Corporate Presentation

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	• **5** clinical trials active and multiple data readouts over the next 18 months
World-Class Talent	• Deeply experienced leadership team with proven track record of developing innovative clinical assets

Deep Therapeutic Area Expertise Leads to Centers of Excellence

Allergy

- Maximize value for oral DP2 franchise
- Leverage pathway across multiple indications
- Develop innovative oral therapies for rheumatology diseases

Autoimmunity

- Gastroenterology: Develop gut-targeted therapies and drive barrier homeostasis with PHD inhibition in IBD
- Neuroinflammation: Focus on CNS-targeted immunomodulatory therapies

Foundation of Immunology and Translational Discovery and Developmental Expertise

Pulmonology / Fibrosis / Vascular Disease

- Own PDGF pathway for PAH
- Promote normalization of organ function to achieve meaningful clinical benefit in advanced diseases of lung and kidney

Oncology

- Discover orthogonal pathways in areas of emerging biology
- Address 1st and 2nd resistance to checkpoint inhibitors

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Experienced Leadership Team at the Helm

Sheila Gujrathi, MD
Chief Executive Officer

receptos
Bristol-Myers Squibb
McKinsey&Company
Ambrx
Genentech
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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Ardea Biosciences

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Managing Director,
ARCH Venture Partners

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

Renée Galá,
Former CFO,
GRAIL, Inc.

Sheila Gujrathi, MD
CEO

Josh Bilenker, MD
CEO,
Loxo Oncology

Russell Cox
President and CEO,
Cardero Therapeutics

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Robust Pipeline with Five Active Clinical Trials

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 Ongoing – LEDA Study	Worldwide (except Japan)
GB001	DP2 Antagonist (Oral)	Chronic Rhinosinusitis (with and without nasal polyps)	Phase 2 Ongoing – TITAN Study	Worldwide (except Japan)
GB001	DP2 Antagonist (Oral)	Chronic Spontaneous Urticaria	Phase 2 Planned	Worldwide (except Japan)
GB002	PDGFR Inhibitor (Inhaled)	Pulmonary Arterial Hypertension	Phase 1b Sites Initiated Phase 2/3 Planned	Worldwide
GB004	HIF-1α Stabilizer (Oral)	Inflammatory Bowel Disease	Phase 1b Ongoing	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 Disease

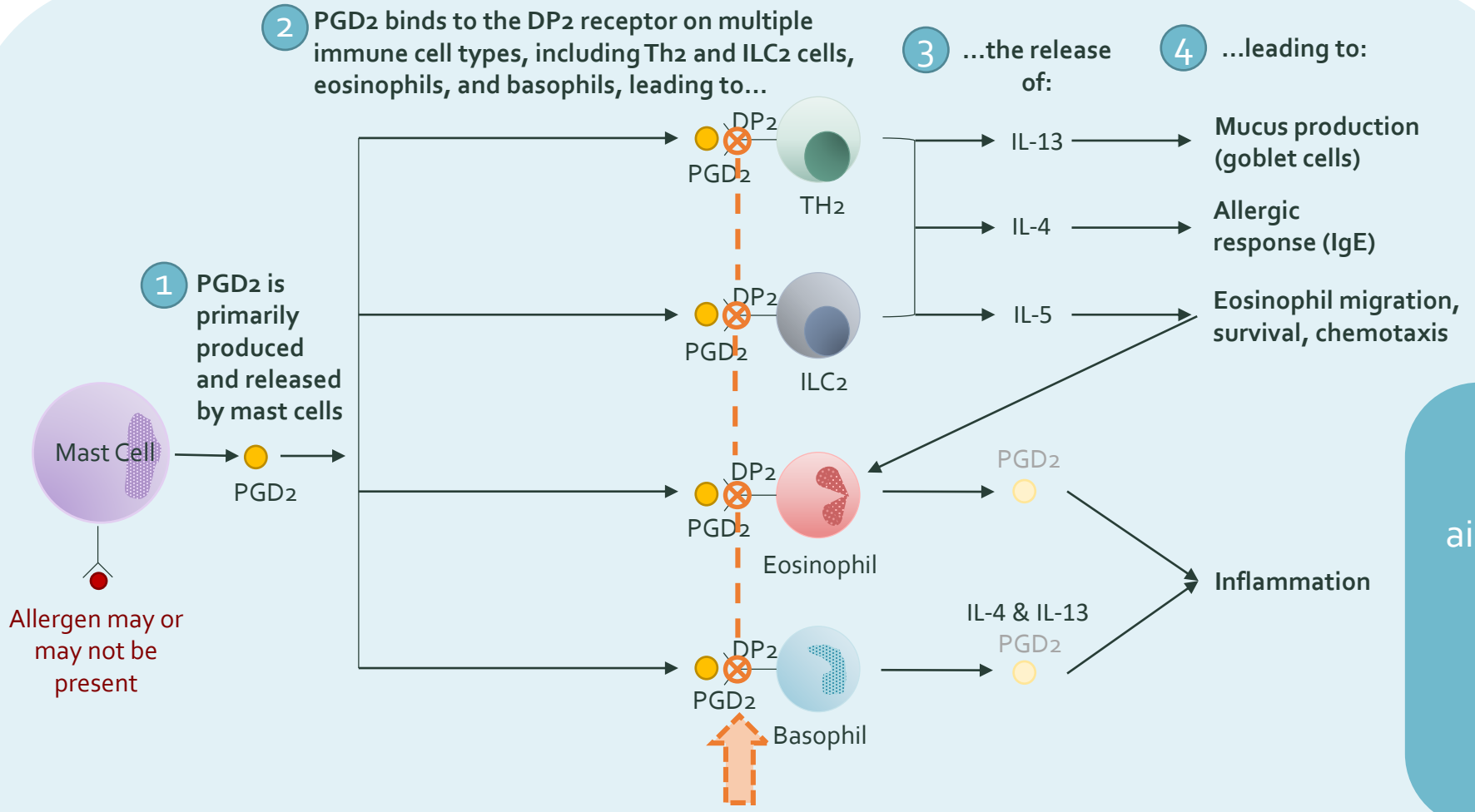
GB001

Product Description	• Oral DP2 antagonist in Phase 2b development for the treatment of moderate-to-severe eosinophilic asthma (LEDA Study – Initiated Q4 2018) • Proof of concept Phase 2 trial for chronic rhinosinusitis with and without nasal polyps underway (TITAN Study – Initiated Q2 2019) • Asthma Phase 2 interim results expected in 1H20; Asthma Phase 2 topline results in 2H20; CRS Phase 2 topline results in 2020 • 409 patients have received at least 1 dose of GB001 with no clinically significant safety findings(1) • Patent protection out to 2031(2)
Mechanism of Action and Scientific Rationale	• DP2 important in Th2 cell activation and upstream of IL-4, IL-5 and IL-13 • Th2 cell activation plays prominent role in asthma and other allergic and inflammatory disorders • Target validation from Teijin's Phase 2 study in Japanese patients and Novartis's fevipiprant program • Anti-inflammatory effect comparable to certain biologics with potential to be used earlier in treatment

CRS = Chronic Rhinosinusitis.

1) As of December 31st, 2018.
2) Includes assumption of maximum 5 years of patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims.

Role and Biology of the DP2 Pathway in Type 2 Inflammation



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

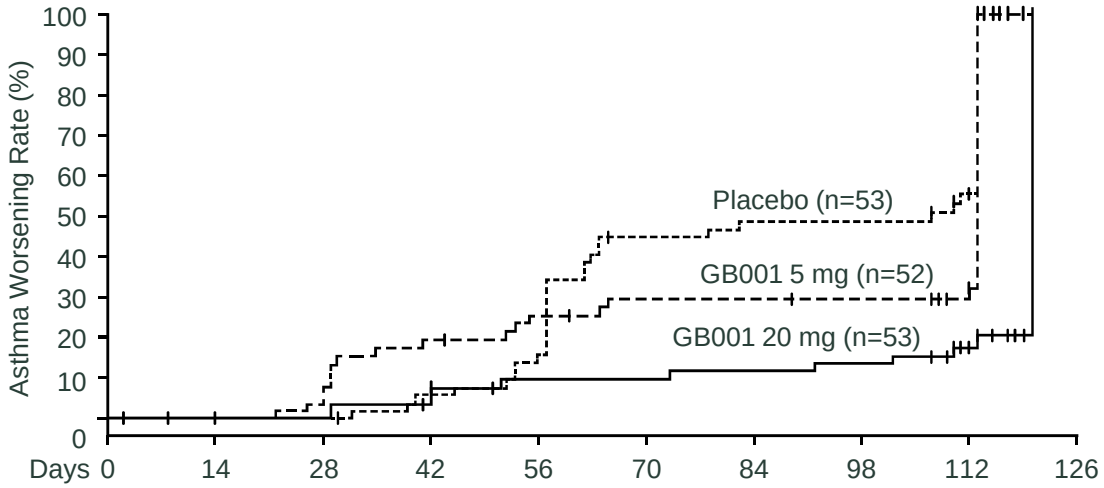
Treatment with GB001, an oral DP2 antagonist, blocks the interaction of PGD2 with DP2, preventing the resulting pro-inflammatory cascade

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.

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

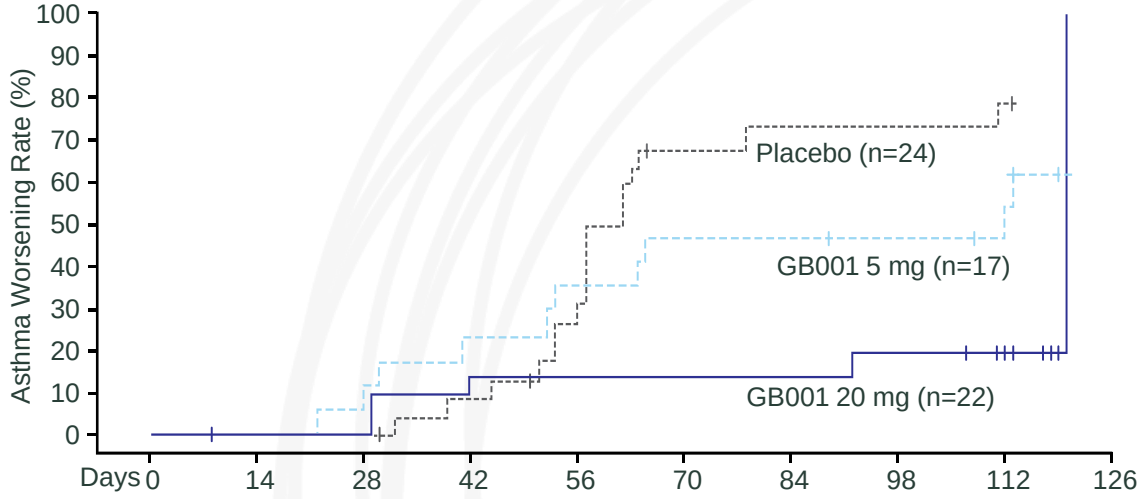
Both doses of GB001 met the primary endpoint of change in morning peak expiratory flow with statistical significance vs placebo

