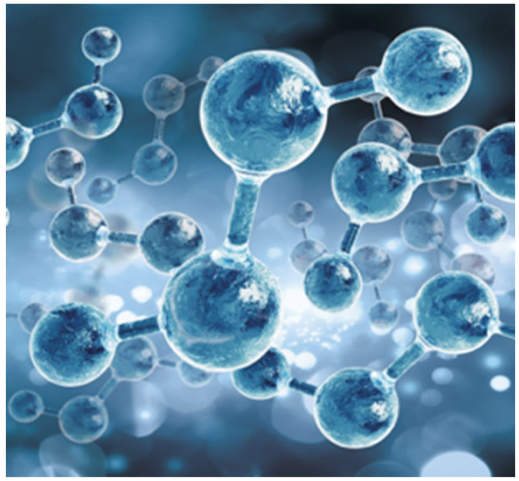
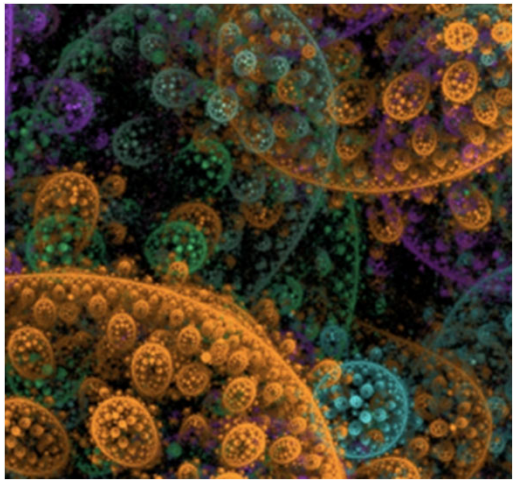
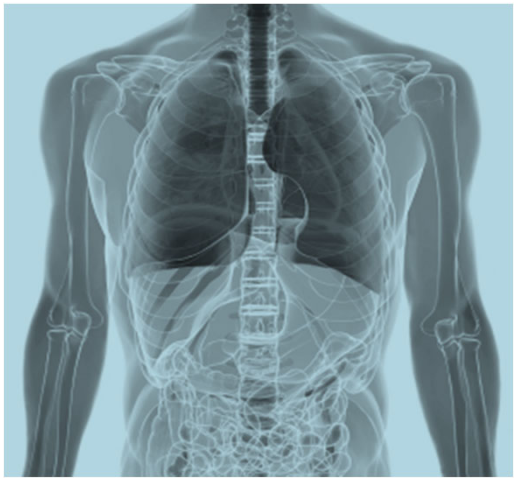
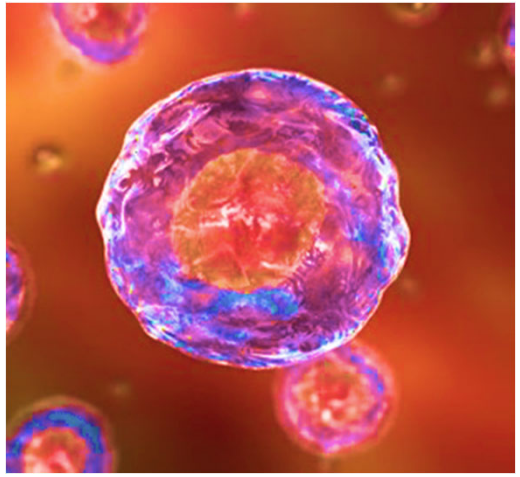
Galecto

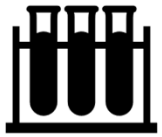
Positive Clinical Data Showing Significant Improvements in Decompensated Cirrhosis Patients

Webcast – Nov 8, 2022 at 8 am EST

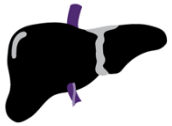
Forward-looking statements

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.

GULLIVER-2: Encouraging top-line results in a decompensated cirrhosis patient population – statistically significant reductions in liver enzymes



- GB1211 was **well-tolerated** with a **predictable PK** profile consistent with the option of repeated dosing in patients with hepatic impairment
- Gal-3 levels were reduced in the GB1211 treated population, demonstrating **target engagement**



- GB1211 reduced Gal-3 levels with a **fast onset** and concordant changes in liver biochemistry, liver stiffness and CAP, over 12 weeks
- Data suggests that GB1211 may **improve liver inflammation and reduce liver injury**

Future studies will focus on exploring GB1211 in an optimal setting of patients with liver disease

CAP, controlled attenuation parameter; Gal-3, galectin-3; PK, pharmacokinetics

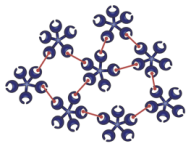
GULLIVER-2

Galecto

Well-capitalized clinical-stage biotech with near-term catalysts

Innovative platform developing next-generation treatments in oncology and fibrosis

INNOVATIVE PLATFORM TARGETING CORE DISEASE PROCESSES



- Pioneers in **galectin-** and **LOXL2**-based pharmacology
- **First-in-class small-molecule inhibitors** targeting galectin-3 and LOXL2

ADVANCING BROAD ONCOLOGY AND FIBROSIS PIPELINE

- **Four** ongoing Phase 2 trials:
  - Non-small cell lung cancer (**NSCLC**)
  - Idiopathic pulmonary fibrosis (**IPF**)*
  - Myelofibrosis (**MF**)
  - Liver **cirrhosis***

ADDRESSING DISEASE AREAS WITH SIGNIFICANT UNMET MEDICAL NEED

- Galecto's programs all address:
  - Diseases characterized by clear **unmet medical need**
  - **Multi-billion-dollar** market opportunities

WELL-CAPITALIZED WITH BROAD PIPELINE AND NEAR-TERM CATALYSTS

- **Newsflow includes four** Phase 2 read-outs through 2023
- Cash balance of ~$86M as of 6/30/2022, funding all Phase 2 trials with **runway into 2H 2024**

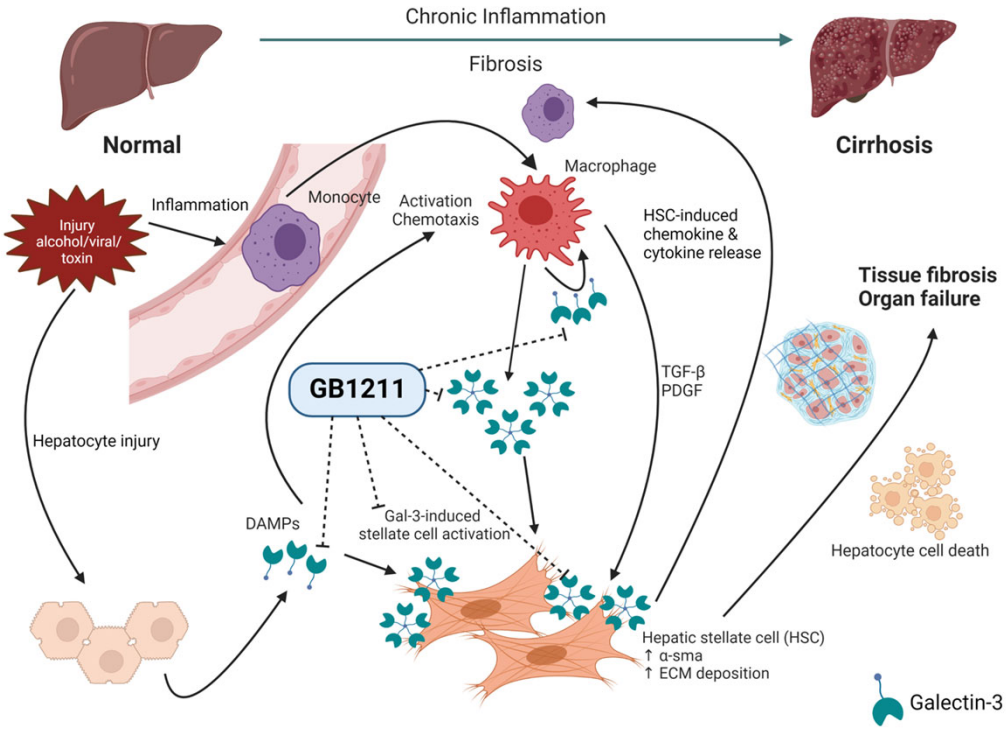
LOXL2: lysyl oxidase-like 2

* Trials fully enrolled

Galectin-3 is a key pro-fibrotic mediator and highly expressed in liver fibrosis

GB1211 targets galectin-3 and affects key elements in the fibrosis cascade

- Galectin-3 is linked to liver fibrosis and inflammation
  - High expression in severe liver disease
  - Strongly affects key cells and cytokines in fibrosis cascade
- GB1211 is a potent, selective Galectin-3 inhibitor
  - Orally active
- GB1211 has successfully completed phase I in healthy volunteers with no apparent safety issues
  - No toxicity observed in preclinical studies
  - Currently in clinical phase 2 studies in cirrhosis and lung cancer (NSCLC) patients

