UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 5, 2021

GALECTO, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-39655**	**37-1957007**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

Ole Maaloes Vej 3
DK-2200 Copenhagen N
Denmark
(Address of principal executive offices, including zip code)

(+45) 70 70 52 10
(Registrant's telephone number, including area code)

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trade Symbol(s)	Name of each exchange on which registered
Common Stock, $0.00001 par value per share	**GLTO**	**The Nasdaq Global Select Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 2.02. Results of Operations and Financial Condition.

As of December 31, 2020, Galecto Inc.'s cash, cash equivalents and investments balance was approximately $164 million.

Item 7.01. Regulation FD Disclosure.

Included as Exhibit 99.1 to this Current Report on Form 8-K is a presentation titled "First-in-class small molecule anti-fibrotic and anti-cancer agents" dated January 2021, which is incorporated herein by reference. We intend to utilize this presentation and its contents in various meetings with securities analysts, investors and others commencing on January 6, 2021.

The information in this Current Report on Form 8-K, including Exhibit 99.1, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Presentation titled "First-in-class small molecule anti-fibrotic and anti-cancer agents", dated January 2021.

SIGNATURE

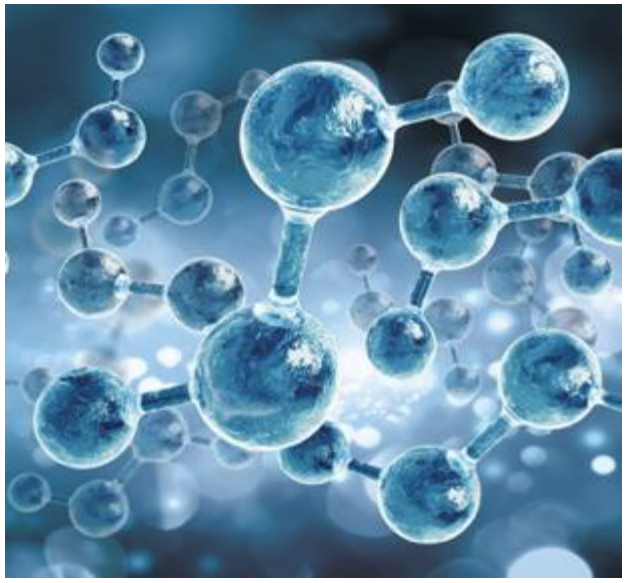
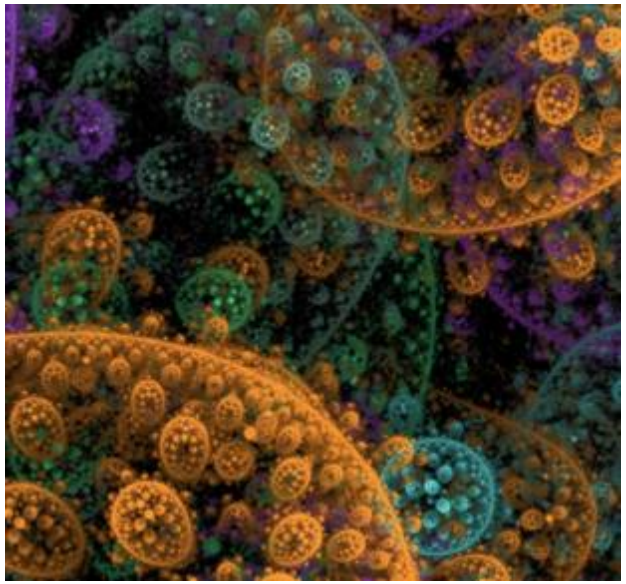
Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

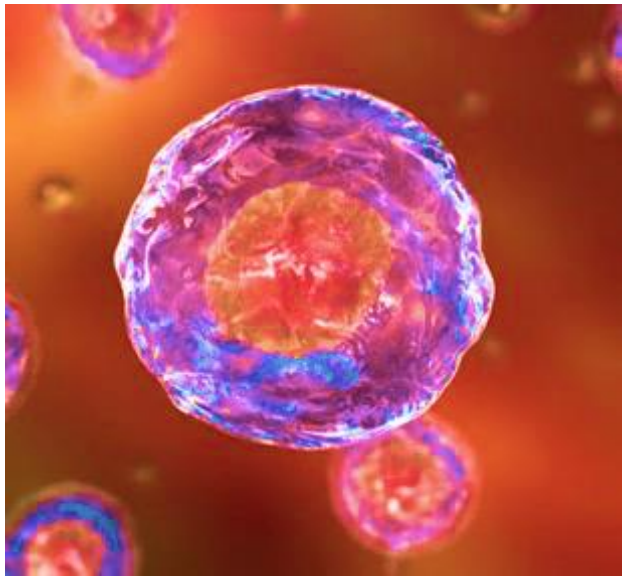
Galecto, Inc.

Date: January 5, 2021

By: /s/ Hans T. Schambye
Hans T. Schambye, M.D., Ph.D.
President and Chief Executive Officer

Galecto



First-in-class small-molecule anti-fibrotic and anti-cancer agents

January 2021

Forward-looking statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding Galecto, Inc.'s (the "Company") strategy, future operations, future financial position, projected costs, prospects, plans, and objectives of management, are forward-looking statements. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "project," "target," "should," "would," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. For such statements, we claim the protection of the Private Securities Litigation Reform Act of 1995. The Company may not actually achieve the plans, intentions, or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements. Factors that could cause actual results to differ materially from such statements include, without limitation: Drug development is expensive, time consuming, uncertain and susceptible to change, interruption, delay or termination; the timing and outcome of research, development and regulatory review and feedback is uncertain; we expect to need additional funds to advance all of our programs, and you and others may not agree with the manner in which we allocate our resources; the amount of our future losses is uncertain and could cause our stock price to fluctuate or decline; topline data may not accurately reflect the complete results of a particle study or trial; results of clinical trials and other studies are subject to different interpretation and may not be predictive of future results; new data may be unexpected or unfavorable; GB0139, GB1211, GB2064 or other drug candidate may not advance in development or be approve for marketing; clinal trial and other studies may not proceed at the time or in the manner expected or at all; enrolling patients in our ongoing and intended clinical trials is competitive and challenging; the duration and severity of the ongoing coronavirus disease (COVID-19) pandemic, including but not limited to the impact on our clinical and other operations, the operations of our suppliers, others and the capital markets, which in each case remains uncertain; clinical and nonclinical data is voluminous and detailed, and regulatory agency may interpret or weigh the importance of data differently and reach different conclusions than we or others, request additional information, have additional recommendations or change their guidance or requirements; data and information related to our program may not meet regulatory requirements or otherwise be sufficient for further development at all or on our projected timeline; other risks related to developing, seeking regulatory approval of and commercializing drugs, including regulatory, manufacturing, supply and marketing issues and drug availability; our and third parties' intellectual property rights; competition; reimbursement and pricing decisions; risk relating to relying on third parties; product liability and other litigation; and legislation and regulations. Additional factors that could cause results to differ materially from those stated or implied by our forward-looking statements are disclosed in our Securities and Exchange Commission (SEC) filings, including under the headings "Risk Factors." In addition, the forward-looking statements included in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing the Company's views as of any date subsequent to the date of this presentation.

Investment Highlights

Public (NASDAQ: GLTO), biotech with differentiated focus on fibrosis & cancer

- Potentially first-in-class with FDA and EMA orphan drug designation (ODD) for lead asset GB0139
- Small-molecule fibrosis inhibitors targeting Galectin-3 & lysyl oxidase-like 2 (LOXL2)

Strong pipeline with visible catalysts

- Phase 2b in idiopathic pulmonary fibrosis (IPF) with potential EU conditional approval
- To launch Phase 2 in myelofibrosis and NSCLC in 2021

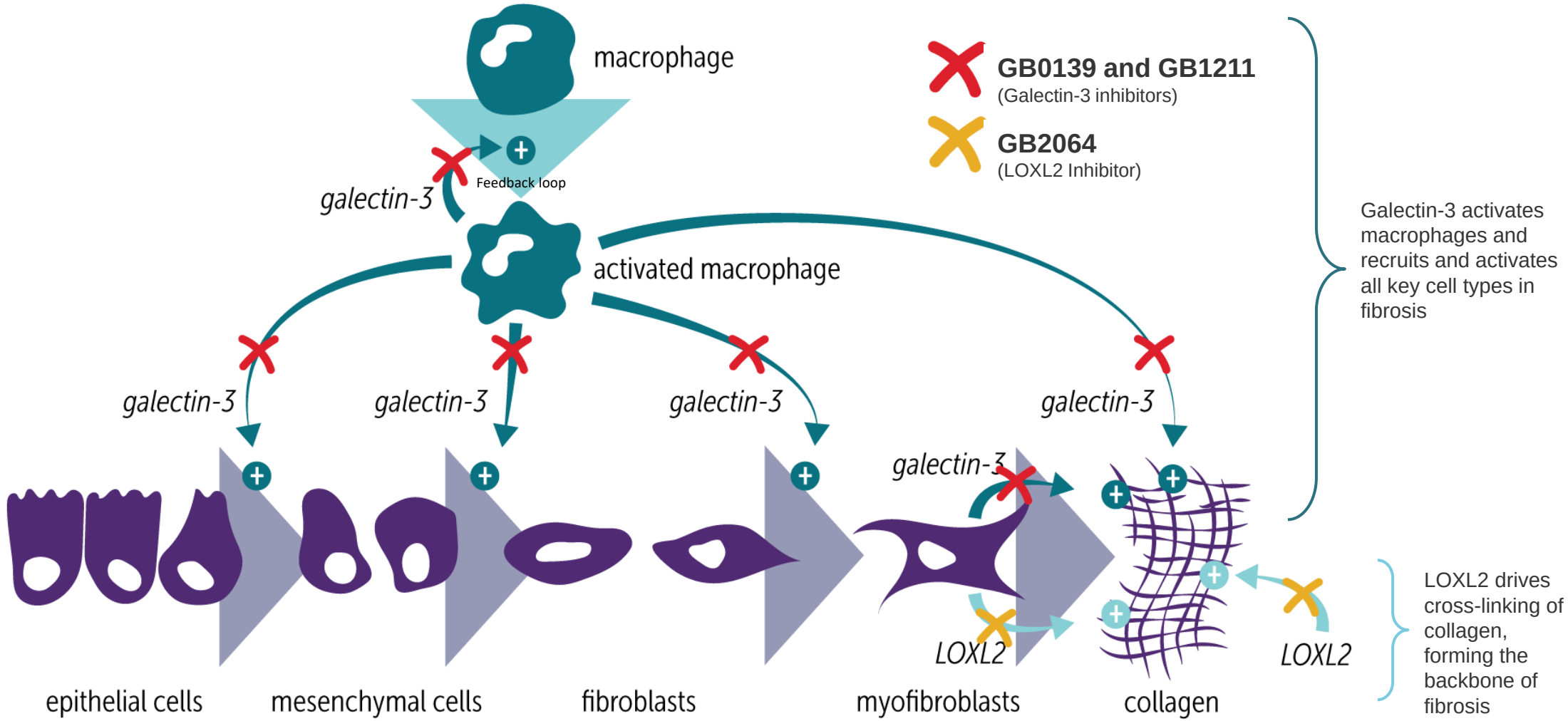
Raised ~$160M in 2H 2020 – Cash balance (Dec 31, 2020) ~$164M, funded into 2024

