# 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 6, 2023**

# 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.)** |

**75 State Street, Suite 100**
**Boston, MA 02109**
**(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, 2022, Galecto, Inc.'s (the "Registrant") cash, cash equivalents and investments balance was approximately $66 million.

**Item 7.01. Regulation FD Disclosure.**

Included as Exhibit 99.1 to this Current Report on Form 8-K is an updated corporate presentation for the Registrant, dated January 2023, 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, 2023.

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 | Updated Corporate Presentation, dated January 2023. |
| 104 | Cover Page Interactive Data File (embedded within the Inline XBRL document) |

**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 6, 2023

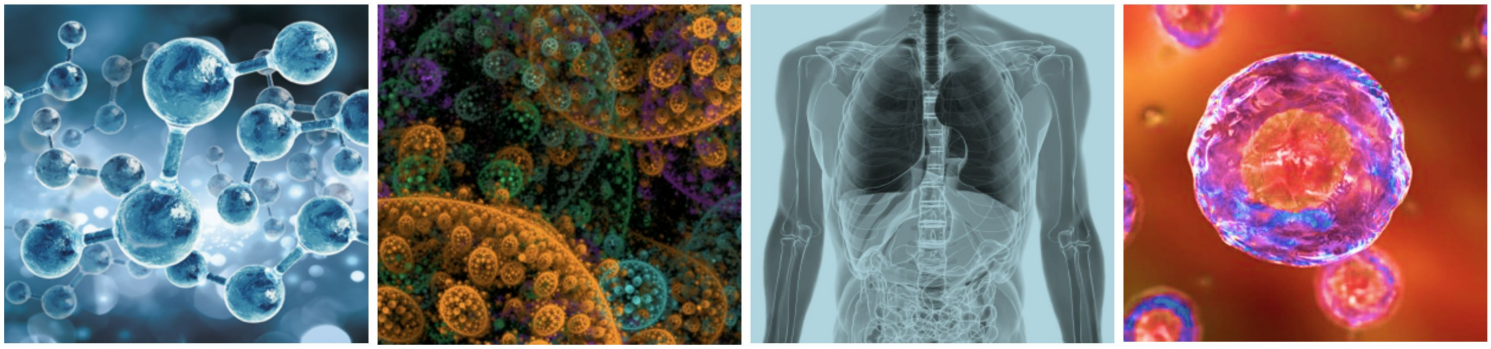
By: /s/ Hans T. Schambye

Hans T. Schambye, M.D., Ph.D.

President and Chief Executive Officer

Exhibit 99.1

Galecto



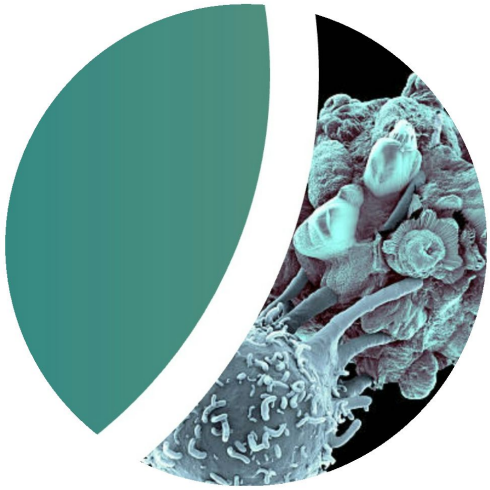
# First-in-class small molecule antifibrotic and anticancer agents

January 2023

# Forward-looking statements

This presentation contains forward-looking statements about Galecto, Inc.'s ("Galecto" or the "Company") strategy, future plans, operations and prospects, including, but not limited to, statements regarding the development of Galecto's compounds and potential opportunities; the expected timing and reporting of results of Galecto's clinical trials; and Galecto's expected cash runway. These statements involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation 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, Galecto claims 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: that drug development is expensive, time consuming, uncertain and susceptible to change, interruption, delay or termination; the duration and severity of the ongoing coronavirus disease (COVID-19) pandemic, including but not limited to the impact on the Company's clinical and other operations, the operations of its suppliers, others and the capital markets, which in each case remains uncertain; enrolling patients in clinical trials is competitive and challenging and the expected timing of Galecto's planned readouts for its ongoing clinical trials may be delayed as a result; that the timing and outcome of research, development and regulatory review and feedback is uncertain; Galecto's need to raise additional capital to advance all of its programs; the amount of Galecto's future losses is uncertain and could cause our stock price to fluctuate or decline; top-line data may not accurately reflect the complete results of a particular study or trial; results of clinical trials and other studies are subject to different interpretation and may not be predictive of future results; Galecto's clinical trials may fail to demonstrate adequately the safety and efficacy of any of its drug candidates; Galecto's drug candidates may not advance in development or be approved for marketing; clinical trial and other studies may not proceed at the time or in the manner expected or at all; clinical and nonclinical data is voluminous and detailed, and regulatory agencies may interpret or weigh the importance of data differently and reach different conclusions than Galecto or others, request additional information, have additional recommendations or change their guidance or requirements; data and information related to the Company's programs may not meet regulatory requirements or otherwise be sufficient for further development at all or on the Company's projected timeline; and other risks related to developing, seeking regulatory approval of and commercializing drugs, including regulatory, manufacturing, supply and marketing issues and drug availability. Additional factors that could cause results to differ materially from those stated or implied by Galecto's forward-looking statements are disclosed in its Securities and Exchange Commission (SEC) filings, including its most recent Annual Report on Form 10-K, filed with the SEC on February 17, 2022, 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.

**Galecto is working to create a world where patients suffering from fibrotic diseases, including cancers, have effective treatment solutions**



# Galecto has already demonstrated proof-of-concept with novel biology

*Cash balance of ~$66.1M as of 12/31/2022 funds all Phase 2 trials with runway into 2H 2024*

### INNOVATIVE PLATFORMS TARGETING CORE DISEASE PROCESSES

- Pioneers in **galectin-3** and **LOXL2** based pharmacology
- **First-in-class oral small-molecule, target specific inhibitors**

### ADDRESSING DISEASE AREAS WITH SIGNIFICANT UNMET MEDICAL NEED

**All programs address**:

- Diseases characterized by clear **unmet medical need**
- **Multi-billion-dollar** market opportunities

### ADVANCING BROAD PIPELINE IN FIBROTIC DISEASE

**Four** Phase 2 programs:

- Non-small cell lung cancer (**NSCLC**)
- Idiopathic pulmonary fibrosis (**IPF**)*
- Myelofibrosis (**MF**)
- Liver **cirrhosis****

* Trial fully enrolled ** Trial complete

### PROOF OF CONCEPT ALREADY ESTABLISHED FOR BOTH APPROACHES

**Galectin-3 Inhibitors**:

- **Target engagement** confirmed
- Positive **biomarker** data in **IPF**
- Evidence of **tumor microenvironment transformation**
- **Significant clinical data** in **cirrhosis** and **COVID**

**LOXL2 Inhibitors:**

- Clinically demonstrated bone marrow collagen **fibrosis reduction** in myelofibrosis

*LOXL2: lysyl oxidase-like 2*

# Core assets all supported by positive clinical data

| Product Candidate | Indication | Preclinical | Phase 1 | Phase 2 | Phase 3 | Next Steps | Demonstrated Results |
|---|---|---|---|---|---|---|---|
| GB0139 | Idiopathic Pulmonary Fibrosis | | | GALACTIC-1 (Inhaled Gal-3 inhibitor) | | Topline Data Phase 2b Mid-2023 | Target engagement and biomarker effects in COVID and IPF patients |
| GB2064 | Oncology and Fibrosis (Initially in Myelofibrosis) | | | MYLOX-1 (Oral LOXL2 inhibitor) | | Topline Date Phase 2a 2H-2023 | Unprecedented reduction in bone marrow collagen fibrosis |
| GB1211 | Liver Fibrosis | | | GULLIVER-2 (Oral Gal-3 inhibitor) | | End of Study Regulatory Discussions 1H-2023 | Clinical efficacy in liver cirrhosis |
| GB1211 | Oncology: NSCLC | | | GALLANT-1 (Oral Gal-3 inhibitor) | | Complete Phase 2a Enrollment Mid-2023 | Evidence for transforming tumor microenvironment and prevention of galectin-3 mediated checkpoint inhibitor resistance |
| GB1211 | Oncology: Melanoma & HNSCC | | | IIT Phase 2 Trial | | Trial Initiation 2023 | |

# Experienced management team & board of directors

**Hans Schambye**
CEO

**Stephanie Oestreich**
CBO

**Bertil Lindmark**
CMO

**Jonathan Freve**
CFO

| BOARD OF DIRECTORS | |
|---|---|
| **Carl Goldfischer, M.D. Chairman** | Bay City Capital Partner<br>Former CFO of ImClone[1] |
| **Jayson Dallas, M.D.** | Former CEO Aimmune[2] |
| **Chau Khuong, MPH** | Former OrbiMed Partner |
| **Søren Møller, Ph.D.** | Novo Holdings Partner<br>Former CSO Exiqon[3] |
| **Amit Munshi, MBA** | Former CEO Arena[4] |
| **Anne Prener, M.D., Ph.D.** | CEO Imbria & venture partner SV Health Investors |
| **David Shapiro, M.D., FRCP, FFPM** | Retired CMO Intercept<br>Former CMO Idun[4] |
| **Hans Schambye, M.D., Ph.D.** | CEO Galecto |

[1]Acquired by Eli Lilly. [2]Acquired by Nestlé. [3]Acquired by Qiagen. [4]Acquired by Pfizer.

