Corporate Presentation

June 2020

Forward Looking Statement

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective products, product approvals, research and development costs, timing and likelihood of success, plans and objectives of management for future operations, and future results of current and anticipated products, are forward-looking statements.

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, with 4 assets in clinical development and multiple preclinical programs underway
Diversified Pipeline	• 4 clinical-stage product candidates and multiple data readouts anticipated before year-end
World-Class Talent	• Deeply experienced leadership team with proven track record of developing innovative clinical assets

Robust Pipeline with Four Clinical-Stage Product Candidates

PROGRAM	CLASS (Route of Admin.)	INDICATION	RESEARCH / PRE-CLINICAL / PHASE 1 / PHASE 2 / PHASE 3	RIGHTS
GB001	DP2 Antagonist (Oral)	Moderate-to-Severe Eosinophilic Asthma	Phase 2b Enrollment Complete – IA Complete – LEDA Study	Worldwide (except Japan)
GB001	DP2 Antagonist (Oral)	Chronic Rhinosinusitis (with and without nasal polyps)	Phase 2 Enrollment Complete – TITAN Study	Worldwide (except Japan)
GB002	PDGFR Inhibitor (Inhaled)	Pulmonary Arterial Hypertension	Phase 1b Ongoing Phase 2 Planned	Worldwide
GB004	HIF-1α Stabilizer (Oral)	Inflammatory Bowel Disease	Phase 1b Complete Phase 2 Planned	Worldwide
GB1275	CD11b Modulator (Oral)	Oncology, Solid Tumors	Phase 1/2 Ongoing	Worldwide

IA = Interim Analysis.

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GB001

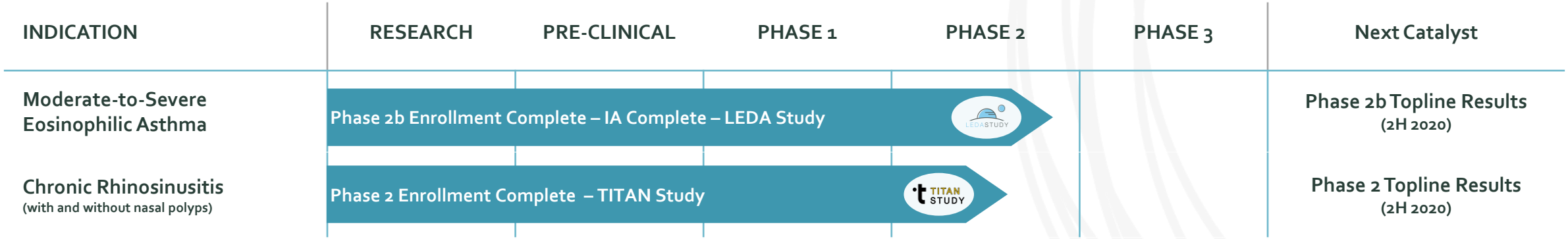
DP2 Antagonist

Asthma and Other Allergic Conditions, including Chronic Rhinosinusitis (CRS)

GB001: Oral Product Candidate with Potential to Disrupt Treatment Paradigms in Allergic and Inflammatory Diseases

Product Candidate Description

- Oral, once-a-day, DP2 antagonist currently in development for the treatment of moderate-to-severe eosinophilic asthma and chronic rhinosinusitis (CRS)
- Target validation from Teijin's GB001 Phase 2 study in Japanese asthmatics
- Anti-inflammatory effect comparable to certain biologics; potential to be used earlier in treatment
- Over 400 patients received at least 1 dose of GB001 with no clinically significant safety findings(1)
- Phase 2b LEDA interim analysis completed in Q2 2020; based on results of interim and IDMC recommendation, Gossamer will complete LEDA; initial Phase 3 planning and supportive activities commenced; final decision to move into Phase 3 dependent on final analysis of full LEDA results
- Composition-of-matter protection to 2031(2); lysine salt composition protection to 2037



CRS = Chronic Rhinosinusitis, IA = Interim Analysis, IDMC = Independent Data Monitoring Committee.

1) As of December 31st, 2018 in completed clinical studies.
2) 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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Eosinophils Play a Critical Role in Type 2 Asthma

GB001

We Estimate that Approximately 50% of Severe Asthmatics in the United States have Elevated Eosinophils

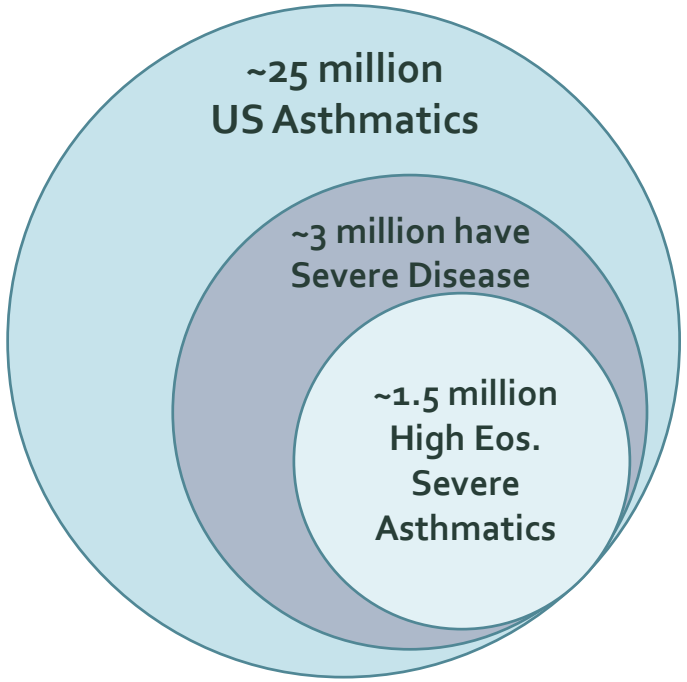
Eosinophils are Key Cells Mediating Inflammation in Asthma

- Asthma is typically characterized by airway inflammation and airway hyperresponsiveness
- The DP2 receptor is expressed by the main cells involved in Type 2 asthma inflammation (eosinophils, basophils, ILC2, and Th2 cells)
- The resulting eosinophilic inflammation contributes to the presence and persistence of asthma symptoms

Symptoms

- Asthma exacerbation ("asthma attack")
- Blocked airways
- Coughing
- Tightness in the chest
- Shortness of breath or hard time breathing
- Wheezing

Asthmatics with Elevated Eosinophils are Still Underserved



CDC, Datamonitor, Literature.

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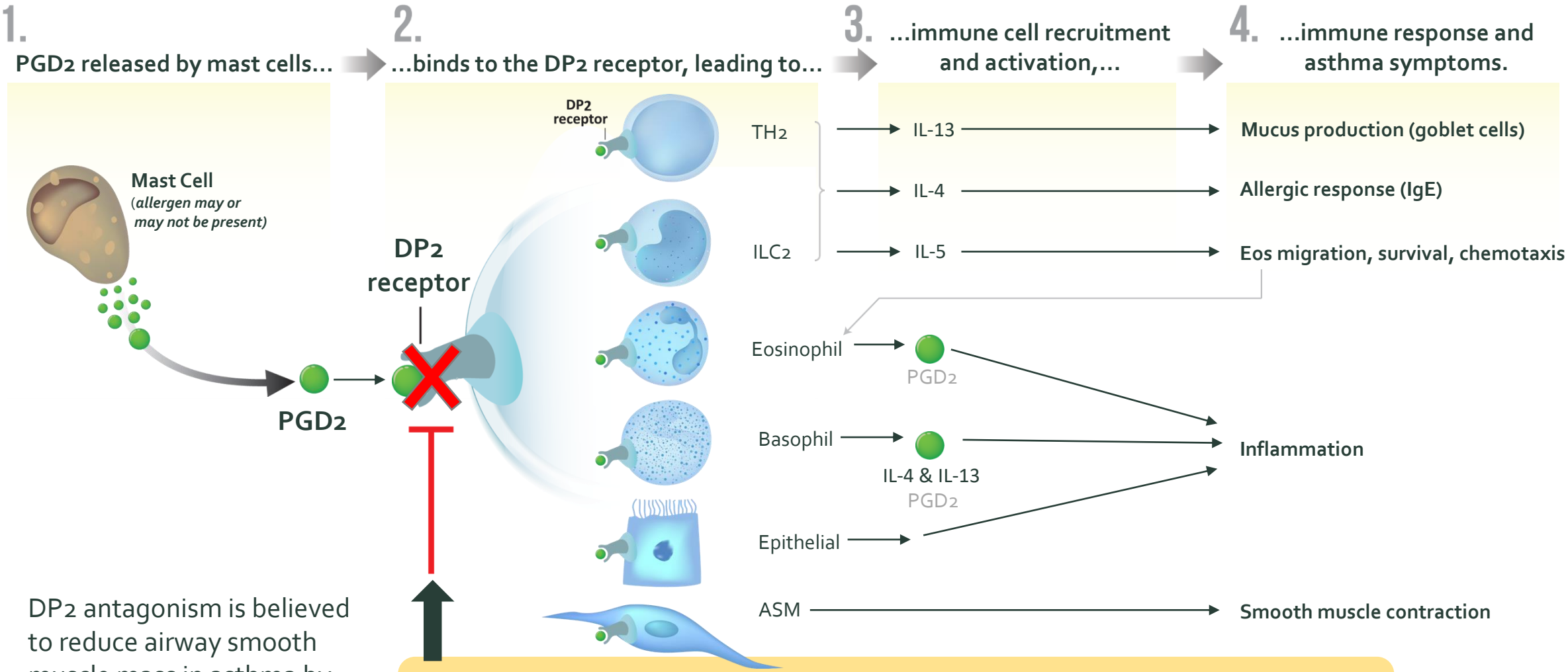
Role and Biology of the PGD2/DP2 Pathway in Type 2 Inflammation

GB001



DP2 antagonism is believed to reduce airway smooth muscle mass in asthma by decreasing eosinophilia and myofibroblast recruitment

GB001, an oral DP2 antagonist, is designed to block the interaction of PGD2 with DP2, preventing the resulting a pro-inflammatory cascade

Sources: Domingo, Respiratory Research 2018; Singh, Clinical Pharmacology: Advances and Applications 2017; Farne, Expert Opinion on Emerging Drugs 2016; Stone, J Allergy Clin Immunol 2010; Saunders, Sci Transl. Med. 2019.

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GB001 Demonstrated a Reduction in FeNO in Post-Hoc Analysis of Phase 2 Study in Mild-to-Moderate Asthma

GB001

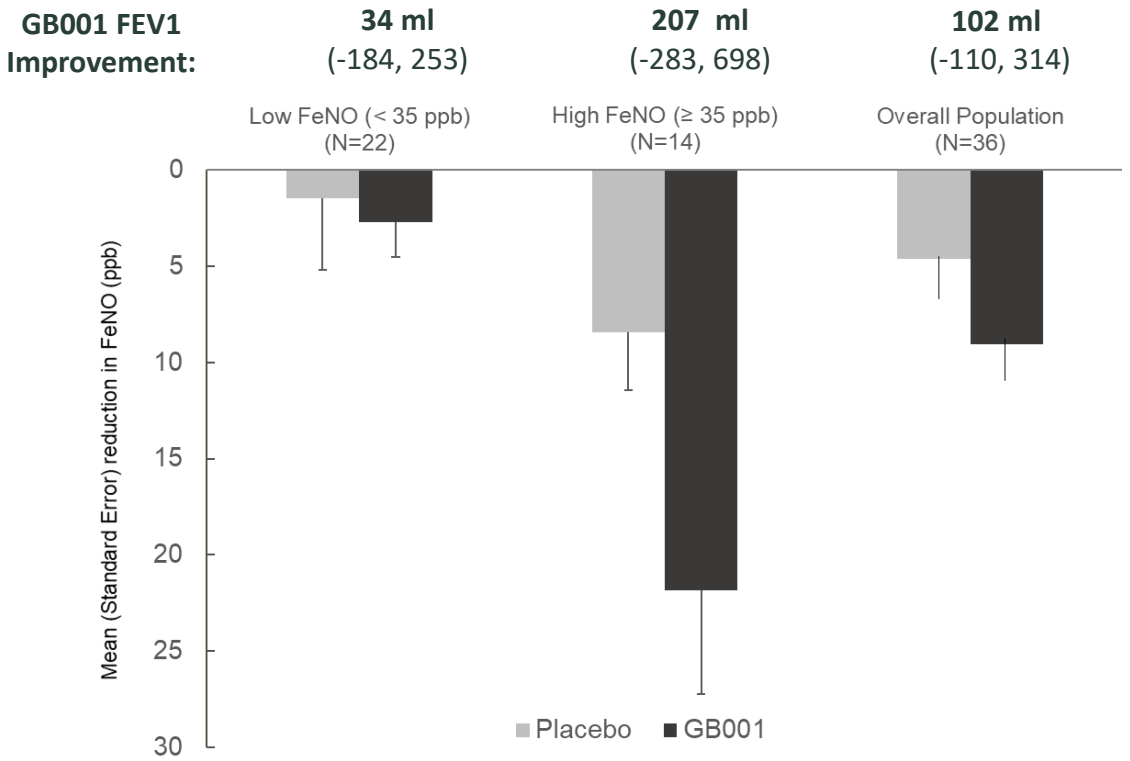
Reduction of Exhaled Nitric Oxide by the DP2 antagonist GB001

Results from a post-hoc analysis evaluating Fractional exhaled Nitric Oxide (FeNO) as marker and outcome following administration of GB001 or placebo over 28 days

Findings

- In a retrospective analysis, GB001 resulted in rapid and greater improvements in lung function relative to placebo in subjects with high FeNO and blood eosinophils(1)
- Marked difference in the magnitude of FeNO reduction and the treatment effect of GB001 relative to placebo in subjects with high (≥35 ppb) versus low (<35 ppb) baseline FeNO
- The FeNO changes from baseline are comparable to the changes observed with Dupilumab in Phase 2 and 3 studies(2)(3)
- FeNO in addition to blood eosinophils may be a useful marker for treatment response to GB001

Mean Reduction in FENO and FEV1 at Day 28



A Phase 2 Study to Evaluate the Safety, Efficacy and Pharmacokinetics of DP2 Antagonist GB001 and to Explore Biomarkers of Airway Inflammation in Mild-to-Moderate Asthma.(1)

1) Ortega H, et al. "A Phase 2 Study to Evaluate the Safety, Efficacy and Pharmacokinetics of DP2 Antagonist GB001 and to Explore Biomarkers of Airway Inflammation in Mild-to-Moderate Asthma." Clinical & Experimental Allergy 50, no. 2 (2019): 189 – 197.
2) Wenzel, Sally et al. "Dupilumab in Persistent Asthma with Elevated Eosinophil Levels." NEJM 368 (2013): 2455 – 2466.
3) Castro, Mario et al. "Dupilumab Efficacy and Safety in Moderate-to-Severe Uncontrolled Asthma." NEJM 378 (2018): 2486 – 2496.