Overall Population



	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 (≥300µL)



- p-value (log-rank test) for placebo vs 20mg GB001 is ***0.0003*** for the high eosinophil subgroup (≥300µL)

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

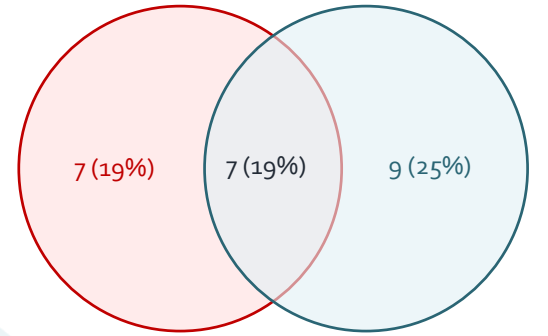
Beyond Eos: Potential for Target Market Expansion

Poster Presentation at AAAAI 2019

Reduction of Exhaled Nitric Oxide by the DP2 antagonist GB001 in Patients with Mild-Moderate Atopic Asthma

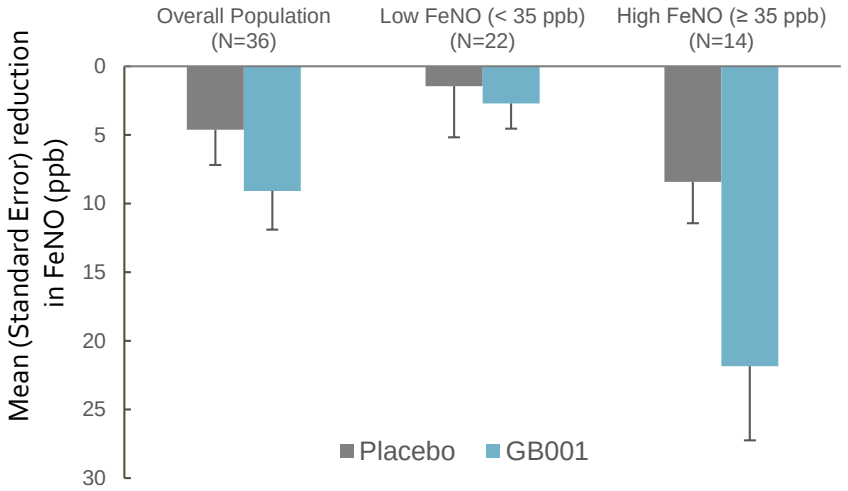
Results from a post-hoc analysis evaluating Fractional exhaled Nitric Oxide (FeNO) as a baseline marker and outcome following administration of GB001 or placebo over 28 days in 36 subjects with partially controlled, atopic asthma

Subjects in High FeNO and Eos. Subgroups



Weak correlation between baseline FeNO and Eos. (r=0.29)

Mean Reduction in FeNO at Day 28



Background	• Fractional exhaled nitric oxide (FeNO) is a marker of airway inflammation. FeNO is elevated in eosinophilic inflammation
Results:	• In a retrospective analysis, GB001 resulted in greater numeric improvements in lung function at Day 28 relative to placebo in subjects with high FeNO in this partly controlled asthma population • 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 • FeNO may be a useful marker for treatment response to GB001

FeNO = fractional exhaled nitric oxide; ppb = parts per billion; PK = pharmacokinetics; PD = pharmacodynamics; eos. = eosinophil.

DP2 Antagonism in Asthma Has Evidence of Clinical Validation

GB001

	Select Clinical Studies in Asthma*				
	Oral (Mild-to-Mod. Asthma)		**Biologics** (Mod.-to-Severe Asthma) 12-month exacerbation studies		
	DP2 antagonist GB001	DP2 antagonist [3]Fevipiprant	anti-IgE [4]Omalizumab	anti-IL-5 [5]Mepo, [6]Benra, [7]Rezi	anti-IL4R [8]Dupilumab
Annualized exacerbation rate reduction vs placebo (%)	TBD (~50% reduction in asthma worsening, steroid withdrawal setting) [1]	30 to 50% Targeted Profile, 52 weeks	38 to 60%	28% to 59%†	66 to 67%†
FEV1 change from baseline difference vs placebo (mL)	102 ml[2] week 4	77 to 164 ml^ week 12	21-98 ml week 16	52 to 137 ml Week 12 / 16†	210-260 ml week 12†
	Select Biomarker Data*				
FeNO reduction from baseline	+++	-	-	-	+++
Reduction in blood eos. from baseline	+	+	++	++++	+
Reduction in sputum eos. from baseline	Not Available	+++	+	++++	++

Note: Results need to be interpreted within context of different trials, study populations, disease severity, study designs, timepoints, and analysis methodologies.

DP2 receptor pathway plays an important role in Type 2 inflammation.
Oral DP2 antagonists have the potential to generate anti-inflammatory effects comparable to biologic therapies.

*Clinical trials were not conducted head to head.
† Eosinophilic phenotype.
^ Study defines high eosinophil levels ≥250 cells/µL. Targeted efficacy profile studied with GINA step 4/5 patients.

1) Phase 2 in Japanese patients with mild-to-moderate eos. asthma.
2) Ortega H, et al. Reduction of Exhaled Nitric Oxide by the DP2 antagonist GB001 in Patients with Mild Atopic Asthma. Presented at: 2019 AAAAI Annual Meeting; 2019 Feb 21-25; San Francisco.
3) Bateman E et al, Eur Respir J, 2017; and Novartis 2018 R&D and investor update.
4) Omalizumab pediatric supplement application.
5) Mepolizumab USPI.
6) Benralizumab USPI.
7) Resilizumab USPI.
8) Dupliumab USPI.

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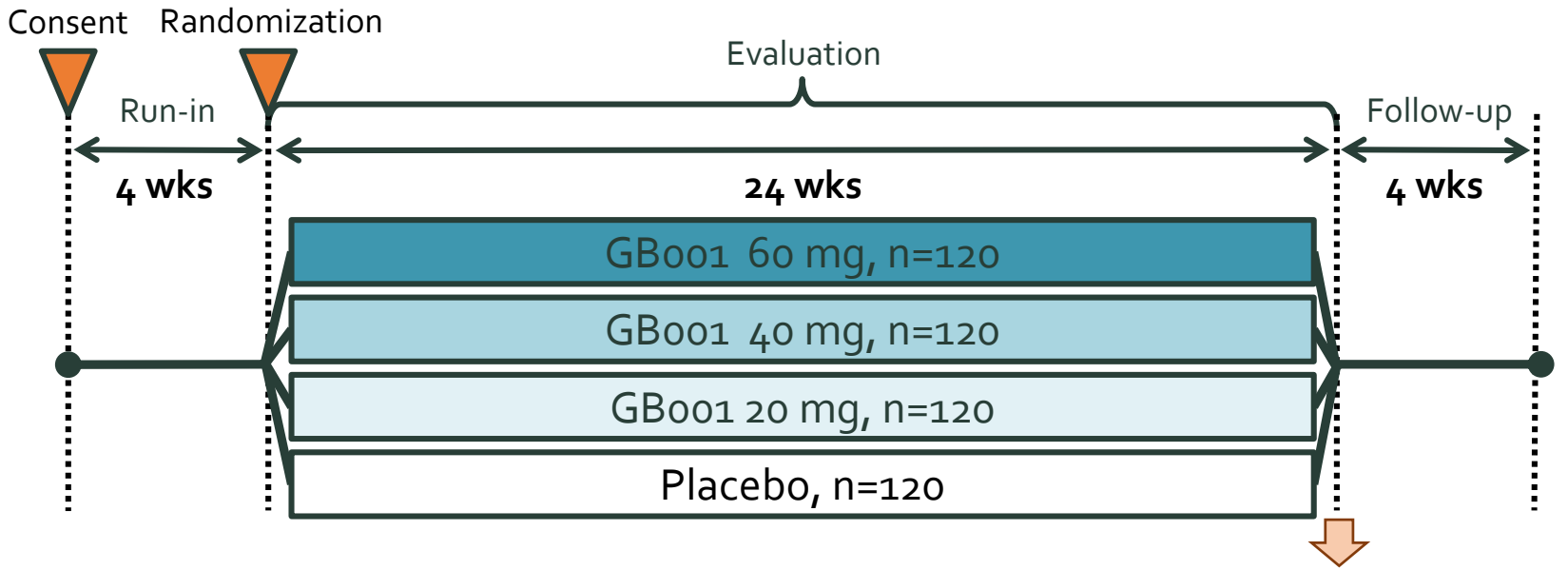
LEDA Study: Phase 2b Study Design Allows for Efficient Transition to Phase 3

LEDASTUDY

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

Status: Enrolling, Initiated Q4:2018



Interim analysis after ~320 subjects complete week 24 or prematurely withdraw from the study (expected in 1H 2020)

Patient Population	480 adult mod.-to-severe eosinophilic asthmatics (eosinophil counts ≥ to 250 cells/µL)
Treatment	60mg, 40mg, 20 mg or placebo, oral administration (QD) on top of background therapy
Duration of Treatment	24 weeks
Endpoint	**Primary:** Reduction in asthma worsening from baseline; asthma worsening composite primary endpoint includes changes in FEV1, AM PEF, rescue medication use, asthma control and severe asthma exacerbations **Secondary:** FEV1, asthma control, asthma quality of life

QD = once daily dosing.

