Galectin Therapeutics Corporate Overview

July 2026



Galectin Therapeutics

Forward-Looking Statements

This presentation contains, in addition to historical information, forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These statements relate to future events or future financial performance and use words such as “may,” “estimate,” “could,” “expect” and others. They are based on our current expectations and are subject to factors and uncertainties that could cause actual results to differ materially from those described in the statements.

These statements include potential therapeutic benefits of our drugs, expectations, plans and timelines related to our clinical trials, supporting activities, potential partnering opportunities and estimated spending for 2026 and beyond. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, any clinical trials may not product positive data.

Future phases or future clinical studies may not begin or produce positive results in a timely fashion, if at all, and could prove time consuming and costly. Plans regarding development, approval and marketing of any of our drugs are subject to change at any time based on the changing needs of our company as determined by management and regulatory agencies. Strategies and spending projections may change. We may be unsuccessful in developing partnerships with other companies or obtaining capital that would allow us to complete our clinical trials or further develop and/or fund any future studies or trials.

To date, we have incurred operating losses since our inception, and our future success may be impacted by our ability to manage costs and finance our continuing operations. For a discussion of additional factors impacting our business, see our Annual Report on Form 10-K for the year ended December 31, 2025, and our subsequent filings with the SEC. You should not place undue reliance on forward-looking statements. Although subsequent events may cause our views to change, we disclaim any obligation to update forward-looking statements.

Investment Highlights

Developing therapeutics to improve the lives of patients with chronic liver diseases and cancer

Focused Pipeline

Belapectin is a novel, potent, galectin-3 inhibitor with Fast Track Designation

Galectin-3 inhibition is a well described target across multiple chronic fibrotic diseases

Carbohydrate-based molecule which is degraded by natural processes resulting in a favorable safety profile

MASH Cirrhosis

Only company to exclusively focus on treatment for MASH cirrhosis and portal hypertension

Belapectin significantly reduced incidence of varices, change in portal pressure and improvement in biomarkers across two large trials

Reached agreement with the FDA on important Phase 3 clinical trial design elements, providing a regulatory pathway forward; final protocol submission expected in Q3 2026

Oncology (Combination Therapy)

Encouraging clinical response in phase 2 trial of difficult-to-treat cancers in combination with checkpoint inhibitor

IND filed and approval to proceed received from FDA (head & neck cancer)

HVPG- Hepatic Vein Pressure Gradient

Highly Experienced Leadership Team



JOEL LEWIS, Chief Executive Officer & President
Executive with over 25 years of management experience in taxation, restructuring, acquisition, and private equity ventures.



KHURRAM JAMIL, M.D., Chief Medical Officer
Has over two decades of experience leading drug development across various stages of clinical trials in the pharmaceutical industry. Led multiple new drug application filings and secured approvals from several regulatory agencies.



JACK W. CALLICUTT, Chief Financial Officer
Over 35 years of public and private company executive experience including more than a decade of audit, tax and SEC registrant experience with Deloitte.



ANDREW VOLOSOV, PH.D. Senior Director, Clinical Pharmacology and Preclinical Research & Development
Over 20 years of experience in clinical pharmacology, DMPK, and bioanalysis across multiple therapeutic areas.



JESSICA KOPACZEWSKI, Senior Director, Clinical Operations
Over 25 years diverse experience in the pharmaceutical research industry supporting global study operations from site to personnel management.



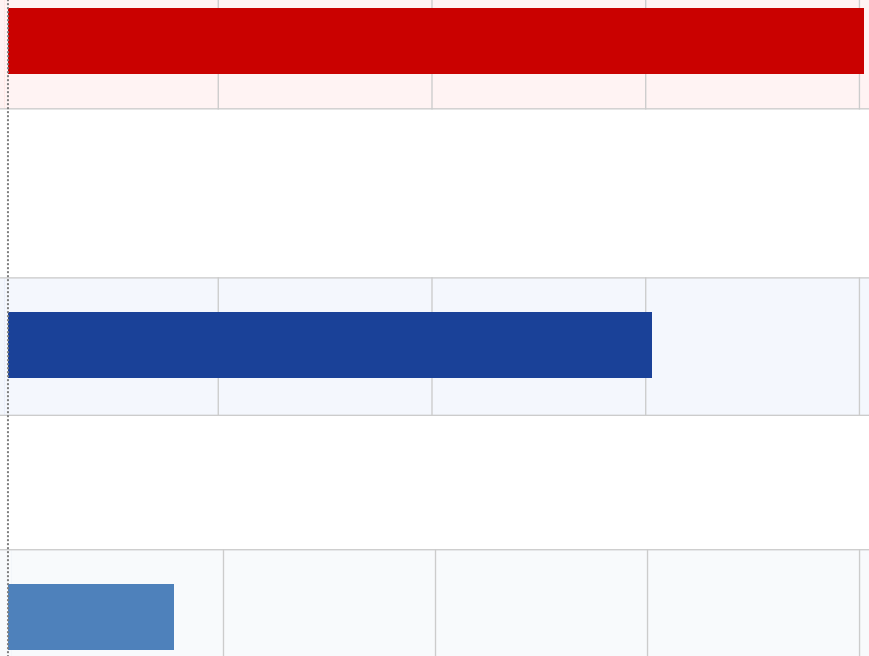
JIM WILKINS, PH.D. Head of CMC and Pharmaceutical Development
Over 30 years of experience in CMC and biopharmaceutical development. Previously served as Chief Technology Officer at Sensorin and Director of Technology Assessment at Genentech.



SETH ZUCKERMAN, Senior Director, Biostatistics
Over 28 years of experience working in the pharmaceutical industry in clinical data and trial management with 23 years as statistician.