FeNO = fractional exhaled nitric oxide; ppb = parts per billion.

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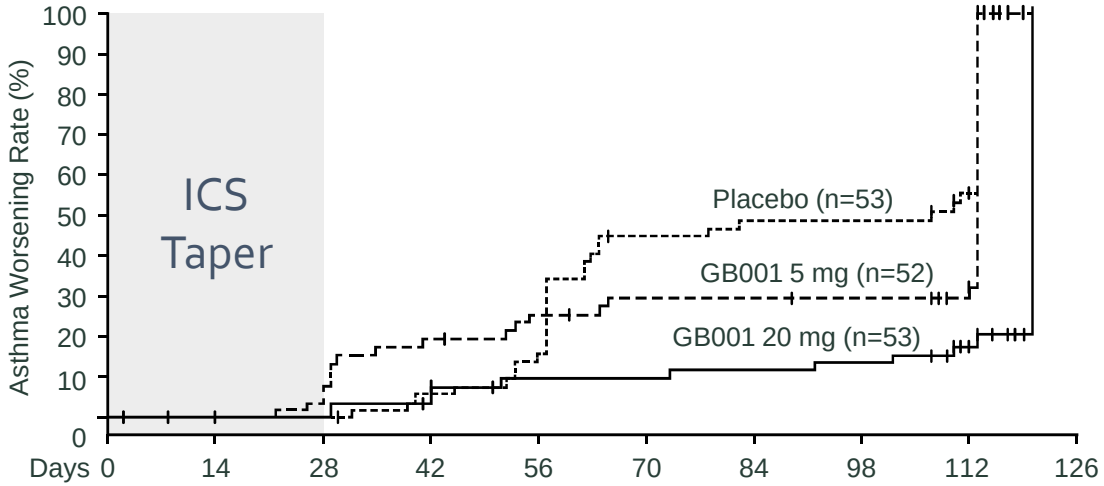
GB001 Demonstrated Statistically Significant Improvements in Time-to-First Asthma Worsening in Japanese Phase 2 Study

Both doses of GB001 met the primary endpoint of change in morning peak expiratory flow with statistical significance vs placebo

Overall Population

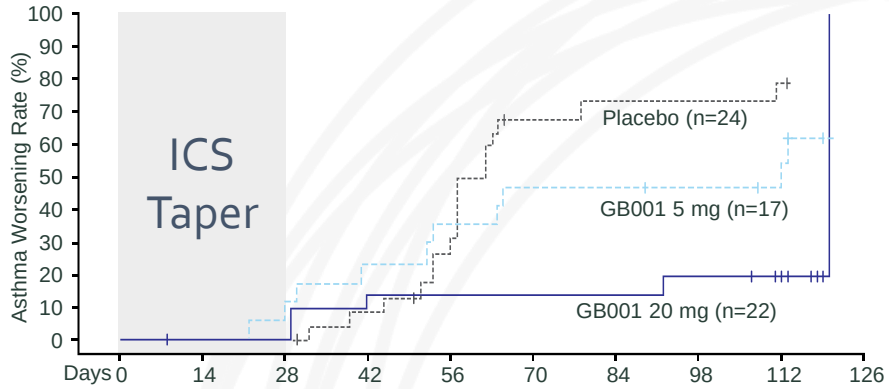
71% reduction in the risk of asthma worsening for 20mg GB001 group



	pbo vs 5 mg	pbo vs 20 mg
p-value (log-rank test)	0.088	**P<0.001**
Hazard Ratio (95% CI)*	0.59 (0.32, 1.07)	0.29 (0.14, 0.58)

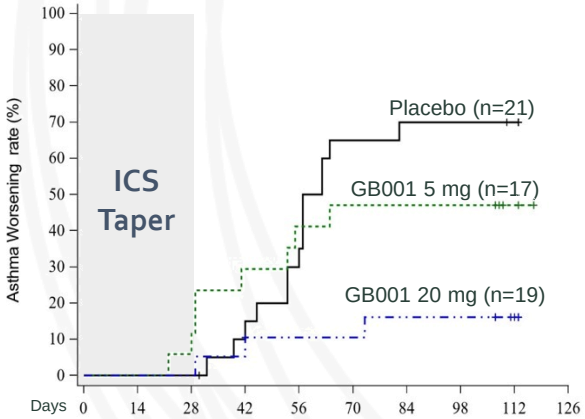
High Eosinophil Population (≥300 µL)

84% reduction in the risk of asthma worsening for 20mg GB001 group



High FeNO Population (≥25 ppb)

84% reduction in the risk of asthma worsening for 20mg GB001 group



pbo = placebo.
*Cox Regression.
Definition of asthma worsening:

1. For 2 or more consecutive days, AM PEF (morning peak expiratory flow) ≤ 0.75 x mean level of AM PEF for the last 7 days of Run-in Period
2. FEV1 (forced expiratory volume in one second) ≤ 0.8 x at the randomization time point
3. For 2 or more consecutive days, using SABA (short-acting beta agonist) at a dose of 5 puffs/day
4. Asthma Control Questionnaire (ACQ) ≥ ACQ at the randomization time point + 0.5
5. Having had asthma exacerbation requiring administration of oral corticosteroids or step 2 or higher treatments of Japan Guidelines 2012 steps of asthma attacks

Source: Asano K, et al. Phase 2 study results of DP2-antagonist GB001 on asthma worsening and other asthma control markers. Presented at: (ACAAI) Scientific Meeting 2019; 2019 Nov 7 - 11; Houston.

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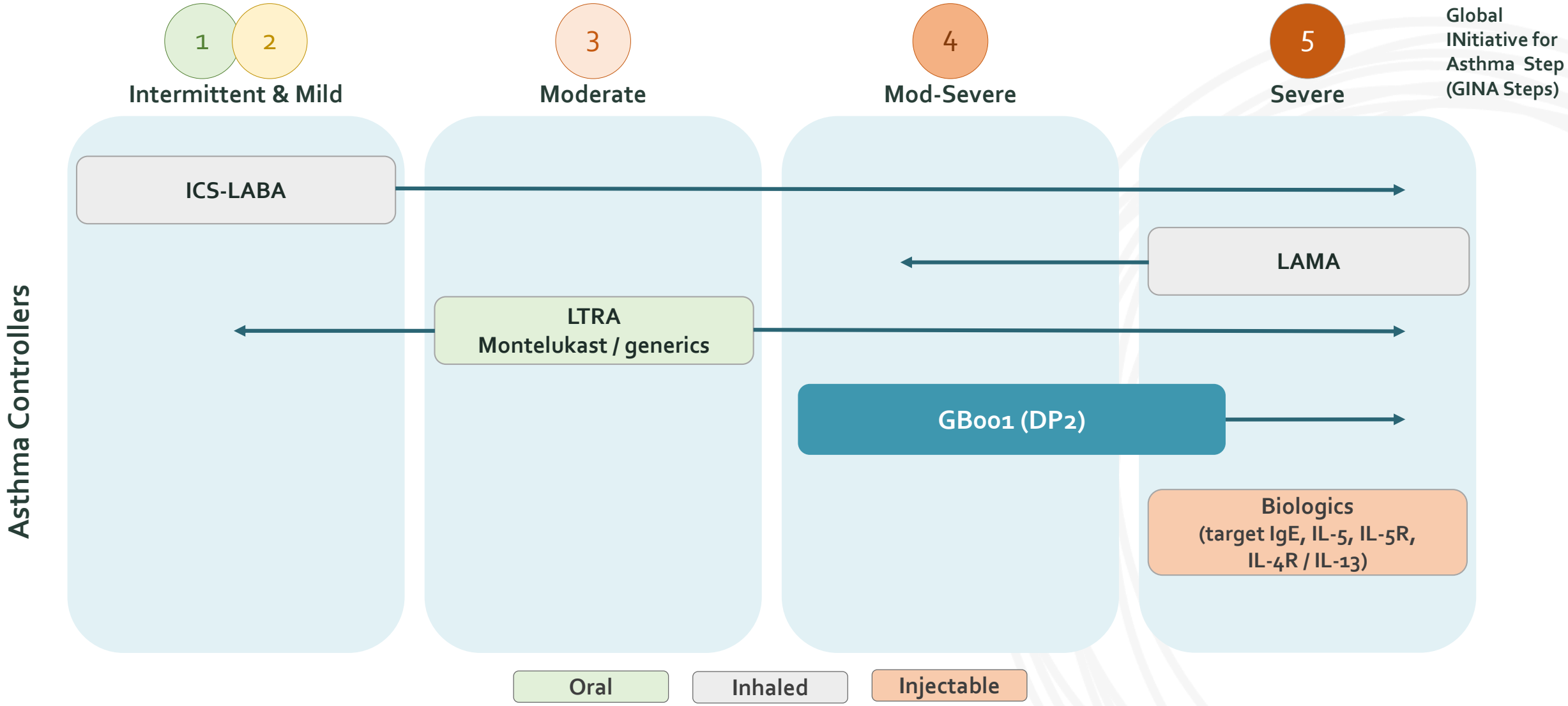
Potential for Market Asthma Positioning Prior to Biologics

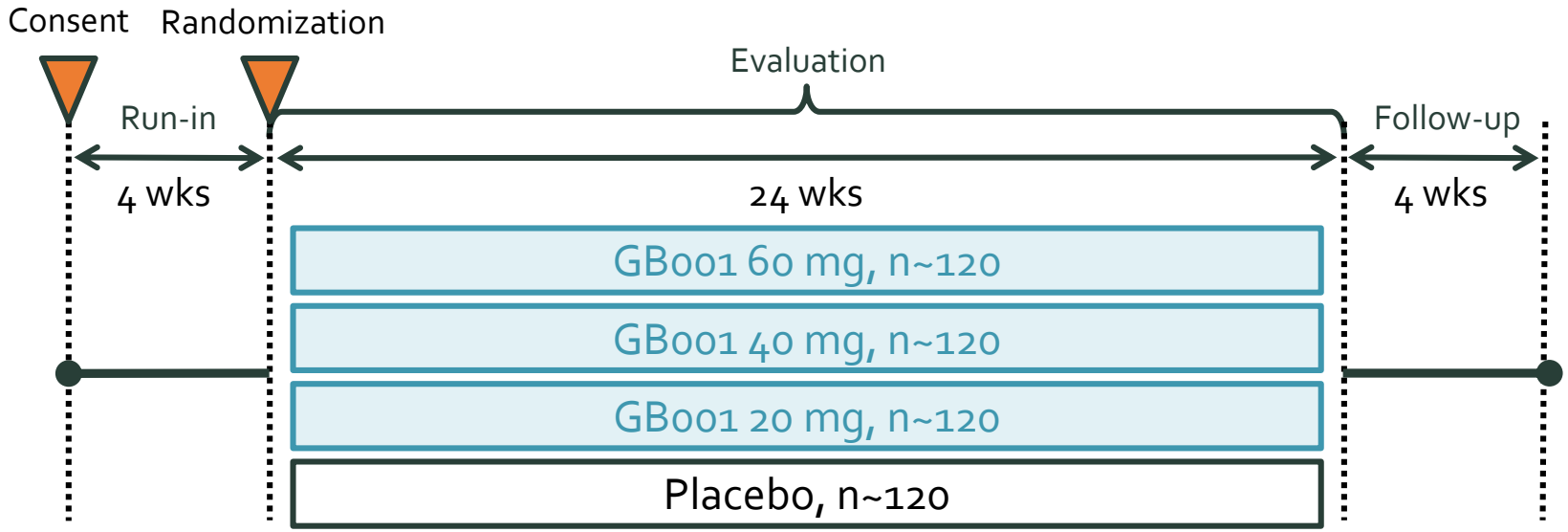


ICS = inhaled corticosteroids; LABA = long-acting beta agonist; LAMA = long-acting muscarinic antagonist; LTRA = leukotriene receptor antagonist.
Source: GINA 2019 guidelines, Datamonitor, open source medical claims, internal analysis.

LEDA Study: Phase 2b Topline Results Expected in 2H:20

A Phase 2b, randomized, double-blind, placebo-controlled, dose-ranging, multi-center study to evaluate the efficacy and safety of GB001 as maintenance therapy in adult subjects with moderate-to-severe asthma

LEDASTUDY



Patient Population	481 adult mod.-to-severe eosinophilic asthmatics (Type 2 phenotype: blood eosinophil ≥ 250 cells/µL)
Endpoints	**Primary:** Reduction in asthma worsening **Secondary:** Annualized Exacerbation Rate, Time to First Asthma Worsening, AM PEF, FEV1, asthma control

Interim analysis completed in Q2:20: based on the results and the recommendation of the IDMC, Gossamer will continue the study to completion and has commenced initial Phase 3 planning and supportive activities

QD = once daily dosing; wks = weeks; IDMC = Independent Data Monitoring Committee.