# GB0139: Inhaled Galectin-3 Inhibitor for IPF

GALACTIC-1

# 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 forced vital capacity (FVC))
  - Lung tissue scars and becomes non-functional
  - Median survival of 2-5 years
  - Death caused by respiratory failure
  - Unknown cause

## Limited Treatment Options

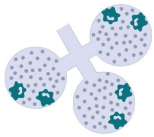
- Only two approved drugs slow disease progression: **pirfenidone** and **nintedanib**
  - Neither has been associated with improvements in overall survival
  - Both have significant side-effects that limit compliance and usage
- **Due to side effects, less than 50% of patients on treatment**
- Despite dose-limiting side effects, sales of pirfenidone and nintedanib exceeded $3.7B and $3.5B in 2021 and 2020, respectively

# 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
- Other clinical development candidates given intravenously, subcutaneously and orally



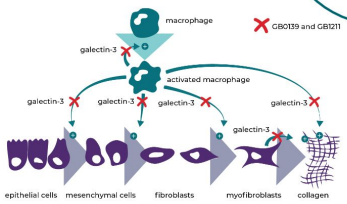
## Indications of Efficacy

- GB0139 delivered to the periphery of the lungs at high concentrations
- GB0139 targets macrophages – the cells driving the fibrosis mechanism

**GB0139**

## Pluripotent MoA

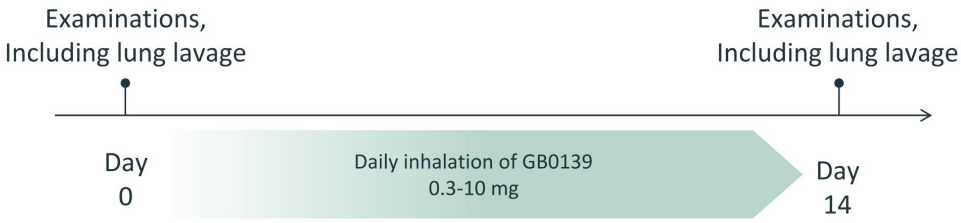
- Unique and pluripotent MoA
- GB0139 inhibits fibrosis by targeting macrophages, fibroblasts, and epithelial cells




## 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: Completed Phase 2a 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

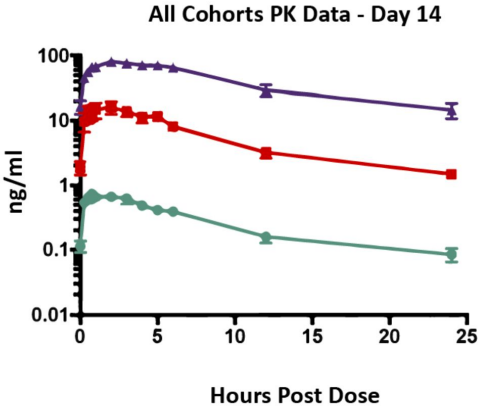
**Four centers in the UK**

**All patients completed 2-week dosing as planned**

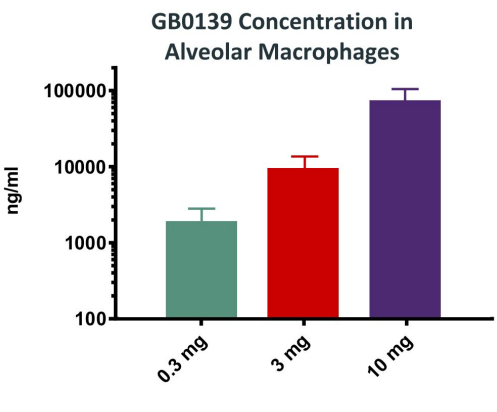
**Evaluable bronchoalveolar lavages (BALs) obtained for all 48 bronchoscopies**

# GB0139: Phase 2a Results – bioavailability & target engagement in IPF patients

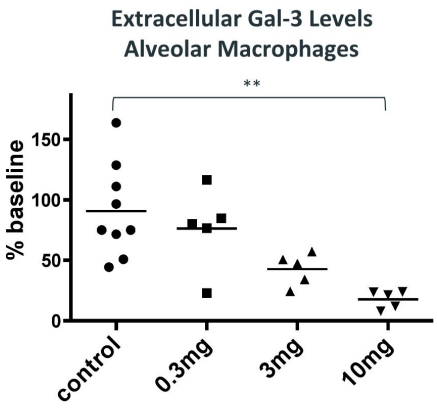
## Exceptionally Consistent PK Data in IPF Patients




## GB0139 Reaches the Alveolar Macrophages Deep within Lung

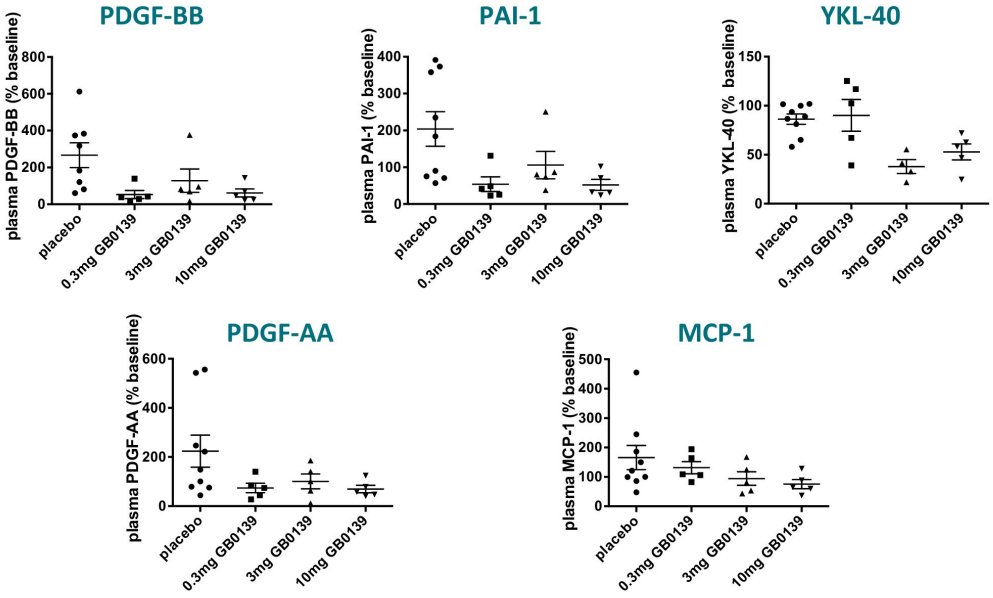



## Induces Profound Reduction of Gal-3 Levels on Alveolar Macs



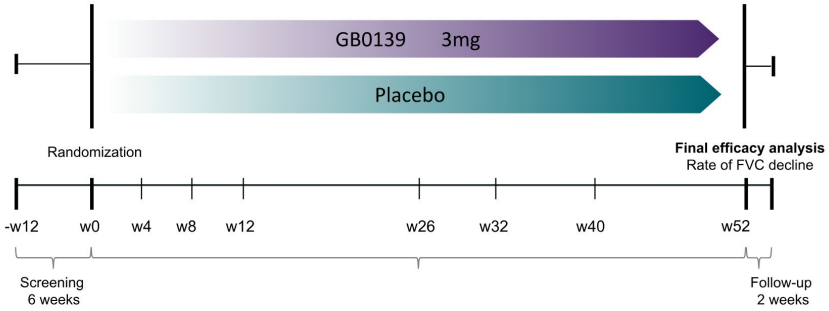
# GB0139: Phase 2a study showed consistent, dose-dependent effects on highly relevant fibrosis biomarkers

- 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 orphan drug designation (ODD)
- BAL fluid and plasma correlation indicates GB0139 directly impacts lung function



# GALACTIC-1: Ongoing Phase 2b study in IPF patients

GALACTIC-1




- Randomized, placebo controlled
- 52-week study
- 100+ centers
- Enrolled 144 patients
  - Randomized 2:1 GB0139 to Placebo

- Primary outcome measure:
  - Annualized rate of Forced Vital Capacity (FVC) decline over 52 weeks
  - Study is powered to see an effect in patients not on pirfenidone or nintedanib
- Key secondary outcomes:
  - Safety, Diffusing Capacity for Carbon Monoxide (DLCO), 6-minute walk test, Quality of Life

# GB0139: Inhaled galectin-3 inhibitor summary

Ground-breaking novel treatment for IPF, an orphan disease with poorly tolerated treatments

