Corporate Presentation

September 2024

disc medicine

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This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including, but not limited to, express or implied statements regarding Disc’s expectations with respect to its AURORA Phase 2 and BEACON Phase 2 clinical trials of bitopertin and the results thereof, its Phase 1b/2 clinical trial of DISC-0974 in patients with MF and NDD-CKD patients with anemia, its initial SAD data in its Phase 1 clinical trial of DISC-3405 in healthy volunteers; projected timelines for the initiation and completion of its clinical trials, anticipated timing of release of data, and other clinical activities; and Disc’s belief about operating expenses and that it will have capital to fund Disc well into 2027. The use of words such as, but not limited to, “believe,” “expect,” “estimate,” “project,” “intend,” “future,” “potential,” “continue,” “may,” “might,” “plan,” “will,” “should,” “seek,” “anticipate,” or “could” or the negative of these terms and other similar words or expressions that are intended to identify forward-looking statements. Forward-looking statements are neither historical facts nor assurances of future performance. Instead, they are based on Disc’s current beliefs, expectations and assumptions regarding the future of Disc’s business, future plans and strategies, clinical results and other future conditions. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

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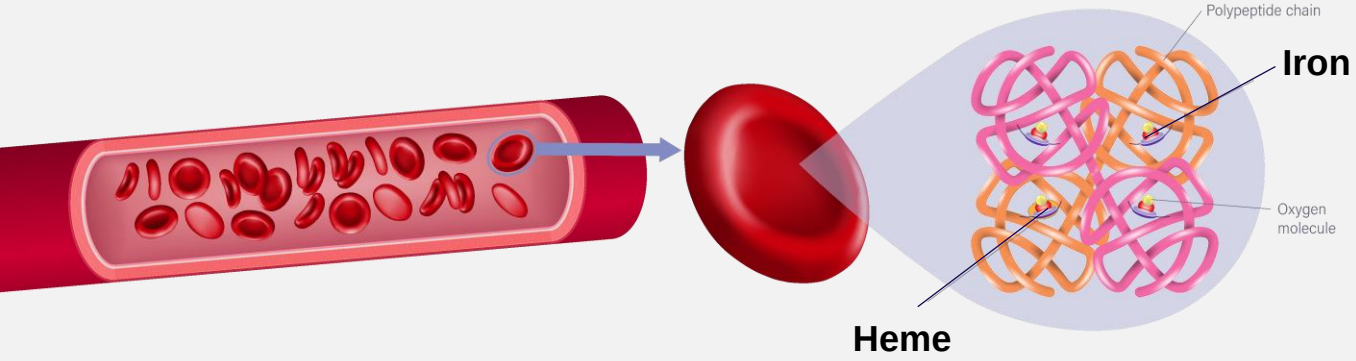
Bitopertin, DISC-0974, and DISC-3405 are investigational agents and are not approved for use as therapies in any jurisdiction worldwide

Targeting Fundamental Pathways of Red Blood Cell Biology using Validated Mechanisms



Iron and heme metabolism are critical pathways in hematology with genetically-validated targets

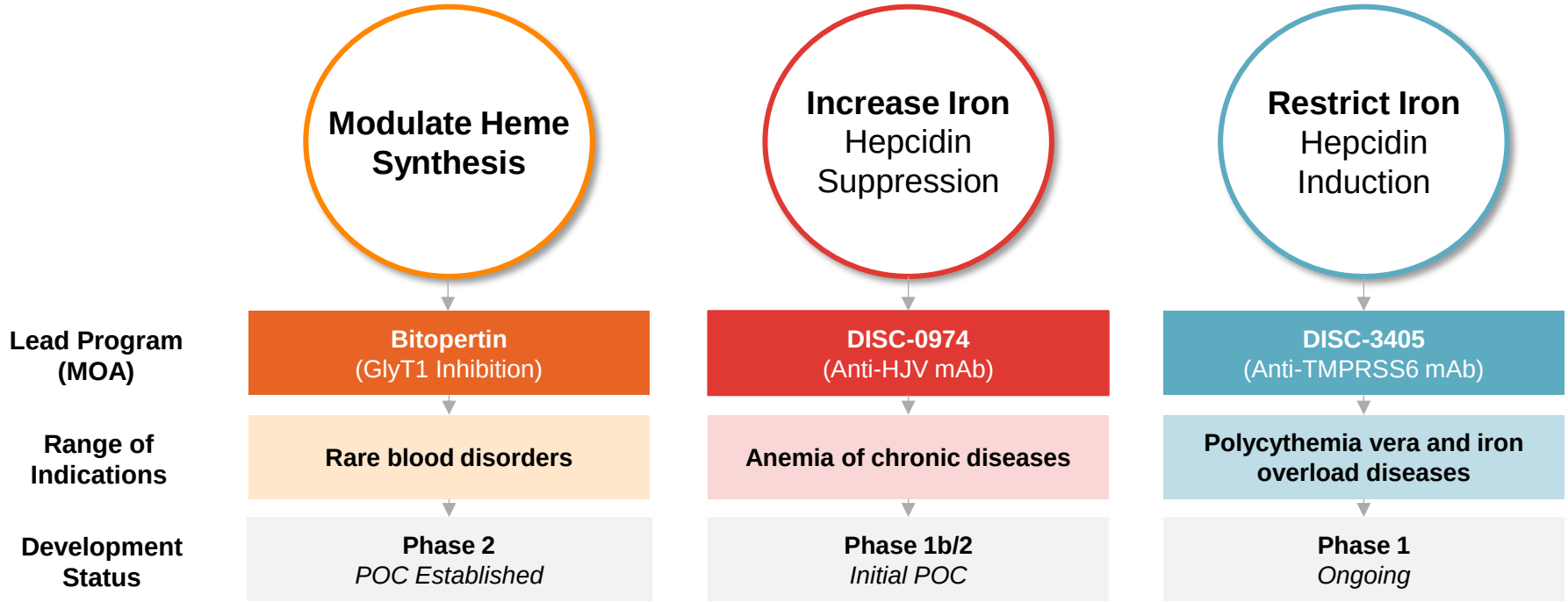
Key points of intervention across a wide range of diseases

Spectrum of Hematologic Diseases Addressable by Disc Portfolio

Severe Rare (000s)			*Moderate Prevalence (100K+)*						*Widely Prevalent (MMs)*
Diamond-Blackfan Anemia	**Erythropoietic Porphyrias**	Beta-Thalassemia	**Anemia of Myelofibrosis**	**Myelodysplastic Syndromes**	Sickle Cell Disease	Polycythemia Vera	Hereditary Hemochromatosis	IBD Anemia	**CKD Anemia**

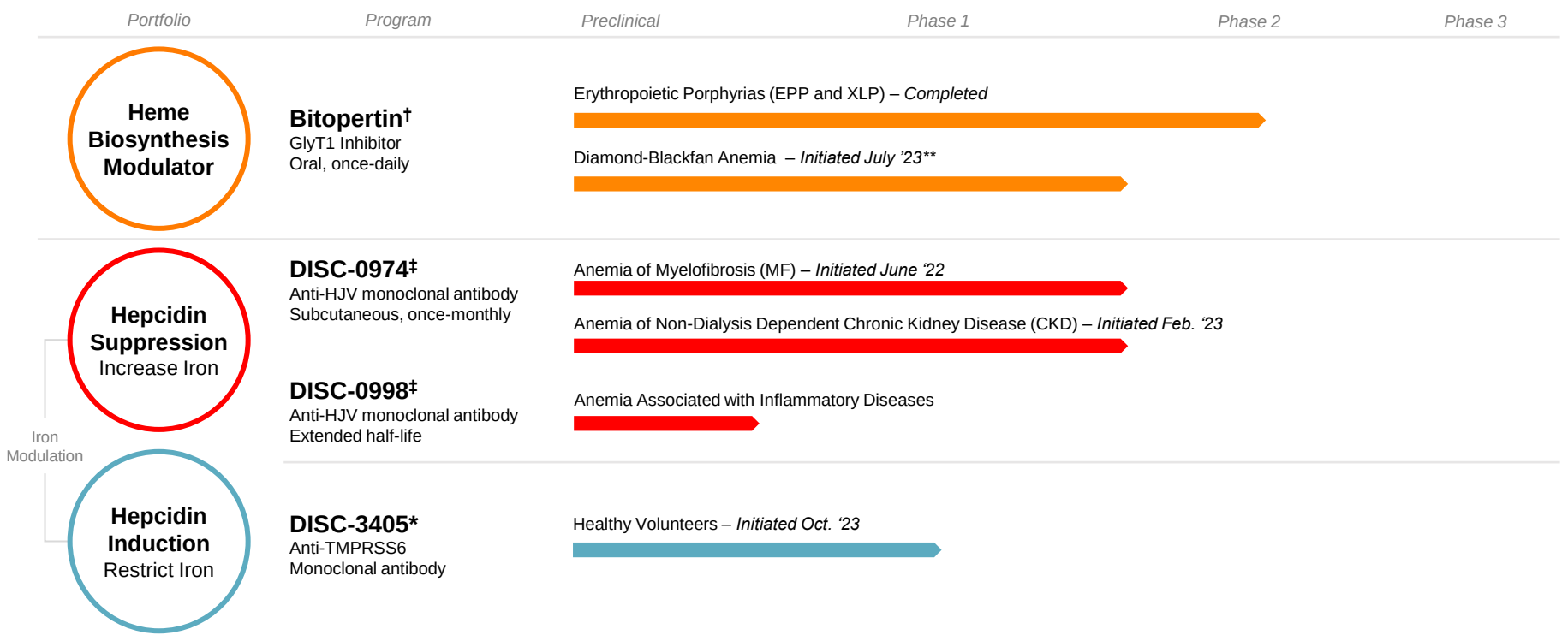
Bold = ongoing Disc trial

By Targeting Heme and Iron, Disc's Portfolio Can Address a Wide Range of Hematologic Disorders



Disc's Hematology-Focused Pipeline

Multiple programs in development with pipeline-in-a-product potential



† Bitopertin in-licensed from Roche; ‡ DISC-0974 and DISC-0998 in-licensed from AbbVie; *in-licensed from Mabwell; formerly MWTX-003; **IIT with the NIH

Projected Upcoming Milestones and Events

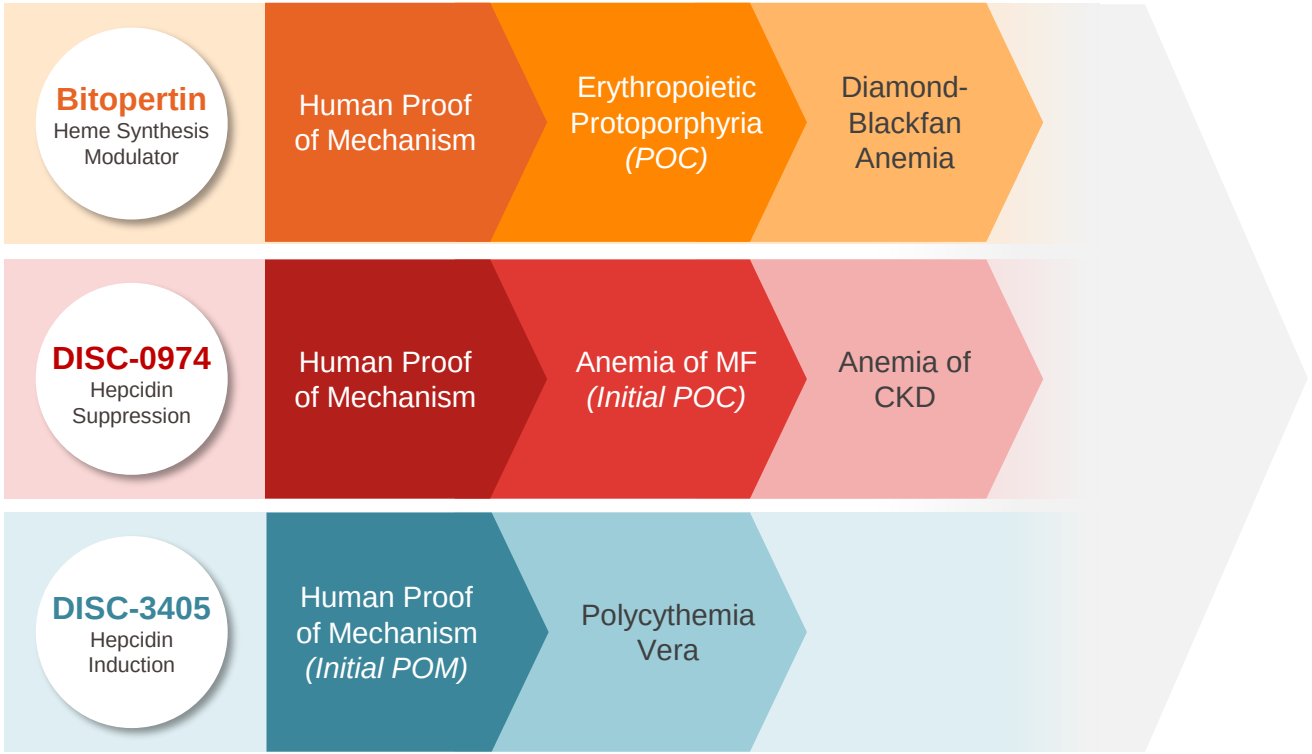
Multiple additional data catalysts anticipated in the next 18 months

Program	Indication	H1 2024	H2 2024	2025
Bitopertin Heme Synthesis Modulator	**Erythropoietic Porphyrias (EPP and XLP)**	• Phase 2 AURORA Data (March-April)	• Guidance from End of Ph 2 Meeting (Q4)	• Phase 3 Initiation Pending Regulatory Feedback
	Diamond-Blackfan Anemia (DBA)		• Initial Phase 2 Data	
DISC-0974 Hepcidin Suppression	**Anemia of Myelofibrosis (MF)**	• Updated Phase 1b Data	• Final Phase 1b Data • Initiate Phase 2 Study	• Phase 2 Topline Data
	Anemia of Chronic Kidney Disease (CKD)		• Phase 1b SAD Data (hemoglobin)	• Phase 1b/2a Multiple Dose Topline Data
DISC-3405 Hepcidin Induction	**Polycythemia Vera and Diseases of Iron Overload/ Ineffective Erythropoiesis**	• Phase 1 SAD Data	• Phase 1 SAD/MAD Data	• Phase 2 in PV Initiation

Supported by a strong cash position with runway well into 2027

SAD = single ascending dose; MAD = multiple ascending dose; PV = polycythemia vera

Disc Portfolio Provides Strong Foundation for Growth



Other Iron and Heme Disorders

- Beta Thalassemia
- Other Porphyrias
- Myelodysplastic Syndromes
- Sickle Cell Disease
- Hereditary Hemochromatosis
- Anemia of IBD
- Anemia of Cancer

Bitopertin

GlyT1 Inhibitor

Heme Biosynthesis Modulation

Bitopertin: Investigational Oral, Selective GlyT1 Inhibitor

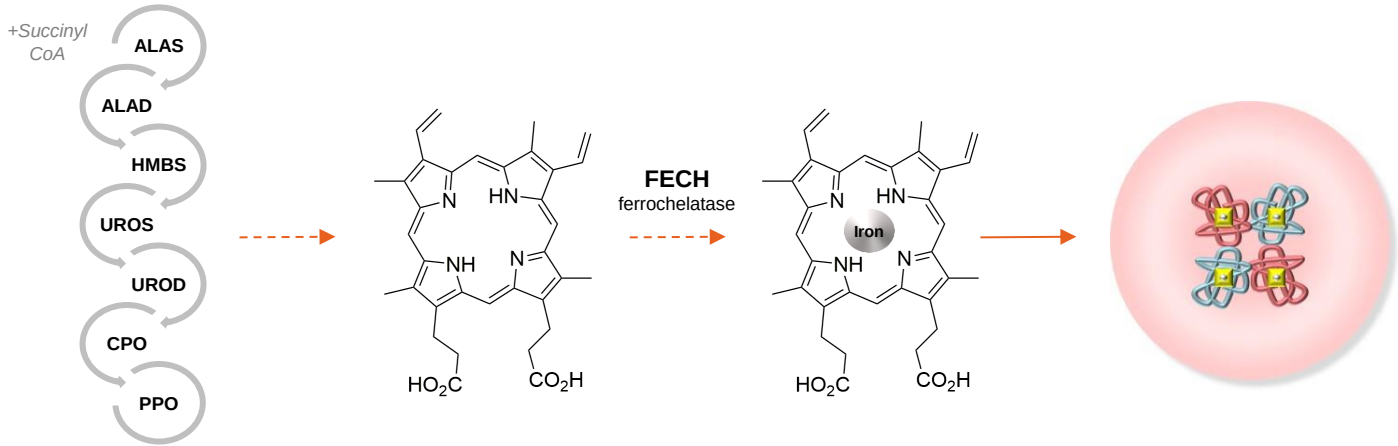
In multiple clinical trials by Roche, bitopertin was observed to modulate heme biosynthesis by blocking uptake of glycine in erythrocytes

Glycine

Critical and initiating precursor for heme biosynthesis and is supplied by **GlyT1 transporter**

Bitopertin

Designed to block glycine uptake in RBCs by inhibiting GlyT1



Heme Biosynthesis Pathway

Erythropoietic Protoporphyria (EPP)

Rare, debilitating and lifelong condition characterized by extreme pain and damage to skin caused by light

Genetic condition caused by defective heme biosynthesis – deficient enzyme ferrochelatase

- Lifelong and presents in early childhood
- Caused by accumulation of toxic metabolite PPIX
- XLP, mechanistically similar disease, also PPIX-related

Debilitating and potentially life-threatening

- Skin: severe, disabling pain attacks (days), edema, burning
- Hepatobiliary disease: gallstones, liver dysfunction or failure
- Psychosocial well-being (fear, anxiety) and development

