Corporate Presentation

March 2020

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, with 4 assets in clinical development and multiple preclinical programs underway
Diversified Pipeline	• 4 clinical-stage product candidates and multiple data readouts over the next 12 months
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 – 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)
GB001	DP2 Antagonist (Oral)	Chronic Spontaneous Urticaria	Phase 2 Under Review					Worldwide (except Japan)
GB002	PDGFR Inhibitor (Inhaled)	Pulmonary Arterial Hypertension	Phase 1b Ongoing					Worldwide
GB004	HIF-1α Stabilizer (Oral)	Inflammatory Bowel Disease	Phase 1b Enrollment Complete					Worldwide
GB1275	CD11b Modulator (Oral)	Oncology, Solid Tumors	Phase 1/2 Ongoing					Worldwide

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GB001

DP2 Antagonist

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

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

Product Description

- Oral, once-a-day, DP2 antagonist in development for the treatment of moderate-to-severe eosinophilic asthma, chronic rhinosinusitis (CRS) and chronic spontaneous urticaria (CSU)
- Target validation from Teijin's GB001 Phase 2 study in Japanese asthmatics
- Anti-inflammatory effect comparable to certain biologics with potential to be used earlier in treatment
- 409 patients have received at least 1 dose of GB001 with no clinically significant safety findings(1)
- Patent protection to 2031(2)

INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	Next Catalyst
Moderate-to-Severe Eosinophilic Asthma	Phase 2b Enrollment Complete – LEDA Study					Phase 2b Interim Analysis (Q2 2020)
Chronic Rhinosinusitis (with and without nasal polyps)	Phase 2 Enrollment Complete – TITAN Study					Phase 2 Topline Results (2H 2020)
Chronic Spontaneous Urticaria	Phase 2 Under Review					Phase 2 Initiation (2H 2020)

CRS = Chronic Rhinosinusitis.

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.

Eosinophils Play a Critical Role in Type 2 Asthma

GB001

There are ~2.4mm – 3mm uncontrolled GINA 4/5 asthmatics in the US, 1.2 – 1.5mm of which are high Eos.

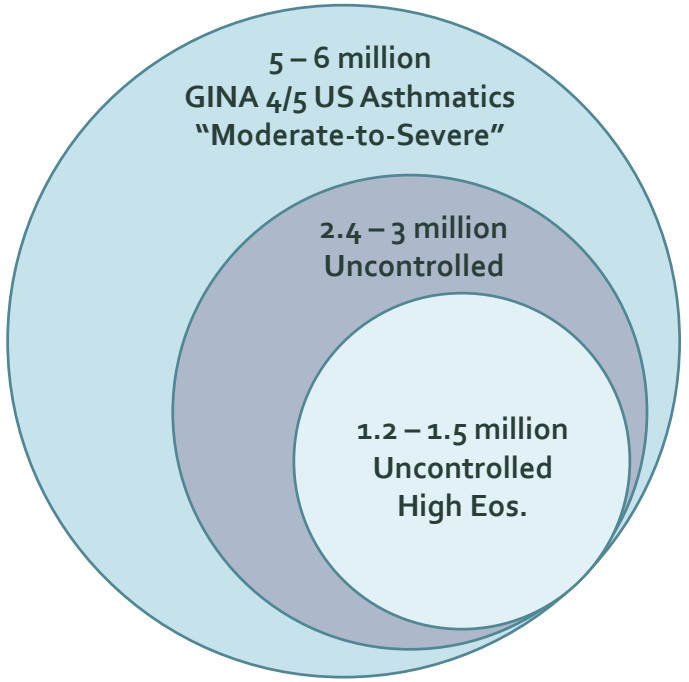
Eosinophils are Key Cells Mediating Inflammation in Asthma

- 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



Datamonitor, Open Source Medical Claims, Internal Analysis.

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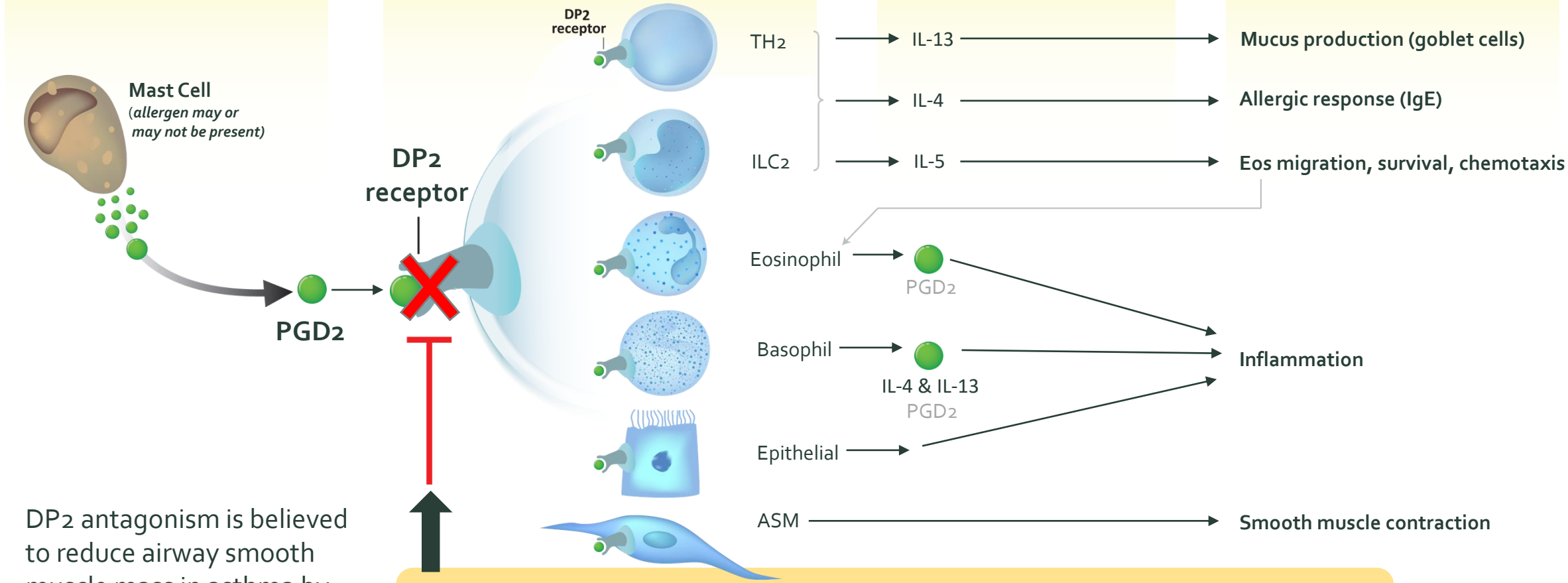
Role and Biology of the PGD2/DP2 Pathway in Type 2 Inflammation

GB001

1. PGD2 released by mast cells…
2. …binds to the DP2 receptor, leading to…
3. …immune cell recruitment and activation,…
4. …immune response and asthma symptoms.



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

Treatment with GB001, an oral DP2 antagonist, blocks 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 Has Demonstrated an Effect on FeNO

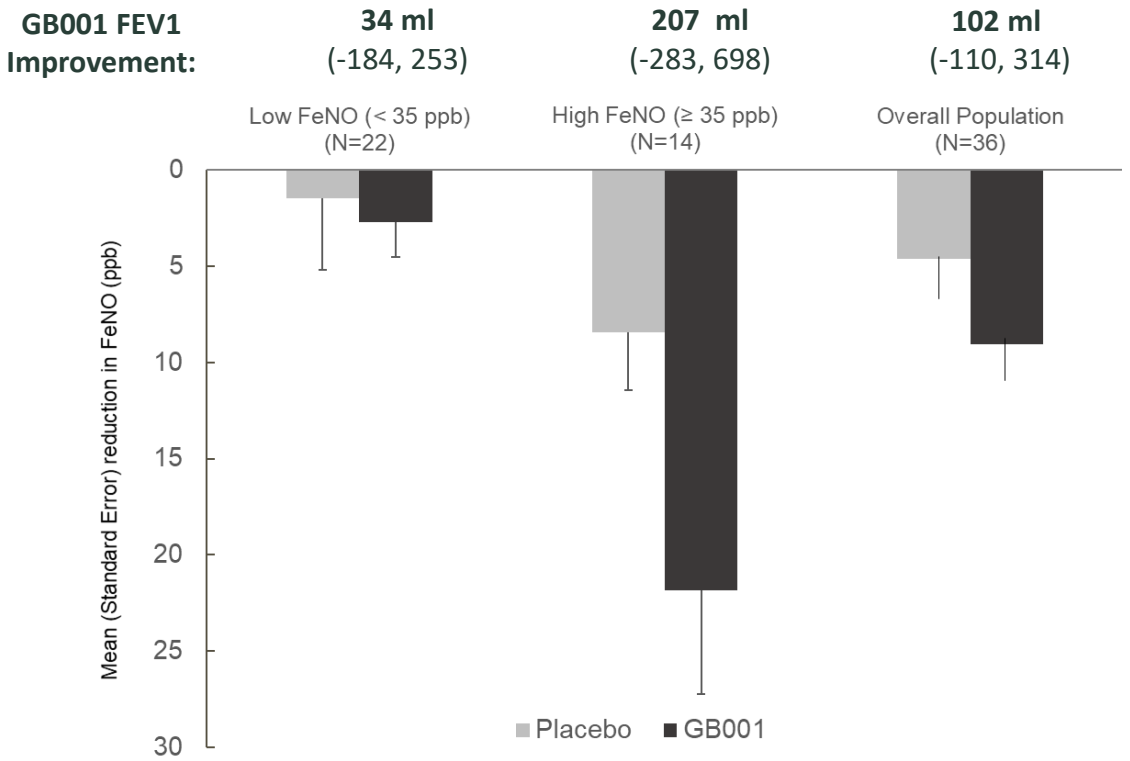
Reduction of Exhaled Nitric Oxide by the DP2 antagonist GB001

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)

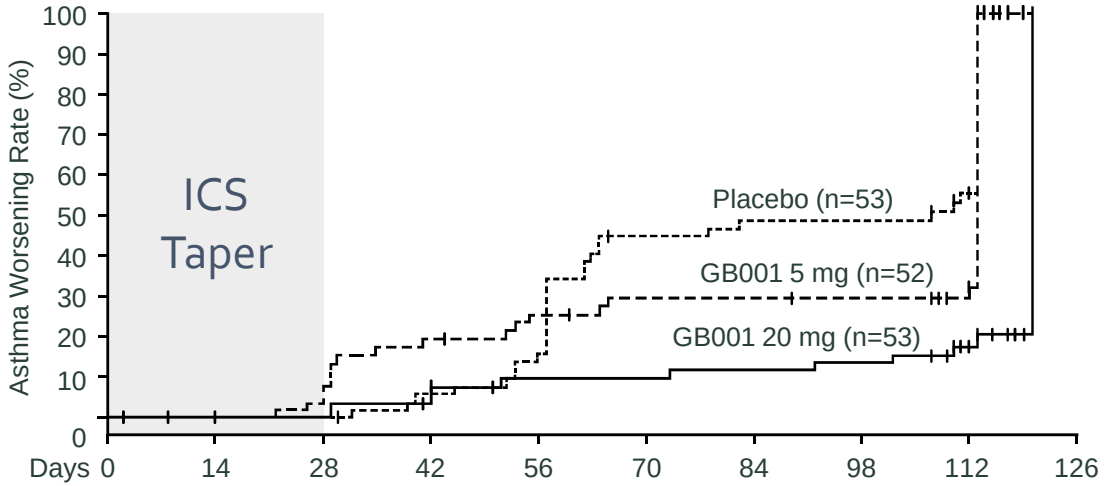
1) Ortega H, et al. Clin Exp Allergy (in press).
2) Wenzel, Sally et al. "Dupilumab in Persistent Asthma with Elevated Eosinophil Levels." NEJM 368 (2013): 2455 – 2466.
3) Castro, Mario et al. "Dupilumab Efficacy and Safety in Moderate-to-Severe Uncontrolled Asthma." NEJM 378 (2018): 2486 – 2496.
FeNO = fractional exhaled nitric oxide; ppb = parts per billion.