Chronic Rhinosinusitis (CRS) is a Heterogeneous, Persistent Inflammatory Condition

GB001

Chronic Rhinosinusitis Affects 4% of the Population

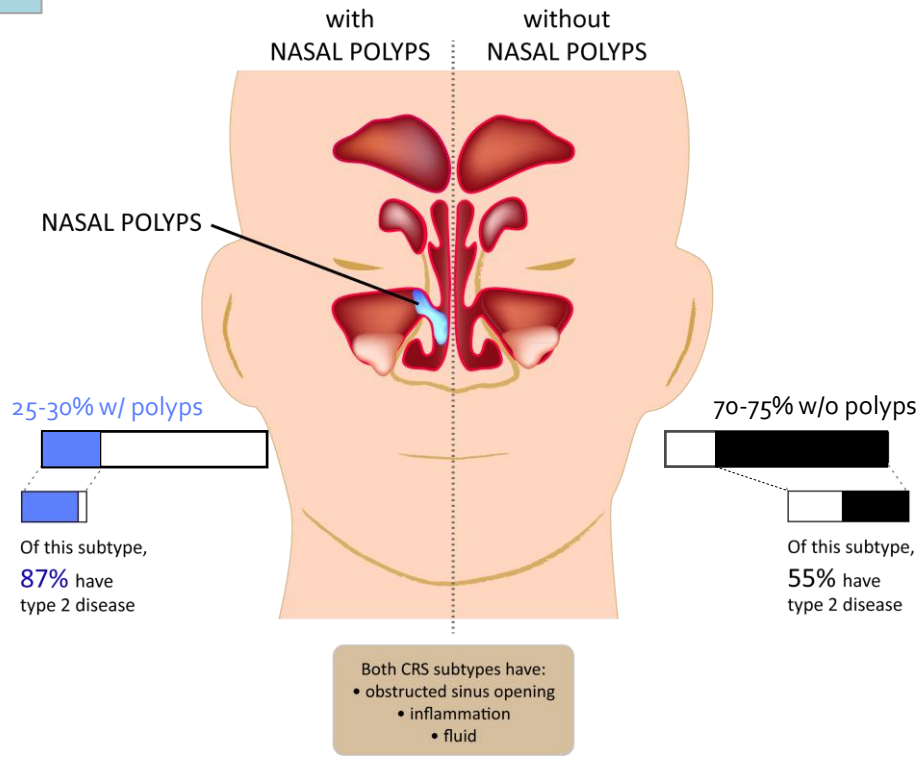
Eosinophilic Inflammation is a Major Pathologic Hallmark of CRS

- Eosinophilic CRS is associated with severe disease resistant to medical and surgical interventions
  - Accumulation of eosinophils, release of cytokines, and mucus secretion implicated in disease pathogenesis
- Expression of PGD2 is increased and an important contributing factor to Type 2 inflammation in Eosinophilic CRS
  - Activated eosinophils secrete tissue-damaging granules and represent an ideal target for selective inhibition

Symptoms

- Difficulty breathing
- Facial pain / pressure
- Chronic headaches
- Loss of smell, taste
- Mucus production
- Nasal discharge
- Nasal infections
- Chronic fatigue

CRS Presents as Two Subtypes



Meltzer, Eli. Update on Sinusitis Management. Presented at: ACAAI 2018 Annual Scientific Meeting; 2018 Nov 7 - 11; Houston.
Hamilos, Daniel "Chronic rhinosinusitis: Epidemiology and medical management." Journal of Allergy and Clinical Immunology 128, no. 4 (2011): 693 – 707.
Hastan, Deniz et al. "Chronic rhinosinusitis in Europe--an underestimated disease. A GA²LEN study." Allergy 66, no. 9 (2011): 1216 – 1223.
Hirsch, Annemarie et al. "Nasal and sinus symptoms and chronic rhinosinusitis in a population-based sample." Allergy 72, no. 2 (2017): 274 – 281.

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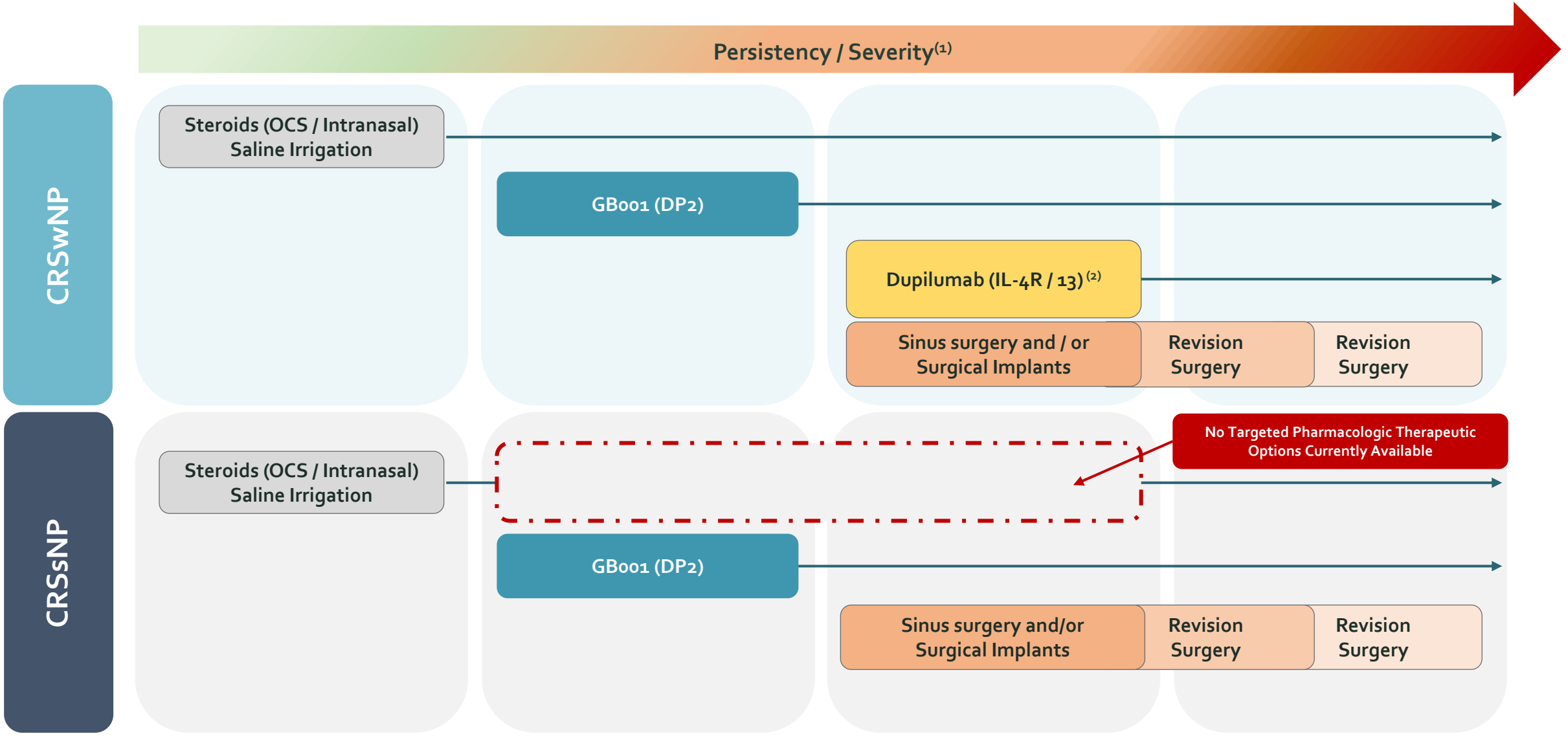
GB001 Has the Potential to be the Only Oral Therapy Available for Severe CRS Patients With and Without Nasal Polyps



1) Excludes antifungals and antibiotics for infectious disease.
2) Multiple biologics in late stage development, including omalizumab (anti-IgE), mepolizumab (anti-IL-5), and benralizumab (anti-IL5R).
NP = nasal polyps; OCS = oral corticosteroids.

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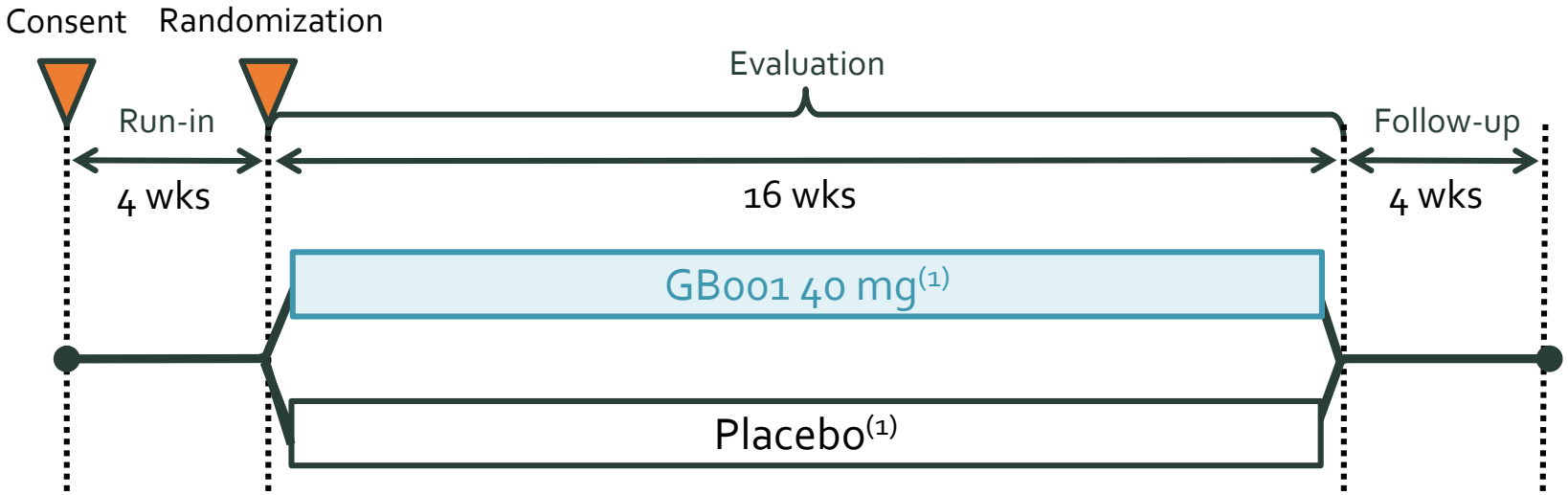
TITAN Study: Phase 2 Proof of Concept in CRS With and Without Nasal Polyps

GB001

A Phase 2, signal seeking, randomized, double-blind, placebo-controlled, multi-center study to evaluate the efficacy and safety of GB001 in combination with intra-nasal steroids in adult patients with CRS

TITAN STUDY



Patient Population	66 adult patients with CRS with nasal polyps (CRSwNP); 31 adult patients with CRS without polyps (CRSsNP)
Endpoint	**Primary:** SNOT-22 (Sino-Nasal Outcome Test-22) **Secondary:** Opacification of sinuses as measured by CT scan, Nasal Polyposis Score (in subset with NP), Nasal Congestion, Incidence of TEAEs, Labs, ECG, vital signs

QD = once daily dosing; NP = nasal polyps; TEAEs = treatment-emergent adverse events; ECG = echocardiogram; wk = week.
1) On top of intra-nasal steroids.

GB002

PDGF Receptor Kinase Inhibitor

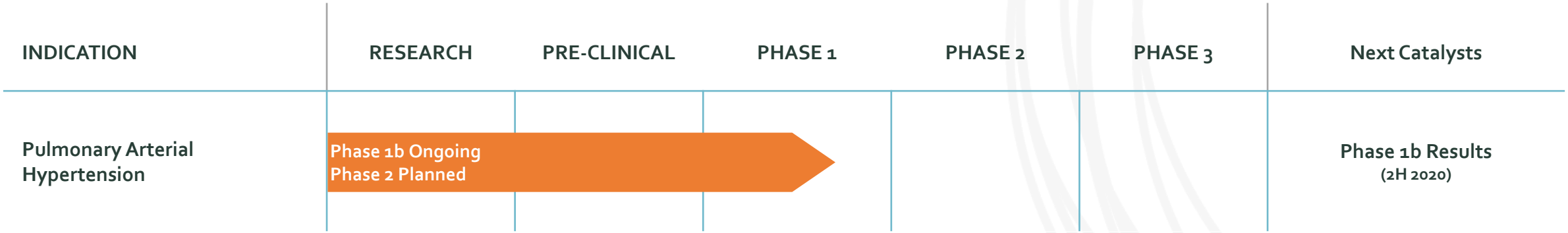
Pulmonary Arterial Hypertension (PAH)

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

GB002

Product Candidate Description

- Selective, inhaled PDGF receptor 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)
- GB002 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 GB002 at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- Patent protection to 2034[(1)]; Orphan Drug Designation from FDA and EMA



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.

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

GB002

PAH affects ~53,000 patients in the United States

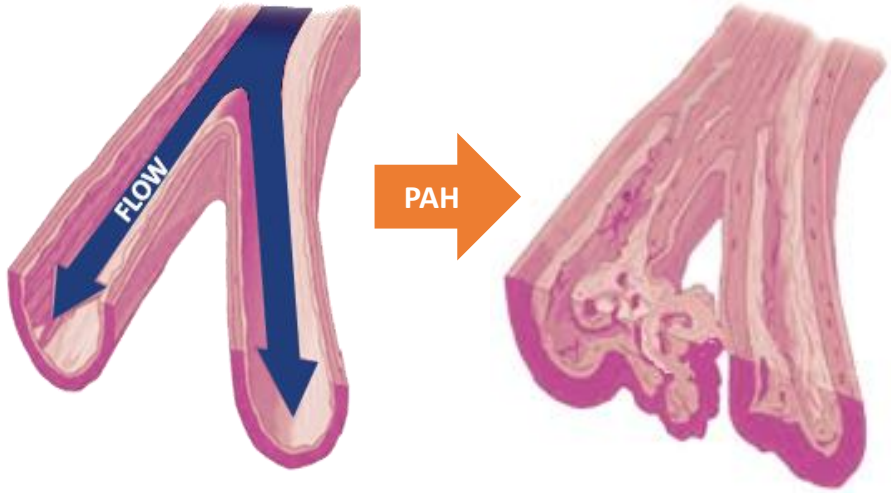
Unabated Cellular Proliferation with Limited Treatment Options

- PAH underlying pathology driven by abnormal cell proliferation related to lung small blood vessels
- Activated PDGF receptor induces cellular proliferation and is known to be upregulated in PAH
- Kinase inhibition was shown to have clinically significant effects in Phase 3 PAH trial of imatinib (Gleevec), with systemic toxicities
- Current therapies function primarily as vasodilators and do not address the abnormal cell proliferation underlying PAH

Symptoms

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

PAH Results in Vascular Remodeling



Source: Datamonitor.