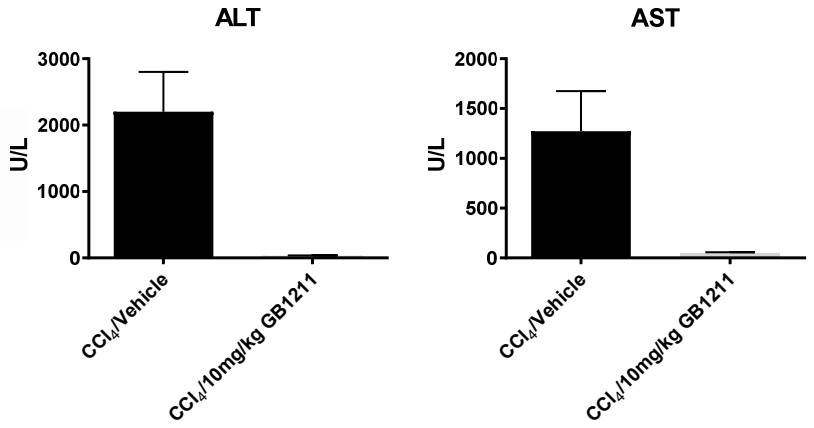


Slack *et al. Int J Biochem Cell Biol* 2020;130:105881
Henderson *et al.* Proc Natl Acad Sci 2006;103(13):5060

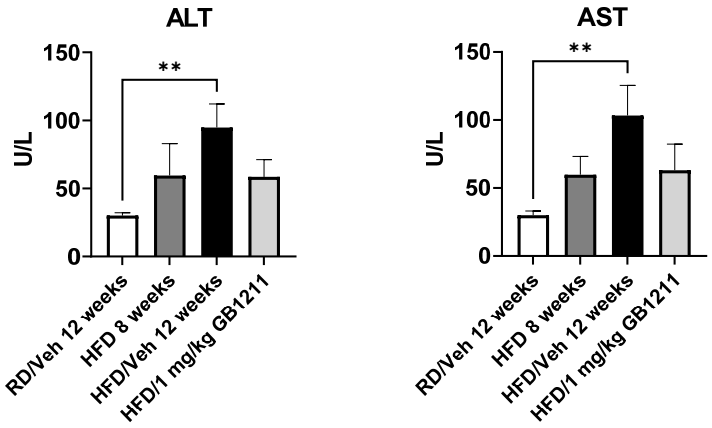
GB1211 reduces liver transaminases & fibrosis in preclinical models of liver disease

Liver transaminases

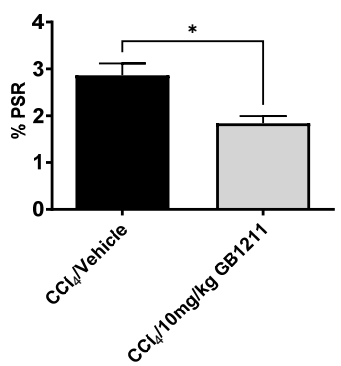
CCl_4

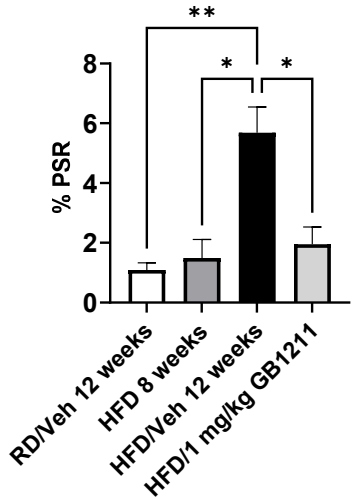


HFD

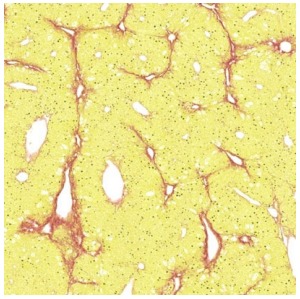


Fibrosis (%PSR)

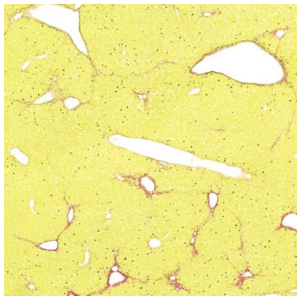




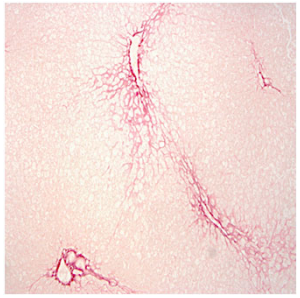
CCl_4/Veh



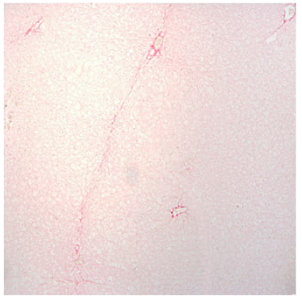
CCl_4/GB1211



HFD/Veh



HFD/GB1211



Introduction to our experts

Massimo Pinzani, MD, PhD, FRCP, FAASLD

Sheila Sherlock Chair of Hepatology

UCL Institute for Liver and Digestive Health

Royal Free Hospital, London, UK

Michael Charlton, MBBS, FRCP

Chief of Hepatology and Medical Director,

Transplant Institute at the University of Chicago

Cirrhosis – An Introduction

Massimo Pinzani, MD, PhD, FRCP, FAASLD

Sheila Sherlock Chair of Hepatology

UCL Institute for Liver and Digestive Health

Royal Free Hospital, London, UK

Cirrhosis - The End-stage of Every Chronic Liver Disease - Overview

Decompensated cirrhosis has the prognosis and features of a deadly disease

>2M prevalent cases[1] *>3M prevalent cases[1]*

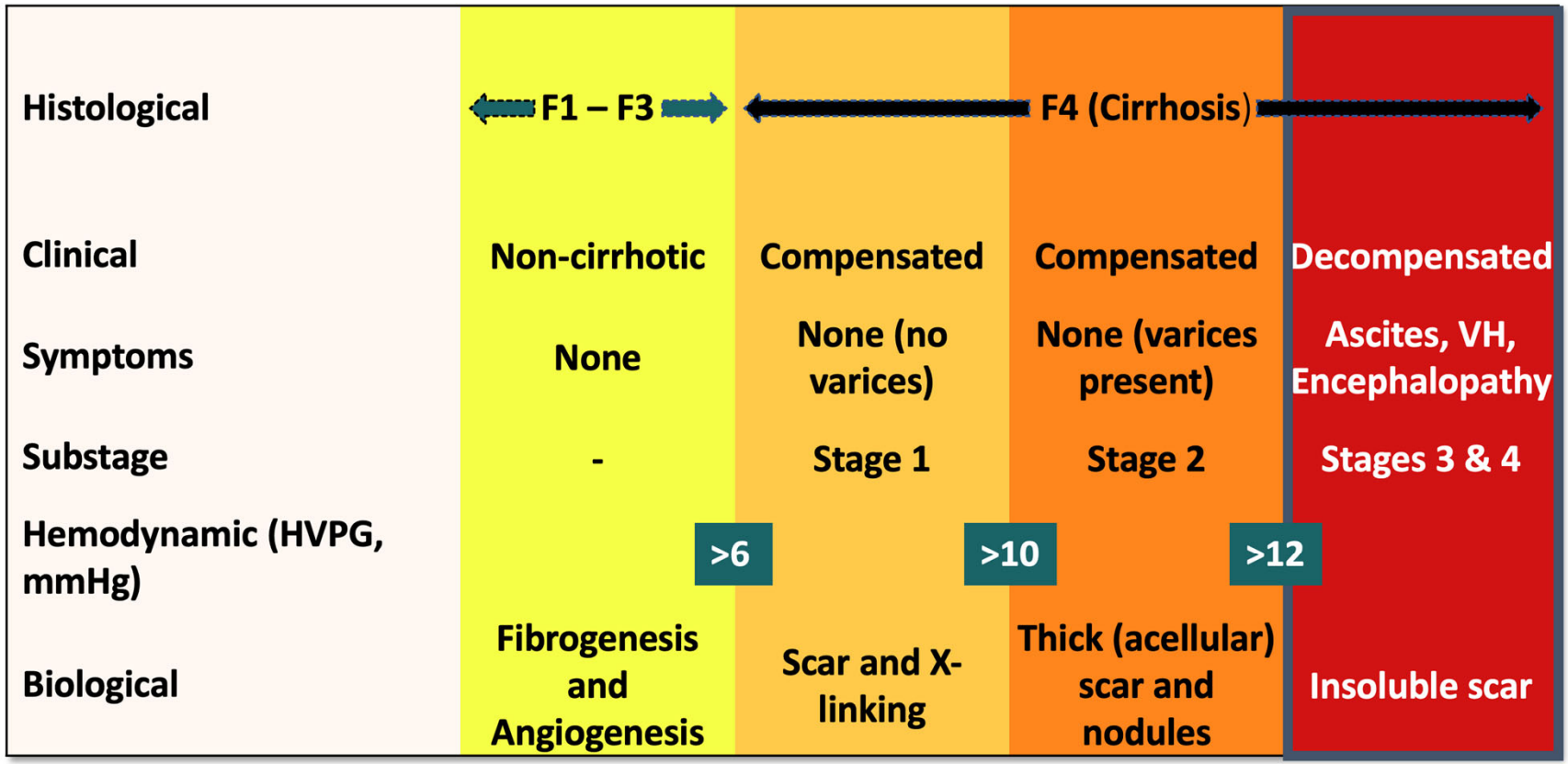
No definitive treatment opportunities other than liver transplantation, but addressing etiological factors (e.g. antivirals or alcohol abstinence) might improve decompensation