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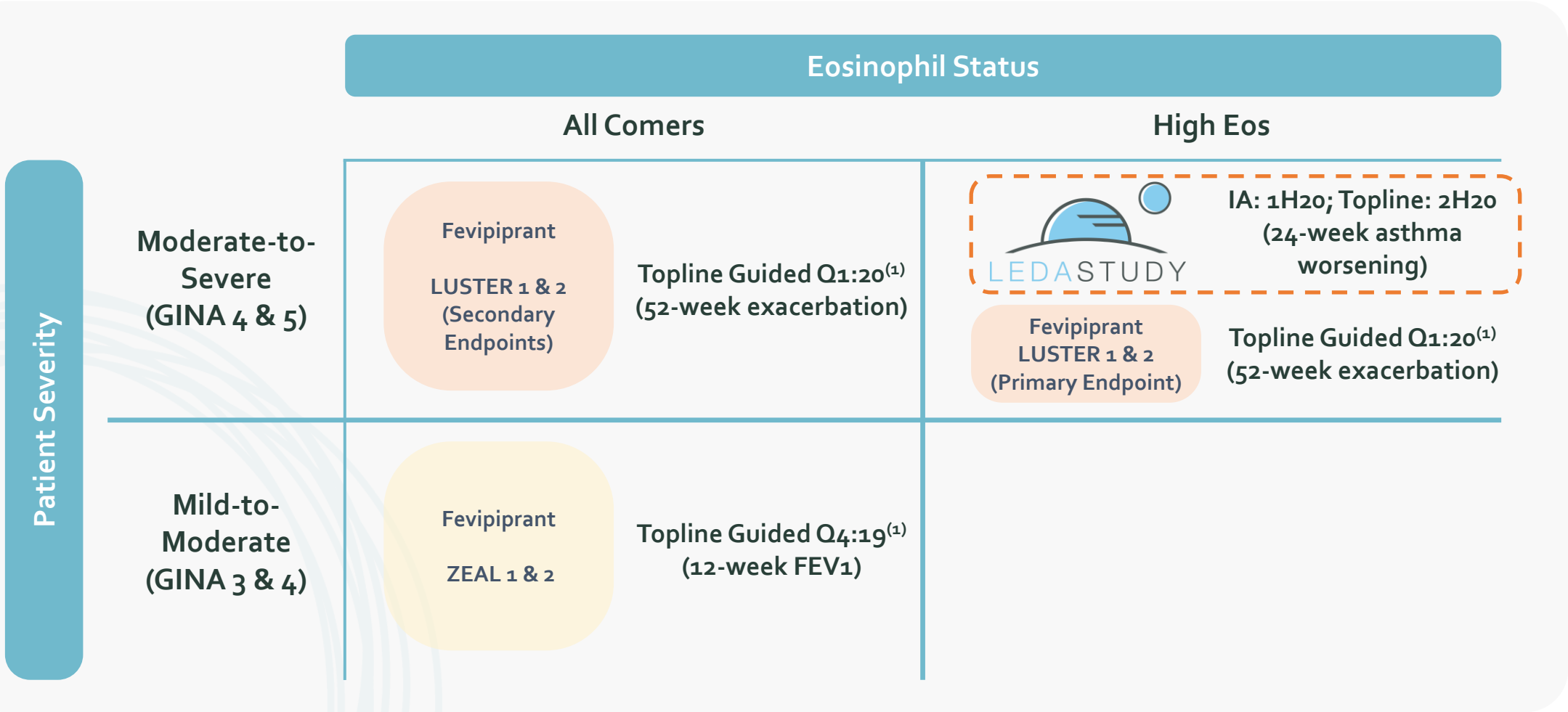
Upcoming Data Readouts for DP2 Antagonists

GB001

- Phase 2b LEDA study designed to enroll a targeted patient population with the most relevant endpoint
- Limited readthrough from near-term ZEAL readouts because of less severe, more heterogeneous study population and more difficult primary endpoint (difference vs. placebo in FEV1 reduction from baseline at 12 weeks in all comer population)



1) Novartis Q2:19 Earnings Call.
IA = interim analysis.

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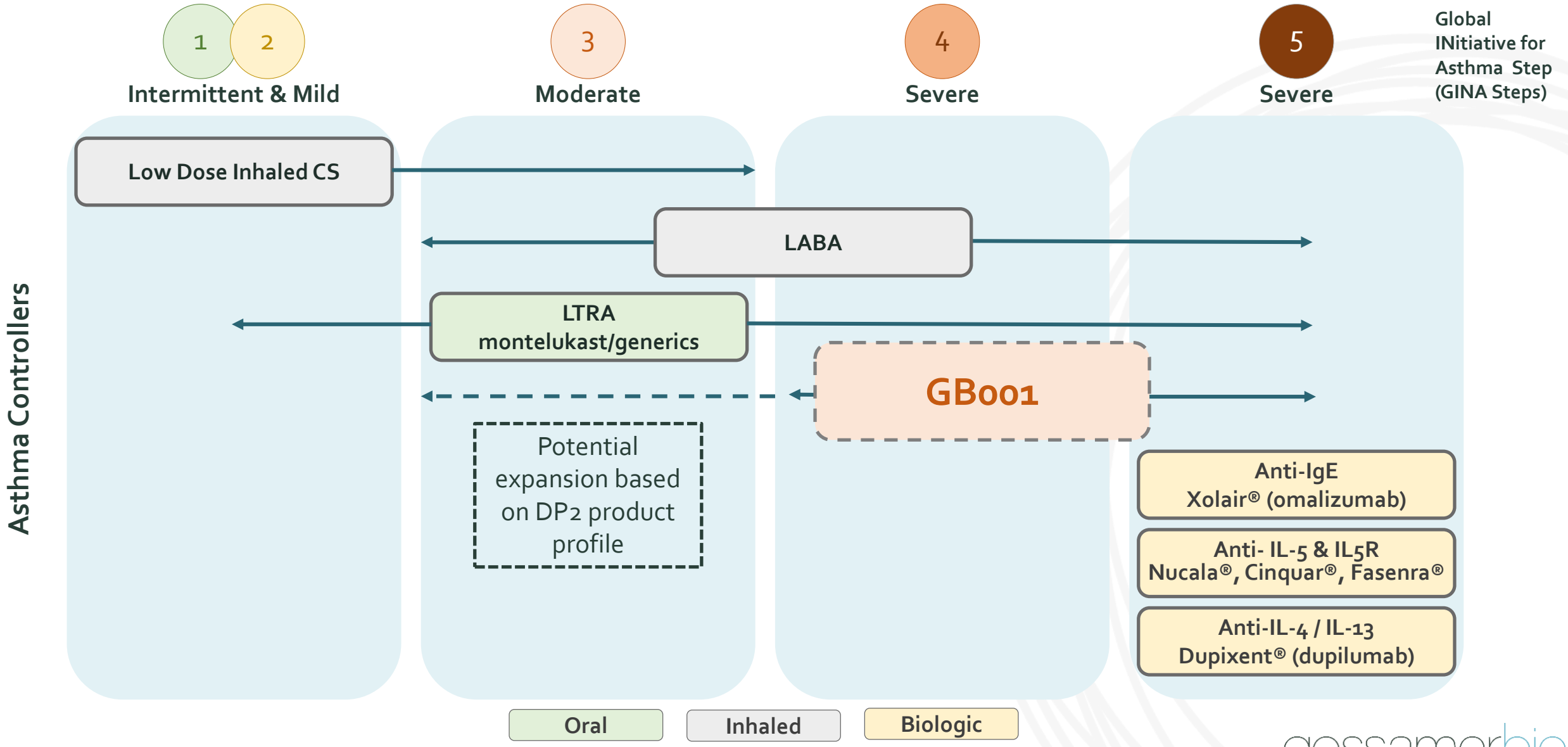
Potential for Market Asthma Positioning Prior to Biologics



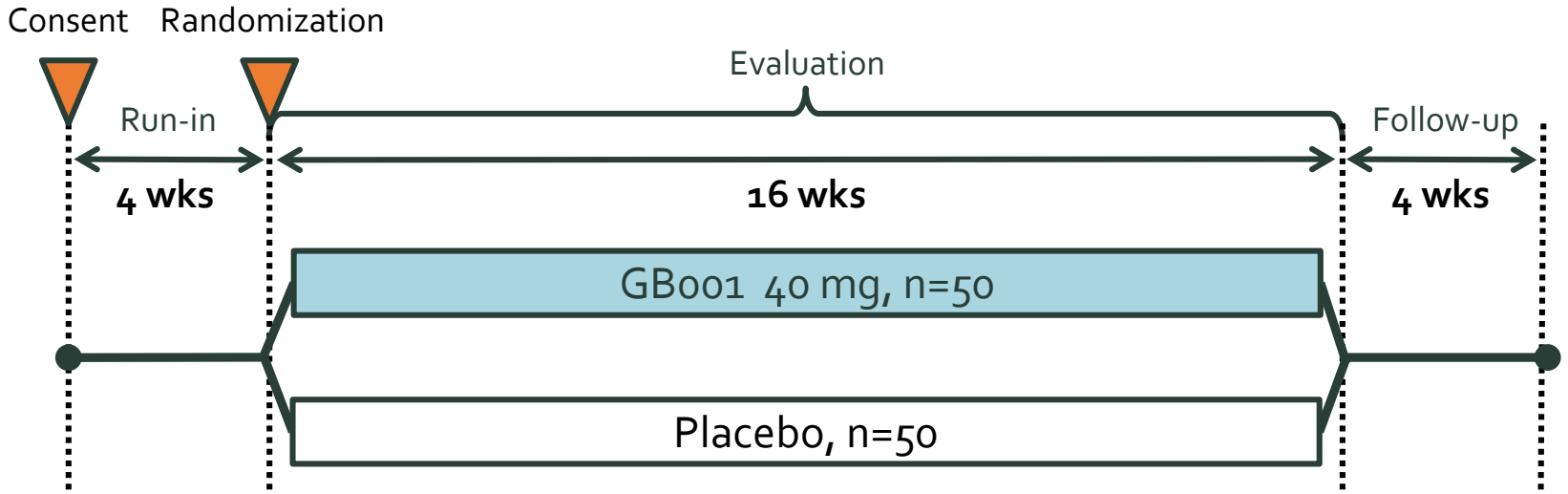
CS = Corticosteroids; LABA = Long-acting beta agonist; LTRA = Leukotriene receptor antagonist.

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

TITAN STUDY

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

Status: Enrolling, Initiated Q2:2019



Patient Population	~64 adult patients with CRS with nasal polyps; ~36 adult patients with CRS without polyps
Treatment	40mg or placebo, oral administration (QD), on top of intra-nasal steroids
Duration of Treatment	16 weeks
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.

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 • Active Phase 1b trial in PAH with expected readout in 1H 2020 • Planned Phase 2/3 trial in PAH, initiating in 2H 2019, with expected readout in 2H 2021 • Patent protection out to 2034[1]; Orphan Drug Designation from FDA and EMA
Mechanism of Action and Scientific Rationale	• 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 be clinically significant in Phase 3 PAH trial of imatinib (Gleevec), with systemic toxicities • GB002 has improved selectivity vs PDGF receptor β compared to imatinib and has demonstrated hemodynamic improvements and reduced occlusive lesions in animal models • Inhaled delivery of GB002 designed to improve side-effect profile (compared to imatinib), provide convenient administration, and maximize drug delivery to lung

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.

