], spondylarthritis[3], inflammatory bowel disease[4]
>2x	Increased suicide risk for patients living with HS compared to the general population[5]

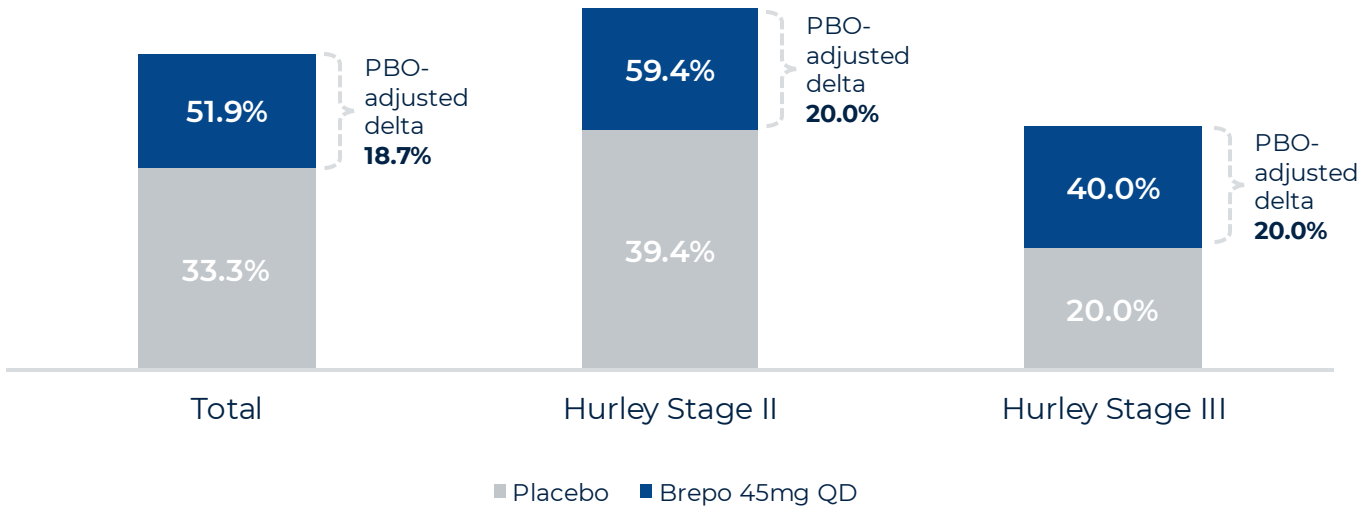
1) Estimates for moderate/severe HS only; based on Phan et al, Biomed Derm 2020
2) Sabat et al, PLoS One (2012)
3) Shlyankevich et al, J Am Acad Derm (2014)
4) Deckers et al, J Am Acad Derm (2017)
5) Thorlacious et al, J Invest Dermatol 2018

BREPOCITINIB IN HS

Clinical Proof-of-Concept

In a 16-week Phase 2 study of brepocitinib in 100 patients with moderate-to-severe hidradenitis suppurativa, brepocitinib was associated with a significantly higher HiSCR response rate than placebo

Patients Achieving HiSCR Response at Week 16
Response Rate by Hurley Stage



27 of 52 (51.9%) brepocitinib-treated patients achieved HiSCR compared to 16 of 48 (33.3%) placebo-treated patients (p = 0.0298)

Effect sizes were consistent across Hurley stages and among patients who had previously demonstrated inadequate response to TNF inhibitors

Key secondary endpoints, including total AN count and time to first flare, also achieved statistical significance