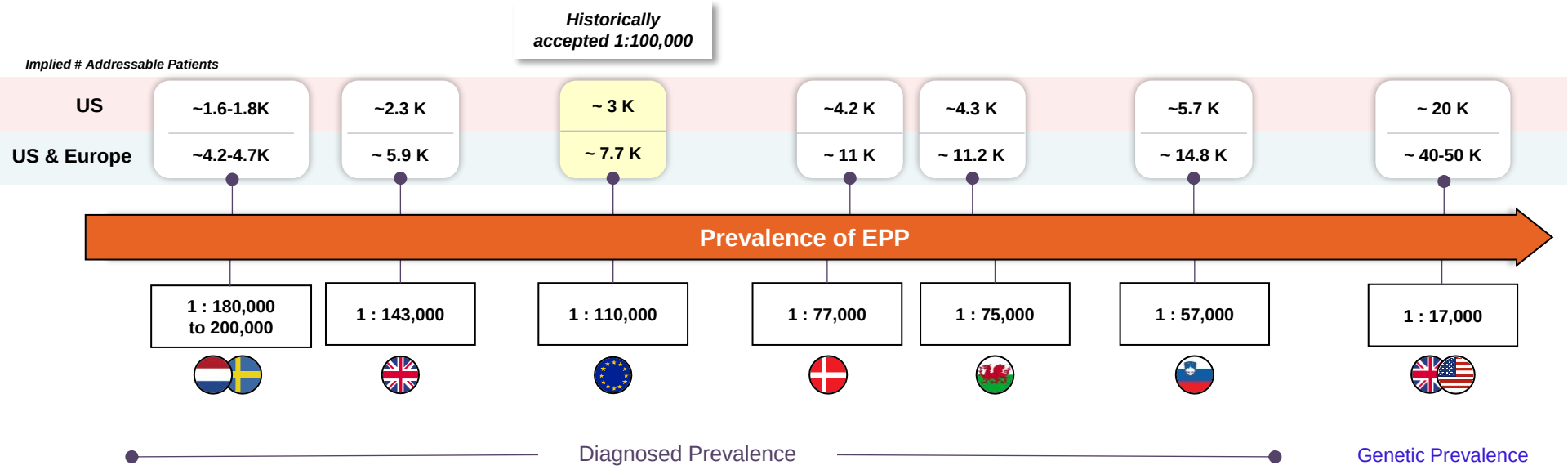
No cure or disease-modifying treatment

- Avoid sun / light, protective clothing, window tinting, Zn/Ti Oxide
- One FDA-approved agent, afamelanotide, a surgically-implanted tanning agent

Image sources: *Daily Mail Australia* (2019); FDA Scientific Workshop on EPP (2016); Buonuomo et al. (2014) *Arch Dis Child*

Historical EPP estimates likely underrepresent prevalence

Based on methodology reported in literature and patient journey

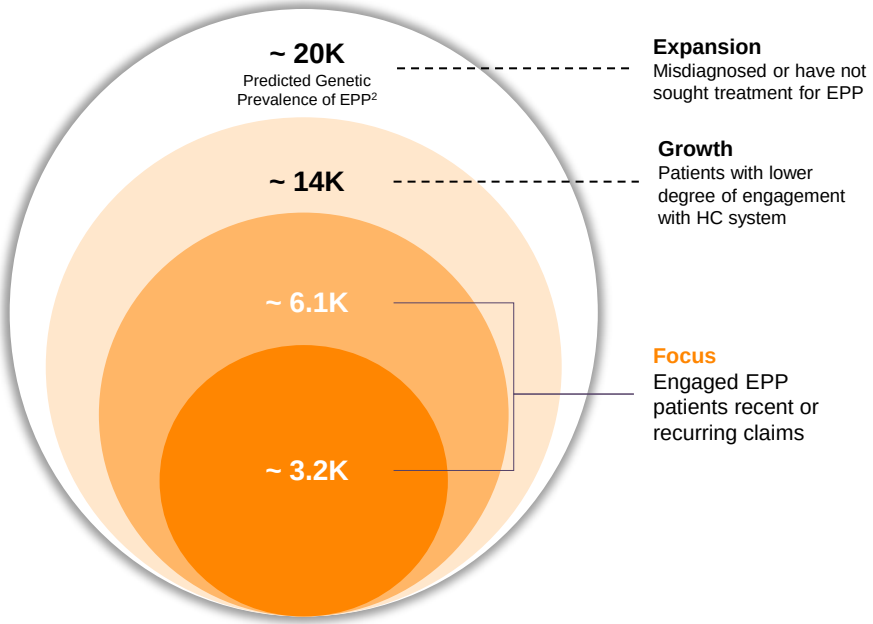


Clearview Healthcare Partners; Went et al (1984); Wahlin et al. (2011); Holme et al. (2006); Elder et al. (2013); Christiansen et al. (2016); Elder et al. (1990); Marko et al. (2007); Dickey et al. (2021)

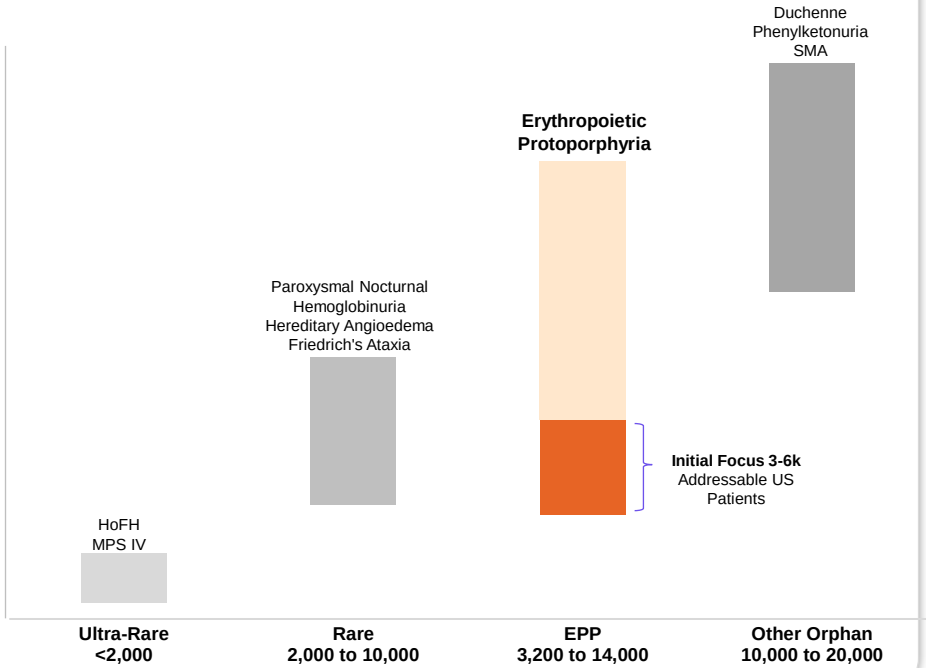
EPP Prevalence: Est. 3-6K addressable patients in the US

Based on analysis of ICD-10 codes in claims data

Prevalence of EPP Patients in the U.S.



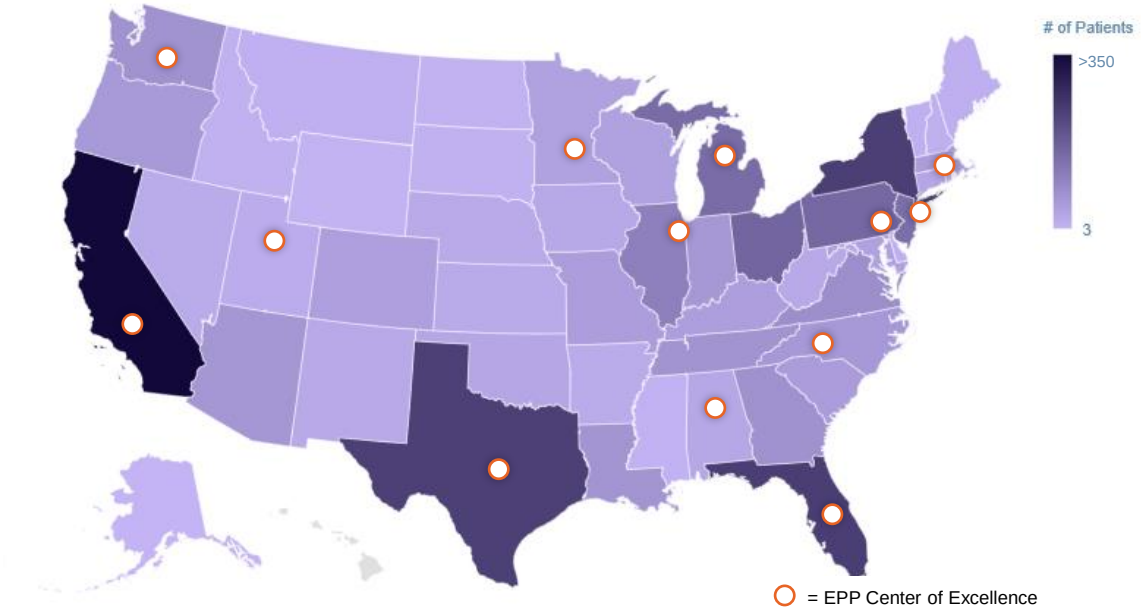
US EPP Prevalence Comparable to Major Rare Diseases



Source: Trinity Life Sciences; Komodo Claims Data (2016-2022); *Dickey et al, Genetic Medicine, 2021; HC = healthcare

EPP patients are identifiable and can be addressed through a highly efficient operating model

Distribution of EPP Patients



Concentration of patients in key accounts enables a targeted and efficient field force

Source: Trinity Life Sciences; Komodo Claims Data (2016-2022); United Porphyria Association; American Porphyria Foundation

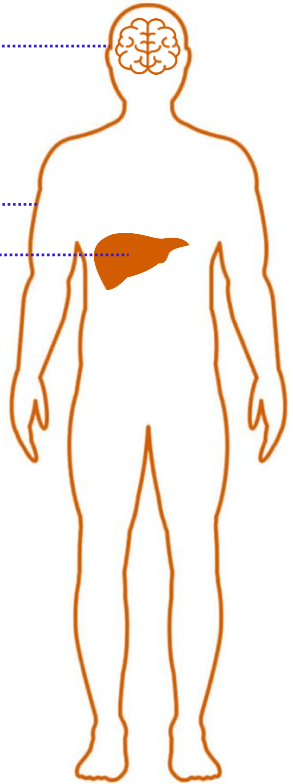
Real world data confirm EPP has a significant impact on patients' lives across multiple domains

56%
have anxiety and/or depression

Psychosocial

<30 min.
sun exposure before symptoms

Skin



34%
have liver and/or biliary issues

Hepatobiliary Complications

Concomitant Medication

57%
receive steroid treatment

30%
treated with narcotics

19%
get prescription Vitamin D

Healthcare Utilization

38%
inpatient stay in past 3 years

32%
visit the ER each year

Source: Trinity Life Sciences; Komodo Claims Data (2016-2022); Balwani et al, JAMA Dermatology, 2017

PPIX is a Driver of Disease in EPP / XLP Patients

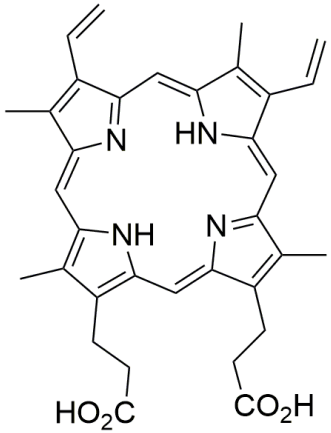
Toxic and photo-active metabolite accumulates in RBCs and is transported to skin and other organs, causing damage

Skin

- Porphyrin ring absorbs light and **emits energy and heat**
- Oxidative damage to endothelial capillaries and surrounding tissue, perivascular edema, complement activation
- Pain, burning sensation, swelling, inflammation, chronic skin lesions

Psychosocial

- Issues with focus and concentration
- Lack of sleep, physical and social isolation
- Significant lifestyle modification, fear and anxiety



Protoporphyrin IX

Hepatobiliary

- PPIX accumulation in bile canaliculae, causing oxidative damage
- Cholelithiasis requiring surgery or impaired liver function (~25%) and end-stage liver disease requiring transplant (2-5%)
- Clinical and biochemical surveillance

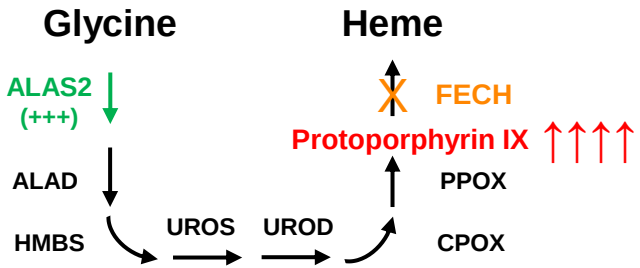
Other Complications

- Nutritional deficiency resulting in osteoporosis and propensity for fractures, chronic alterations to skin (e.g. fragile), mild anemia

Bitopertin: Potential Disease-Modifying Treatment

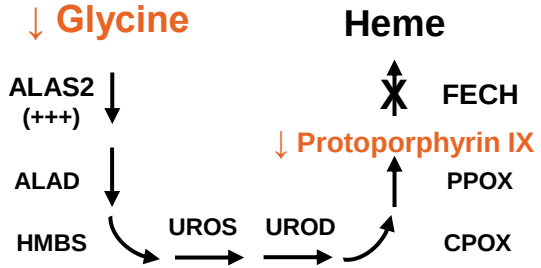
Designed to reduce disease-causing PPIX by limiting uptake of glycine into developing erythrocytes

EPP and XLP Patients
High PPIX Levels



Mutations result in reservoir of pathologically high levels of PPIX

Bitopertin Treatment
Designed to Reduce PPIX Levels



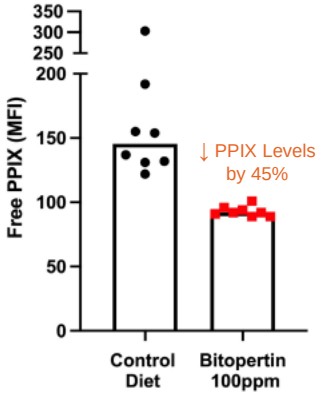
Potential first disease-modifying treatment for EPP and XLP

Figures adapted from Halloy et al. (2021) Cell Chem Biol

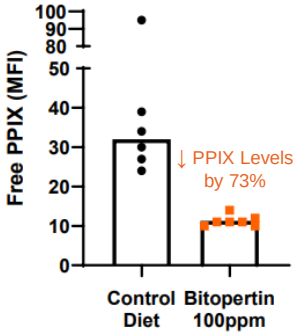
Bitopertin Reduced PPIX in Models of EPP / XLP

Effects on PPIX have the potential to be disease-modifying

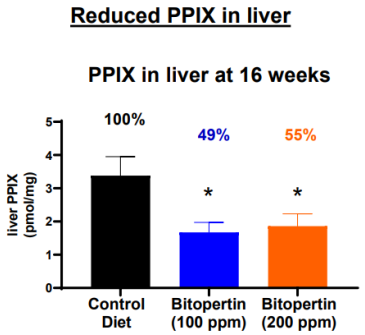
In vivo - EPP Model (Mouse)
FECH^{m1pas} Missense Mutation

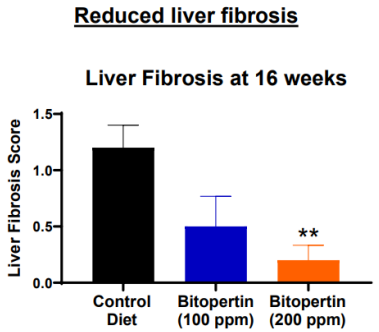


In vivo - XLP Model (Mouse)
ALAS2^{Q548X} Gain-of-Function Mutation



In vivo - EPP Model (Mouse)
FECH$^{m1pas/m1pas}$ Mutation





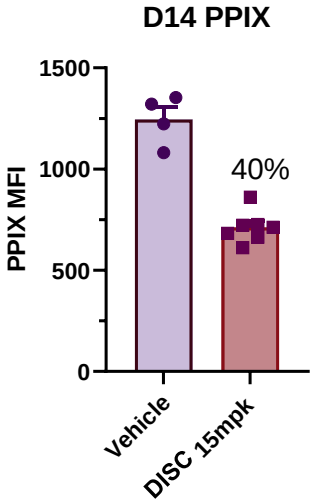
Bitopertin reduced PPIX, the driver of disease pathophysiology, and, based on the data, is expected to be disease-modifying

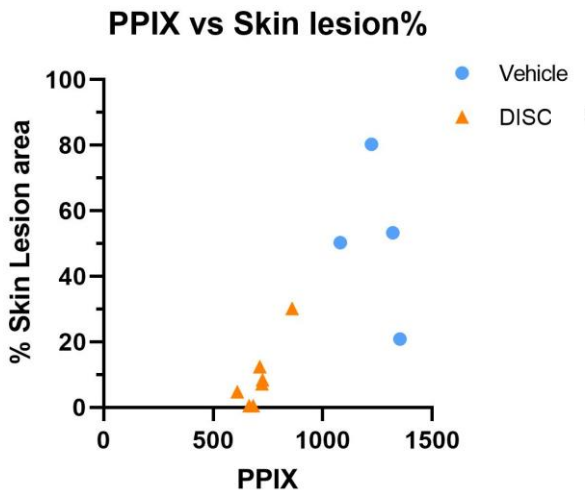
- Reductions in PPIX levels of ≥30% reported in literature to have a major impact on photosensitivity in patients[†];
- Bitopertin has been shown in an animal model of EPP (data presented at ASH 2022) to reduce liver fibrosis

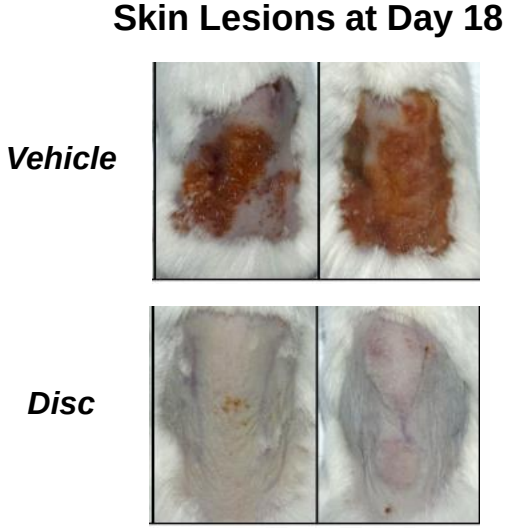
Data presented at the 63rd ASH Annual Meeting (December 2021); Studies performed in collaboration with Boston Children's Hospital (PI: Paul Schmidt, Advisor: Mark Fleming)
Sources: † Heerfordt et al. (2016) Br J. Dermatol.; Wulf et al. (2019) Photodiagn and Photodyn Ther; Poh-Fitzpatrick (1997) J Am Acad Derm

PPIX in EPP: Phototoxicity in Mice

GlyT1 inhibition significantly ameliorated skin lesions after UV exposure and degree of skin lesion correlated with PPIX levels







Note: Initial study data; EPP mice were dosed with either vehicle (N=4) or DISC (N=7) 15 mpk BID for 18 days; mice were exposed to UV light on Day 14; study measured PPIX, skin lesions, and pain sensitivity; DISC is a research-grade GlyT1 inhibitor; This study was performed with approval from an IACUC. Adequate measures were taken to minimize pain and distress for the animals and still accomplish the objectives for the study.