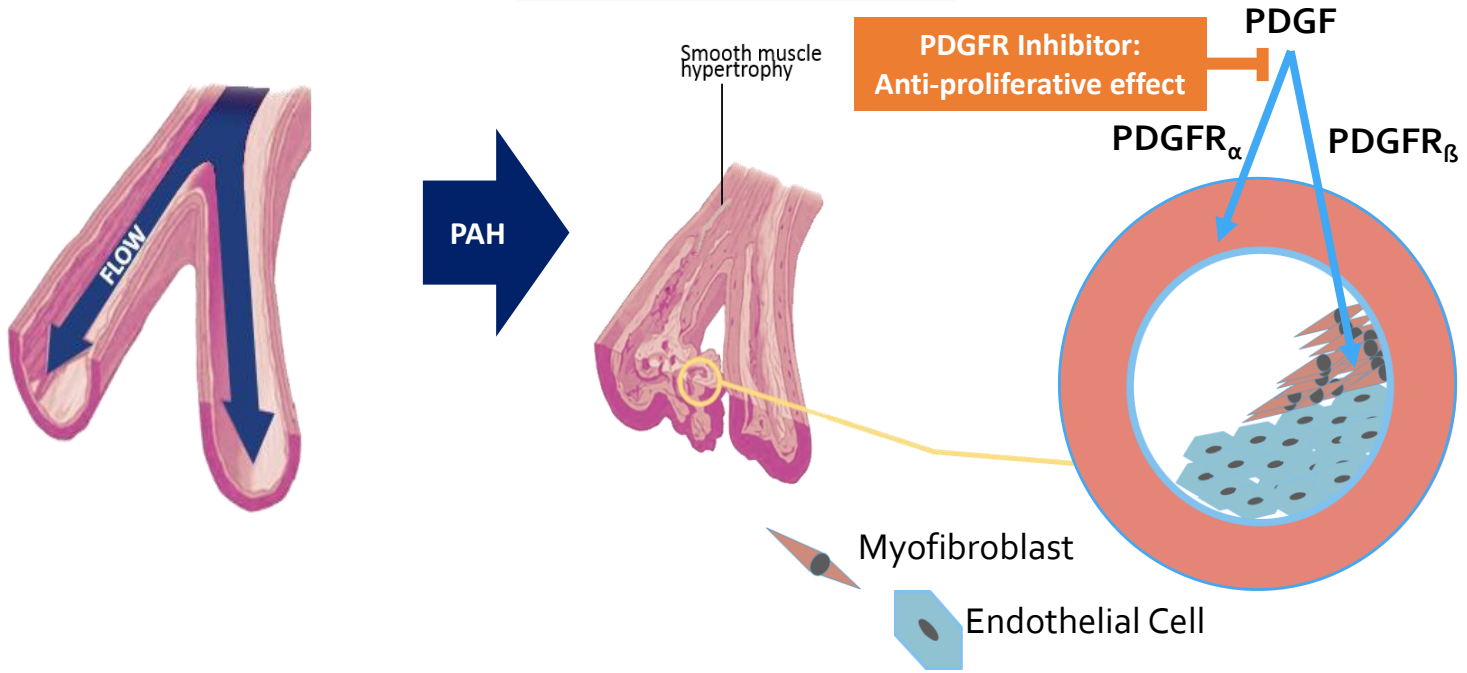
PDGF Pathway Drives Pulmonary Arteriolar Remodeling – an Underlying Problem in PAH

GB002

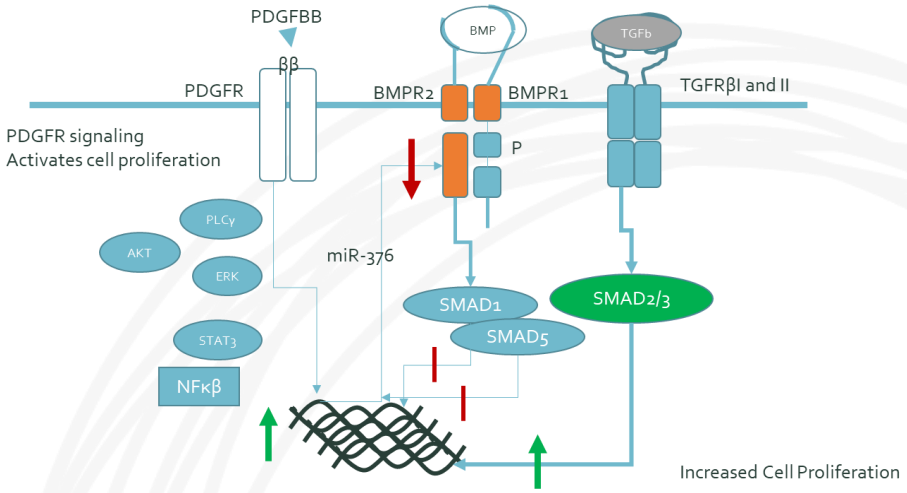
Normal Pulmonary Artery

Vascular Remodeling

PDGFR Inhibitor: Anti-proliferative effect



- PDGF signaling causes overgrowth of cells in lung blood vessels
- PDGF Receptor is activated by phosphorylation in human PAH



- BMPR2 dysregulation can lead to endothelial changes to smooth muscle like cells
- PDGFR inhibition modulates BMPR2 in pulmonary artery smooth muscle cells
- Primary lesions occur in the small blood vessels of the lung (pulmonary arterioles)

Sources: Hopper, et al., Circulation, 2016; Chen et al., BMC Genomics, 2016.
AKT = protein kinase B; TGFb = transforming growth factor beta; $NF_k\beta$ = nuclear factor-kappa beta; BMP = bone morphogenetic protein.

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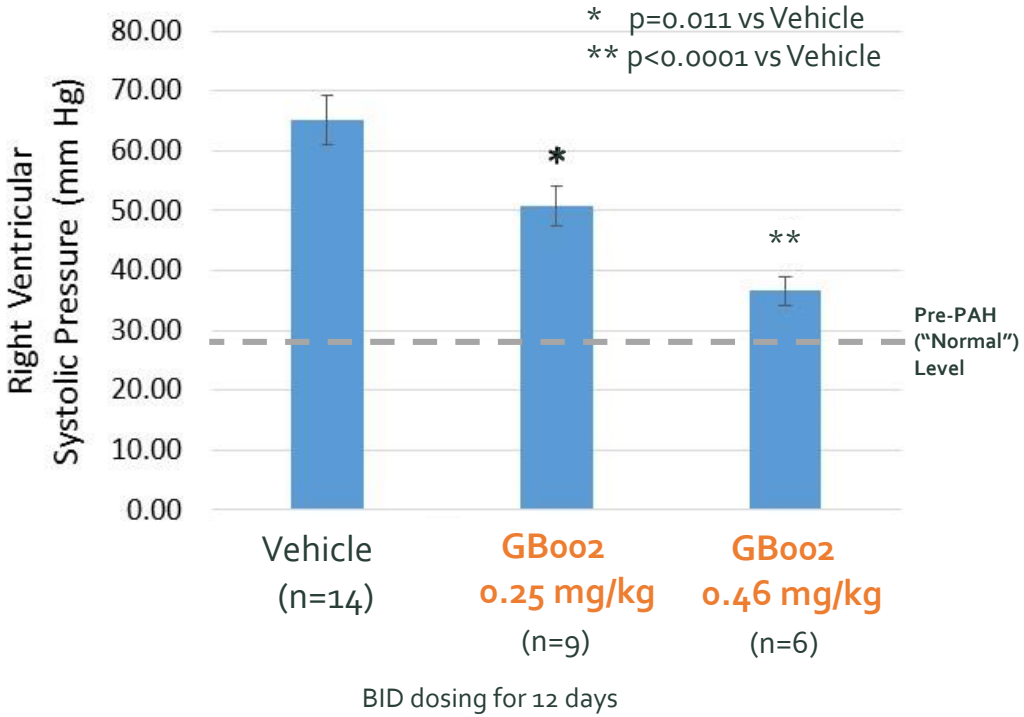
GB002 Improves Hemodynamics and Reverses Vascular Remodeling Through Inhibition of PDGF in Animal Models

GB002

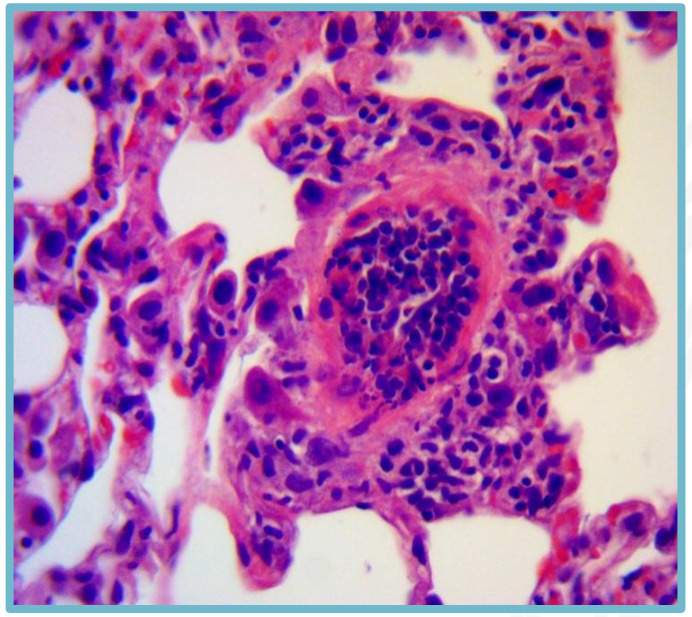
Pre-Clinical Data Right Heart Pressure



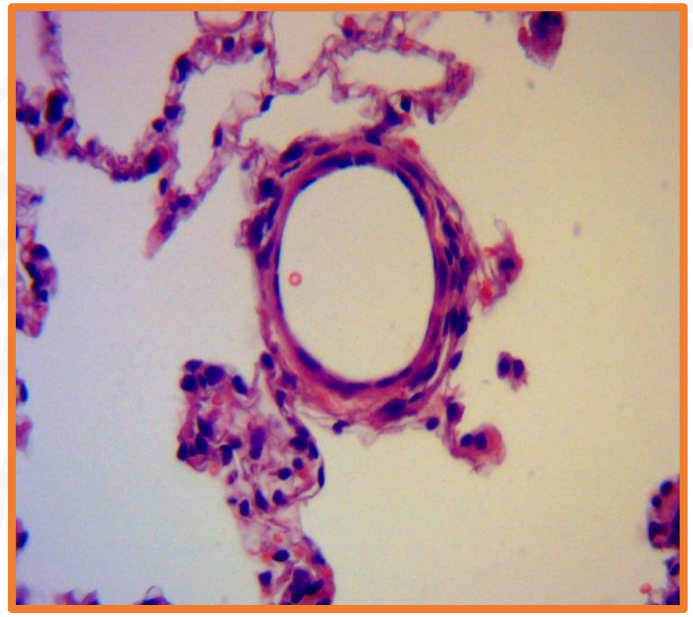
BID dosing for 12 days

- Dose dependent hemodynamic improvement seen in animal models

Pre-Clinical Data of Histology Samples From Rat Model of PAH



Vehicle



GB002

- GB002 inhibits both PDGF α and β, and inhibited and reversed cell overgrowth in lung blood vessels in PAH in a rat model
- Rat model replicates many features of human PAH, including the abnormal cell proliferation that can block the small vessels of the lung

BID = twice daily dosing.