Inhaled, delivered directly to the site of active lung destruction

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

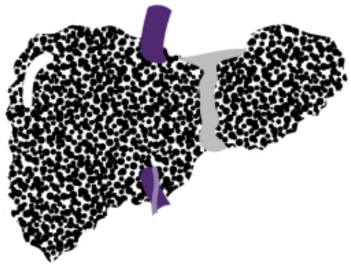
# GB1211: Oral Galectin-3 Inhibitor for Liver Cirrhosis

Gulliver-2

# Advanced liver disease is a massive indication and a growing medical concern

## Disease Overview

- Cirrhosis prevalence: ~2 million patients in US, ~3 million in EU[1]
- Severe, progressive liver fibrosis ultimately leads to liver failure
- Caused primarily by NASH, alcoholic liver disease and 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

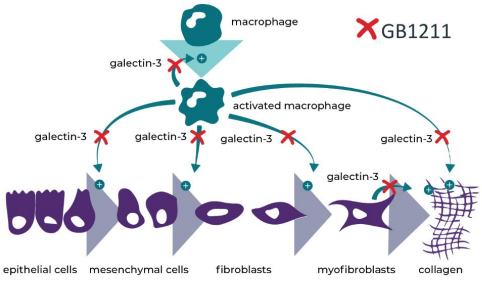


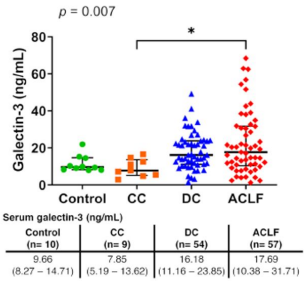
[1] https://www.thelancet.com/journals/langas/article/PIIS2468-1253(19)30349-8/fulltext Sepanlou, et al Lancet Gastroenterol Hepatol 2020; 5: 245–66
[2] https://www.hepatitis.va.gov/cirrhosis/background/stages.asp

# GB1211: Oral galectin-3 inhibitor for advanced liver disease and cirrhosis

*Evidence links galectin-3 to cirrhosis progression*

- Galectin-3 is elevated in decompensated cirrhosis and in acute on chronic liver failure
- Galectin-3 is prognostic biomarker of hepatocellular carcinoma, a known 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-ß mediated myofibroblast activation and matrix production in liver fibrosis
  - Pre-clinical and clinical evidence for reduction in transaminases by galectin-3 inhibitors suggesting hepatocyte protection




Serum galectin-3 (ng/mL)

| Control (n= 10) | CC (n= 9) | DC (n= 54) | ACLF (n= 57) |
|---|---|---|---|
| 9.66 (8.27 – 14.71) | 7.85 (5.19 – 13.62) | 16.18 (11.16 – 23.85) | 17.69 (10.38 – 31.71) |

[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
[3]Cervantes-Alvarez *et al.*, 2022

# Galecto's oral galectin-3 inhibitors

- Galecto has developed a series of orally active, specific and potent inhibitors of Galectin-3
- The lead oral compound, GB1211, reduces fibrosis and cancer growth in several different models
  - Appears safe and well tolerated in IND-enabling studies
  - SAD/MAD in healthy volunteers successfully completed (N=78)
  - GULLIVER-2 (N=54) completed, GALLANT-1 in 1st line NSCLC ongoing
- GB1211 addresses a large number of currently poorly treated indications

| Fibrosis | Cancer |
|---|---|
| • **Cirrhosis** | • **Hepatocellular Carcinoma** |
| • NASH | • Lung Cancer |
| • Chronic Kidney Disease (CKD) | • Pancreatic Cancer |
| • Heart Failure | • Gastric Cancer |
| • Scleroderma | • Renal cell Carcinoma |

# GULLIVER-2 – Part 2

*A randomized, double blind, placebo-controlled 12-week study in Child-Pugh B patients*

GULLIVER-2

**Part 2:** Repeat dose hepatic impairment study (Child-Pugh B)

**GB1211 100mg BID (n=15)**

**Placebo BID (n=15)**

Biochemistry on Day 1, 7, 42, and follow up Day 96
PK samples on Day 1, 7, 21, 42, 63, 84

**Primary endpoints:**
- Safety and tolerability
- PK

**Exploratory endpoints**
- Biochemistry
- Liver fibrosis (VCTE)
- Steatosis (CAP)
- Exploratory biomarkers

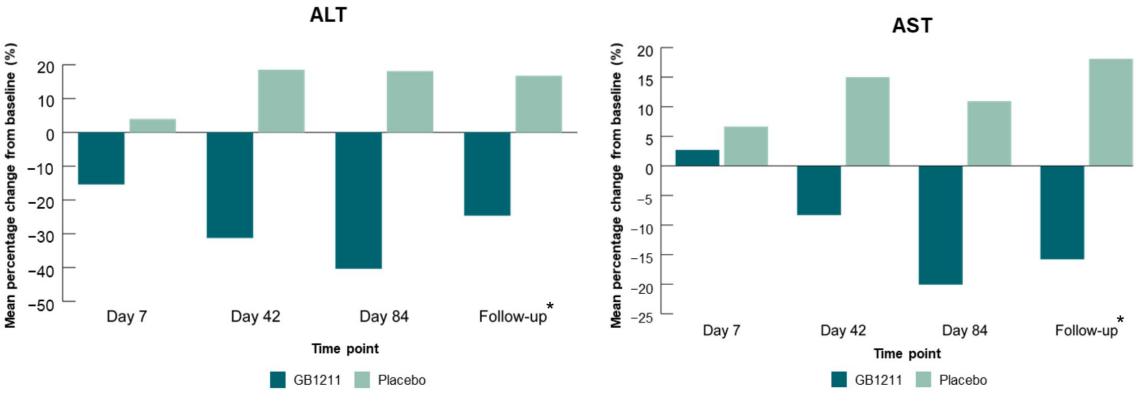
**Part 1:** Single dose hepatic impairment study (Child-Pugh B) → **Part 3:** Single dose hepatic impairment study (Child-Pugh C)

• BID, twice a day; CAP, controlled attenuation parameter; PK, pharmacokinetics; VCTE, vibration controlled transient elastography

# Liver-related biochemistry results

GULLIVER-2

*Consistent and increasing reduction in liver enzymes for GB1211 patients*

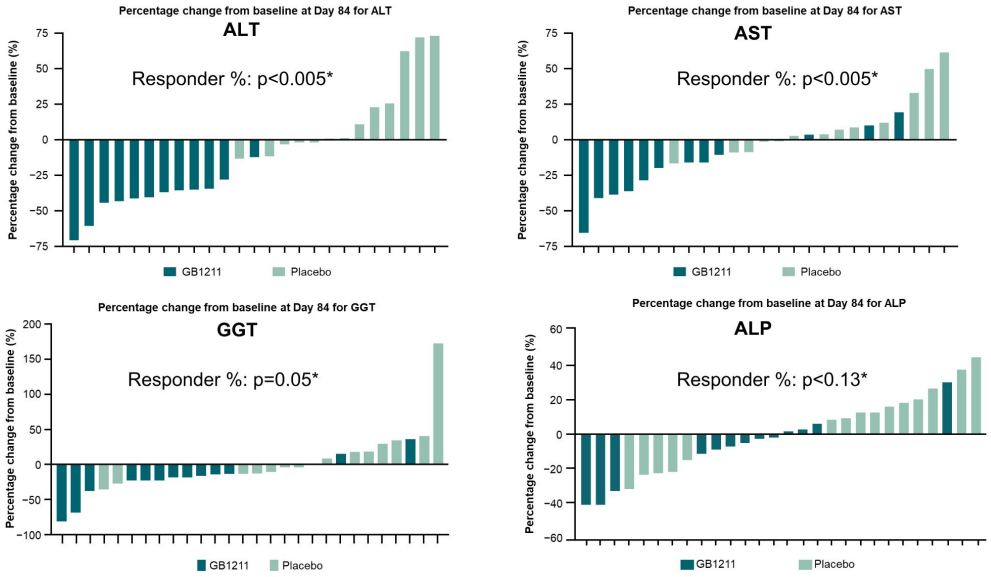



| Treatment effect (GB1211-Placebo) [%] at Day 84 | ALT | AST | GGT | ALP |
|---|---|---|---|---|
| Mean | -58.44 | -32.40 | -37.77 | -14.76 |
| 95% confidence interval | (-79.00, -37.88) | (-51.63, -13.17) | (-69.47 , -6.06) | (-31.92 , -2.40) |
| p-value | 0.0001 | 0.002 | 0.0214 | 0.0889 |

*Follow up took place two weeks after the last dose. ALT: alanine transferase; AST: aspartate transferase