Bitopertin Robust Data Package

Extensive non-clinical, CMC and clinical development has already been completed

Non-Clinical	CMC	Clinical
✓ Genetic toxicity and Safety pharmacology	✓ Commercial-scale production	✓ Healthy volunteer studies
✓ Long-term GLP toxicology	✓ Optimized oral formulation (tablet and capsule)	✓ Drug-drug interaction studies
✓ Juvenile GLP toxicology studies supporting patients ≥2 y/o	✓ Highly stable molecule (at least 5 years)	✓ Hepatic impairment
✓ Carcinogenicity studies		✓ Renal impairment
✓ Full reproductive GLP toxicology		✓ TQT (heart rhythm) study
✓ Metabolites fully qualified		✓ Pharmacokinetics in patients of Asian descent
		✓ 30+ Other clinical trials

Note: Total clinical experience of bitopertin is extensive and includes 700+ HV and 4,000 patients in over 30 clinical trials; all trials referenced conducted by Roche

EPP Phase 2 Development Program

BEACON and AURORA Studies

BEACON

- **EPP and XLP**; N = 26 (22 adults, 4 adolescents)
- **Australia** (study opened July ’22)
- **Open-Label, randomized, 24-week study**

AURORA

- **EPP**; N = 75 (fully enrolled)
- **US** (study opened October ’22)
- **Double-blind, placebo-controlled, 17-week study**

Trial endpoints: Changes in blood PPIX levels, time in daylight without pain, light tolerance, time to prodromal symptom (TTPS), QOL, safety / tolerability

Data availability: Fully enrolled; Updated data presented June 2024; Guidance from end of Phase 2 regulatory interaction to be provided in Q4 2024

AURORA Study: Disposition and Baseline Characteristics

	Placebo (n=24)	Bitopertin 20 mg (n=26)	Bitopertin 60 mg (n=25)
Randomized	24	26	25
Completed Study	24	26	22
Discontinued Prior to Day 121	0	0	3
Characteristic			
Mean Age, years	42.3	45.0	47.8
Female, n (%)	12 (50%)	14 (54%)	12 (48%)
White, n (%)	24 (100%)	24 (92%)	24 (96%)
Baseline PPIX, Mean ± SE (ng/mL)	8,691 ± 903	8,155 ± 1,337	10,597 ± 983
Daily Sunlight Exposure (hr), Mean (range)	1.29 (0.18, 3.31)	1.17 (0.26, 4.03)	1.07 (0.04, 2.78)
Time to Prodrome, n (%)			
< 30 min	9 (38%)	9 (35%)	8 (32%)
≥ 30 min	15 (63%)	17 (65%)	17 (68%)

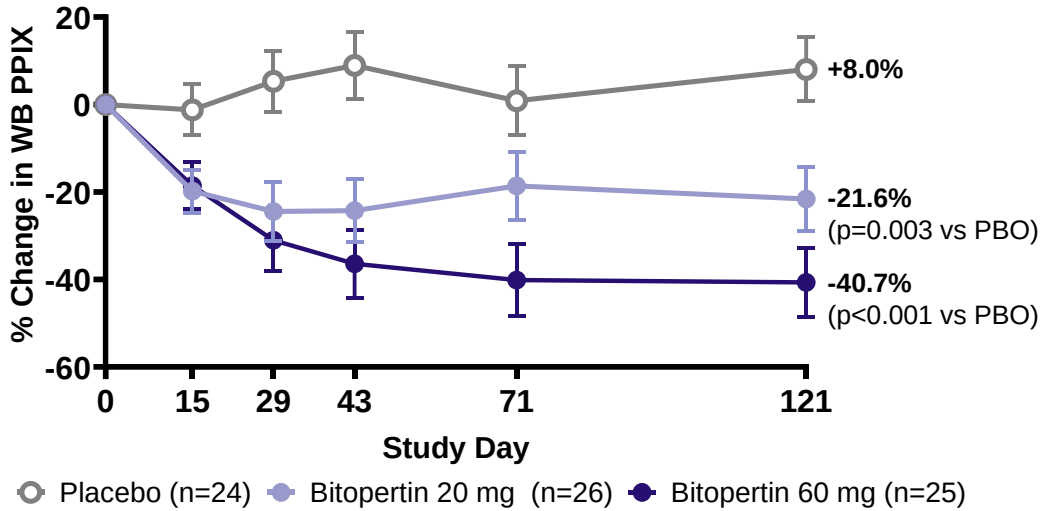
[a] Season that encompasses majority of participant's double-blind treatment period

AURORA Met Primary Endpoint

Statistically significant reductions in whole-blood (WB) metal-free PPIX

- Bitopertin reduced PPIX levels consistent with BEACON, taking ~6-8 weeks to reach max reduction
- Significant reductions observed in both 20 mg and 60 mg doses

PPIX Over Time

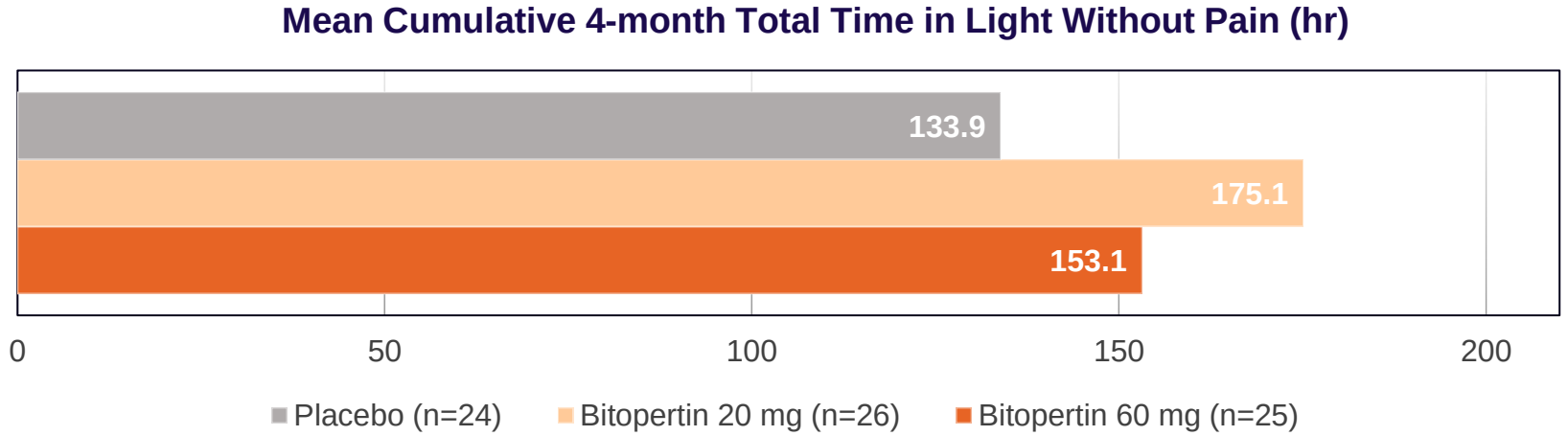


Least-squares means and p-values for percent changes in PPIX analyzed using a mixed model repeated measures (MMRM) to compare 20 mg and 60 mg bitopertin dose groups versus placebo.

Updated AURORA Data: Key Secondary Endpoint

Cumulative time in light without pain

- Bitopertin treatment effect similar to BEACON results
- Did not meet statistical significance due to strong performance of placebo arm

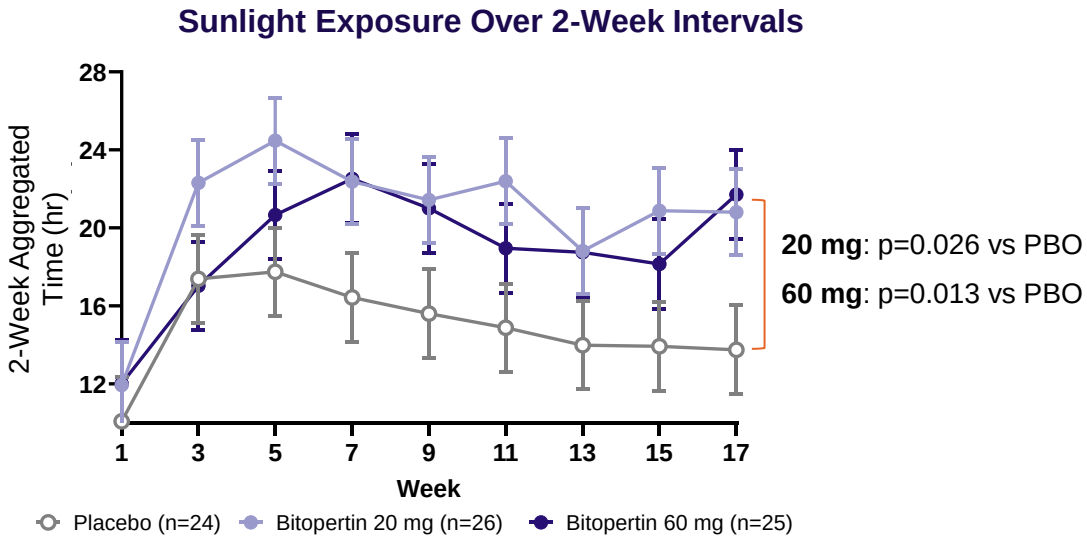


Least-squares means for cumulative time in light measured via daily diary, adding all time in light between the hours of 10:00 am and 6:00 pm on days without any pain, and analyzed using an analysis of variance model to compare 20 mg , 60 mg bitopertin dose groups versus placebo.

Updated AURORA Data: Time in Light Without Pain

Post-hoc longitudinal analysis adjusted for baseline

- Statistically significant improvements in daily time in light compared to placebo
- Meaningful changes in daily time in light relative to baseline



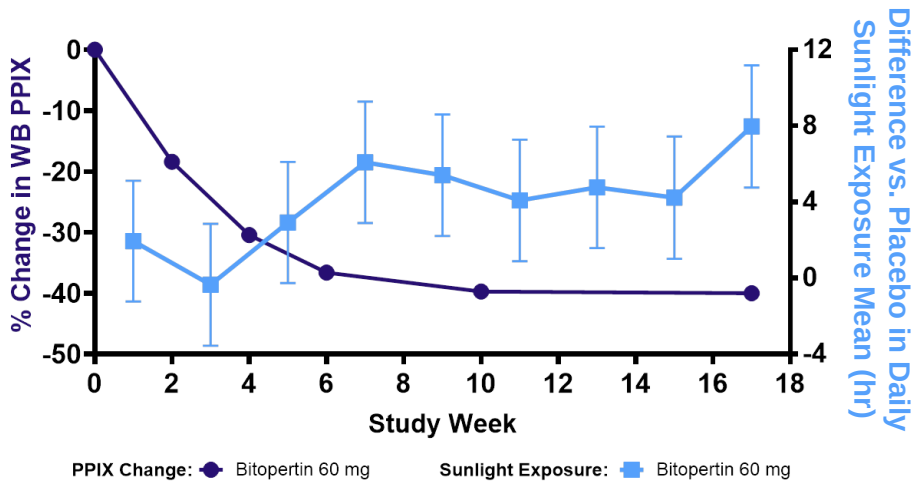
Change from Baseline	
Bitopertin 60 mg (n=25)	2.0x
Bitopertin 20 mg (n=26)	1.9x
Placebo (n=24)	1.1x

Total daily sunlight exposure from 10:00am to 6:00pm on days without pain aggregated over 2-week periods and analyzed using MMRM to compare 20 mg and 60 mg bitopertin dose groups vs placebo. Mean ± SE baseline daily sunlight exposure (hr) during 14-day screening period: 11.1 ± 1.9 for 20 mg, 10.7 ± 1.7 for 60 mg, 12.2 ± 2.1 for placebo.

Updated AURORA Data: Light Tolerance

- Timing of PPIX reduction aligns with the time course of increases in sunlight tolerance

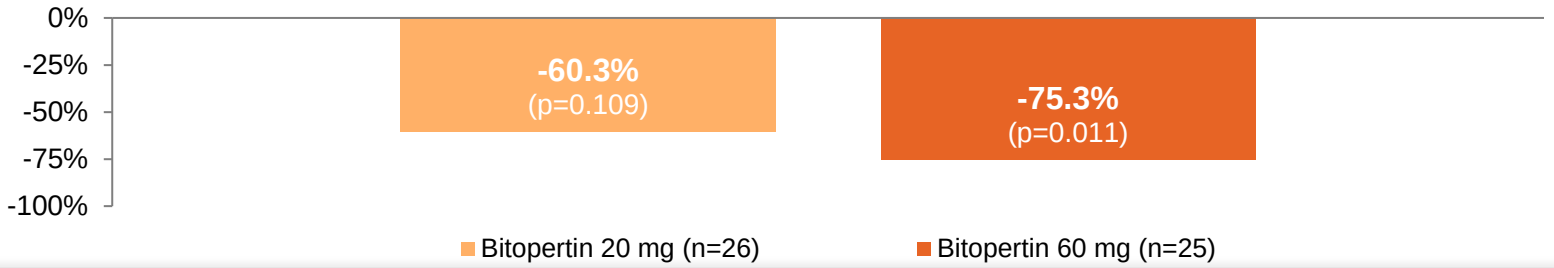
Change in PPIX and Placebo-Corrected Sunlight Exposure for 60 mg Group



Updated AURORA Data: Phototoxic Reactions with Pain

- Dose-dependent reduction in rate of phototoxic reactions with pain, reaching statistical significance in the 60 mg dose group
- Max pain score reduced with bitopertin

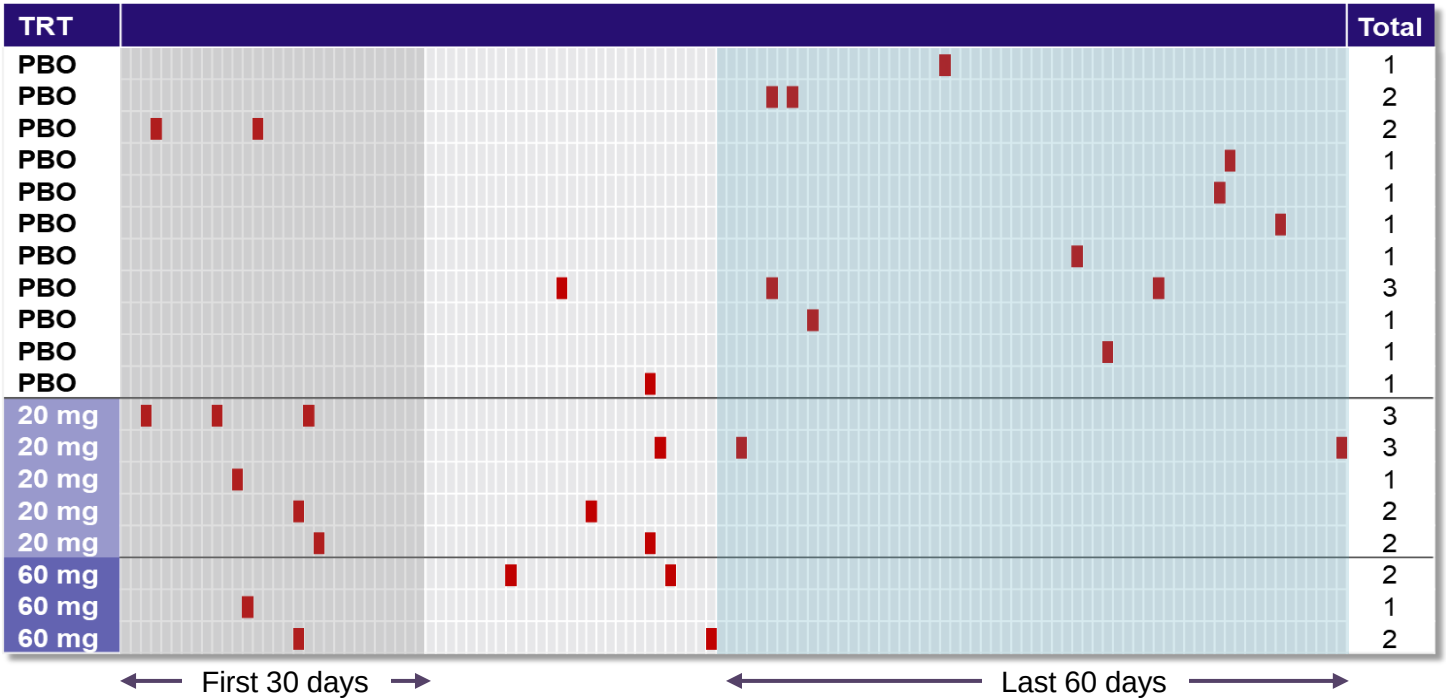
Incidence Rate Ratio of New Phototoxic Reactions with Pain vs. Placebo



