Corporate Presentation

May 2021

Forward Looking Statement

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In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target," "project," "contemplates," "believes," "estimates," "predicts," "potential" or "continue" or the negative of these terms or other similar expressions. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These known risks and uncertainties are described in detail in our filings with the Securities and Exchange Commission (the "SEC") from time to time. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995 and we undertake no obligation to revise or update this presentation to reflect events or circumstances after the date hereof.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Building The Next Generation Biotechnology Company

Immunology Focus	• Disruptive research and development engine focused on the disease areas of immunology, inflammation and oncology
Diversified Pipeline	• **2** ongoing proof-of-concept Phase 2 studies in PAH and UC – topline results for both expected in the 1H22, subject to developments in COVID pandemic
World-Class Talent	• Deeply experienced leadership team with proven track record of developing innovative clinical assets

Robust Pipeline with 2 Clinical-Stage Product Candidates in Ongoing PoC Phase 2 Trials

PROGRAM	CLASS (Route of Admin.)	INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	RIGHTS
Seralutinib (GB002)	PDGFR, CSF1R, c-KIT Inhibitor (Inhaled)	Pulmonary Arterial Hypertension	Phase 2 Ongoing – TORREY Study					Worldwide
GB004	Gut-Targeted, HIF-1α Stabilizer (Oral)	Inflammatory Bowel Disease	Phase 2 Ongoing – SHIFT-UC Study					Worldwide
GB1275	CD11b Modulator (Oral)	Oncology, Solid Tumors	Phase 1/2 Ongoing					Worldwide
Internal Research Programs	Multiple Programs in Development	Autoimmune, Inflammation, and Oncology Indications						Worldwide

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Seralutinib (GB002)

PDGF Receptor, CSF1R, & c-KIT Kinase Inhibitor

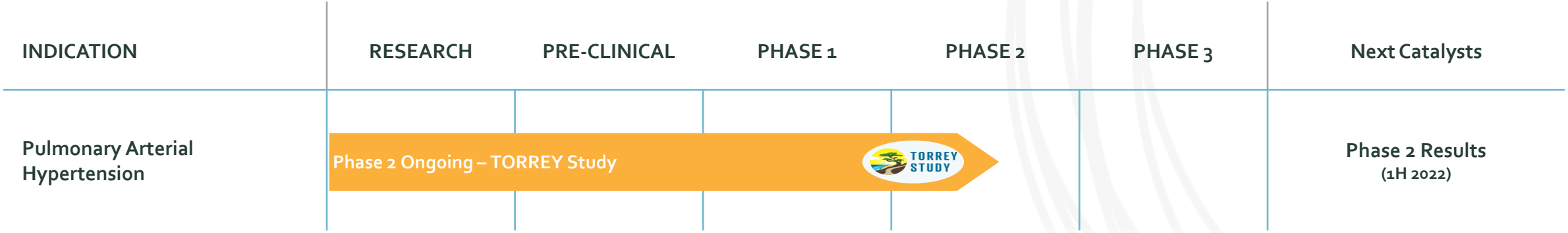
Pulmonary Arterial Hypertension (PAH)

Seralutinib (GB002): Potential To Deliver Disease-Modifying Effects to Patients with PAH

GB002

Product Candidate Description

- Selective, inhaled PDGF receptor, CSF1R, and c-KIT kinase inhibitor designed to address the disease pathogenesis of PAH
- Kinase inhibition was shown to be clinically significant in Phase 3 PAH trial of imatinib (Gleevec), with systemic toxicities (IMPRES Study)
- Seralutinib formulation and administration via dry powder inhaler (DPI) designed to reach areas of the deep lung; DPI device is small, convenient and currently used in commercial products
- Designed to deposit inhaled seralutinib at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- Patent protection to 2039[1]; Orphan Drug Designation from FDA and EMA



1) Includes assumption of maximum 5 years of patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims. Total patent life with patent term extension cannot exceed 14 years from approval.

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PAH is an Orphan Disease With High Unmet Need and Significant Disease Burden

GB002

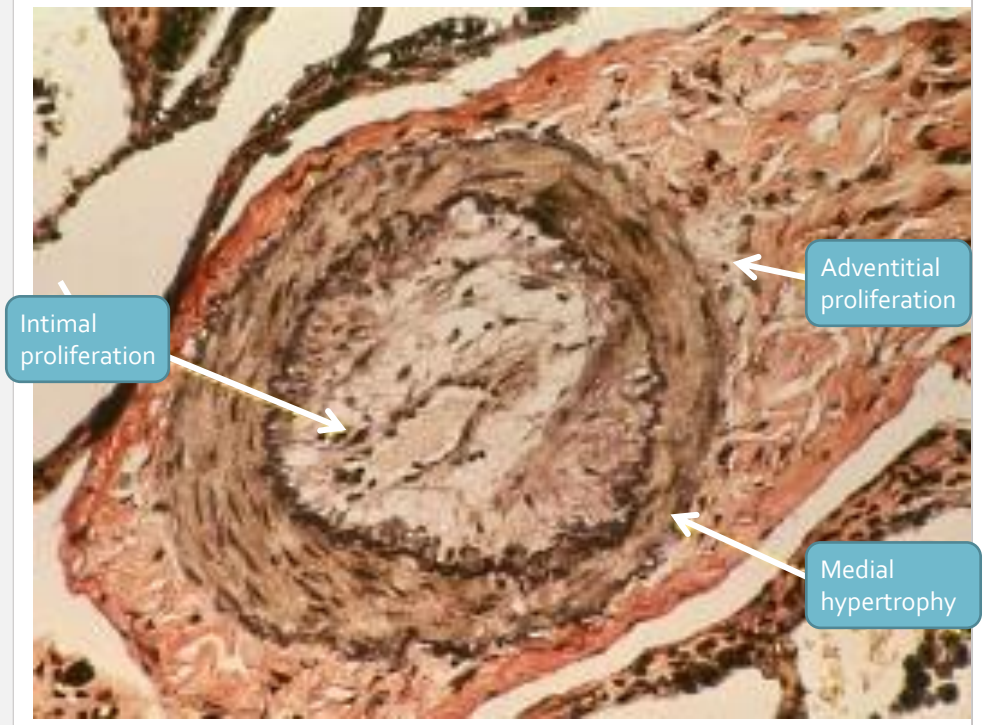
Pulmonary Arterial Hypertension (PAH)

- Rare, orphan disease
- Characterized by high blood pressure in the blood vessels carrying deoxygenated blood from the right side of the heart to the lungs
- Caused when the arteries in the lungs become narrowed, thickened and / or stiff as a result of pathological remodeling and vasoconstriction
- Heart works harder to pump blood to the lungs, potentially leading to right heart failure
- Progressive disease and often fatal

Symptoms

- **Dyspnea**
- **Fatigue**
- **Dizziness**
- **Chest pressure / pain**
- **Edema in ankles, legs, abdomen**
- **Cyanosis**
- **Heart palpitations**

PAH is Characterized by Vascular Remodeling



Muscular pulmonary artery from iPAH patient[1]

Source: [1]Gaine S and Rubin L: Lancet 1998; 352: 719-725

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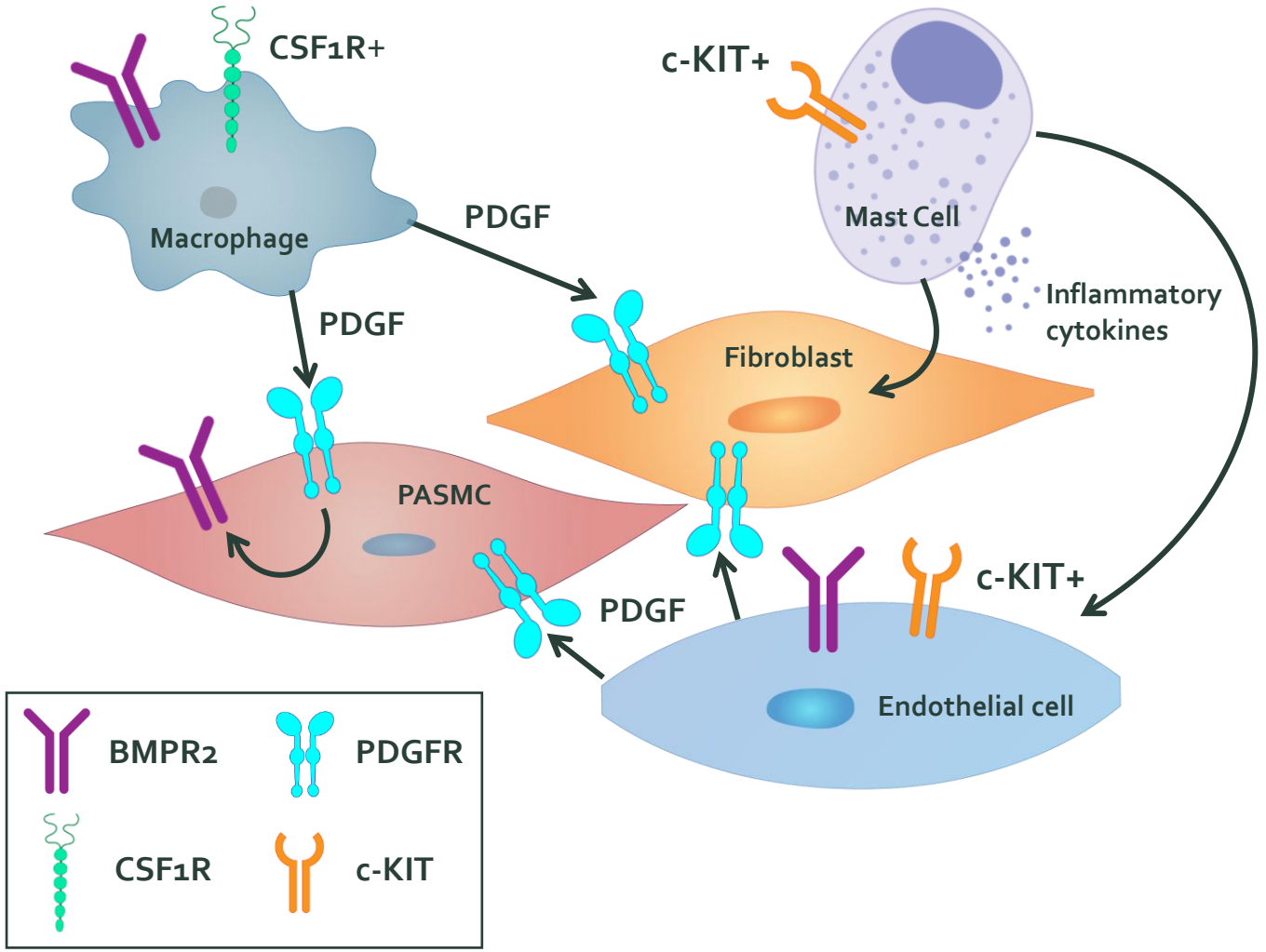
PDGFR,CSF1R, and c-KIT Activation Play a Role in the Pathological Remodeling of Lung Blood Vessels in PAH and Interact with BMPR2

GB002

- Aberrant PDGFR signaling drives overgrowth of smooth muscle cells myofibroblasts leading to medial hypertrophy, neointimal lesions, and fibrosis
- CSF1R+ macrophages secrete PDGF and contribute to inflammation and vascular remodeling
- c-KIT+ cells are increased and further drive the inflammatory and proliferative disease process
- PDGF activation decreases BMPR2, which further drives PASMC proliferation



Source: Grimminger et al dv Exp Med Biol 2010; 661:435; Zhou et al Cell 2018;172:744; Montani et al AJRCCM 2011; 184:116; Chen et al BMC Genomics 2016 17:781.

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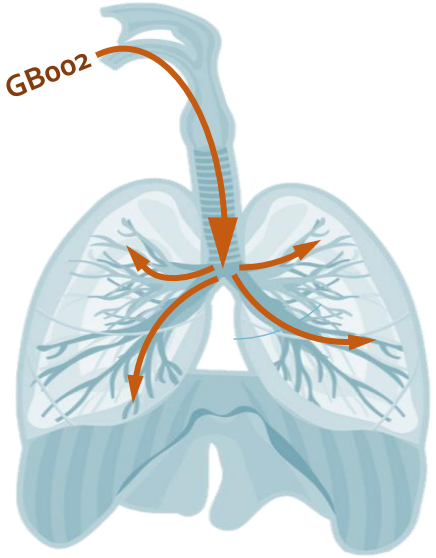
Seralutinib Administration via Dry Powder Inhaler is Designed to be Convenient and to Deliver Drug Directly to the Site of Disease

GB002

Dry Powder Inhaler from Plastiape



- Seralutinib formulation and administration via dry powder inhaler (DPI) designed to reach areas of the deep lung
- Designed to deposit inhaled seralutinib at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- In pre-clinical studies, has resulted in 30x ratio of lung-to-systemic exposure, potentially providing for an improved therapeutic index in the clinic
- DPI device is small, convenient and currently used in commercial products

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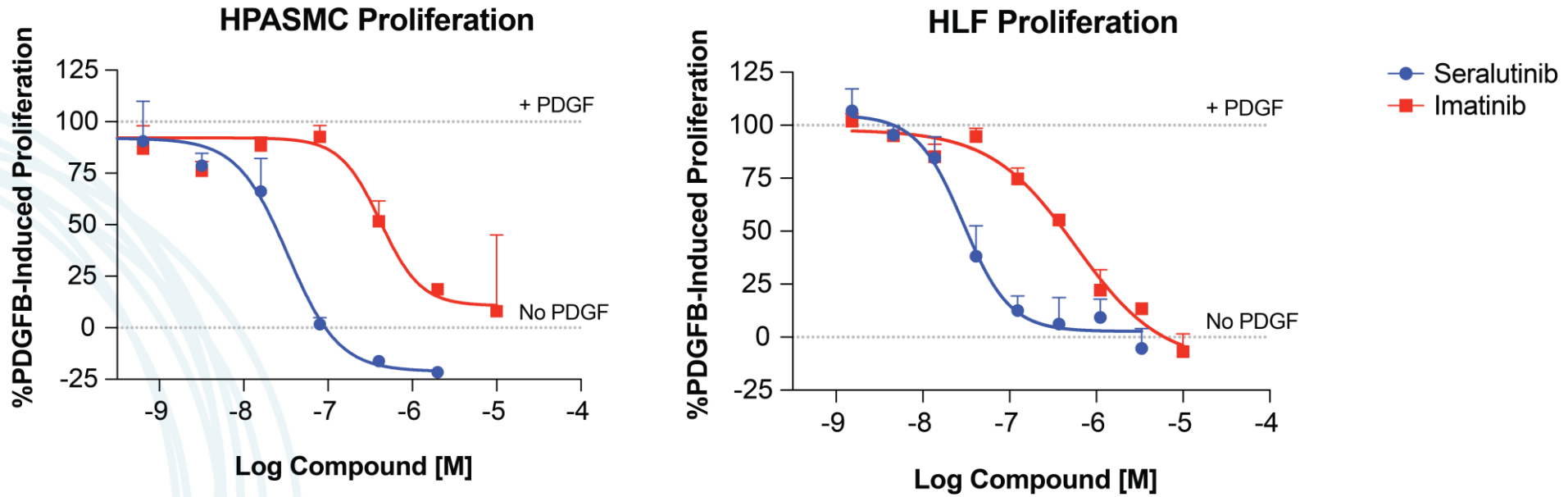
Seralutinib In Vitro Profile