# Encouraging reductions in ALT, AST, GGT and ALP at day 84

GULLIVER-2



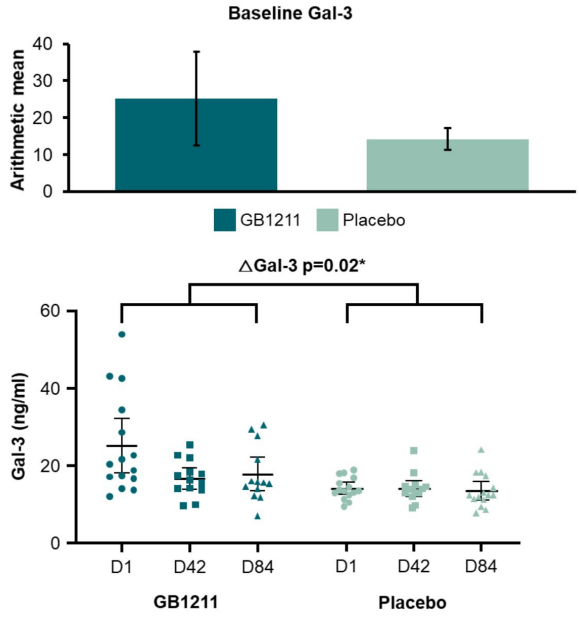

No adverse changes in standard safety laboratory parameters, including bilirubin albumin, or INR

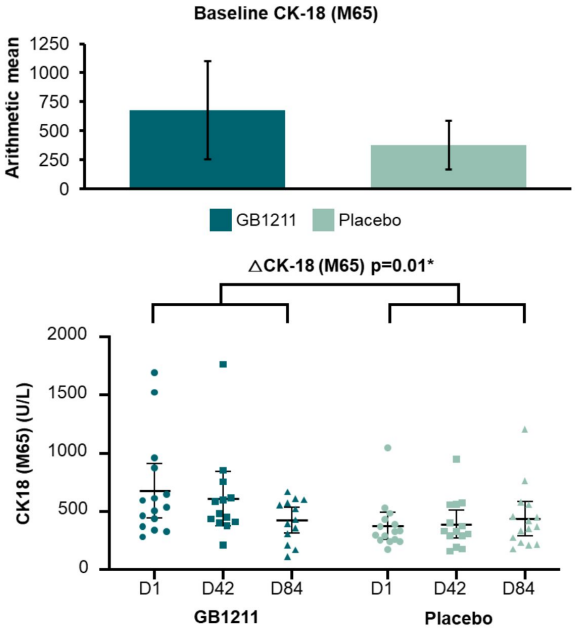
*Post-hoc analysis: Fischer's Exact test. Percentage of patients experiencing reduction liver enzyme values at Day 84. ALP: alkaline phosphatase; ALT: alanine transferase; AST: aspartate transferase; D: day; GGT: gamma-glutamyl transferase; INR: international normalized ratio

# GB1211 reduces galectin-3 and CK-18 (M65)

*GB1211 demonstrated target engagement and potential anti-apoptotic properties*

Galectin-3




CK-18 (M65)

*p value from unpaired t-test. Δ: change; CK-18: cytokeratin-18; D: day; Gal-3: galectin-3

# Putting GULLIVER-2 data Into perspective

GULLIVER-2

| Outcome | Selonsertib (48w)[1] MELD 7 | PBO | Emricasan (24w)[2] Child-Pugh A | PBO | Simtuzumab (48w)[3] Child-Pugh A | PBO | Belapectin (52w)[4] Child-Pugh A | PBO | GB1211 (12w)* Child-Pugh B | PBO* |
|---|---|---|---|---|---|---|---|---|---|---|
| MELD | NC | NC | 0.2 | 0.4 | NC | NC | NC | NC | **−1.4†** | **0.5†** |
| ALT (U/L) | −3 | −4 | NC | NC | −5 | −1 | NC | NC | **−12.2** | **3.9** |
| GGT (U/L) | −8 | −4 | NC | NC | −7 | −8 | NC | NC | **−54.2** | **17.9** |
| Total bilirubin (µmol/L) | NC | NC | −0.5 | 0.3 | 0.1 | 0.1 | NC | NC | **−1.2** | **−0.5** |
| Transient elastography (kPa) | −0.7 | −0.7 | −6.7 | −0.3 | NA | NA | −2.3 | −0.5 | **−9.7** | **−7.6** |
| CAP (dB/m) | NA | NA | NA | NA | NA | NA | NA | NA | **−20.2** | **4.1** |

***Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.***

*Data are absolute changes from baseline to Day 84. †Modified ITT population.
ALT: alanine transferase; CAP: controlled attenuation parameter; dB/m: decibels per meter; GGT: gamma-glutamyl transferase; ITT: intent to treat; kPa: kilopascal; MELD: model for end-stage liver disease; NA: not available; NC: no change; PBO: placebo; U/L: units per litre; w: week


1. Harrison et al. J Hepatol 2020;73(1):26–39;
2. Garcia-Tsao et al. J Hepatol 2020;72(5):885–895;
3. Harrison et al. Gastroenterology 2018;155(4):1140–1153;
4. Chalasani et al. Gastroenterology 2020;158(5):1334–1345.e5

# GULLIVER-2 - topline results

*Unprecedented study results in a decompensated cirrhosis patient population*

GULLIVER-2

**Galectin-3 in liver disease**

- Carbohydrate binding protein shown to drive fibrosis via TGF-ß receptor
- Elevated in decompensated cirrhosis, alcoholic hepatitis and ACLF
- GB1211 is a potent, selective, oral inhibitor of Galectin-3

**Well tolerated**

- GB1211 was well-tolerated with no drug-related adverse events identified
- Predictable PK profile consistent with the option of repeated dosing in patients with hepatic impairment

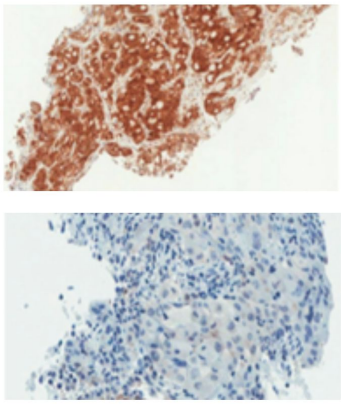
**Clinical**

- Galectin-3 reduction demonstrates target engagement
- Consistent and statistically significant reductions in ALT, AST and GGT
- Concordant changes in liver biochemistry, liver stiffness & steatosis observed
- Data suggests that GB1211 improves liver inflammation and reduces liver injury

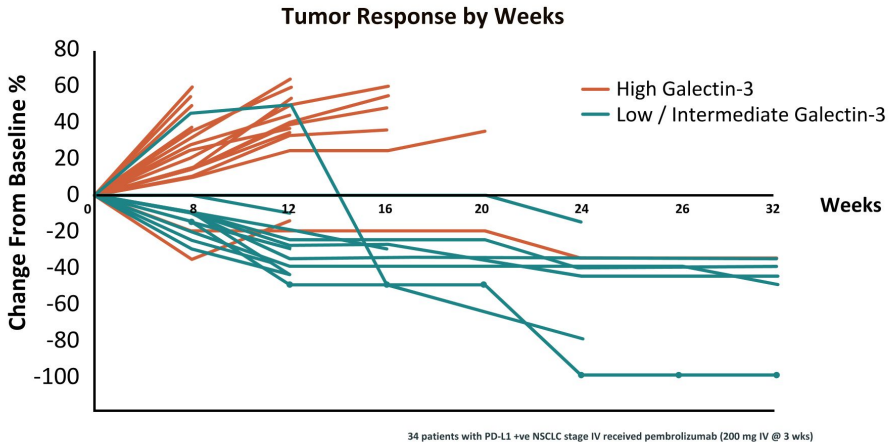
**Results strongly support progressing to phase II/III studies in severe liver diseases**