	Screening (2-4 weeks)		Double-Blind Period (17 weeks)		
	# of New Reactions	# of Participants	# of New Reactions	# of Participants	Median Max Pain Score
Placebo (n=24)	4	2 (8%)	15	11 (46%)	5.0
Bitopertin 20 mg (n=26)	11	8 (31%)	11	5 (19%)	4.0
Bitopertin 60 mg (n=25)	8	6 (24%)	5	3 (12%)	3.5

Updated AURORA Data: Phototoxic Reactions with Pain

- Consistent with profile for PPIX reductions reaching a nadir, time course of phototoxic reactions showed greater bitopertin treatment effect during the last 60 days of study



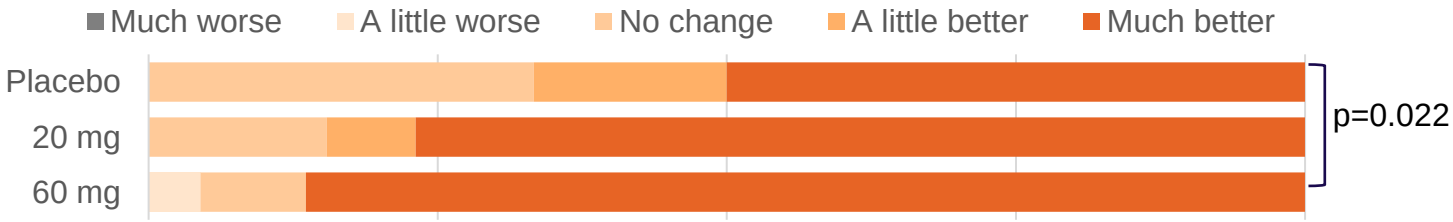
Participants reporting new phototoxic reactions during double-blind period and corresponding study day for new reaction (shown in red)

= New Phototoxic Reaction

Updated AURORA Data: Patient-Reported Outcomes

- Dose-dependent improvements in Patient Global Impression of Change (PGIC), reaching statistical significance in the 60 mg dose group at end of study
- Improved PGIC responses are associated with greater reductions in PPIX

PGIC: *"Since the start of the study, how would you rate the change in your EPP?"*



% PPIX Change	PGIC Response				
	Much worse	A little worse	No change	A little better	Much better
N	0	1	14	6	48
Mean (SD)	-	43.8	6.7 (64.9)	-0.4 (15.2)	-25.9 (31.7)

Percentages calculated using data for PGIC from participants who completed the study (D121): n=26 for 20 mg bitopertin, n=22 for 60 mg bitopertin; n=24 for placebo

Updated AURORA Data: PPIX Change and Light Tolerance

- Greater PPIX reductions in bitopertin participants reporting no phototoxic reactions (-36.5%) vs phototoxic reactions (-4.0%)
- PPIX reductions associated with improvements in multiple measures of light tolerance

Tertiles of PPIX Change

PPIX Decreased ← → PPIX Increased

Light Tolerance Measure (Mean ± SD)	Tertile 1 (-88% to -38%)	Tertile 2 (-38% to -7%)	Tertile 3 (-7% to 190%)
Cumulative total time in sunlight without pain (hr)	161.1 ± 142.6	124.5 ± 68.3	117.5 ± 83.2
Average time in sunlight without pain (hr)	1.61 ± 1.32	1.20 ± 0.72	1.16 ± 0.83
Change from baseline in time to prodrome (min)	117.4 ± 148.6	109.4 ± 121.1	64.1 ± 123.8

Tertile 1 = 6 participants at 20 mg, 16 at 60 mg, and 2 on placebo; Tertile 2 = 13 participants at 20 mg, 6 at 60 mg, and 5 on placebo; Tertile 3 = 7 participants at 20 mg, 1 at 60 mg, and 17 at placebo

Safety and Tolerability

- No serious adverse events reported with bitopertin
- Stable hemoglobin levels
- Favorable safety profile consistent with prior studies enrolling >4000 participants

	Placebo (n=24)	Bitopertin 20 mg (n=26)	Bitopertin 60 mg (n=25)
Participants with any TEAE, n (%)	18 (75%)	20 (77%)	22 (88%)
TEAEs leading to discontinuation, n (%)	0	0	2 (8%)
SAEs, n (%)	1 (4%)	0	0
Common TEAEs			
Dizziness, n (%)	4 (17%)	4 (15%)	11 (44%)
Median Duration (days)	2.0	4.5	5.0
Nausea, n (%)	2 (8%)	1 (4%)	4 (16%)
Alanine aminotransferase increased, n (%)	3 (13%)	1 (4%)	2 (8%)

TEAEs leading to discontinuation: rash and dizziness; common TEAEs include those reported in >5 participants during study regardless of treatment assignment.
SAE = serious adverse event; TEAE = treatment-emergent adverse event

Summary of EPP Bitopertin Data

BEACON and AURORA Studies

AURORA

- Significant reductions in PPIX 40% vs placebo
- Time-dependent, 2x improvements in pain-free time in sunlight
- Significant 75% reduction in rate of phototoxic reactions vs placebo
- Significant improvement in PGIC vs placebo

Targets underlying pathophysiology of EPP

Significant improvement in sunlight tolerance

Functional benefit by reducing debilitating phototoxic reactions

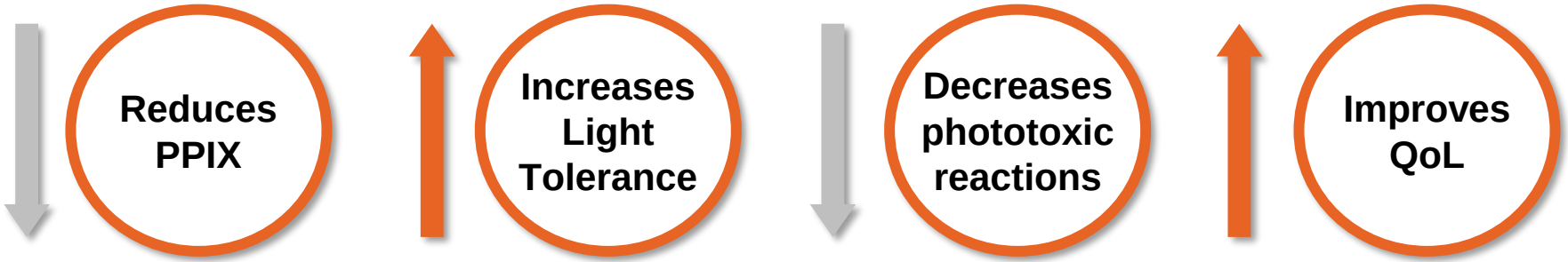
Significantly improved how patients feel

BEACON

- Significant reductions in PPIX >40% vs baseline
- Significant 3x increase in sunlight tolerance (time to prodrome)
- 92% reduction in number of phototoxic reactions vs baseline
- Nearly all (95%) participants reported improvements in PGIC

Summary of Updated Bitopertin Data

Bitopertin demonstrated meaningful impact on key aspects of EPP



➢ **Next Steps**

- Guidance from end of Phase 2 meeting expected to be provided in Q4 2024; initiation of a pivotal study in 1H 2025
- Range of available endpoints to bring to regulators that address the placebo effect
  - *Options include*: longitudinal analysis of time in sunlight, phototoxic pain reactions, PPIX, composites of multiple endpoints, and others

Diamond Blackfan Anemia

Genetic condition caused by defective erythropoiesis

- Mutations in ribosomal protein genes (classically RPS19)
- Heme/globin imbalance: excess heme accumulation leading to toxicity as globin synthesis is delayed

Characterized by severe anemia that presents in infancy

- Anemia, fatigue, delayed growth, cardiac or renal defects, risk of malignancy
- Patients may also have distinct physical features / congenital abnormalities (i.e., cleft palate, thumb and upper limb abnormalities, short stature, microcephaly)

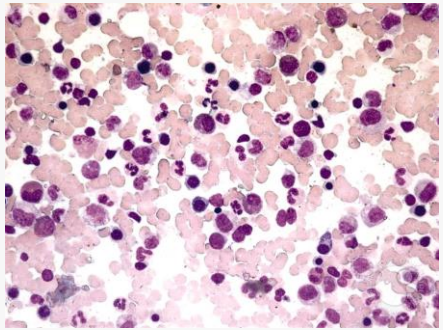
No approved treatments for DBA

- Patients receive steroids and blood transfusions to manage their condition
- Median life expectancy is 38 years, with 25% mortality by age 50

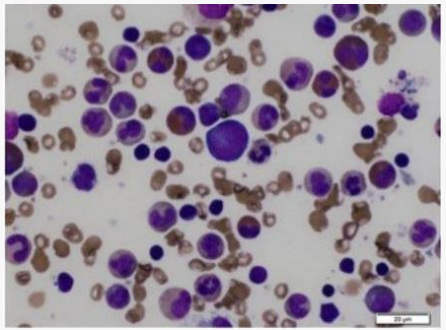
Rare disease with an incidence rate of 5-7 per 1 million live births

- Estimated worldwide prevalence of 5,000

Normal



DBA

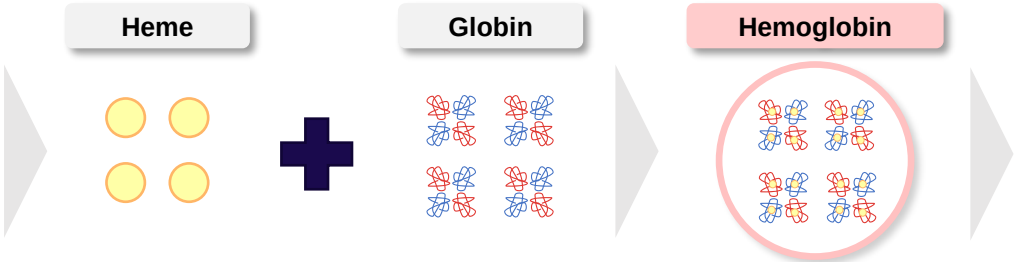


St. Jude's Children Hospital; Fatima et al, Pediatric Hematology Oncology Journal, March 2021

Diamond Blackfan Anemia: Heme Toxicity

Normal Erythropoiesis

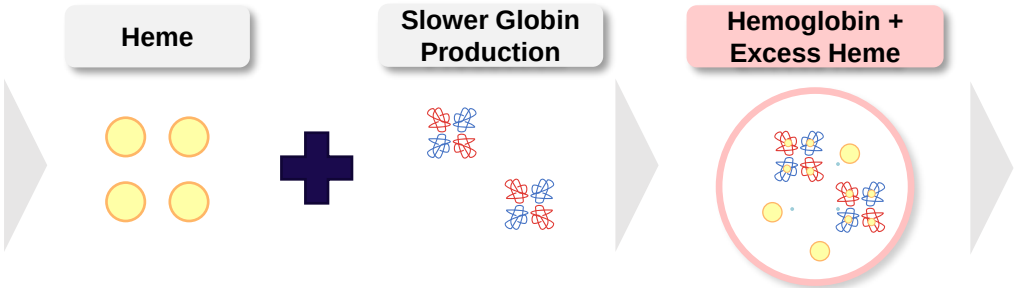
Glycine
Critical and initiating precursor for heme biosynthesis and is supplied by **GlyT1 transporter**



Complete Maturation

DBA Erythropoiesis

Glycine
Critical and initiating precursor for heme biosynthesis and is supplied by **GlyT1 transporter**



Heme toxicity and cell death

Yang et al, 2016 (Jan Abkowitz lab), Sci Transl Med. 8:338ra67, 2016

Bitopertin in Diamond Blackfan Anemia

By slowing the influx of glycine, bitopertin lowers heme production, reducing the amount of excess heme and preventing cell death

Glycine

Critical and initiating precursor for heme biosynthesis and is supplied by **GlyT1 transporter**

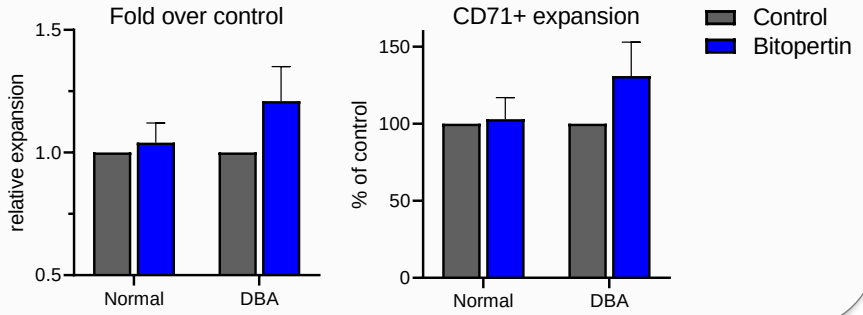
Less Heme

Slower Globin Production

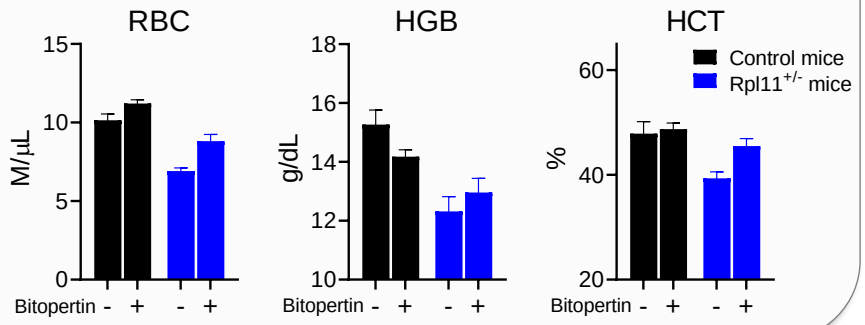
Hemoglobin

Complete Maturation

Primary human marrow in erythroid differentiation cultures treated with 10 ng/ml bitopertin for 7 days



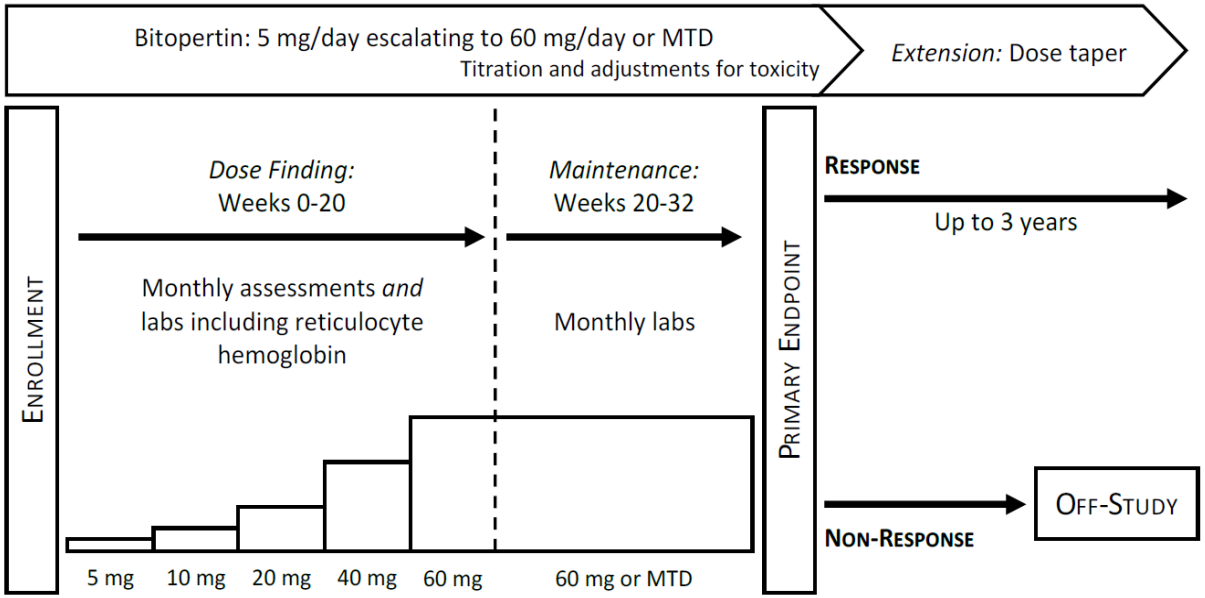
Rpl11 haploinsufficient mice were treated with 100 ppm bitopertin in chow (20 mg/kg/d) for 8 weeks



Diamond Blackfan Anemia Phase 2 Clinical Trial

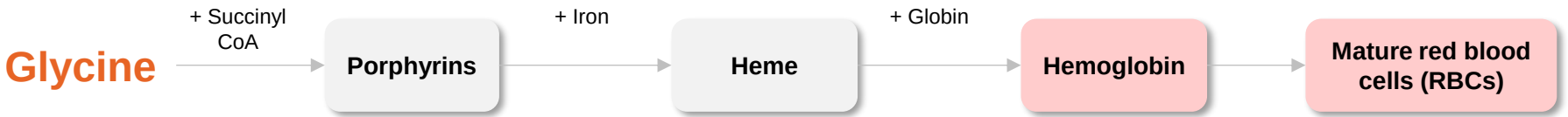
IIT conducted by the NIH under CRADA with Disc

- Single-arm, dose-escalation study with extension
- N=15-25 patients with steroid-refractory and/or relapsed disease, or steroid intolerant
- Response defined as >50% reduction in RBC transfusions over 8-week period or an increase in pre-transfusion hemoglobin of >1.5 g/dL



Multiple Additional Potential Applications of Bitopertin

Inhibiting heme synthesis with bitopertin has potential to address a wide range of hematologic diseases



Porphyrin Toxicity

Erythropoietic Protoporphyria
X-Linked Protoporphyria
Congenital Erythropoietic Porphyria
Hepatic Porphyrias

Heme Toxicity

Diamond-Blackfan Anemia
Myelodysplastic Syndromes

Globin Toxicity

Beta-Thalassemia
Sickle Cell Disease

Excess RBCs

Polycythemia Vera

bold (trial ongoing)