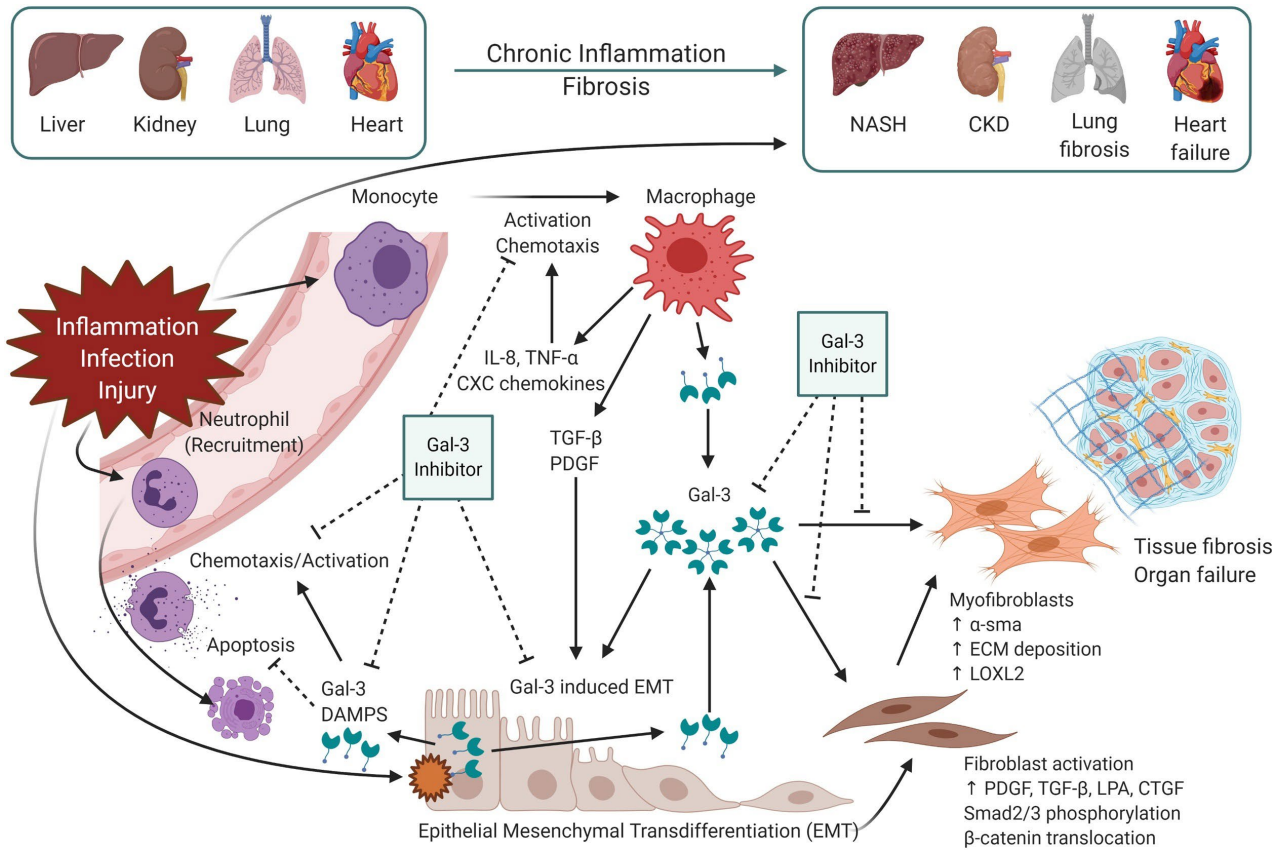
Laser-Focused Pipeline

Clinical Program		Development Stage					
Drug	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Recent & Upcoming Milestones
Fibrosis							
Belapectin	**MASH Cirrhosis with Portal Hypertension**						NAVIGATE trial completed; Phase 3 protocol submission in Q3 2026
Cancer Immunotherapy (Combination therapy)							
Belapectin + Keytruda	**Melanoma + Head & Neck Cancer**						
Oral Galectin-3 Inhibitors							
Discovery program oral small molecules to inhibit Galectin-3							



Galectin-3 Inhibition a Therapeutic Target in Chronic Liver Disease

- Galectin-3 is upregulated in liver fibrosis, integrating injury-driven inflammation and scar formation across all major chronic liver diseases
- Galectin-3 is increased in diseased liver, with levels tracking fibrosis severity, portal hypertension, and progression to cirrhosis
- Directly associated with scar formation, activating hepatic stellate cells and sustaining collagen production.
- Compelling disease-modifying target: Gal-3 inhibition consistently reduces fibrosis and enables regression in multiple preclinical models.



Slack, R. J., et al. *Int J Biochem Cell Biol.* 2021;130: 105881.

MASH Cirrhosis



Galectin Therapeutics

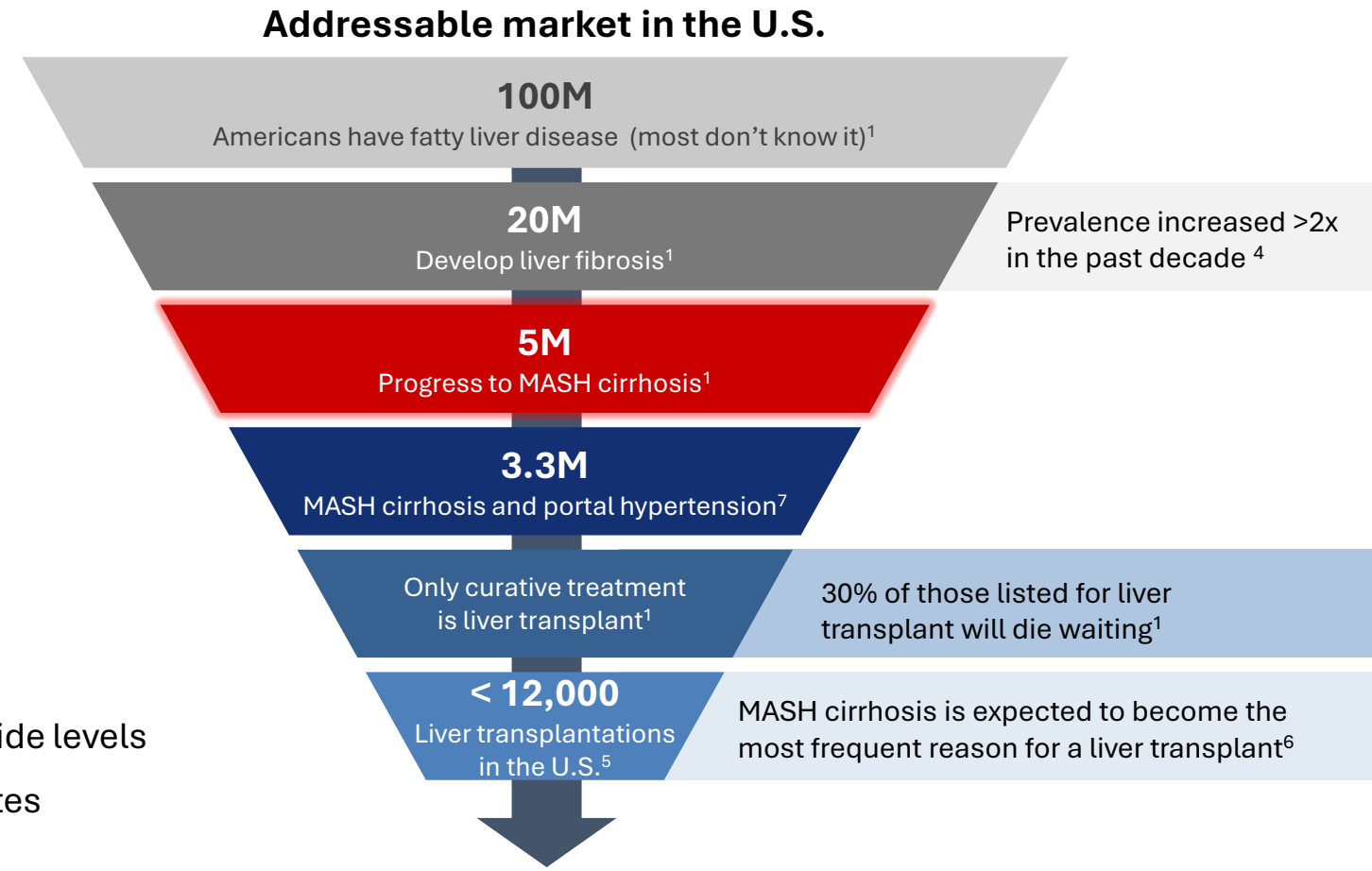
MASH Cirrhosis Represents a Significant Market Opportunity in the U.S. with No FDA-Approved Treatment

Metabolic dysfunction-associated steatohepatitis (MASH), previously known as non-alcoholic steatohepatitis (NASH), is characterized by fat accumulation, inflammation and fibrosis of the liver[1]

3%-5% of the global population is estimated to be affected by MASH, though the disease is considered to be underdiagnosed[2]

There are genetic predisposition to MASH, yet certain health conditions put patients at increased risk:[3]