- *Cirrhosis is the ultimate final outcome of chronic liver damage and suboptimal repair*
- *Severe, progressive liver fibrosis that ultimately leads to liver failure*
- *Caused primarily by NASH, alcoholic liver disease, viral hepatitis*
- *Median survival of appr. 2 years for patients with decompensated cirrhosis[2]*
- *Implies an enormous amount of highly specialized clinical care*
- *Prevalence on the rise in the US and the EU*

1. Sepanlou et al. Lancet Gastroenterol Hepatol 2020;5:245–66
2. D'Amico et al. J Hep 2006;44:217-31

Classification of Chronic Liver Disease

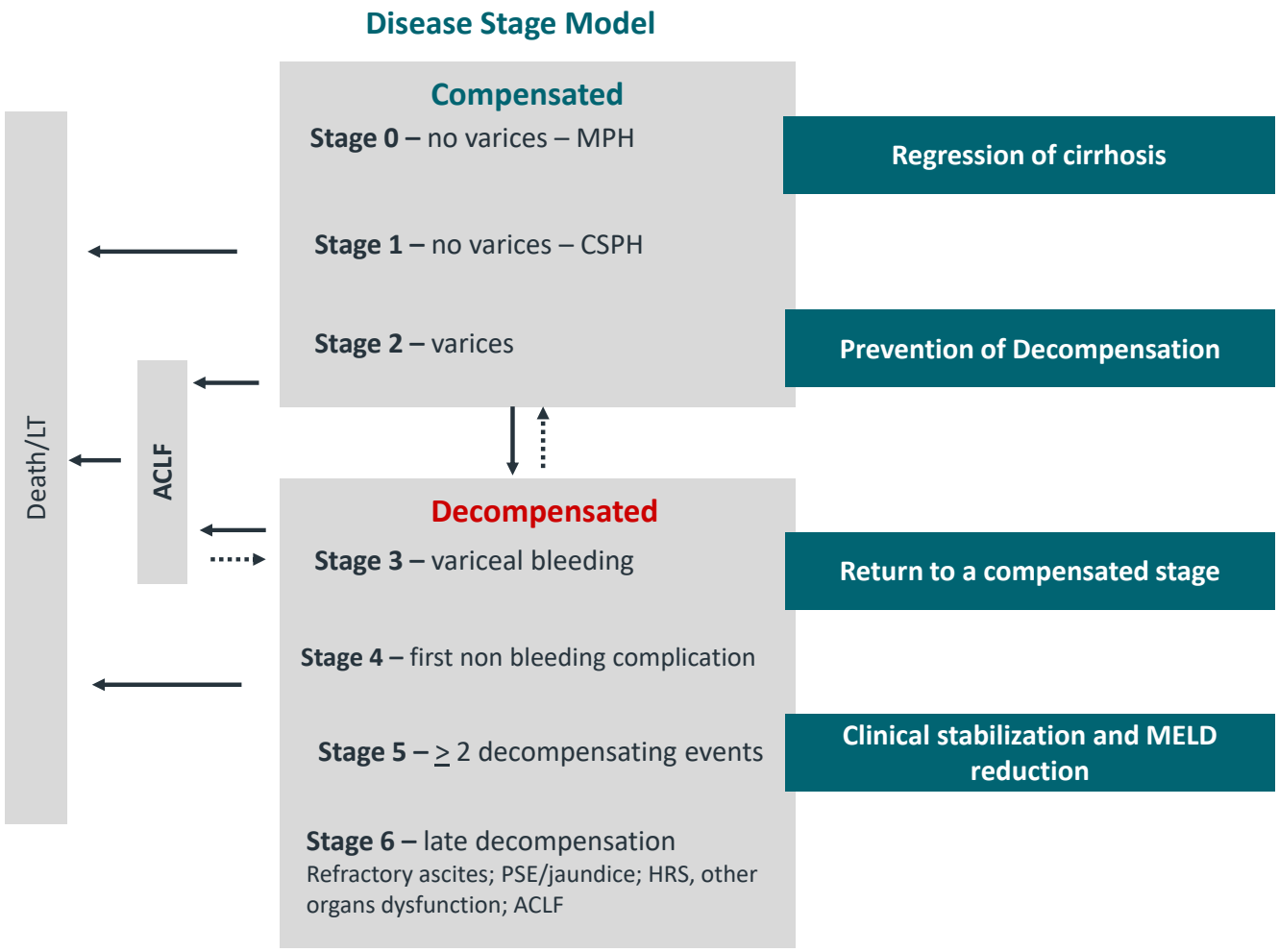
Patients with decompensated cirrhosis have an overall survival on par with difficult-to-treat cancer patients



GULLIVER-2

Garcia-Tsao et al. Hepatology. 2010 Apr;51(4):1445-9

Cirrhosis - Clinical Endpoints of Treatment



Previous Studies in a Child-Pugh A Population Have Not Shown Benefit on Various Markers

A massive unmet medical need for the treatment of cirrhosis still exists

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
MELD	NC	NC	0.2	0.4	NC	NC	NC	NC
ALT (U/L)	−3	−4	NC	NC	−5	−1	NC	NC
GGT (U/L)	−8	−4	NC	NC	−7	−8	NC	NC
Total bilirubin (µmol/L)	NC	NC	−0.5	0.3	0.1	0.1	NC	NC
Transient elastography (kPa)	−0.7	−0.7	−6.7	−0.3	NA	NA	−2.3	−0.5

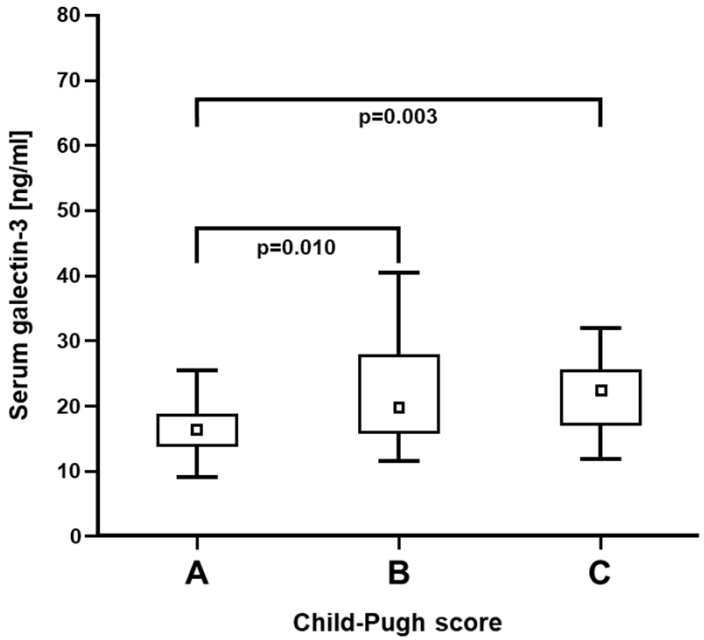
Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