Hepcidin Modulation

Iron Homeostasis

Iron is Fundamental to RBC Biology

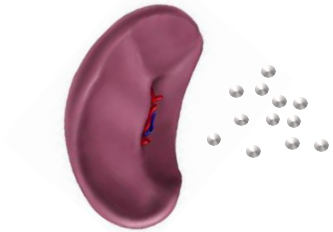
Hepcidin is a regulatory hormone that plays a central role in iron metabolism and homeostasis

Induced by Inflammation

Hepcidin

Gatekeeper Function: Blocks iron absorption and recycling

GI Tract
Iron Intake



Spleen
Iron Storage

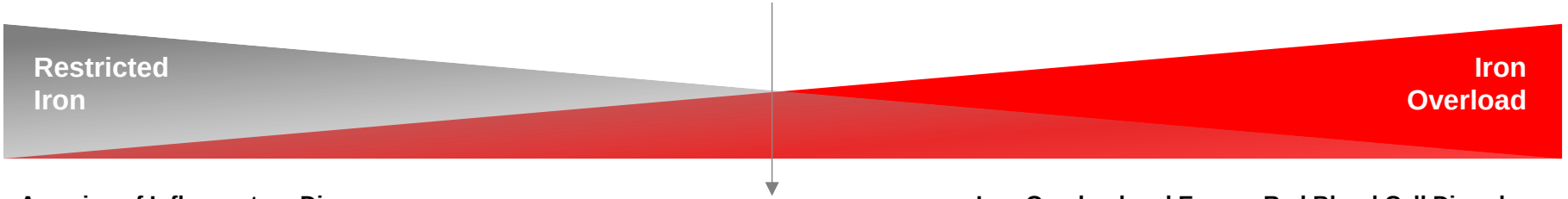
RBC Production in Bone Marrow

Hepcidin is a Therapeutic Target for Diseases

Dysregulated hepcidin drives a wide range of hematologic diseases

High Hepcidin ← **Normal Hepcidin** → **Low Hepcidin**

Regulated erythropoiesis



Regulated iron

Anemias of Inflammatory Disease

- Myelofibrosis
- Chronic Kidney Disease
- Autoimmune / Inflammatory Disorders

DISC-0974 (anti-HJV mAb)
Reduce Hepcidin / Increase Iron

Iron Overload and Excess Red Blood Cell Disorders

- Polycythemia Vera
- Hereditary Hemochromatosis
- Myelodysplastic Syndromes

DISC-3405 (anti-TMPRSS6 mAb)
Induce Hepcidin / Restrict Iron

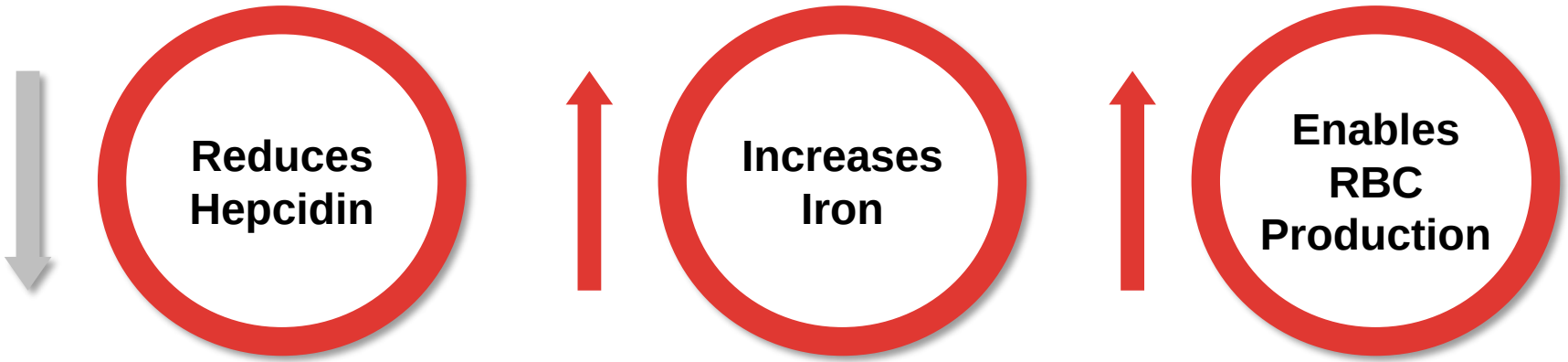
DISC-0974

Anti-HJV mAb

Hepcidin Suppression

DISC-0974: Novel Anti-HJV mAb to Suppress Hepcidin

Designed to enhance iron availability to address a wide range of hematologic disorders



Inhibits endogenous production of hepcidin

Releases iron stores
Enables GI absorption

Potential to treat wide range of anemias

Significant Opportunity in Anemia of Inflammation

Numerous chronic diseases associated with anemia from high hepcidin

Anemia Types	US Prev.	Est. % Anemic
Myelofibrosis (MF)	16-18.5K	87%
Chronic Kidney Disease (CKD)	37 MM	17-50%
Inflammatory Bowel Disease	1.6 MM	25-35%
Anemia of Cancer	17 MM	35-80%
Systemic Lupus Erythematosus	210K	50%

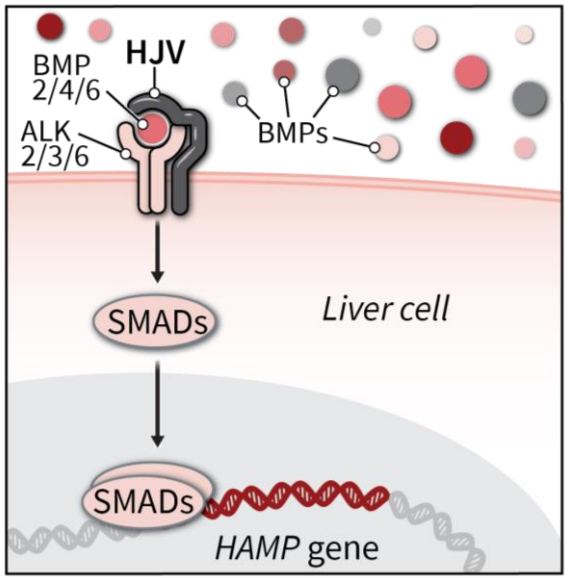
- **Anemia of inflammation** is the 2nd most common form of anemia
- **Estimated 40% of all anemias** are driven by or have an inflammatory component
- **Hepcidin is up-regulated** and correlates with anemia, driven by inflammation

Bold = ongoing Disc trial

Sources: Weiss (2019); Maccio (2014); Tefferi (2012); Lupus Foundation; Stauffer (2014); Filmann (2014); Koutroubakis (2015); Crohn's and Colitis Foundation

Targeting Hemojuvelin (HJV) to Suppress Hepcidin

Critical and specific target for hepcidin expression



Inhibiting Hemojuvelin (HJV) Prevents Hepcidin Expression and Increases Iron

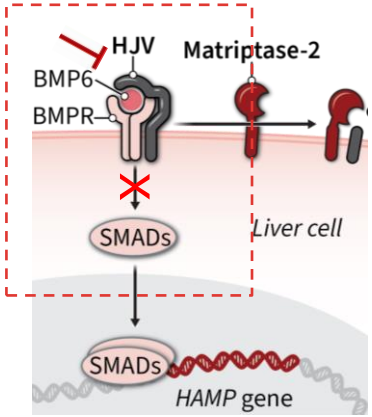
- **Genetic validation** in patients with Juvenile Hemochromatosis (lower hepcidin and elevated iron levels)
  - Loss-of-function mutations in HJV are phenotypically indistinguishable from mutations in *HAMP* (hepcidin) gene
- **Functionally specific** to hepcidin / iron
- **Tissue specific** expression primarily in the liver

Sources: Finberg et al, (2010) Blood; Zhang et al, (2010) J Biol Chem

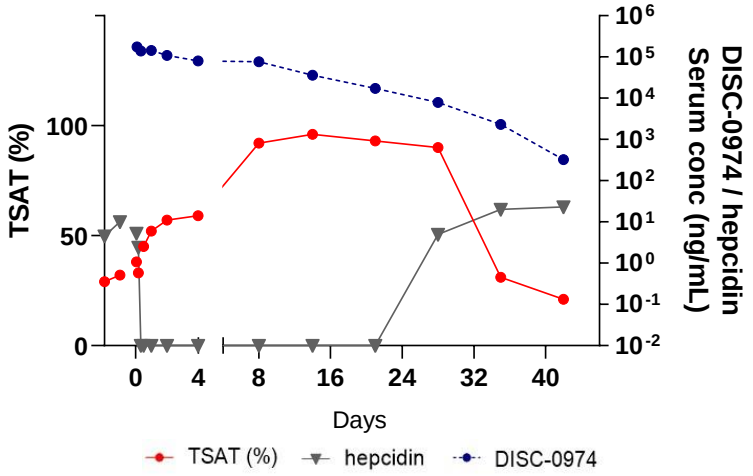
DISC-0974 Mechanism of Action

Designed to reduce hepcidin and increase serum iron levels

DISC-0974 mAb binds to and prevents signaling through hemojuvelin (HJV) co-receptor



Potent and rapid effects on hepcidin and iron with single 5 mg / kg dose (NHP)

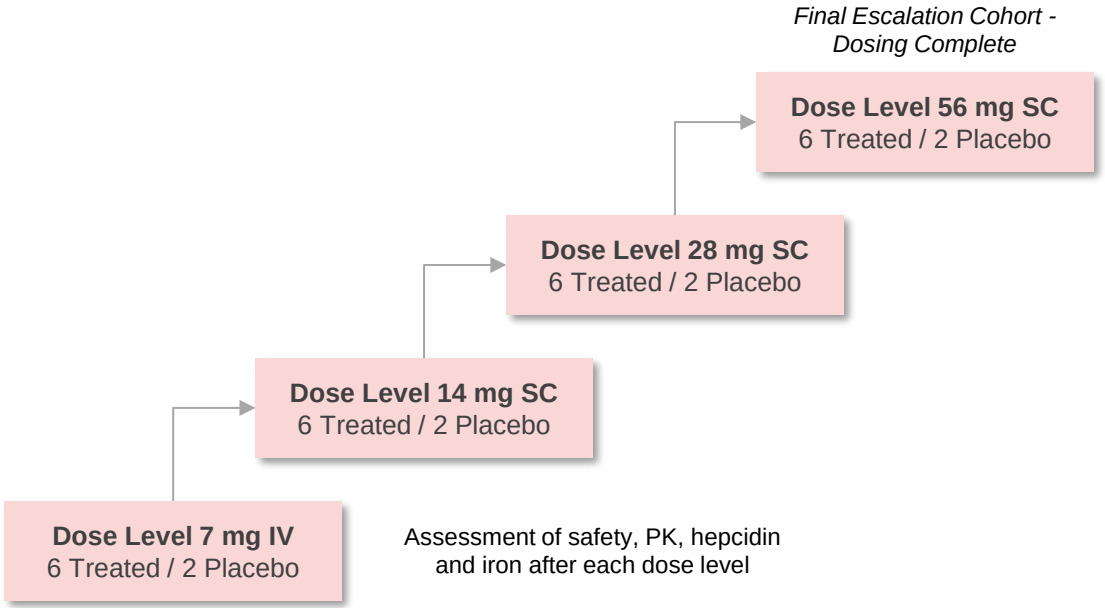


Phase 1 SAD Trial in Healthy Volunteers

Established proof-of-mechanism based on hepcidin and iron parameters

Trial Design

- Single-ascending dose in ≥32 healthy volunteers
- Key outcome measures:
  - Safety and PK
  - Hepcidin level, serum iron level, % TSAT
- Dose escalation until TSAT > 40% for at least 2 weeks
- Dose levels: 7 mg dose (IV); 14, 28 and 56 mg doses (SC)

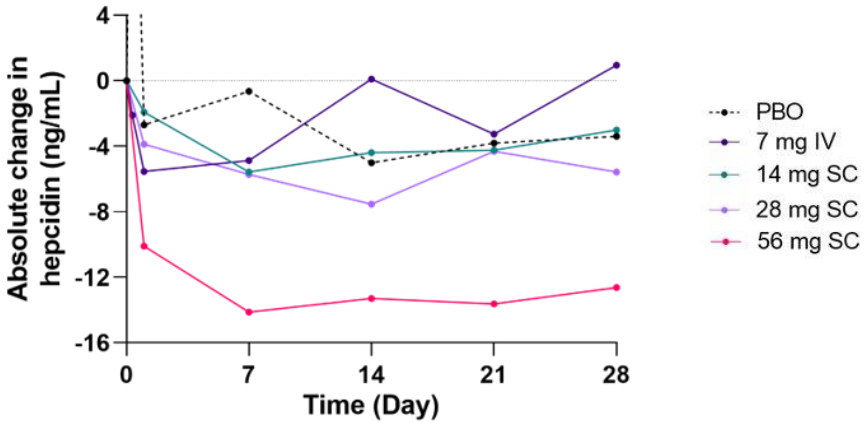


TSAT = transferrin saturation; IV = intravenous; SC = subcutaneous

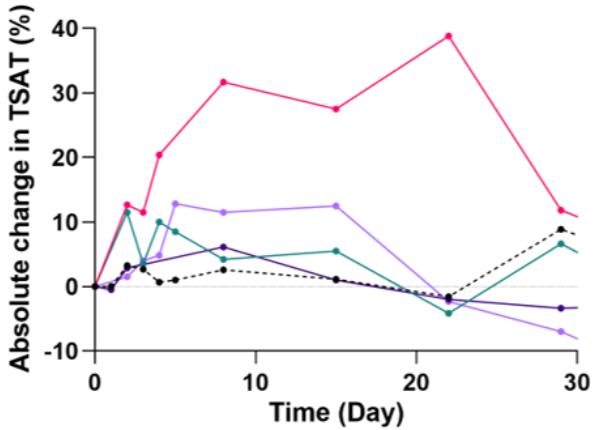
DISC-0974 Phase 1 SAD Data

Dosing of DISC-0974 demonstrated a reduction of hepcidin and iron mobilization

DISC-0974 Reduced Hepcidin Production



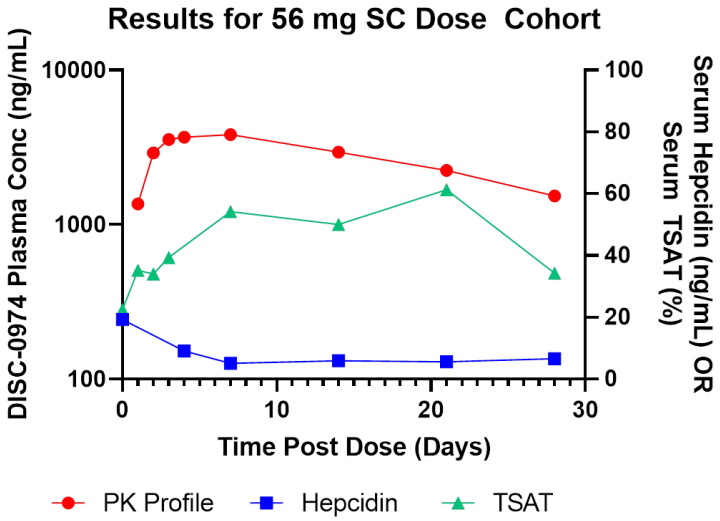
DISC-0974 Increased TSAT

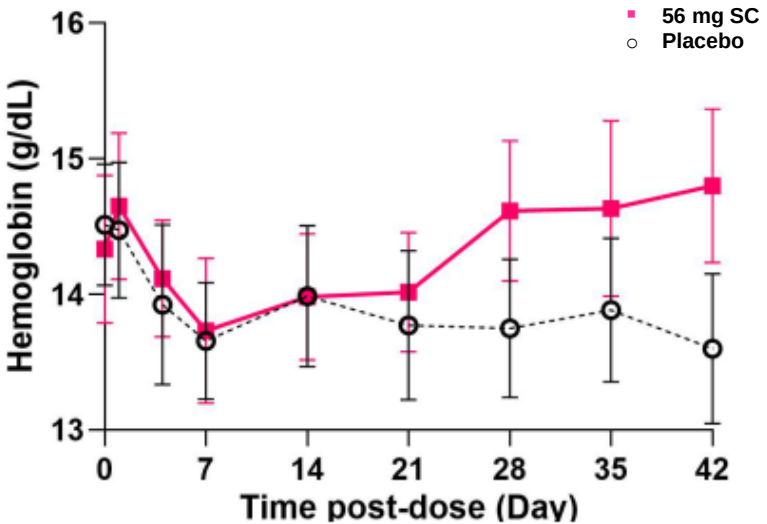


TSAT = transferrin saturation; SAD = single ascending dose

DISC-0974 Phase 1 SAD Data (cont.)