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

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

Overall Population

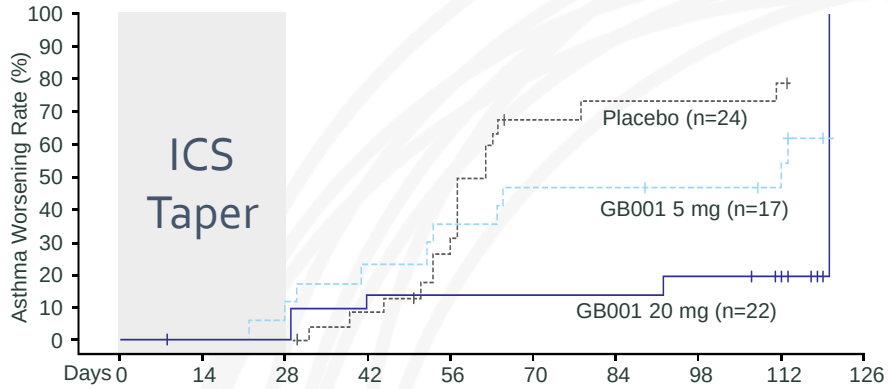
71% reduction in the risk of asthma worsening for 20mg GB0001 group



	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)

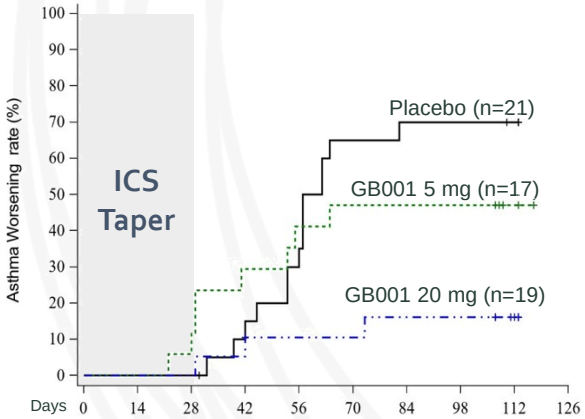
High Eosinophil Population (≥300 µL)

84% reduction in the risk of asthma worsening for 20mg GB0001 group



High FeNO Population (≥25 ppb)

84% reduction in the risk of asthma worsening for 20mg GB0001 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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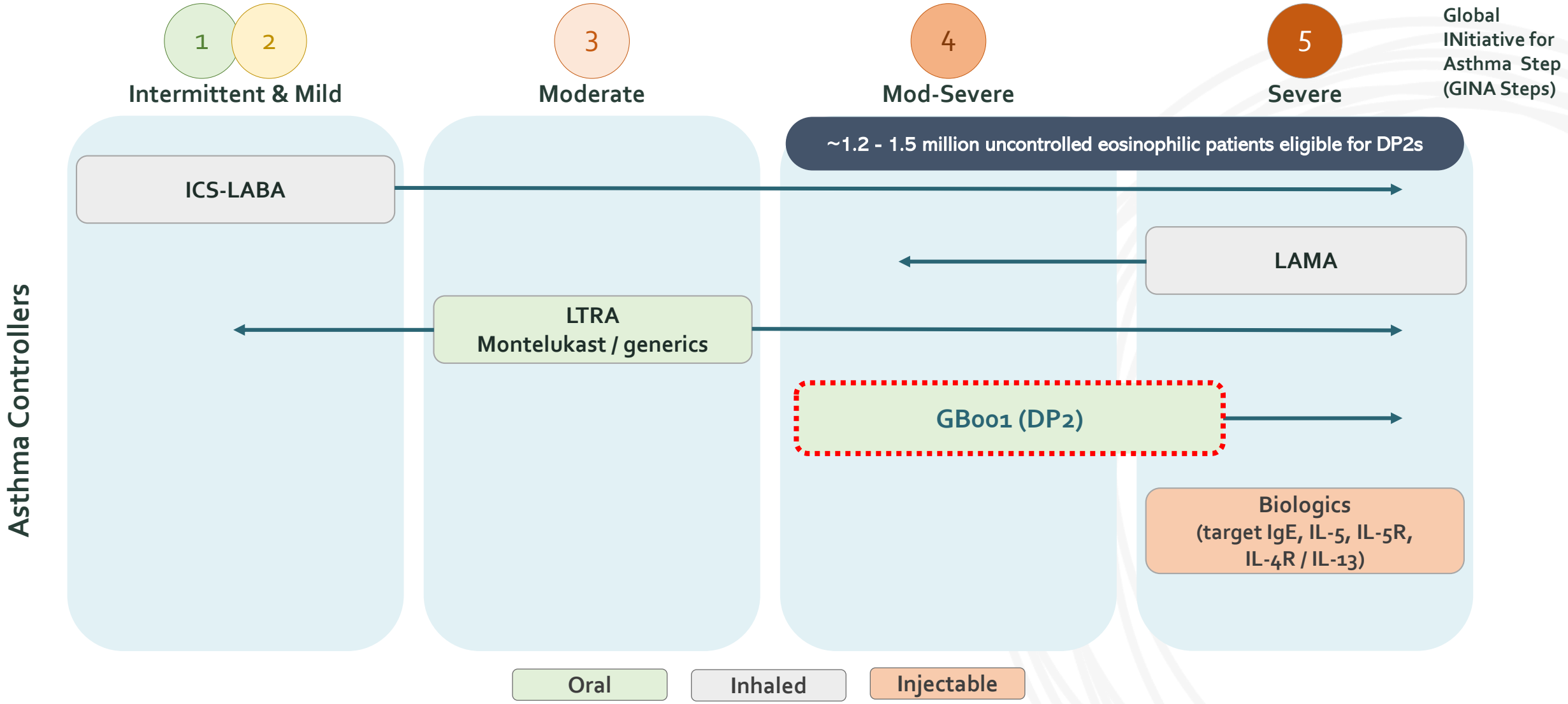
Potential for Market Asthma Positioning Prior to Biologics

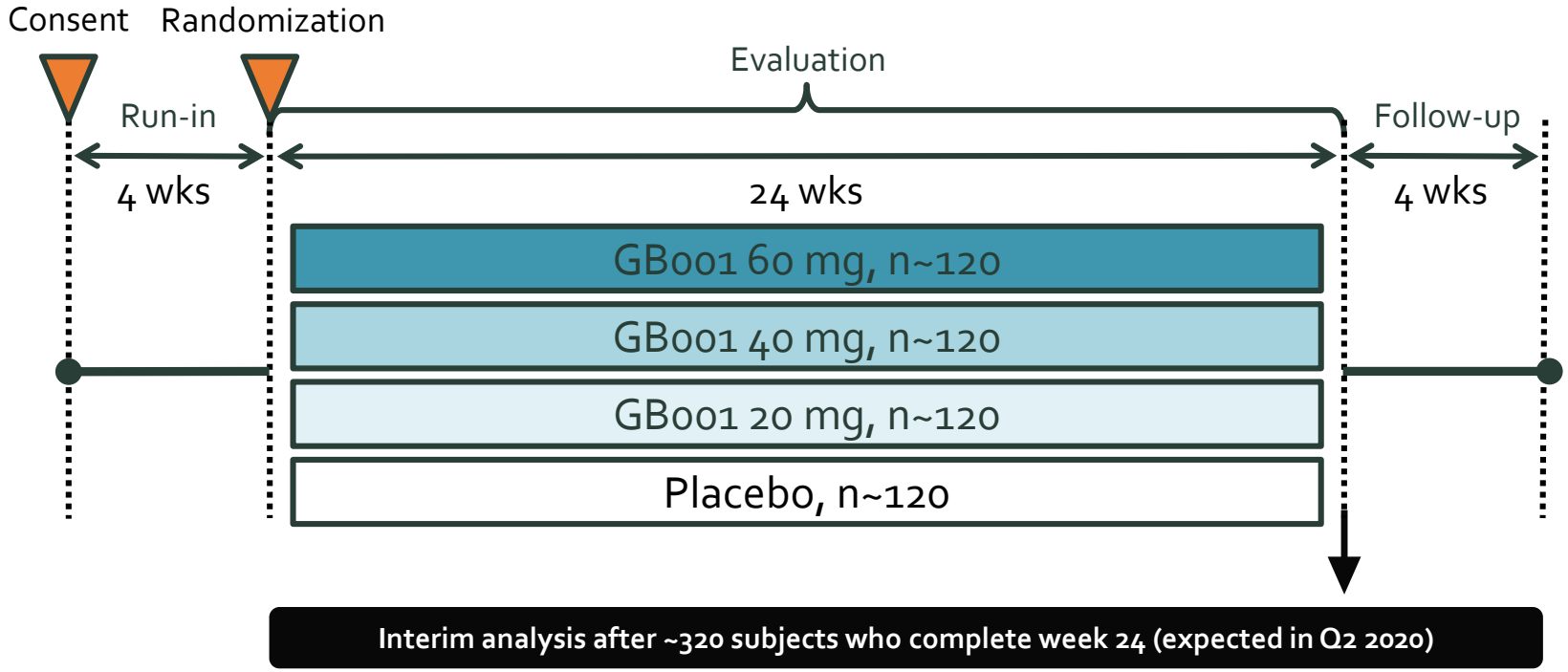


ICS = inhaled corticosteroids; LABA = long-acting beta agonist; LAMA = long-acting muscarinic antagonist; LTRA = leukotriene receptor antagonist.
Source: GINA 2019 guidelines, Datamonitor, open source medical claims, internal analysis.

LEDA Study: Phase 2b Study Design Allows for Efficient Transition to Phase 3

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:** FEV1, asthma control, asthma quality of life

QD = once daily dosing; wks = weeks.

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

GB001

CRS Affects 10 – 15% of the population (>25 million US adults)

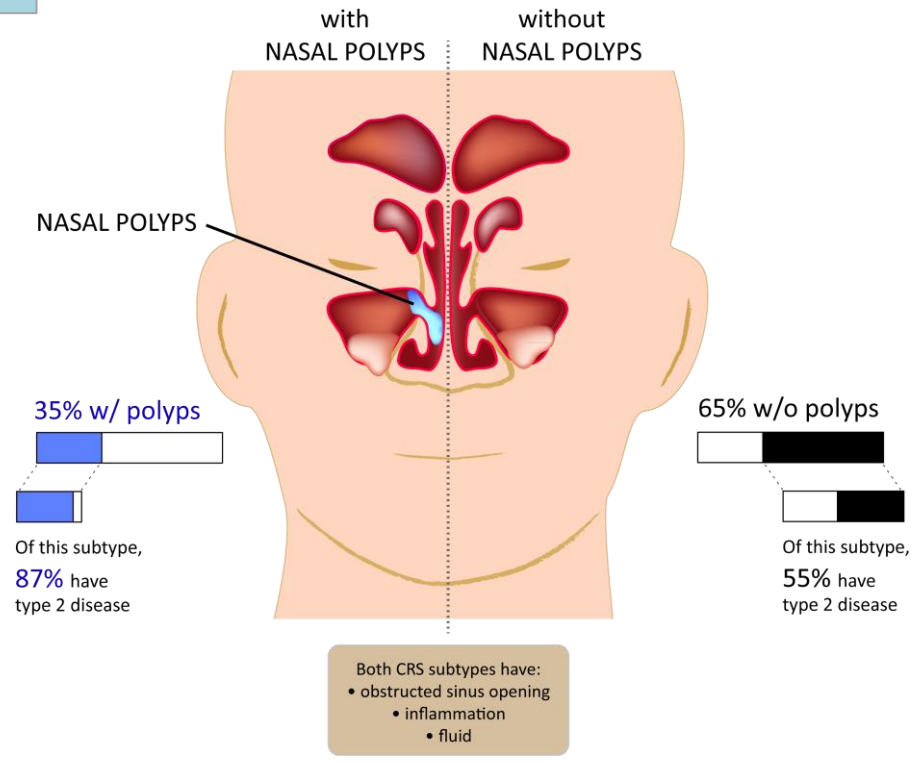
Eosinophilic Inflammation is a Major Pathologic Hallmark of CRS

- 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



Notes: Small subset of 8-12% is allergic fungal disease.