GB002

Seralutinib is a potent PDGFR, CSF1R and c-KIT inhibitor

Compound	Cell Based IC50 (nM)				
	H1703 PDGFRα	HLF PDGFβ>α	PASMC PDGFRα=β	CSF1R	c-KIT
Seralutinib	32	29	33	8	14
Imatinib	62	579	419	1032	230

Seralutinib is highly potent in PASMC and HLF proliferation assays



HPASMC: human pulmonary arterial smooth muscle cell; HLF: human lung fibroblast

Source: Ten Freyhaus, *Arterioscler Thromb Vasc Biol* 2015, 35(5):1236; Barst, *J Clin Invest* 2005, 115(10):2691; Gomez-Arroyo, et al, *Am J Physiol* (2012) 302(10):L1014; Sawada, et al, *J Exp Med* (2014) 211 (2):263; Talati, et al, *PLoS One* (2014) 9(4):e94119; Abid, et al, *Eur Respir J* 2019, 10;54(4):1802308; Savai, et al., *Am J Respir Crit Care Med* 2012, 186(9):897; Montani, et al., *Am J Respir Crit Care Med* 2011, 184(1):116

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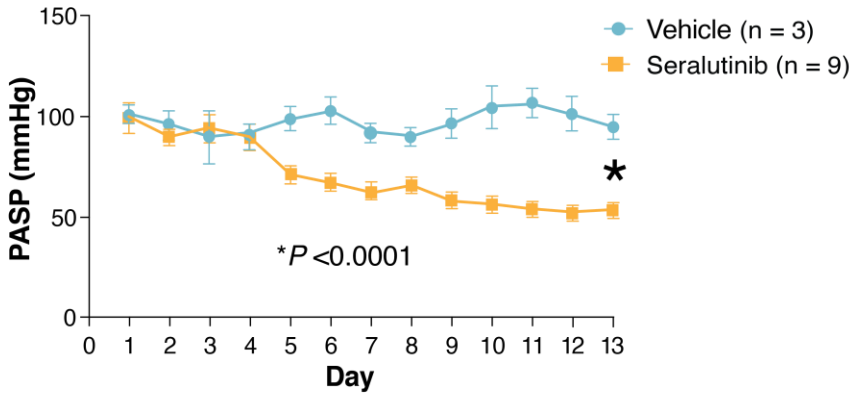
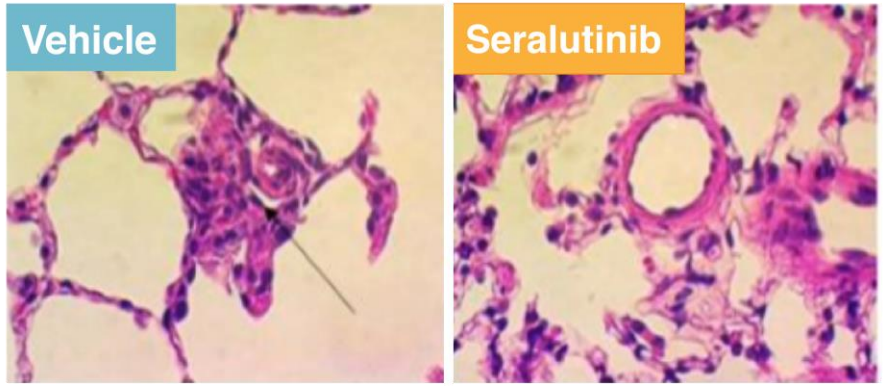
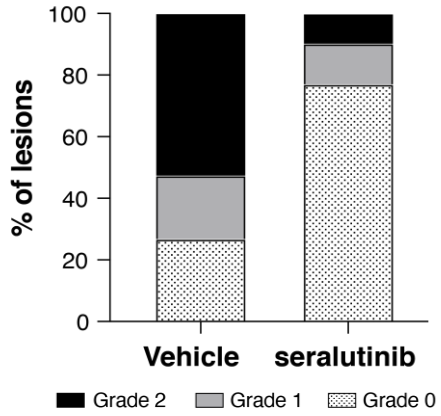
Seralutinib Demonstrates Efficacy in the SU5416/Hypoxia and MCT/PN Models

GB002

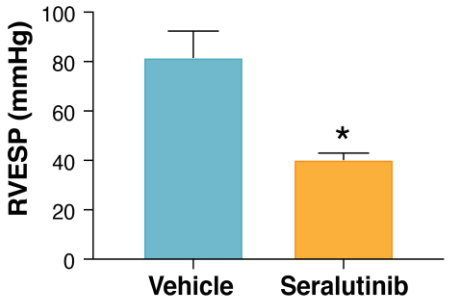
SU5416/H telemetry study

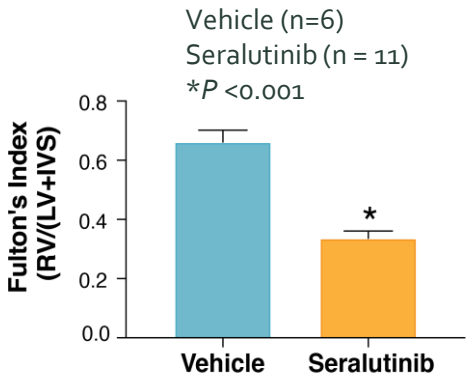


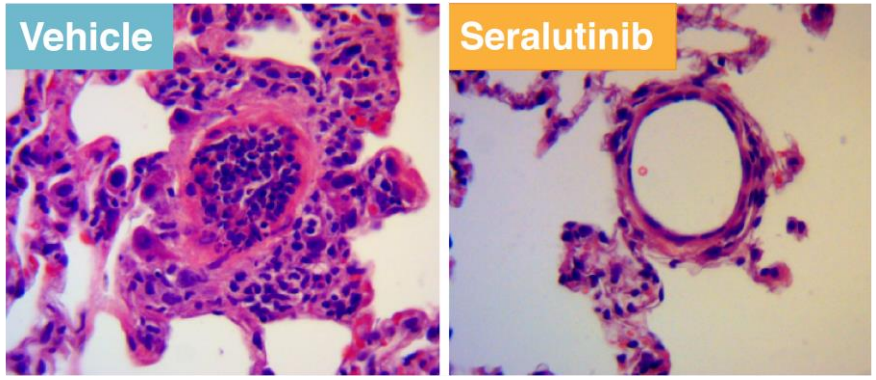


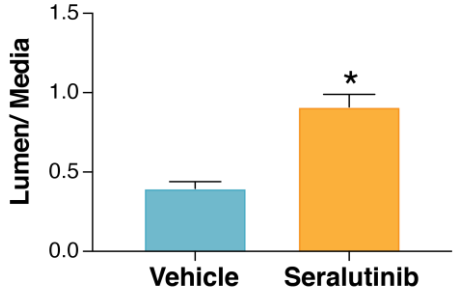


MCT/PN study









SU5416: Sugen 5416 MCT/PN: monocrotaline pneumonectomy; PASP: pulmonary artery systolic pressure; RVESP: right ventricular end-systolic pressure-volume; RV/(LV+IVS): right ventricular weight/weight of left ventricle plus interventricular septum

Source: Galkin, et al., Manuscript in preparation; Sitapara, et al., *Circulation* 2019;140: A12947.

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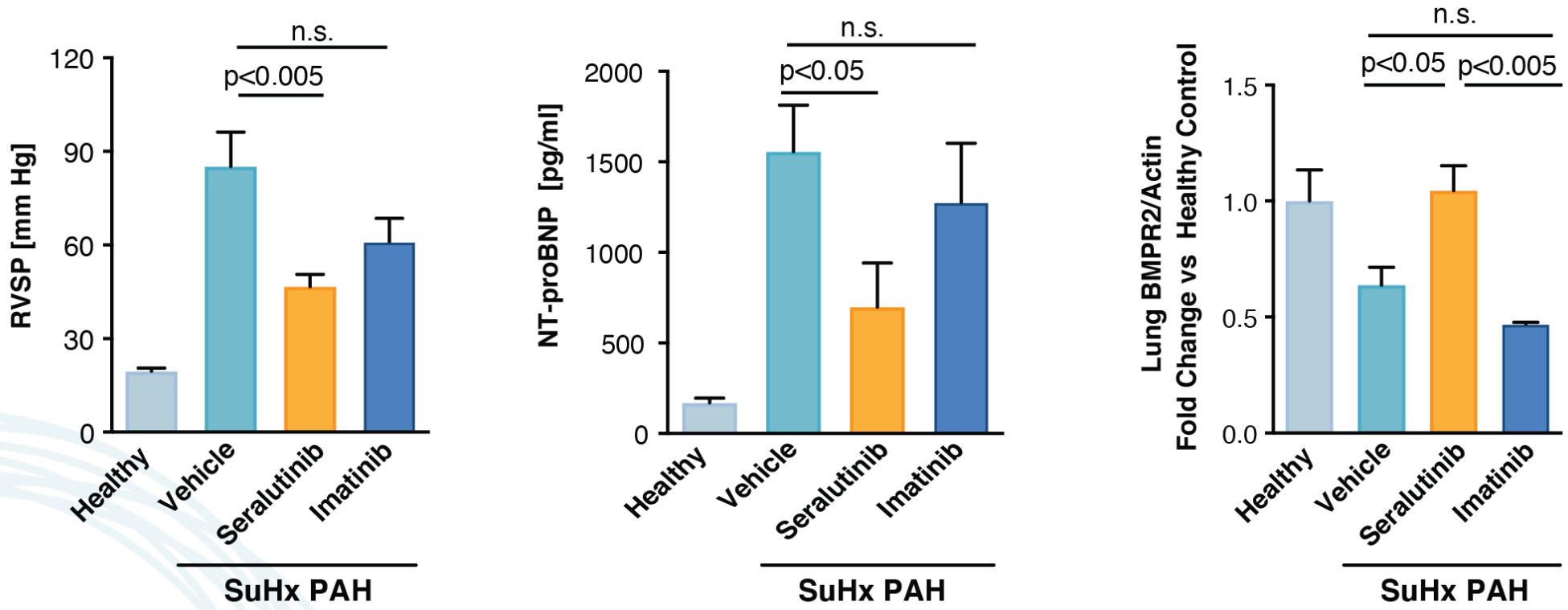
Inhaled Seralutinib Outperformed Oral Imatinib in a Head-to-Head Preclinical SuHx PAH Study

GB002



Data presented as Mean +/- SEM. Statistical analysis was performed using one-way ANOVA with Dunnett's multiple comparisons test (Healthy n=8; Vehicle n=7; Seralutinib n=9; Imatinib n=7)

- Seralutinib treatment led to a significant improvement in RVSP
- Seralutinib reduced circulating levels of NT-proBNP and increased lung BMPR2 protein expression

SuHx: Sugen hypoxia; RVSP: right ventricular systolic pressure
Source: Galkin, et al. Circulation 2019, 140: A11102

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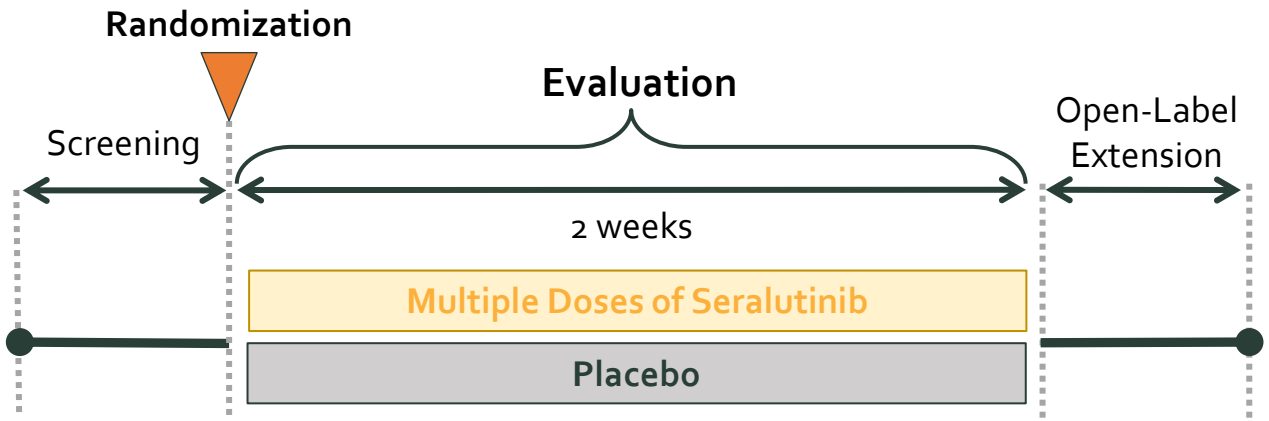
Phase 1b Study in Patients with PAH

GB002



- First patient enrolled Q1:20; prior to pandemic-related site closures, 5 patients (4 active and 1 placebo) completed two weeks of treatment
- Study was re-opened with COVID precautions in Q3:20, allowing enrollment of 3 additional patients (N = 8 total)

Study Objectives

Primary

- To evaluate the safety and tolerability of inhaled seralutinib

Secondary

- To evaluate pharmacokinetics (PK) of seralutinib

Exploratory

- To evaluate pharmacodynamic (PD) biomarker analysis on blood samples and/or circulating cells and/or airway samples