Top dose (56 mg) pharmacodynamic activity improved key clinical parameters (> 1g/dL Hgb)





Hgb = hemoglobin; SAD = single ascending dose

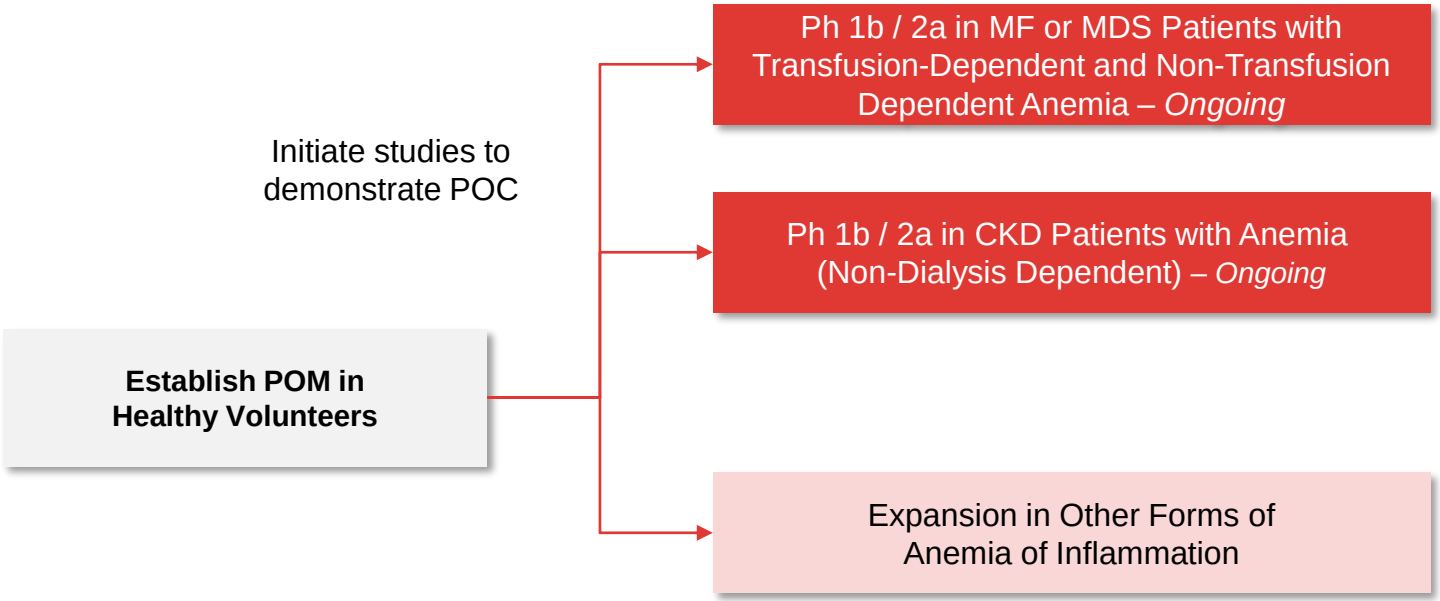
DISC-0974 Phase 1 SAD Safety

Safety profile was consistent with selective target biology and preclinical studies; no serious or AEs > Grade 1

	Total n=42	Pooled Placebo n=10	7 mg IV n=8	14 mg SC n=6	28 mg SC n=6	28 mg IV n=6	56 mg SC n=6
Diarrhea	1 (2.4)	1 (10.0)	0	0	0	0	0
Dizziness	2 (4.8)	0	0	0	0	1 (16.7)	1 (16.7)
Dyspepsia	1 (2.4)	0	0	0	0	0	1 (16.7)
Eye pruritis	1 (2.4)	0	0	0	1 (16.7)	0	0
Peripheral swelling	1 (2.4)	0	0	0	0	1 (16.7)	0
Headache	1 (2.4)	0	0	0	1 (16.7)	0	0
Myalgia	1 (2.4)	0	0	0	0	0	1 (16.7)
Nasal congestion	1 (2.4)	0	0	0	0	0	1 (16.7)
Pain in extremity	1 (2.4)	1 (10.0)	0	0	0	0	0
Seasonal allergy	1 (2.4)	0	0	0	1 (16.7)	0	0
Vessel puncture site bruise	1 (2.4)	1 (10.0)	0	0	0	0	0
Vomiting	1 (2.4)	1 (10.0)	0	0	0	0	0

DISC-0974 Development Strategy

Aim to demonstrate POC in anemia of MF and CKD

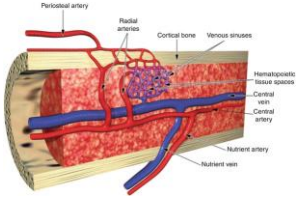


Plan to assess safety, PK, hepcidin, iron, hemoglobin and transfusion burden (MF and MDS) and others

Hepcidin is a Key Driver of Myelofibrosis (MF) Anemia

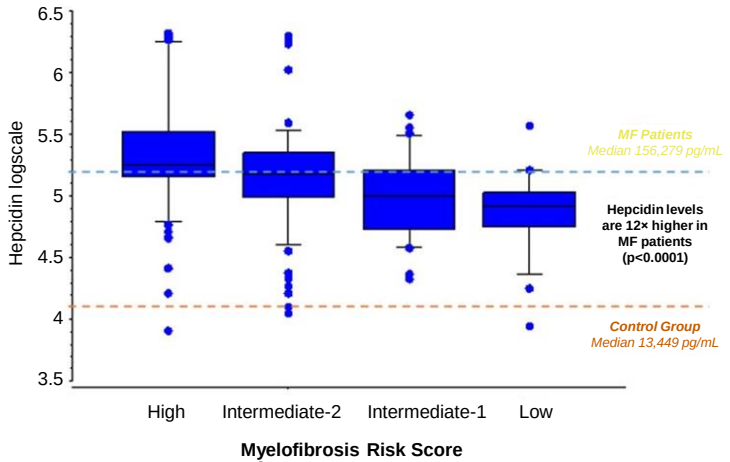
Anemia is severe and prevalent in MF and can limit treatment

Anemia of MF



- **Est. # Patients**
  - 25,000 patients (US)
  - ~87% are anemic; severe and require transfusion
- **Etiology of Anemia**
  - High hepcidin from inflammation
  - JAK inhibitors worsen anemia; loss of marrow function
- **Unmet Medical Needs**
  - Severe and difficult to treat; high transfusion burden
  - No approved or effective anemia therapy
  - Anemia limits optimal JAK inhibitor treatment

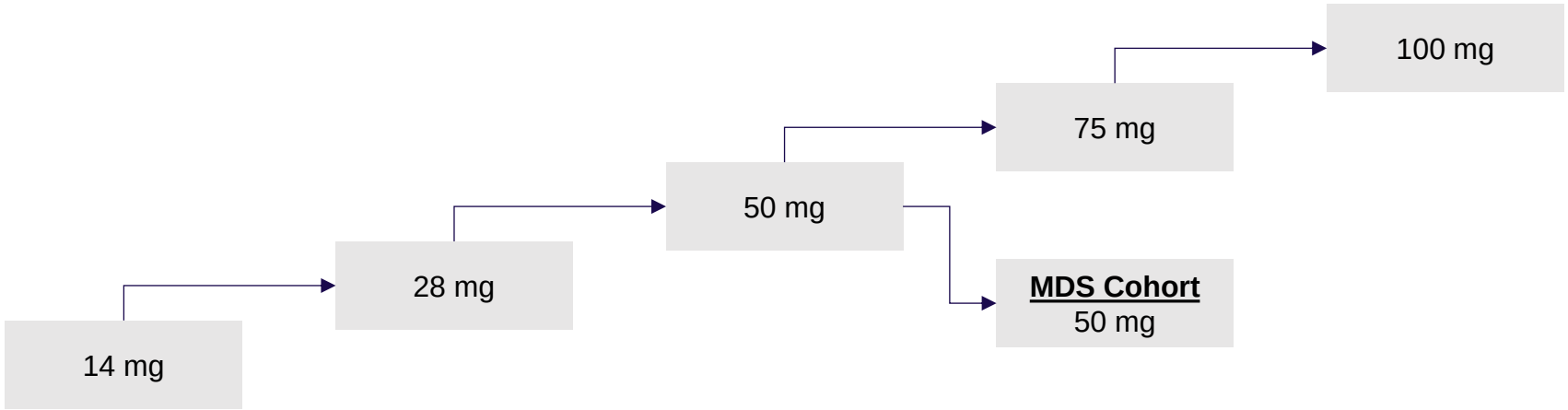
Hepcidin Levels are Elevated in MF
~ 12× higher than control and associated with severity of anemia and transfusion burden



Source: Pardanani et al (2013) Am. J. Hematol

DISC-0974 Phase 1b Anemia of MF Study Overview

Subcutaneous dosing once monthly for 6 months



Key Endpoints/Measures: Iron, hepcidin, and other hematologic parameters, safety/tolerability

Data Availability: Data presented in June 2024; initiation of Phase 2 study expected by the end of 2024

Updated DISC-0974 MF Data: Baseline and Demographics

Data as of April 29, 2024

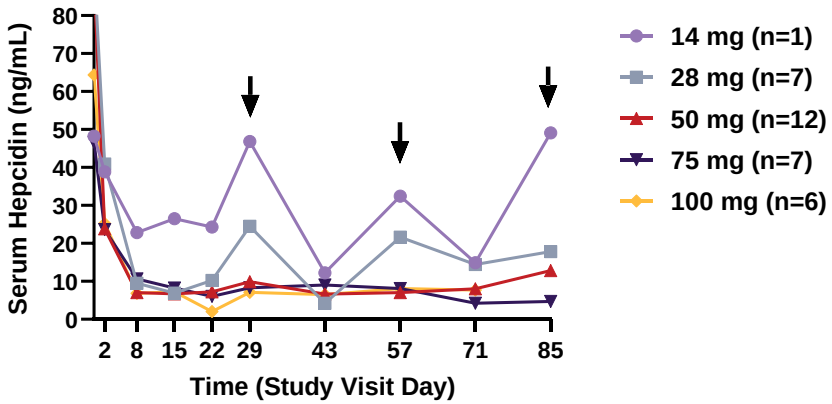
	DISC-0974 14 mg (N=1)	DISC-0974 28 mg (N=7)	DISC-0974 50 mg (N=12)	DISC-0974 75 mg (N=8)	DISC-0974 100 mg (N=6)
Age, median (range), years	70	71 (57, 89)	70.5 (31, 83)	74 (58, 84)	67.5 (53, 79)
Time since MF diagnosis, median (range), years	1	6 (0,18)	2.5 (0,14)	4 (0, 12)	1 (0,2)
Concomitant medication, n (%)					
JAK inhibitor	0	4 (57.1)	5 (41.7)	1 (12.5)	0
Hydroxyurea	1 (100)	0	0	1 (12.5)	0
Transfusion dependent, n (%)*	0	2 (28.6)	1 (8.3)	0	1 (16.7)
Baseline hepcidin, median (range), ng/mL	48.2	93.3 (21.4, 171.1)	90.2 (8.7, 155.7)	46.6 (23.7, 188.2)	64.4 (11.5, 374.7)
Baseline hemoglobin, median (range), g/dL	8.2	8.4 (6.8, 9.3)	8.6 (6.1, 10.3)	8.9 (6.7, 9.9)	8.2 (5.5, 9.4)

Defined as an RBC transfusion frequency of ≥6 units PRBC over the 84 days immediately prior to Screening. There must not be any consecutive 42-day period without an RBC transfusion in the 84-day period, and the last transfusion must be within 28 days prior to Screening. One participant treated with 28 mg discontinued DISC-0974 early due to physician decision. JAK = Janus kinase. Baseline is defined as: (1) Participants transfused within 84 days of screening; (1.a) transfusion dependent then use lowest hemoglobin level recorded in the 84 days before screening initiation (one reading). (1.b) Non-transfusion dependent then {1.b.i} participants transfused within 30 days before screening use the lowest pre-transfusion hemoglobin level (one reading). {1.b.ii} participants transfused within 84 days but not within the month before screening use average of the pre-transfusion hemoglobin level, screening hemoglobin level, and Day -1 level (3 readings); (2) Participants not transfused within 84 days of Screening use Screening and Day -1 average

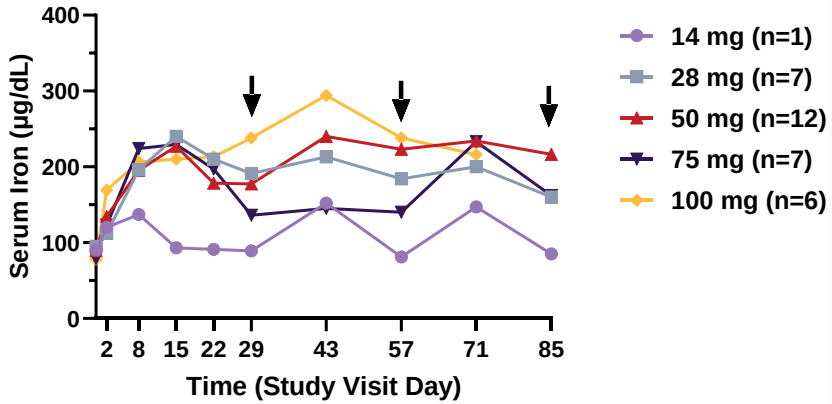
Updated DISC-0974 Anemia of MF Data: Hepcidin and Iron

- DISC-0974 demonstrated decreases in hepcidin and increases in serum iron
- Impact was consistent across all treated participants

Median Serum Hepcidin



Median Serum Iron

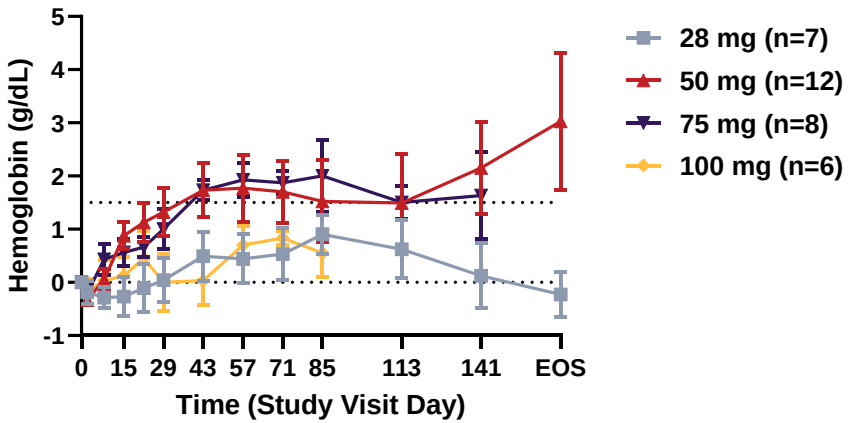


Arrows represent dosing days

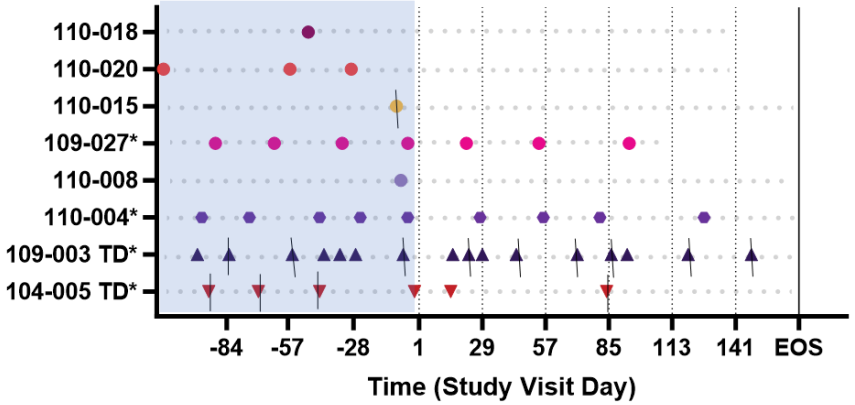
Updated DISC-0974 Anemia of MF Data: Hematologic Response

- DISC-0974 demonstrated sustained increases in hemoglobin across dose groups
- All evaluable participants with baseline transfusion requirement had at least a 50% reduction in transfusions over a rolling 8-week window on study compared to baseline

Hemoglobin Increase from Baseline in All Patients



Transfusion Frequency Over Time[1]

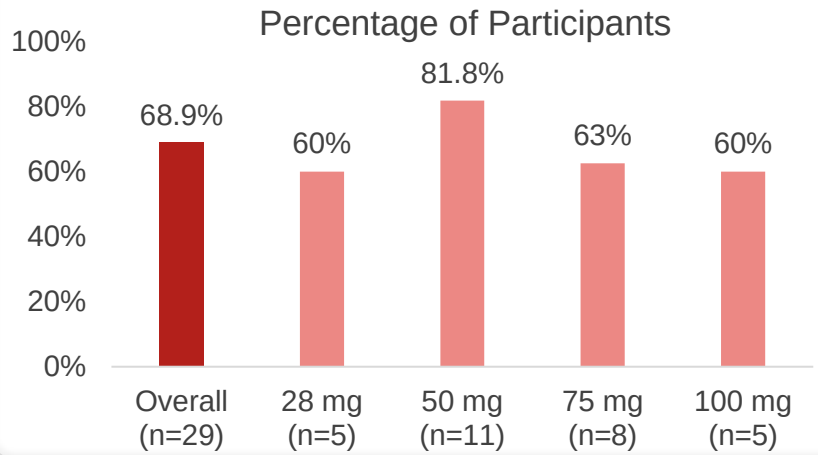


[1]Transfusion frequency of participants with baseline transfusion requirement and at least 12-weeks of follow up. I indicates 2 units transfused; all other transfusions are 1 unit. One of 2 evaluable TD participants (104-005) achieved a TI per Gale criteria. * denotes concomitant MF-directed therapy. EOS = end of study; TD = transfusion dependent; TI = transfusion independent.