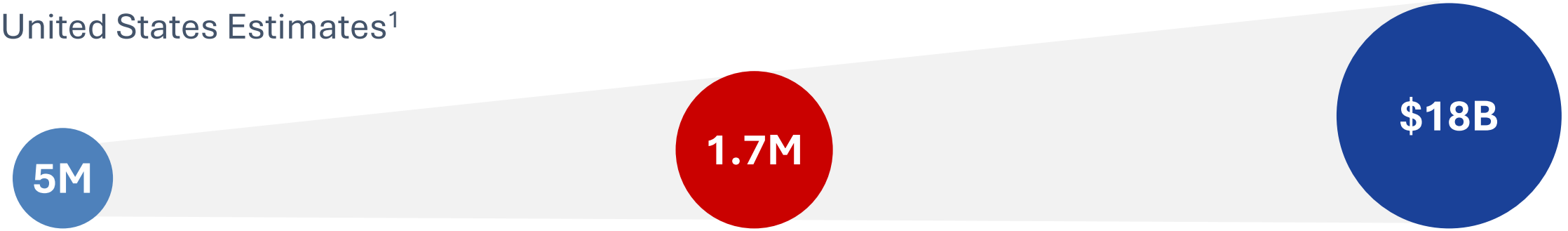
- Being overweight or obese
- Having hypertension, high cholesterol or high triglyceride levels
- Having type 2 diabetes, insulin resistance or prediabetes



1. Fatty Liver Foundation. https://www.fattyliverfoundation.org/#gsc.tab=0. .2. Sherif ZA, et al. *Dig Dis Sci*. 2016;61(5):1214-25. 3. NIDDK. NAFLD and MASH. https://www.niddk.nih.gov/health-information/liver-disease/nafld-nash/symptoms-causes. 4. Datamonitor Healthcare. MASH Disease Analysis. 5. Scientific Registry of Transplant Recipients. OPTN/SRTR 2024 Annual Data Report: Liver. https://srtr.transplant.hrsa.gov/annual_reports/2024/Liver.aspx. 6. Stepanova M, et al. *Hepatol Commun*. 2022;6(7):1506-1515. 7. Zobair M. Younossi, et al, Prevalence and predictors of cirrhosis and portal hypertension in the United States, American Association for the Study of Liver Disease, DOI: 10.1097/HEP.0000000000001243.

Belapectin's Novel Mechanism Positions It to Address MASH Cirrhosis & Portal Hypertension

United States Estimates[1]



Patients with compensated MASH cirrhosis in 2024

Patients with compensated cirrhosis and portal hypertension with no varices in 2024

Peak belapectin sales in the U.S.

3rd Party Market Opportunity Assessment Suggests[1]

Potential 35-100% Adoption Rate

Limited current treatment options:

- Cirrhotic management focuses on stabilization and delaying progression
- Management directed towards comorbidities

Highly favorable perception of belapectin indication, MoA and safety by HCPs

Payers believe in the high unmet need in MASH cirrhosis

A significant unmet need exists for compensated MASH cirrhosis patients with portal hypertension due to disease severity and risk of decompensation

1. LifeSci Consulting Belapectin Commercial Opportunity Assessment contracted by the Company.

When to Intervene in Cirrhosis

Compensated cirrhosis | **Decompensated cirrhosis**

No Portal Hypertension | **Portal Hypertension**

No varices

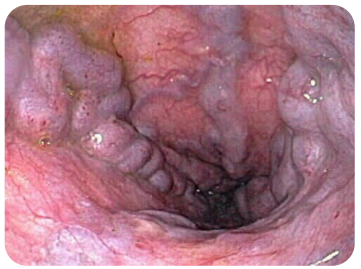


≥6

No varices

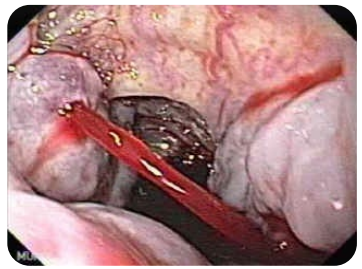


Varices,
small to large



≥10

Varices Bleeding, ascites, encephalopathy

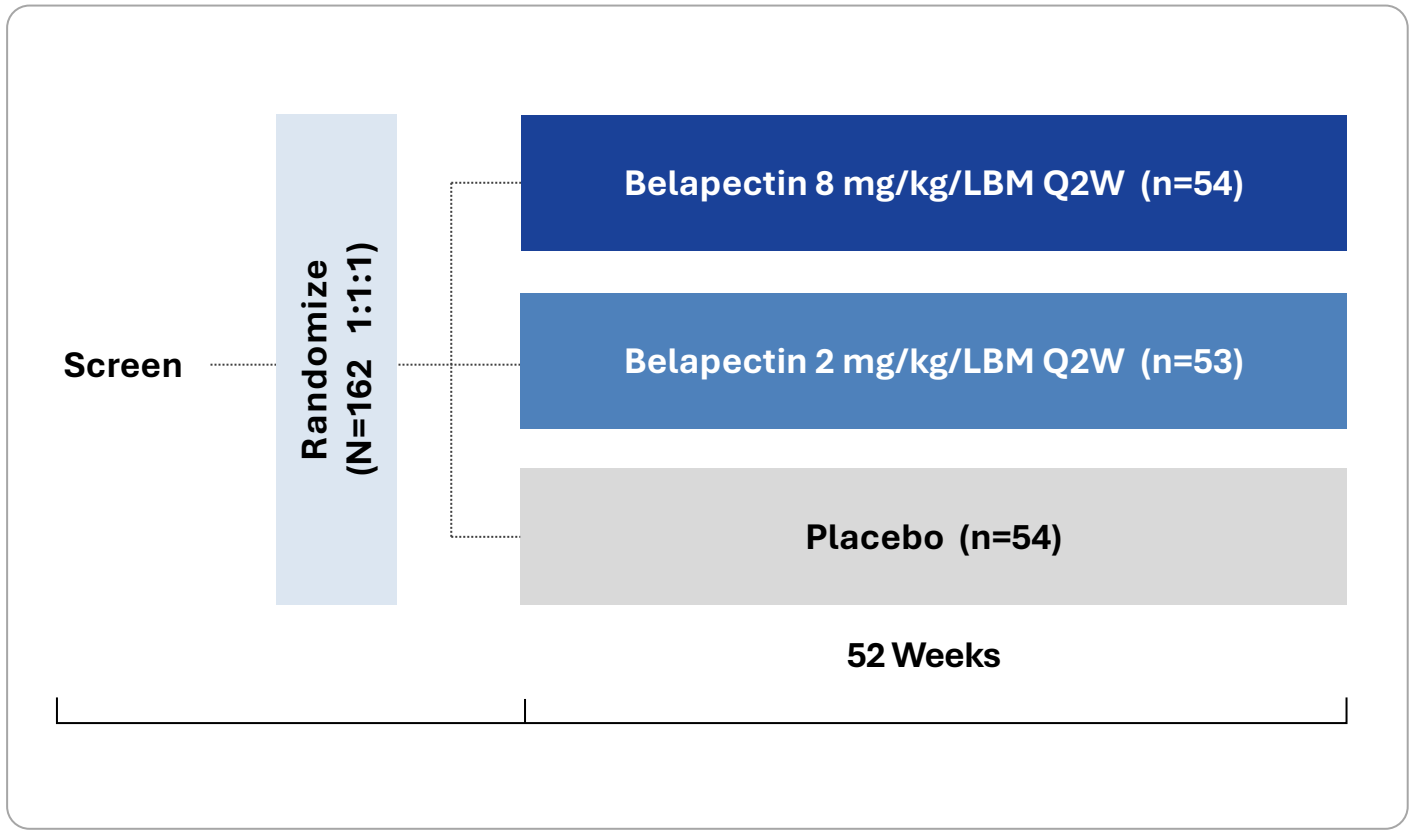


One year mortality 1-3%

One year mortality ~50%

There are no approved therapies to reverse portal hypertension once it develops in MASH Cirrhosis

Phase 2b Study of Belapectin in Patients with MASH Cirrhosis: GT-026 Trial



Main inclusion criteria

- MASH cirrhosis (biopsy)
- Portal Hypertension: HVPG ≥ 6 mmHg
- No cirrhosis complications
- No varices/varices (50:50)