Key Inclusion Criteria

- **Diagnosis PAH,** WHO group 1, FC II-IV
- Prior cardiac catherization data c/w PAH
- Baseline 6MWT >100 m
- On PAH background medications

Dosing

- 45 mg to 90 mg BID dose escalation at PI discretion

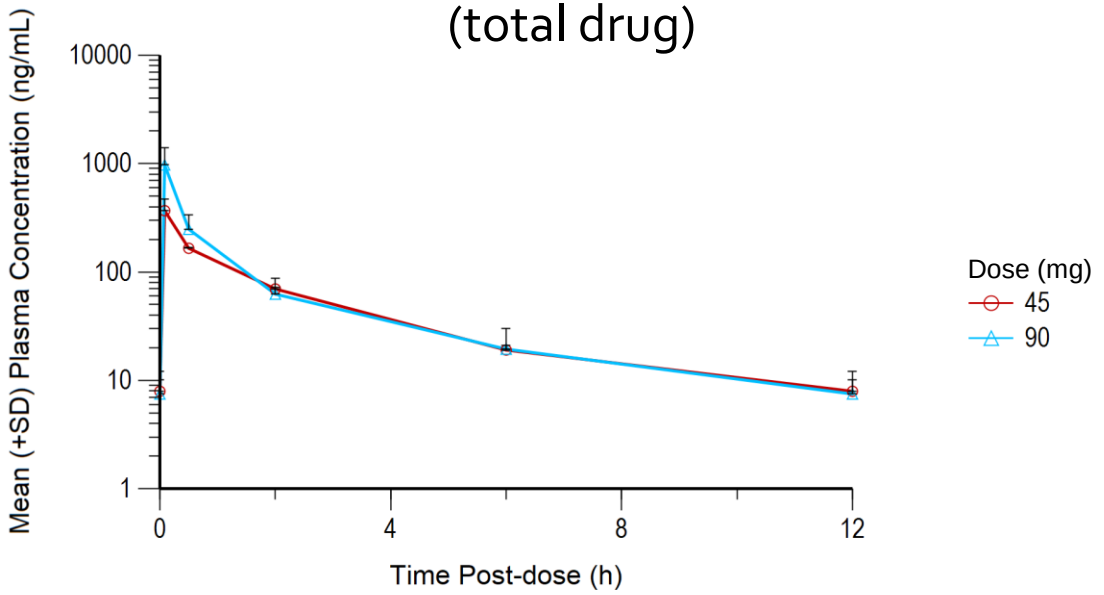
Source: clinical trials.gov/NCT03926793

Preliminary Phase 1b Results: Safety and Pharmacokinetics

GB002

- Seralutinib generally well tolerated in PAH patients
- All 8 subjects completed the 2-week study
- No SAEs were reported
- The most frequently reported AEs were:
  - Cough (mild-moderate)
  - Headache (mild)
- There were no clinically significant changes in labs, ECGs, PFTs, and vital signs.
- PK in PAH patients consistent with PK data from Healthy Volunteers
- Systemic PK characterized by low systemic exposure and rapid clearance

Systemic PK
(total drug)



- Profile is consistent with an inhaled therapy and the potential for a favorable therapeutic index

ECG: electrocardiogram; PFT: pulmonary function test
Source: Data on File

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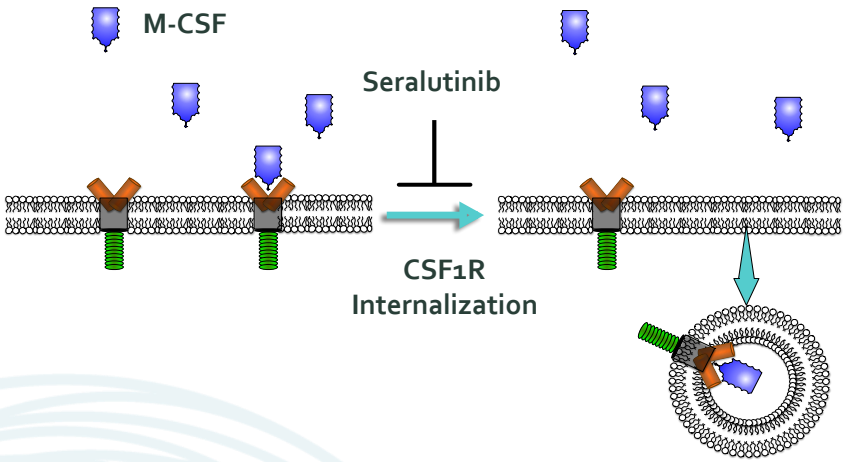
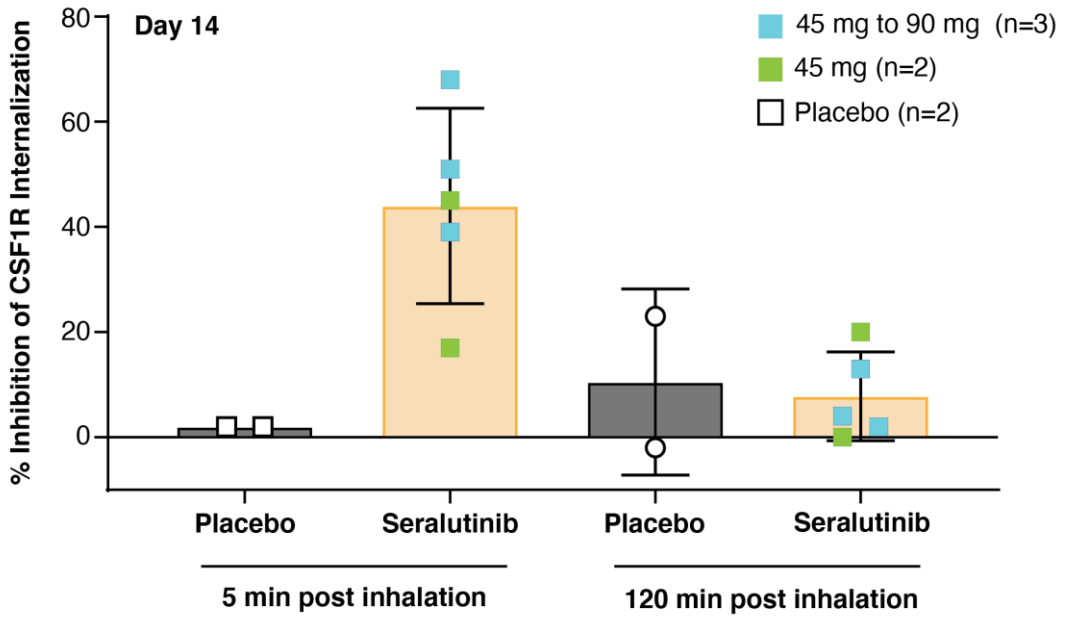
Preliminary Phase 1b Results: Seralutinib Demonstrated Target Engagement

GB002





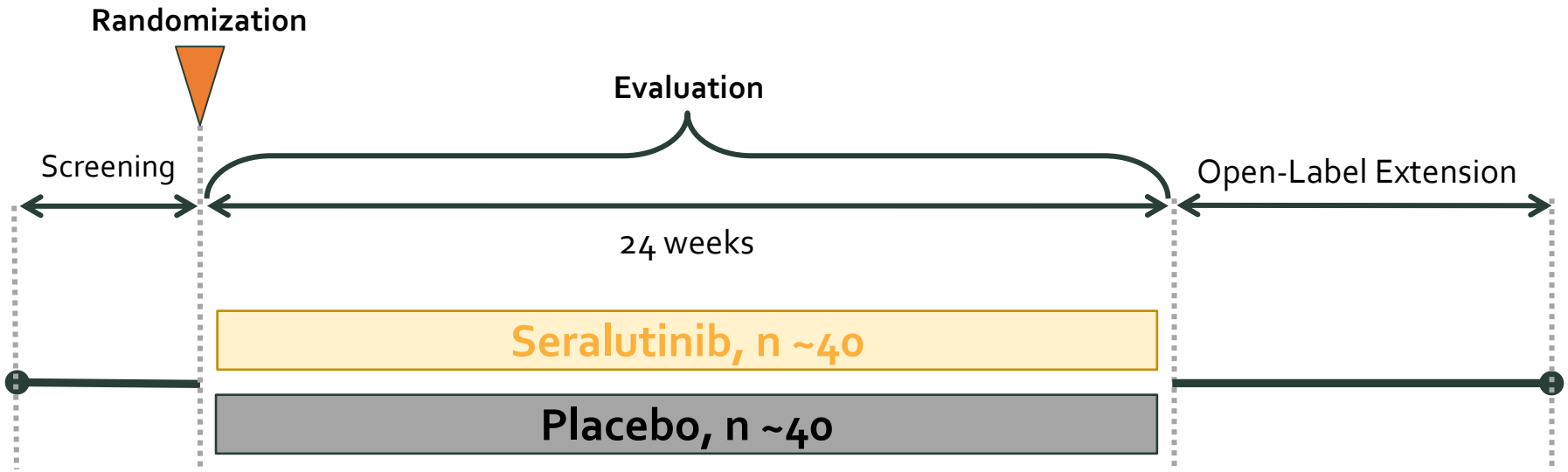
- Seralutinib blocks CSF1R internalization at 5 min post inhalation demonstrating successful Target Engagement, followed by designed rapid systemic clearance (lack of signal at 120 min)
- Rapid systemic clearance combined with sustained lung PK seen in animal studies potentially provides for greater therapeutic index

Source: Data on File

TORREY: Phase 2 Study of Seralutinib in Patients With Functional Class II and III PAH

GB002

TORREY STUDY



Patient Population	Functional Class II and III PAH patients on standard background therapy (including triple therapy); PVR >/= 400 dyne*s/cm^5
Endpoints	**Primary:** PVR Change from Baseline at Week 24 **Key Secondary:** 6MWD Change from Baseline at Week 24

PVR: pulmonary vascular resistance; 6MWD: six-minute walk distance
Source: clinical trials.gov/NCT04456998

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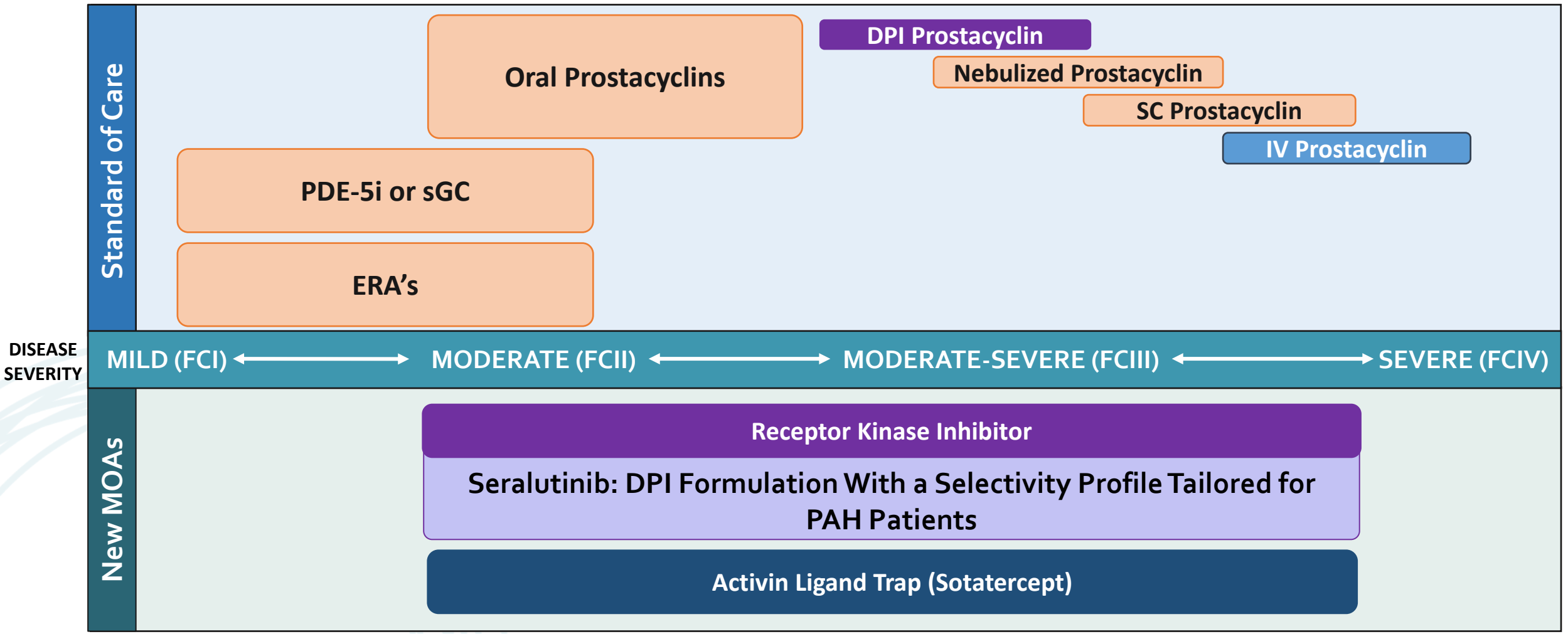
Seralutinib Offers a New, Multifaceted Approach to Treating PAH, Differentiating it From the Competitive Landscape

GB002



Seralutinib is being evaluated on-top of background therapy (≥1 therapy)

KEY

- Approved Self-Admin. Tx
- Novel Self-Admin. Tx*
- Approved Physician Admin. Tx
- Novel Physician-Admin. Tx*

*Hypothesized positioning based on internal analysis

GB004

Gut-Targeted,
Hypoxia Inducible Factor 1-Alpha (HIF-1α) Stabilizer

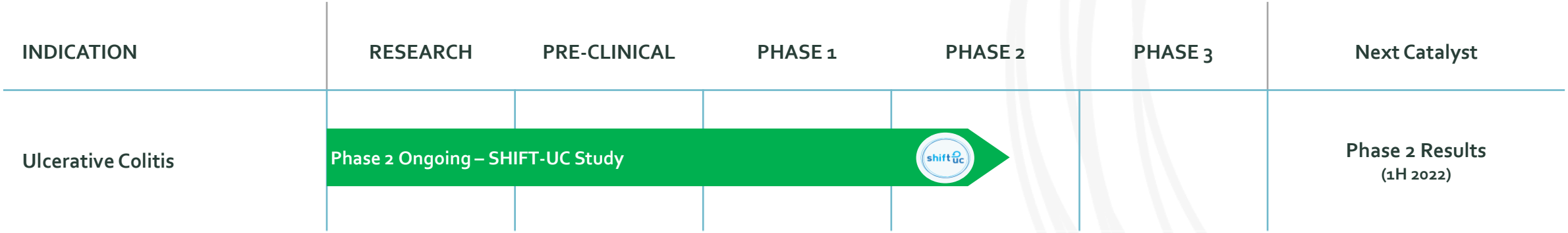
Inflammatory Bowel Disease (IBD),
including Ulcerative Colitis (UC) and Crohn's Disease (CD)

GB004: Gut-targeted, HIF Stabilizer in Development for the Treatment of IBD

GB004

Product Candidate Description

- Oral, small molecule, gut-targeted, prolyl hydroxylase inhibitor that is designed to stabilize HIF-1α for the treatment of inflammatory bowel disease (IBD)
- HIF-1α stabilization restores epithelial barrier function and exerts innate immunomodulatory effects, which is expected to reduce inflammation and enhance mucosal healing in human IBD
- Potential for use as mono or combo therapy for IBD
- Promising results from 4-week Phase 1b study announced in Q2:20
- Patent protection to 2035(1)



1) Includes assumption of maximum 5 years of patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims.