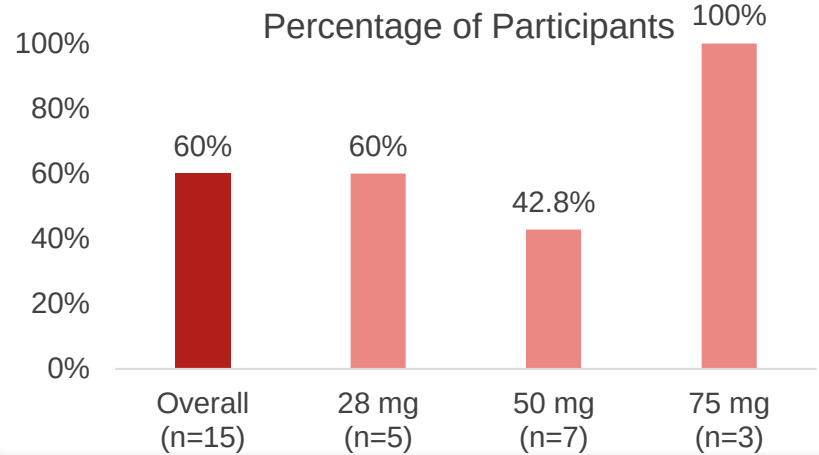
Updated DISC-0974 Anemia of MF Data: Hemoglobin Response in NTD Participants

- Hemoglobin responses of ≥1.5 g/dL increase were achieved in 68.9% of NTD participants
- For participants who have completed at least 16 weeks of the study, 60% of NTD had a mean hemoglobin response of 1.5 g/dL above baseline sustained for at least 12-weeks

NTD Participants with Hgb Δ ≥1.5 g/dL



NTD Participants with Hgb Δ ≥1.5 g/dL for ≥12 Weeks[1]



[1]Percent of non-transfusion dependent participants with mean hemoglobin ≥ 1.5 g/dL above baseline for at least 12 weeks (minimum of 16 week follow up required for inclusion and maximal follow up of 169 days). NTD = non-transfusion dependent; Hemoglobin response was achieved in 62.5% of participants on concomitant JAKi and 71.4% in those not on concomitant JAKi

Updated DISC-0974 Anemia of MF Data: Safety

- Generally well tolerated at all evaluated dose levels

Adverse events at least possibly related to DISC-0974	14 mg (N=1)	28 mg (N=7)	50 mg (N=12)	75 mg (N=8)	100 mg (N=6)
Participants with event (n)	**0**	**3**	**5**	**1**	**1**
Diarrhea	0	1	2	1	0
Injection site bruising	0	1*	0	0	0
Pyrexia	0	1*	0	0	0
Blood bilirubin increased	0	0	0	0	1
Platelet count decreased	0	0	1*	0	0
Anemia	0	0	1*	0	0
Urinary tract infection	0	1*	0	0	0
Headache	0	0	1	0	0
Hot flush	0	0	1	0	0

AE = adverse event; JAKi = Janus kinase inhibitor
Grade 3 AEs include headache reported in 1 participant treated at 28 mg (unlikely related to DISC-0974) and Grade 3 anemia reported in 2 participants treated at 28 mg and 4 participants treated at 50 mg (one at 50 mg was deemed related to DISC-0974; all others were deemed not related). Serious AE: Grade 2 arthralgia was reported in 1 participant treated at 28 mg (not related to DISC-0974). There were no ≥ Grade 4 AEs reported. Liver iron concentration was obtained at baseline and end of study; for available participants (n=10), median change from baseline was 0.3 mg/g dry weight, range (-0.5 to 16.2). * indicates AE in a participant receiving concomitant JAKi therapy.

Summary of Updated DISC-0974 MF Data

Decreased hepcidin & increased iron

68.9% of NTD pts had Hgb response ≥1.5g/dL

60% of NTD pts had Hgb response sustained for ≥12 weeks*

100% of pts with baseline transfusions had ≥50% reduction

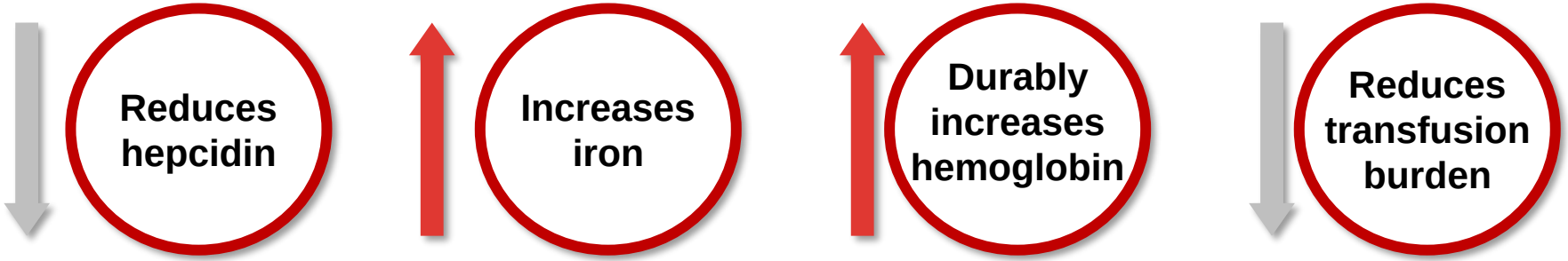
1 of 2 TD pts reached TI

Generally well tolerated

*for participants who have completed at least 16 weeks on study

Summary of DISC-0974 in MF Anemia

DISC-0974 demonstrated improved hemoglobin response and transfusion burden in MF



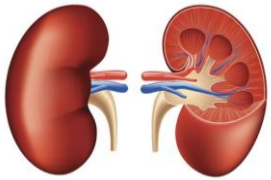
➢ **Next Steps**

- End of Phase 1b meeting with regulators in H2 2024
- Initiation of Phase 2 study by the end of 2024

Hepcidin is a Key Driver of CKD Anemia

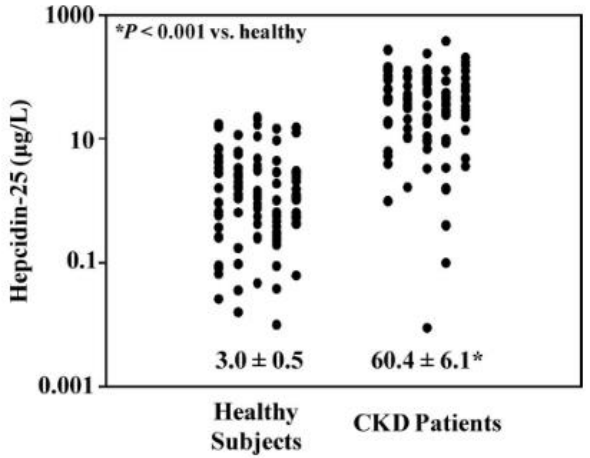
Pervasive issue that is currently highly under-treated

Anemia of CKD



- **Est. # Patients**
  - 5 to 6 million anemic NDD-CKD patients in the US alone
- **Etiology of Anemia**
  - High hepcidin from inflammation & poor renal clearance
  - Compromised erythropoietin production
- **Unmet Medical Needs**
  - Majority patients untreated or under-treated
  - ESAs restricted due to safety and black box
  - Mean Hb 9.3 g/dL in patients initiating dialysis

Hepcidin Levels Elevated in CKD Patients
~20x higher than healthy subjects and increases with disease severity

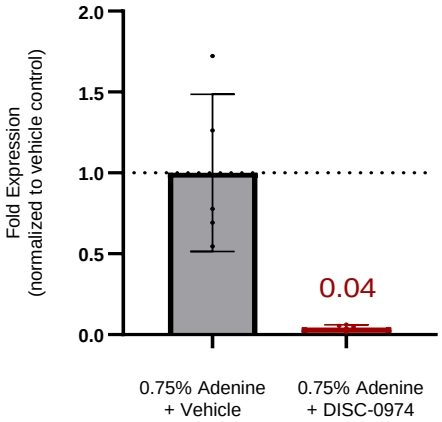


Sources: Troutt et al. (2013), J Clin Lab Analy; Sheetz et al. (2019) Br J Clin Pharmacol. (2019); *from third party; NDD: Non-Dialysis Dependent; DD: Dialysis Dependent

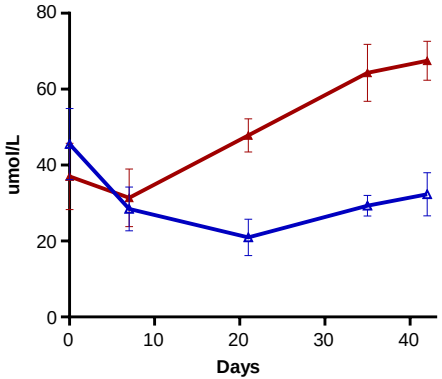
DISC-0974 Improved Anemia in Model of CKD

Rat Model of Adenine Diet-Induced CKD

DISC-0974 Reduced Hepcidin Expression

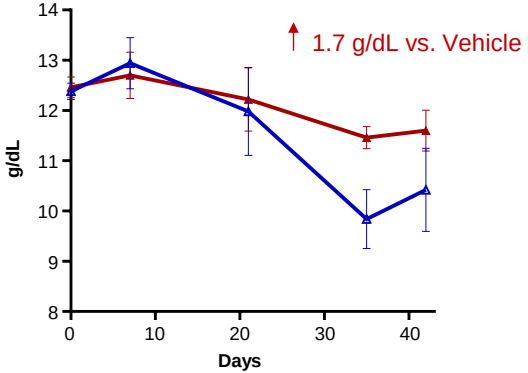


DISC-0974 Increased Serum Iron



Red: 0.75% Adenine + DISC-0974 (20 mg/kg)
Blue: 0.75% Adenine + Vehicle

DISC-0974 Increased Hemoglobin Levels



Red: 0.75% Adenine + DISC-0974 (20 mg/kg)
Blue: 0.75% Adenine + Vehicle

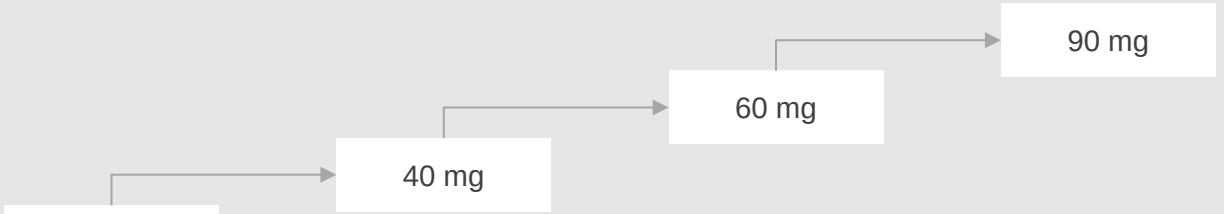
DISC-0974 NDD-CKD Anemia Trial Overview

Data as of October 20, 2023

Trial Population

- Stage II-V CKD; Adult
- Not receiving dialysis
- Hgb (g/dL) <10.5 (F), 11 (M)
- Exclude iron-deficient anemia by ferritin and TSAT

Phase 1b | Single-Ascending Dose



N = 8 per Cohort (6 Active, 2 Placebo)
Assessment of safety, PK, hepcidin, and iron after each dose level

Key Endpoints/Measures: Change in hemoglobin; iron, hepcidin, and other hematologic parameters, safety / tolerability

Data availability: Initial data presented at ASH 2023; updated Phase 1b data to be presented 2H 2024

	DISC-0974 28 mg	Placebo
Enrolled	6	2
Median Age (range), years	69.5 (55, 78)	74.5 (73, 76)
Median Baseline Hemoglobin (range), g/dL	9.7 (7.9, 10.5)	9.5 (9, 10)

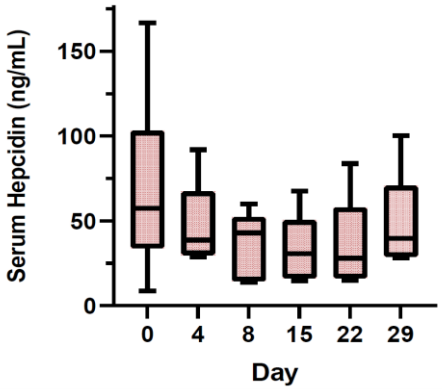
CKD = chronic kidney disease; Hgb = hemoglobin; NDD = non-dialysis dependent; PK = pharmacokinetics; TSAT = transferrin saturation

ASH 2023 DISC-0974 Anemia of CKD Data: Hepcidin and Iron

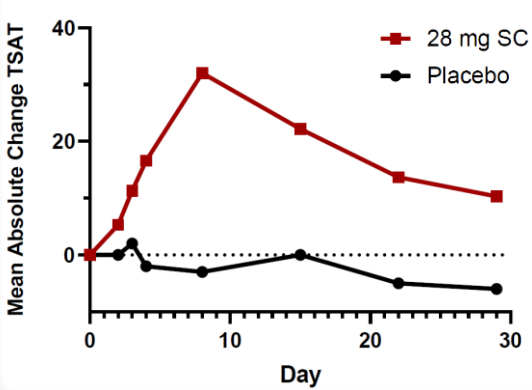
First Cohort: 28 mg SC

- Meaningful reduction in serum hepcidin with corresponding increase in serum iron
- Similar PK/PD relationship as seen in healthy volunteers

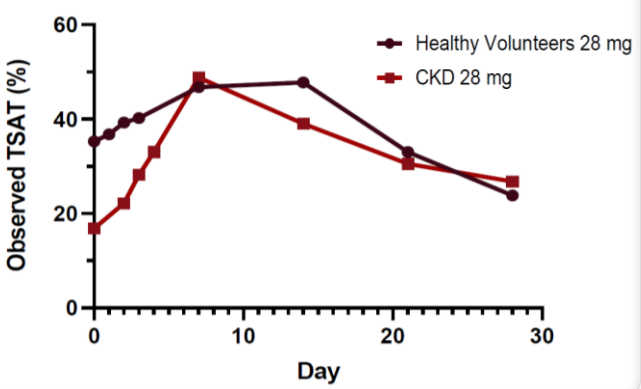
Hepcidin Changes Over Time



Iron Changes Over Time



Iron Changes Over Time vs. HV



Safety: DISC-0974 was generally well tolerated to date; 2 subjects treated with DISC-0974 28 mg had a TEAE (33%) vs. 2 on placebo (100%); 2 treated subjects had SAEs deemed not related to DISC-0974*

1 patient received IV iron during study week 4. For this patient, the iron and hepcidin data through week 3 only was included in this analysis; *1 "atrial fibrillation" and 1 "worsening of end stage renal disease; CKD = chronic kidney disease; HV = healthy volunteer; IV = intravenous; PK/PD = pharmacokinetic/pharmacodynamic; SAE = serious adverse event; SC = subcutaneous; TEAE = treatment-emergent adverse event

DISC-3405

Anti-TMPRSS6 mAb

Hepcidin Induction

Anti-TMPRSS6 mAb Induces Hepcidin

Designed to limit iron levels with potential to address a wide range of hematologic disorders

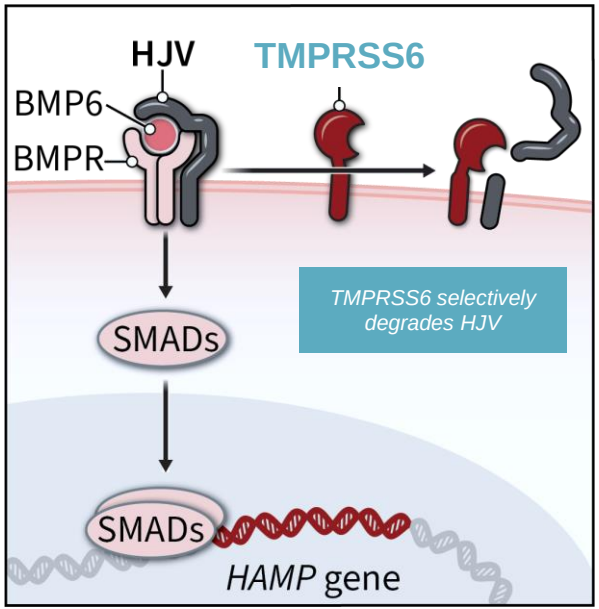
Enables Endogenous Production of Hepcidin

Promotes Iron Restriction
Decreases GI Absorption

Erythrocytosis (PV)
Ineffective Erythropoiesis
Iron Overload

Targeting TMPRSS6 to Increase Hepcidin

Potent, specific target controls endogenous hepcidin production



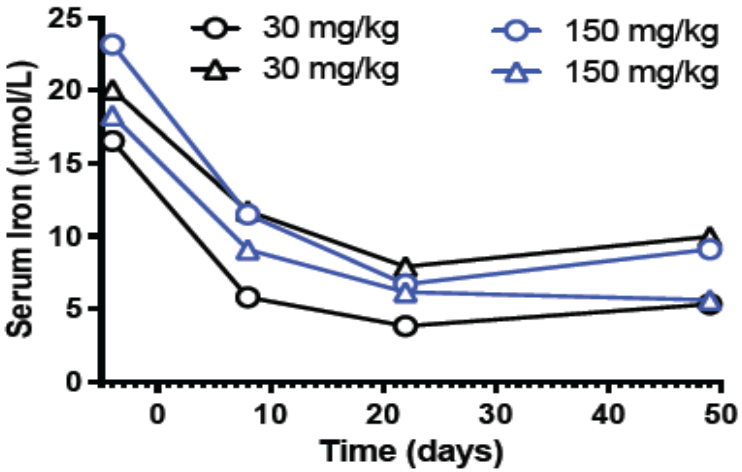
Inhibiting TMPRSS6 with an Antibody Enables Hepcidin Production to Suppress Iron

- **Genetic validation** in patients with IRIDA (Iron-Refractory Iron Deficiency Anemia)
  - LOF TMPRSS6 mutation increases hepcidin and reduces iron availability
- **Functionally specific** to hepcidin / iron
- **Tissue specific** expression primarily in the liver

DISC-3405 Effects in Non-Human Primates

Resulted in deep and sustained suppression of serum iron levels

Single dose of DISC-3405 resulted in ~ 70% suppression of serum iron lasting 3 weeks



- Potent PD effects observed across multiple preclinical studies consistent with TMPRSS6 inhibition
  - Hepcidin: 3-4 fold induction
  - Serum iron: ~60-70% suppression
- DISC-3405 demonstrated excellent safety profile in non-clinical GLP safety studies

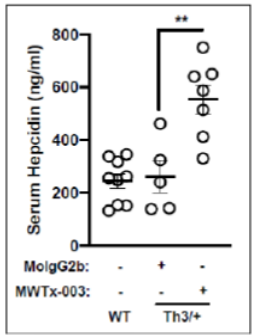
Chen B. et al Blood (2021) 138 (Supplement 1): 941, ASH 2021 Annual Meeting

DISC-3405 in Beta Thalassemia and Polycythemia Vera

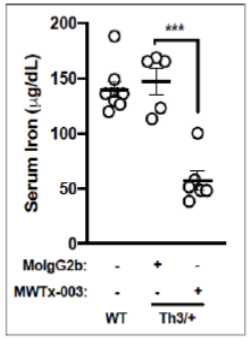
Significant effects on hallmarks of disease