Primary endpoint

- Portal pressure (HVPG) change from baseline to Week 54

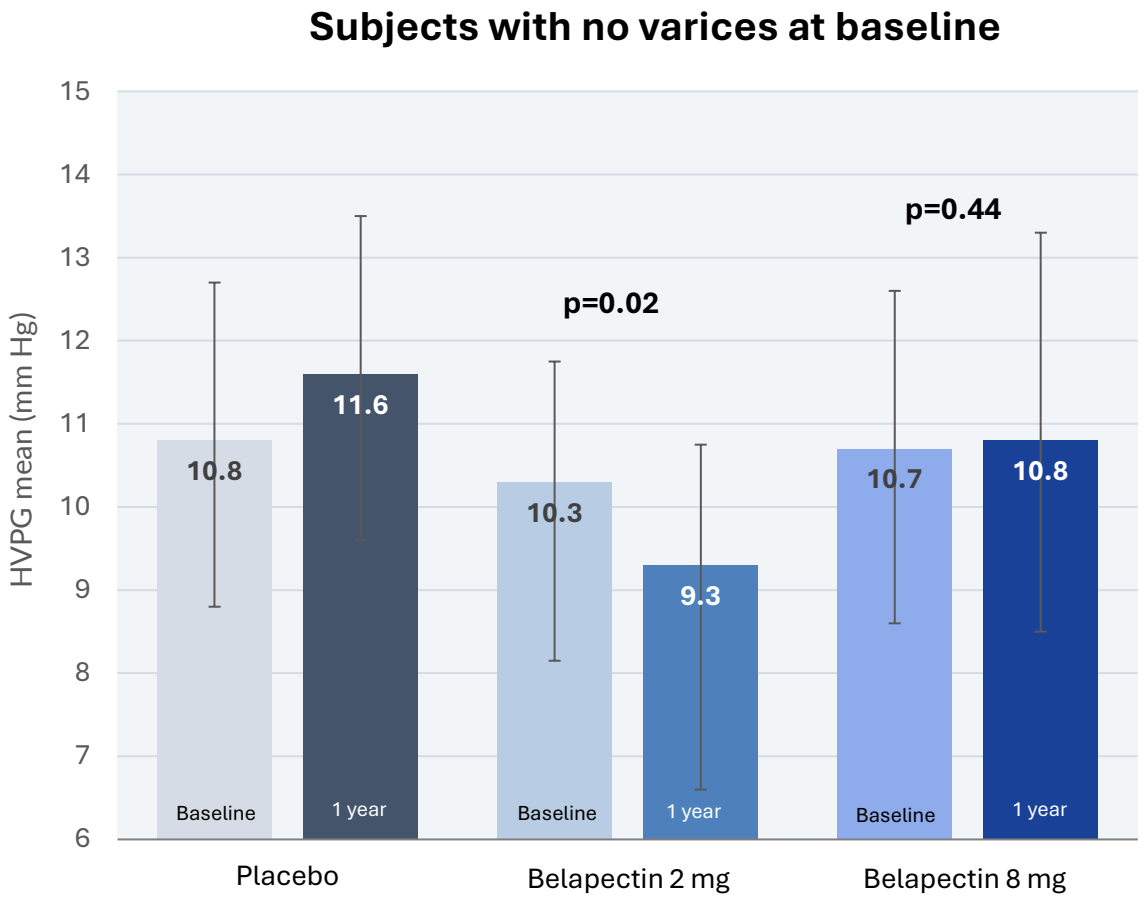
Secondary endpoints at Week 54

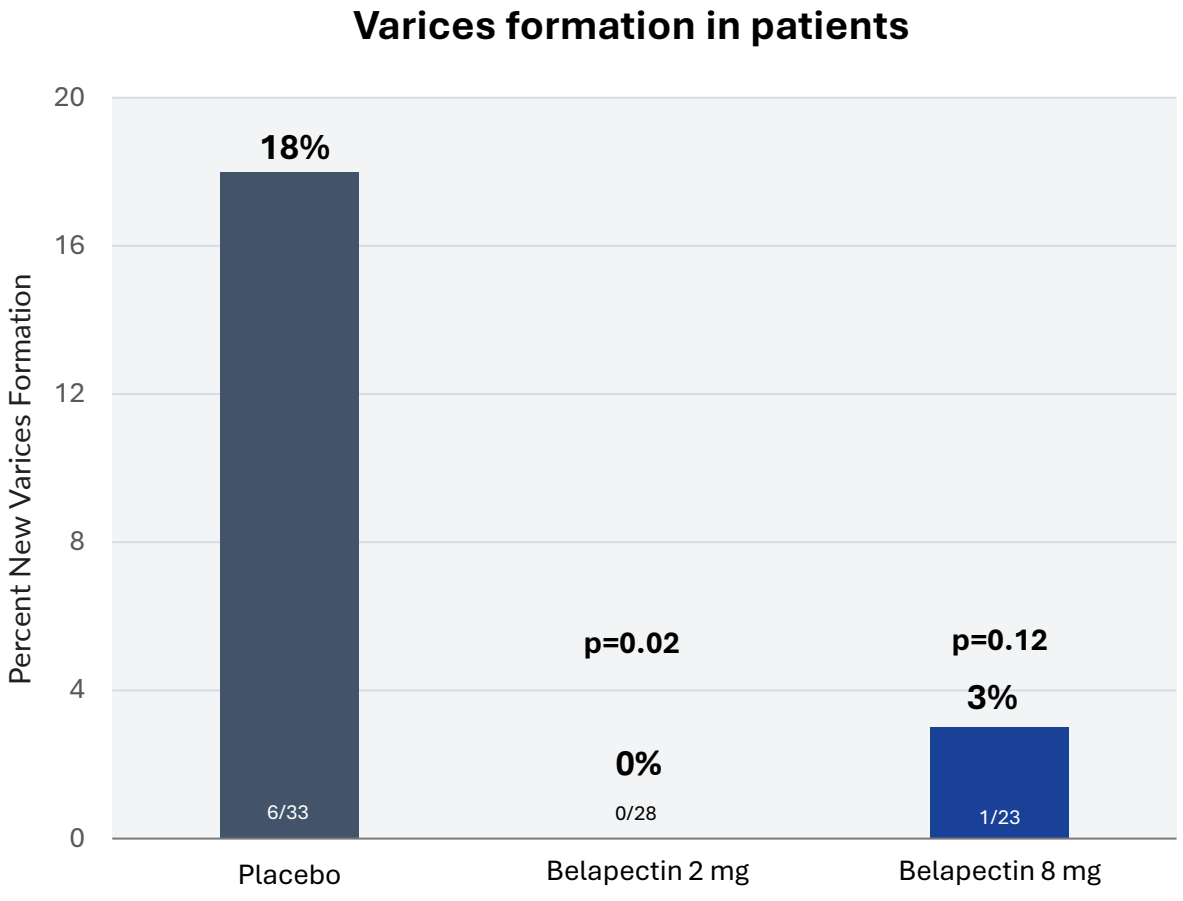
- Liver biopsy
- Varices (esophago-gastric endoscopy)
- Cirrhosis decompensation

HVPG = Hepatic Venous Pressure Gradient; LBM=lean body mass.

1. Chalasani N, et al. *Gastroentrol.* 2020;158:1334-45.

Belapectin Impact on HVPG and Varices Formation at One Year[1,*]





Belapectin demonstrated efficacy on a clinically-meaningful endpoint where no current therapies exist

HVPG = Hepatic Venous Pressure Gradient; LBM=lean body mass, N.S.=non significant. *ITT with LOCF, ANCOVA with baseline as covariate and treatment as factors, Bonferroni-Holm.