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

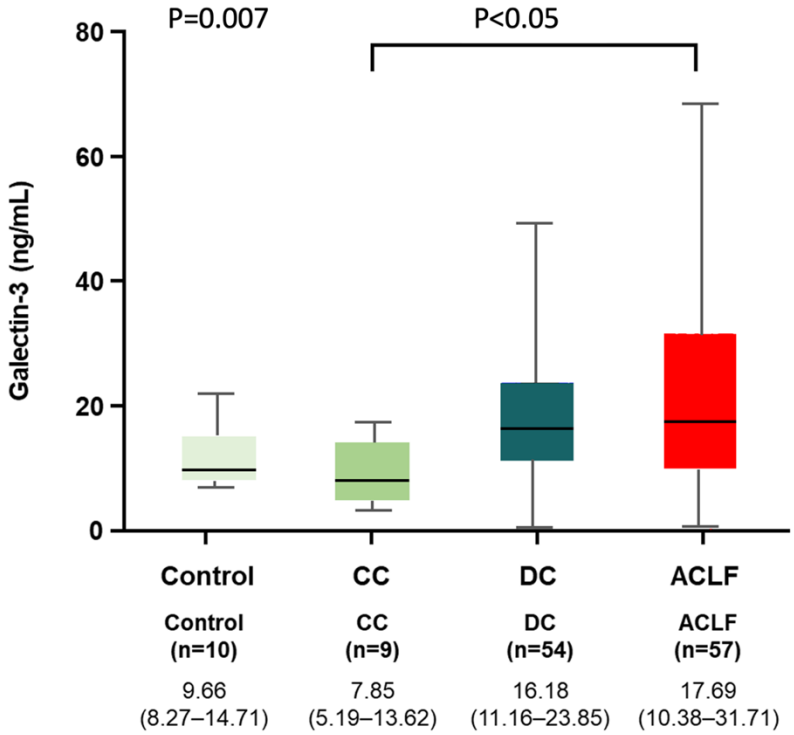
1. Harrison et al. J Hepatol 2020;73(1):26–39;
2. Garcia-Tsao et al. J Hepatol 2020;72(5):885–895;
3. Harrison et al. Gastroenterology 2018;155(4):1140–1153;
4. Chalasani et al. Gastroenterology 2020;158(5):1334–1345.e5

Galectin-3 is Overexpressed in Advanced Chronic Liver Disease

High Galectin-3 levels in liver disease have been correlated with severe disease[1,2]



Gudowska *et al.*, 2015



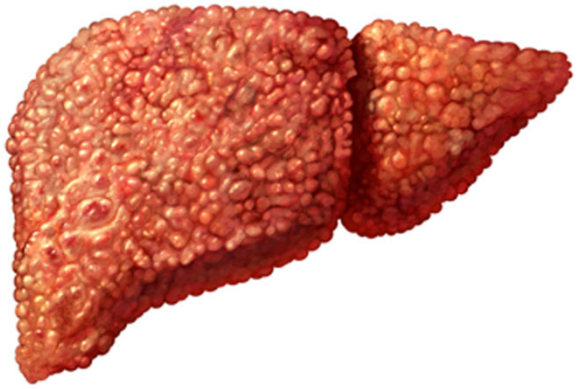
Adapted from Cervantes-Alvarez et al., 2022

ACLF, acute-on-chronic liver failure; CC, compensated cirrhosis; DC, decompensated cirrhosis; Gal-3, galectin-3

1. Gudowska et al. *Ann Clin Lab Sci* 2015;45:669–73;
2. Cervantes-Alvarez et al. *Liver Int* 2022; 00:1–14;

Summary

- Cirrhosis is a major challenge for the health care system worldwide
- Apart from direct acting antiviral agents, no disease modifying treatments are currently available
- There is an urgent need for safe and effective therapies addressing the significant unmet medical need that cirrhosis accounts for
- Galectin-3 is over-expressed in chronic liver disease and is correlated with more severe disease



GB1211, an oral galectin-3 inhibitor, in decompensated cirrhotic patients: initial findings from the Phase 2 randomized, placebo-controlled GULLIVER-2 trial

Bertil Lindmark, MD, PhD

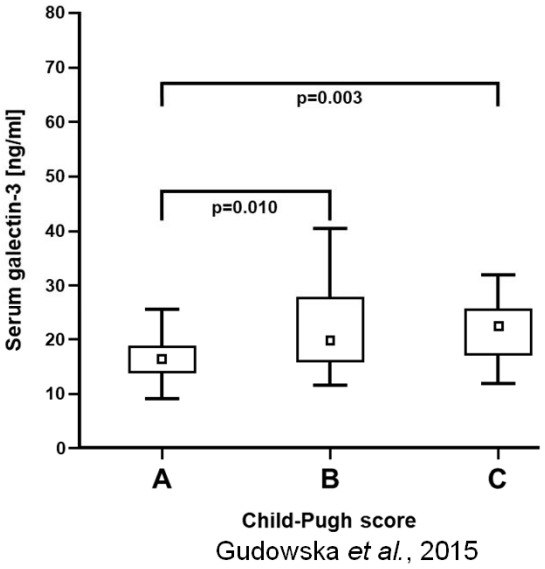
Chief Medical Officer, Galecto Inc.

Background of Galectin-3 and GB1211

Galectin-3 is Overexpressed in Advanced Chronic Liver Disease

High Galectin-3 levels in liver disease have been correlated with severe disease[1,2]

- Gal-3 is a beta-galactoside-binding protein which regulates inflammation and fibrosis in the liver[3]
- Gal-3 has a potential role as a prognostic indicator of liver fibrosis[2]
- GB1211 is a specific and high-affinity, small molecule, oral Gal-3 inhibitor that has shown anti-fibrotic and anti-cancer effects in various pre-clinical models including preclinical models of liver fibrosis[4]



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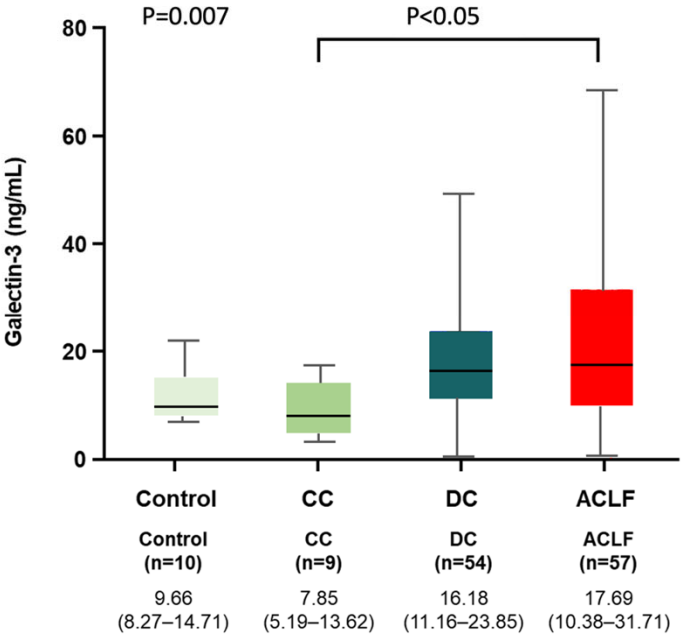

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2. Cervantes-Alvarez et al. *Liver Int* 2022; 00:1–14;
3. Henderson et al. *Proc Natl Acad Sci USA* 2006;103:5060–5;
4. Zetterberg et al. *J Med Chem* 2022;65(19):12626–38

GULLIVER-2

Trial design

GULLIVER-2 – Part 2

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

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

GULLIVER-2

Results

Patients Were Well-matched Based on Child-Pugh Score

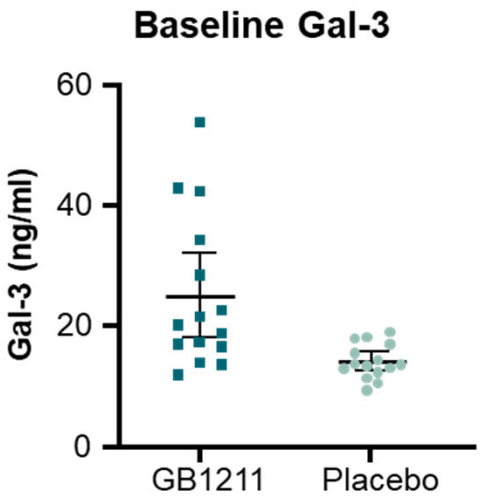
		GB1211 (N=15)	Placebo (N=15)
Age, years; median (range)		58 (48–66)	51 (45–66)
Sex, male; n (%)		10 (66.7)	10 (66.7)
Race, White/Caucasian; n (%)		15 (100)	15 (100)
Body mass index, kg/m^2; median (range)		31.3 (19.1–39.8)	24.9 (18.1–32.9)
Child-Pugh score, median (range)		8 (7–11)	8 (7–9)
Encephalopathy grade	None	14 (93.3%)	13 (86.7%)
	1	1 (6.7%)	1 (6.7%)
	2	0	1 (6.7%)
Ascites	Absent	3 (20.0%)	5 (33.3%)
	Slight	9 (60.0%)	9 (60.0%)
	Moderate	3 (20.0%)	1 (6.7%)
Etiology of cirrhosis	Alcoholic cirrhosis	12 (80.0%)	9 (60.0%)