- $95 million in October 2020 IPO led by BoA, Leerink & Credit Suisse
- $64 million crossover in September 2020

Unique Pipeline Targeting Fibrosis and Cancer

Product Candidate	Indication	Preclinical Testing	Phase 1/2a	Phase 2b	Phase 3	Expected Next Steps	Potential Data Readout
GB0139	Idiopathic Pulmonary Fibrosis	(Inhaled Galectin-3 inhibitor)				*Potentially registrational in US and EU*	2022
GB2064	Fibrotic Indications (Initially in Myelofibrosis)	(Oral LOXL2 inhibitor)				Phase 2 start	2022
GB1211	Oncology	(Oral Galectin-3 inhibitor)				Phase 2a start	2022
GB1211	Fibrotic Indications (Initially in Cirrhosis)	(Oral Galectin-3 inhibitor)				Phase 2a start	2022

Galectin-3 and LOXL2: Targeting Serious Fibrotic Diseases Through Two Different Mechanisms of Action



GB0139: Inhaled Galectin-3 Inhibitor for IPF

IPF is a Large Orphan Indication with Suboptimal Solutions

Disease Overview

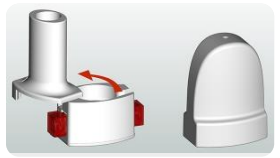
- **Approximately 100K patients in US**
- IPF is a progressive, irreversible, ultimately fatal lung disease characterized by decline in lung function (as measured by FVC)
- Lung tissue scars and becomes non-functional
- Median survival of 2-5 years
- Death caused by respiratory failure
- Unknown cause

Limited Treatment Options

- Only two approved drugs to slow disease progression: **Pirfenidone** and **Nintedanib**
  - Neither has been associated with improvements in overall survival
  - Both have significant side-effects that limit compliance and usage
- **Due to side effects, less than 50% of patients on treatment**
- Despite dose-limiting side effects, sales of pirfenidone and nintedanib in 2019 exceeded $2.8B in the aggregate

GB0139: Inhalable, Once-Daily Treatment for IPF
Potential for Accelerated Approval

Superior Delivery



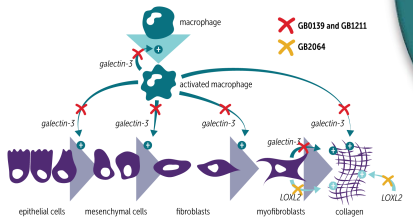
- Inhaled therapy via generic inhaler delivers therapy directly to target tissue with low systemic exposure
- Competing clinical development candidates given intravenously, subcutaneously and orally

Well Tolerated

- Well tolerated in clinical trials and long-term toxicology studies to date
- Existing therapies marred by significant side effects leading to approximately 50% discontinuations

GB0139

Pluripotent MoA



- Unique and pluripotent MoA
- GB0139 inhibits fibrosis by targeting macrophages, fibroblasts, and epithelial cells

Confirmed Target Engagement

- GB0139 reduced macrophage galectin-3 levels in lungs of IPF patients
- Dose-response effects on several fibrosis plasma biomarkers
- No other therapy in development has demonstrated similar consistent effects

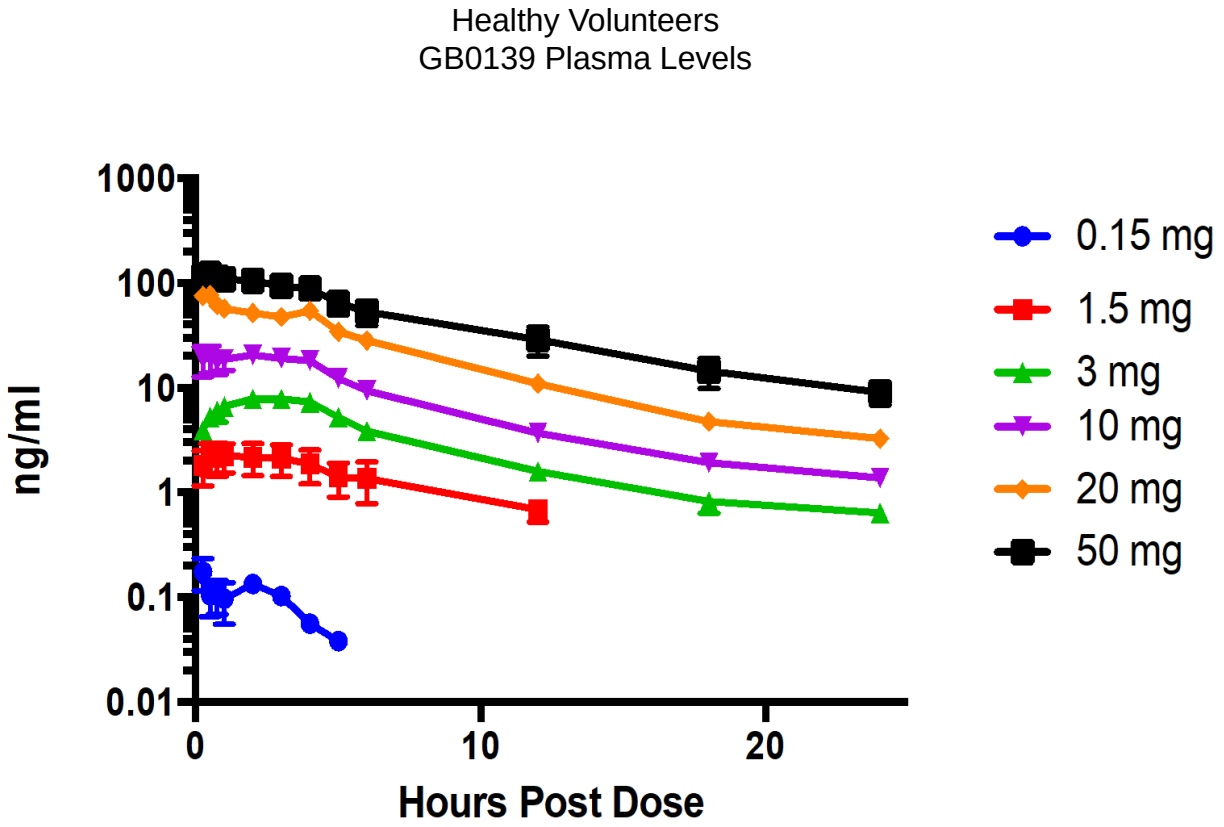
GB0139: Results of Phase 1 SAD Study

Phase 1 Trial Design

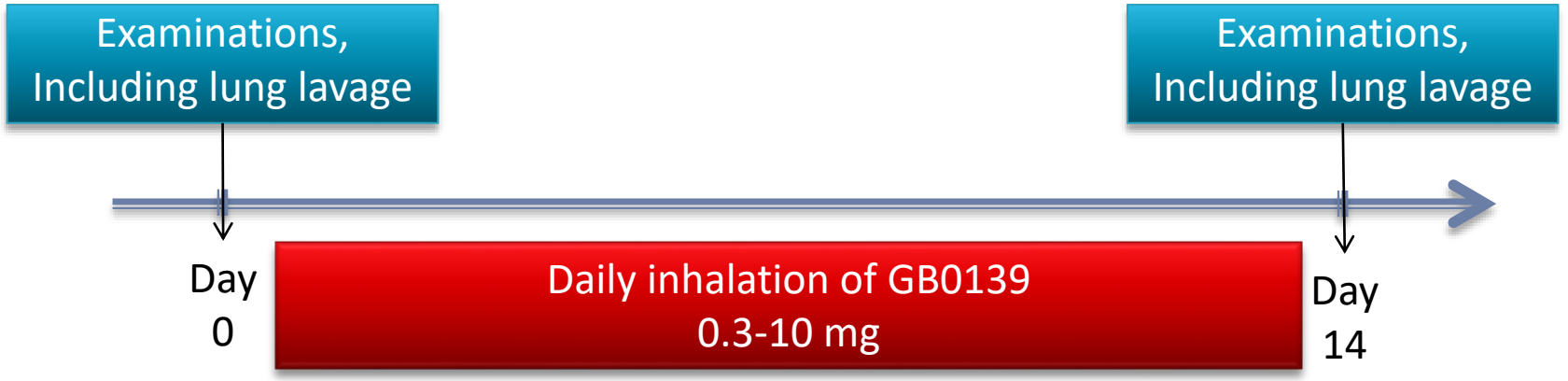
- 6 dose groups (0.15, 1.5, 3, 10, 20 and 50 mg)
- 4 active patients and 2 placebos in each group

Key Phase 1 SAD Study Results

- Highly reproducible pharmacokinetic (PK) profile and dose-dependent exposure
- Mild adverse events (AE) only (cough & headache)
- All lab and other clinical parameters satisfactory
- Generic inhaler performing well



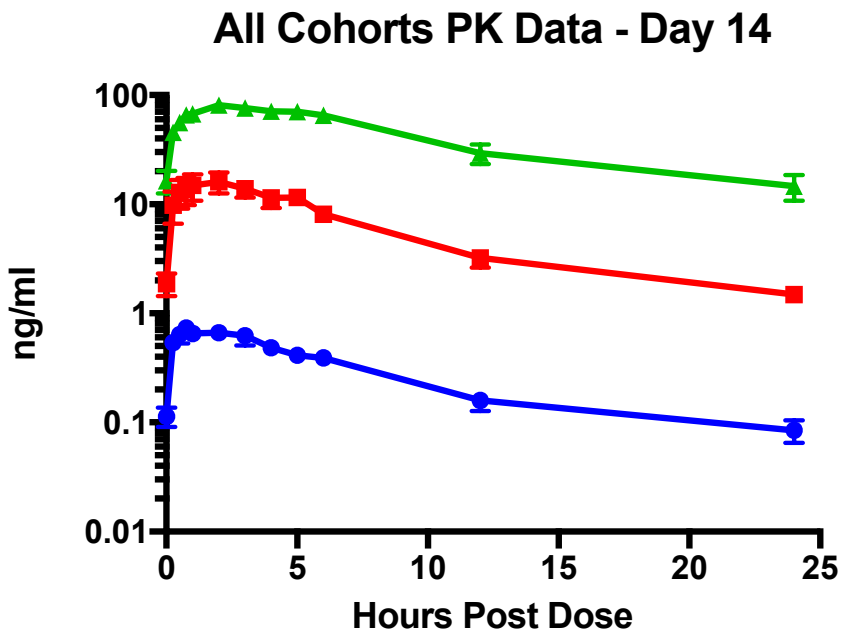
GB0139: Phase 2a Patient Study in IPF Patients