Sources: CDC
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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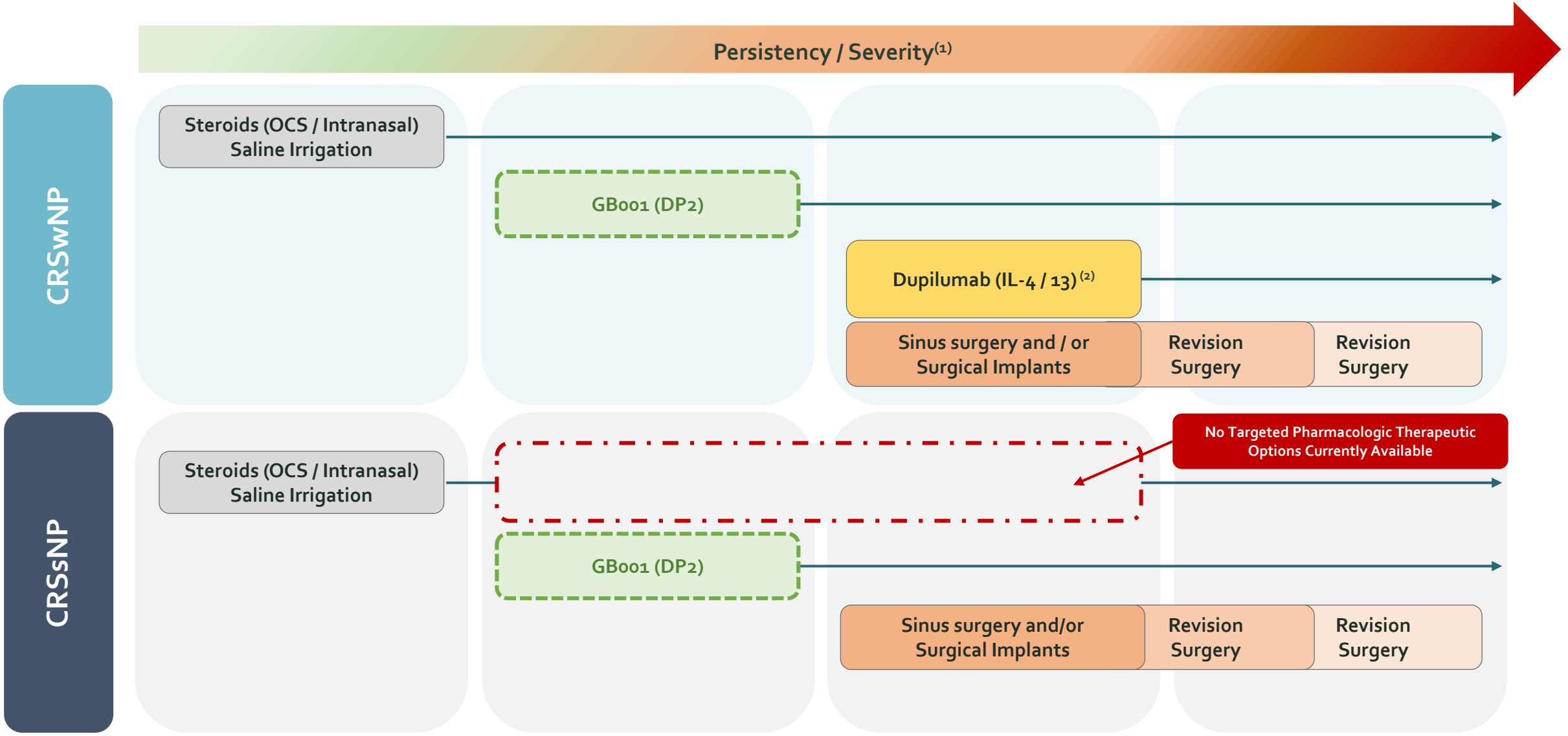
GB001 is Positioned to be the Only Oral Therapy Available for Severe CRS Patients With and Without Nasal Polyps



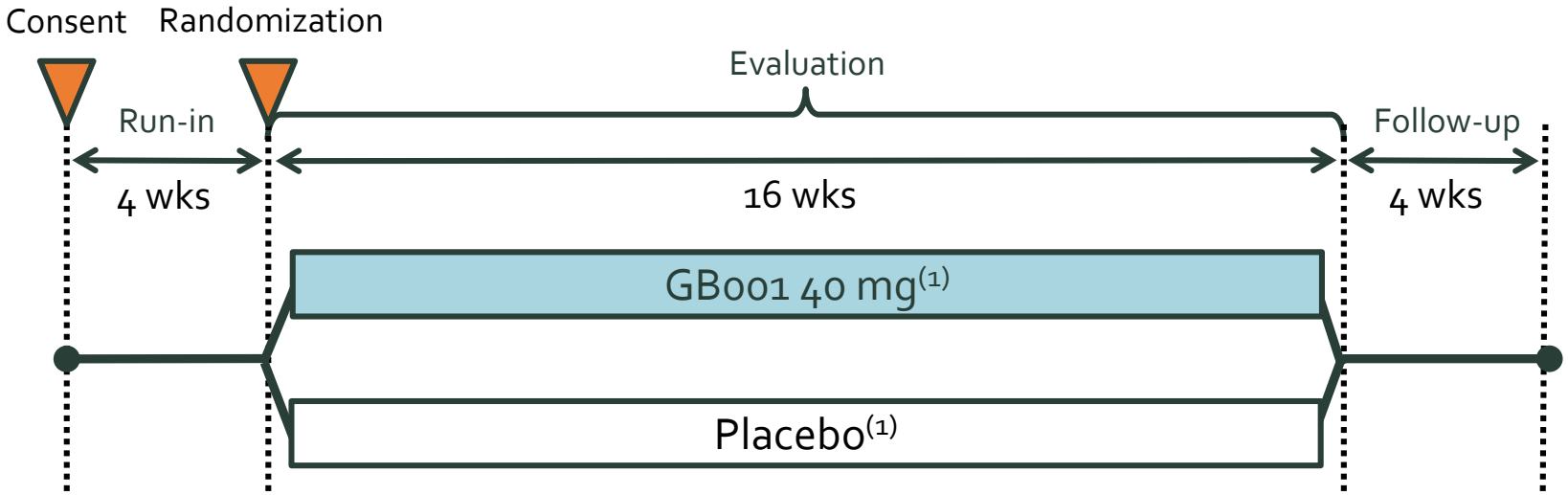
1) Excludes antifungals and antibiotics for infectious disease.
2) Multiple biologics in late stage development, including omalizumab (anti-IgE), mepolizumab (anti-IL-5), and benralizumab (anti-IL5R).
NP = nasal polyps; OCS = oral corticosteroids.

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

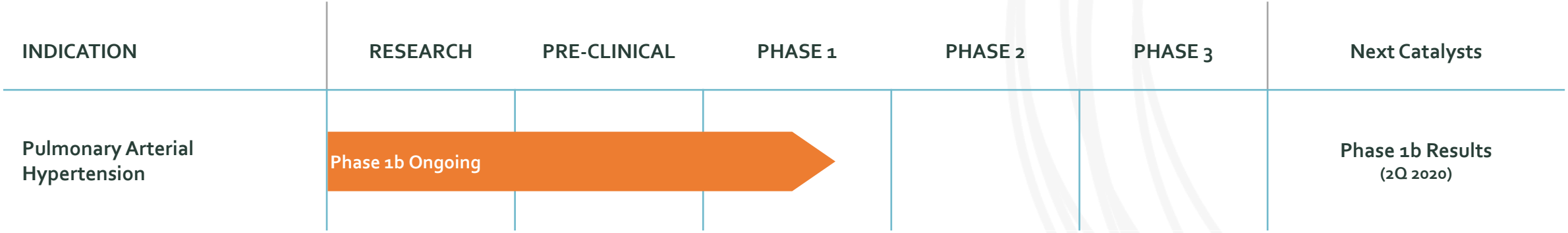
Pulmonary Arterial Hypertension (PAH)

GB002: Potential To Be the First Treatment for PAH with Disease-Modifying Effects

GB002

Product Description

- Selective, inhaled PDGF receptor kinase inhibitor 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
- Inhaled GB002 deposits 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

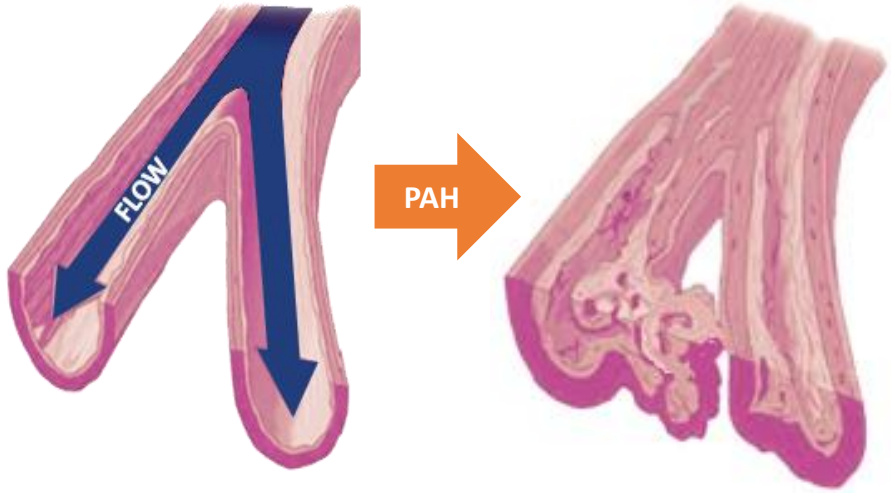
Unabated Cellular Proliferation with Limited Treatment Options

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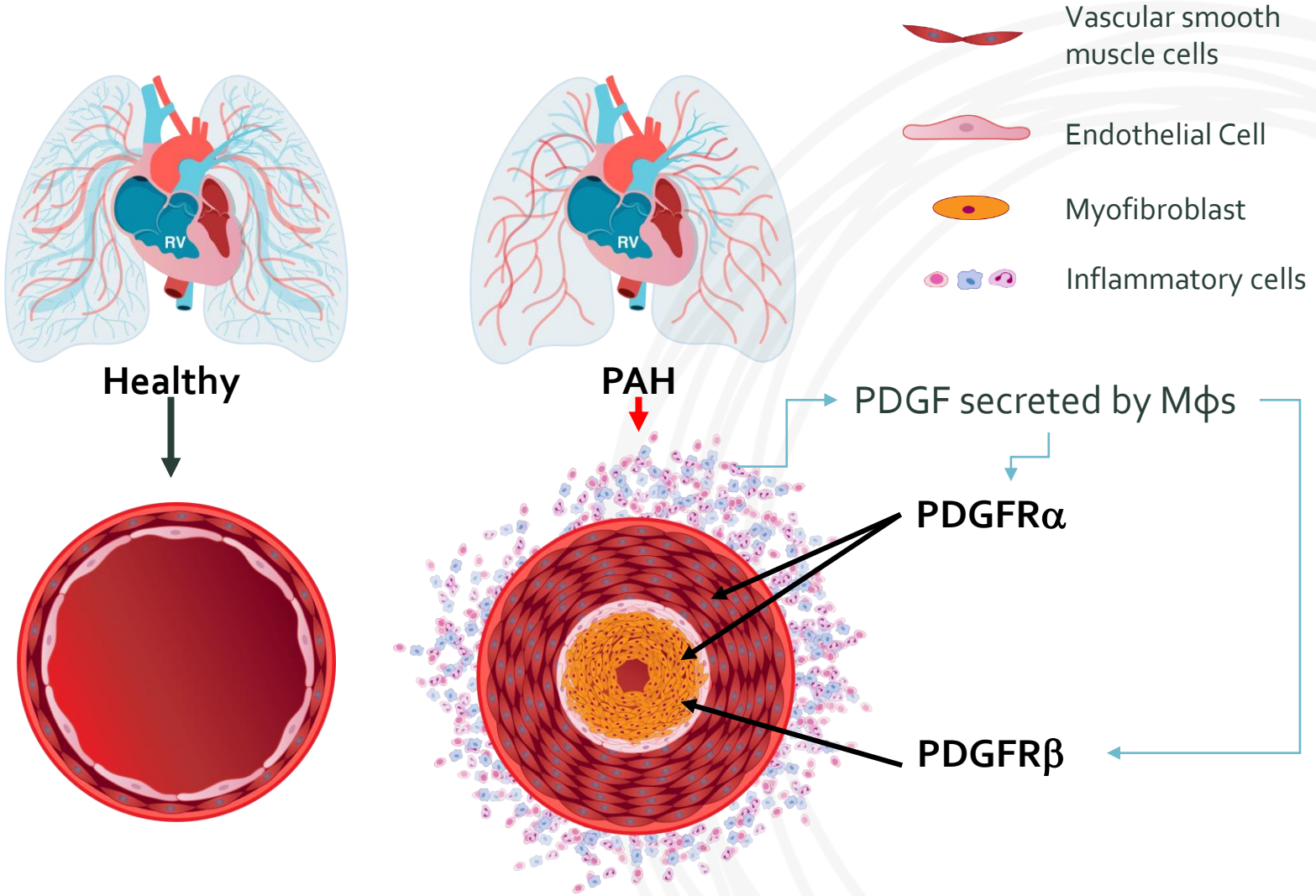
PDGFR Activation Plays a Role in the Pathological Remodeling of Lung Blood Vessels in PAH