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

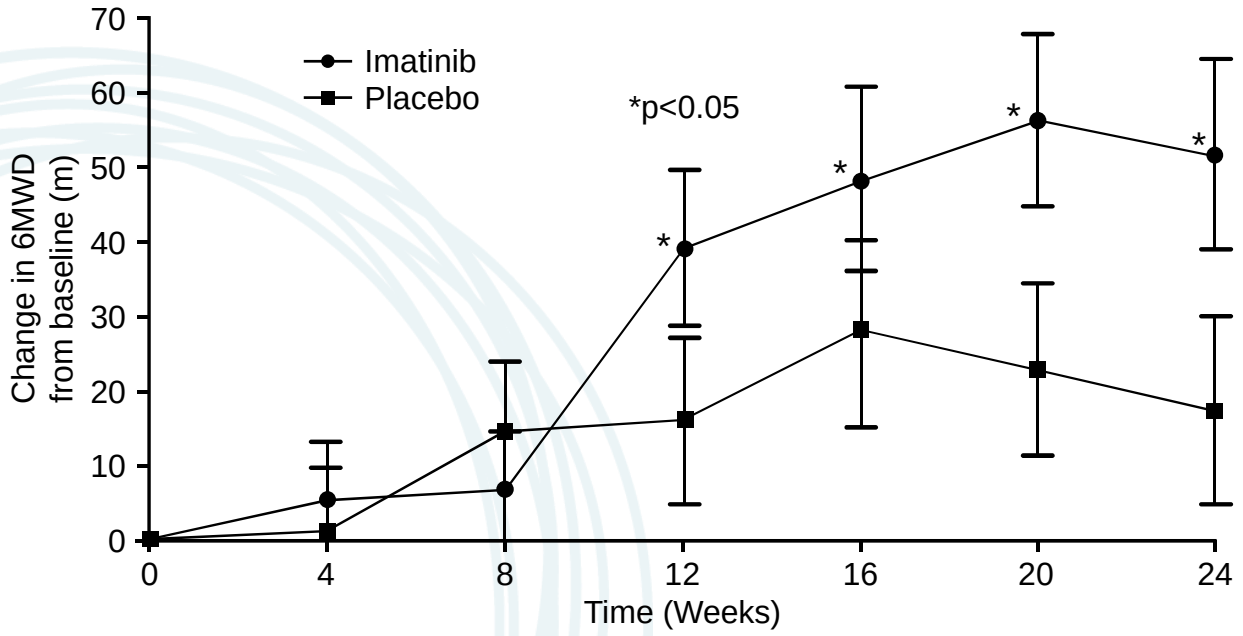
GB002

Imatinib Mesylate as Add-on Therapy For Pulmonary Arterial Hypertension

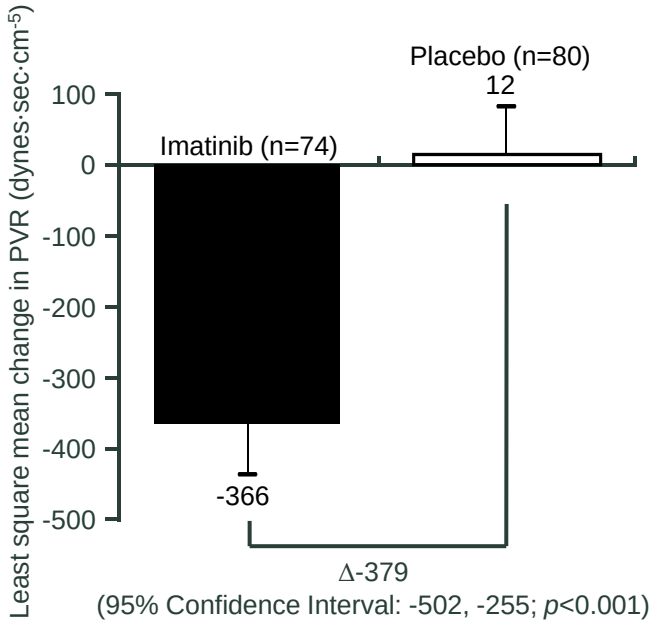
Results of the Randomized IMPRES Study

- 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
- Serious adverse events, including 8 subdural hematomas and high drop-out rates

Exercise Tolerance



Peripheral Vascular Resistance (PVR)



Source: Circulation, March 2013.

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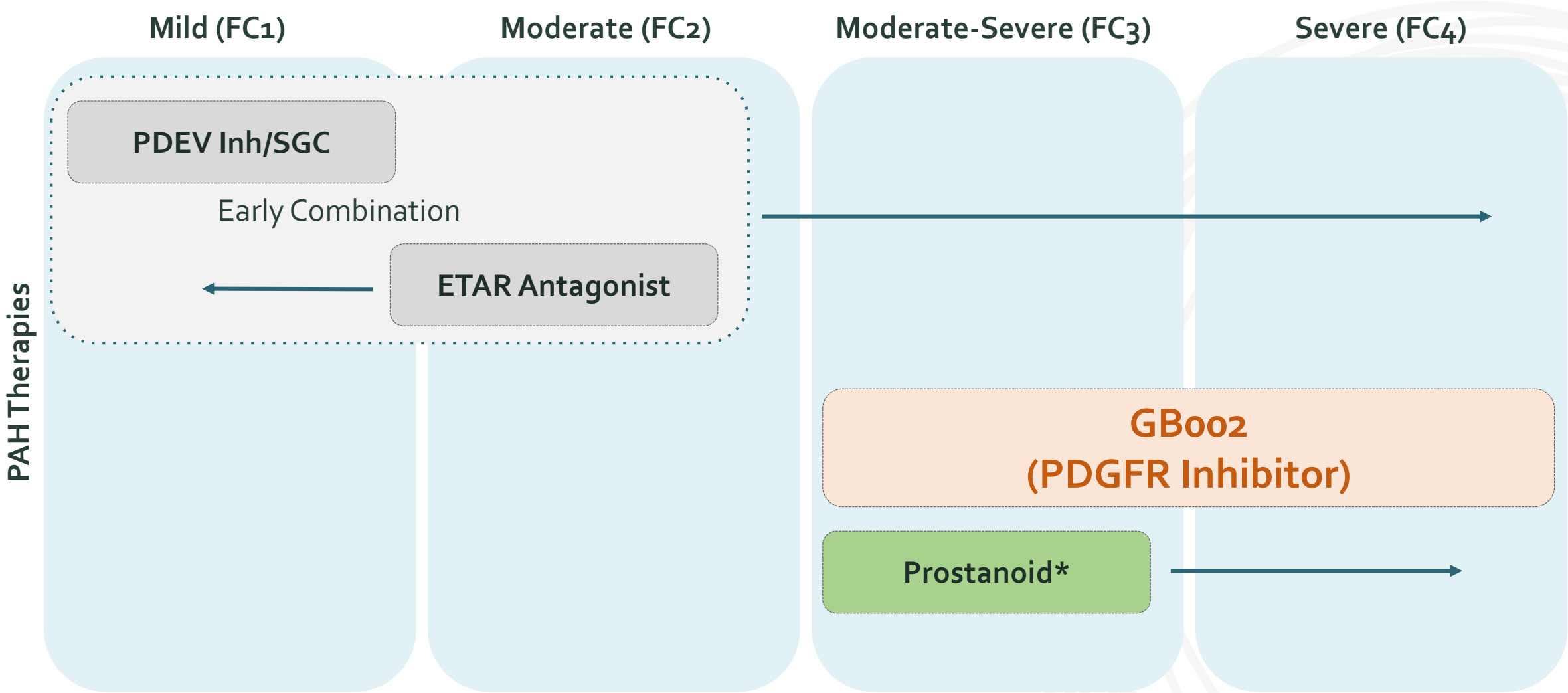
GB002 Could Provide a Much Needed Treatment Option for High Risk PAH Patients

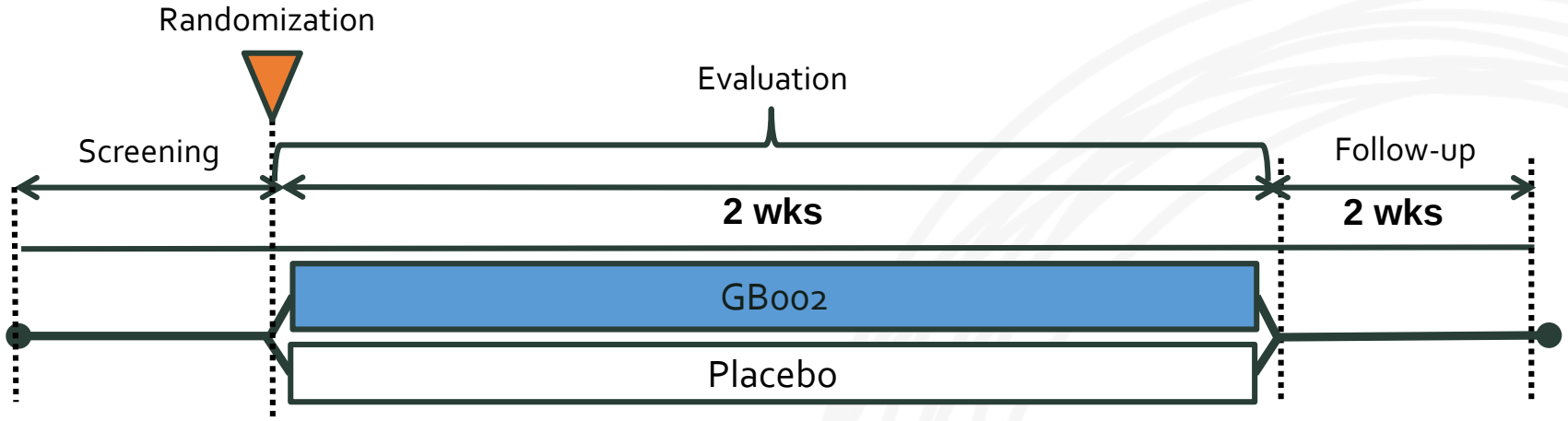


ETAR = endothelin receptor type A; FC = Functional Class; PDEV = phosphodiesterase type V; SGC = soluble guanylate cyclase.
Sources: 2015 ESC/ERS Guidelines

Active 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
Treatments	Multiple doses of GB002, placebo
Duration of Treatment	2 weeks
Key Study Objectives	Safety, tolerability, PK profile, peripheral blood biomarkers, markers of disease modification through imaging
Endpoints	AE Profile, changes in safety lab values, PK parameters, NTproBNP, Right Ventricular Ejection Fraction (based on cardiac MRI), high resolution CT Scan reconstruction of pulmonary vasculature

Status: Sites Initiated Q3:2019

NTproBNP = biomarker for heart failure.