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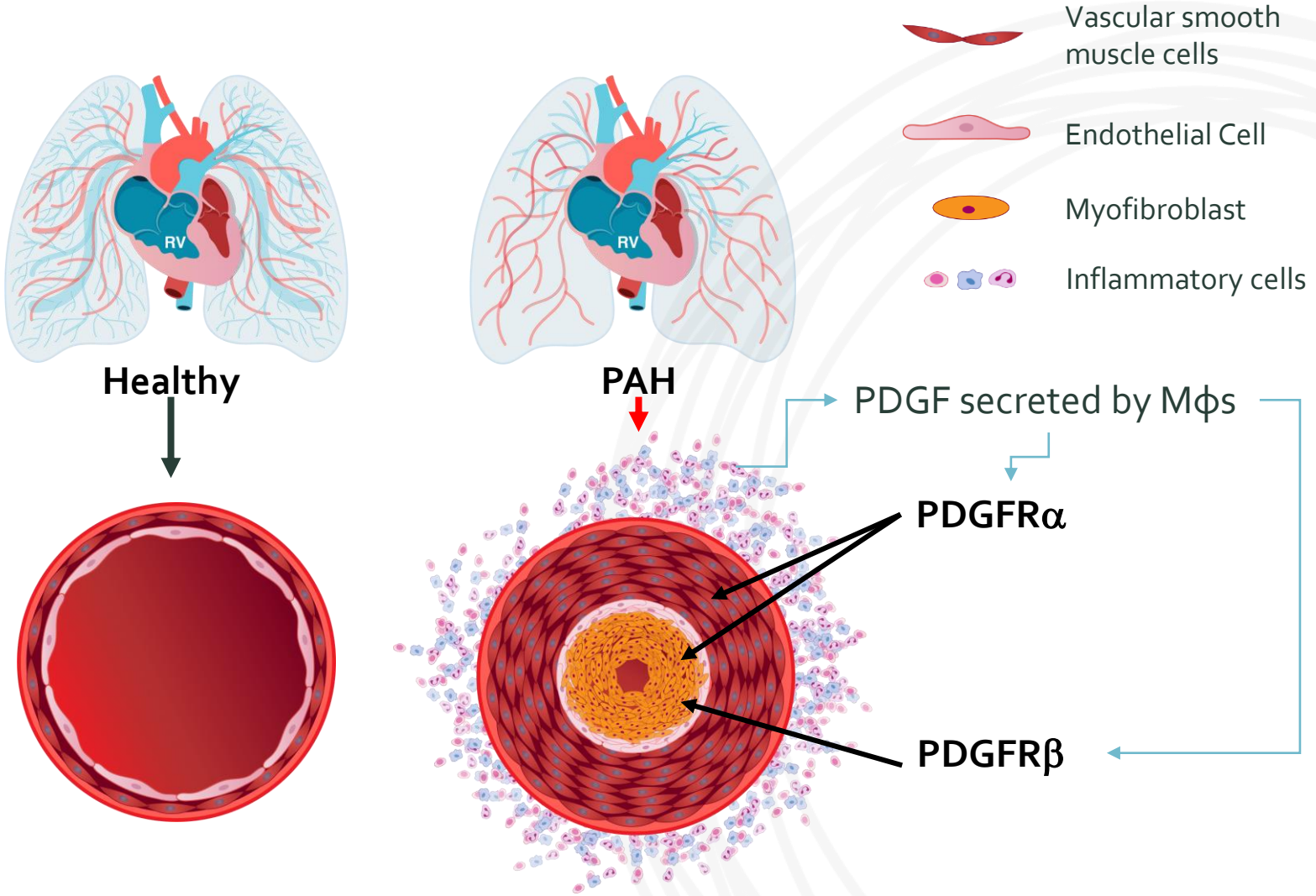
PDGFR Activation Plays a Role in the Pathological Remodeling of Lung Blood Vessels in PAH

GB002

- Aberrant PDGFR signaling drives overgrowth of smooth muscle cells and fibroblasts (via NFkβ, ERK1/2, PLCγ, STAT3 pathways)
- PDGFRα is highly expressed in pulmonary arteriole vascular smooth muscle cells, while PDGFRβ is more highly expressed in myofibroblasts
- Obstructed pulmonary arteries increase pressure, leading to right ventricle thickening and right heart failure
- PDGFR inhibition can normalize BMPR2 expression in arterial smooth muscle cells



RV = right ventricle, Mϕ = macrophage.

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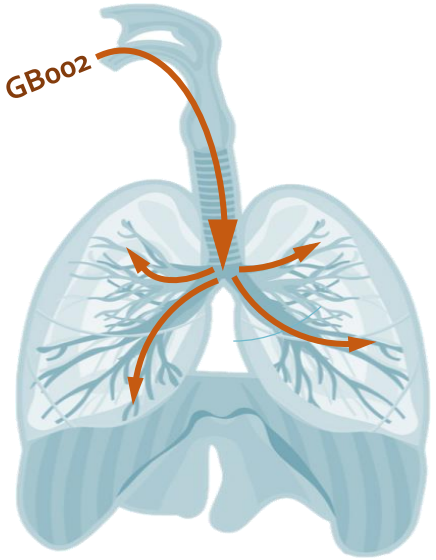
GB002 Administration via Dry Powder Inhaler is Designed to be Convenient and to Deliver Drug Directly to the Site of Disease

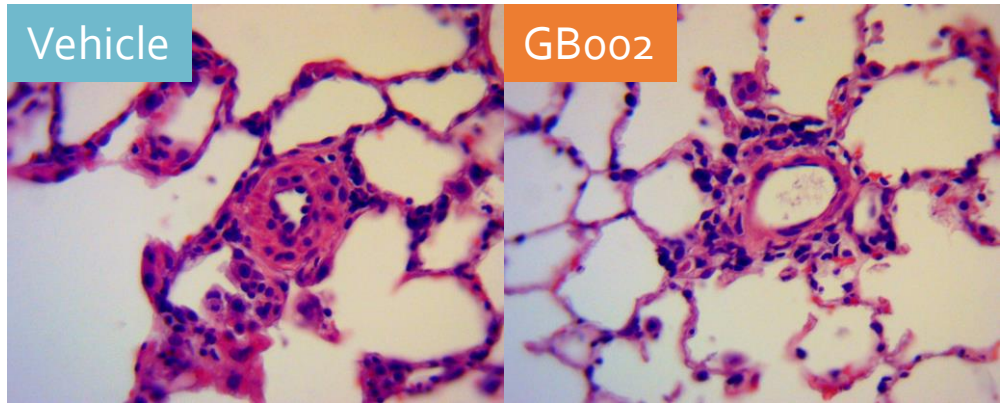
Dry Powder Inhaler from Plastiape

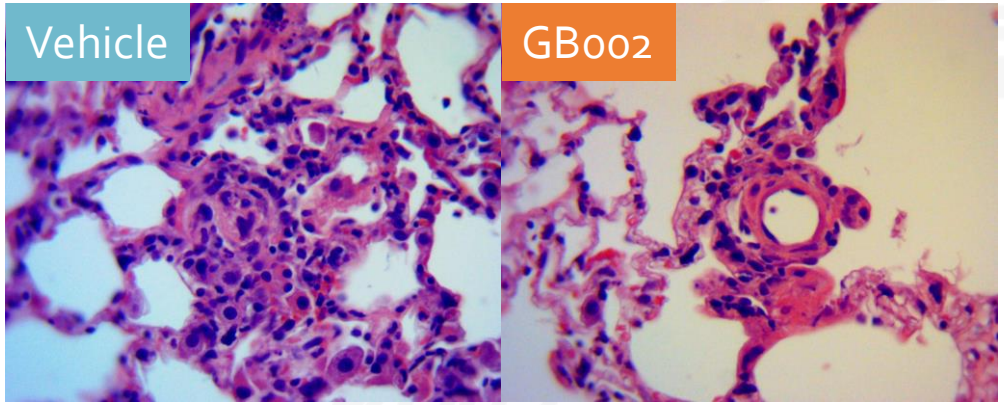


- GB002 formulation and administration via dry powder inhaler (DPI) designed to reach areas of the deep lung
- Designed to deposit inhaled GB002 at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- In pre-clinical studies, has resulted in higher 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

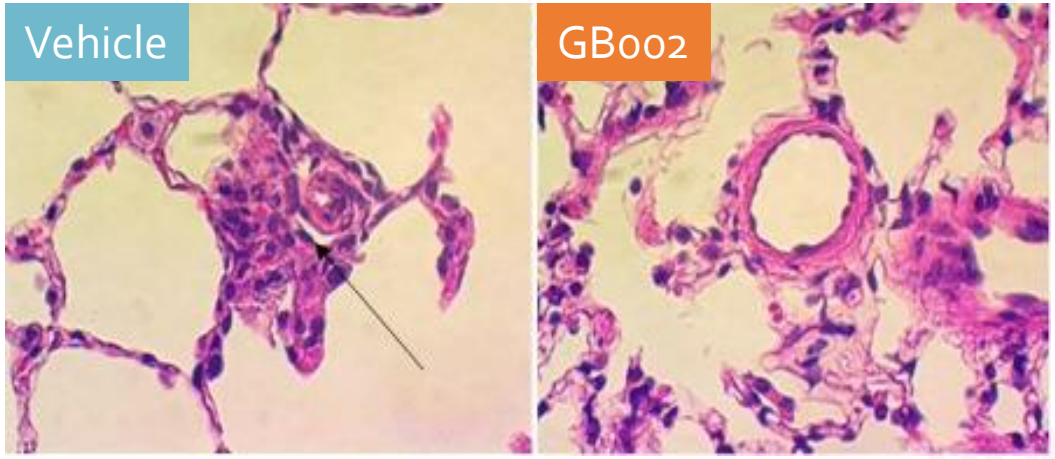
GB002 Reversed Vascular Remodeling Through Inhibition of PDGFR in Animal Models of PAH

GB002 Reversed Remodeled Pulmonary Arterioles in Rat Monocrotaline Plus Pneumonectomy Model(1)





GB002 Restored Healthy Blood Vessel Architecture in Rat SU5416 / Hypoxia PAH Model(2)



1) Sitapara, Ravikumar et al. In Vivo Efficacy of a Novel, Inhaled PDGFRα/β Inhibitor, GB002, in the Rat Monocrotaline and Pneumonectomy Model of Pulmonary Arterial Hypertension. Presented at: American Heart Association Scientific Sessions 2019; 2019 Nov 16 - 18; Philadelphia.
2) Galkin, Anna et al. GB002, A Novel, Inhaled PDGFR Kinase Inhibitor, Demonstrates Efficacy in the SU5416 Hypoxia Rat Model of Pulmonary Arterial Hypertension. Presented at: American Heart Association Scientific Sessions 2019; 2019 Nov 16 - 18; Philadelphia.

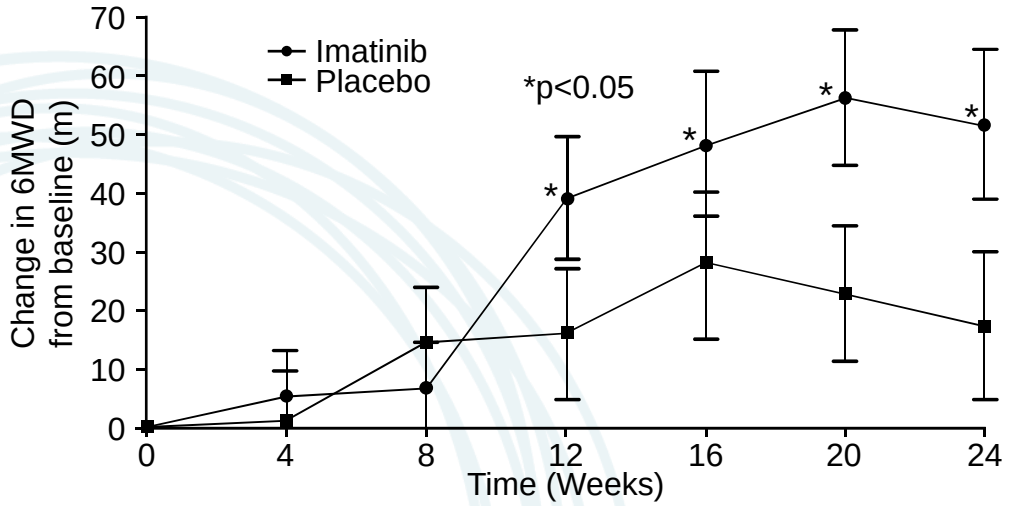
Imatinib Demonstrated Clinical Proof of Principle for Targeting PDGF in PAH in the Phase 3 IMPRES Trial

GB002

Imatinib Mesylate as Add-on Therapy For Pulmonary Arterial Hypertension: Results of the Randomized IMPRES Study

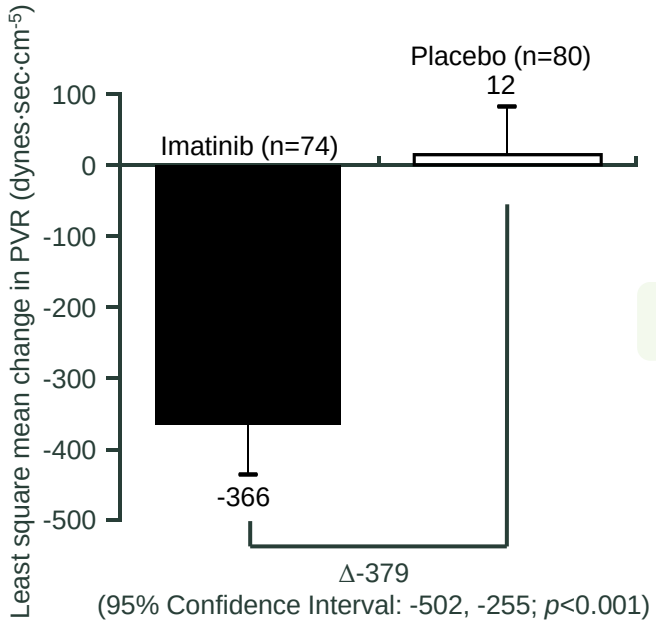
- Imatinib mesylate, as add-on therapy in PAH patients who remain inadequately treated on at least two PAH-specific drugs, improved exercise capacity and hemodynamics
- Of 202 patients enrolled, 41% had failed three classes of therapies, ~70% were on prostacyclins
- Serious Adverse Events: 44% imatinib vs 30% placebo, including 8 subdural hematomas and high drop-out rates
- Discontinuations (overall): 33% imatinib vs 18% placebo

Exercise Tolerance



32m Improvement at 24 Weeks

Pulmonary Vascular Resistance (PVR)



32% reduction

Source: Hoeper, Marius et al. "Imatinib mesylate as add-on therapy for pulmonary arterial hypertension: results of the randomized IMPRES study." Circulation 127, no. 10 (2013): 1128 – 1138.