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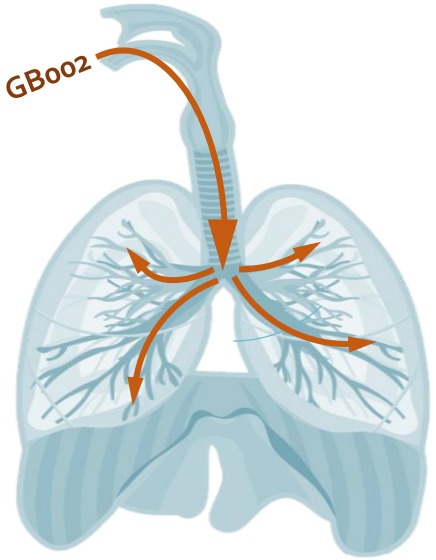
GB002 Administration via Dry Powder Inhaler is Convenient and Delivers Drug Directly to the Site of Disease

GB002

Dry Powder Inhaler from Plastiape



- GB002 formulation and administration via dry powder inhaler (DPI) designed to reach areas of the deep lung
- Inhaled GB002 deposits at site of disease due to proximity of terminal bronchiole and alveolar space to affected pulmonary arteries
- Results in higher ratio of lung to systemic exposure, potentially providing for an improved therapeutic index
- DPI device is small, convenient and currently used in commercial products

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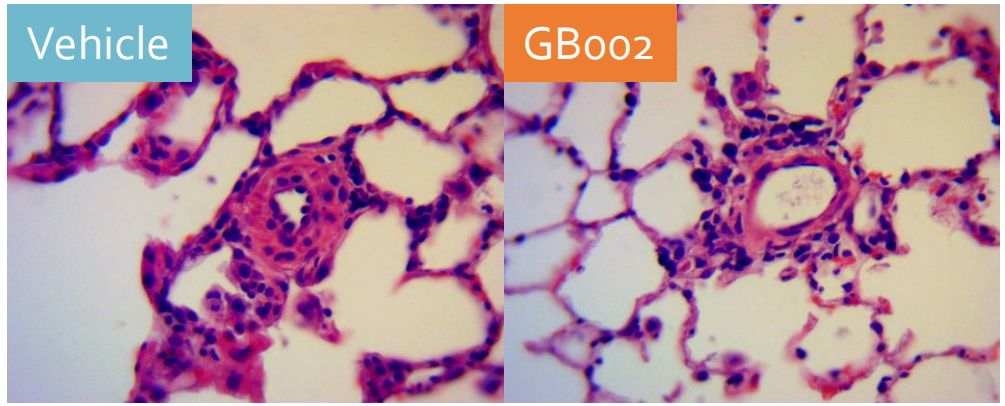
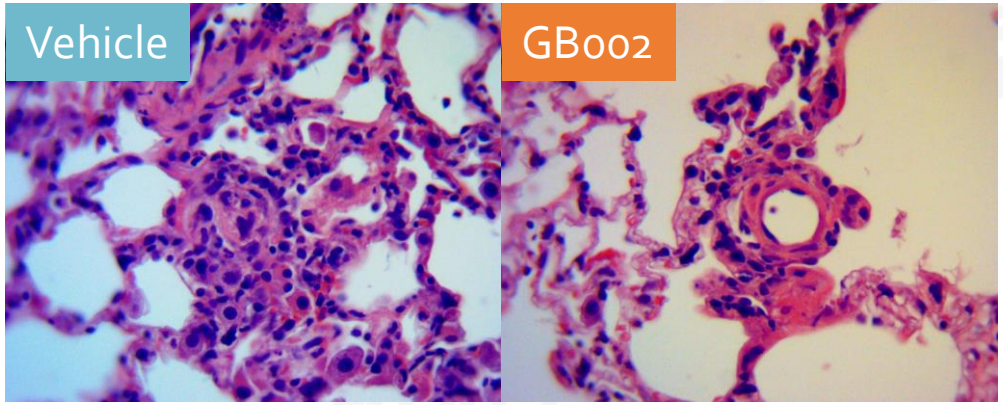
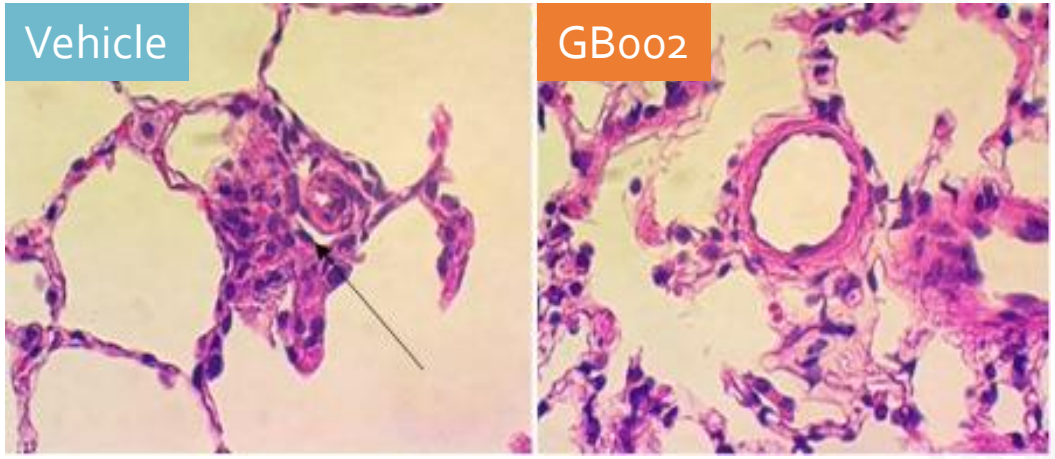
GB002 Reverses Vascular Remodeling Through Inhibition of PDGFR in Animal Models of PAH

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





GB002 Restores 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.

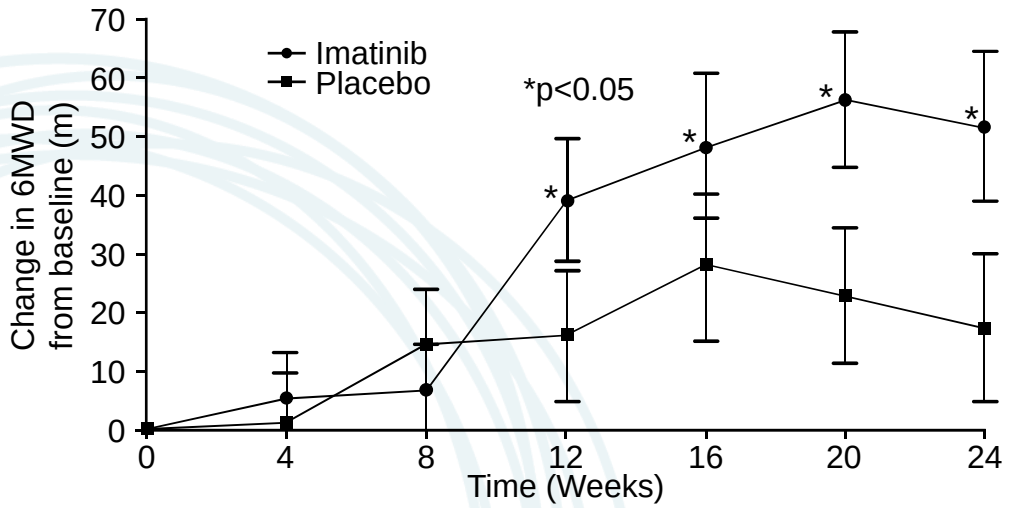
Imatinib Demonstrated Clinical Proof of Principle for Targeting PDGF in PAH in the Phase 3 IMPRES Trial

GB002

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

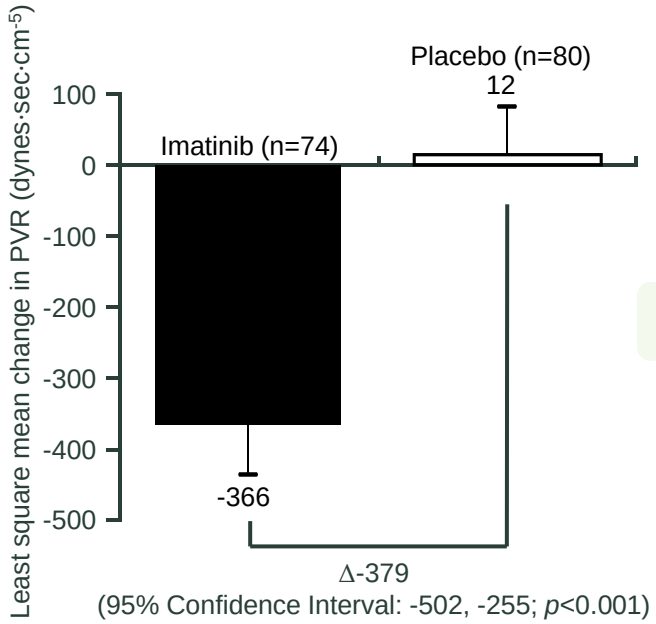
- Imatinib mesylate, as add-on therapy in PAH patients who remain inadequately treated on at least two PAH-specific drugs, improves exercise capacity and hemodynamics
- Of 202 patients enrolled, 41% had failed three classes of therapies, ~70% were on prostacyclins
- Serious Adverse Events: 44% imatinib 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

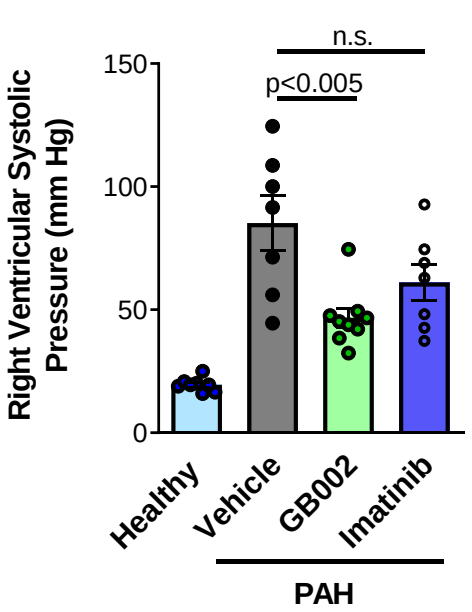
GB002 is a Potent PDGFRα/β Inhibitor with Limited Systemic Exposures