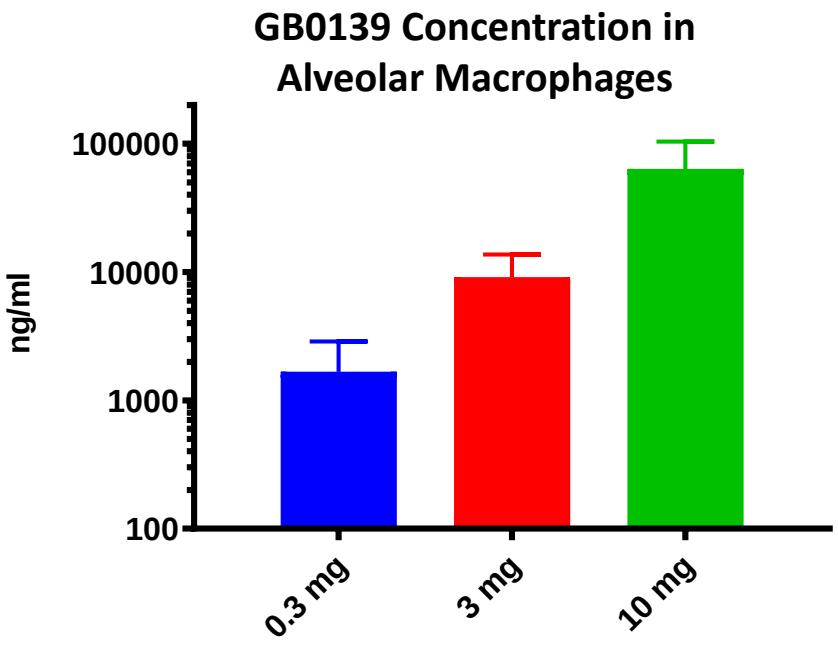
- 24 patients in 3 dose groups
  - Double-blind, placebo-controlled, multicenter
  - Doses: 0.3, 3 and 10 mg per day
  - 5 active patients and 3 placebos per group
- Four centers in the UK
- All patients completed 2 week dosing as planned
- Evaluable bronchoalveolar lavages (BALs) obtained for all 48 bronchoscopies

GB0139: Phase 2a Result - Bioavailability & Target Engagement in IPF Patients

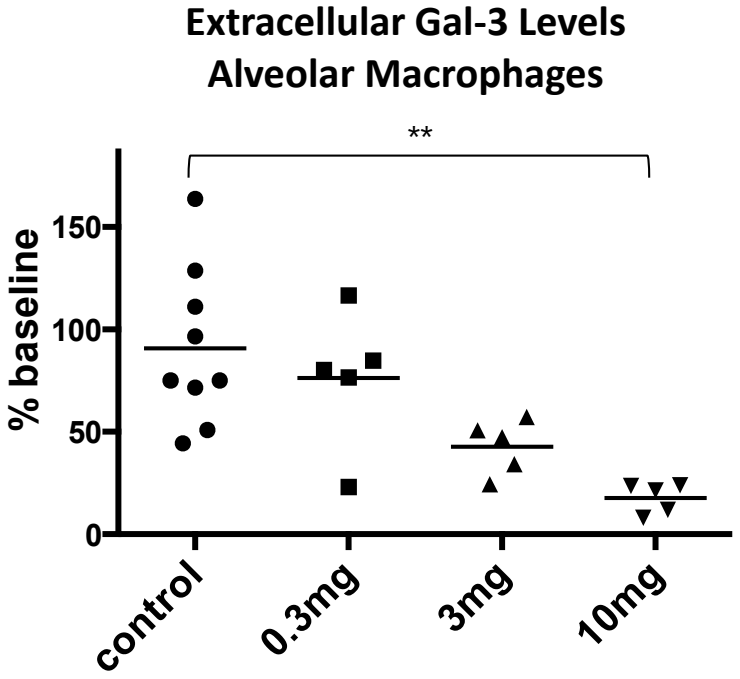
Exceptionally Consistent PK Data in IPF Patients



GB0139 Reaches the Alveolar Macrophages Deep within Lung

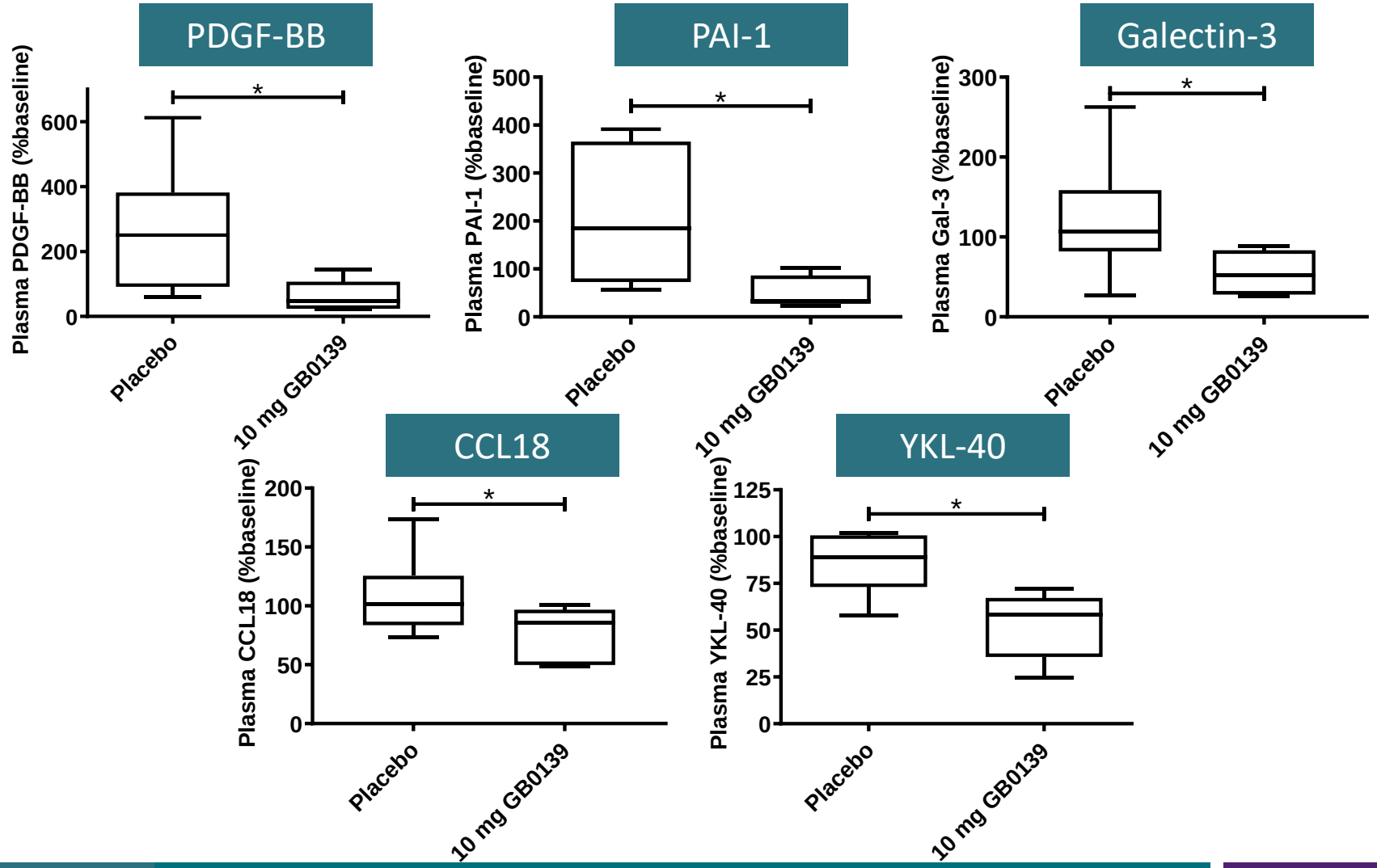


Induces Profound Reduction of Gal-3 Levels on Alveolar Macs



GB0139: Phase 2a Study Showed Consistent, Dose-Dependent Effects on Highly Relevant Fibrosis Biomarkers

- Biomarkers associated with IPF disease severity and progression had biggest impact
- Biomarker effects cited by EMA as clinically relevant in IPF patients and basis for ODD
- BAL fluid and plasma correlation indicates GB0139 directly impacts lung function