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Inhaled GB002 Outperformed Imatinib in Head-to-Head Pre-Clinical Studies

GB002

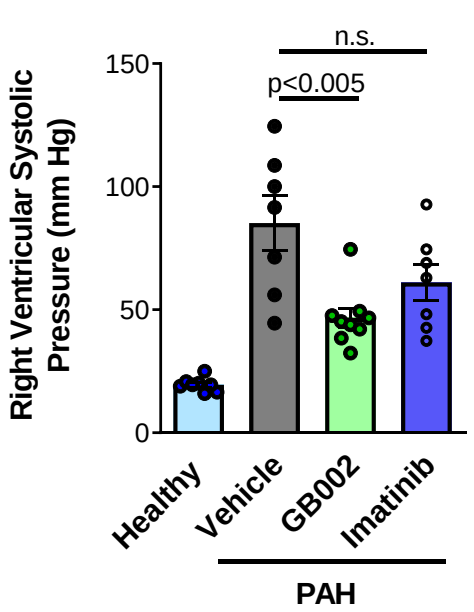
GB002 is a Potent PDGFRα/β Inhibitor with Limited Systemic Exposures

	GB002	Imatinib
PDGFRα IC_{50} (nM)	7	12
PDGFRβ IC_{50} (nM)	6	74
Lung Exposure	++++	+++
Systemic Exposure	+	++

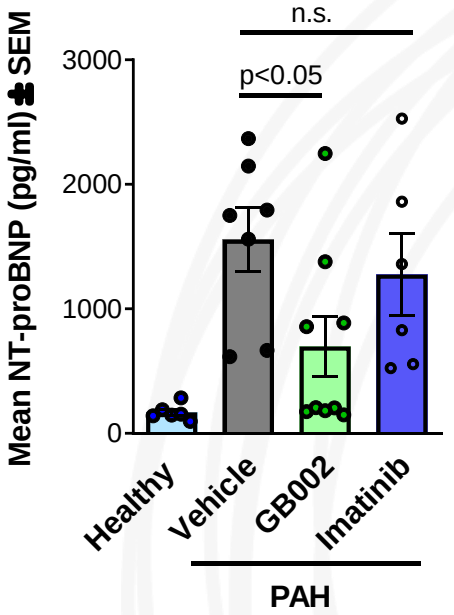
Imatinib in vivo exposures matched to published clinical exposures at the 400 mg dose

GB002 is a new chemical entity

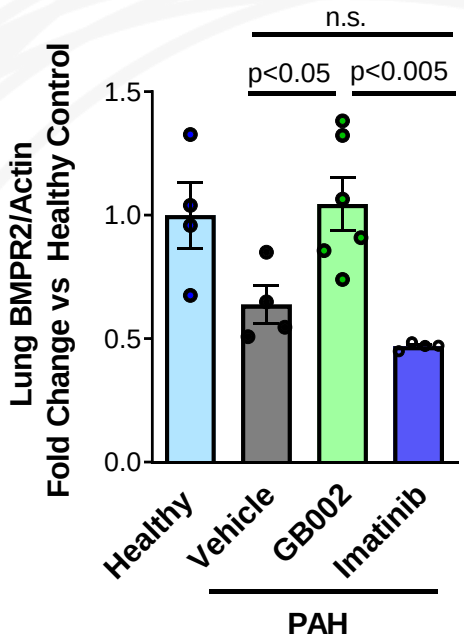
GB002 Generated Greater Reductions in PAH-Related Measures vs. Imatinib and Restored BMPR2 Expression in Rat SU5416 / Hypoxia PAH Model



RVSP:
direct measure of right heart strain used to diagnose PAH



NT-proBNP:
peripheral PAH prognostic biomarker



BMPR2:
receptor implicated in heritable forms of PAH

Source: Galkin, Anna et al. GB002, A Novel, Inhaled PDGFR Kinase Inhibitor, Demonstrates Efficacy in the SU5416 Hypoxia Rat Model of Pulmonary Arterial Hypertension. Presented at: American Heart Association Scientific Sessions 2019; 2019 Nov 16 - 18; Philadelphia.
n.s. = not significant.

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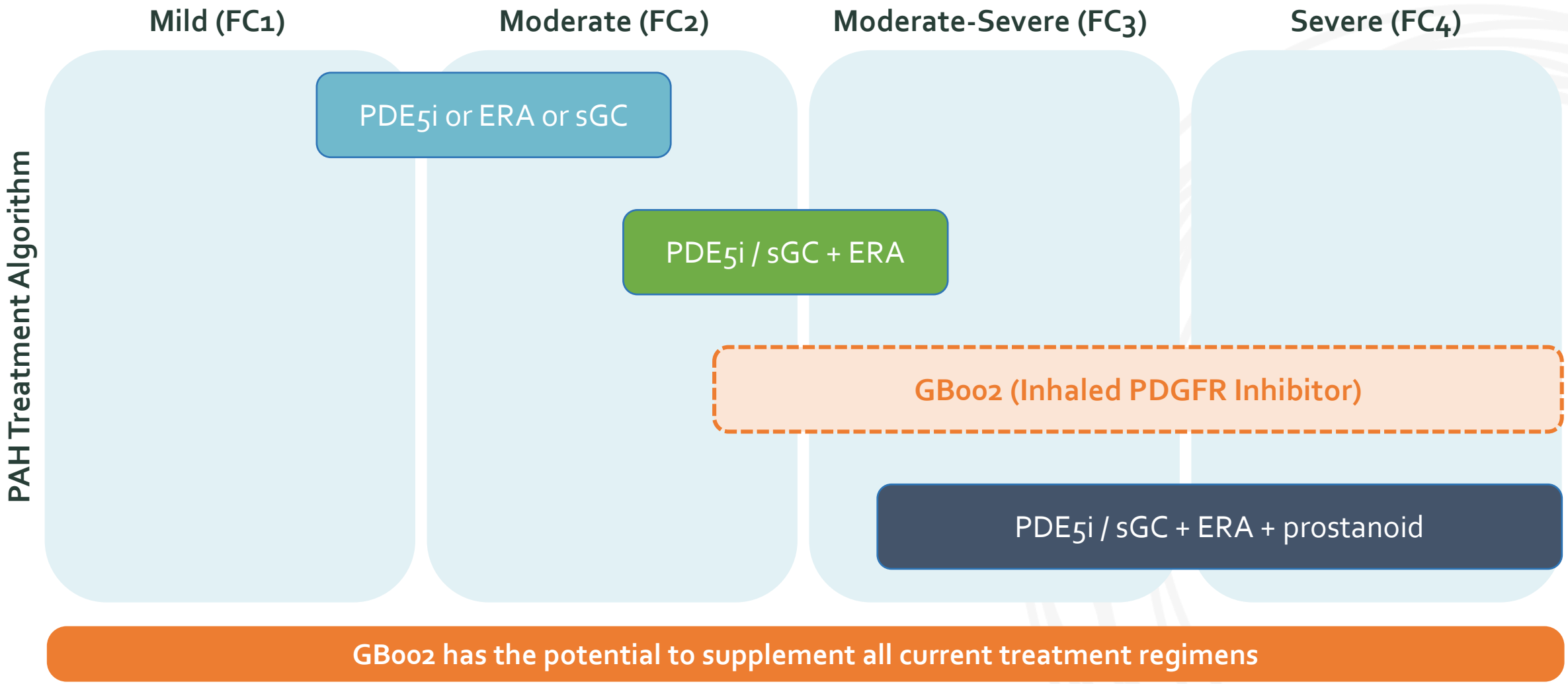
The PAH Treatment Paradigm Increasingly Supports Addition of Therapies as Severity and Functional Class Increase

GB002



ERA = endothelin receptor antagonist; FC = Functional Class; PDE5i = phosphodiesterase type 5 inhibitor; sGC = soluble guanylate cyclase stimulator.
Source: 2015 ESC/ERS Guidelines.

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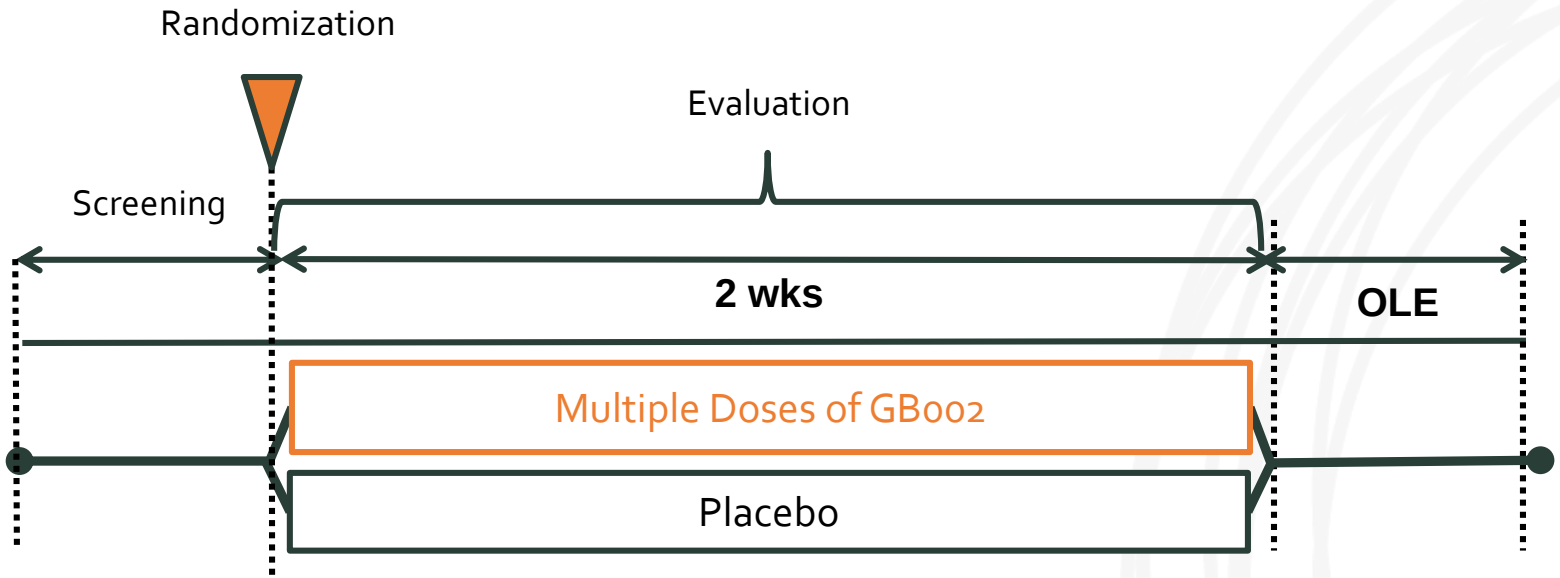
Ongoing Phase 1b Study in Pulmonary Arterial Hypertension

GB002

A Phase 1b, signal seeking, placebo-controlled, dose-ranging study to evaluate the safety and pharmacokinetic profile of GB002 in adult patients with PAH



Patient Population	Adult PAH patients
Endpoints	AE Profile, changes in safety lab values, PK parameters, NTproBNP, Right Ventricular Ejection Fraction (based on ECHO)

NTproBNP = biomarker for heart failure; OLE = open label extension; wk = week.

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GB004

Hypoxia Inducible Factor 1$^{\alpha}$ (HIF-1α) Stabilizer

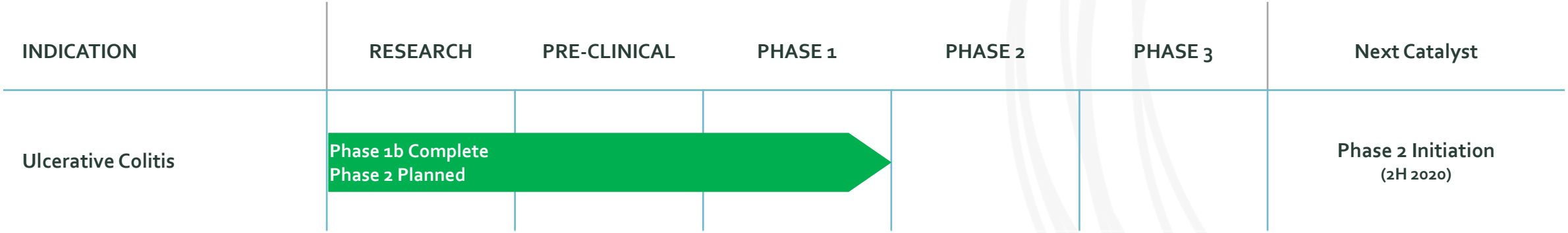
Inflammatory Bowel Disease (IBD), including Ulcerative Colitis (UC) and Crohn's Disease (CD)

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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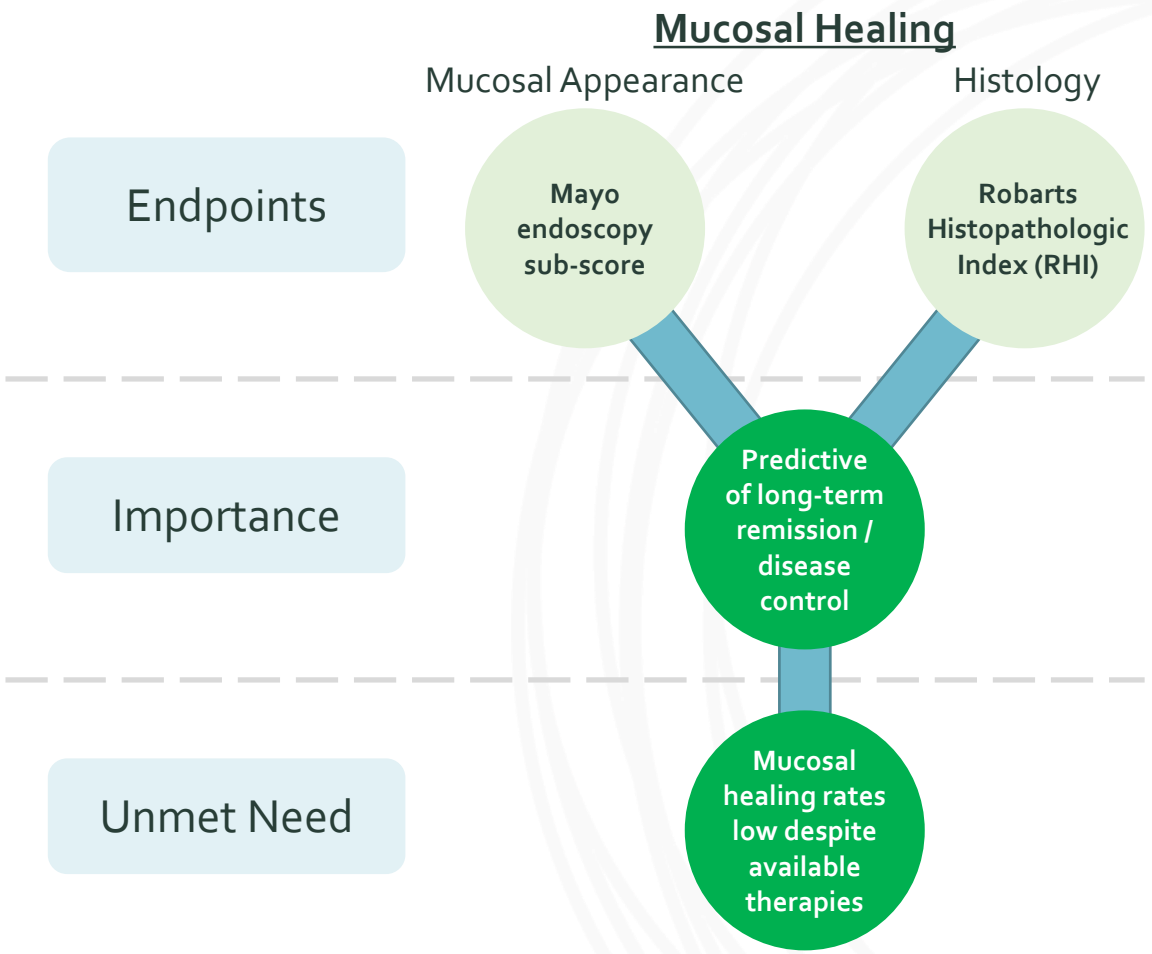
Improved Mucosal Healing is a High Unmet Need in UC