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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 for IBD

GB004

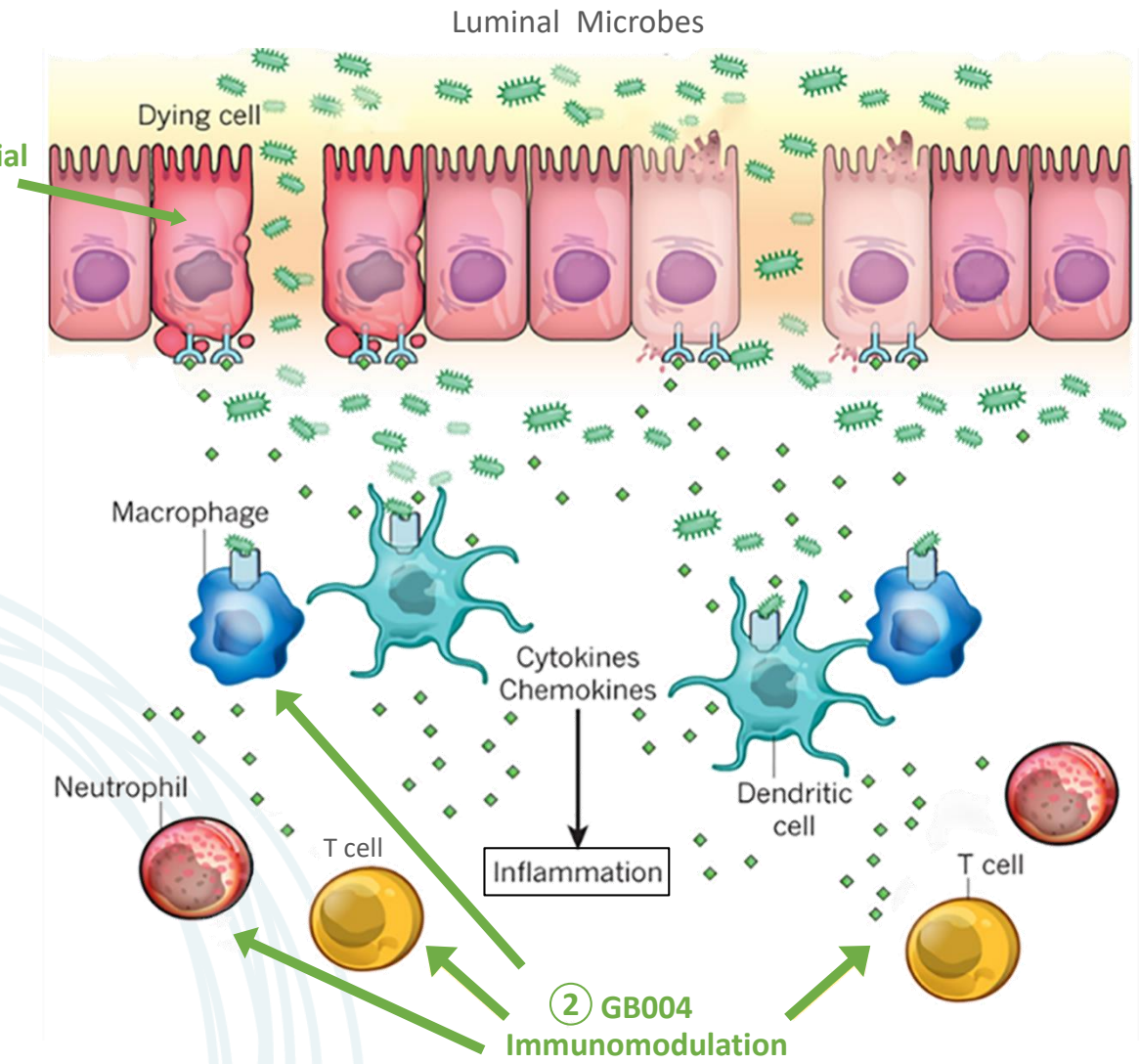
Product Description	• Oral, small molecule, gut-targeted, prolyl hydroxylase inhibitor that for the treatment of IBD • Phase 1b trial in UC ongoing, initiated in 2Q 2019, with expected readout in 1H 2020 • Planned Phase 2 trial in UC, initiating in 1H 2020, with expected readout in 1H 2022 • Patent protection out to 2035(1)
Mechanism of Action and Scientific Rationale	• Designed to restore epithelial barrier function, in addition to immunomodulatory effects • High degree of hypoxia in inflamed gut due to vascular disruption and chronic inflammation • 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 • GB004 stabilizes hypoxia inducible factor (HIF-1α) locally, and has been shown to reduce weight loss and restore epithelial barrier function in animal models of IBD

IBD = inflammatory bowel disease.

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

Mechanism of PHD Inhibitor to Restore Epithelial Barrier Function

GB004



- IBD represents a state of chronic tissue injury
- HIFs play an important role in protecting cells in times of stress and low oxygen levels
- GB004 inhibits PHDs, which break down HIFs, preferentially stabilizing HIF-1α
- HIF-1α stabilization in IBD leads to two primary effects: restoration of epithelial barrier function and immunomodulation
  1. HIF-1α expression leads to increases in genes known to promote epithelial integrity and mucosal barrier function
  2. Additionally, HIF-1α is thought to be critical for regulatory immune cell function, and its stabilization can lead to reduced inflammation
- GB004 is gut-targeted, and has thus far avoided systemic effects of other PHD inhibitors, including erythropoiesis

Adapted from Pasparakis *et al.* (2015) *Nature.*

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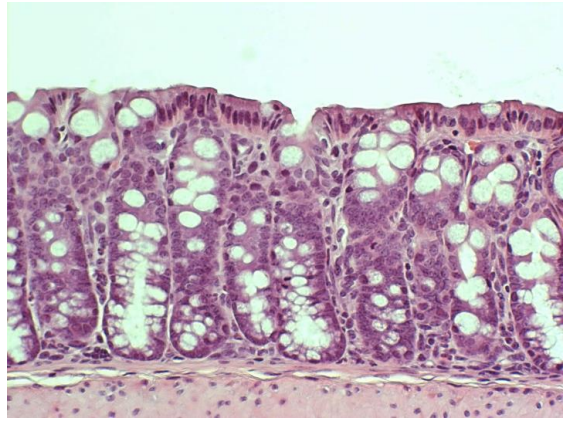
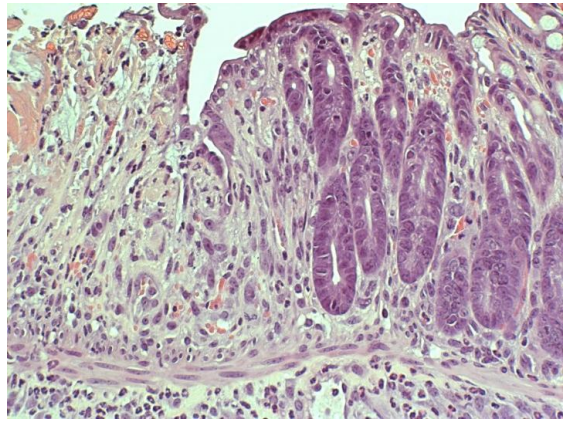
Oral GB004 Demonstrates Restitution of the Epithelial Barrier and Effects on Mucosal Healing in TNBS-Induced Colitis Model

GB004

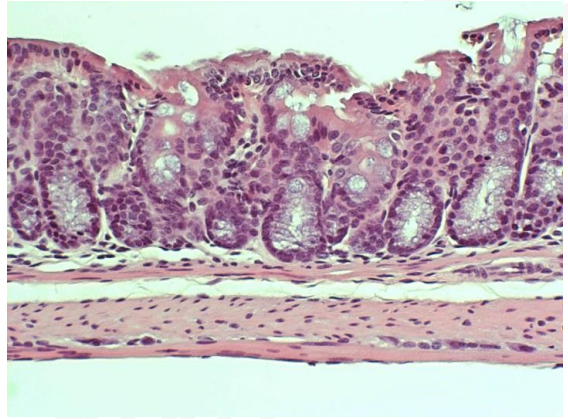
Vehicle in Cyclodextrin



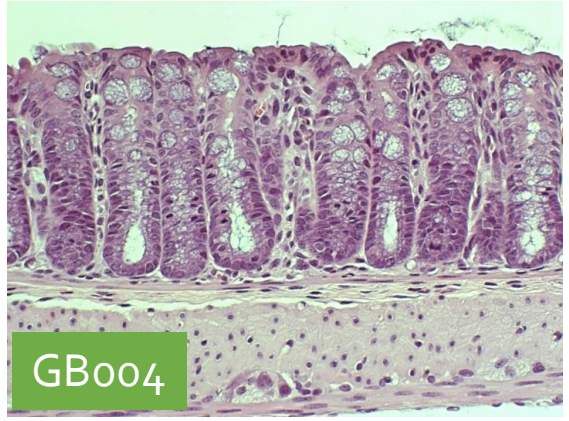
TNBS + Cyclodextrin



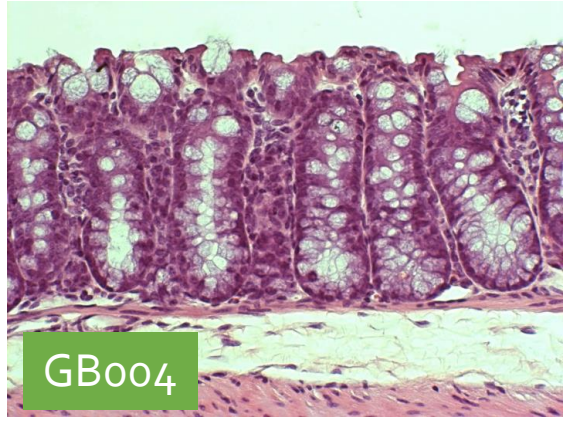
TNBS + Dexamethasone



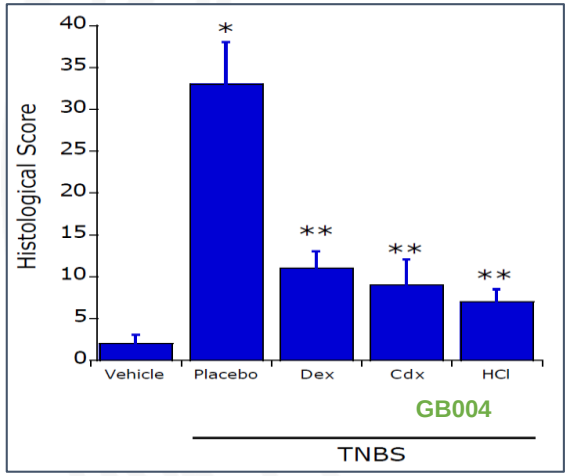
TNBS + GB004 in Cyclodextrin



TNBS + GB004 in HCl



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
Dex = dexamethasone; Cdx = cyclodextrin.