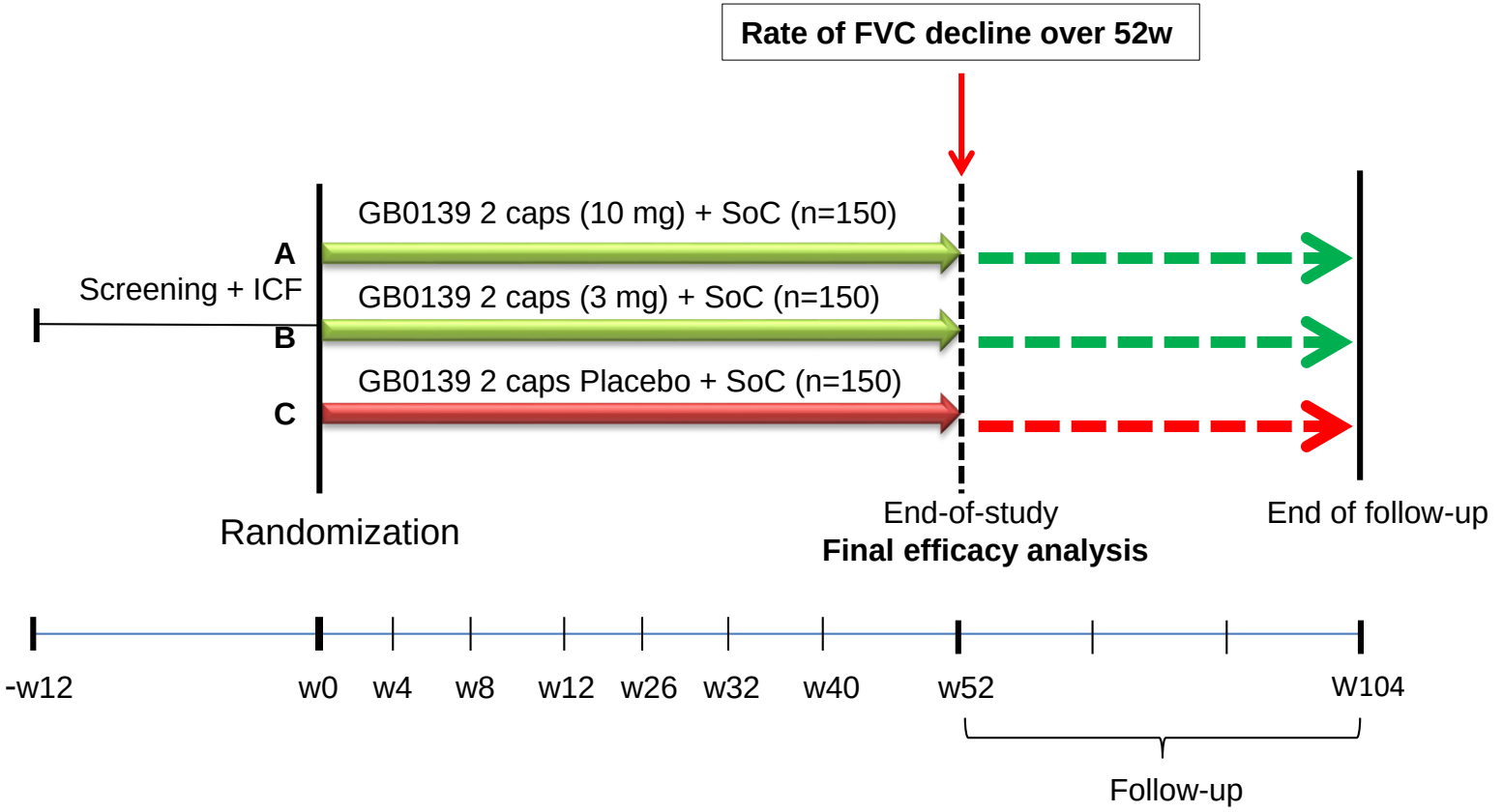


Comparison: GB0139 MoA to alternative programs

MoA	GB0139	Alternative programs
Targets macrophages	Yes	1 program
Targets fibroblasts	Yes	Most programs
Targets epithelial cells	Yes	No
Lowers TGF-ß	No	Some programs
Modulates TGF-ß	Yes	No
Affects multiple cytokines (PDGF, CTGF, TGF-ß, LPA, VEGF, etc.)	Yes	1 program (nintedanib)

GB0139: Phase 2b IPF Study In Progress

- Randomized, placebo controlled 52 week study
- 450 IPF patients in 3 arms
- ~100 centers
- First-patient, first-visit in Q1 2019
- Expected completion in mid 2022
- Primary outcome measure:
  - Annualised rate of FVC decline over 52 weeks
  - Study is also powered to see an effect in the patients on neither pirfenidone nor nintedanib
- Key secondary outcomes:
  - Safety, DLCO(*), 6 minute walk test, Quality of Life



ICF - Informed Consent Form, FVC - Forced Vital Capacity, R - Randomization, w - week, n - sample size, SoC - standard of care
"2 caps" – GB0139 5mg x 2 or TD139 1.5mg x 2, respectively GB0139 Placebo

* DLCO – Diffusing Capacity for Carbon Monoxide

GB0139: Inhaled Galectin-3 Inhibitor Summary

- Ground-breaking novel treatment of IPF, an orphan disease with poorly tolerated treatments
- Inhaled, delivered directly to the site of active lung destruction
- Well tolerated in trials to date
- Reaches the target cell in the lung at high concentrations
- Causes a dose-related reduction in cell surface galectin-3 deep within patient lungs
- Promising biomarker trends observed in Phase 2a study - validated by EMA as clinically relevant in IPF patients and basis for ODD
- Phase 2b study underway – FDA and Medicines and Healthcare Products Regulatory Agency (MHRA) approved design
- **Conditional approval possible after Phase 2b completion**

GB2064: LOXL2 Inhibitor for Myelofibrosis and Other Fibrotic Diseases

GB2064: Oral LOXL2 Inhibitor in Myelofibrosis

Overview and Treatment Opportunity

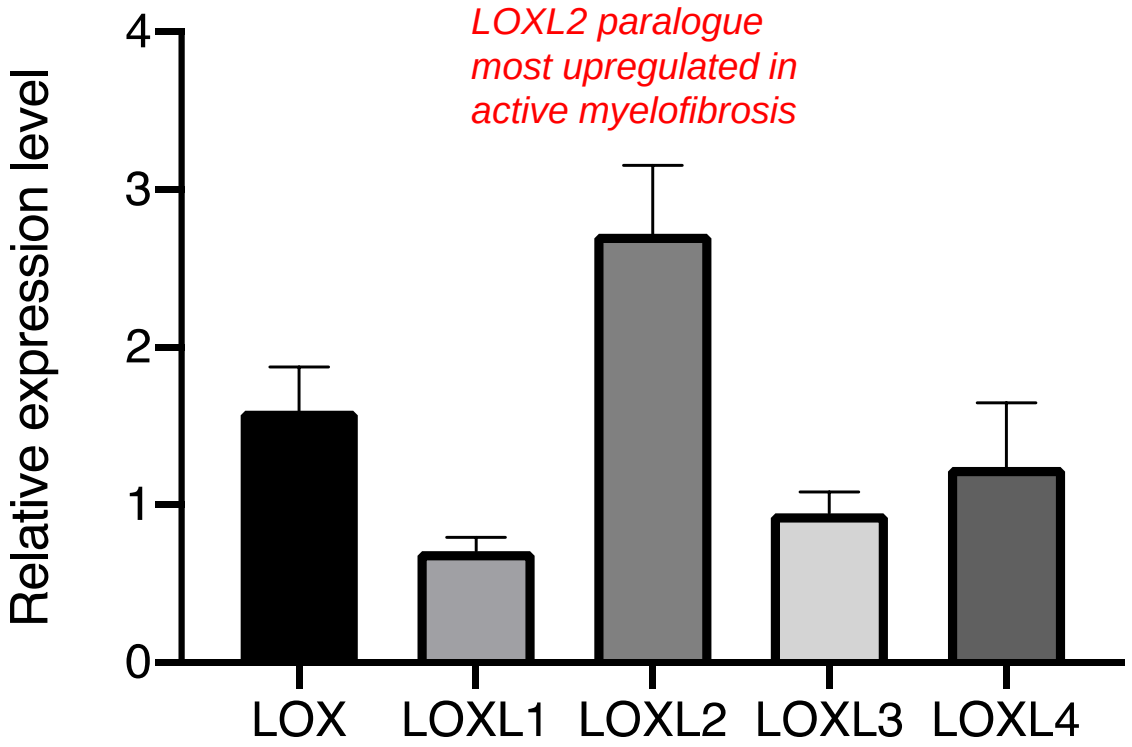
GB2064 (previously PAT-1251)

- A small-molecule inhibiting LOXL2, an enzyme that catabolizes the formation of lysine cross-linking in fibrillar collagens
- Potentially disease modifying
- Opportunity in multiple fibrotic indications

Myelofibrosis:

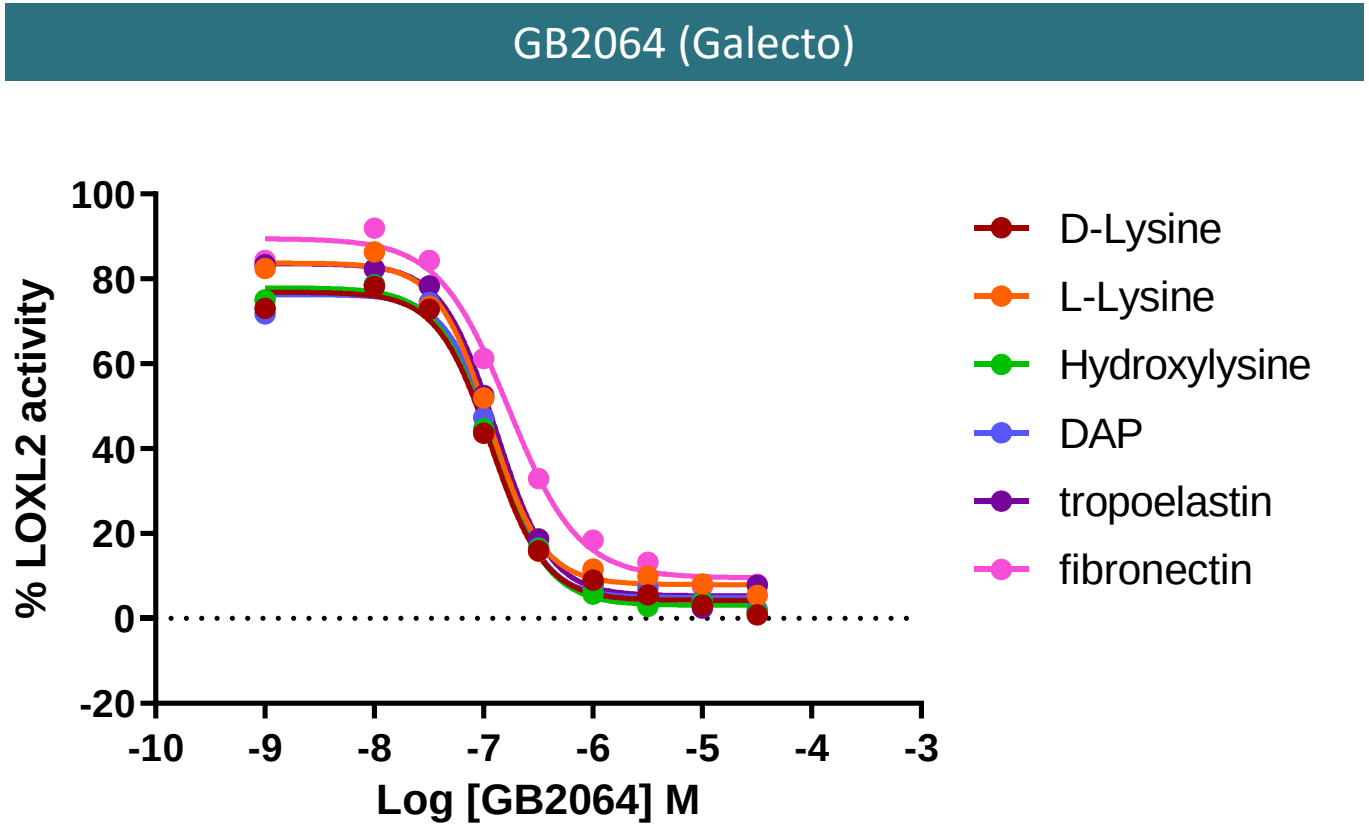
- Orphan indication: 16,000 - 18,500 patients in US
- Current therapies (JAK inhibitors) are not disease modifying
- Large market - Incyte's Jakafi achieved $1.7bn in 2019 sales