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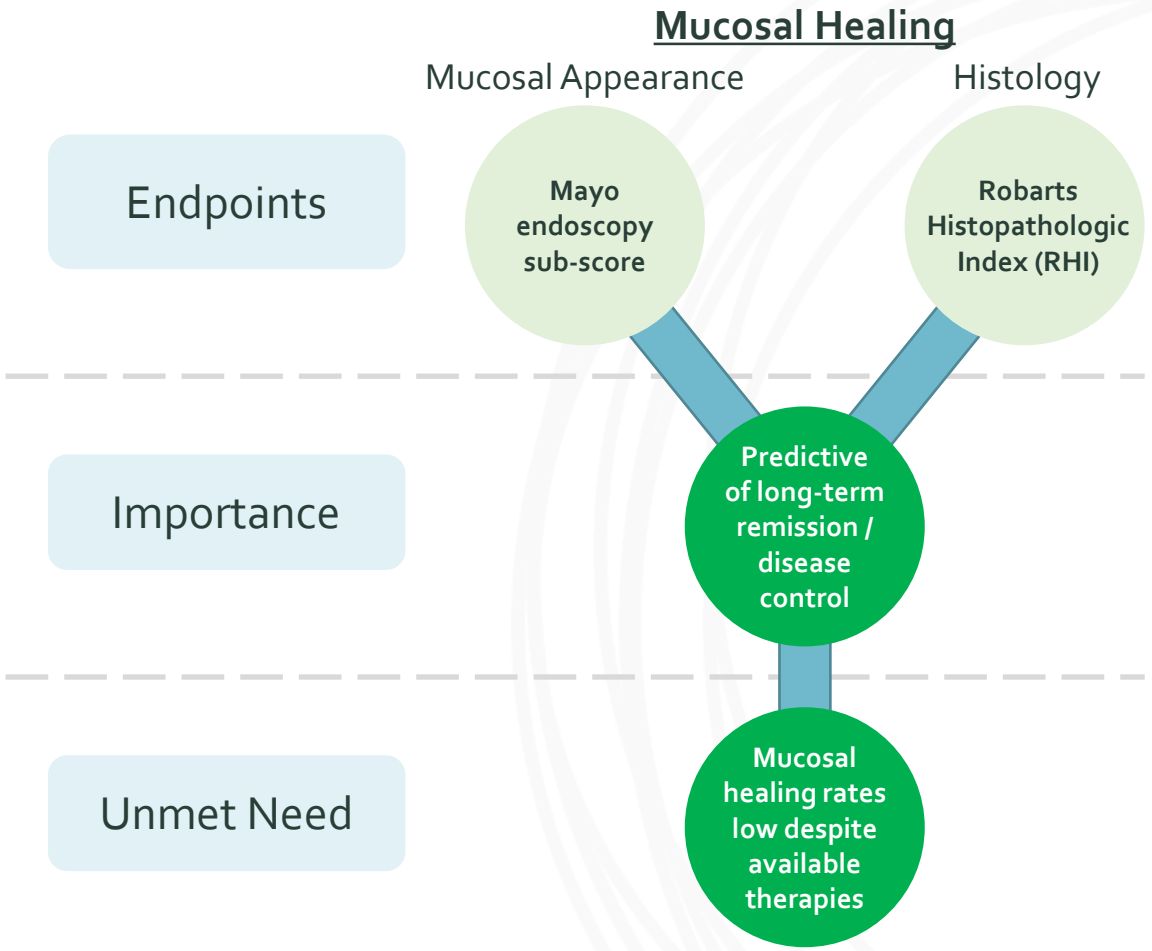
Improved Mucosal Healing is a High Unmet Need in UC

GB004



- Historically, endoscopy alone had been used to evaluate "mucosal healing"
  - Following data and guidance from KOLs, the FDA, and the EMA, histology has emerged as an important third therapeutic target and second component of mucosal healing
- Histology has been shown to be a better predictor of long-term rates of disease relapse, hospitalizations, colectomy, and corticosteroid use than endoscopy
  - While endoscopy assesses mucosal improvement at the tissue level, histology magnifies to the cellular level
- Current therapies do not adequately achieve mucosal healing

Patients in Gossamer's Phase 1b study of GB004 in UC were required to have active disease as evaluated by RHI

Source:
Pai, Rish et. al. "The emerging role of histologic disease activity assessment in ulcerative colitis." Gastrointestinal Endoscopy 88, no. 6, 887 – 898.
Ungaro, Ryan et al. "A Treat-to-Target Update in Ulcerative Colitis: A Systematic Review." The American Journal of Gastroenterology 114 (2019) 874 – 883.

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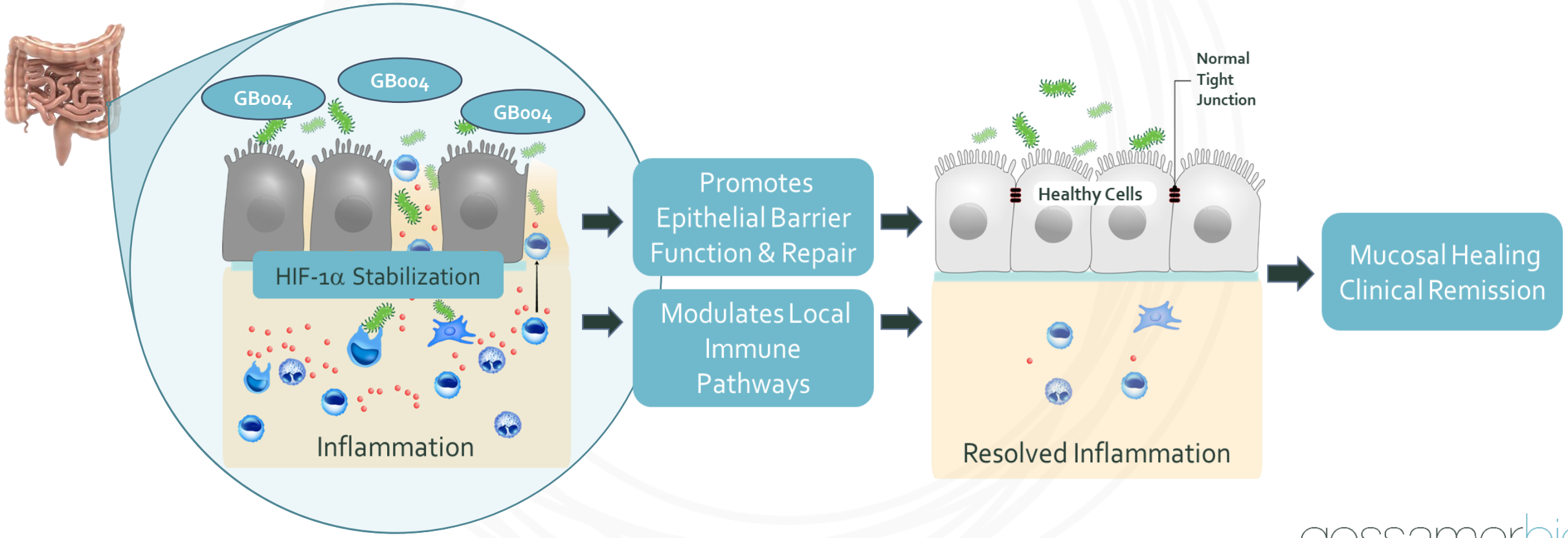
GB004 Targets the HIF-1α Pathway, Demonstrating a Unique Mechanism for Mucosal Healing in Ulcerative Colitis

GB004

- GB004 stabilizes HIF-1α specifically in gut tissue and sustains HIF-1-driven gene expression
- HIF-1α plays a crucial role in the maintenance of epithelial barrier integrity and function
- GB004 facilitates local immune homeostasis in intestinal epithelial cells and promotes mucosal healing



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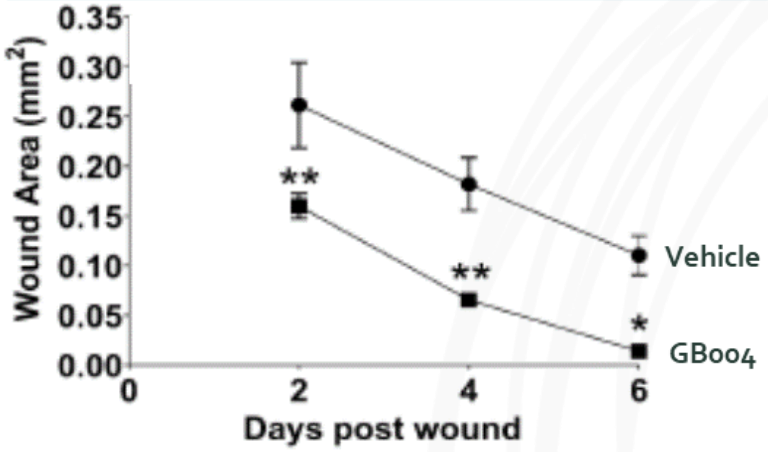
Treatment with GB004 Leads to Accelerated Barrier Repair and Intestinal Wound Healing in Mouse Model

GB004

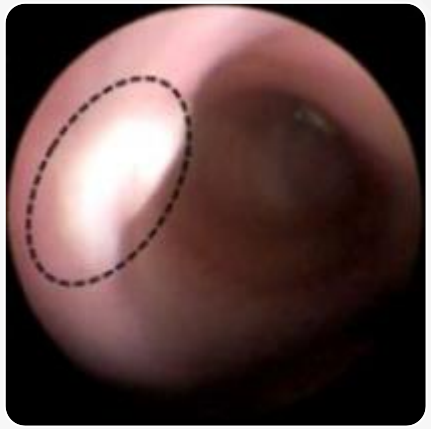
In Vivo Wound Healing Assay

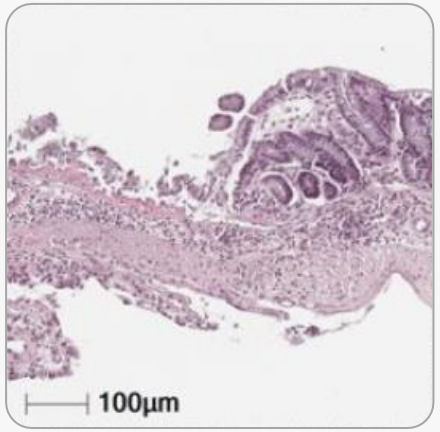


Day 2 Endoscopy

Day 2 Histology

Vehicle





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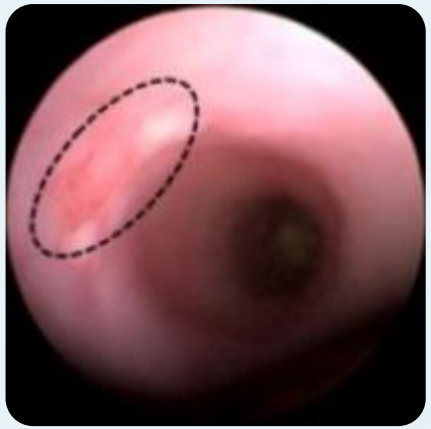

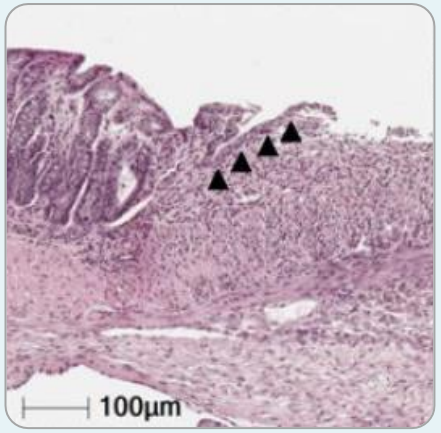

▲ Areas of pathological healing, incl. re-epithelialization and crypt reformation

Key Takeaway

GB004 leads to accelerated re-epithelialization and crypt reformation of disrupted intestinal epithelial tissue

Goggins et al, Am J Physiology 2021

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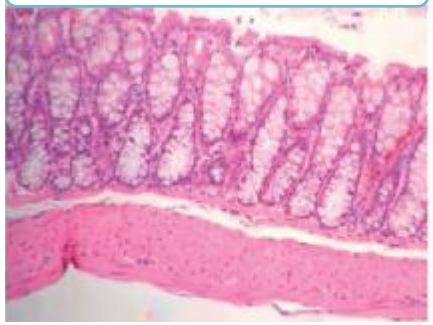
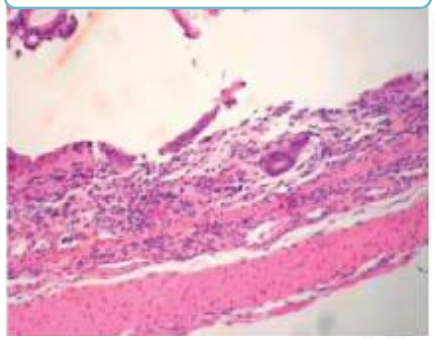
GB004 Leads to Epithelial Reconstitution and Disease Activity Improvement in TNBS Colitis Mouse Models