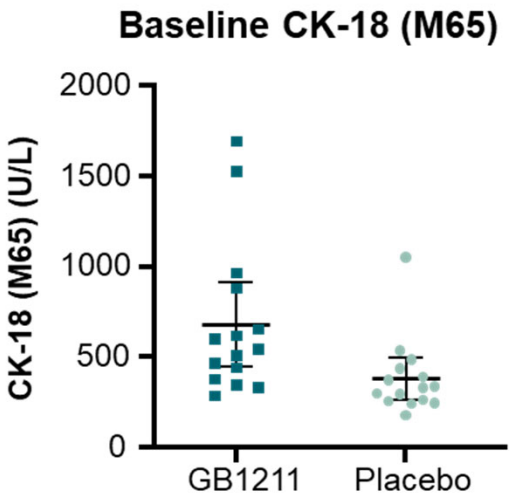
GULLIVER-2

Baseline Laboratory and Key Biomarker Characteristics

Patients in the GB1211 group had higher baseline Gal-3 and CK-18 (M65) values, compared with placebo

Baseline laboratory and key biomarker characteristics	GB1211 (N=15) (mean ± SD)	Placebo (N=15) (mean ± SD)
AST (IU/L)	47.27 ± 16.38	40.55 ± 36.23
ALT (IU/L)	30.05 ± 8.33	29.54 ± 33.12
Total bilirubin (mg/dL)	1.79 ± 1.38	2.00 ± 0.93
Creatinine (mg/dL)	0.99 ± 0.39	0.78 ± 0.12
WBC (k/uL)	6.64 ± 2.99	5.25 ± 2.06
Platelets (k/uL)	139.21 ± 61.38	114.11 ± 55.11
INR	1.48 ± 0.29	1.45 ± 0.24
MELD	15.07 ± 6.49	12.93 ± 3.28
Gal-3 (ng/mL)	25.13 ± 12.68	14.25 ± 2.86
CK-18 (M65) (U/L)	679.11 ± 423.83	379.29 ± 209.50





ALT, alanine transferase; AST, aspartate transferase; CK-18, cytokeratin-18; Gal-3, galectin-3; INR, international normalized ratio; (I)U/L, (international) units per liter; MELD, model for end-stage liver disease; SD, standard deviation; WBC, white blood cell

GULLIVER-2

Galecto

Safety

Seventeen TEAEs were reported (9 with GB1211 and 8 with placebo)

No related adverse events were observed in patients receiving GB1211

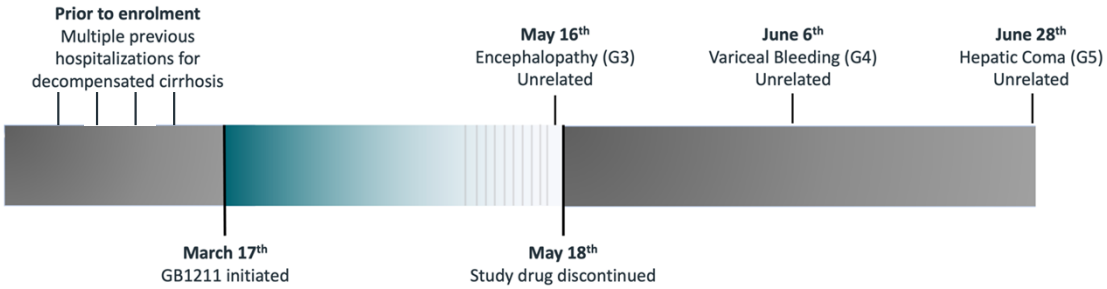
Non-serious adverse events

Preferred Term	Statistic	GB1211 (N = 15)	Placebo (N = 15)	Grade
Headache	n (%) E	0	2 (13.33) 3	1,1,1
Blood loss anemia	n (%) E	1 (6.67) 1	0	2
Pyrexia	n (%) E	0	1 (6.67) 1	1
COVID-19	n (%) E	1 (6.67) 1	0	1
Nasopharyngitis	n (%) E	1 (6.67) 1	0	1
Blood creatinine increased	n (%) E	1 (6.67) 1	0	1
Blood urea increased	n (%) E	1 (6.67) 1	0	1
SARS-CoV-2 test positive	n (%) E	0	1 (6.67) 1	1
Back pain	n (%) E	0	1 (6.67) 1	1
Epistaxis	n (%) E	0	1 (6.67) 1	1
Hemoptysis	n (%) E	0	1 (6.67) 1	1
Pruritus	n (%) E	1 (6.67) 1	0	1

E, number of events; G, grade; n, number of patients; TEAE, treatment-emergent adverse event

Serious adverse events – 1 patient; 3 unrelated SAEs

- One patient treated with GB1211 experienced **three** separate serious adverse events, none of which were considered related to the drug by the investigator



GULLIVER-2

Pharmacokinetics

- Exposure to GB1211 was **moderately increased** in patients with hepatic impairment
  - Child-Pugh B: AUC increased by 60%, C_{max} unaffected, half-life prolonged after single dose
  - Child-Pugh C: AUC increased by 60%, C_{max} increased by 35%, half-life prolonged after single dose
- Steady-state already **reached by Day 7** in Child-Pugh B patients after repeated (BID) doses
- Accumulation at steady-state (vs. Day 1) is **3-fold** in Child-Pugh B patients (vs. 2-fold in healthy subjects) after BID doses
- Free (unbound) fraction was **not affected** in Child-Pugh B or C patients after single dose

- Predictable and moderate build up of GB1211 levels over the course of treatment allows for repeated dosing of at least 100mg GB1211 BID in patients with liver impairment in future studies

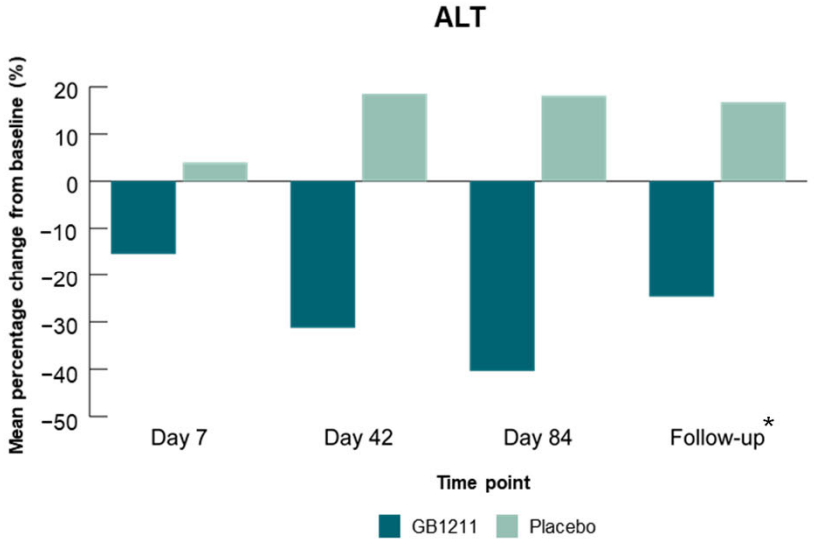
AUC, area under the plasma concentration versus time; BID, twice a day; C_{max}, maximum plasma concentration