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

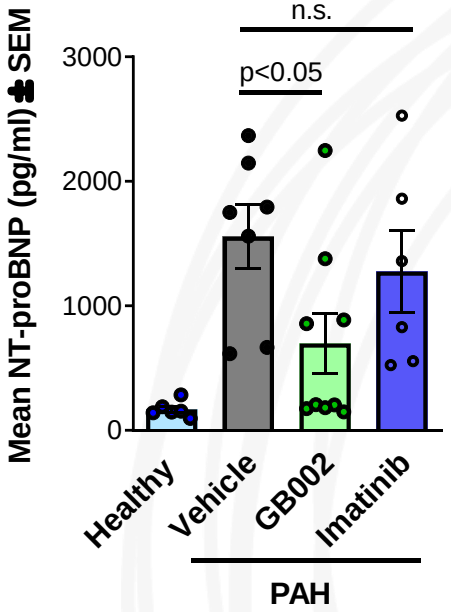
Imatinib in vivo exposures matched to published clinical exposures at the 400 mg dose

GB002 is a new chemical entity

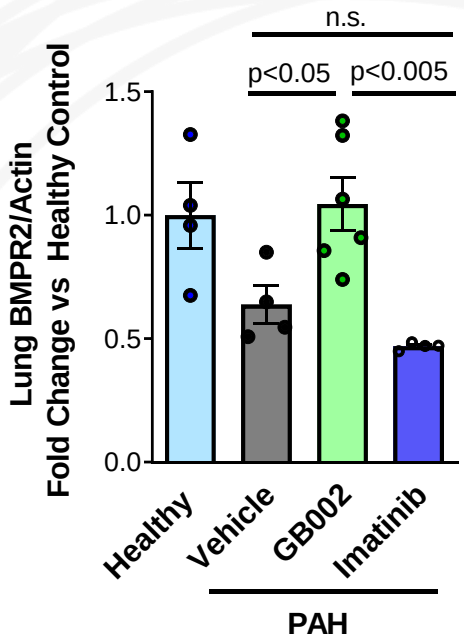
GB002 Demonstrates Greater Efficacy Than Imatinib and Restores BMPR2 Expression in the Established rat SU5416 / Hypoxia PAH Model



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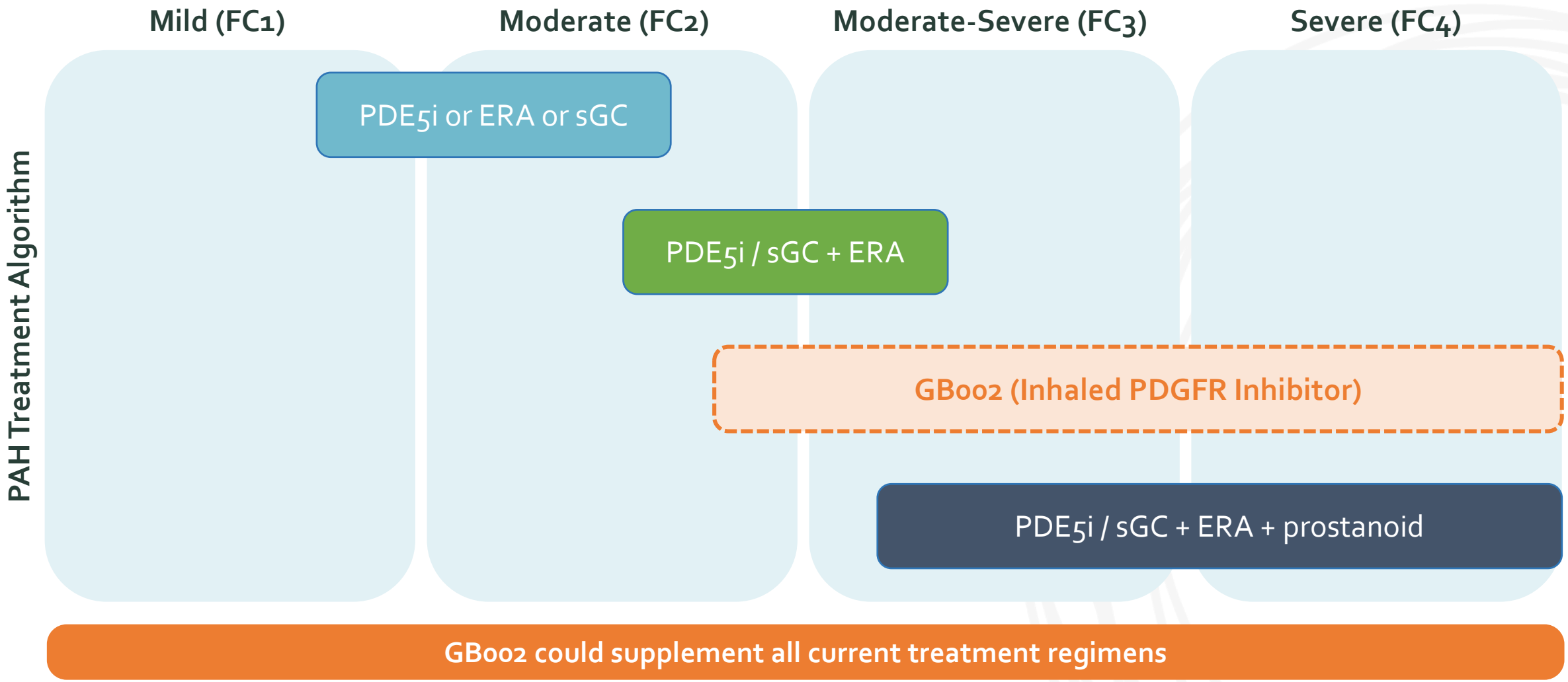
The PAH Treatment Paradigm Increasingly Supports Addition of Therapies as Severity and Functional Class Increase

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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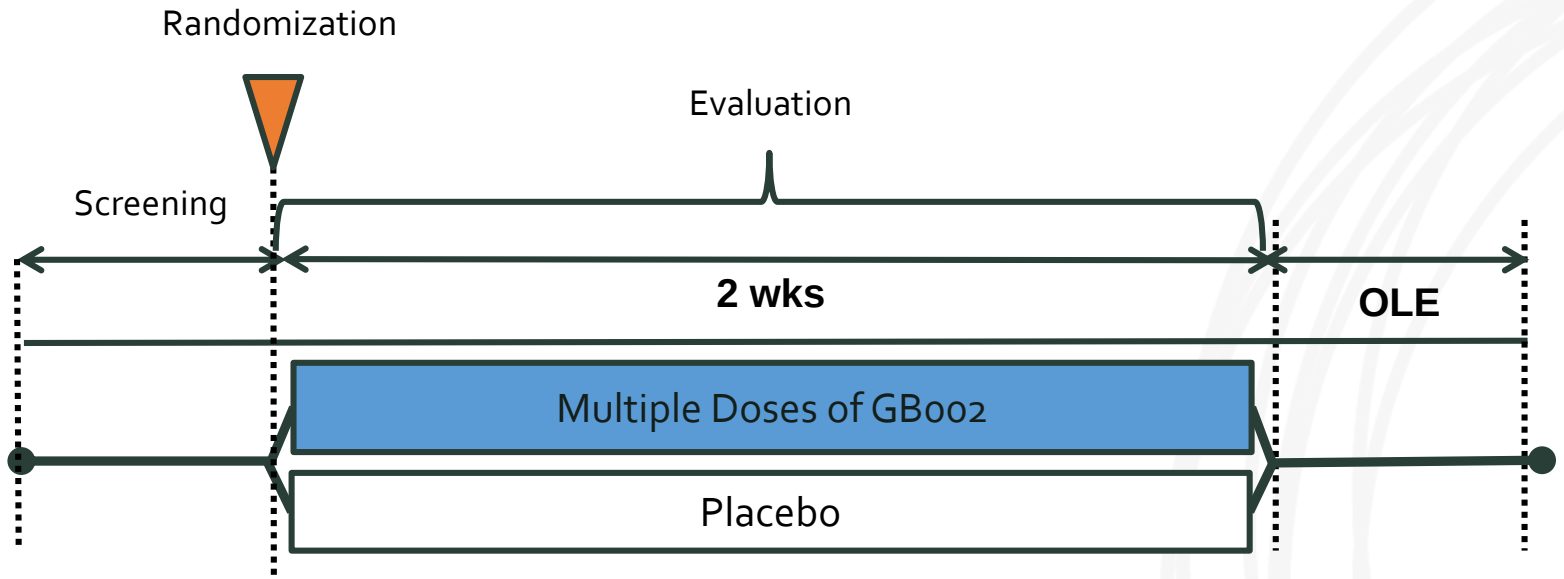
Ongoing Phase 1b Study in Pulmonary Arterial Hypertension

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

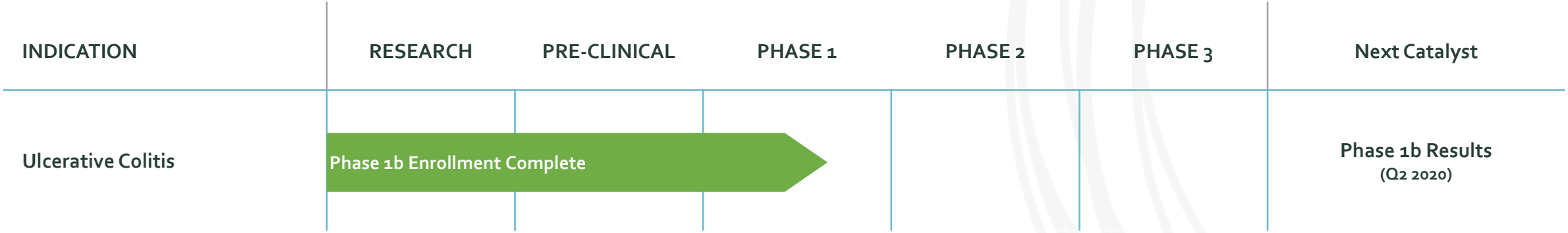
Inflammatory Bowel Disease (IBD), including Ulcerative Colitis (UC) and Crohn's Disease (CD)

GB004: Gut-targeted, HIF Stabilizer with Potential to Deliver Superior Efficacy in IBD

GB004

Product Description

- Oral, small molecule, gut-targeted, prolyl hydroxylase inhibitor that stabilizes 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
- Designed leveraging the Nobel Prize winning science studying the impact of oxygen regulation
- Potential for use as mono or combo therapy for IBD
- 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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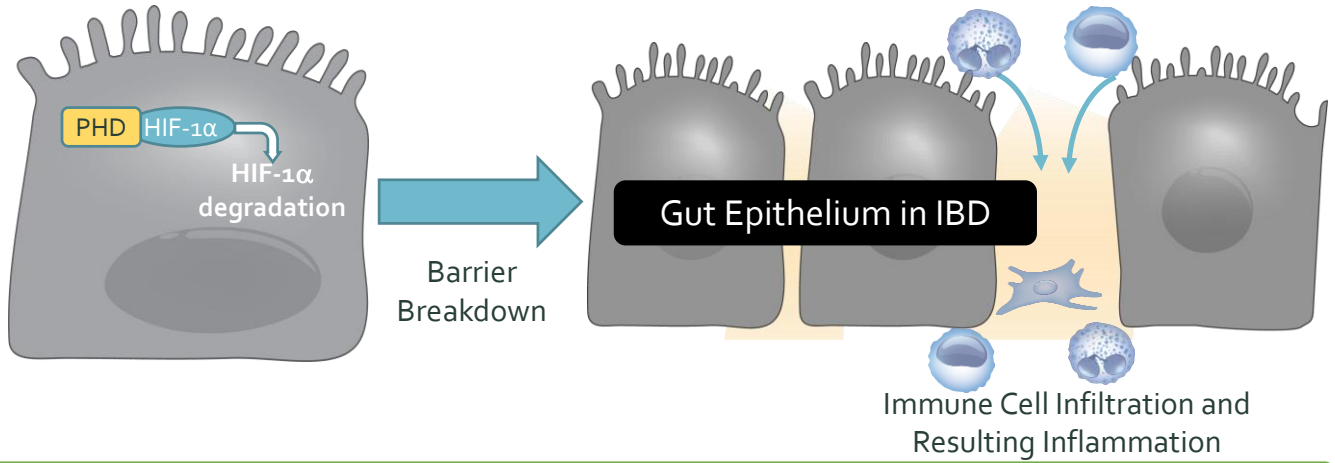
Mechanism of PHD Inhibitor to Restore Epithelial Barrier Function in IBD