# GB1211: Oral Galectin-3 Inhibitor for Cancer

# Galectin-3 expression predicts response to pembrolizumab in NSCLC



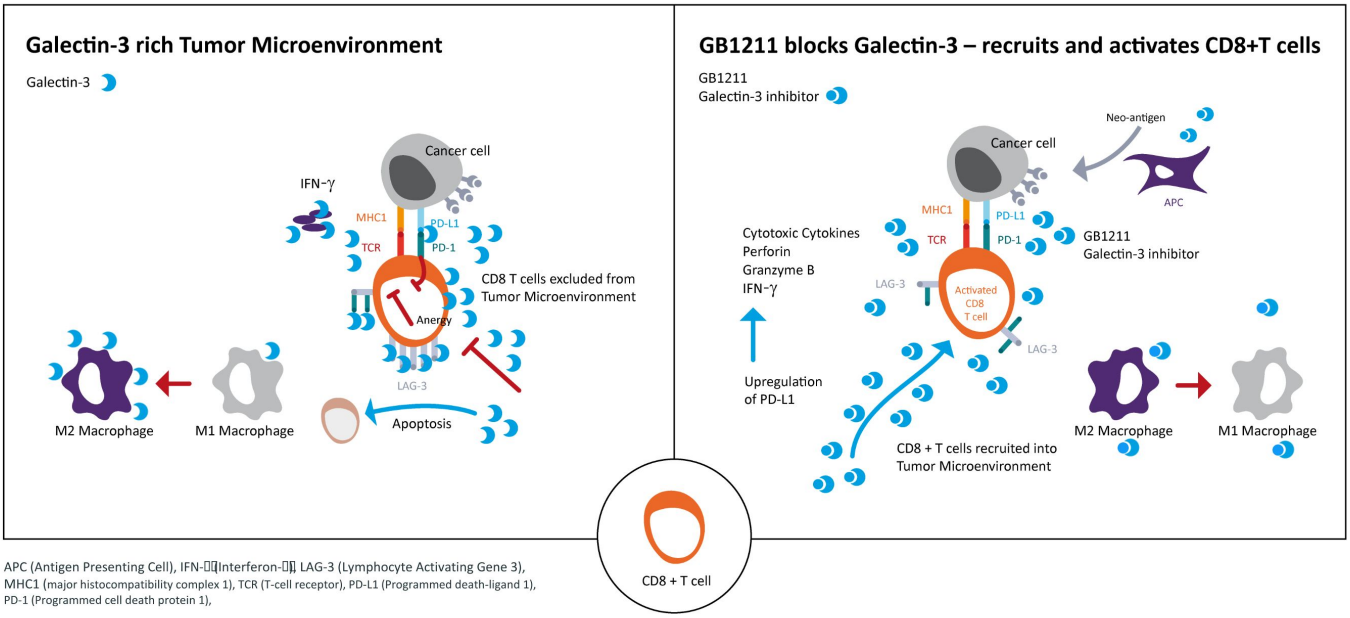
Galectin-3 expression in NSCLC biopsies




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

- High galectin-3 expression in patients with NSCLC strongly correlated with tumor resistance to pembrolizumab
- A clinical response was seen in tumors with a negative, low or intermediate galectin-3 expression

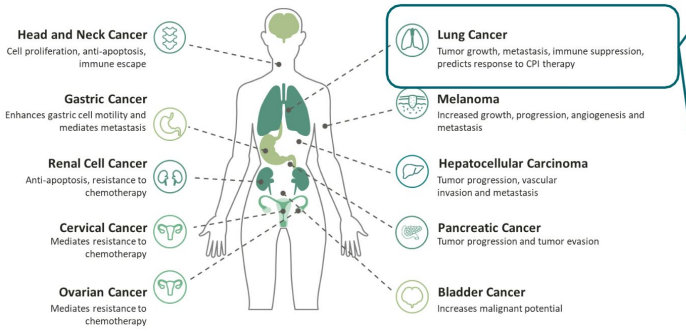
# GB1211 increases CD8+ T cell recruitment and activation in galectin-3 rich tumor microenvironment, and potentiates checkpoint inhibitors




APC (Antigen Presenting Cell), IFN-γ (Interferon-γ), LAG-3 (Lymphocyte Activating Gene 3), MHC1 (major histocompatibility complex 1), TCR (T-cell receptor), PD-L1 (Programmed death-ligand 1), PD-1 (Programmed cell death protein 1),

# Galecto has chosen NSCLC as first development target

*NSCLC represents a significant unmet medical need with a strong rationale for anti-Galectin-3 therapy*




ASCO: Cancer.net (01-2021)
Ebrahim et al (2014); Ann Transl Med;2(9):88
Kuou et al (2015); Cancer Immunol Res;3: 412
Ou et al (2021); Ther Adv Med Oncol;13: 1
Capalbo et al. (*2019); Int. J. Mol. Sci.;20*
Vuong et al (2019); Cancer Res;79: 1480

**High unmet need**

- Lung cancer is 2nd most common cancer and leading cause of cancer death
  - More than 130,000 death/year in US
  - 1.59 million deaths/year globally
- NSCLC has a poor prognosis – 5-year survival <25%
  - Metastatic NSCLC: 5-year survival rate < 7%
- Billion-dollar market opportunity

**Galectin-3 is a promising target** that

- Predicts overall poor survival
- Predicts response to CPI therapy

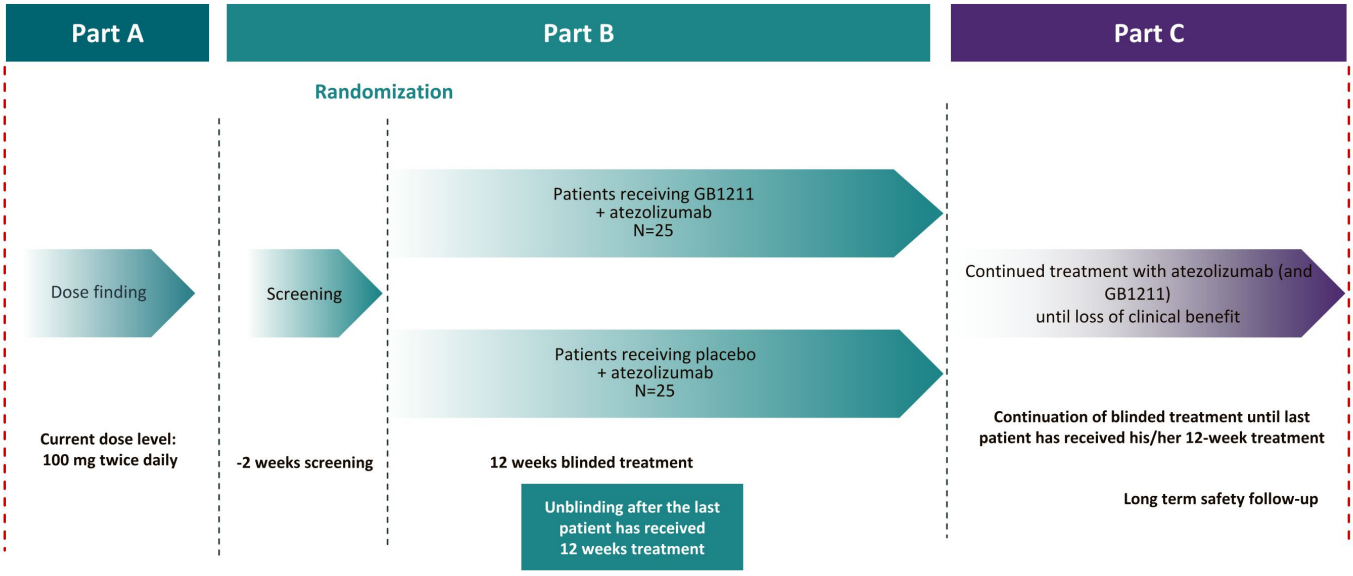
**CPI therapy for treatment of NSCLC is well established**

- However, 40-60% of patients don't respond to therapy
- Gal-3 inhibitors show:
  - Anti-tumor effects
  - T cell activation – LAG3 blockade
  - Macrophage polarizations
  - Increased apoptosis

# GALLANT-1 (Phase 2a) study design

*Primary endpoint is tumor shrinkage*





# Galecto oncology opportunities

Galectin-3 plays central role for the hallmarks of cancer and is linked to poor survival for many solid tumors

Inhibition of galectin-3 may have anti-tumor activity as monotherapy and in combination with CPIs, chemo- and radiotherapy

Galectin-3 is a negative regulator of immune cell functions and drives low CPI response rate

GB1211 is a specific oral galectin-3 inhibitor ready for Phase 2

- Anti-tumor effects in preclinical models
- Well-tolerated and no observed adverse safety or drug interaction signals

Galecto collaborating with Roche on upcoming NSCLC first line trial with GB1211

- Randomized, placebo-controlled trial in combination with Tecentriq® (atezolizumab)
- Topline data expected mid-2023

GB1211 marks Galecto's first entry into the solid tumor space

# GB2064: LOXL2 Inhibitor for Myelofibrosis and Other Oncology and Fibrotic Diseases

# GB2064: Oral LOXL2 inhibitor in myelofibrosis

## Overview and Treatment Opportunity

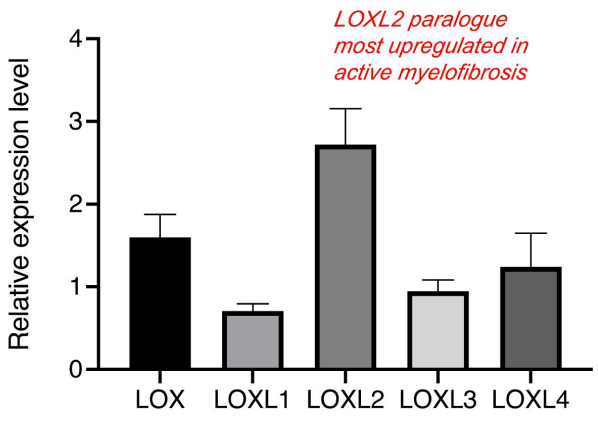
### GB2064

- 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® and Novartis's Jakavi® achieved aggregate sales of $3.7B and $3.3B in 2021 and 2020, respectively