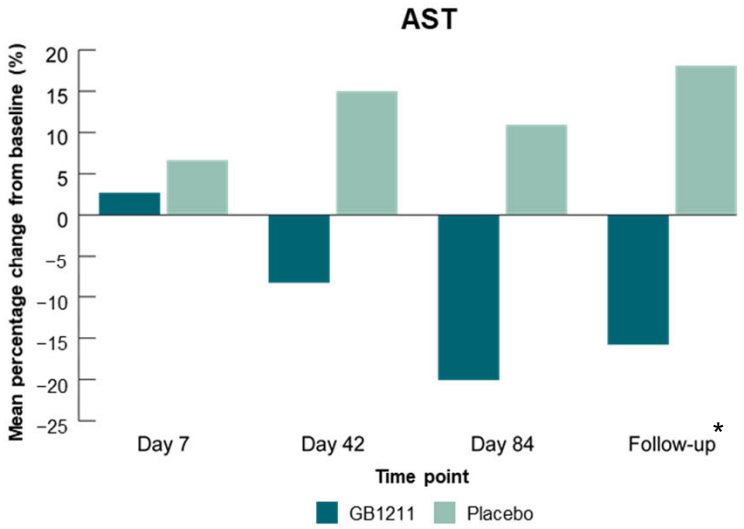
GULLIVER-2

Clinical biochemistry

Liver-related Biochemistry Results

Consistent and increasing reduction in liver enzymes for GB1211 patients





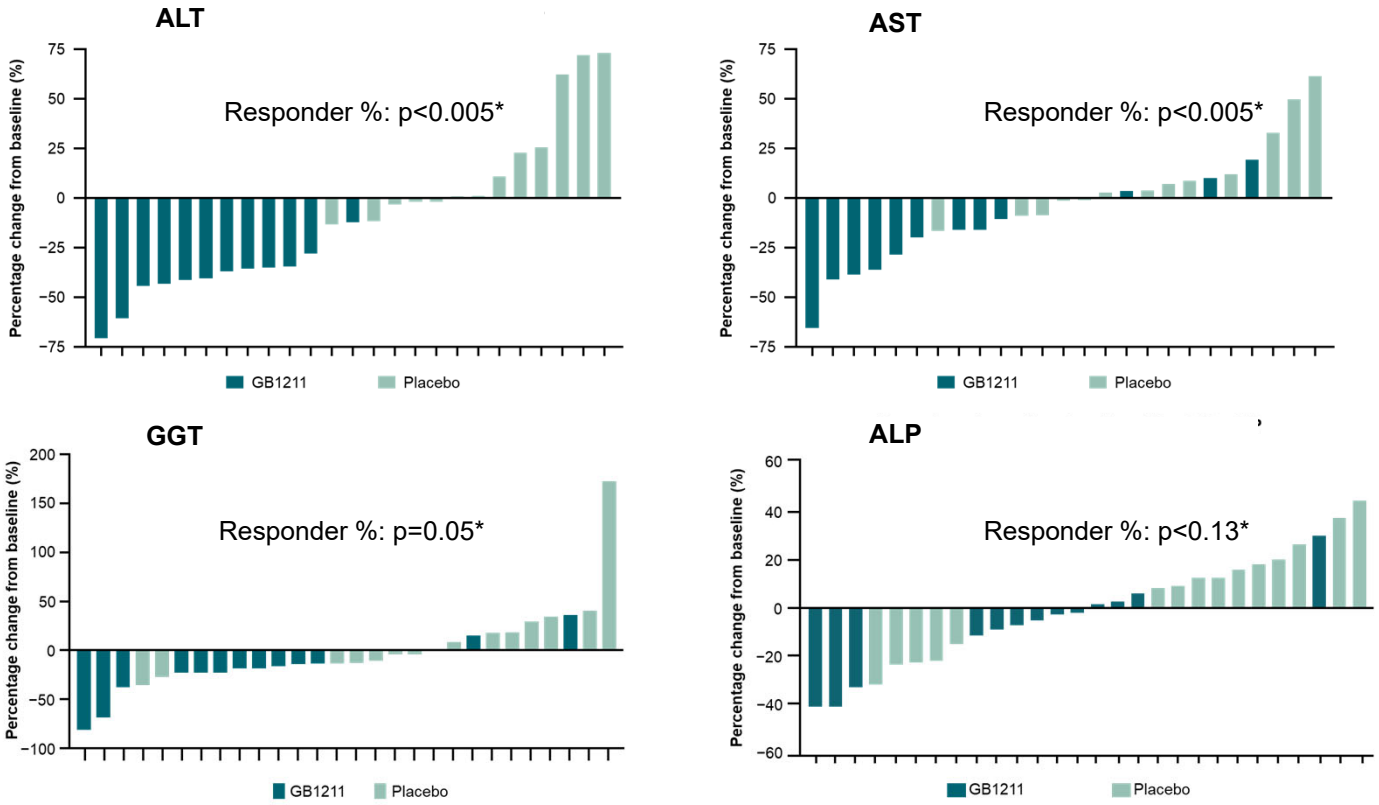
Treatment effect (GB1211-Placebo) [%] at Day 84	ALT	AST	GGT	ALP
Mean	-58.44	-32.40	-37.77	-14.76
95% confidence interval	(-79.00, -37.88)	(-51.63, -13.17)	(-69.47 , -6.06)	(-31.92 , -2.40)
p-value	0.0001	0.002	0.0214	0.0889

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

GULLIVER-2

Galecto

Encouraging Reductions in ALT, AST, GGT and ALP at Day 84 in Patients Receiving GB1211, Compared With Placebo



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

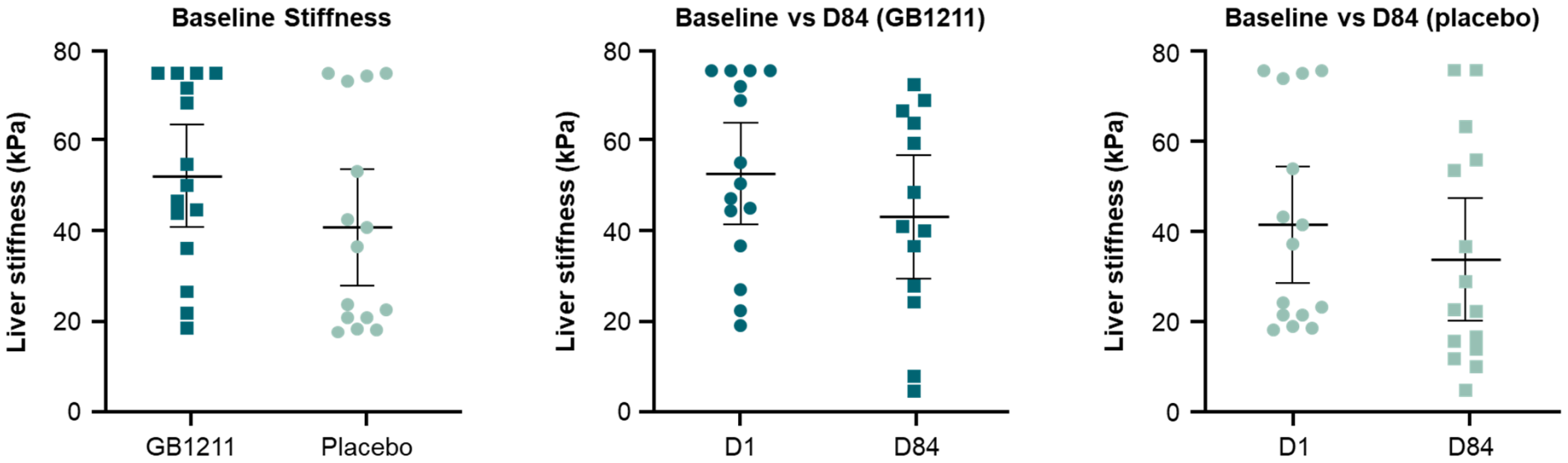
GULLIVER-2

Galecto

GULLIVER-2 : Results

Exploratory analysis: Vibration-controlled transient elastography

More Patients Demonstrated Improvements in Liver Stiffness* in GB1211 Versus Placebo



- Mean change from baseline to Day 84: **GB1211: −9.66 (SD 22.52) kPa**; **placebo: −7.62 (SD 11.34) kPa**
- In the GB1211 group, **10/13 (77%)** of evaluable patients showed a **≥4kPa reduction in liver stiffness**, compared with **8/15 (53%)** of patients in the placebo group

*Liver stiffness measured with FibroScan at baseline and Day 84. D, day; kPa, kilopascal; SD, standard deviation