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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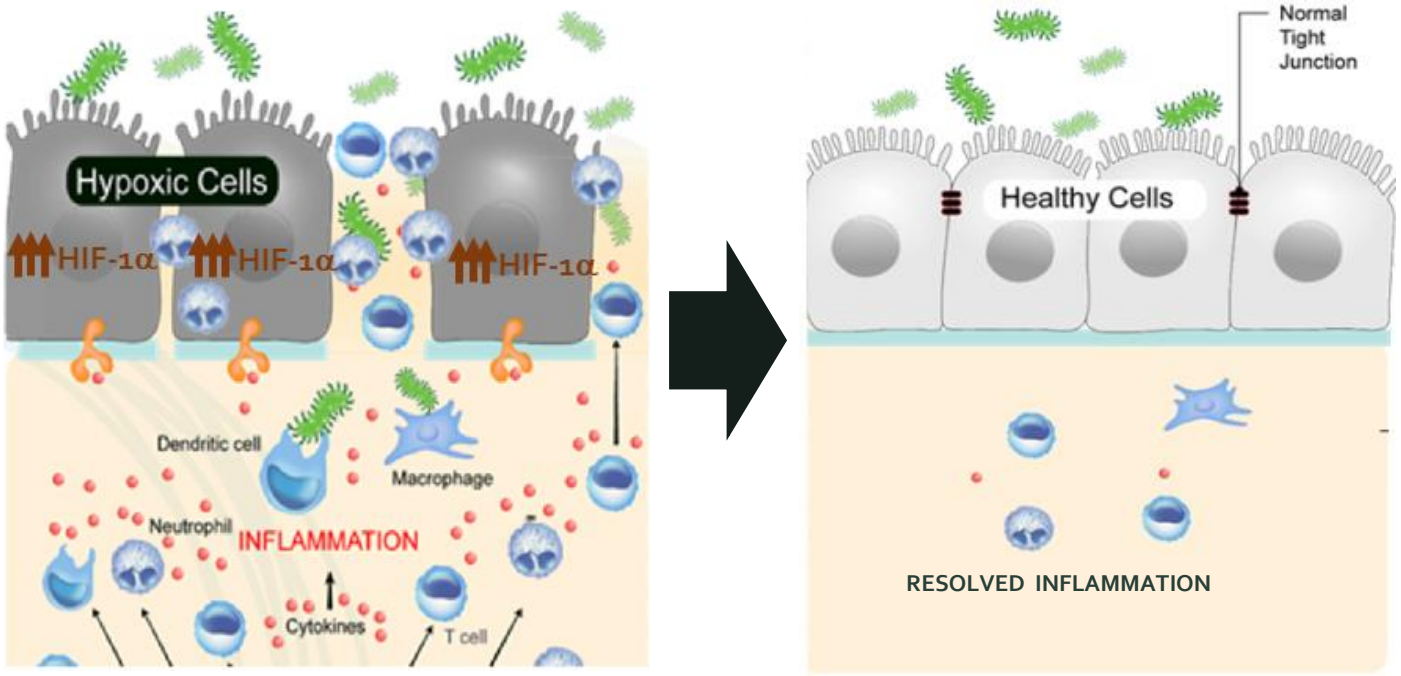
Mechanism of PHD Inhibitor to Restore Epithelial Barrier Function in IBD

GB004

- In patients with IBD, a disrupted epithelial barrier allows for the entry and re-entry of microbes and inflammatory immune cells
- GB004 stabilizes epithelial cell HIF-1α by inhibiting prolyl hydroxylase domain proteins (PHDs)
- HIF-1α promotes expression of epithelial protective molecules and improves barrier function
- The restored gut epithelium limits neutrophil infiltration and activity
  - The RHI assesses neutrophils as a key parameter so histologic remission reflects significant reduction in neutrophilic inflammation



RHI = Robarts Histopathology Index.

Transmigrating Neutrophils Shape the Mucosal Microenvironment through Localized Oxygen Depletion to Influence Resolution of Inflammation, Campbell, et al., Immunity 40, 66–77, January 16, 2014
Neutrophils as Components of Mucosal Homeostasis, Caroline H. T. Hall, et al, Cell Mol Gastroenterol Hepatol 2017;4:329–337
HIF-dependent induction of apical CD55 coordinates epithelial clearance of neutrophils, Nancy A. Louis, et al, FASEB J. 19, 950–959 (2005)

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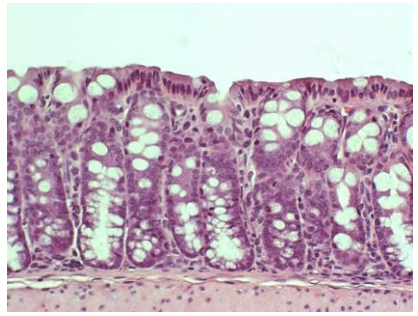
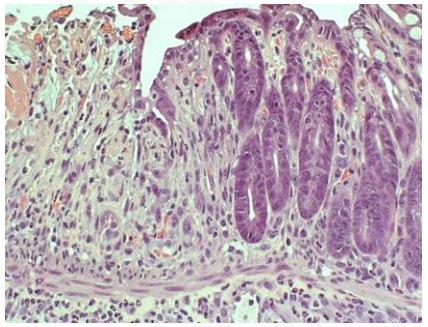
Oral GB004 Reconstituted the Epithelial Barrier and Improved Mucosal Healing in Pre-Clinical Studies

GB004

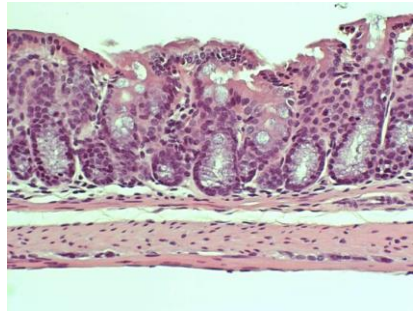
Naive



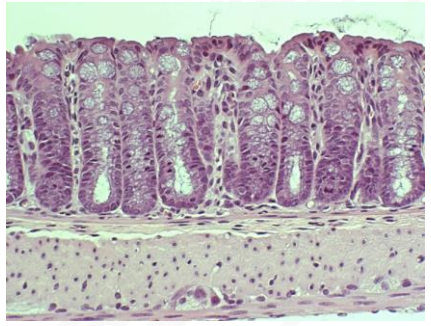
Vehicle



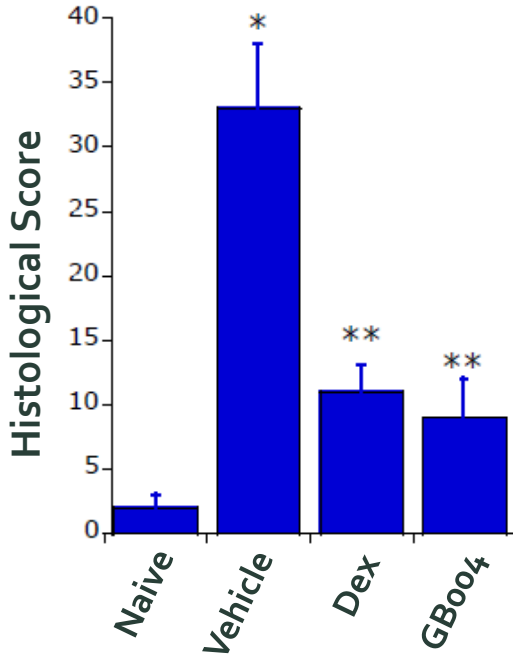
Dexamethasone



GB004 10mg/kg

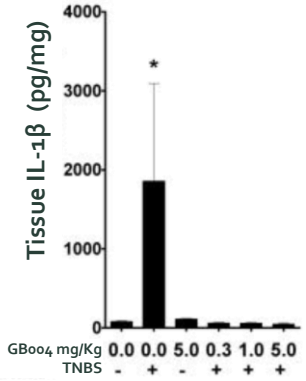


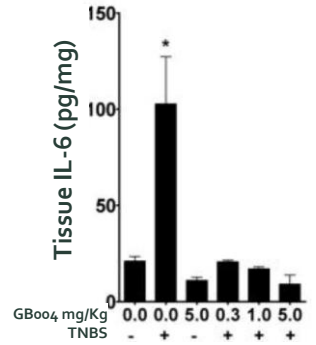
Histological Score Improvement in TNBS-Induced Colitis Model

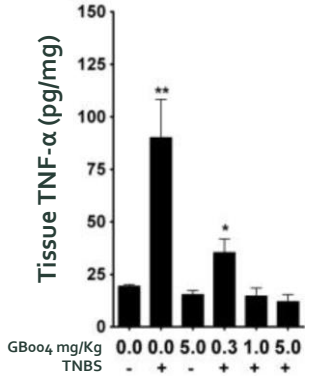


* p<0.01 compared to all other groups
** p<0.025 compared to placebo treated TNBS animals

GB004 Reduced Tissue Cytokines in TNBS-Induced Colitis Model[1]







*p<0.05
**p<0.01

Dex = dexamethasone.
Source: Internally generated data.
1) Simon, Keely et al. "Contribution of epithelial innate immunity to systemic protection afforded by prolyl hydroxylase inhibition in murine colitis." Mucosal Immunology 7, no. 1 (2014), 114–123.

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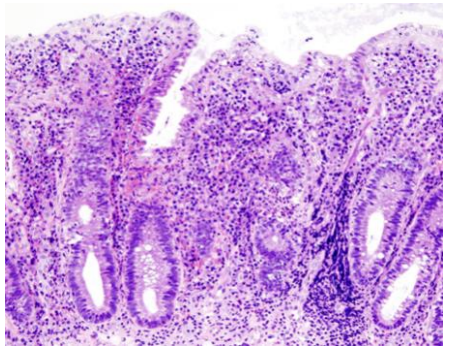
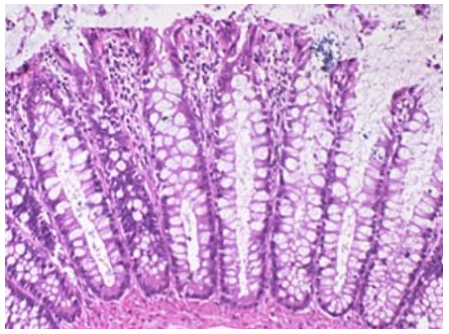
Histologic Healing is Predictive of Favorable Patient Outcomes

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.

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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)

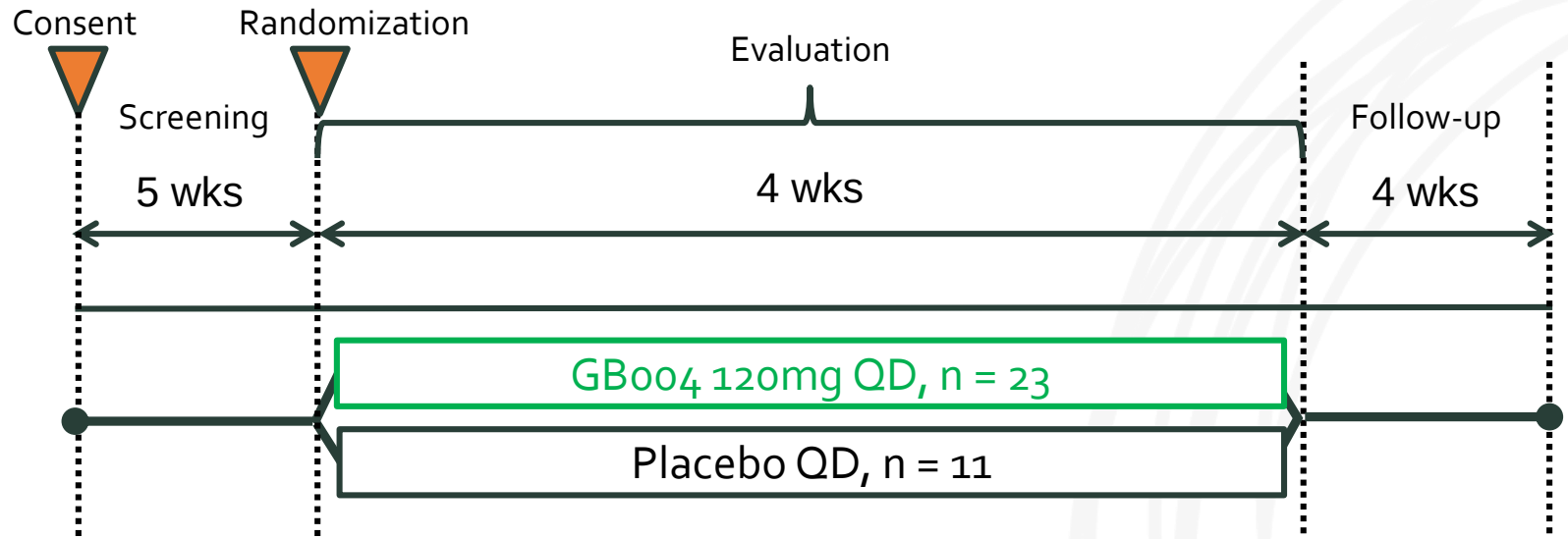
Chronic inflammatory infiltrate (1 x score [0-3])	0 = No increase
	1 = Mild but unequivocal increase
	2 = Moderate increase
	3 = Marked increase
Lamina propria neutrophils (2 x score [0-3])	0 = No increase
	1 = Mild but unequivocal increase
	2 = Moderate increase
	3 = Marked increase
Neutrophils in epithelium (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.