GB004

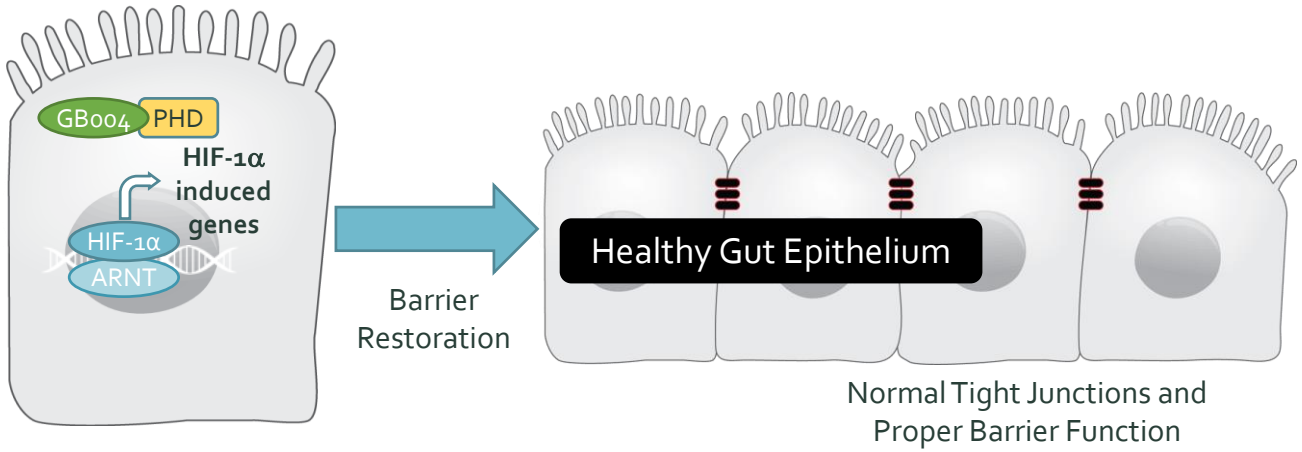
Epithelial Barrier State in IBD (↑Hypoxia ↓HIF-1α ↓Protective Genes ↑Barrier Disruption)

- Patients with IBD experience increased hypoxia throughout their mucosa and dysregulation of hypoxia induced transcription factor (HIF)
- HIF-1α is degraded by prolyl hydroxylase, leading to decreased expression of protective genes and an increase in barrier apoptosis and disruption
- The disrupted epithelial barrier allows for the entry and re-entry of microbes and inflammatory immune cells, leading to chronic disease remission



Healthy Epithelial Barrier (↓Hypoxia ↑HIF-1α ↑Protective Genes ↓Barrier Disruption)

- GB004 inhibits PHD-induced degradation of HIF-1α
- Stabilization of HIF-1α results in accumulation and translocation to the nucleus where HIF-1α drives protective gene expression in the epithelium
- HIF-1α-mediated protective pathways (such as TFF3, CD73, MDR1) are critical for barrier integrity and function
- Emerging data suggests important role of oxygen regulation in IBD control; significantly decreased oxygen levels have been associated with inflamed mucosa in UC patients



Source:
Fraser, Gerald et al. "Six patients whose perianal and ileocolic Crohn's disease improved in the Dead Sea environment." Journal of Clinical Gastroenterology 21, no. 3 (1995): 217 – 219.
Dulai, Parambir et al. "Systematic review: The safety and efficacy of hyperbaric oxygen therapy for inflammatory bowel disease." Alimentary Pharmacology & Therapeutics 39, no. 11 (2014): 1266 – 1275.
Dulai, Parambir et al. "Hyperbaric oxygen therapy is well tolerated and effective for ulcerative colitis patients hospitalized for moderate-severe flares: a phase 2A pilot multi-center, randomized, double-blind, sham-controlled trial." American Journal of Gastroenterology 113, no. 10 (2018): 1516 – 1523.
Brown, Eric et al. "Inflammation-dependent transcriptional re-programming of the HIF pathway in the mucosa of ulcerative colitis patients ." Gastroenterology, 156, no. 6 (2019), S-606.

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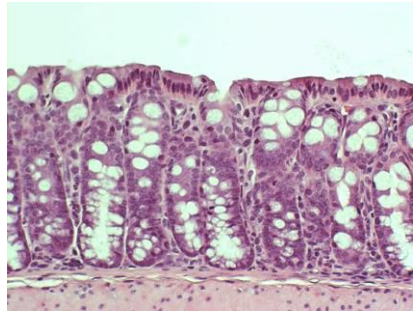
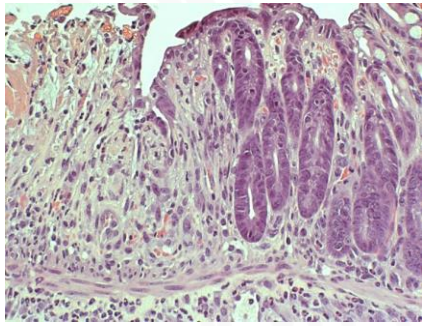
Oral GB004 Reconstitutes the Epithelial Barrier and Improves Mucosal Healing

GB004

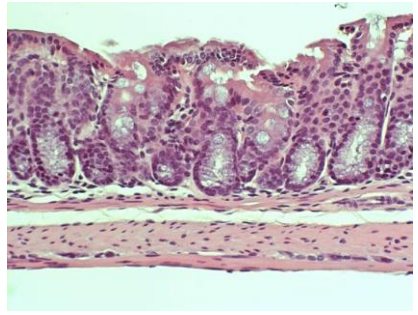
Naive



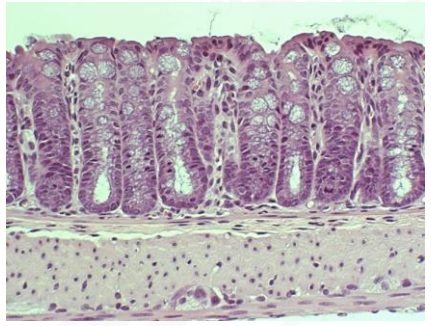
Vehicle



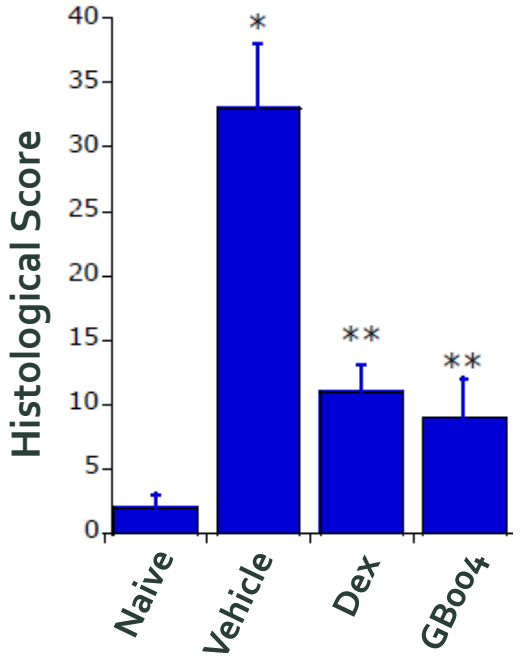
Dexamethasone



GB004 10mg/kg

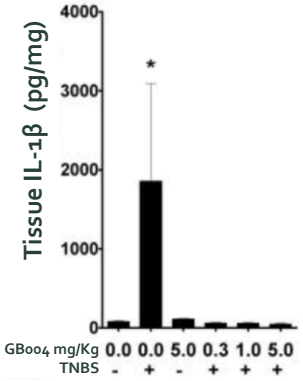


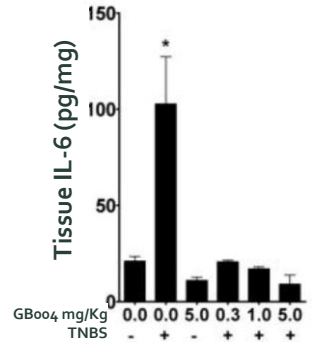
Histological Score Improvement in TNBS-Induced Colitis Model

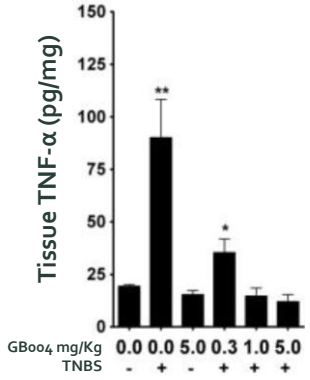


* p<0.01 compared to all other groups
** p<0.025 compared to placebo treated TNBS animals

GB004 Reduces 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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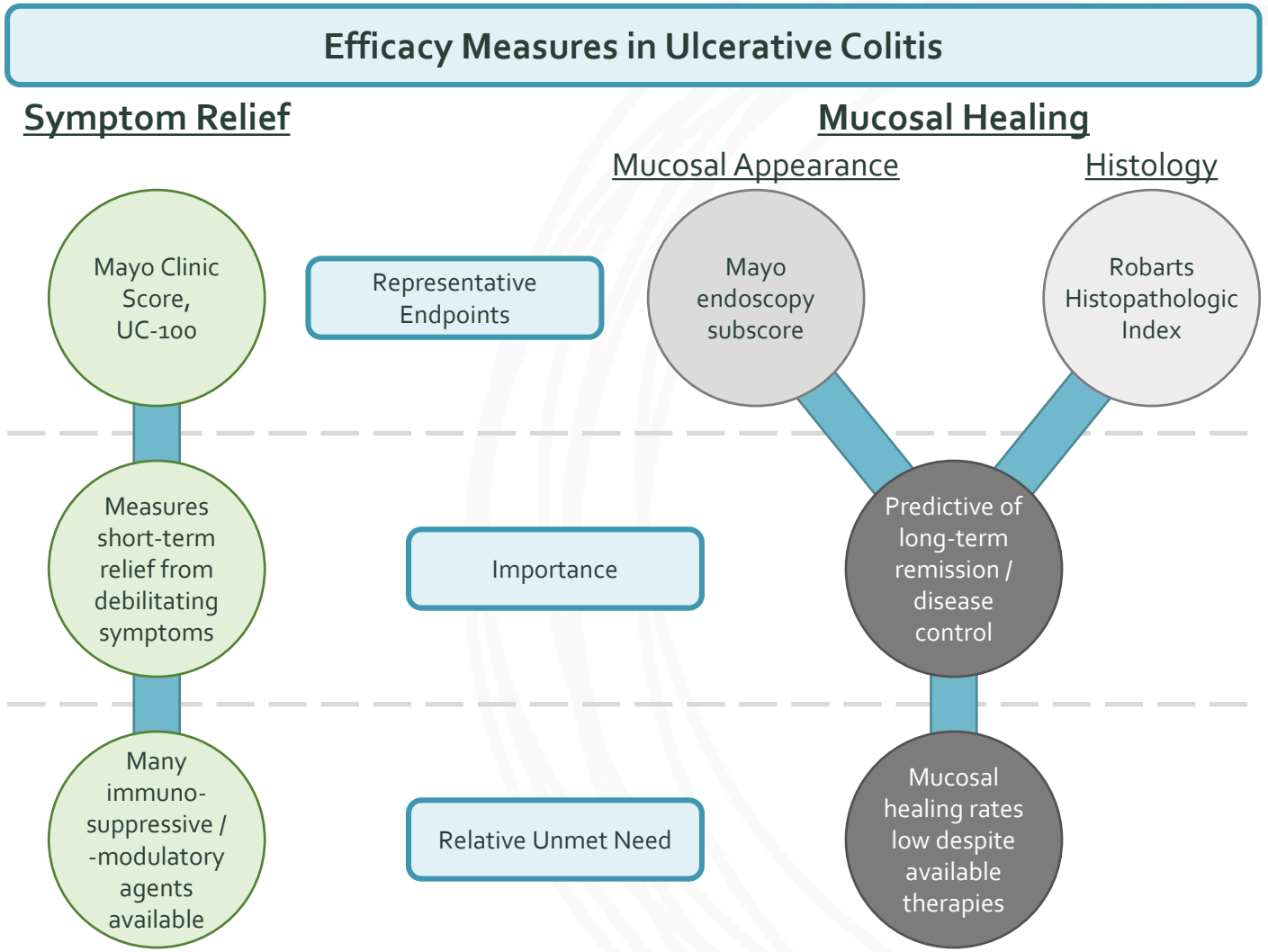
Improvement in Histology is Emerging as a Key Therapeutic Target in UC