GULLIVER-2

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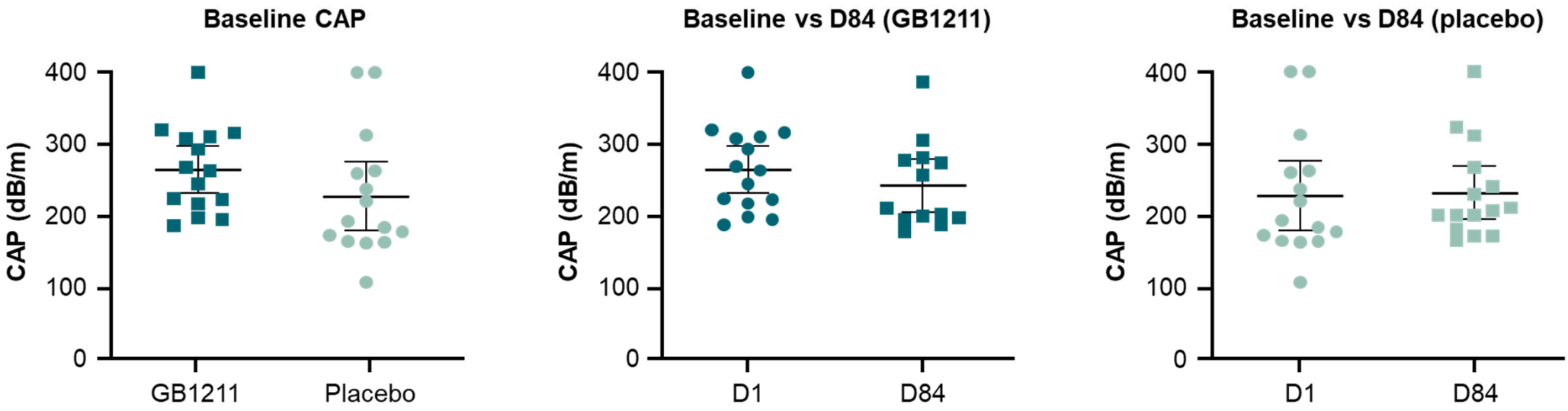
Improvements in CAP* Were Observed With GB1211 Versus Placebo



- Mean change from baseline to Day 84: **GB1211: −20.23 (SD 42.81) dB/m**; **placebo: 4.13 (SD 63.35) dB/m**
  - GB1211 patients had higher CAP at baseline compared with placebo patients (mean **265** vs. **229 dB/m**)

*CAP measured with SmartExam®. CAP, controlled attenuation parameter; D, day; dB/m, decibels per meter; SD, standard deviation

GULLIVER-2

Galecto

GULLIVER-2

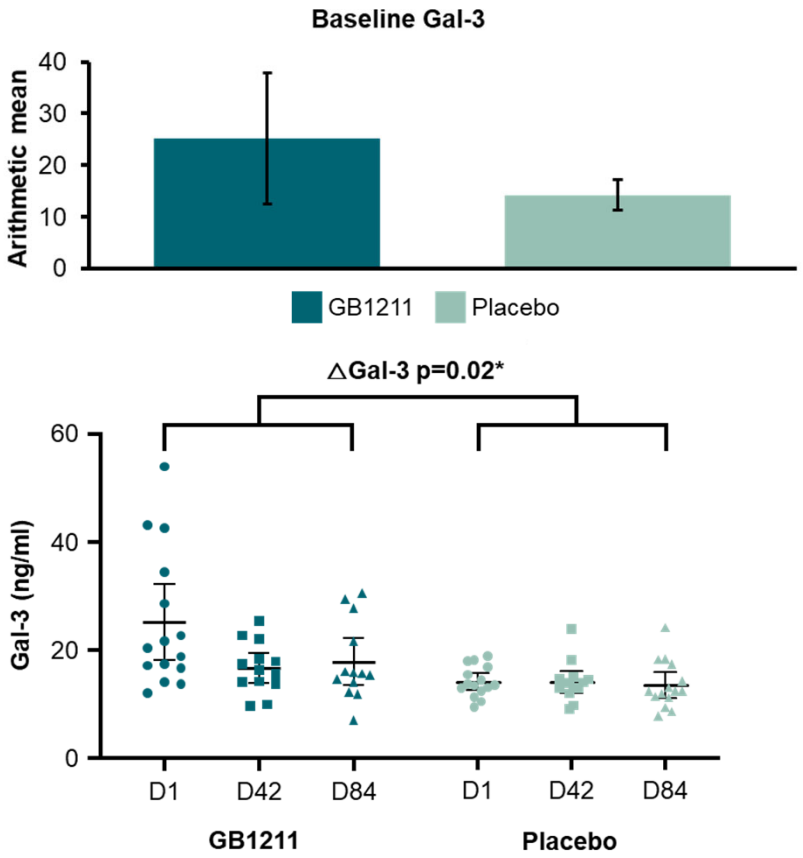
Exploratory analysis: Biomarkers

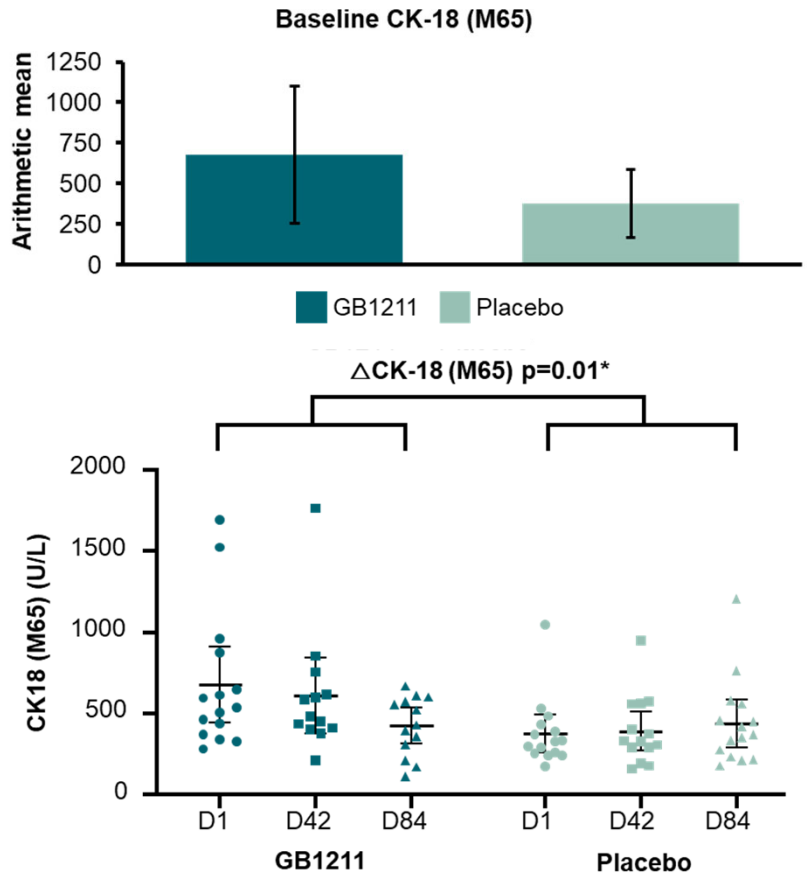
GB1211 Reduces Galectin-3 and CK-18 (M65)

GB1211 demonstrated target engagement and potential anti-apoptotic properties

GULLIVER-2

Galectin-3





CK-18 (M65)

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

Putting 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.

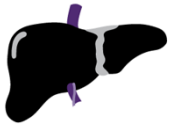
*Data are absolute changes from baseline to Day 84. †Modified ITT population.
ALT, alanine transferase; CAP, controlled attenuation parameter; dB/m, decibels per meter; GGT, gamma-glutamyl transferase; ITT, intent to treat; kPa, kilopascal; MELD, model for end-stage liver disease; NA, not available; 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

Conclusions and next steps

GULLIVER-2: Encouraging top-line results in a decompensated cirrhosis patient population

- GB1211 was **well-tolerated** with a **predictable PK** profile consistent with the option of repeated dosing in patients with hepatic impairment
- Gal-3 levels were reduced in the GB1211 treated population, demonstrating **target engagement**



- GB1211 reduced Gal-3 levels with a **fast onset** and concordant changes in liver biochemistry, liver stiffness and CAP, over 12 weeks
- Data suggests that GB1211 may **improve liver inflammation and reduce liver injury**

Future studies will focus on exploring GB1211 in an optimal setting of patients with liver disease