$Hbb^{Th3/+}$ Model of Beta-Thalassemia

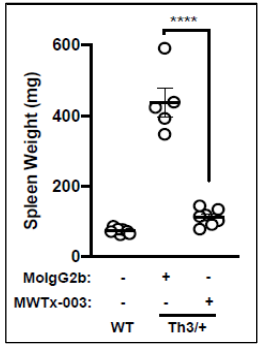
↑ Hepcidin Production



↓ Iron

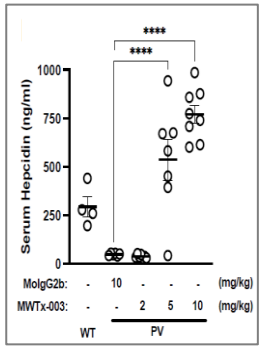


↓ Spleen Weight

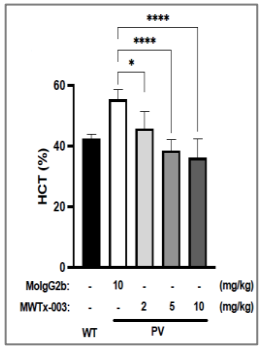


$Jak2^{V617F}$ model of Polycythemia Vera

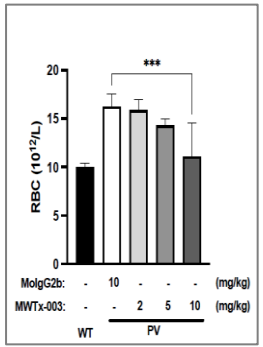
↑ Hepcidin Production



↓ Hematocrit



↓ RBC Production



Chen B. et al Blood (2021) 138 (Supplement 1): 941, ASH 2021 Annual Meeting; HbbTh3/+ mice were treated with the lead anti-TMPRSS6 antibody at 10 mg/kg IP for 4 weeks; Chen B. et al, ASH 2023 Annual Meeting; JAK2V617F mice were treated with anti-TMPRSS6 mAb MWTx-003 at 2, 5, or 10 mg/kg IP every 4 days for 3 weeks

DISC-3405 Development Plans

Phase 1 in healthy volunteers ongoing; aim to advance program into POC studies with focus on polycythemia vera

Phase 1 SAD/MAD in HV
Initiated October 2023

Demonstrate proof-of-mechanism (hepcidin, iron, hematologic parameters)

Phase 2 Proof-of-Concept Study in Polycythemia Vera

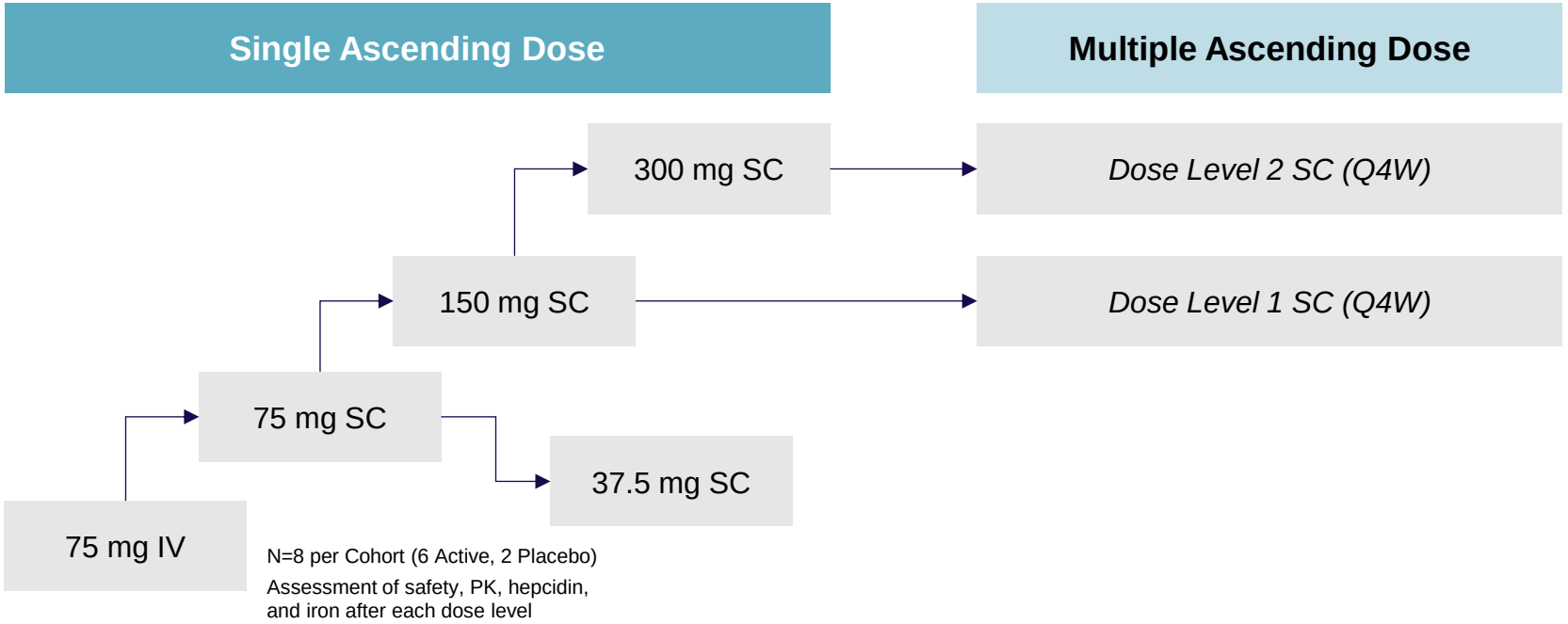
- Strong proof of therapeutic hypothesis; clarity on regulatory development path
- Assess safety, PK, hepcidin, iron, hematologic parameters; % Hct and requirement for phlebotomy

Additional POC Studies in a Range of Indications

- Hereditary Hemochromatosis
- Beta-Thalassemia
- Myelodysplastic Syndromes

Hct = hematocrit; HV = healthy volunteers

DISC-3405 Phase 1 Healthy Volunteers Study Overview



Key Endpoints/Measures: Iron, hepcidin, and other hematologic parameters, safety/tolerability

DISC-3405 Phase 1 Healthy Volunteer SAD: Baseline and Demographics

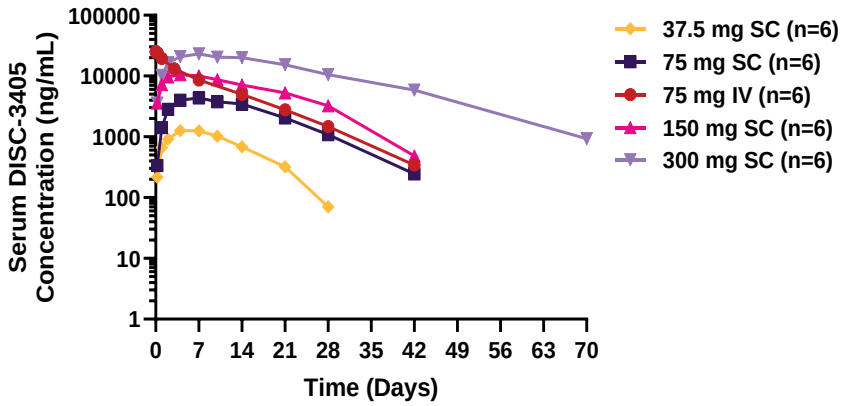
Characteristic	Placebo n = 10	37.5 mg SC n = 6	75 mg IV n = 6	75 mg SC n = 6	150 mg SC n = 6	300 mg SC n = 6
Age, years	48.6 (39-62)	52.7 (42-64)	36.8 (23, 49)	57.3 (49, 61)	44.0 (25, 57)	34.0 (22, 38)
Gender, Female, n (%)	2 (20)	5 (83.3)	3 (50.0)	4 (66.7)	2 (33.3)	0 (0)
Hepcidin, ng/mL	14.1 (5.2, 28.8)	41.7 (6.1, 177.2)	19.4 (2.0, 36.6)	32.6 (7.2, 69.8)	15.2 (8.7, 20.2)	18.7 (8.6, 45.0)
Serum Iron, ug/dL	97.2 (50, 180)	88.7 (43, 127)	99.2 (74, 127)	95.7 (67, 137)	85.7 (43, 138)	106.2 (54, 135)
Hemoglobin, g/dL	14.9 (13.1, 16.0)	13.2 (10.7, 17.7)	13.8 (12.1, 15.6)	13.8 (12.7, 16.0)	14.2 (13.0, 14.9)	15.4 (14.4, 16.7)
Hematocrit, %	43.6 (38.9, 47.1)	39.7 (34.3, 50.2)	41.5 (37.1, 45.5)	41.0 (38.7, 45.0)	42.3 (39.4, 46.2)	45.2 (42.3, 48.2)
RBC, 10^{12}/L	4.9 (4.2, 5.8)	4.5 (3.9, 5.7)	4.6 (3.8, 5.2)	4.5 (4.2, 5.0)	4.7 (3.9, 5.1)	5.1 (4.8, 5.8)

IV = intravenous; RBC = red blood cell; SC = subcutaneous

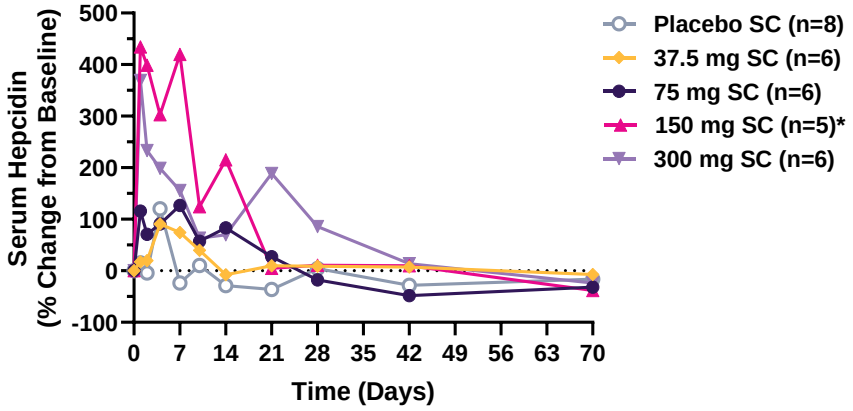
Initial DISC-3405 HV Data: PK and Hepcidin

- Dose-dependent PK profiles
- DISC-3405 demonstrated dose-related hepcidin increases

Pharmacokinetics



Mean Change from Baseline in Serum Hepcidin

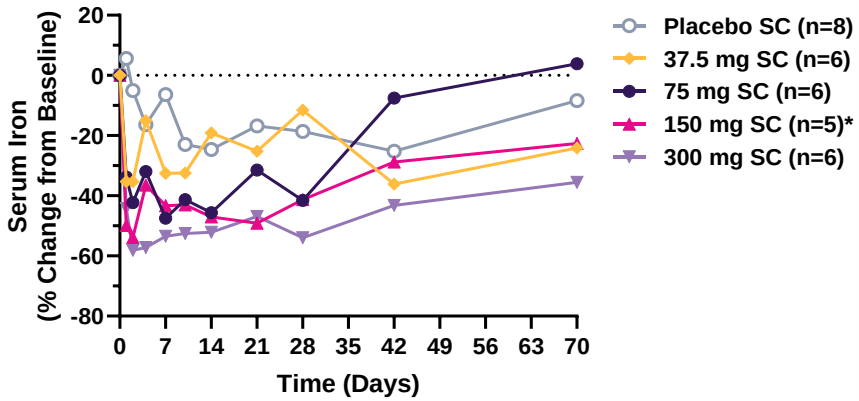


*One participant randomized to 150 mg SC was excluded from PD analysis due to history of anemia and recent hemorrhoidal bleeding, not disclosed prior to enrollment, deeming the participant ineligible; data as of 19 April 2024

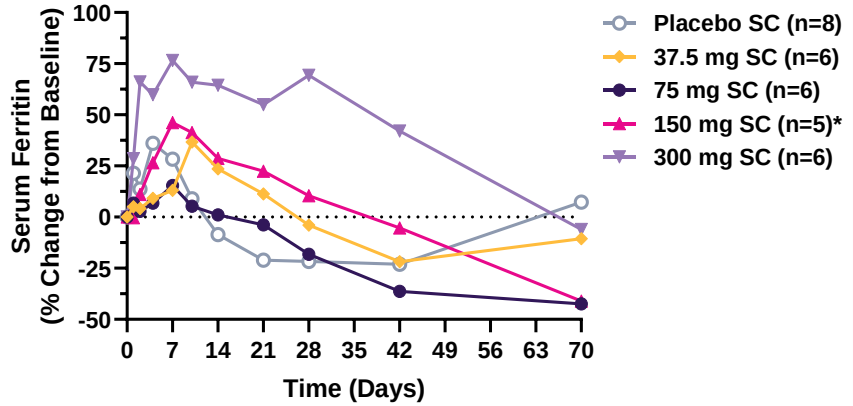
Initial DISC-3405 HV Data: Iron Parameters

- Mean serum iron reduction of more than 50% from baseline was achieved for both 150- and 300-mg doses
- Serum iron reductions were sustained for at least 4 weeks, supportive of monthly SC dosing

Mean Change from Baseline in Serum Iron



Mean Change from Baseline in Serum Ferritin

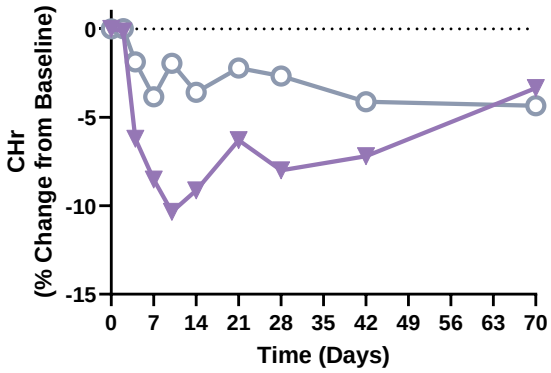


*One participant randomized to 150 mg SC was excluded from PD analysis due to history of anemia and recent hemorrhoidal bleeding, not disclosed prior to enrollment, deeming the participant ineligible; SC = subcutaneous

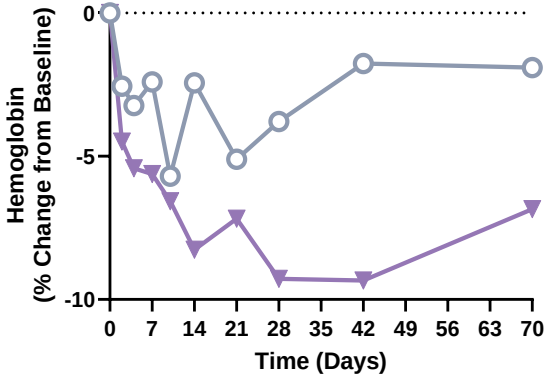
Initial DISC-3405 HV Data: Hematologic Response

- A single 300-mg dose of DISC-3405 demonstrated meaningful reductions in hematologic parameters (reticulocyte hemoglobin, hemoglobin, and hematocrit)

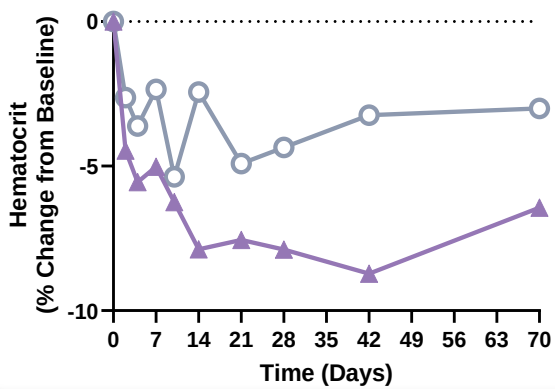
Reticulocyte Hemoglobin (300 mg)



Hemoglobin (300 mg)



Hematocrit (300 mg)



300 mg SC (n=6) Placebo SC (n=8)

CHr = reticulocyte hemoglobin; SC = subcutaneous

Initial DISC-3405 HV Data: Safety

- Generally well tolerated at all evaluated dose levels; no serious AEs, > Grade 2 AEs, or AEs leading to study withdrawal were reported

Adverse Event	Placebo n = 10	37.5 mg SC n = 6	75 mg IV n = 6	75 mg SC n = 6	150 mg SC n = 6	300 mg SC n = 6
Sore Throat	0	0	1	0	0	0
Nausea	0	1	0	1	0	0
Headache	1	1*	0	0	0	0
Cough	0	0	0	0	1	0
Rhinorrhea	0	0	0	0	1	0
Lightheadedness	0	0	0	1	0	0
Increased ALT	0	0	0	0	1*	0
Increased AST	0	0	0	0	1*	0

*Grade 2 AEs; one participant reported a self-limited headache; one participant observed to have isolated, self-limited elevations of AST and ALT.

Summary of Phase 1 Healthy Volunteer SAD Data

- Single-dose SC administration of DISC-3405 demonstrated dose-dependent increases in hepcidin and corresponding reductions in serum iron levels across all dose levels
- >50% reductions in mean serum iron were observed in patients that received 150 mg and 300 mg doses
- PK/PD profile is supportive of monthly subcutaneous dosing in polycythemia vera and iron overload conditions
- DISC-3405 was well tolerated
- **Next Steps**: Phase 1 multiple-ascending dose (MAD) data expected by EOY; initiation of a Phase 2 study in PV expected in 1H 2025

Disc Continues Strong Growth Trajectory Towards Becoming a Leading Hematology Company

	Significant Accomplishments in 1H 2024	*Important Catalysts in 2H 2024-2025*
Bitopertin	Positive data across two Phase 2 studies	• EPP Phase 3 Study pending regulatory feedback • POC in DBA
DISC-0974	Updated positive data in anemia of MF	• Additional POC data in MF and CKD anemia • Preclinical efforts on additional indications
DISC-3405	Initial positive SAD healthy volunteer data	• MAD healthy volunteer data • Polycythemia vera as first indication

Supported by a strong cash position with runway well into 2027

Thank You

disc medicine