1. Chalasani N, et al. *Gastroentrol.* 2020;158:1334-45.

NAVIGATE Trial Design

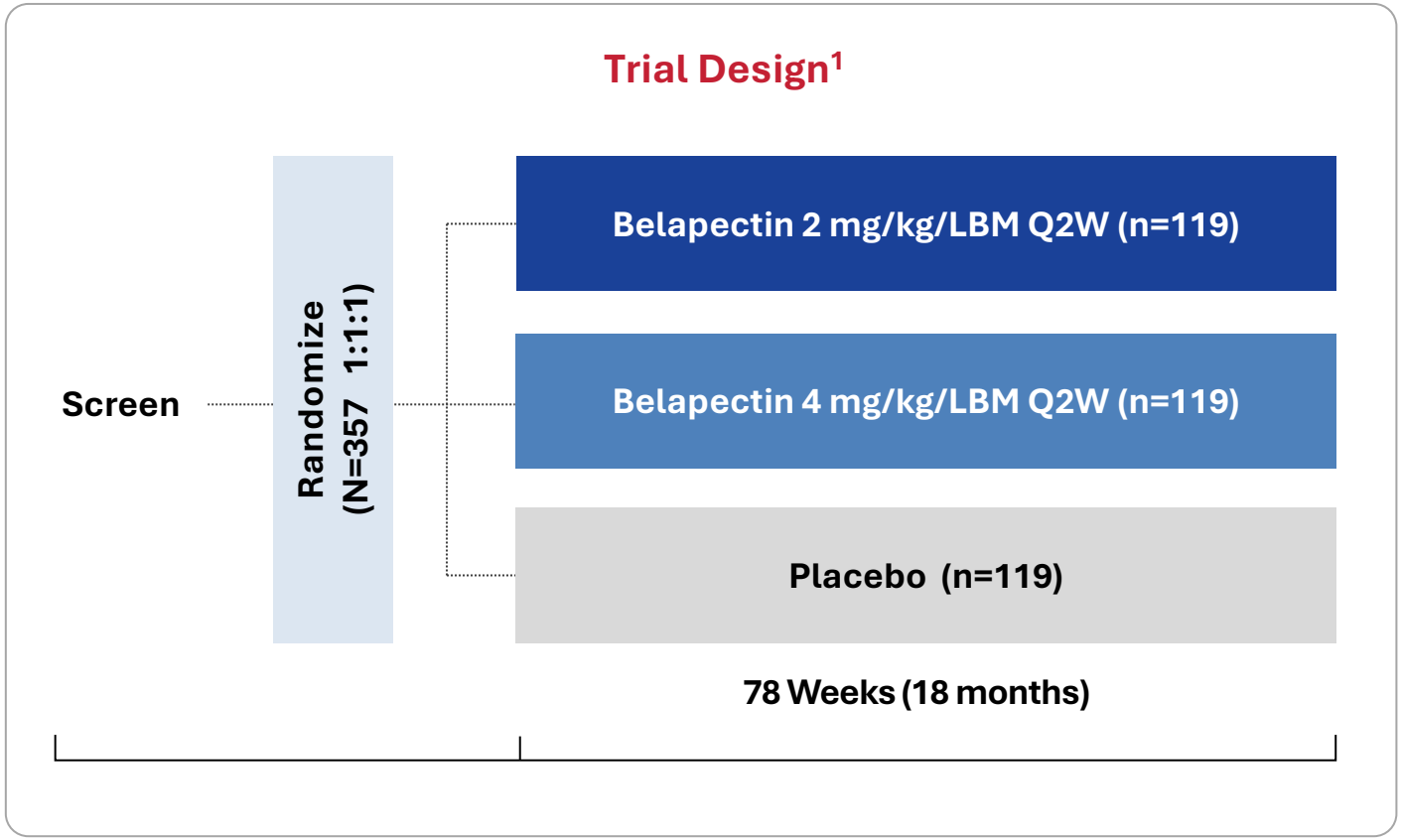
Patient Population

MASH cirrhosis based on Liver Forum recommended criteria for clinical trials

Diagnosis of Portal Hypertension as per Baveno VI criteria (via non-invasive markers)

No gastroesophageal varices by endoscopy at baseline

Assessment of varices through central adjudication of endoscopy videos by multiple blinded reviewers based on standardized protocol[2]



1. Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774. 2. Alkhouri N, et al. *Gastro Hep Adv*. 2026;5(8):101004.

NAVIGATE Study: Patient Population and Efficacy Endpoints

Key inclusion criteria



MASH cirrhosis

No varices on EGD

CTP Scores <7

Evidence of Portal Hypertension:

Platelet count <150,000/mm^3 or at least two of the following

I. AST/ALT > 1
II. Spleen ≥ 14 cm
III. Collaterals by imaging
IV. Stiffness ≥ 20 kPa

Primary endpoint



Composite primary end point in ITT population

Incidence of varices in per protocol population

Composite secondary endpoint



Hepatic decompensation events

All-cause mortality

Proportion of patients with large varices or red wale sign

Varices requiring treatment

MELD ≥ 15

Liver transplant

ALT = alanine aminotransferase ; AST = aspartate transaminase; CTP = Child-Turcotte-Pugh; EGD = Esophagogastroduodenoscopy; ITT=intent to treat; MELD = model for end-stage liver disease. Chalasani N, et al. *Hepatology.* 2026; DOI: 10.1097/HEP.0000000000001774.

Key Populations for Assessment of Varices Outcome

ITT All randomized subjects minus two subjects who had varices at baseline;

Per Protocol All subjects who completed 18 months of therapy and had an EGD at baseline and 18 months

- Subjects were encouraged to complete the study even after development of varices unless subject dropped out for other reasons

Composite Primary Endpoint Any subject who developed esophageal varices or had an intercurrent event or dropouts without an EGD/intercurrent event

- Intercurrent events included;
  - Liver related clinical events
  - AE leading to discontinuation
  - TIPS-Trans-jugular intrahepatic portosystemic shunt
  - ≥12-month use of GLP-1 or NSBB

EGD = Esophagogastroduodenoscopy; ITT=intent to treat.

Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774

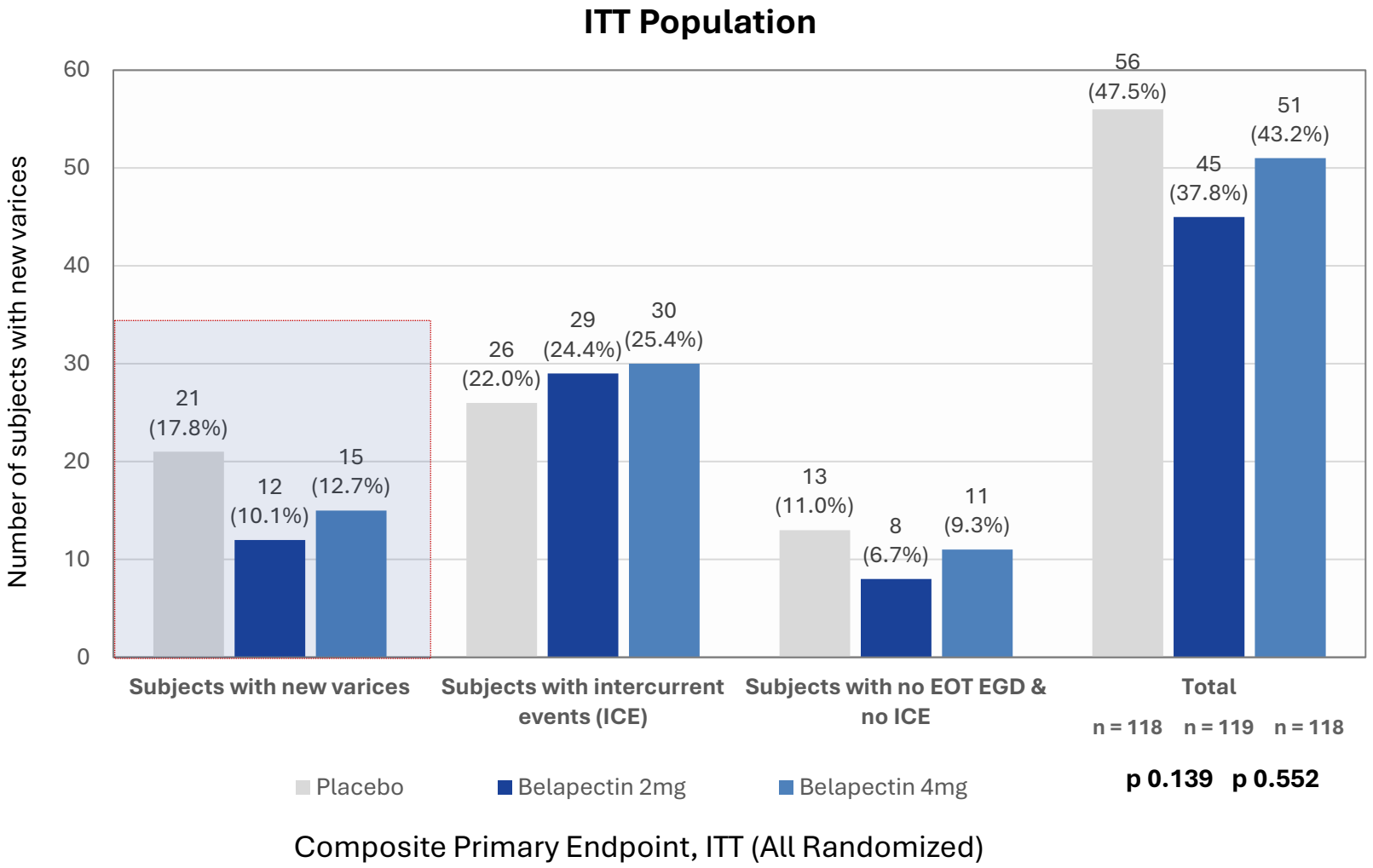
NAVIGATE Trial: Baseline Demographics

Baseline Demographics and Clinical Characteristics (N=355*)

	Placebo (n = 118)	Belapectin 2 mg (n = 119)	Belapectin 4 mg (n = 118)
	Mean (Standard Deviation)	Mean (Standard Deviation)	Mean (Standard Deviation)
Age (years)	60.4 (8.50)	60.6 (8.82)	59.0 (9.14)
Gender (female), n (%)	72 (61.0)	75 (63.0)	83 (70.3)
Ethnicity (Hispanic), n (%)	34 (28.8)	39 (32.8)	33 (28.0)
Race (white), n (%)	104 (88.1)	107 (89.9)	111 (94.1)
Weight (kg)	94.2 (21.68)	98.1 (24.30)	94.6 (20.95)
BMI (Kg/m^2)	33.82 (6.46)	34.88 (6.68)	34.53 (6.22)
Hypertension	89 (75.4)	89 (74.8)	82 (69.5)
Type 2 Diabetes	80 (67.8)	79 (66.4)	79 (66.9)
HbA1C %	6.4 (1.27)	6.3 (1.13)	6.4 (1.09)
Alanine Aminotransferase (ALT), U/L	46.3 (29.92)	38.9 (26.88)	39.7 (20.22)
Aspartate Aminotransferase (AST), U/L	46.7 (23.52)	41.8 (24.40)	43.6 (21.90)
Platelets (per µL)	130.1 (39.66)	127.6 (48.39)	136.4 (53.62)
Liver Stiffness Measurement (kPa)	24.22 (12.17)	24.63 (13.54)	25.67 (13.19)
Spleen (cm)	13.79 (2.7)	13.97 (2.6)	13.87 (2.4)
MELD Score	7.6 (1.65)	7.9 (2.46)	7.5 (1.55)
Child Pugh Score	5.1 (0.29)	5.1 (0.31)	5.0 (0.18)
Statins (n)	49 (41.5)	55 (46.2)	47 (39.8)
GLP-1 agonist (n)	24 (20.3)	26 (21.8)	27 (22.9)

Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774. * 2 patients with varices at baseline were excluded from database

NAVIGATE 18-Month Primary Analyses Result – ITT Population



Key points

Intent to Treat (ITT) -All randomized subjects

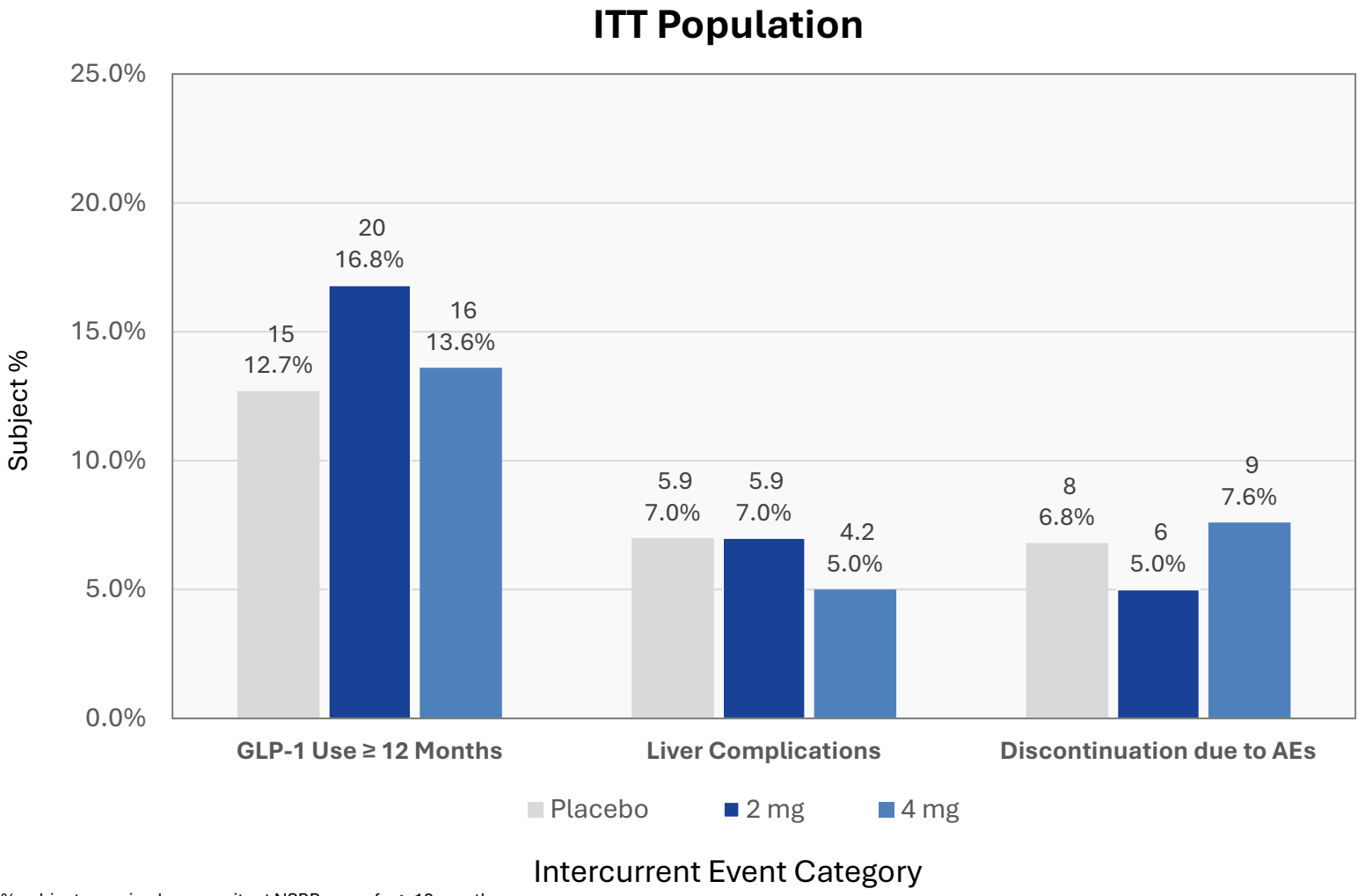
Primary endpoint composite strategy i.e. new varices and/or intercurrent events or drop out