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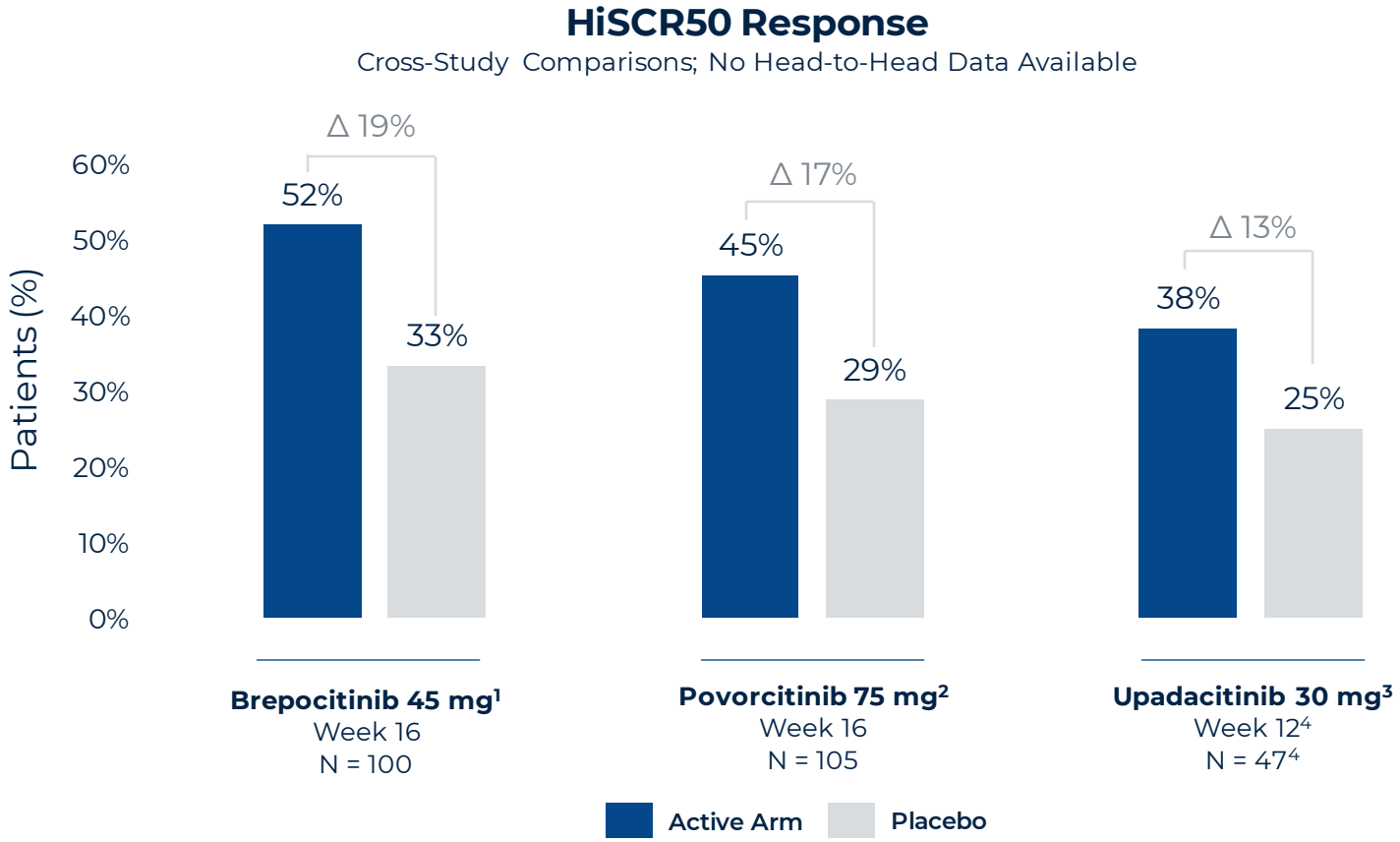
BREPOCITINIB IN HS

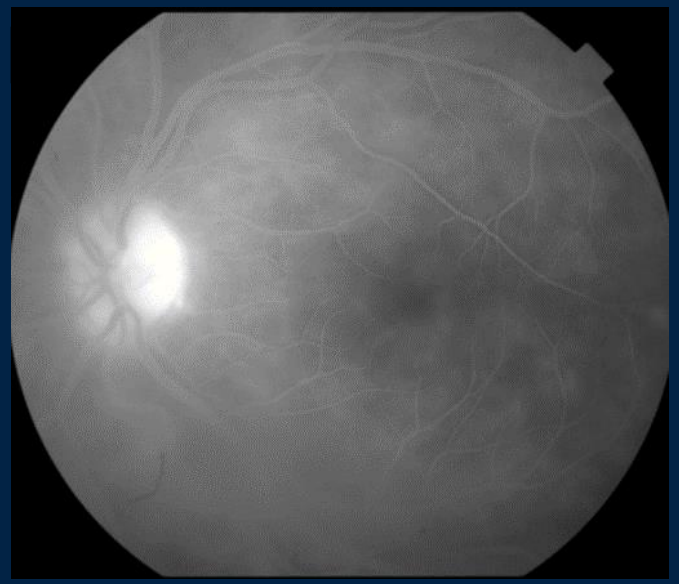
Dual TYK2/JAK1 Inhibition May Provide Greater Efficacy than Inhibition of JAK1 Alone



Absolute and placebo-adjusted results numerically better than other oral agents and comparable to leading biologics

Results suggest that addition of TYK2 inhibition may contribute to better efficacy in HS; inhibition of Th17 (IL-23) axis in HS extensively validated by IL-17-targeting biologics (eg Cosentyx)

1) Kimball et al, EADV 2022
2) Kirby et al, EADV 2022 Poster P0004
3) Kimball et al, AAD 2023 Poster 43799
4) Primary endpoint of upadacitinib Phase 2 study was HiSCR at 12 weeks. N represents active arm only as primary statistical analyses were performed in reference to prespecified historical placebo control (N = 259). Active arm performance at Week 16 for UPA30 was 40%.