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; wk = week.

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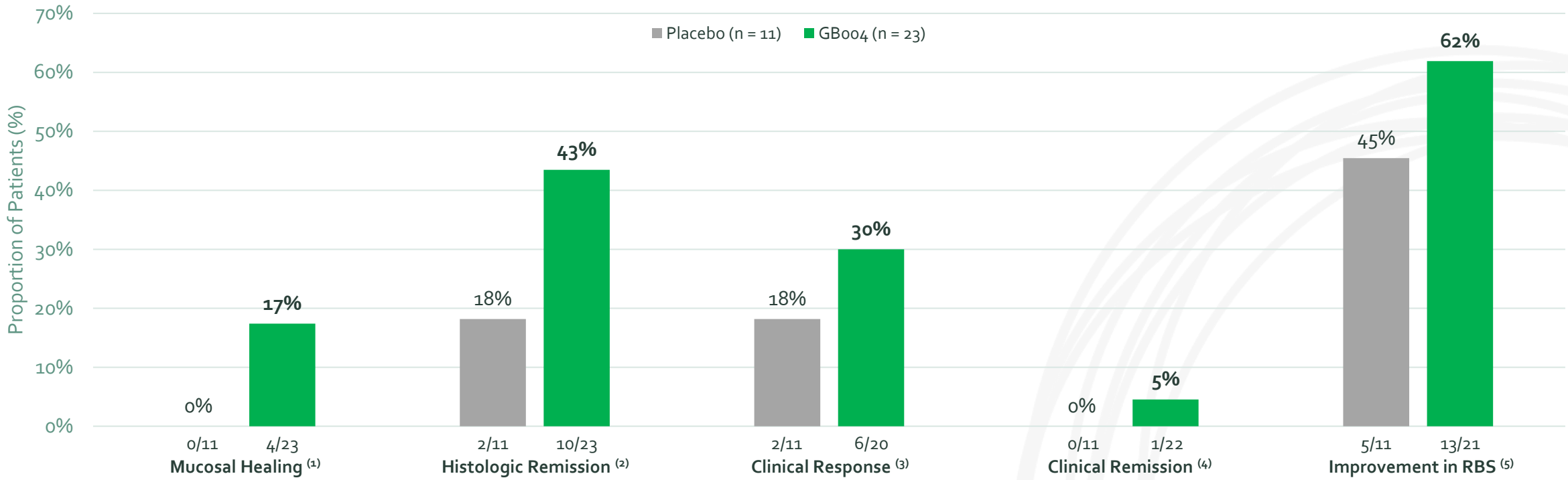
Phase 1b Results: Promising Trends in Exploratory Efficacy Outcomes Observed, Especially in Endpoints Related to Epithelial Lining Healing and Repair

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

Presentation of full results at a future medical meeting; GB004 progressing into Phase 2 in UC in 2H:20

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

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 RBS improvement (baseline RBS of 0).

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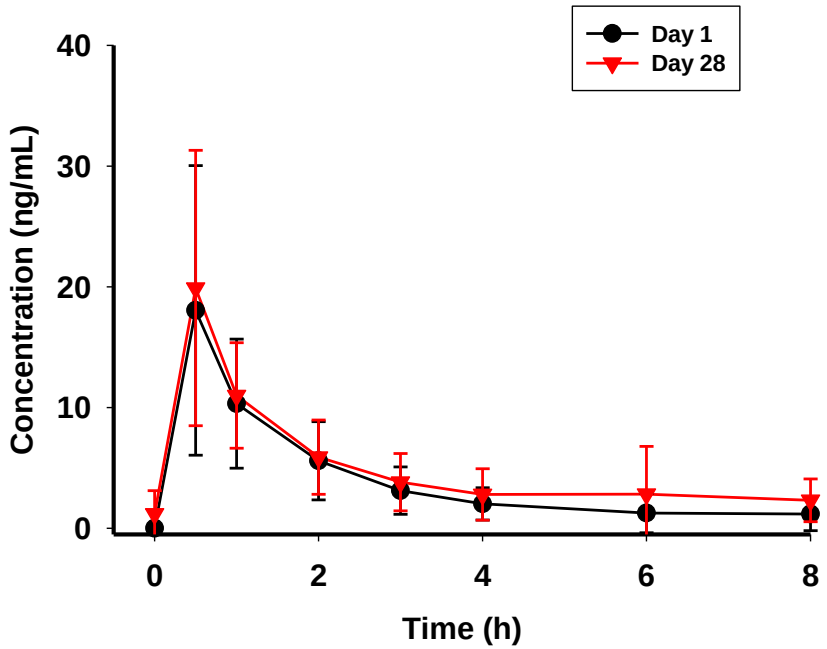
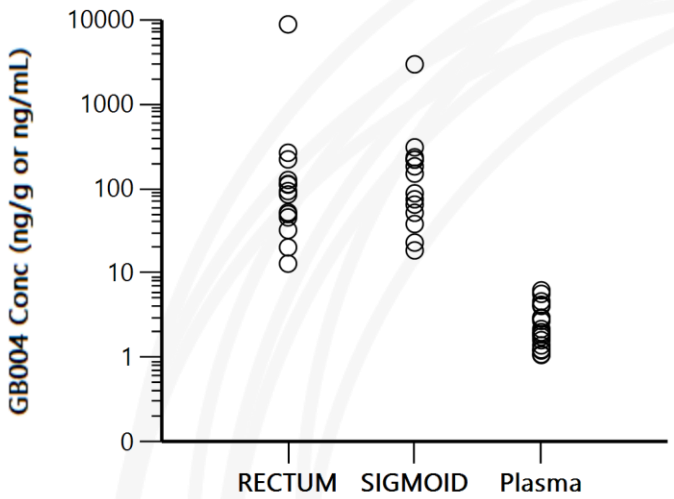
Phase 1b Results: Well-Tolerated With Evidence of Gut-Targeted PK and Target Engagement in Gut

GB004

Rapid Clearance and Minimal Accumulation Observed



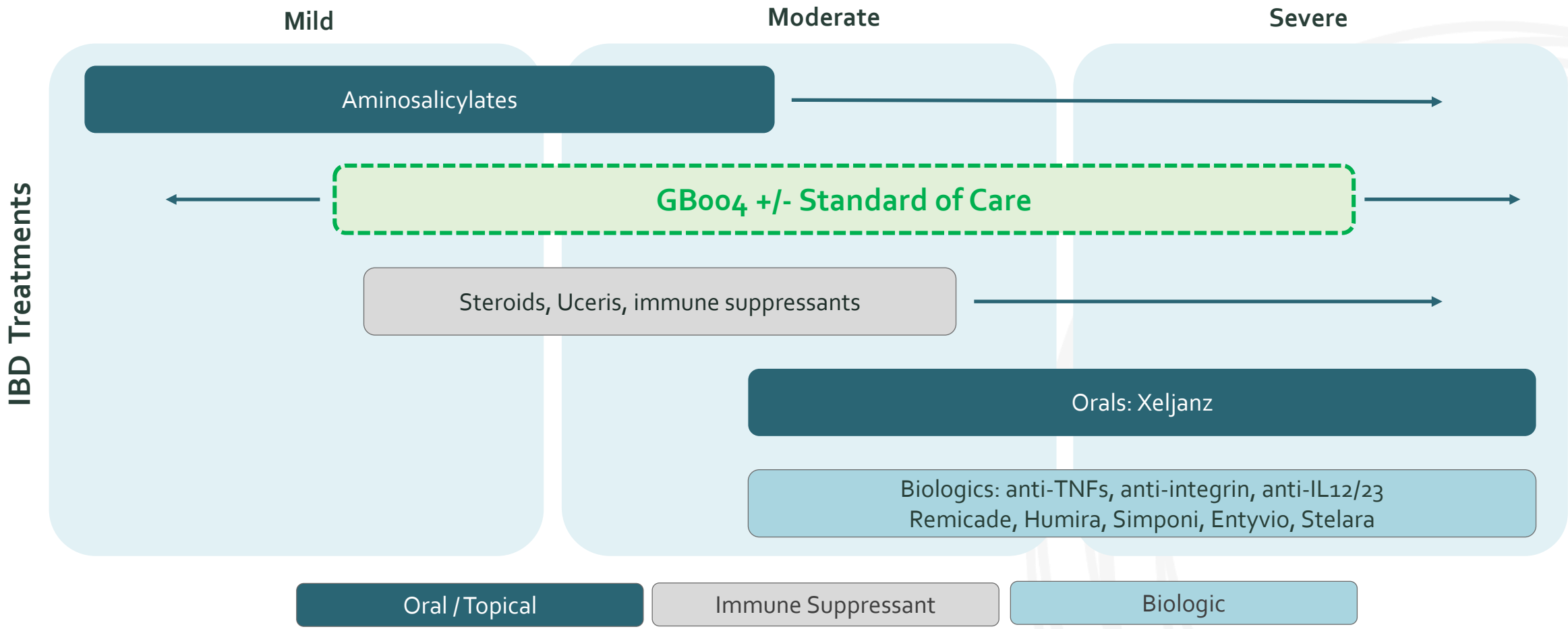
Drug Concentrations Eight Hours After Dosing



Well-Tolerated	• No impact on systemic EPO of VEGF levels • Most frequent AEs of nausea and dysgeusia, all of which were mild or moderate in severity
Gut-Targeted PK Profile	• Rapid clearance from systemic circulation • Multi-fold higher concentration of drug in the gut vs. plasma 8 hours after dosing
Evidence of Target Engagement	• Increased expression of TJP1 and CLD1 genes in GB004 arm, consistent with epithelial healing and barrier repair • Reduced neutrophil activity in gut vs. placebo as measured by preliminary MPO immunohistochemistry staining

EPO = erythropoietin; VEGF = vascular endothelial growth factor; TJP1 = tight junction protein 1; CLDN1 = Claudin 1; MPO = myeloperoxidase

GB004 Represents a Potential New, Gut-Targeted Transformative Approach in IBD



GB004 has the potential to be positioned as pre-biologic therapy across the spectrum of disease activity segment as monotherapy or in combination

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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

INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	Next Catalyst
Oncology, Solid Tumors	Phase 1/2 Ongoing – KEYNOTE-A36 (Clinical Supply Agreement with Merck for KEYTRUDA)(2)					Initial Phase 1 Data (Q2 2020 - ASCO) Updated Phase 1 Data (2H 2020)

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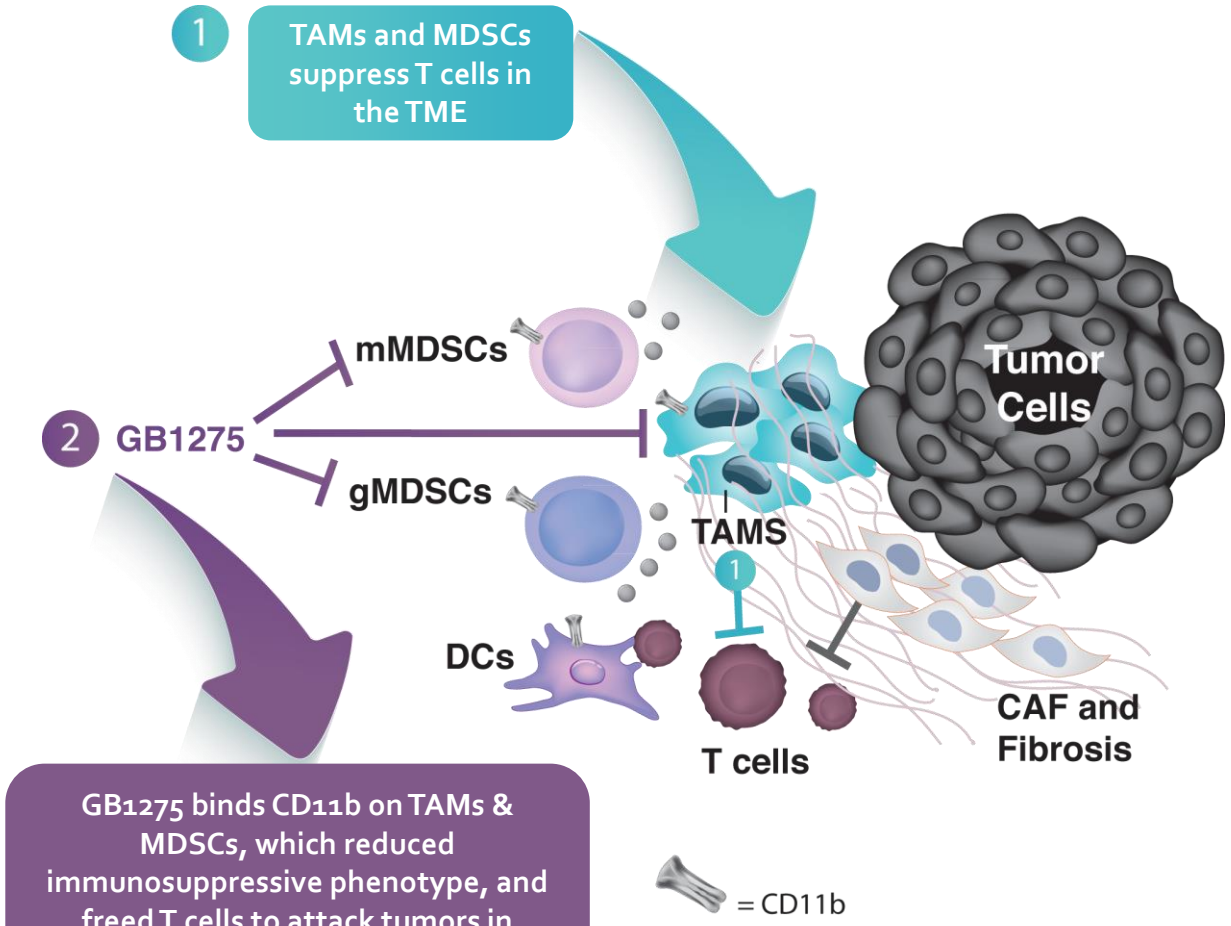
MDSCs and M2 Macrophages Generate an Immunosuppressive Tumor Microenvironment and Hinder Response to IO Agents



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.