LOX Family Gene Expression in Myelofibrosis Stromal Cells



GB2064: Demonstrated *In Vitro* Inhibition of LOXL2

- GB2064 is a small-molecule inhibitor of the LOXL2 catalytic site, not an antibody approach
- GB2064 therefore has the potential to avoid the *in vivo* target low tissue penetration and target engagement encountered by Gilead's simtuzumab
- GB2064's superior efficacy to simtuzumab has been observed in cell-based assays and preclinical models



- GB2064 fully inhibits LOXL2 activity using a variety of substrates

MYLOX-1: GB2064 in Myelofibrosis

- Study to be led by Professor Srdan Verstovsek, MD Anderson
- Single arm open label study, allowing real time read of safety and activity
- Patients who are ineligible for JAK-inhibitors or who do not tolerate JAKi
- Biomarkers for:
  - blood formation
  - bone marrow general histology and fibrosis
  - PK to show drug levels in target tissue
  - imaging for spleen and liver volume

GB2064: Phase 2a in Myelofibrosis Summary

- Ample evidence for central role of LOXL2 in fibrosis
- GB2064 potently inhibits LOXL2 and shows antifibrotic activity in numerous models

- Upcoming Phase 2a could generate both target engagement and efficacy data in the same study as repeated biopsies are already standard practice
  - Opportunity for both orphan designation and fast track following data in this indication

- IND approved October 2020, and Phase 2a to start in coming months
  - Phase 1 SAD/MAD study already completed
  - Chronic toxicology studies completed
  - Robust efficacy in lung, liver and kidney models

GB1211: Oral Galectin-3 Inhibitor for Cancer and Fibrosis

Galecto's Oral Galectin-3 Inhibitors

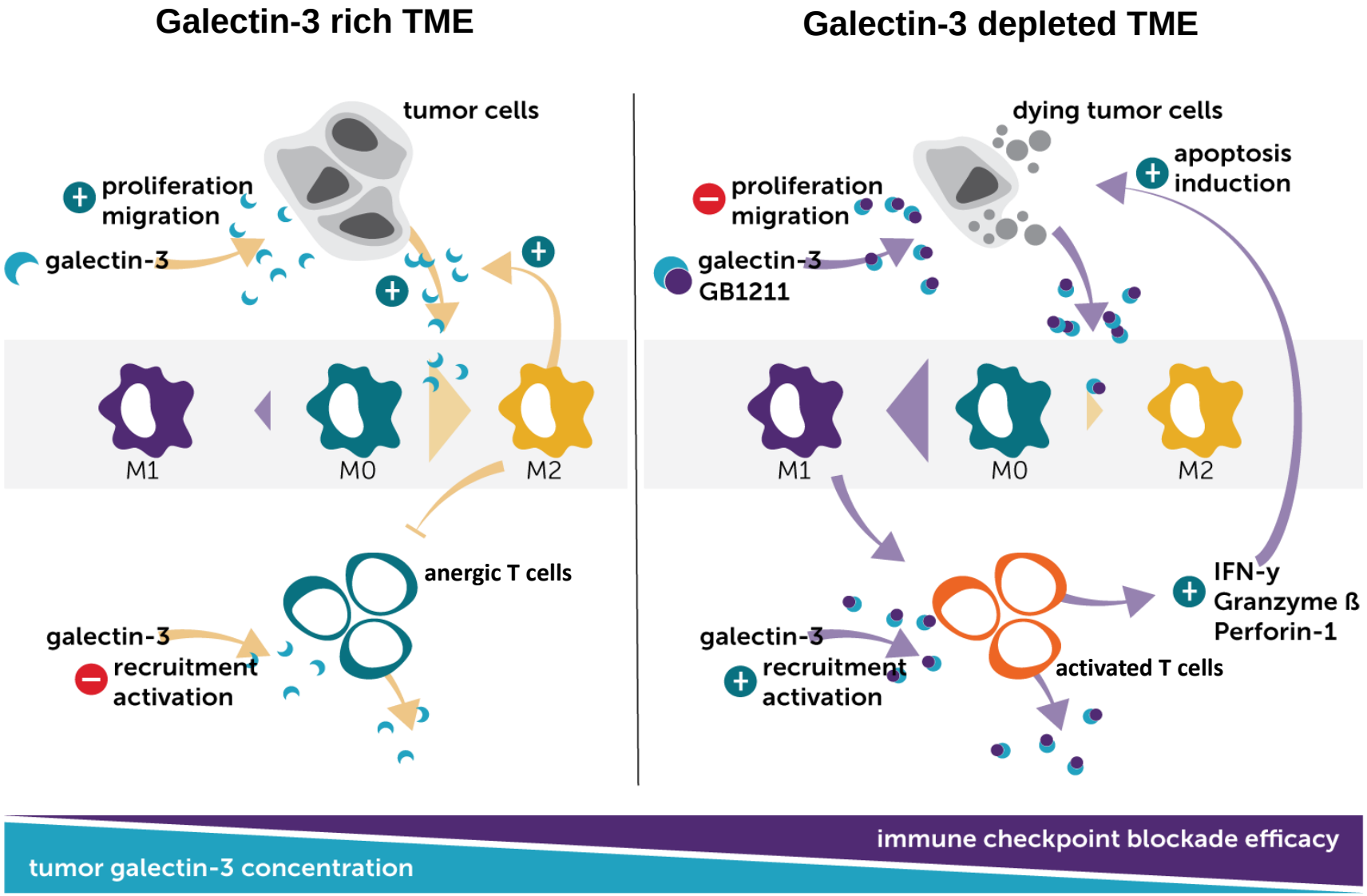
- Galectin-3 is a very attractive target for a series of currently poorly treated indications, including many cancer types and fibrosis
- Galecto is pioneering galectin field and has developed a series of orally active, specific and potent inhibitors of galectin-3
  - These bind to the carbohydrate recognition domain and inhibit the galectin-3 related effects on several key cell types
- The lead compound, GB1211, reduces fibrosis and cancer growth in several different animal models
  - Well tolerated in IND-enabling studies
- SAD/MAD in healthy volunteers successfully completed
- Galecto plans to develop GB1211 for both cancer and fibrotic diseases

Galectin-3 Inhibitors Are an Exciting Novel Therapy for Cancer

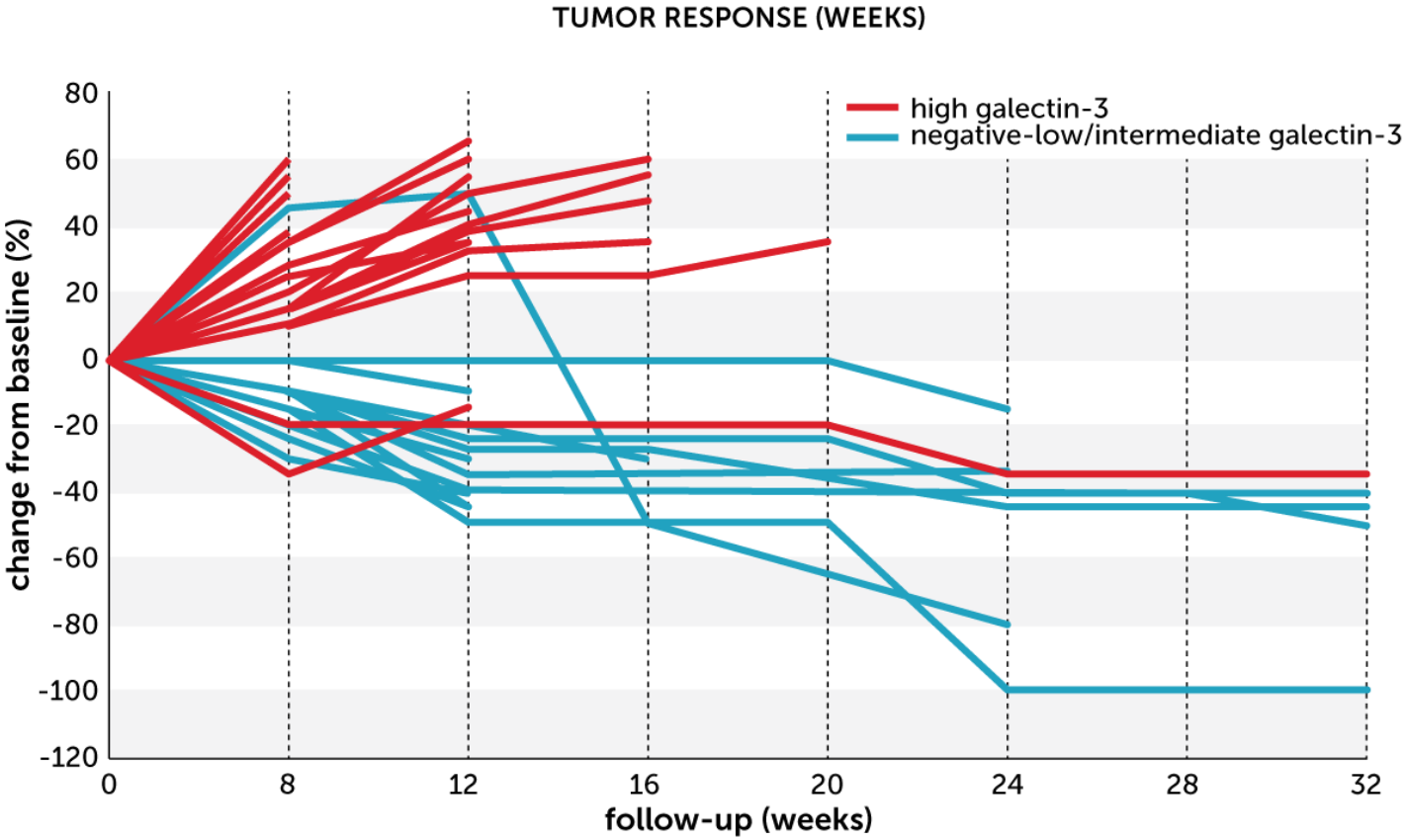
- Galectin-3 acts as a guardian of the tumor micro-environment (TME) and a negative regulator (checkpoints) of immune cell functions
- *In vivo* galectin-3 inhibition reprograms tumor associated macrophages, increases T-cell recruitment and cytotoxic T-cell function
  - Expression is linked to metastasis, progression and prognosis of lung cancer and other highly fatal cancers
  - Galectin-3 causes T-cell disorientation by removing interferon-gamma gradient and T-cell anergy by binding to LAG-3
  - Involved in K-Ras signaling
- Inhibiting galectin-3 has direct anti-cancer effects particularly in adenocarcinomas and RAS mutated cancers including lung, pancreas and gastric
- High galectin-3 expression is linked to poor prognosis and poor response to checkpoint inhibitors in non-small cell lung cancer (NSCLC)

LAG-3 – *lymphocyte-activation gene 3;* K-Ras – *protein produced by the Kirsten rat sarcoma gene;* RAS – *reticular activating system*

Galectin-3 Creates an Immune Compromised Tumor Micro-Environment (TME)



Galectin-3 Predicts Poor Response to Pembrolizumab in NSCLC



34 patients with PD-L1-positive NSCLC stage IV received pembrolizumab (200 mg IV @ 3 wks).