POSTERIOR SEGMENT INFLAMMATION

Illustrative fluorescein angiography image indicating diffuse areas of capillary leakage and disc hyperfluorescence

NON-INFECTIOUS UVEITIS (NIU)

A Sight-Threatening Ocular Disease

Non-anterior NIU is a chronic autoimmune disease characterized by intraocular inflammation that affects approximately 75,000[1] people in the United States

30,000	New cases of legal blindness attributable to NIU in the US each year[1]
Most Common Symptoms	Light sensitivity, pain, redness and floaters
Etiology	Idiopathic, or secondary to systemic autoimmune diseases[2]
1	Approved targeted therapy (Humira)

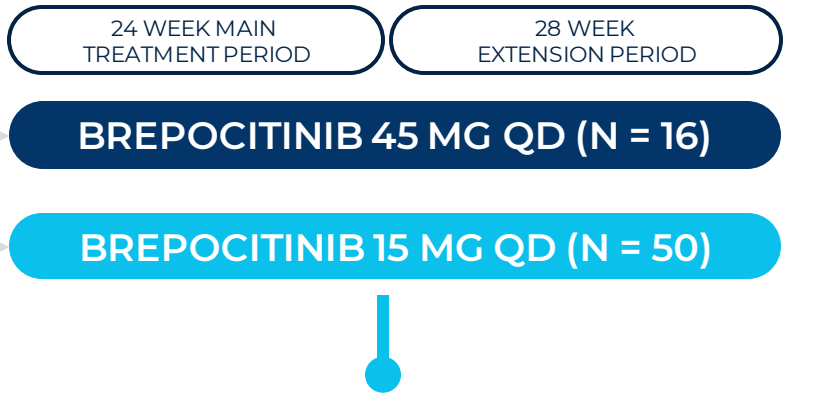
1) Thorne et al, JAMA Ophthalmol. (2016)
2) De Smet et al, Prog in Ret and Eye Res (2011)

ONGOING

Phase 2 Study in NIU

Two dose-arm study designed to provide rapid validation of TYK2/JAK1 approach in NIU. Enrollment now complete; topline data expected in Q1 2024

Subjects with active, non-anterior NIU (N = 24)



Week 24
Primary Efficacy Assessment
(Treatment Failure)

Eligible Patients

Adult subjects with active intermediate, posterior, or panuveitis

Primary Endpoint

Proportion of subjects meeting treatment failure criteria on or after Week 6 up to Week 24

Predefined success criterion: 45 mg treatment failure rate of no greater than 70%*

Other Endpoints

- Treatment failure rate at Week 52
- Change in best corrected visual acuity

* Assumed synthetic placebo rate of 80-90%; based on historical placebo rates, adjusted for more aggressive mandatory corticosteroid taper in brepocitinib study

prIovant


priovant
therapeutics

APPENDIX 1

Additional References

Slide 5: Clinical Program and Safety Overview

TEAEs Incidence Rate Chart

Brepocitinib source data: Brepocitinib Clinical Study Reports (Priovant data on file)

Tofacitinib source data: Pfizer ORAL Surveillance Study (malignancy, MACE); Cohen et al (2020) (thrombosis, serious infections, and herpes zoster)

Baricitinib source data: FDA Risk Review (May 2018) (thrombosis); Smolen et al (2019) (malignancy, MACE, serious infections, herpes zoster)

Upadacitinib source data: FDA Risk Review (Aug 2019)

Multiple Slides: Comparative Cytokine Inhibition

Cytokine Inhibition at Modeled Therapeutic Exposures

Methodology: Using the modeling approach described in Dowty et al, Pharmacol Res Perspect (2019), estimated ICxx (% levels of cytokine inhibition) values were calculated for brepocitinib, tofacitinib, baricitinib, upadacitinib, deucravacitinib, and filgotinib at various therapeutic dose levels.

Brepocitinib source data: Brepocitinib Investigator's Brochure; Priovant data on file

Tofacitinib source data: Dowty et al, Pharmacol Res Perspect (2019); Dowty et al, J Pharmacol Exp Ther (2013)

Baricitinib source data: Dowty et al, Pharmacol Res Perspect (2019)

Upadacitinib source data: Dowty et al, Pharmacol Res Perspect (2019); EMA Risk Assessment Report – RINVOQ (June 2021)

Deucravacitinib source data: Priovant data on file; Chimalakonda et al, Dermatol Ther (2021); Wrobleski et al, J Med Chem (2019)

Filgotinib source data: Dowty et al, Pharmacol Res Perspect (2019); Scholze et al, PLoS ONE (2014)