Intercurrent events (ICEs) include; Liver related clinical events, AE leading to discontinuation, TIPS; ≥12-month use of GLP-1 or NSBB

Overall Target Significance level– 2-sided p value of 0.05; using CMH test, stratified by Type 2 diabetes status at randomization.

Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774.

NAVIGATE: Intercurrent Events Breakdown by Category



Key points

No subject met intercurrent event category for Trans-jugular intrahepatic portosystemic shunt(TIPS) or ≥12-month use of non-selective beta-blocker NSBB

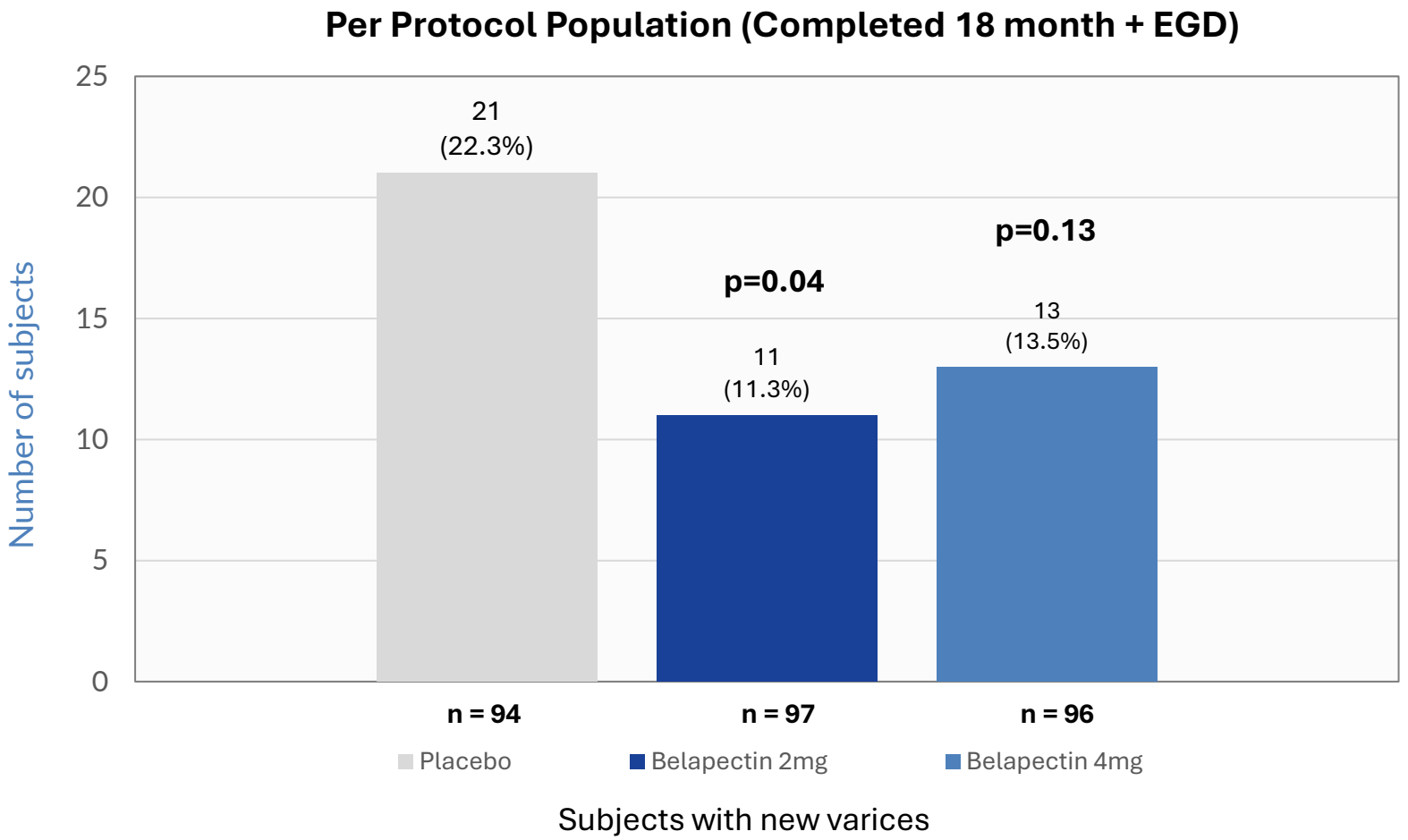
Placebo n = 118

2 mg n = 119

4 mg n = 118

6.3% subjects received concomitant NSBB, none for ≥ 12 months.

NAVIGATE: Significantly Lower Incidence of Varices in Completers at 18 Months



Key points

NAVIGATE 18-month Primary Analyses Result; Per protocol population n=287

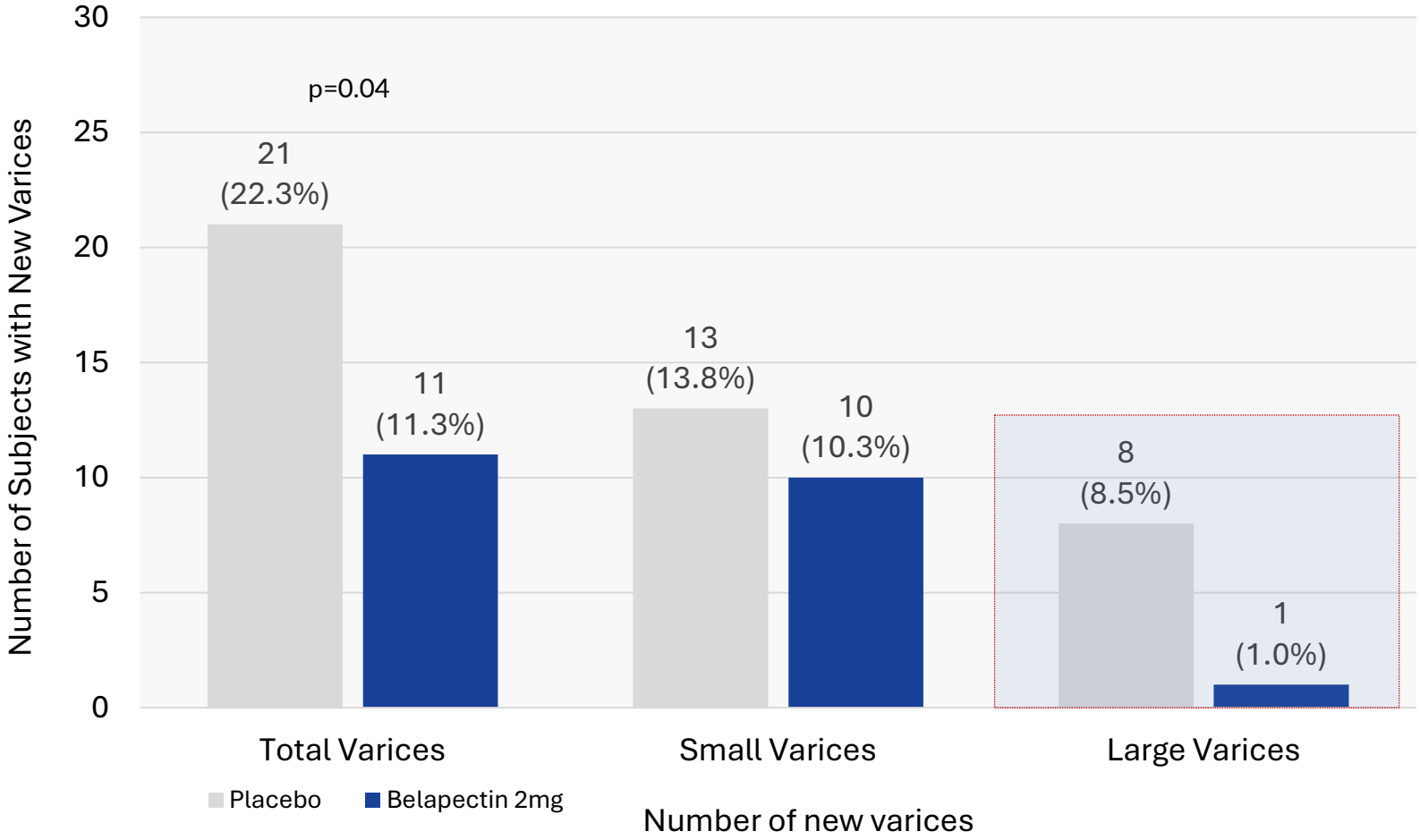
Completer/Per Protocol: All ITT subjects who completed 18 months of treatment with an end of treatment (EOT) EDG