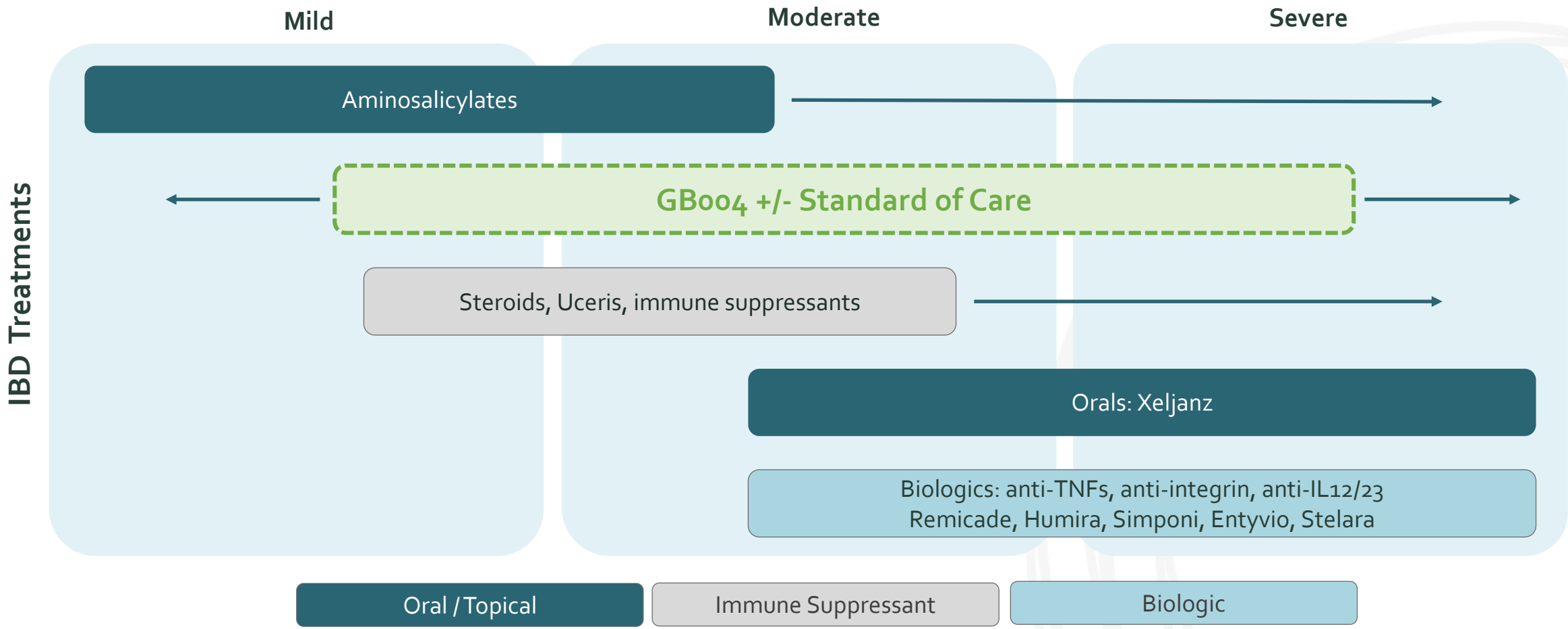
GB004



- Historically, endoscopy alone had been used to evaluate "mucosal healing"
  - Following pushback from KOLs and the FDA, histology has emerged as an important third therapeutic target and second component of mucosal healing
  - While endoscopy assesses mucosal improvement at the tissue level, histology magnifies to the cellular level
- Both improvement of mucosal appearance and improvement in histology have been associated with reduced disease relapse, hospitalizations, and corticosteroid use
  - Evidence suggests histology may be a more predictive measure[1]
- Multiple histologic scoring systems have been validated, including the Robarts Histopathological Index (RHI), Geboes Score (GS), and the Nancy Index (NI)
- Patients in Gossamer's Phase 1b study of GB004 in UC were required to have active disease as evaluated by RHI
- RHI scores range from 0 to 33 and are calculated examining 4 components: chronic inflammatory cell infiltrate, lamina propria neutrophils, neutrophils in epithelium, and erosions or ulceration

1) Pai, Rish et. al. "The emerging role of histologic disease activity assessment in ulcerative colitis." Gastrointestinal Endoscopy, Volume 88, Issue 6, 887 - 898

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



GB004 is well suited as a pre-biologic therapy across the spectrum of disease activity segment as monotherapy or in combination

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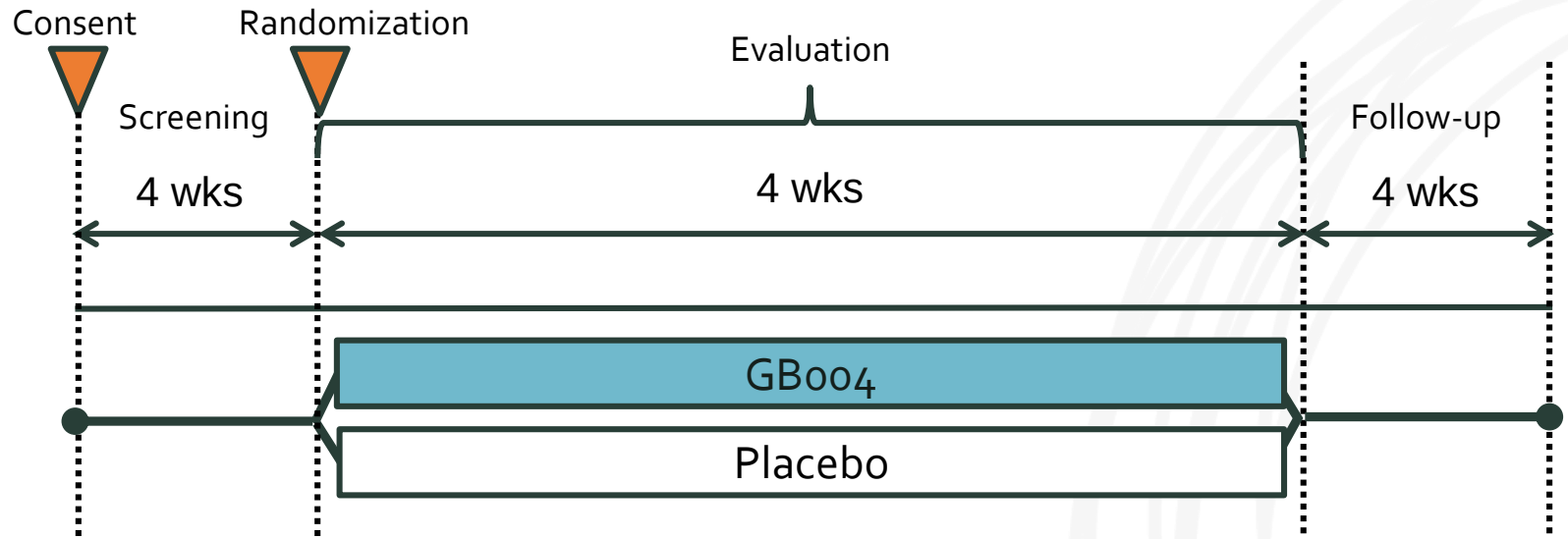
GB004 Phase 1b in Ulcerative Colitis to Allow for PK Assessment in Patients and Potential Initial Assessment of Biological Effect

GB004

A Phase 1b, signal seeking, placebo-controlled, dose-ranging 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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GB1275

CD11b Modulator

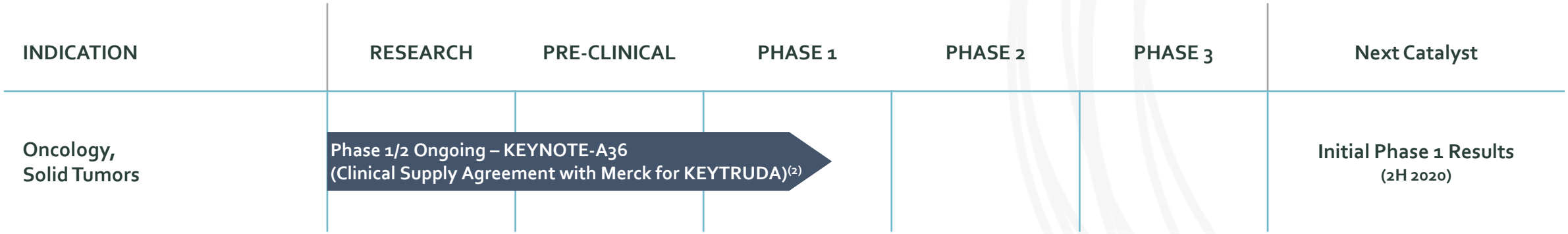
Solid Tumors

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

GB1275

Product Description

- Oral, small molecule, first-in-clinic CD11b modulator for the treatment of solid tumors
- Disrupts multiple immunosuppressive myeloid cell subsets, including MDSCs and TAMs
- PC efficacy 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 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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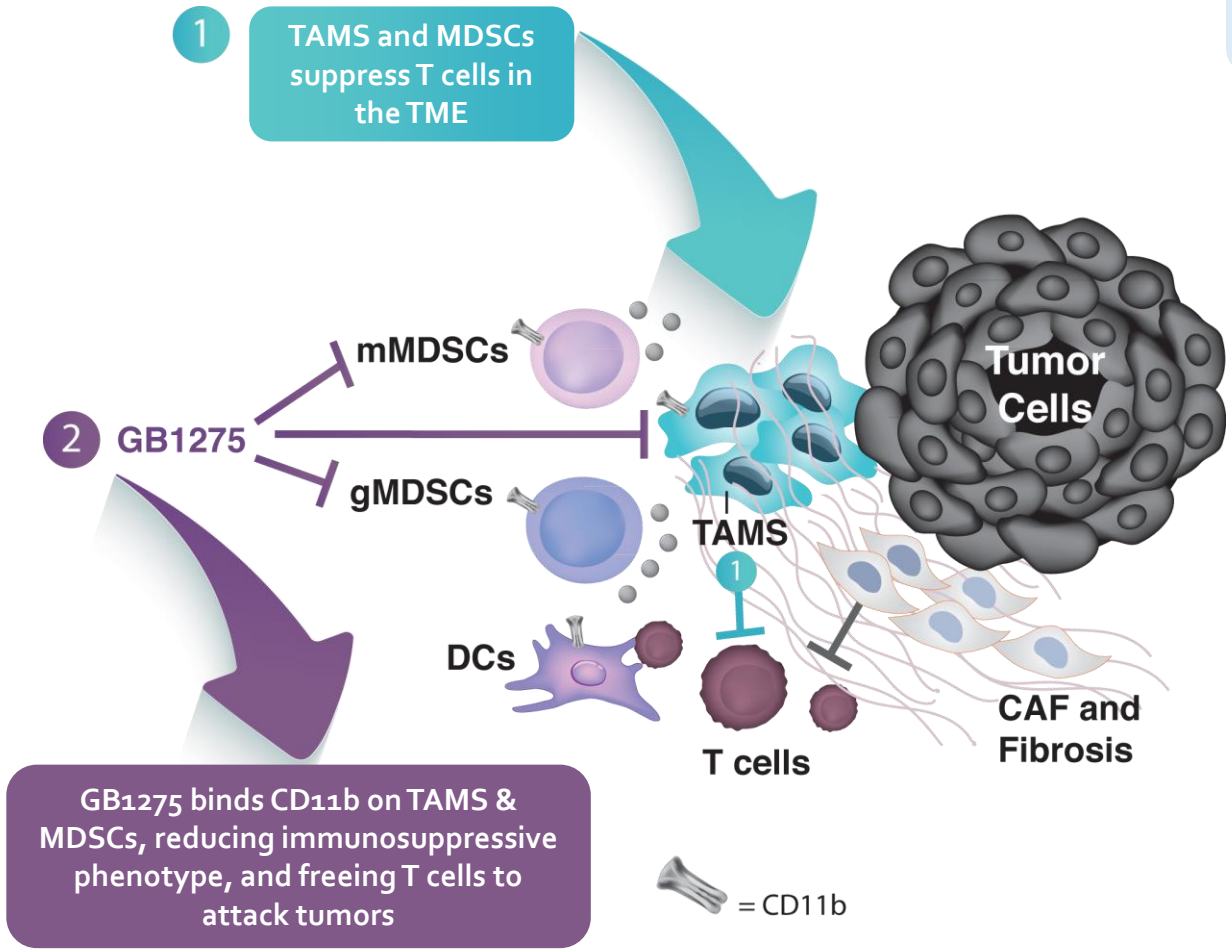
MDSCs and M2 Macrophages Generate an Immunosuppressive Tumor Microenvironment and Hinder Response to IO Agents