## LOX Family Gene Expression in Myelofibrosis Stromal Cells



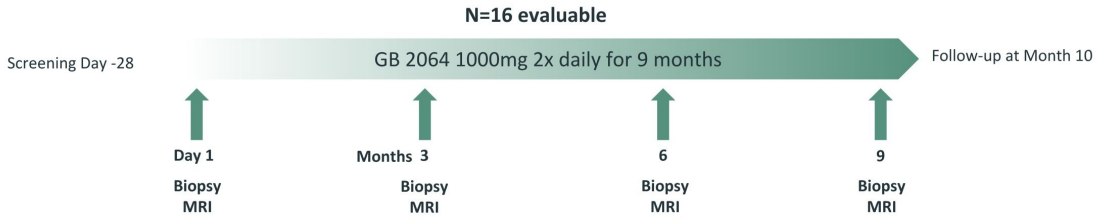
# Major unmet needs remain in myelofibrosis

*Key categories potentially worsened by existing JAKi therapy*

| | | | |
|---|---|---|---|
| **Prevention or reduction in cancer cell growth** | Improve/stabilize hemoglobin count | ▶ | **Unaffected or worsened by JAKi** |
| | Improve/stabilize thrombocyte count | ▶ | **Unaffected or worsened by JAKi** |
| | Stabilize leukocyte counts | ▶ | **Unaffected by JAKi** |
| **Reduction in bone marrow fibrosis** | Reduce spleen volume | ▶ | **Surrogate endpoint addressed by JAKi** |
| | Reduce MF symptoms | ▶ | **Improved by JAKi** |

# MYLOX-1: GB2064 monotherapy in myelofibrosis



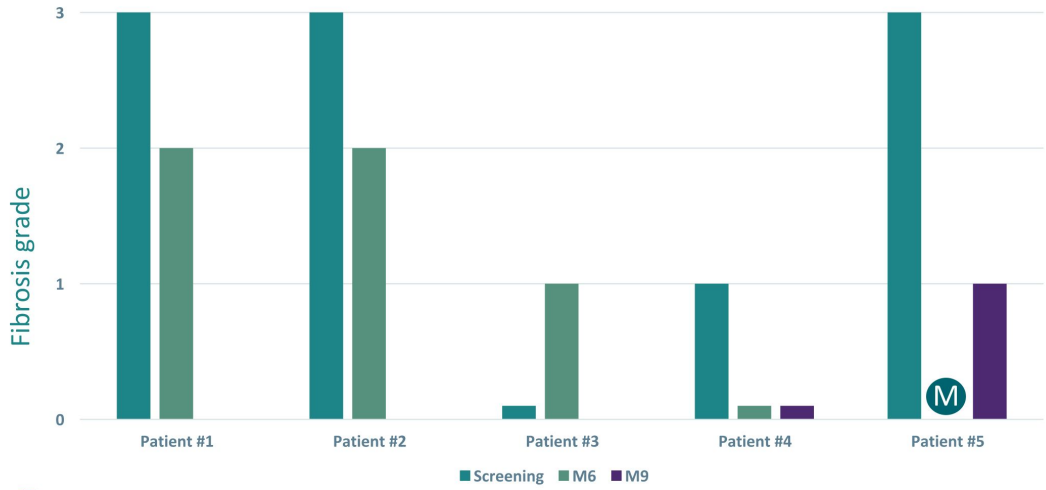



- Study led by Professor Srdan Verstovsek, MD Anderson
- First patient dosed in Q3 2021
- Single arm, open label study allowing real-time read of safety and activity
  - Planned for 16 evaluable patients initially for 9 months of treatment
  - Opportunity for entering an extension phase of the study in case of clinical benefit as evaluated by the treating physician
- Patients who are ineligible for, refractory to or who do not tolerate or have progressed on JAK inhibitors

**Intermediate analysis:**
Five evaluable patients have passed six months of treatment with GB2064

# 80% (4/5) of patients experienced a ≥1 grade reduction in collagen fibrosis

*Bone marrow biopsy - collagen fibrosis grade (trichrome) for patients passing month six*




Ⓜ Missed biopsy due to elective surgery

"It is wholly unprecedented and very encouraging to observe a reduction in collagen fibrosis in this patient population"

"It is exciting to see the first clinical validation of LOXL2 as a fibrosis target"

**Prof. Srdan Verstovsek, MD Anderson Cancer Center**

# Unparalleled reduction in collagen fibrosis

*Stable disease and hematology – acceptable tolerability*

MYLOX-1

**Safety and Clinical Pharmacology**

- GI (predominantly grade 1-2) side effects were observed; acceptable tolerability profile
- GB2064 demonstrated penetration into the fibrotic bone marrow
- Plasma LOXL2 assay showed target engagement

**Clinical and Bone Marrow Findings**

- Four of five evaluable patients (80%) showed ≥ 1-grade improvement in collagen fibrosis
- Two patients currently in the extension phase due to clinical benefit
- All four responders demonstrated disease stabilization with spleen volumes and hematological parameters remaining stable

"

”It is exciting and encouraging to see a clear reduction in collagen fibrosis following the administration of a selective LOXL2 inhibitor in four of the five evaluable patients combined with stabilization of hematological parameters and spleen volume“

“Stable disease is excellent in a progressive disease such as myelofibrosis”

**Prof. Claire Harrison**
**Guy's & St Thomas**
**NHS Foundation Trust**

# MYLOX-1 – Unparalleled reduction in collagen fibrosis

MYLOX-1

### Repeat Bone Marrow Biopsy Trial

- MYLOX-1 is an open-label phase 2a study
- GB2064 administered as monotherapy in myelofibrosis
- Patients are ruxolitinib (JAK inhibitor) refractory/ relapsed/ineligible

### Intermediate Assessment

- Five evaluable patients have passed six months of treatment with GB2064
- GB2064 has shown an acceptable safety and tolerability profile to date

### Key Takeaways

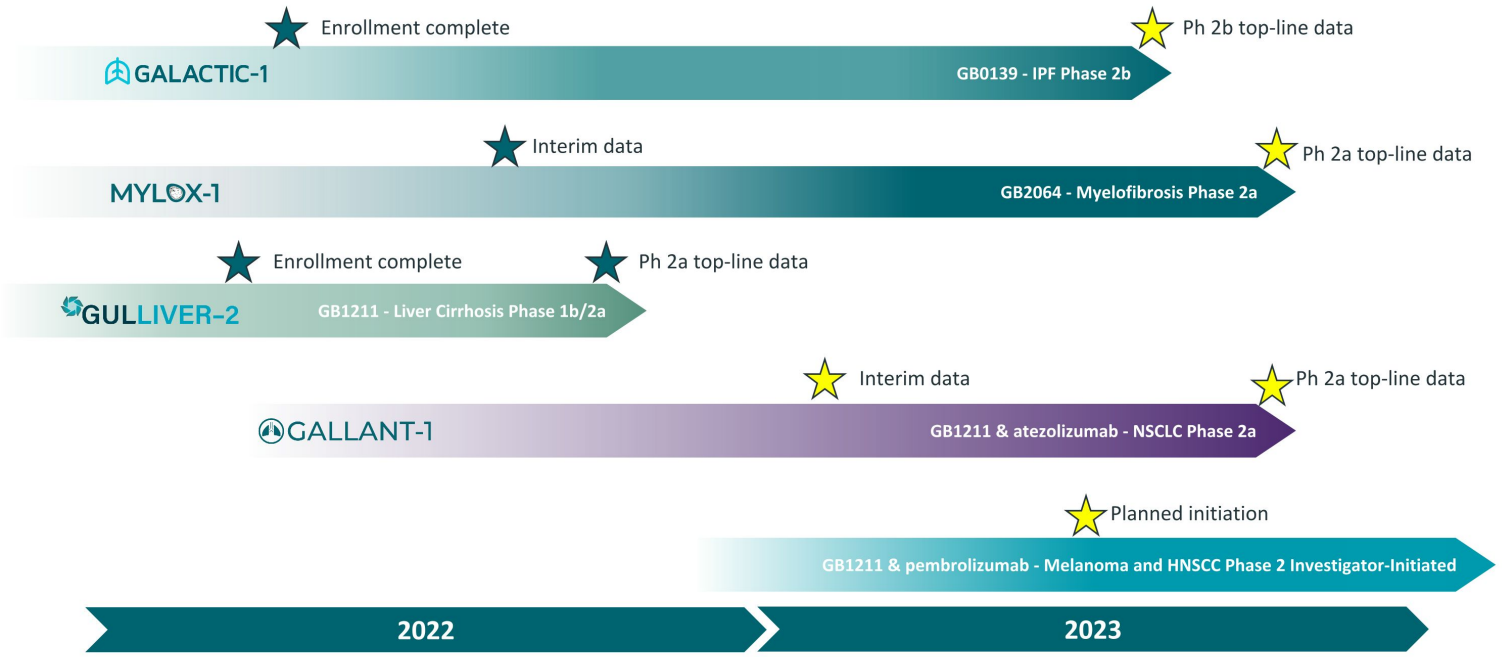
- Unique reduction in bone marrow collagen fibrosis
- Four out of five patients were responders to GB2064 therapy
- Responders showed disease stabilization when progression would have been expected
- Potential for disease modifying effects of GB2064