- Tumour resistance to pembrolizumab strongly correlated with high expression of galectin-3 in NSCLC
- A clinical response was seen in tumours with a negative, low or intermediate galectin-3 expression
- We aim to use an oral galectin-3 inhibitor GB1211 to increase clinical response to Pembrolizumab in galectin-3 high

From Capalbo et al., 2019, Int. J. Mol. Sci. 20.

GB1211: Oral Galectin-3 Inhibitor Cancer Program

- Extensive preclinical evidence for galectin-3 as a key target in many cancers
- GB1211 has completed clinical Phase 1 testing
  - Demonstration of anti-cancer effect in several preclinical models
  - Chronic toxicology studies (dog and mouse) completed with no drug related adverse events
  - SAD/MAD in healthy volunteers successfully completed
- NSCLC selected as initial indication
  - Major unmet medical need
  - Clinical data suggest galectin-3 high NSCLC tumors are resistant to anti-PD-1
  - Potential for turning cold tumors hot with GB1211 in combination with checkpoint inhibitors
- Current status & next steps
  - Design of phase 2 combination study in NSCLC ongoing
  - First patient dosing planned for 2H 2021

GB1211: Oral Galectin-3 Inhibitor for Liver Cirrhosis

GB1211: Oral Galectin-3 Inhibitor for Advanced Liver Cirrhosis

Disease Overview

- Cirrhosis prevalence: ~2 million patients in US, ~3 million in EU[1]
- Severe, progressive liver fibrosis that ultimately leads to liver failure
- Caused primarily by NASH, alcoholic liver disease, viral hepatitis
- Median survival of ~2 years for decompensated cirrhosis[2]
- Limited treatment options:
  - Resolving etiology may improve decompensation e.g., alcohol abstinence, HCV/HBV antivirals
  - Liver transplantation

Galectin-3 in advanced cirrhosis

- Evidence links galectin-3 to cirrhosis progression:
  - Galectin-3 is elevated in alcoholic and non-alcoholic cirrhosis and in toxic hepatitis
  - Galectin-3 is prognostic biomarker of hepatocellular carcinoma, a know complication of liver cirrhosis
- Preclinical data suggest galectin-3 inhibition may address cirrhosis:
  - Inhibition of galectin-3 reduces development of fibrosis
  - Galectin-3 is required for TGF-beta mediated myofibroblast activation and matrix production in liver fibrosis
  - Galectin-3 inhibition reduces YKL-40, a biomarker that is elevated with progressive liver fibrosis

[1] https://www.thelancet.com/journals/langas/article/PIIS2468-1253(19)30349-8/fulltext Sepanlou, et al Lancet Gastroenterol Hepatol 2020; 5: 245–66
[2] https://www.hepatitis.va.gov/cirrhosis/background/stages.asp

GB1211 Hepatic Impairment Study

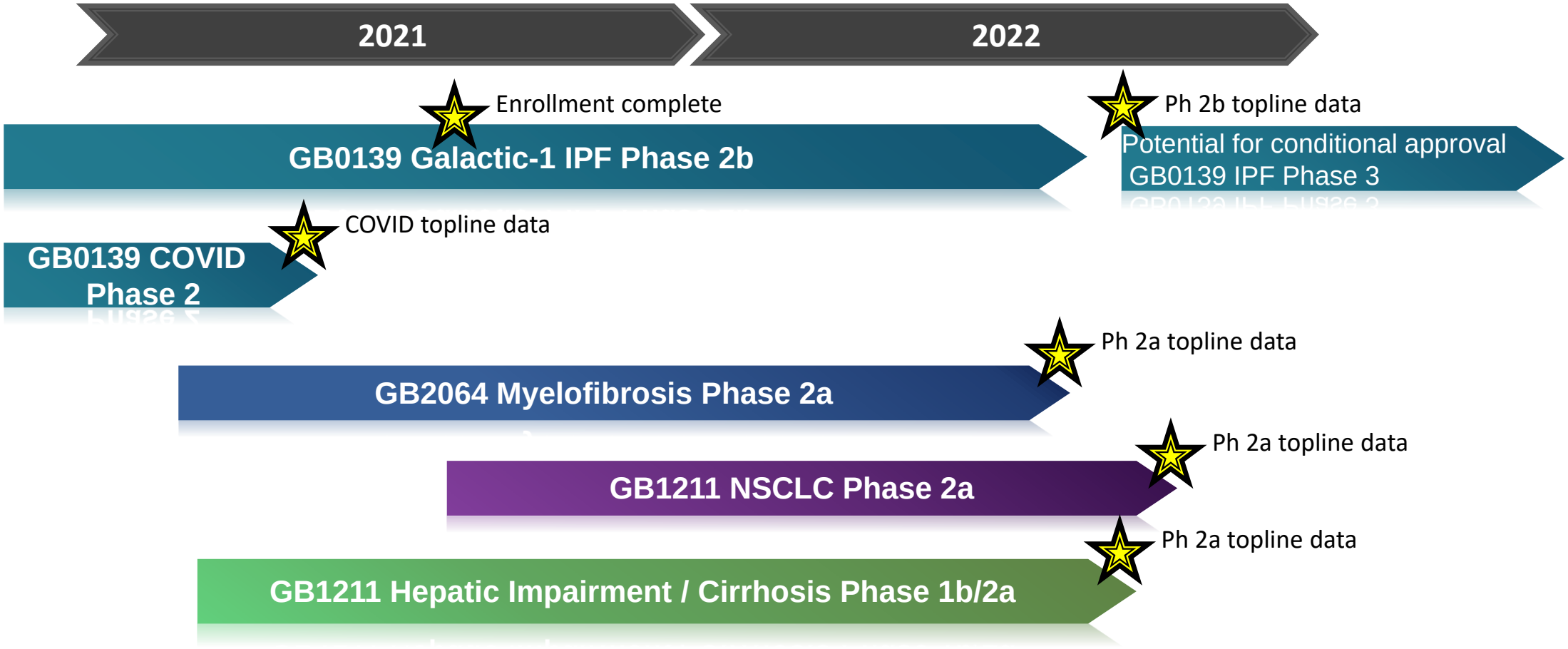
- Single and multidose PK in Child-Pugh B/C patients, including NASH and non-NASH cirrhosis
- Expansion cohort with biomarkers of:
  - Fibrosis
  - Portal hypertension
  - Liver function
- This study will illustrate both safety and drug activity on fibrosis and liver function
- If safe and initial signs of activity are present, we will plan placebo-controlled study

GB1211: Oral Galectin-3 Inhibitor for Liver Cirrhosis

- Extensive preclinical evidence for galectin-3 as a key driver in fibrosis in many organs
  - Directly addressing fibrosis not targeted by current experimental therapies
- GB1211 has completed clinical Phase 1 testing
  - SAD/MAD in healthy volunteers successfully completed
- Current status & next steps
  - IND opened in Q1 2020
  - Hepatic impairment study planned for 1H 2021
  - Study in liver cirrhosis is currently being designed

Wrap-Up

Pipeline and Clinical Development Timeline



Galecto Investment Opportunity - Well-funded with Multiple Shots on Goal

- Publicly-traded (NASDAQ: **GLTO**), US-based biotech developing small-molecule fibrosis inhibitors targeting two essential proteins:
  - **Galectin-3**: activator and recruiter of many cell types involved in fibrosis, guardian of tumor micro-environment
  - **LOXL2**: a key enzyme involved in the formation of the extracellular matrix, linked to worse prognosis in several cancers

- Galecto expects to have **three Phase 2 programs** underway in the coming months and **funded into 2024**

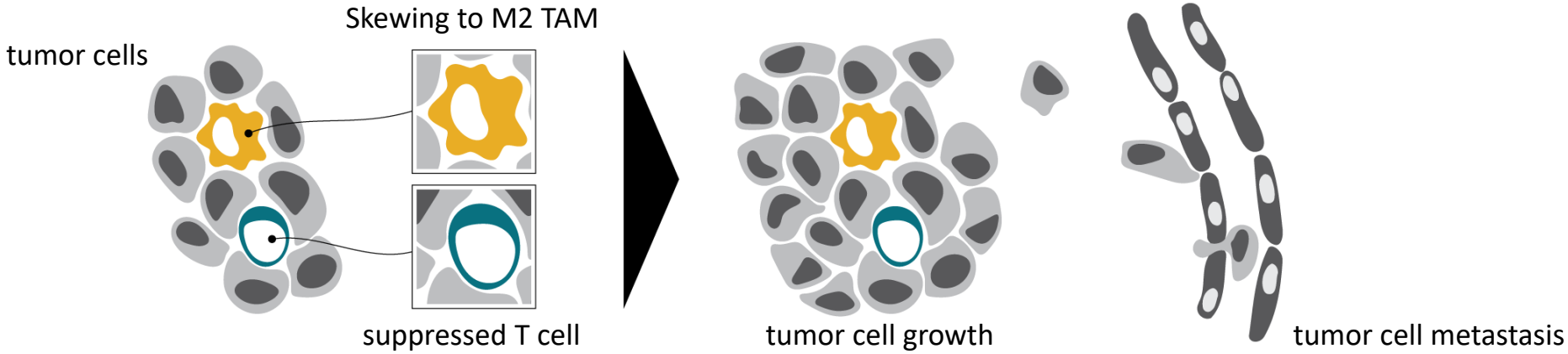
- Lead program, the inhaled GB0139, is in a randomized, controlled pivotal size Phase 2b study in IPF, a poorly treated orphan disease
  - Potential for EU conditional approval following positive Phase 2b results, completion expected mid 2022
  - Prior Phase 2a results in IPF show strong target engagement, deep inside the lungs, and effects on key fibrosis plasma biomarkers, along with a safe clinical profile