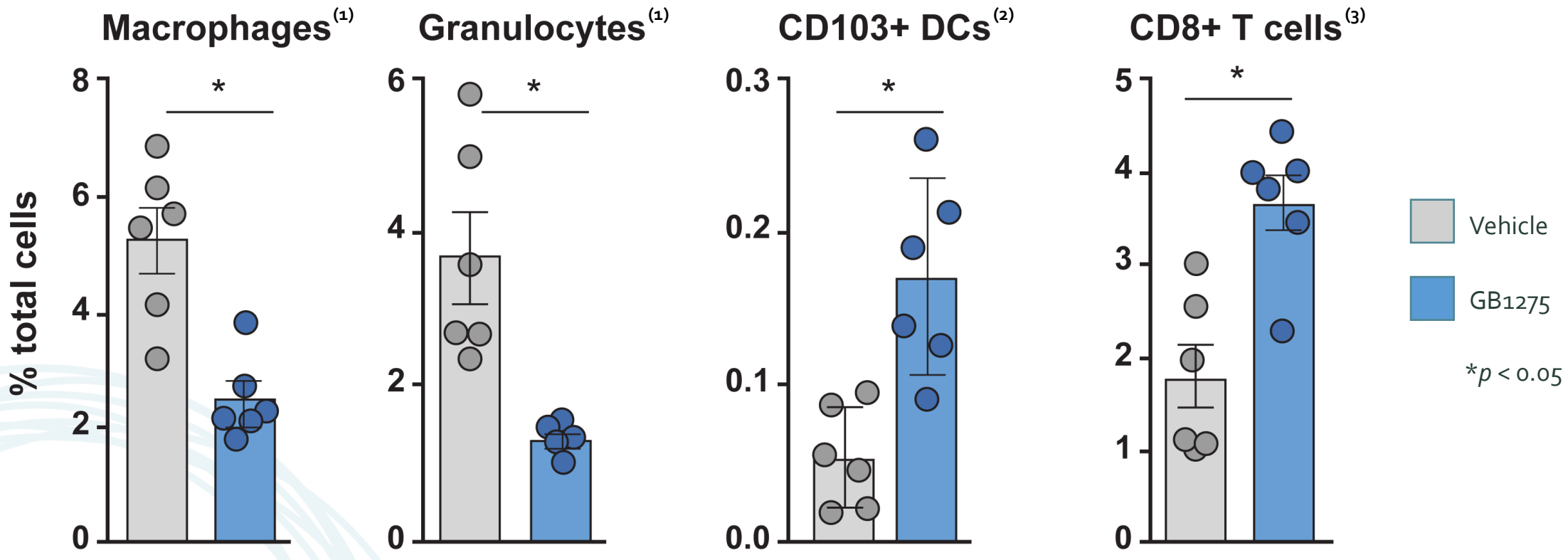
GB1275 Has the Potential to Inhibit Multiple Immunosuppressive Myeloid Cell Types

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).

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



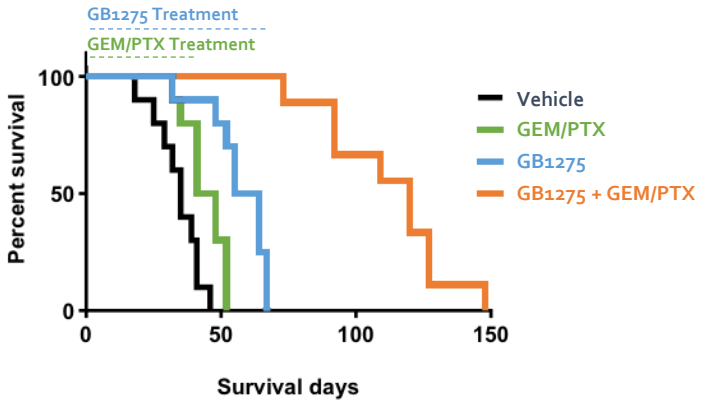
- GB1275 immuno-modulatory effects were tumor-localized
- GB1275 did not alter immune cell phenotypes in the periphery, spleen or bone marrow

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).

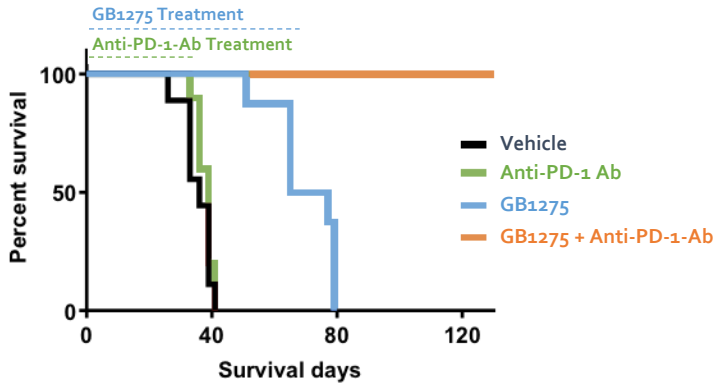
GB1275 Single Agent or in Combination Improved Tumor Responses and Survival Outcomes in Multiple Difficult-to-Treat Tumor Models

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

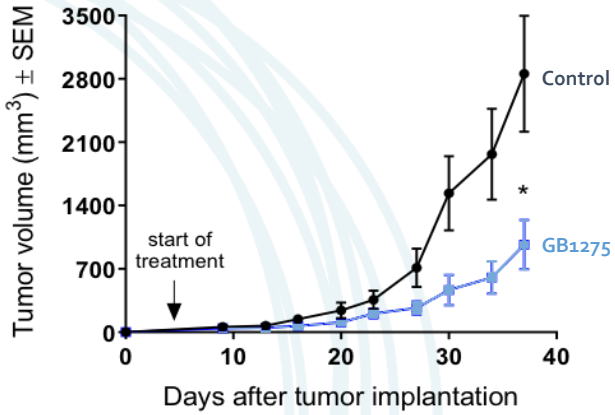


GB1275 Sensitized PDAC Model to anti-PD-1 Ab Checkpoint Blockade

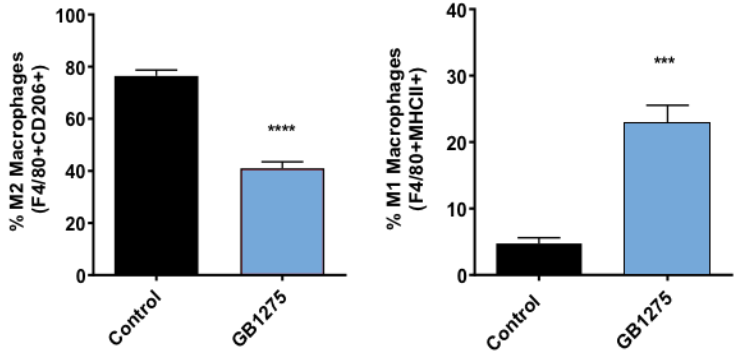


GB1275 Modulated Breast Cancer Tumor Microenvironment via Repolarization of Tumor Associated Macrophages[2]

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.

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

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
- Anti-PD1 combo
- Gem / Abraxane combo

Phase 2

3 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

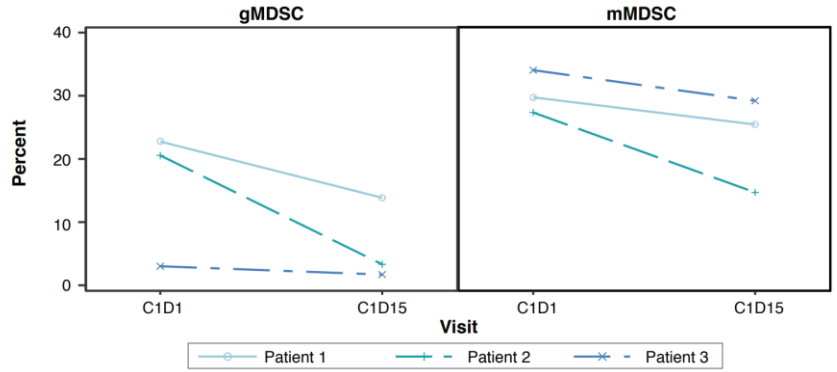
PDAC = pancreatic ductal adenocarcinoma; TNBC = triple negative breast cancer; CRC = colorectal cancer; MSS = micro-satellite stable; Gem = gemcitabine; mPanc = metastatic pancreatic cancer; Gastric = gastric cancer.

Preliminary Phase 1/2 Data Presented at ASCO20

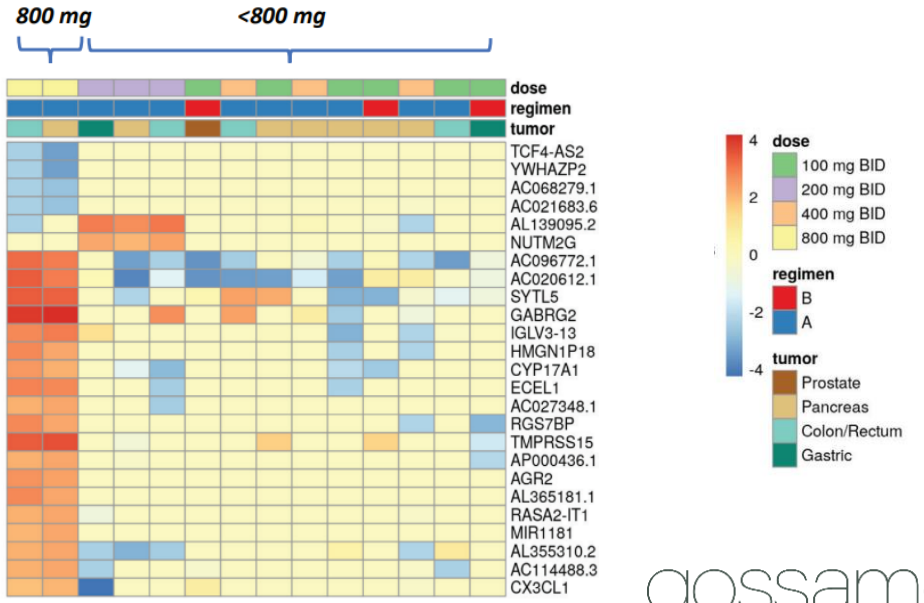
GB1275

- Enrollment as of data cutoff on March 27, 2020:
  - Regimen A (GB1275 monotherapy): 14 patients
  - Regimen B (combination with KEYTRUDA): 8 patients
- No dose-limiting toxicities and all treatment-related AEs mild in severity – no immune-related AEs were reported
- Preliminary biomarker results:
  - Modulation of peripheral levels of MDSCs
  - Dose-dependent changes in peripheral gene expression observed
  - Assessment of tumor and blood biomarker data ongoing
- 7-hour $t_{1/2}$ compatible with BID dosing
- In Regimen B, a mCRPC patient experienced a 52% max decrease from baseline in PSA (from 3730 ng/ml to 1790 ng/ml) and 52% max decrease from baseline in neutrophil-to-lymphocyte ratio
  - Previously progressed on 10 lines of therapy, including atezolizumab (anti-PD-L1) and olaparib (PARP inhibitor)
- Dose escalation ongoing in both Regimen A and Regimen B; updated results to be presented in 2H:20

Reductions in Peripheral MDSCs(1)



Preliminary Dose-Dependent Changes in Gene Expression



MDSC = myeloid-derived suppressor cell; mMDSC = monocytic MDSC; gMDSC = granulocytic MDSC; mCRPC = metastatic-resistance prostate cancer
1) Regimen B 400mg cohort.

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Corporate Overview and Milestones

Financial Overview

Cash, Cash Equivalents and Marketable Securities — ~$642mm

(As of 3/31/2020; pro forma for May 2020 Equity and Convertible Offerings and GB004 license restructuring)[1]

Debt, *Related to Line of Credit* — ~$30mm

(As of 3/31/2020; initial tranche of $150 million debt facility, announced 5/2/19)

Additional Debt Capacity, *Related to Line of Credit* — ~$120mm

(As of 3/31/2020; remaining capacity of $150 million debt facility, announced 5/2/19)[2]

Principal of Convertible Notes Outstanding — ~$200mm

(Pro Forma for May 2020 Offering)

Common Shares Outstanding — ~75.8mm

(As of 3/31/2020; pro forma for May 2020 Equity and Convertible Offerings)

mm = millions.
1) Pro forma as adjusted cash balance following May 2020 offerings, less $15mm GB004 license restructuring payment.
2) Accessible subject to the achievement of certain clinical development milestones and other customary conditions.

Upcoming Milestones

Indication	Milestone	Timing
GB001 (Asthma and Other Allergic Conditions, Including Chronic Rhinosinusitis)		
Asthma	Phase 2b Interim Analysis (LEDA Study)	Q2 2020 ✓
Asthma	Phase 2b Topline Results (LEDA Study)	2H 2020
CRS	Phase 2 Topline Results (TITAN Study)	2H 2020
GB002 (Pulmonary Arterial Hypertension)		
PAH	Phase 1b 2 Week Results	2H 2020
PAH	Phase 2 Initiation	2H 2020
GB004 (Inflammatory Bowel Disease)		
UC	Phase 1b Results	Q2 2020 ✓
UC	Phase 2 Initiation	2H 2020
GB1275 (Oncology, Solid Tumors)		
Solid Tumors	Initial Phase 1 Data (KEYNOTE-A36)	Q2 2020 (ASCO) ✓
Solid Tumors	Updated Phase 1 Data (KEYNOTE-A36)	2H 2020

Experienced Leadership Team at the Helm

Sheila Gujrathi, MD
Chief Executive Officer

receptos · Bristol-Myers Squibb · Genentech · McKinsey&Company · Turning Point Therapeutics · Brigham and Women's Hospital · UCSF · Stanford

Bryan Giraudo
Chief Financial Officer

LEERINK · Merrill Lynch

Luisa Salter-Cid, PhD
Chief Scientific Officer

Bristol-Myers Squibb · La Jolla Pharmaceutical

Christian Waage
EVP and General Counsel

receptos · Ardea Biosciences

Board of Directors

Faheem Hasnain
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Kristina Burow
Managing Director,
ARCH Venture Partners

Tom Daniel, MD
Former Celgene Research Chair,
Pres. of Res. & Early Dev.

Renée Galá,
CFO,
Jazz Pharmaceuticals

Sheila Gujrathi, MD
CEO

Josh Bilenker, MD
Head of Loxo Oncology,
Eli Lily

Russell Cox
CEO,
Epirium Bio

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