GB1275 is an Allosteric Modulator of CD11b, which Impacts 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 which repolarizes myeloid suppressive cells (mMDSCs & gMDSCs) and TAMs, reducing their immunosuppressive roles and allowing T cells to attack tumors
- **GB1275** also prevents migration of additional CD11b+ monocytes to the tumor microenvironment

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

GB1275 Offers Unique & Complementary Opportunity to Inhibit Immunosuppressive Myeloid Cell Types

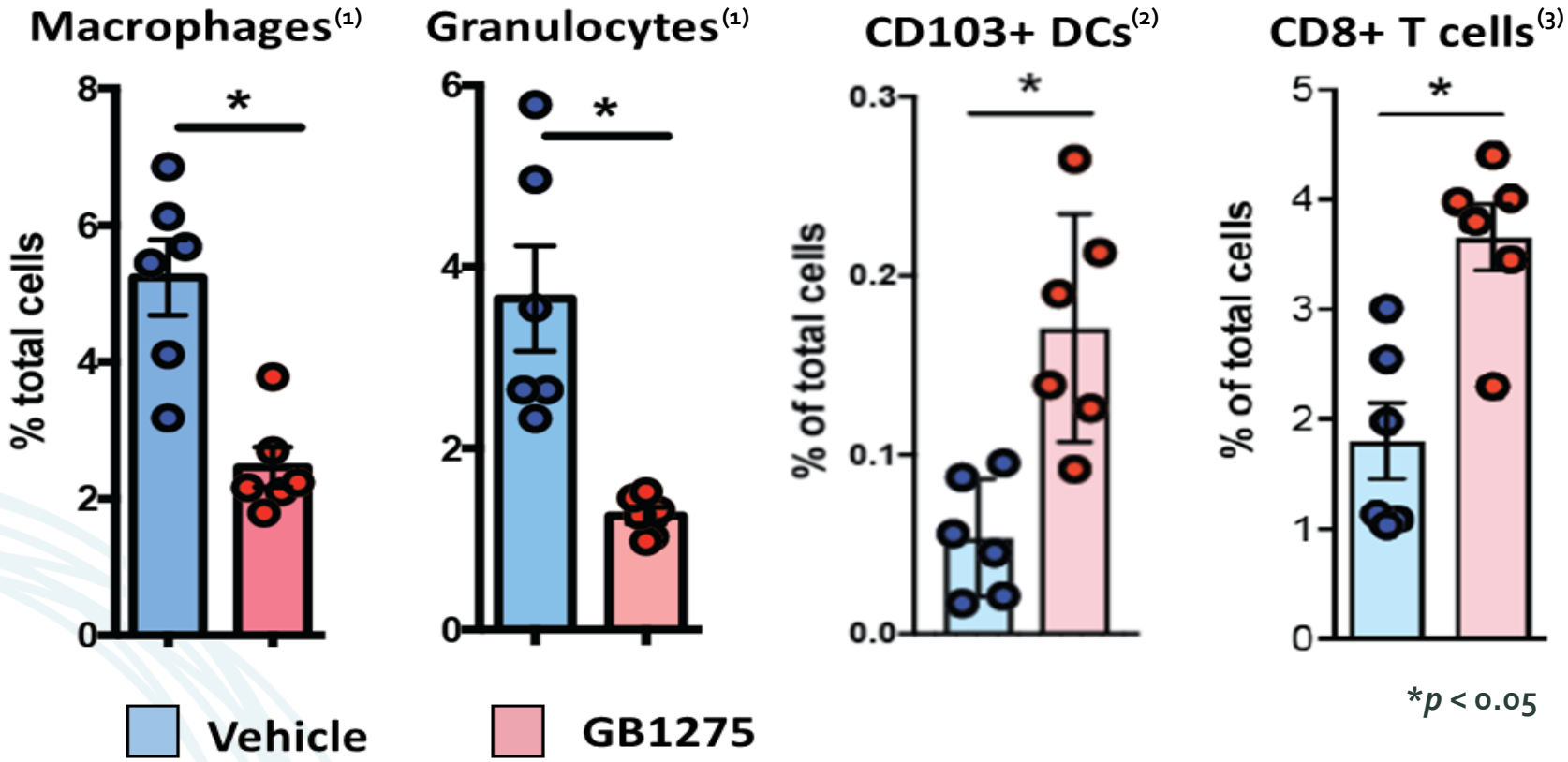
GB1275

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]			✓	

- CCR2 inhibitors may be limited by compensatory recruitment of gMDSCs
- GB1275 blocks gMDSC and mMDSC cell recruitment to the tumor site and blocks M2 polarization
- GB1275 MDSC/TAM modulation effects are tumor-localized, without impact on myeloid cells in the periphery, 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, Markus 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.

GB1275 Reduces Tumor Infiltration of Myeloid Cells and Increases Influx of Activated CD8+ T cells



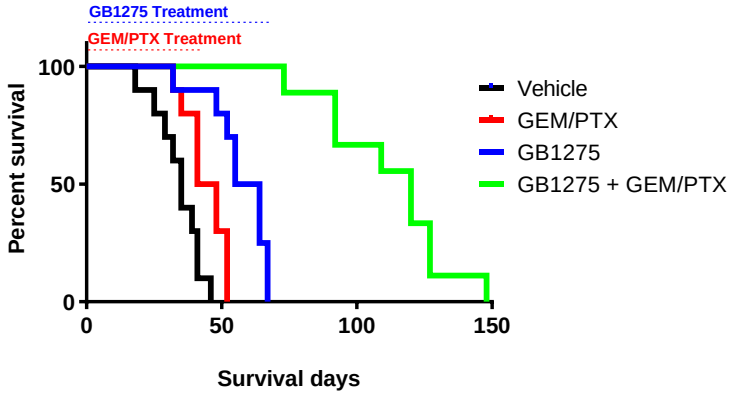
- GB1275 immuno-modulatory effects were tumor-localized
- GB1275 did not alter immune cell phenotypes in the periphery, spleen or bone marrow

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).

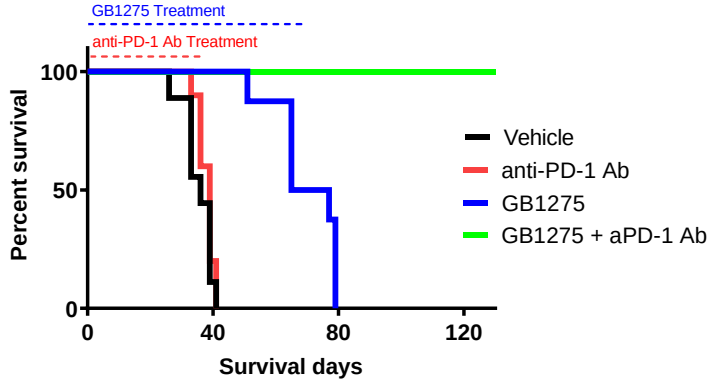
GB1275 Single Agent or in Combination Improves Efficacy and Survival Outcomes in Multiple Difficult-to-Treat Tumor Models

GB1275 Single Agent or in Combo with SoC or anti-PD-1 Ab Treatment Improves Efficacy and Survival Outcomes in the Pancreatic Adenocarcinoma Mouse Tumor Models(1)

GB1275 in Combination with Chemotherapy Extends Survival in the PDAC Model

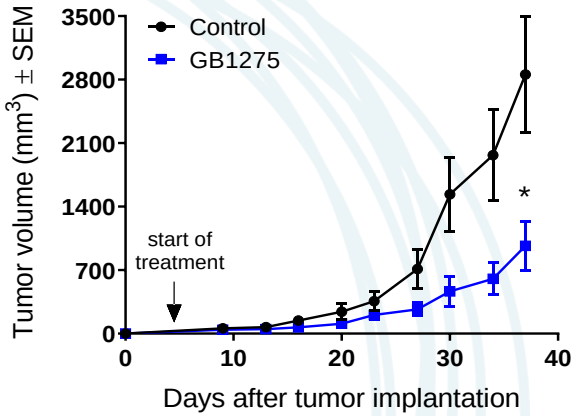


GB1275 Sensitizes PDAC Model to anti-PD-1 Ab Checkpoint Blockade

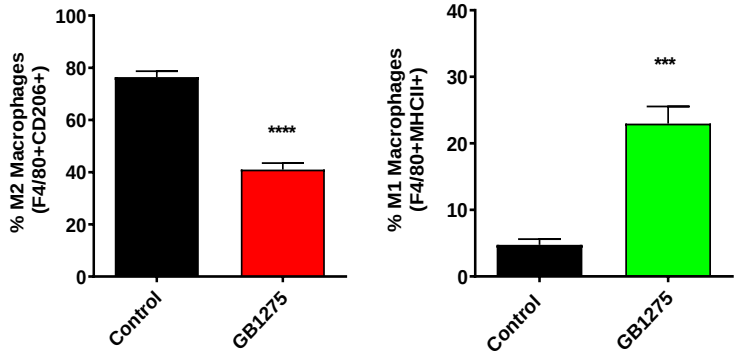


GB1275 Modulates Breast Cancer Tumor Microenvironment via Repolarization of Tumor Associated Macrophages(2)

GB1275 Displays Efficacy in the Orthotopic CL66 Breast Tumor Model



GB1275 Repolarizes 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, Markus 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
- 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.

Corporate Overview and Milestones

Financial Overview

Cash, Cash Equivalents and Marketable Securities (As of 12/31/2019)	$402mm
Debt (Initial tranche of $150 million debt facility, announced 5/2/19)	$30mm
Additional Debt Capacity (Remaining capacity of $150 million debt facility, announced 5/2/19)[1]	$120mm
Common Shares Outstanding (As of 3/18/2020)	66.3mm

mm = millions.

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

Upcoming Milestones

Indication	Milestone	Timing
GB001 (Asthma, Chronic Rhinosinusitis & Chronic Spontaneous Urticaria)		
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	Q2 2020
PAH	Phase 2 Initiation	2H 2020
GB004 (Inflammatory Bowel Disease)		
UC	Phase 1b Results	Q2 2020
GB1275 (Oncology, Solid Tumors)		
Solid Tumors	Phase 1 Results (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

Jakob Dupont, MD
Chief Medical Officer

Genentech · Roche · Stanford Medicine · OncoMed Pharmaceuticals · Memorial Sloan Kettering Cancer Center

Luisa Salter-Cid, PhD
Chief Scientific Officer

Bristol-Myers Squibb · La Jolla Pharmaceutical

Christian Waage
EVP and General Counsel

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Josh Bilenker, MD
CEO,
Loxo Oncology (Eli Lilly)

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CEO,
Epirium Bio

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