- Two additional programs ready for launch of Phase 2a trials
  - GB2064 in myelofibrosis: Phase 2 potentially will show both efficacy and target engagement data; possibility for orphan designation and fast track
  - GB1211 in NSCLC in combination with checkpoint inhibitor in galectin-3 high lung tumors

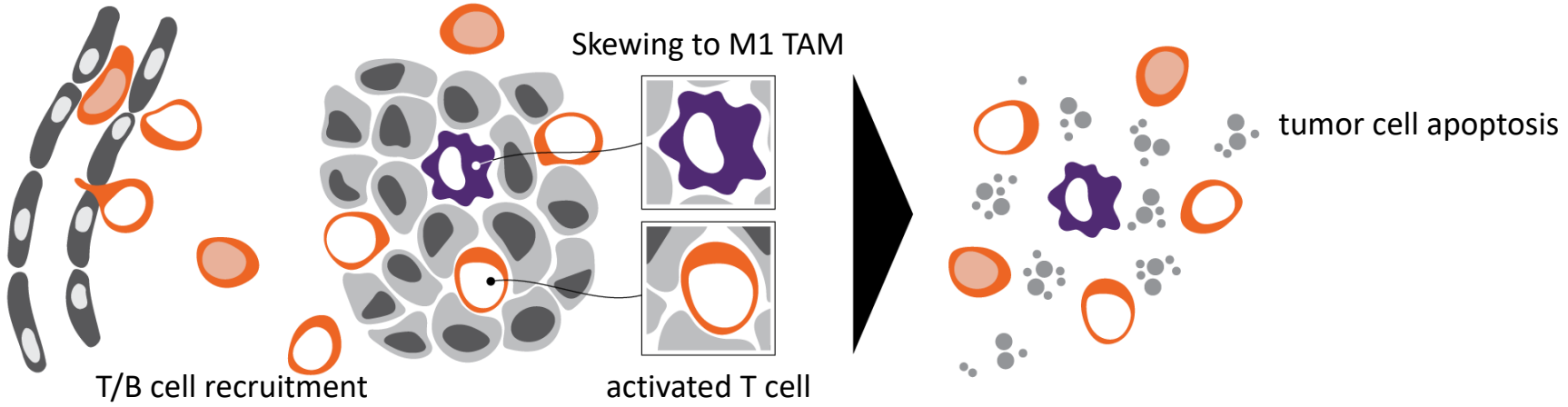
Appendix

Galectin-3 Inhibition Modulates the TME

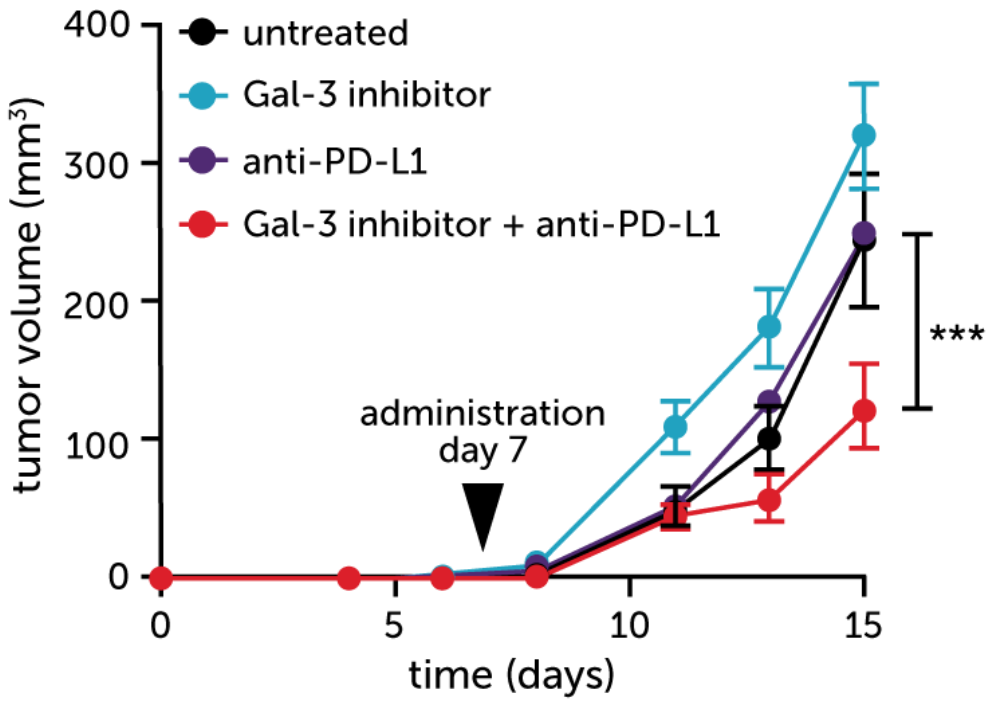
Galectin-3 rich Tumor Microenvironment

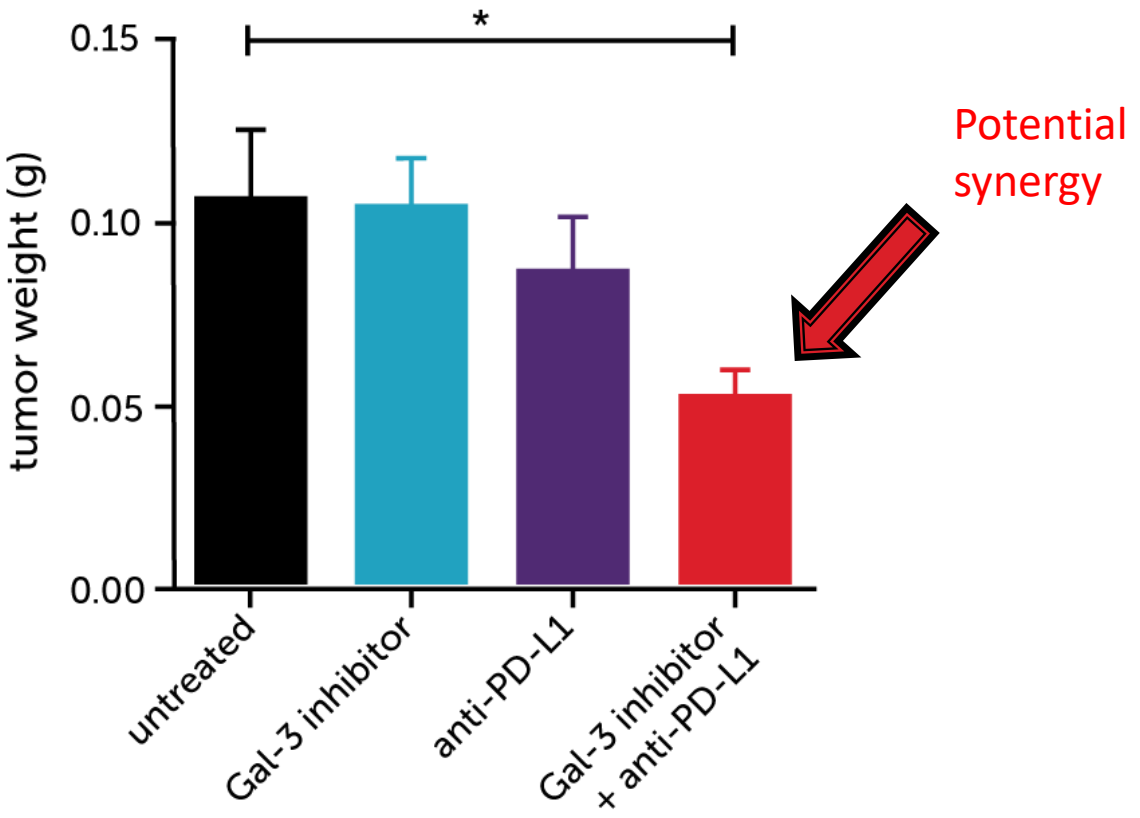


Treatment with GB1211 and anti-PD1



Therapeutic Administration of Galectin-3 Inhibitor in Combination with Anti-PD-L1 Inhibits Lewis Lung Carcinoma Growth

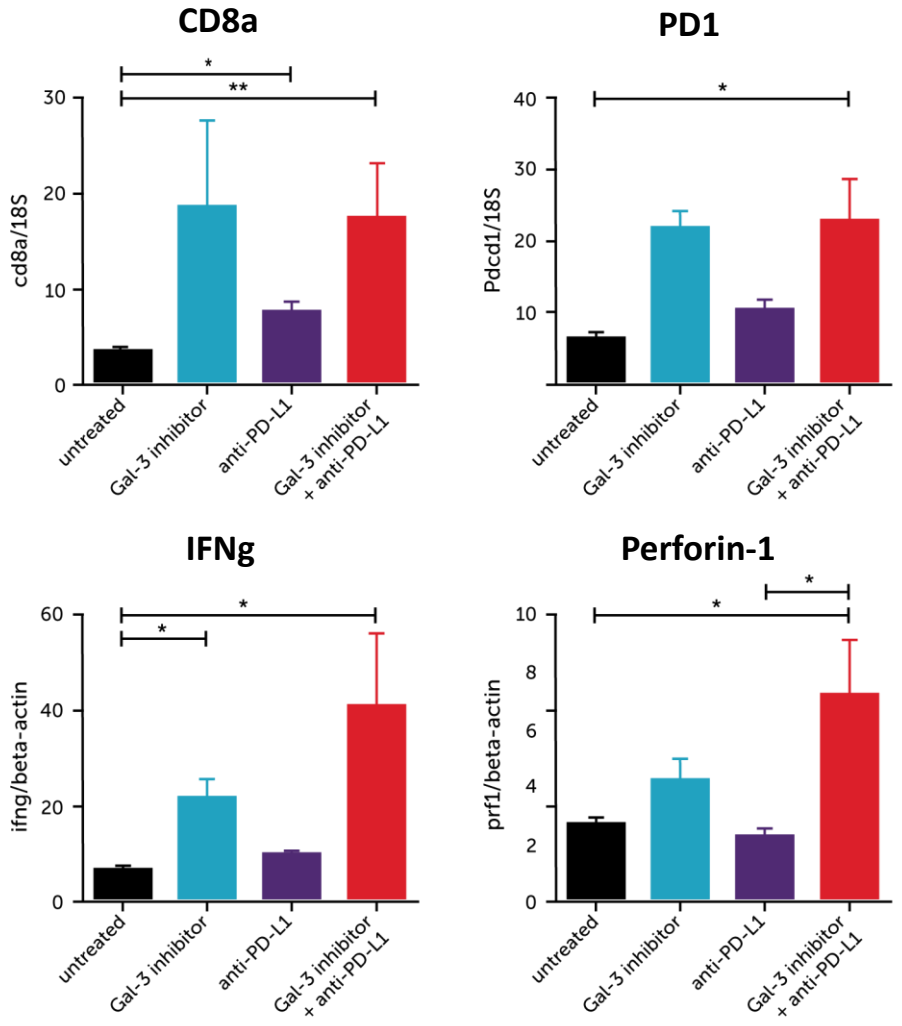




Vuong, L.,et al. (2019) Cancer Res 79, 1480-1492

Galectin-3 Inhibitor Increases Immune Response

- Galectin-3 inhibitor + anti-PD-L1 increases proliferating ki-67+ CD8 cells
- Galectin-3 inhibitor increases recruitment of CD8+ T cells and tumour cytotoxic T cell function
- Galectin-3 inhibitor increases INF-γ and PD-1 both associated with increased response to checkpoint inhibitors