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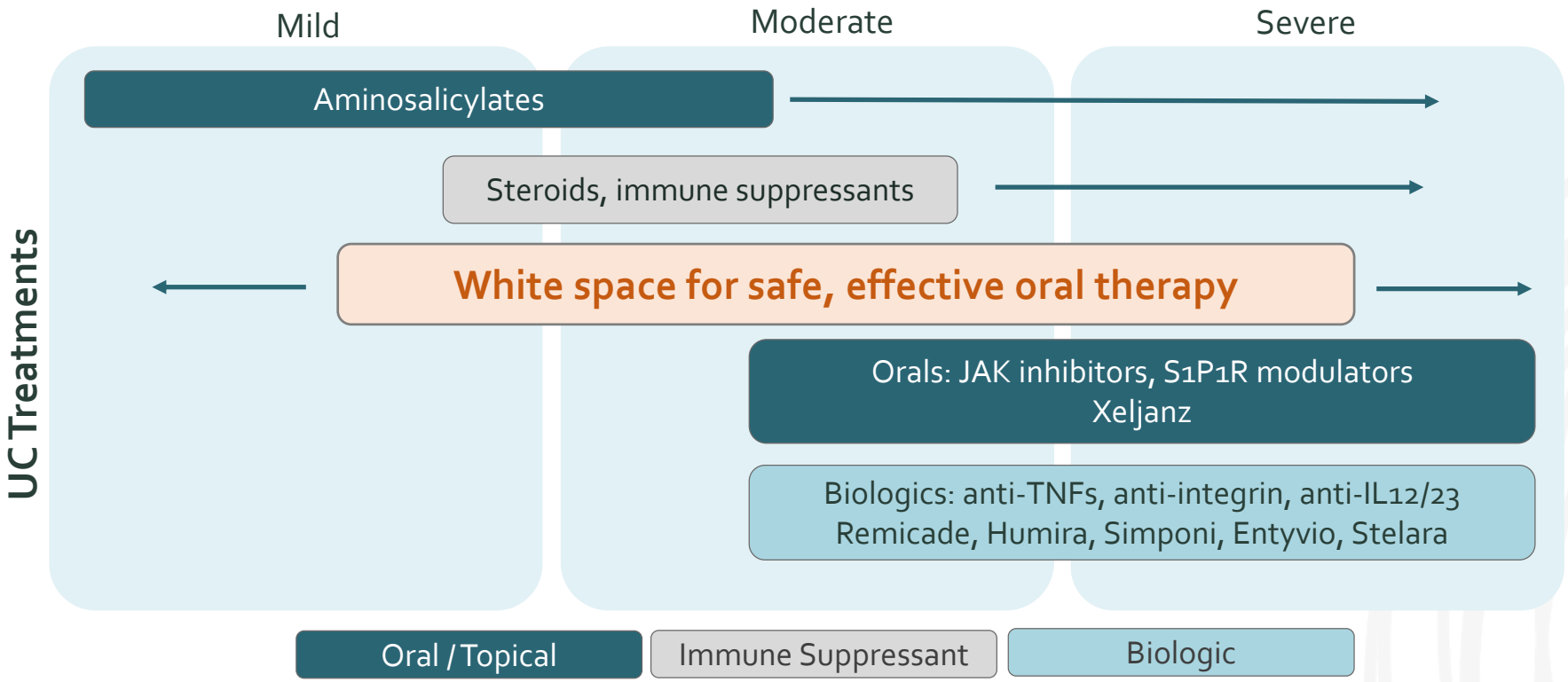
GB004 Represents a New, Potentially Transformative Approach in UC



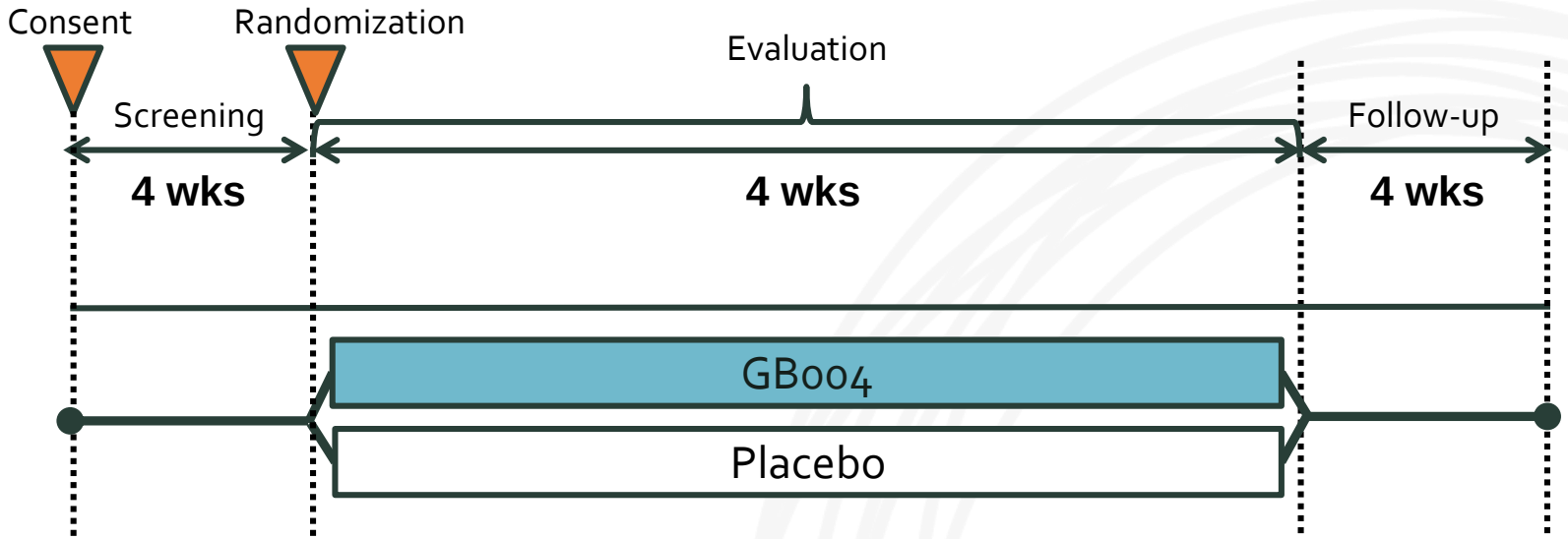
- Current IBD therapies typically target the "overactive" immune response
- Rates of clinical remission, mucosal healing, and durability remain suboptimal even with available therapies
- Development of GB004 will occur within the context of a changing treatment paradigm, evolving regulatory endpoints, competitive clinical trial environment, and the imperative for differentiation in a crowded market

Depending on clinical profile, GB004 is well suited as a pre-biologic therapy for the mild-moderate disease activity segment as monotherapy or in combination

Ongoing 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 ulcerative colitis, who have had an inadequate response or intolerance to 5-ASA or steroids; mild disease or greater; evidence of active inflammation by histology
Treatments	GB004 doses, placebo; QD dosing
Duration	4 weeks
Endpoints	Primary: Safety, tolerability Secondary: PK Exploratory: biomarker analysis, and histologic, endoscopic, and clinical indices to evaluate biological effect

Status: Enrolling, Initiated Q2:2019

QD = once daily dosing

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GB1275

CD11b Modulator

Solid Tumors

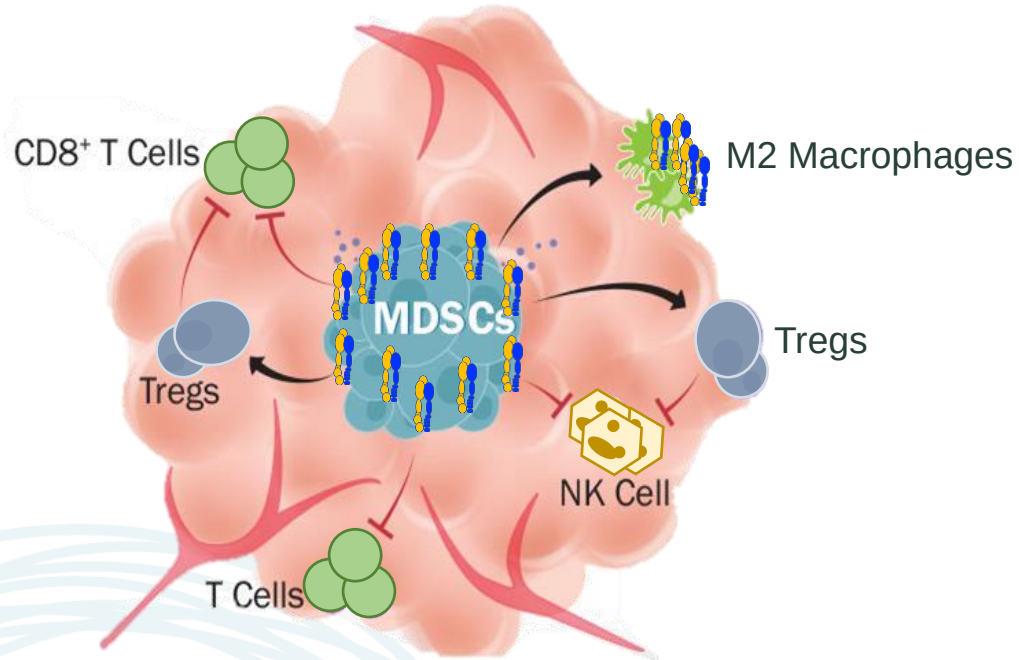
GB1275: Opportunity to Improve Response to Cancer Therapy through Targeting Multiple Myeloid Immunosuppressive Mechanisms of Action

Product Description	• Oral, small molecule, CD11b modulator for the treatment of solid tumors • Ongoing GB1275 Phase 1/2 trial, both as monotherapy and in combination with anti-PD-1 or chemotherapy, targeting selected solid tumors; Phase 1 readout expected in 2H 2020; Phase 2 readout expected in 2H 2021 • Patent protection out to 2036(1)
Mechanism of Action and Scientific Rationale	• Disrupts multiple immunosuppressive myeloid cell subsets, including MDSCs and TAMs • Efficacy observed as single agent and synergistically in combination with chemotherapy and immuno-oncology therapies • Preclinical data suggest differentiation from other approaches targeting immunosuppressive mechanisms • Opportunity to target immuno-oncology resistant tumors including PDAC, CRC, TNBC, CRPC and others

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.

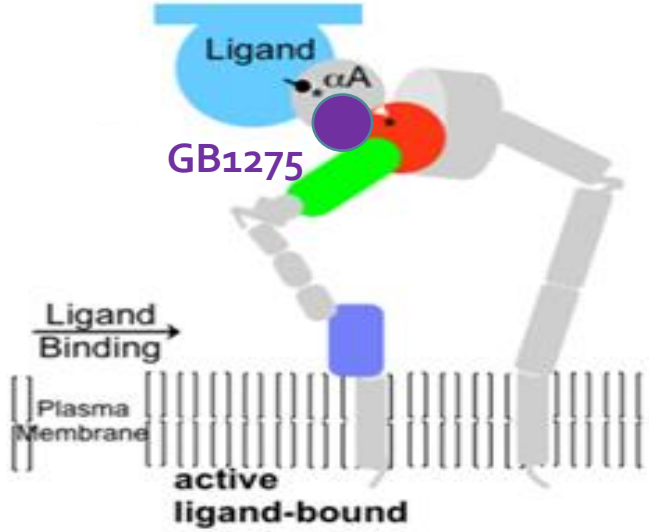
1) Does not include available patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims. Total patent life with patent term extension cannot exceed 14 years from approval.