GB004

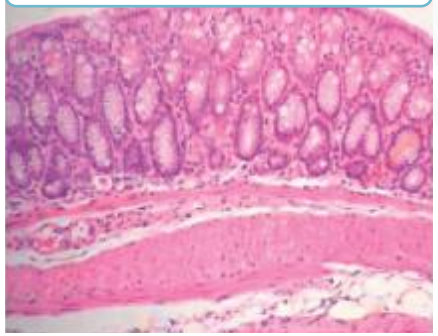
Naive



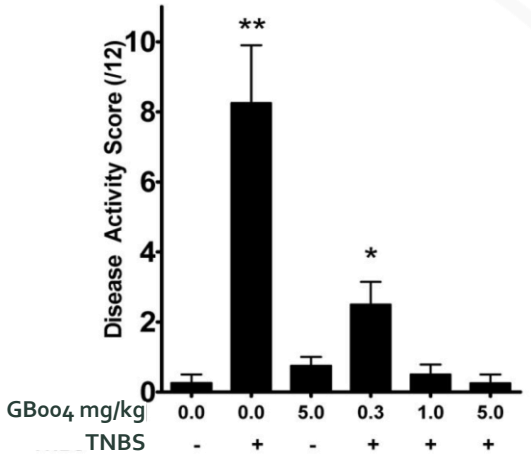
Colitis + Vehicle

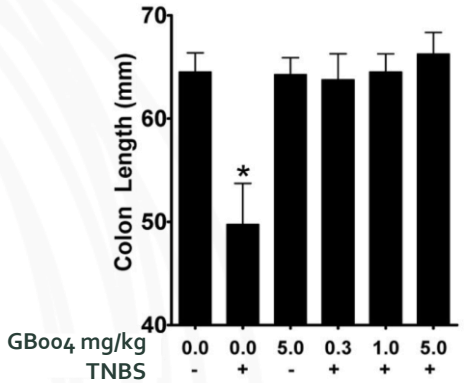


Colitis + GB004

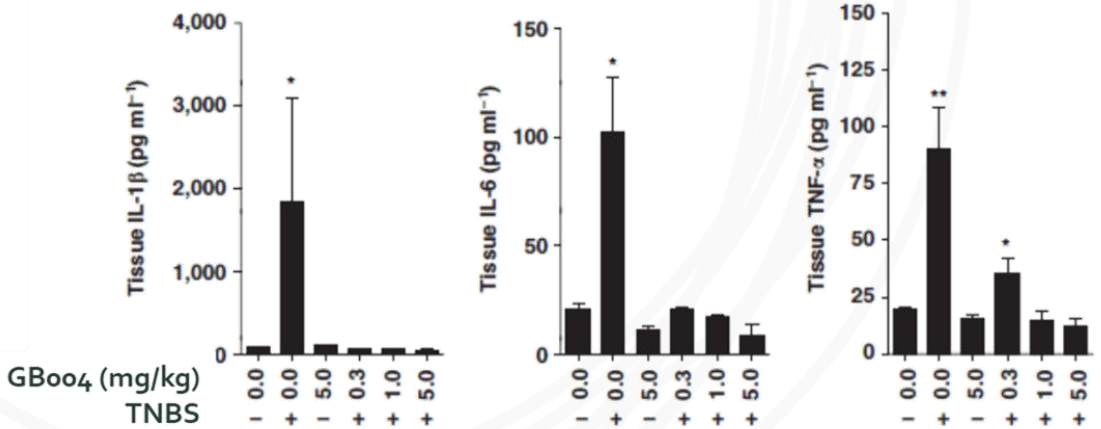


Disease Activity Score and Colon Length





Colon Inflammatory Cytokines



*p<0.05; **p<0.01

Keely et al, Mucosal Imm 2014

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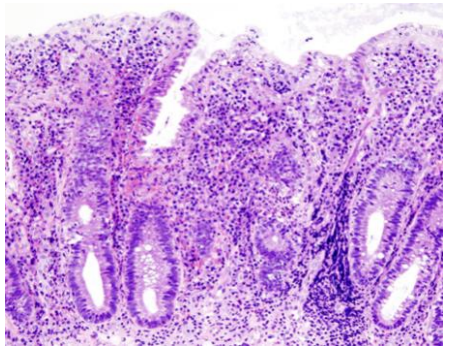
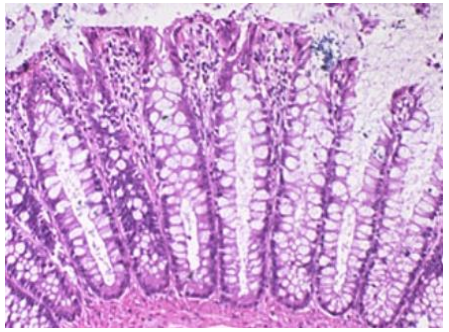
Histologic Healing is Predictive of Favorable Patient Outcomes

GB004

Severe Histologic Activity



Histologic Remission



Histological Remission and Healing in UC is Predictive of…

- Steroid free clinical remission
- Reduction in disease relapse
- Reduction in hospitalizations
- Reduction in corticosteroid use

→ Long-term clinical, endoscopic, & histological remission → More favorable disease course

Source:
Pai, Rish et. al. "The emerging role of histologic disease activity assessment in ulcerative colitis." Gastrointestinal Endoscopy 88, no. 6, 887 – 898.
Ungaro, Ryan et al. "A Treat-to-Target Update in Ulcerative Colitis: A Systematic Review." The American Journal of Gastroenterology 114 (2019) 874 – 883.

Phase 1b Utilized the Robarts Histopathology Index

GB004

- Entry criteria for GB004 Phase 1b required active histology as assessed by RHI
- RHI developed using 4 most reliable indices of Geboes Score
- Emphasizes presence of active neutrophils, the defining histologic hallmark of ulcerative colitis
- Scored 0-33
- Achievement of histological remission in the GB004 Phase 1b required:
  - Total score less than or equal to 3
  - Zero on both neutrophil scores

Calculation of Robarts Histopathology Index (RHI) (1)

Chronic inflammatory infiltrate (1 x score [0-3])	0 = No increase
	1 = Mild but unequivocal increase
	2 = Moderate increase
	3 = Marked increase
Lamina propria neutrophils (2 x score [0-3])	0 = No increase
	1 = Mild but unequivocal increase
	2 = Moderate increase
	3 = Marked increase
Neutrophils in epithelium (3 x score [0-3])	0 = None
	1 = <5% of crypts involved
	2 = <50% of crypts involved
	3 = >50% of crypts involved
Erosions or ulcerations (5 x score [0-3])	0 = No erosion, ulceration, or granulation tissue
	1 = Recovering epithelium + adjacent inflammation
	2 = Probable erosion – focally stripped
	3 = Ulcer or granulation tissue

Source:
Pai, Rish et. al. "The emerging role of histologic disease activity assessment in ulcerative colitis." Gastrointestinal Endoscopy 88, no. 6, 887 – 898.

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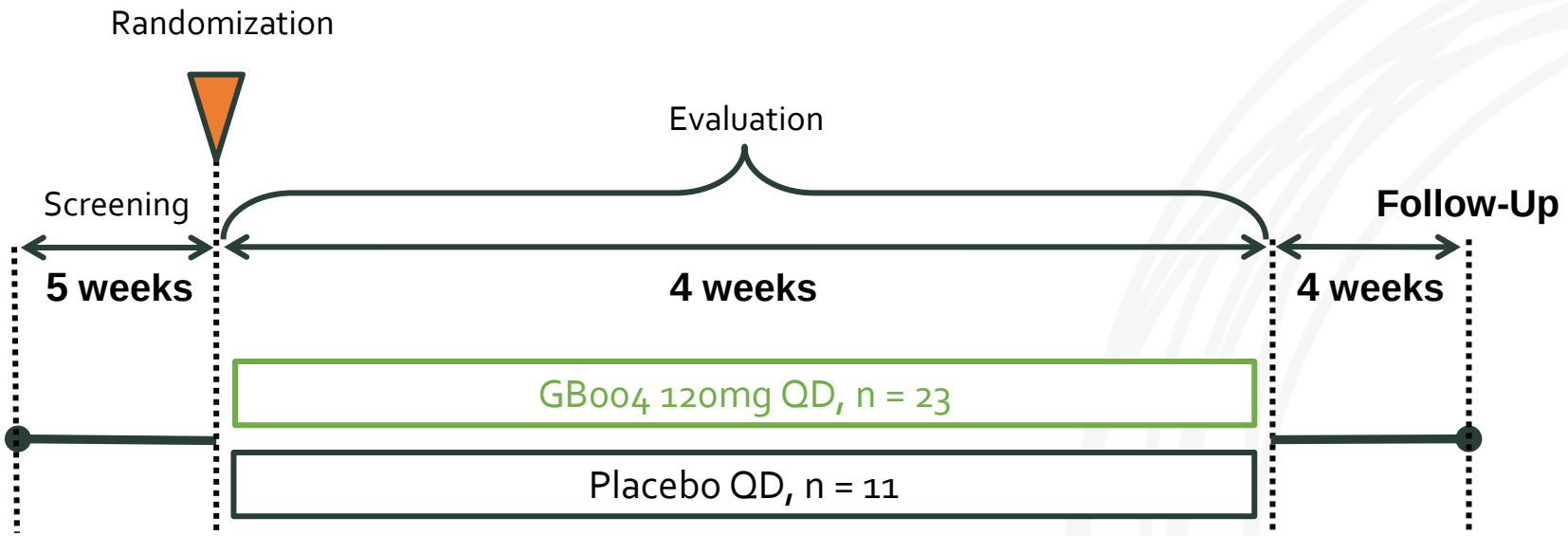
Completed GB004 Phase 1b Study Design

GB004

A Phase 1b, signal seeking, placebo-controlled study to evaluate the safety and pharmacokinetic profile of GB004 in adult patients with UC

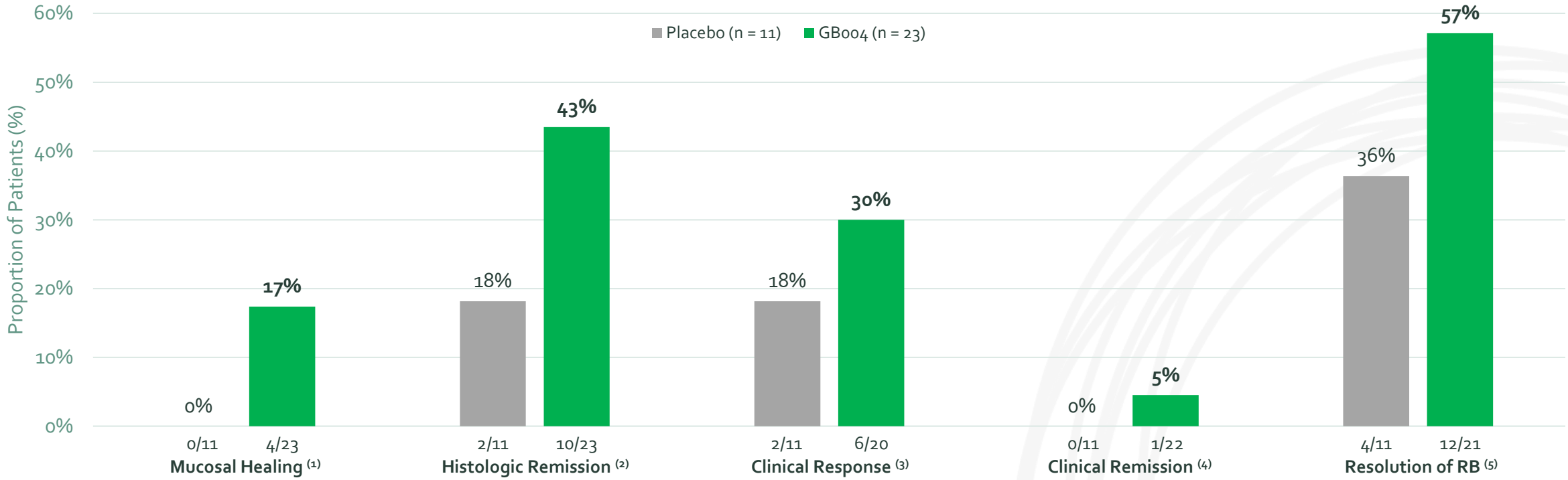


Patient Population	Adult patients with active (mild disease or greater) UC, with inadequate response or intolerance to 5-ASA or steroids and evidence of active inflammation by histology
Endpoints	**Primary:** Safety, tolerability **Secondary:** PK **Exploratory:** biomarker analysis, and histologic, endoscopic, and clinical indices to evaluate biological effect

QD: once daily dosing.

Phase 1b Results: Promising Trends in Exploratory Efficacy Outcomes Observed, Especially in Endpoints Related to Epithelial Lining Healing and Repair

GB004



Evidence of Clinical Activity with 120mg QD Dose

- **Mucosal healing** (histologic remission + endoscopic improvement) and **clinical remission** reported in GB004 arm; none reported in placebo arm
- Higher rates of **histologic remission, clinical response,** and improvement in **rectal bleeding** in GB004 arm compared to placebo

Full results presented at UEGW Virtual 2020

Histology, endoscopic improvement, and mucosal healing were evaluated individually in two segments of the large intestine: the sigmoid colon and rectum; RB = rectal bleeding

1) Mucosal healing: achievement of both histologic remission and endoscopic improvement in the same segment. Analysis of patients with mucosal healing in sigmoid or rectum.
2) Analysis of patients with histologic remission in sigmoid or rectum.
3) Three patients on the GB004 arm were unevaluable for clinical response (2 w/baseline rectal bleeding scores of 0, 1 w/baseline sigmoid endoscopic score of 0).
4) One patient on the GB004 arm was unevaluable for clinical remission (baseline sigmoid endoscopic score of 0).
5) Two patients on the GB004 arm were unevaluable for RB resolution (baseline RBS of 0).