Vuong, L.,et al. (2019) Cancer Res 79, 1480-1492

GB1211: Clean Safety Profile in Phase 1 SAD/MAD Studies

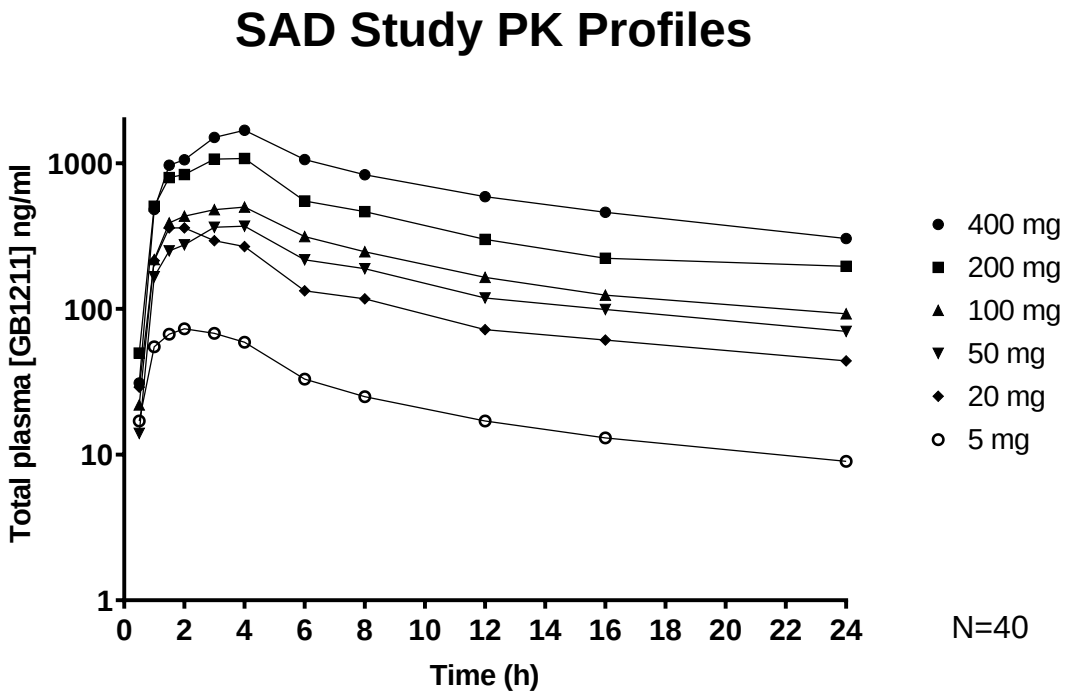
Part A: SAD in HV
N=40 across 5 cohorts

↓

Part B: 10-day MAD in HV
N=20+ across 2-3 cohorts

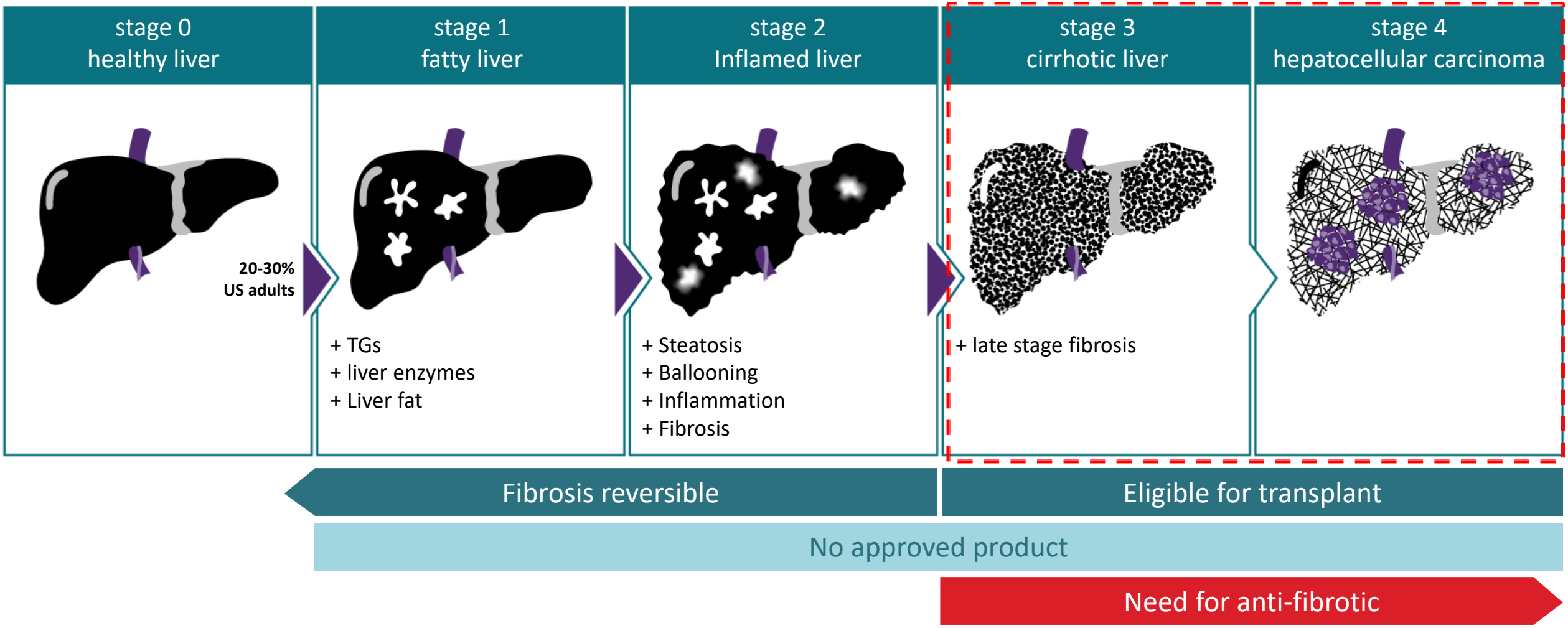
Completed

- Well tolerated:
  - Part A: <400mg in SAD studies
  - Part B: 50mg and 100mg in MAD studies, 2x daily
- Potential for once-daily dosing
- Exposure close to predicted with dose proportional PK

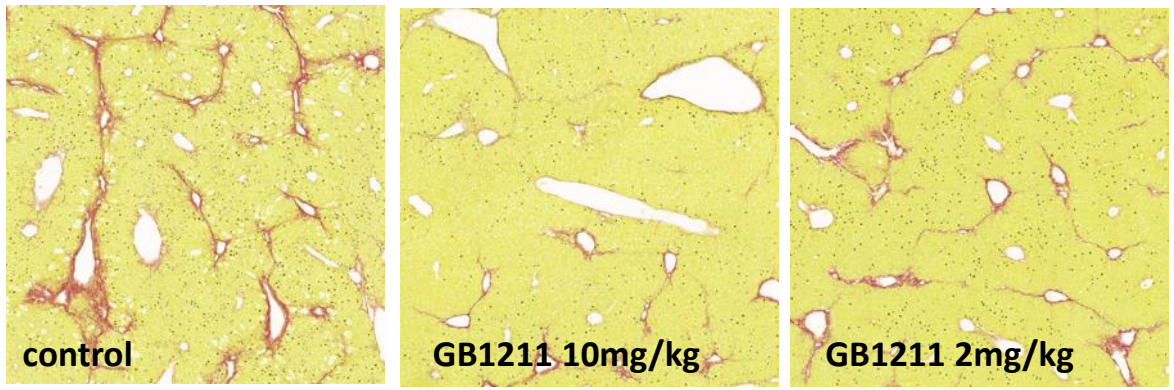


h = hours, N = sample size

Liver Cirrhosis: High Unmet Need with No Available Treatments



GB1211: Blocks CCl_4-Induced Liver Fibrosis

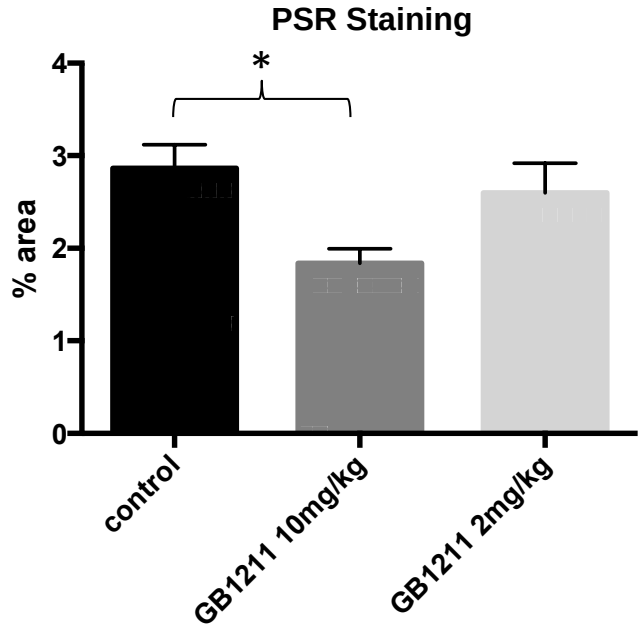


Collagen reduction at 10mg/kg, as measured by PSR (picosirius red staining)

Commence twice weekly CCl_4 1ul/g I.P. for 8 weeks

0 1 2 3 4 5 6 7 8

Vehicle PO OD
GB1211 10mg/kg PO *b.i.d.*
GB1211 2mg/kg PO *b.i.d.*



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