**LOXL2 validated as a clinical fibrosis target**

**GB2064 is an exciting opportunity that may be further developed for myelofibrosis and other fibrotic disease states**

# Summary

# Pipeline and clinical development timeline



# Galecto has already demonstrated proof-of-concept with novel biology

*Cash balance of ~$66.1M as of 12/31/2022 funds all Phase 2 trials with runway into 2H 2024*

### INNOVATIVE PLATFORMS TARGETING CORE DISEASE PROCESSES

- Pioneers in **galectin-3** and **LOXL2** based pharmacology
- **First-in-class oral small-molecule, target specific inhibitors**

### ADDRESSING DISEASE AREAS WITH SIGNIFICANT UNMET MEDICAL NEED

**All programs address**:

- Diseases characterized by clear **unmet medical need**
- **Multi-billion-dollar** market opportunities

### ADVANCING BROAD PIPELINE IN FIBROTIC DISEASE

**Four** Phase 2 programs:

- Non-small cell lung cancer (**NSCLC**)
- Idiopathic pulmonary fibrosis (**IPF**)*
- Myelofibrosis (**MF**)
- Liver **cirrhosis****

* Trial fully enrolled ** Trial complete

### PROOF OF CONCEPT ALREADY ESTABLISHED FOR BOTH APPROACHES

**Galectin-3 Inhibitors**:

- **Target engagement** confirmed
- Positive **biomarker** data in **IPF**
- Evidence of **tumor microenvironment transformation**
- **Significant clinical data** in **cirrhosis** and **COVID**

**LOXL2 Inhibitors:**

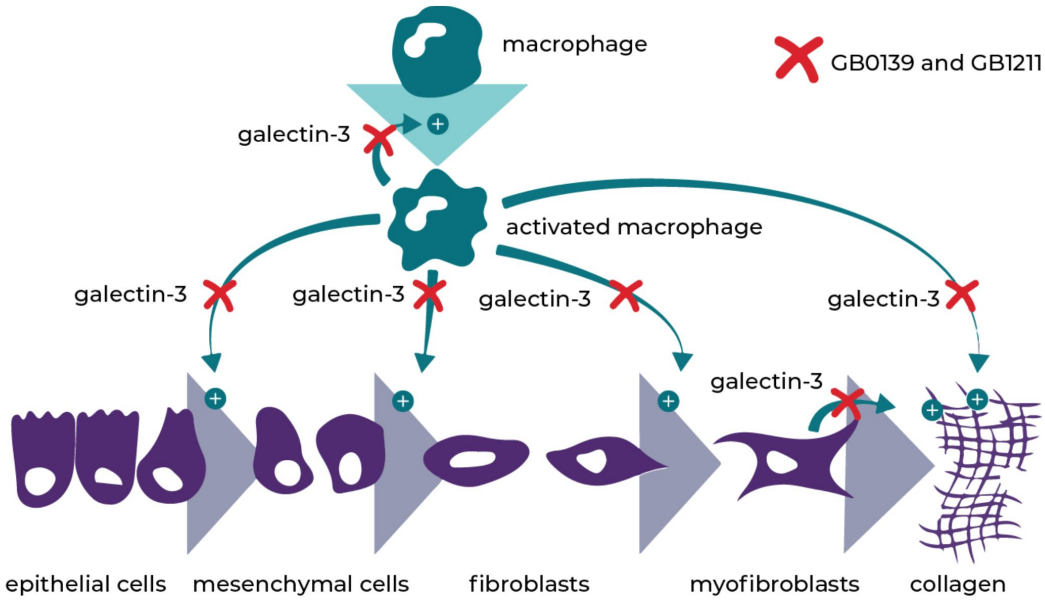
- Clinically demonstrated bone marrow collagen **fibrosis reduction** in myelofibrosis

*LOXL2: lysyl oxidase-like 2*

# Appendix

# Galectin-3 impacts key elements in the fibrosis cascade

## - inhibited by our galectin-3 inhibitors



# Comparison: GB0139 MoA to alternative programs

| MoA | GB0139 | Competitors |
|---|---|---|
| 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) |

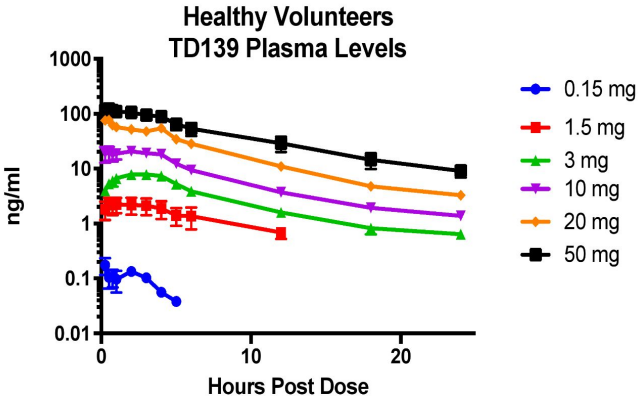
# GB0139: Results of Phase 1 SAD study

## 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



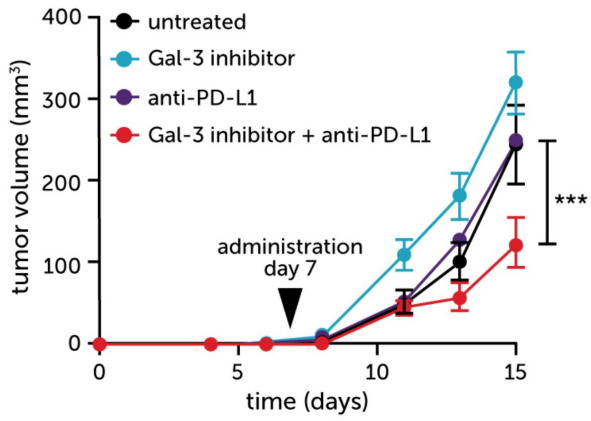
# SAEs in GALACTIC-1 patients

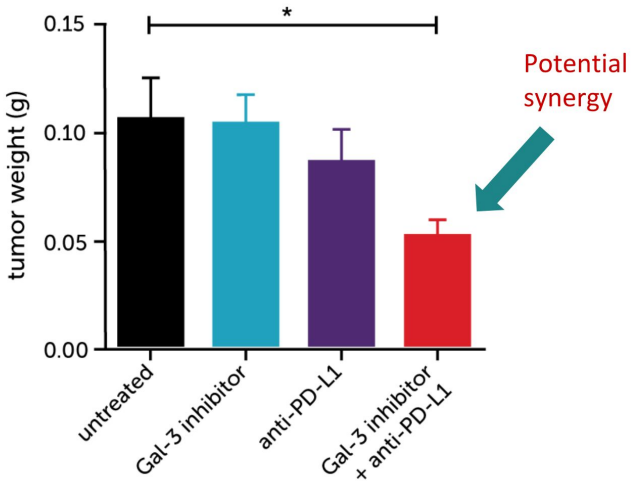
- Following a DSMB interim review and recommendation in Q1 2021, we discontinued GB0139 10mg arm and GB0139 plus SOC arms
- Preliminary safety data from the DSMB review included below continues to show a promising safety profile for monotherapy treatment with GB0139

| System Organ Class/Preferred Term | Patients on nintedanib or pirfenidone N=193 | | | Patients NOT on nintedanib or pirfenidone N=85 | | |
|---|---|---|---|---|---|---|
| | n | % | SAEs | n | % | SAEs |
| Subjects with at least one serious adverse event (SAE) | 38 | 19.7 | 59 | 9 | 10.6 | 13 |
| | | | | | | |
| Respiratory, thoracic and mediastinal disorders | 21 | 10.9 | 24 | 5 | 5.9 | 6 |
| Infections and infestations | 14 | 7.3 | 17 | 4 | 4.7 | 5 |
| General disorders and administration site conditions | 4 | 2.1 | 4 | 1 | 1.2 | 1 |
| Cardiac disorders | 4 | 2.1 | 5 | 0 | 0 | 0 |
| Blood and lymphatic system disorders | 2 | 1.0 | 2 | 0 | 0 | 0 |
| Other non-respiratory conditions | 6 | 3.0 | 7 | 1 | 1.2 | 1 |

The above table shows an overview of the interim *blinded* safety data in the study. As the study is ongoing, this data has not been fully cleaned and this table should not be relied on as the final and definitive adverse event table but is included here to provide an impression of the character of the serious adverse events that have been reported in the study as treatment emergent (i.e., starting after the onset of dosing of the experimental drug inhalations). Since the table represents a blend of patients on placebo, the 3 mg dose of GB0139 and the 10 mg dose of GB0139, including those taking concomitant nintedanib and pirfenidone, no firm conclusion can be made that these reported treatment emergent serious adverse events are related to administration of GB0139 and, conversely, that they are unrelated to the administration of GB0139.