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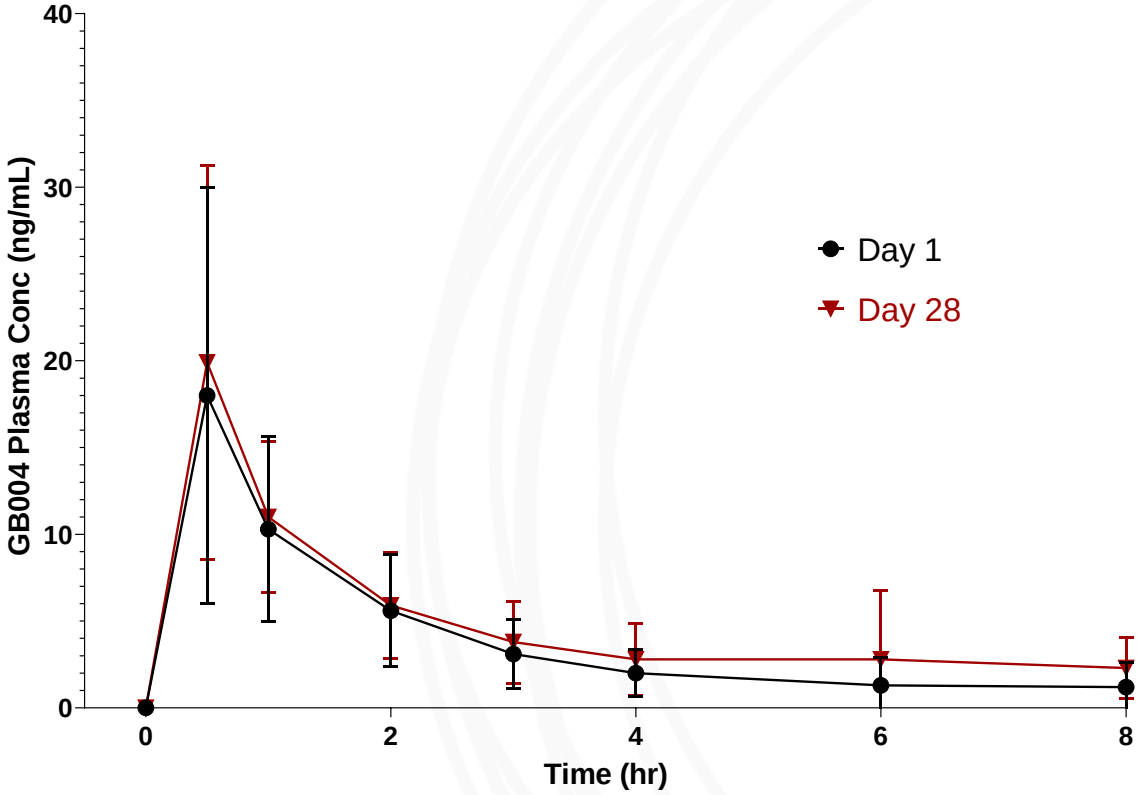
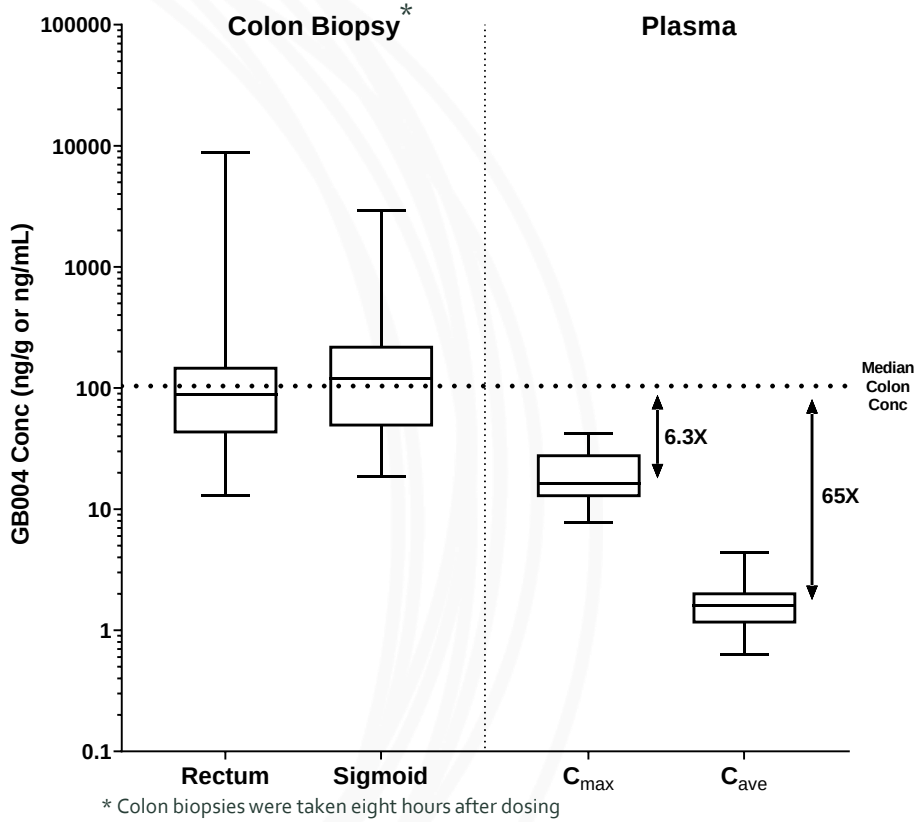
Phase 1b Results Consistent with Expectations: Gut-Targeted PK

GB004

Rapid Clearance and Minimal Accumulation Observed



Multi-fold Higher Concentrations Observed in the Gut



* Colon biopsies were taken eight hours after dosing

Gut-Targeted PK Profile

- Minimal to no accumulation of GB004 after 28 days QD dosing
- Colon tissue concentrations exceed plasma concentrations
  - ~6 times C_{max} and ~65 times $C_{average}$

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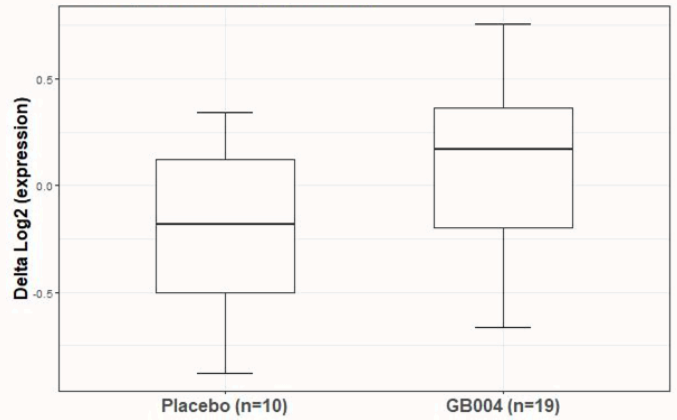
Increased Expression of Genes Associated with Barrier Restitution and Integrity Observed in GB004 Arm Compared to Placebo

GB004

Epithelial Restitution

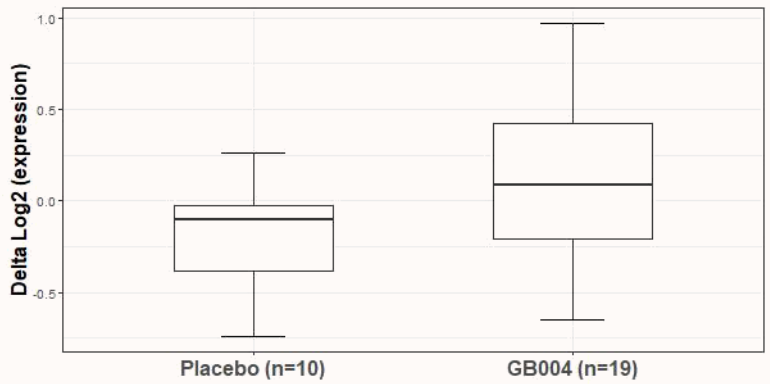
Integrin-α6 (ITGA6)



- Increase in ITGA6 consistent with increased barrier restitution and migration

Barrier Integrity

Tight Junction Protein-1 (TJP1)



- Increases in TJP1 consistent with restoring the integrity of the gut mucosal barrier

*Horizontal line represents the median, the top and bottom of the box represent first and third quartiles, respectively, and the whiskers a range of maximally 1.5 times the distance between the first and third quartile.
Internal data

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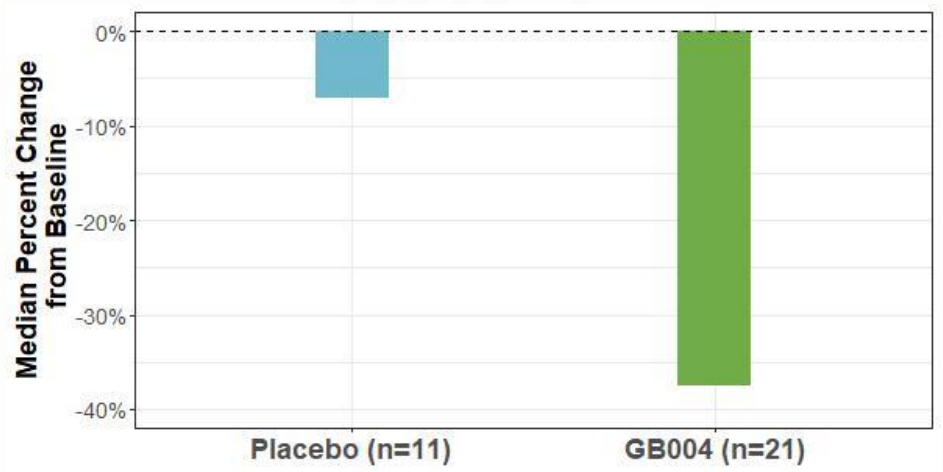
Stool Biomarkers in GB004 Group Indicate Decreased Inflammation and Improved Epithelial Integrity

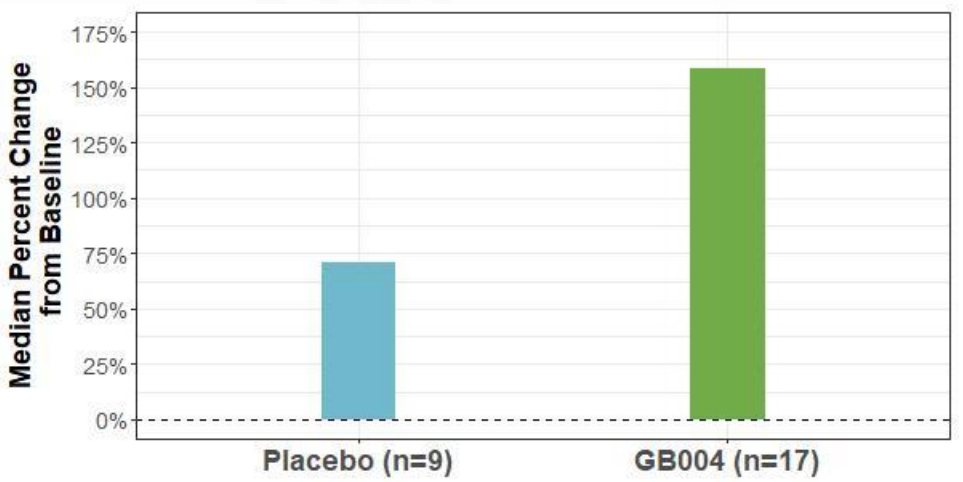
GB004

Stool

Decrease in Fecal Calprotectin



Increase in Secretory IgA (sIgA)



- Decrease in fecal calprotectin is consistent with decreased bowel inflammation
- Secretory IgA (sIgA) increase is consistent with improved gut epithelium and local immune defense

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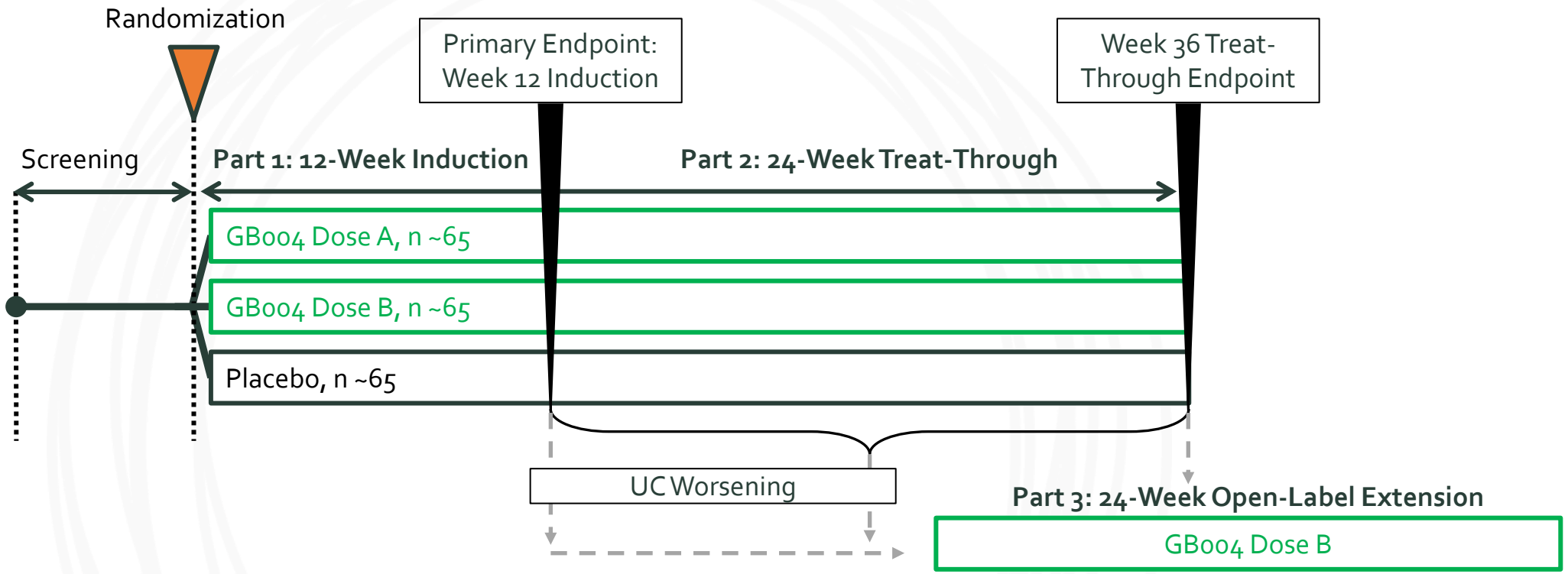
SHIFT-UC Phase 2 Study In Active UC: Measuring Short- and Long-Term Results of Therapy