CAP, controlled attenuation parameter; Gal-3, galectin-3; PK, pharmacokinetics

GULLIVER-2

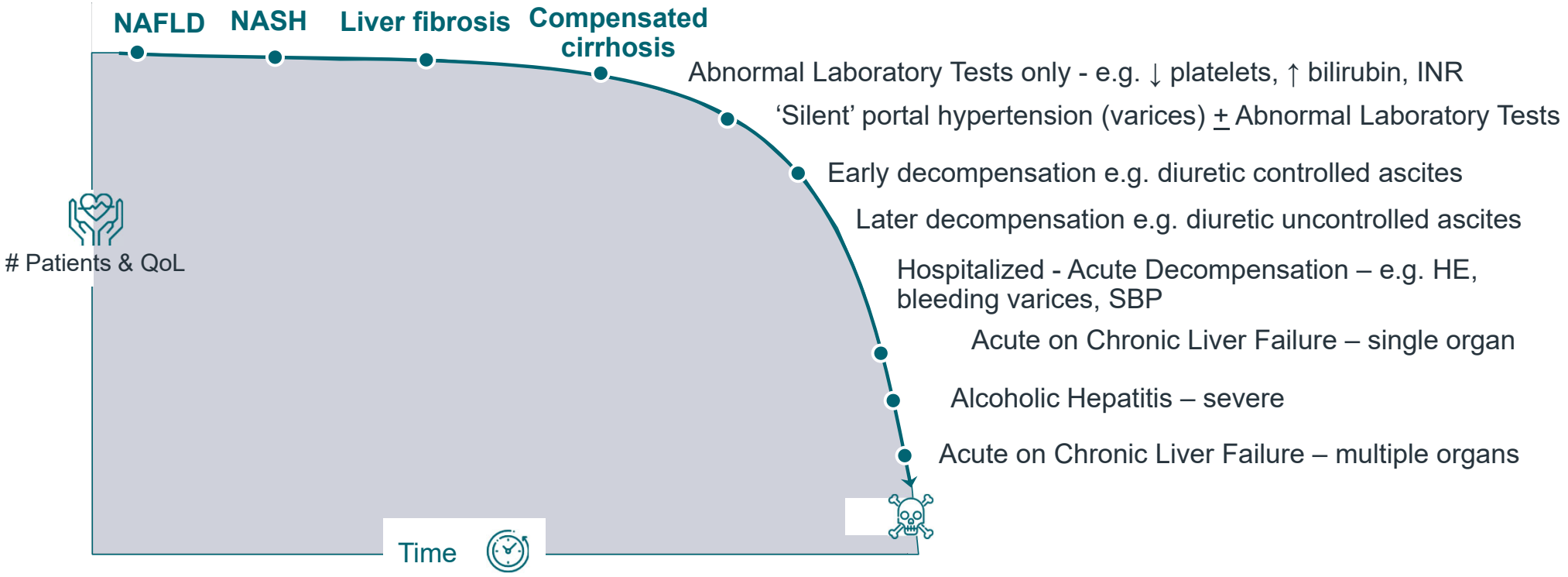
Galecto

GULLIVER-2 – A Clinician's Take

Michael Charlton, MBBS, FRCP

Chief of Hepatology and Medical Director,
Transplant Institute at the University of Chicago

Spectrum of Clinical Manifestations and Complications in Liver Patients – Significant Unmet Medical Need



Putting Data Into Perspective

GULLIVER-2

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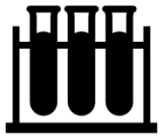

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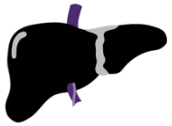

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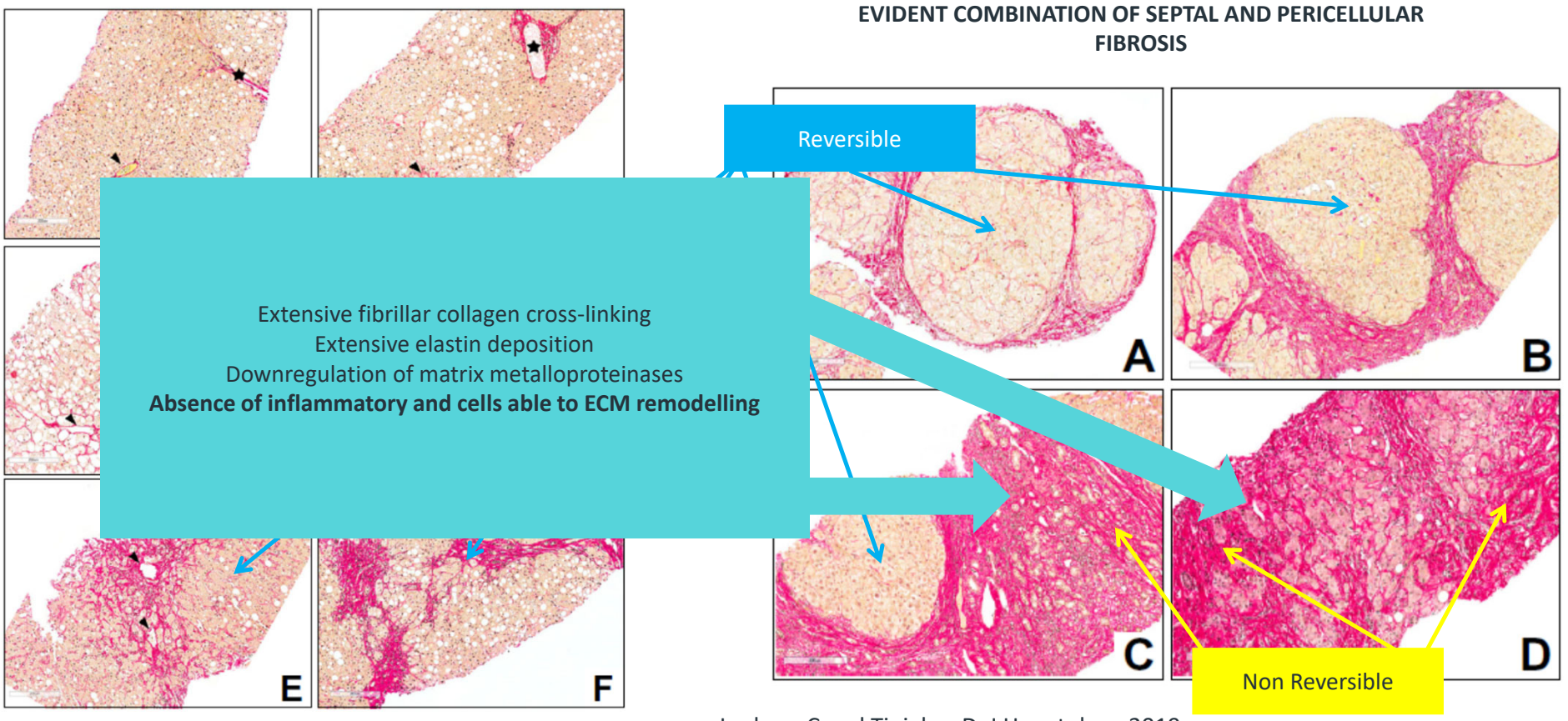
GULLIVER-2

Galecto

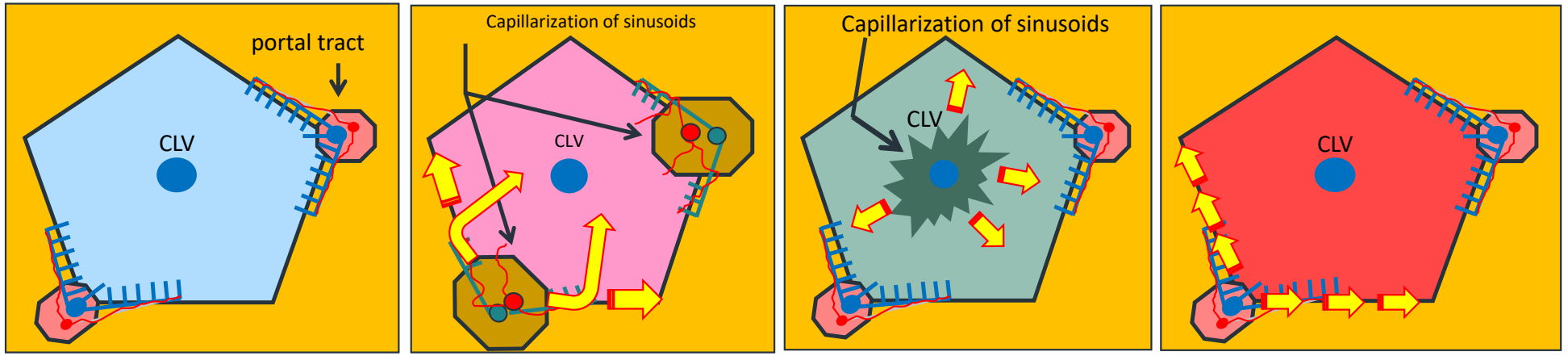
BACK-UP

Reversible and Irreversible Fibrosis in Cirrhotic Liver



Lackner C and Tiniakos D, J Hepatology 2019

Etiology-Driven Liver Fibrosis



e.g. : chronic viral hepatitis, autoimmune hepatitis

e.g. : chronic alcoholic hepatitis, NASH

e.g. : Primary Biliary Cholangitis, Primary Sclerosing Cholangitis

Direction of fibrosis development

Porto-central with early involvement of CLV – Sinusoidal arterialization- Neo-angiogenesis

Zone 3 fibrosis with progressive pan-lobular capillarization of sinusoids

Portal-portal with progressive cholestasis

Friedman SL and Pinzani M. Hepatology February 2022