# Therapeutic administration of galectin-3 inhibitor in combination with anti-PD-L1 inhibits Lewis Lung Carcinoma Growth

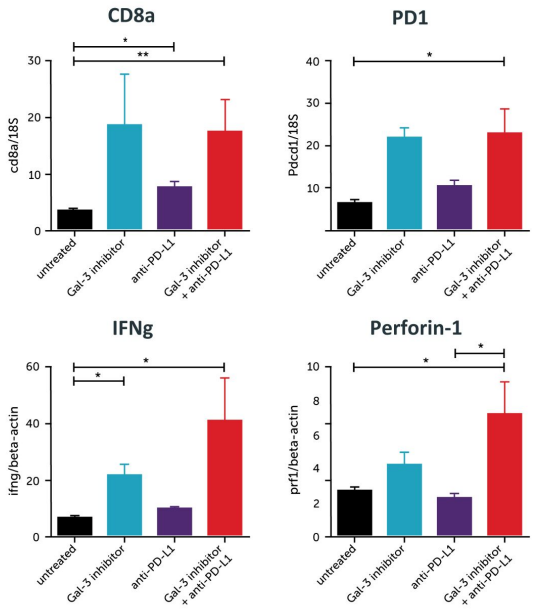



Vuong, L.,et al. (2019) Cancer Res 79, 1480-1492

# 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 tumor cytotoxic T cell function
- Galectin-3 inhibitor increases INF-g and PD-1 both associated with increased response to checkpoint inhibitors



# GB1211: Clean safety profile in Phase 1 SAD/MAD studies

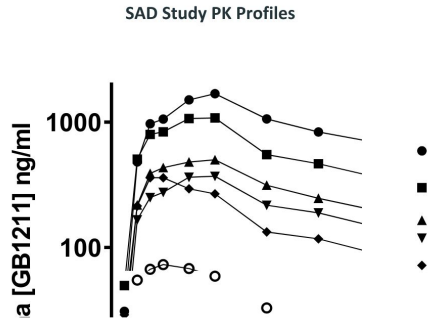
Part A: SAD in HV
N=40 across 5 cohorts

↓

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

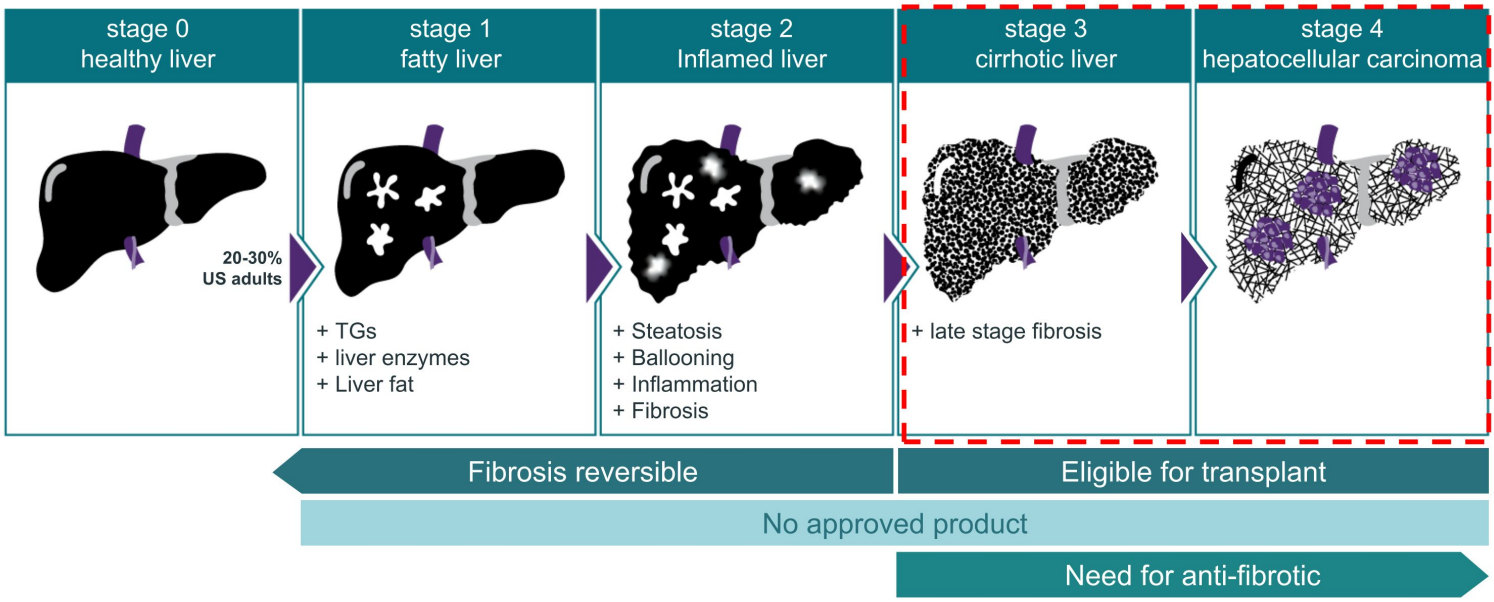
*Completed*

- 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

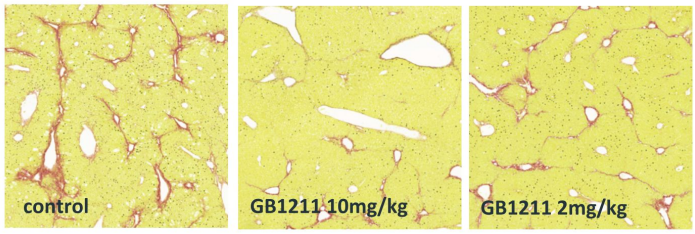



h = hours, N = sample size

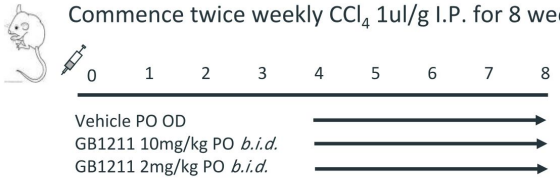
# Liver Cirrhosis: High unmet need with no available treatments

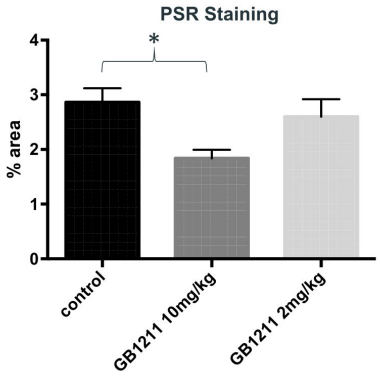


# GB1211: Blocks $CCl_4$-induced liver fibrosis




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



# World leading advisors

## Galectin Biology

**Hakon Leffler, MD PhD**
Co-founder Galecto,
Professor, Microbiology, Immunology and Glycobiology, Lund University

**Ulf Nilsson, PhD**
Co-founder Galecto,
Professor, Organic Chemistry, Lund University

**Tariq Sethi, MD PhD**
Co-founder Galecto,
Professor Emeritus, King's College London

## IPF

**Toby Maher, MD PhD**
Professor, USC, California and Imperial College, London

**Kevin Brown, MD**
Professor, National Jewish Health, Colorado

**Nik Hirani, MD PhD**
Clinical Director, Edinburgh University

**Tariq Sethi, MD PhD**
Co-founder Galecto,
Professor Emeritus, King's College London

**Seamas Donnelly, MD PhD**
Professor, Trinity College Dublin

**Patricia Sime, MD PhD**
Professor, VCU, VA

## Liver Fibrosis

**Frank Tacke, MD PhD**
Professor, University Hospital Aachen,
Germany

**Derek Mann, MD**
Professor, Newcastle University

**Arun Sanyal, MD PhD**
Professor, Virginia Commonwealth University

**Jonathan Fallowfield, MD PhD**
Professor, Edinburgh University

**Neil Henderson, MD PhD**
Professor, Edinburgh University

**Yury Popov, MD PhD**
Assistant Professor, Harvard Medical School

## Myelofibrosis

**Srdan Verstovsek, MD**
Professor, MD Anderson Cancer Center, Houston, TX

**Claire Harrison, MD**
Professor, Guy's and St Thomas, London, UK

**Dr Adam Mead**
Professor of Haematology, University of Oxford

**Dr John O Mascarenhas**
Associate professor, Mount Sinai, NY

**Dr Raajit K. Rampal**
Hematologic Oncologist, Memorial Sloan Kettering, NY

**Dr Aron Gerds**
Assistant Professor, Cleveland Clinic, Cleveland

## Oncology

**Alexander M.M. Eggermont, MD, PhD.**
Professor Utrecht University
The Netherlands

**William L. Redmond, PhD**
Director Providence Cancer Institute
Portland, OR

**Roy S. Herbst, MD, PhD**
Professor,
Yale University, CT

**Federico Cappuzzo, M.D.**
Director, IRCCS,
Rome, Italy