GB004

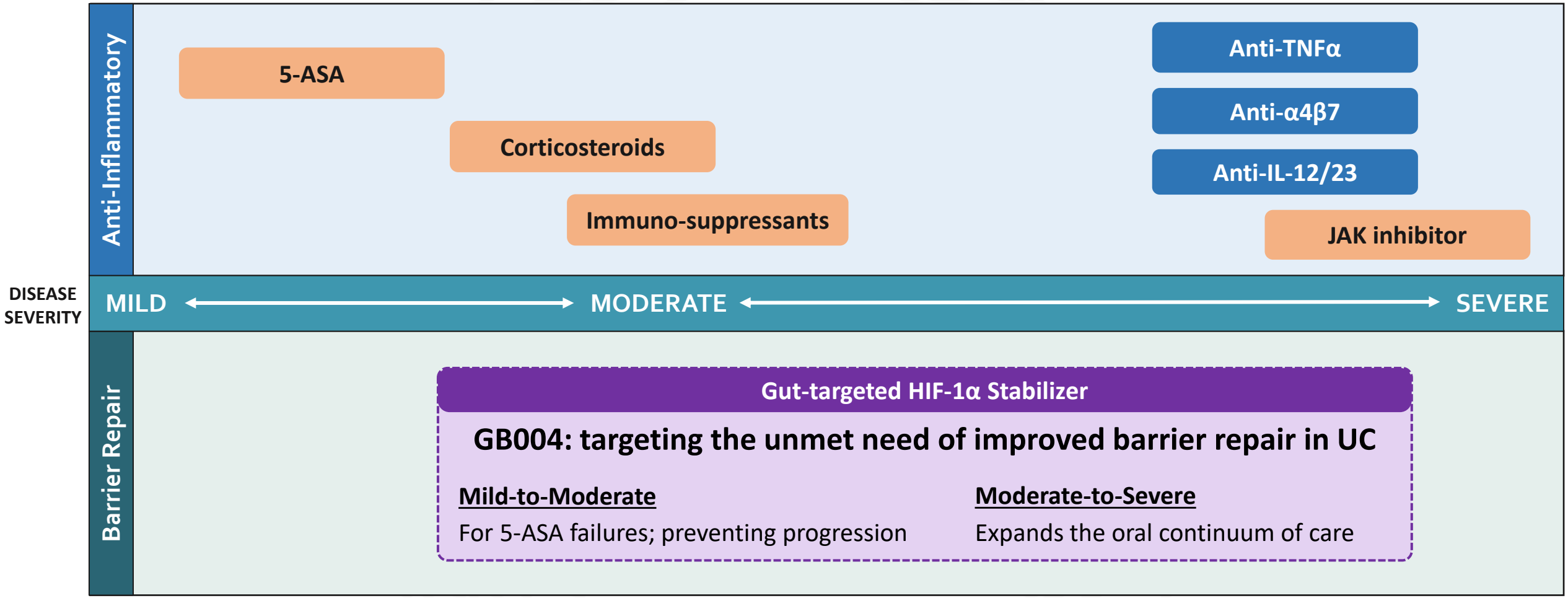


Patient Population:	• Adult UC patients with mild-to-moderate UC, Mayo 5-10, Endoscopic score (MES) ≥=2, RBS=≥1, Stool Freq=≥1
Objectives:	• Assess efficacy and safety of tablet formulation in UC • Demonstrate proof of concept for GB004 MoA
Endpoints:	• **Primary**: Clinical Remission at 12 weeks • **Secondary**: Clinical Response, Mucosal Healing, Endoscopic Improvement, Histologic Remission • **Other**: Safety and tolerability, Disease Clearance, serum, fecal, tissue biomarkers

MES = Mayo endoscopic score; RBS = rectal bleeding sub-score; MoA = mechanism of action.

Given Epithelial Barrier Disruption Characterizes UC at All Stages of Severity, GB004 Has the Potential to Address Unmet Needs Across the Severity Spectrum

GB004



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GB1275

CD11b Modulator

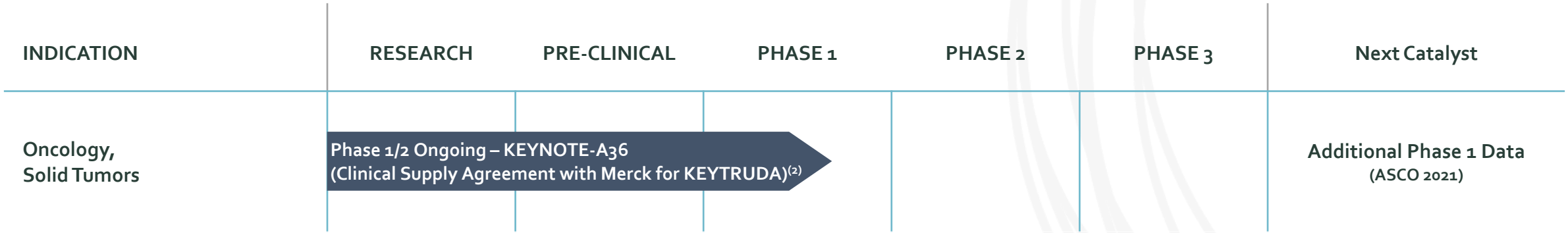
Solid Tumors

GB1275: Opportunity to Improve Response Rates in Difficult-to-Treat Tumors Through Targeting Immunosuppressive Myeloid Cells

GB1275

Product Candidate Description

- Oral, small molecule, first-in-clinic CD11b modulator in development for the treatment of solid tumors
- Designed to disrupt multiple immunosuppressive myeloid cell subsets, including MDSCs and TAMs
- PC activity observed as single agent and synergistically in combo with chemo and IO therapies
- Clinical trial collaboration and supply agreement with Merck & Co. to study GB1275 in combination with KEYTRUDA (pembrolizumab) in the ongoing Phase 1/2 study for selected solid tumors
- Currently targeting immuno-oncology resistant tumors incl., PDAC, CRC, TNBC, CRPC and others
- Patent protection to 2036(1); Orphan Drug Designation from FDA and EMA for pancreatic cancer



PC = preclinical; chemo = chemotherapy; IO = immuno-oncology; PDAC = pancreatic ductal adenocarcinoma; CRC = colorectal cancer; TNBC = triple negative breast cancer; CRPC = castrate-resistant prostate cancer; MDSC = myeloid-derived suppressor cells; TAM = tumor-associated macrophage.

1) Does not include available patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims. Total patent life with patent term extension cannot exceed 14 years from approval.
2) Gossamer Bio maintains full worldwide rights to GB1275.

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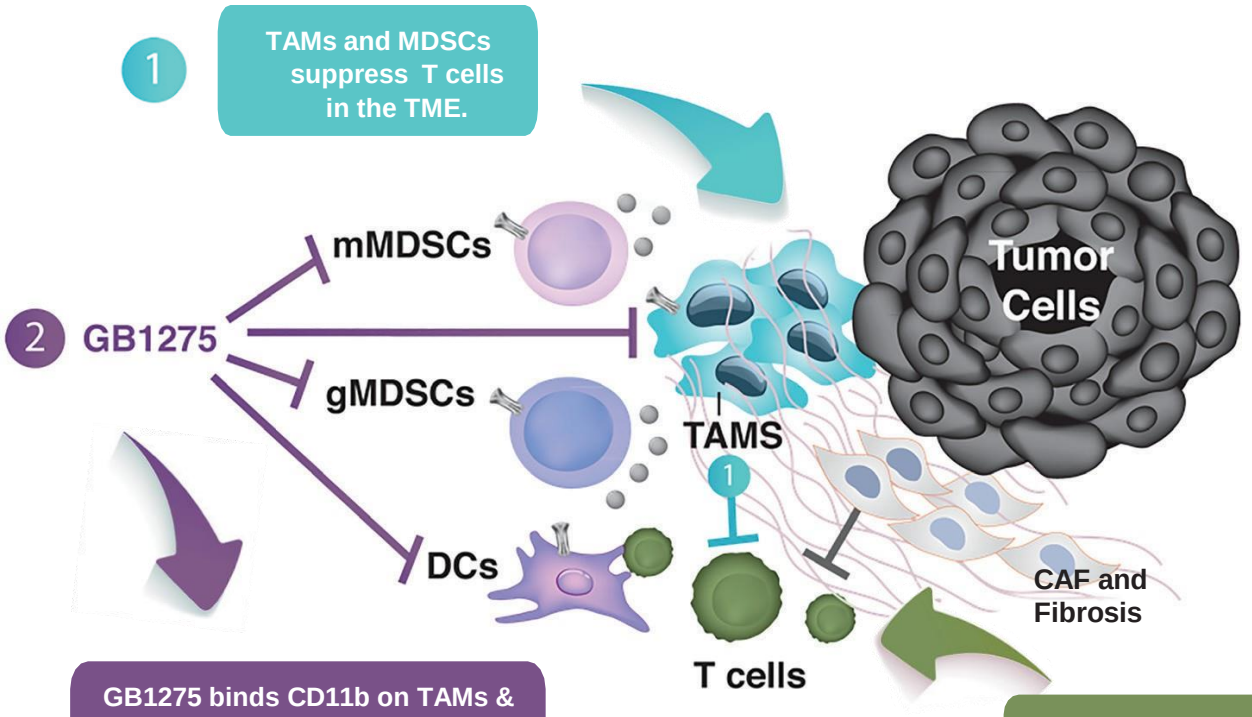
MDSCs and M2 Macrophages Generate an Immunosuppressive Tumor Microenvironment and Hinder Response to IO Agents



GB1275 is an Allosteric Modulator of CD11b Designed to Impact Myeloid Cell Migration and Polarization

- MDSC- and TAM-mediated immunosuppression of T cells in the tumor microenvironment is associated with worse clinical outcome
- **GB1275** is an allosteric modulator of CD11b
- In pre-clinical studies, **GB1275** has been observed to:
  - Repolarize myeloid suppressive cells (mMDSCs & gMDSCs) and TAMs, reducing their immunosuppressive roles and allowing T cells to attack tumors
  - Reduce tumor influx of CD11b+ MDSCs

DC = dendritic cells; MDSC = myeloid-derived suppressor cells (m = monocytic, g = granulocytic); TAM = tumor-associated macrophage; CAF = cancer associated fibroblasts; TME = tumor microenvironment.

GB1275 Has the Potential to Inhibit Multiple Immunosuppressive Myeloid Cell Types

GB1275

Example Product Candidate	Target	TAM polarization	Monocytic (m) MDSC	Granulocytic (g) MDSC	T-reg
Gossamer GB1275 (Phase 1/2)	**CD11b**[1][2]	✓	✓	✓	
BMS-813160 (Phase 2)	**CCR2**[3][4] **/ CCR5**[5]	✓	✓		✓
Pfizer PF-04136309 (Phase 2)	**CCR2**[3][4]	✓	✓		
AstraZeneca AZD5069 (Phase 2)	**CXCR2 Inhibitor**[3]			✓	
Five Prime/BMS Cabiralizumab (Phase 2)	**CSF1R**[6]	✓			

- GB1275 is designed to block gMDSC and mMDSC cell recruitment to the tumor site and blocks M2 polarization
- CCR2 inhibitors may be limited by compensatory recruitment of gMDSCs
- In pre-clinical studies, GB1275 MDSC/TAM modulation effects were tumor-localized, without impact on myeloid cells in the periphery, potentially avoiding concerns for dose-limiting neutropenia as observed with other myeloid cell therapies

1) Panni, Roheena et al. "Agonism of CD11b reprograms innate immunity to sensitize pancreatic cancer to immunotherapies" Science Translational Medicine 11 (2019).
2) Schmid, Michael et al. "Integrin CD11b activation drives anti-tumor innate immunity." Nature Communications 9, no. 10 (2018): 1516 – 1523.
3) Nywening, Timothy et al. "Targeting both tumour-associated CXCR2+ neutrophils and CCR2+ macrophages disrupts myeloid recruitment and improves chemotherapeutic responses in pancreatic ductal adenocarcinoma." Gut 67, no. 6 (2018): 1112-1123.
4) Deci, Michael et al. "Modulating macrophage polarization through CCR2 inhibition and multivalent engagement." Molecular Pharmaceutics 15, no. 7 (2018): 2721-2731.
5) Tan, Marcus et al. "Disruption of CCR5-dependent homing of regulatory T cells inhibits tumor growth in a murine model of pancreatic cancer." Journal of Immunology 182, no. 3 (2009): 1746-1755.
6) Cannarile, Michael et al. "Colony-stimulating factor 1 receptor (CSF1R) inhibitors in cancer therapy." Journal of ImmunoTherapy of Cancer (2017).