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



Adapted from R&D Systems

- Targeting MDSCs **or** M2 macrophages is one of the key strategies to help overcome resistance to T-cell activating therapies in the clinic
- **GB1275** mediated CD11b modulation Impacts myeloid cell recruitment and macrophage polarization at the tumor site
- **GB1275** is a first-in-class agent that can impact both MDSCs and M2 TAMs in the tumor microenvironment



CD11b expressed on myeloid cells

- Monocytes
- Neutrophils
- MDSCs
- Tumor associated macrophages

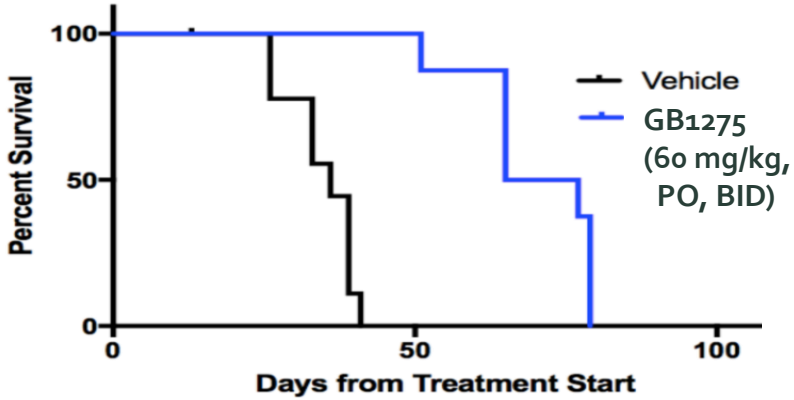
Mac1 = CD18/CD11b Integrin

MDSC = myeloid-derived suppressor cells; NK Cell = natural killer cells; Tregs = regulatory T cells.

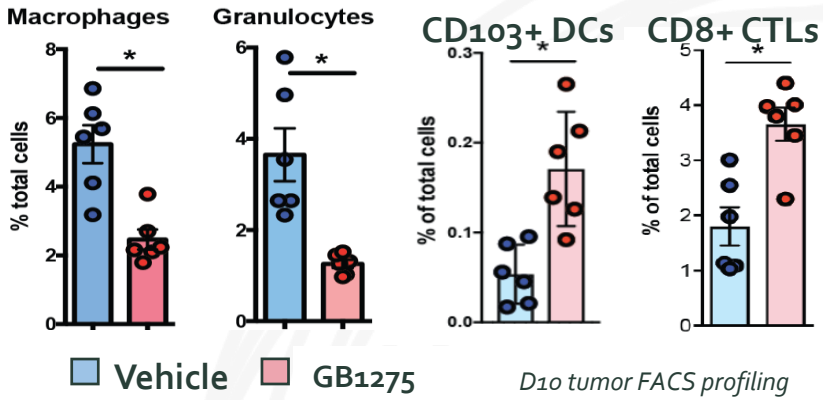
GB1275 Pre-clinical Data: Single Agent and Combination Activity

Single Agent GB1275 in Pancreatic Cancer Mouse Model

Survival: Control vs GB1275



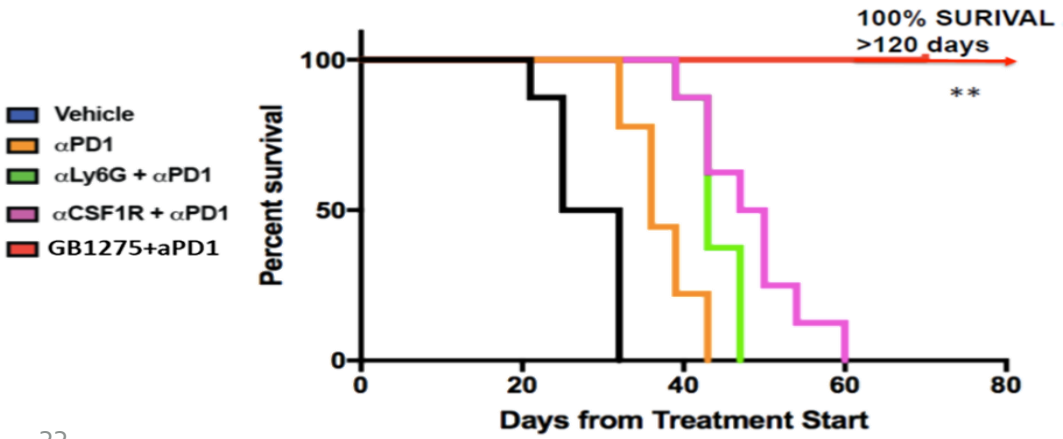
Tumor Biopsy: Biomarker Data



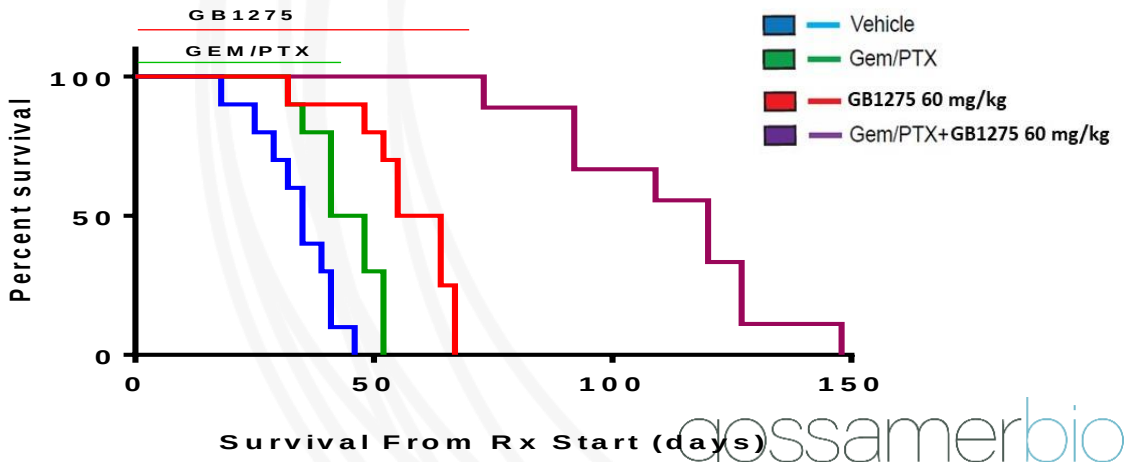
D10 tumor FACS profiling

Combination Data: GB1275+ Chemotherapy; GB1275 + anti-PD1 in Pancreatic Cancer Mouse Model

Survival: GB1275 + anti-PD1



Survival: GB1275 + Chemo



BID = twice daily dosing; PO = oral dosing; Gem = gemcitabine; PTX = paclitaxel; CTL = cytotoxic T lymphocytes.

Ongoing GB1275 Phase 1/2 Study of GB1275 In Select Solid Tumor Indications

A Phase 1/2, dose-ranging, signal seeking, clinical trial of GB002 in adult subjects with various types of solid tumors, including PDAC, gastric, esophageal, prostate, TNBC and CRC

Status: Enrolling, Initiated Q3:2019

Phase 1 of GB1275

3 Dose Escalations:
Monotherapy
Anti-PD1 combo
Gem / Abraxane combo

Phase 2 of GB1275

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

Patient Population	**Targeting IO resistant tumor types including:** Adult solid tumor patients with pancreatic cancer, gastric cancer, esophageal cancer, prostate cancer, triple negative breast cancer, and colorectal cancer
Treatments	Multiple doses of GB1275
Key Study Objectives	PK / PD, safety, tolerability, efficacy signals

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.

Financial Highlights and Milestones

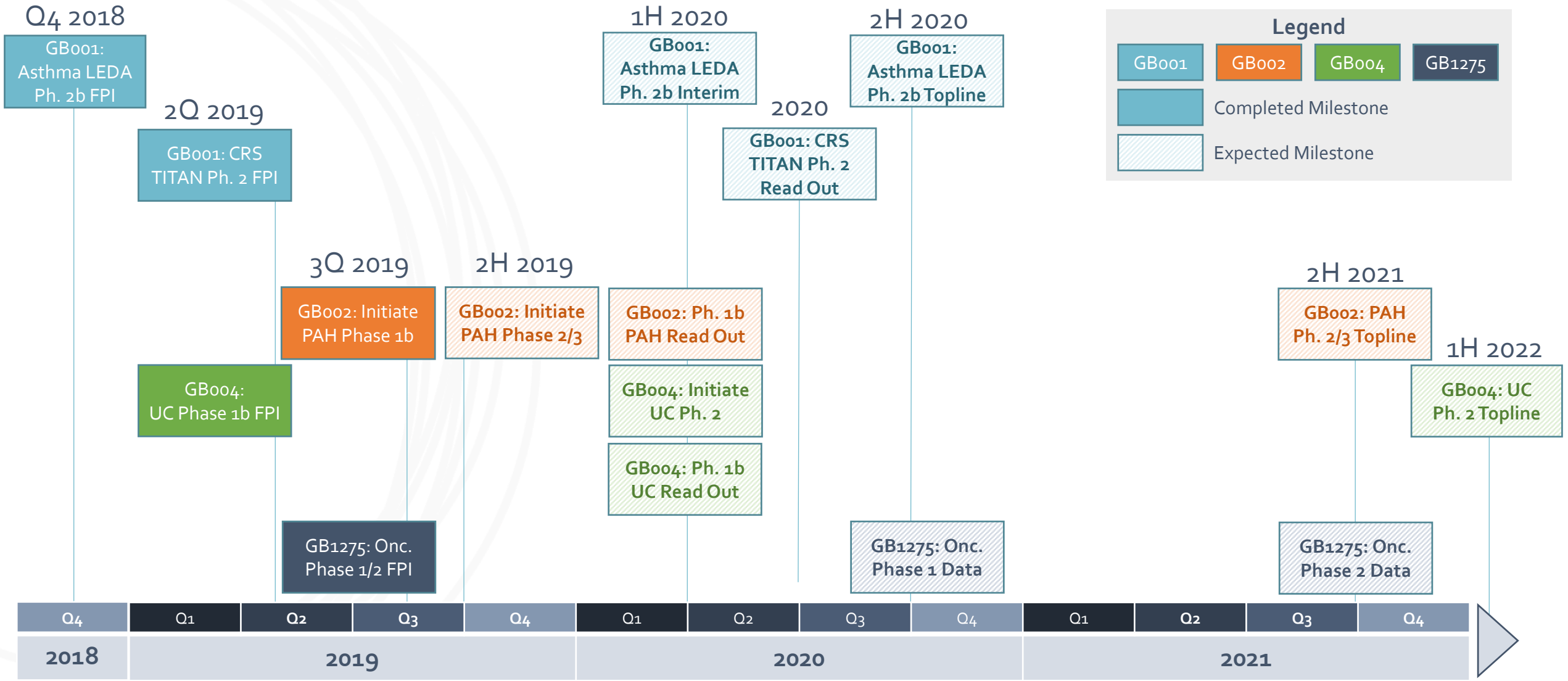
Financial Highlights

Cash, Cash Equivalents and Marketable Securities (As of 6/30/2019)	$464.0mm
Debt (Initial tranche of $150 million debt facility, announced 5/2/19)	$30mm
Debt Capacity (Remaining capacity of $150 million debt facility, announced 5/2/19)[1]	$120mm
Common Shares Outstanding (As of 8/5/2019)	65.9mm

mm = millions.

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

Multiple Near-Term Expected Clinical Trial Initiations and Readouts



Note: All trial bars and milestone flags are shown at the center of estimated timing. Trial initiation to occur at first patient dosed in trial.
Ph. = Phase; Interim = interim analysis; FPI = first patient in, Onc.= oncology.

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