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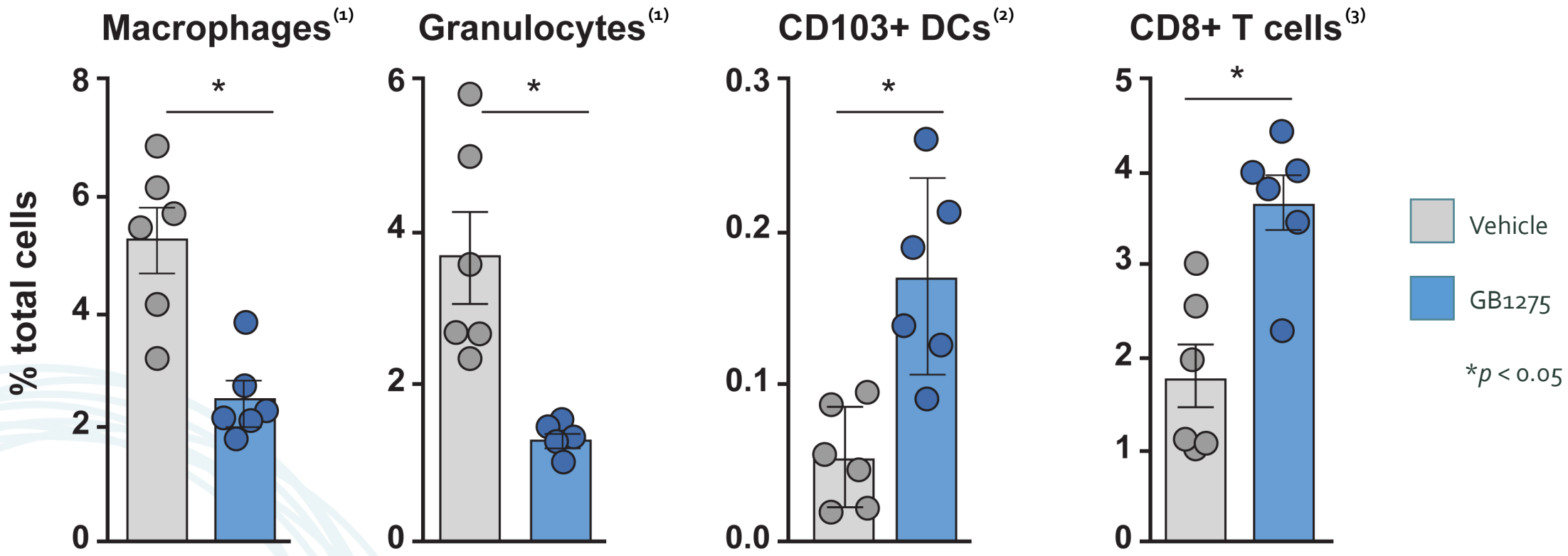
GB1275 Reduced Tumor Infiltration of Myeloid Cells and Increased Influx of Activated CD8+ T cells in Pre-Clinical Studies



- GB1275 immuno-modulatory effects were tumor-localized
- GB1275 did not alter immune cell phenotypes in the periphery, spleen or bone marrow

1) Frequencies of tumor-infiltrating granulocytes and macrophages in orthotopic KP2 PDAC models 10 days after treatment with GB1275 or vehicle.
2) Frequencies of CD103+ DCs in orthotopic KP2 PDAC tissues from mice treated for 12 days with GB1275 or vehicle.
3) Frequencies of tumor-infiltrating CD8a+ CTLs in orthotopic KP2 PDAC tissues from mice treated 10–12 days with GB1275 or vehicle.
Source: Panni, Roheena et al. "Agonism of CD11b reprograms innate immunity to sensitize pancreatic cancer to immunotherapies" Science Translational Medicine 11 (2019).

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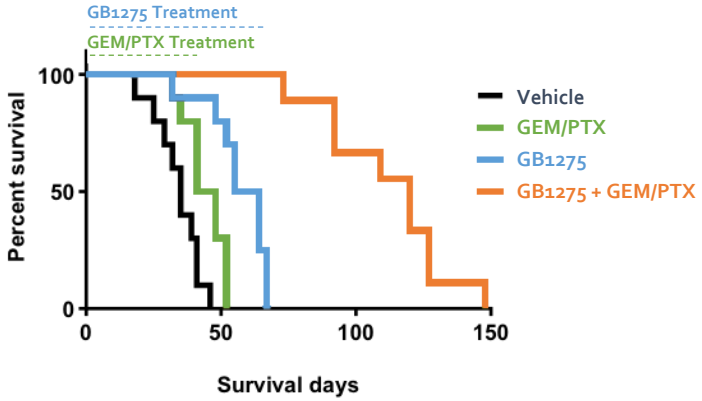
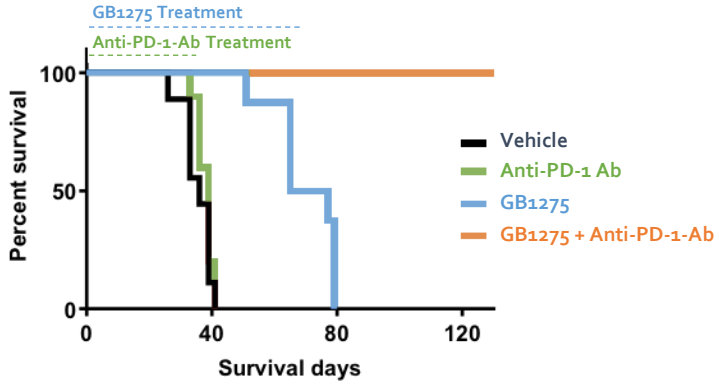
GB1275 Single Agent or in Combination Improved Tumor Responses and Survival Outcomes in Multiple Difficult-to-Treat Tumor Models

GB1275 Single Agent or in Combo with SoC or anti-PD-1 Ab Treatment Improved Tumor Response and Survival Outcomes in the PDAC Mouse Tumor Models[1]

GB1275 in Combination with Chemotherapy Extended Survival in the PDAC Model

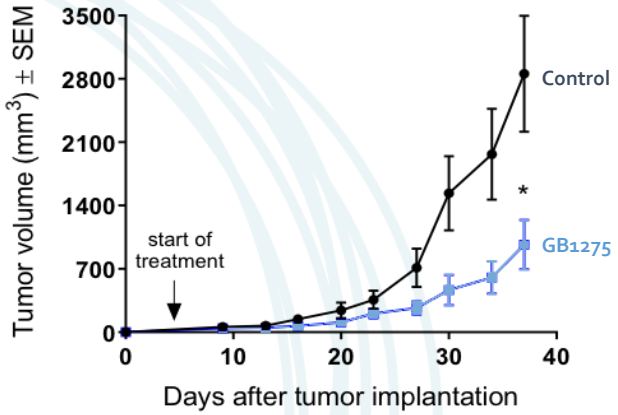


GB1275 Sensitized PDAC Model to anti-PD-1 Ab Checkpoint Blockade

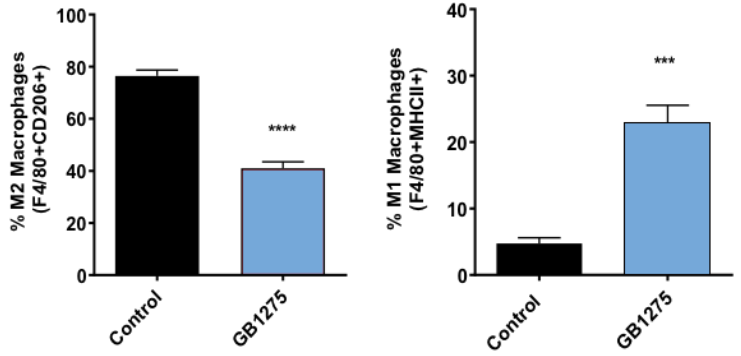


GB1275 Modulated Breast Cancer Tumor Microenvironment via Repolarization of Tumor Associated Macrophages[2]

GB1275 Reduced Tumor Volumes in the Orthotopic CL66 Breast Tumor Model



GB1275 Repolarized TAMs in the CL66 Breast Tumor Model



GEM/PTX = gemcitabine / paclitaxel; PDAC = Pancreatic Ductal Adenocarcinoma Model, Ab = antibody, aPD-1 = anti-PD-1, SEM = standard error of mean; SoC = standard of care.

1) Panni, Roheena et al. "Agonism of CD11b reprograms innate immunity to sensitize pancreatic cancer to immunotherapies" Science Translational Medicine 11 (2019).
2) Schmid, Michael et al. "Integrin CD11b activation drives anti-tumor innate immunity." Nature Communications 9, no. 10 (2018): 1516 – 1523.

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KEYNOTE-A36: Ongoing Phase 1/2 Study of GB1275 In Select Solid Tumor Indications

GB1275

A Phase 1/2, dose-ranging, signal seeking, clinical trial of GB1275 in adult subjects with various types of solid tumors, including PDAC, gastric, esophageal, prostate, TNBC and CRC

Phase 1

3 Dose Escalations:
- Monotherapy
- Pembrolizumab combo
- Gem / Abraxane combo

Dose Expansion at RP2D

Phase 2

3 Planned Expansion Cohorts:
- 1L mPanc (+ chemo)
- 2-4L MSS CRC (+ anti-PD-1)
- 3-4L PDL1+ Gastric (+ anti-PD-1)

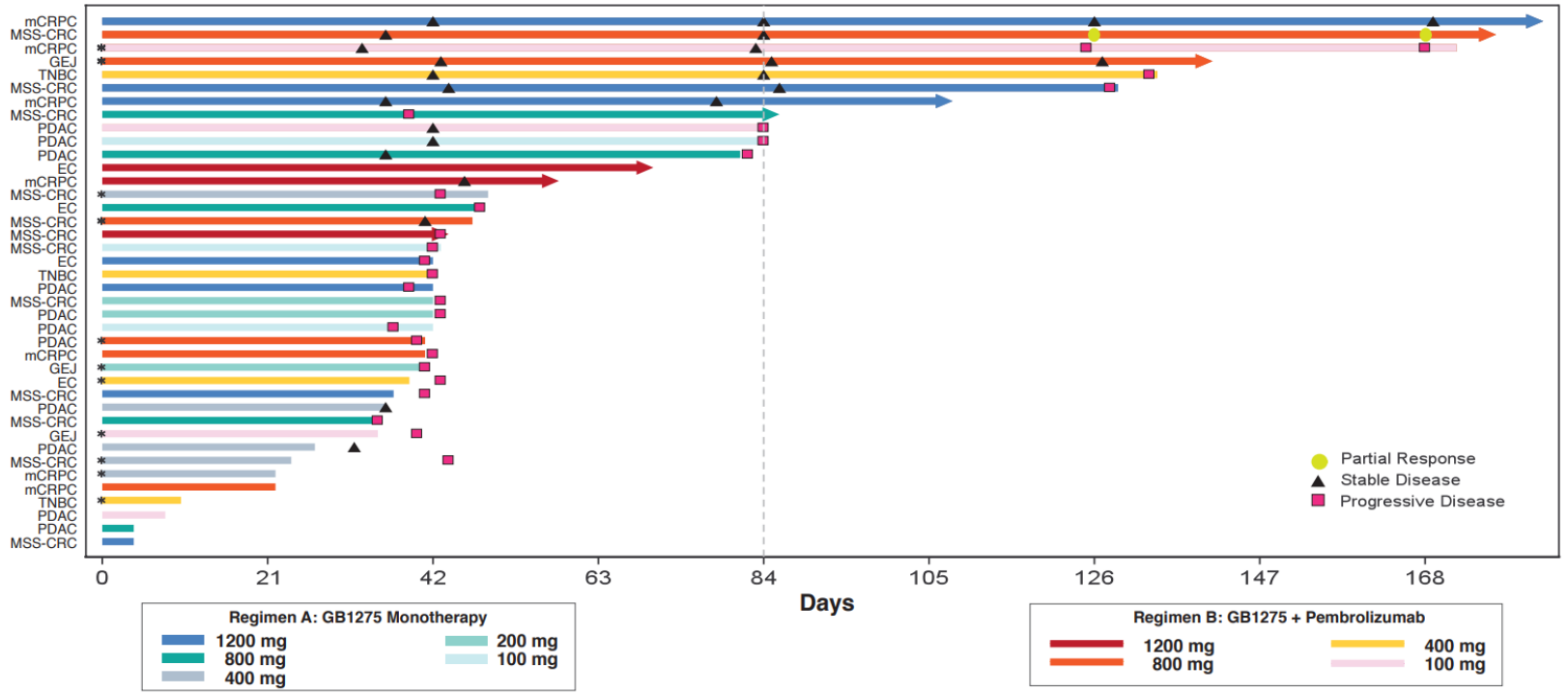
Endpoints	**Primary and Secondary (Phase 1):** Safety, tolerability, PK, PD **Primary (Phase 2):** Objective Response Rate **Secondary (Phase 2):** Duration of Response, Time to Response, Clinical Benefit Rate, Progression Free Survival, Time to Progression, Overall Survival, Safety, PK

PDAC = pancreatic ductal adenocarcinoma; TNBC = triple negative breast cancer; CRC = colorectal cancer; MSS = micro-satellite stable; Gem = gemcitabine; mPanc = metastatic pancreatic cancer; Gastric = gastric cancer; RP2D = recommended Phase 2 dose.

Preliminary Phase 1/2 Data Presented at SITC 2020

- Encouraging clinical and biological activity in dose escalation, especially at doses ≥ 800mg BID
  - 1 partial response observed in patient with MSS CRC
  - 7 cases of prolonged stable disease greater than 84 days (5 of at doses ≥ 800mg BID)
- Biomarker data suggests that GB1275, alone or in combination with pembrolizumab, may modulate myeloid cell biology in the TME, inducing a more inflamed tumor phenotype
- Data to date shows GB1275 alone or in combination with pembrolizumab (up to 1200mg BID) is well tolerated

Encouraging Clinical Activity Observed, Especially at Higher Doses



GC, Gastric cancer; GEJ, gastroesophageal junction; MSS-CRC, microsatellite stable-colorectal cancer; TNBC, triple negative breast cancer; mCRPC, metastatic castrate-resistant prostate cancer; PDAC, adenocarcinoma of the pancreas; EC: esophageal cancer

*Indicates prior CPI treatment; Data as of 14 October 2020.

Recommended Phase 2 dose of GB1275 in combination with pembrolizumab will be evaluated in an expansion cohort of up to 40 patients with selected tumor types

TME = tumor microenvironment; BID = twice daily dosing.

Corporate Overview and Milestones

Financial Overview

Cash, Cash Equivalents and Marketable Securities (As of 3/31/2021)	~$453mm
Debt, *Related to Line of Credit* (As of 3/31/2021; initial tranche of $150 million debt facility, announced 5/2/2019)	~$30mm
Additional Debt Capacity, *Related to Line of Credit* (As of 3/31/2021; remaining capacity of $150 million debt facility, announced 5/2/2019)[1]	~$120mm
Principal of Convertible Notes Outstanding (As of 3/31/2021)	~$200mm
Common Shares Outstanding (As of 5/3/21)	~75.9mm

mm = millions.
1) Accessible subject to the achievement of certain clinical development milestones and other customary conditions.

Upcoming Milestones for Clinical Programs

Indication	Milestone	Timing
Seralutinib (Pulmonary Arterial Hypertension)		
PAH	Phase 2 TORREY Study Topline Results	1H 2022*
GB004 (Inflammatory Bowel Disease)		
UC	Phase 2 S**HIF**T-UC Study Topline Results	1H 2022*
GB1275 (Oncology, Solid Tumors)		
Solid Tumors	Additional Phase 1 Data	ASCO 2021
Internal Research Programs		
Autoimmune, Oncology, Inflammation	Initiate Clinical Trial for One Additional Product Candidate	Within 12 months

*Subject to further developments in the ongoing COVID-19 pandemic.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