Overall Target Significance level – 2-sided p value of 0.05; using CMH test, stratified by Type 2 diabetes status at randomization.

Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774.

NAVIGATE: Incidence of New Varices by Size at 18 months

Per Protocol Population



Key points

Placebo: n = 94

Belapectin 2 mg: n = 97

Varices grade definition

- Large > 5 mm in diameter
- Small < 5 mm in diameter

Lowest Rates of Liver-Related Events and MACE Observed with the 2 mg Dose

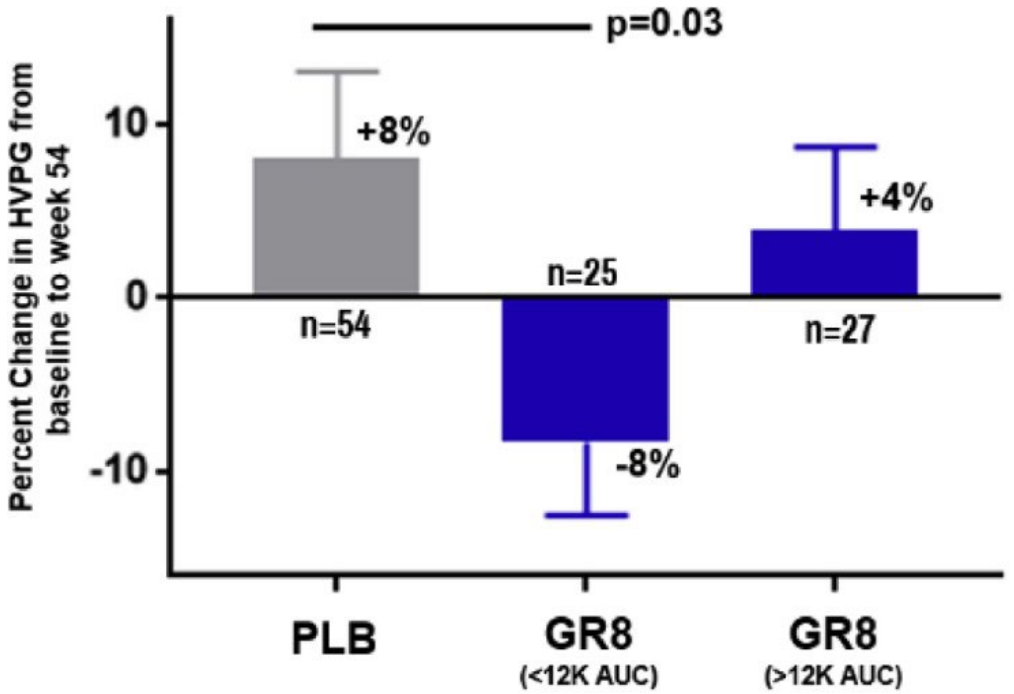
Per Protocol

		Belapectin	
	Placebo (n = 94) n (%)	2 mg (n = 97) n (%)	4 mg (n = 96) n (%)
Subjects with Composite Clinical Outcomes, n (%)	**5 (5.3)**	**3 (3.1)**	**9 (9.4)**
Varices (Esophageal or Gastric) Requiring Treatment	3 (3.2)	3 (3.1)	4 (4.2)
Variceal Bleed Requiring Hospitalization	0	0	0
Clinically Significant Ascites Requiring Hospitalization	0	0	1 (1.0)
Spontaneous Bacterial Peritonitis	0	0	1(1.0)
Overt Hepatic Encephalopathy (West Haven Score ≥2 and Requiring Hospitalization)	1(1.1)	0	1 (1.0)
Liver Transplant	0	0	0
Model End Stage Liver Disease (MELD) Score ≥ 15	1(1.1)	0	2 (2.1)
MI or Hospitalization for Unstable Angina	0	0	1 (1.0)
Stroke or Transient Ischemic Attack	1 (1.1)	0	1 (1.0)

MACE- major adverse cardiovascular events; subjects with multiple events counted once.

Lack of Dose Response at Higher Doses of Belapectin in GT-026 were Also Observed in NAVIGATE Trial

- Based on findings from preclinical and clinical trials to date, Belapectin likely demonstrates target-mediated drug disposition (TMDD)
- Once Galectin-3 binding sites within macrophages are saturated, additional drug molecules do not enhance efficacy
- Higher doses may exceed the macrophage-specific uptake mechanisms, resulting in altered drug distribution and clearance
- Higher drug concentrations have been associated with reduced efficacy, as observed in the GT-026 cohort, where subjects receiving 8 mg/kg (with higher AUC) exhibited lower PD effects.
- Similar PK profile shown by monoclonal antibodies and interferon among other agents.
- 2 mg/kg dose demonstrated consistent and most optimum efficacy response
- Similar PK-PD effects were observed across the GT-026 trial and the NAVIGATE 18-month results



PD=pharmacodynamic; PK=pharmacokinetic.

Safety Summary

Adverse Events

Discontinuation of the study due to Adverse Events was similar across 3 cohorts:

- 7 (5.9%) in the Placebo
- 5 (4.2%) in 2 mg Belapectin
- 8 (6.7%) in 4 mg Belapectin
- One subject in each of the three cohorts discontinued the study due to death

- No drug related SAE reported in the entire trial
- No Adjudicated Drug-Induced Liver Injury (DILI) Events.

Treatment-Emergent Adverse Events (TEAEs)

Similar proportion of subjects reported **Treatment-Emergent Adverse Events TEAEs** across 3 cohorts:

- 112 (94.9%) in Placebo
- 116 (97.5%) in 2 mg Belapectin
- 116 (96.7%) in 4 mg Belapectin

Treatment-Emergent Serious Adverse Events (TESAEs)

Similar proportion of subjects reported **Treatment-Emergent Serious Adverse Events** across 3 cohorts:

- 23 (19.5%) in Placebo
- 27 (22.7%) in 2 mg Belapectin
- 25 (20.8%) in 4 mg Belapectin

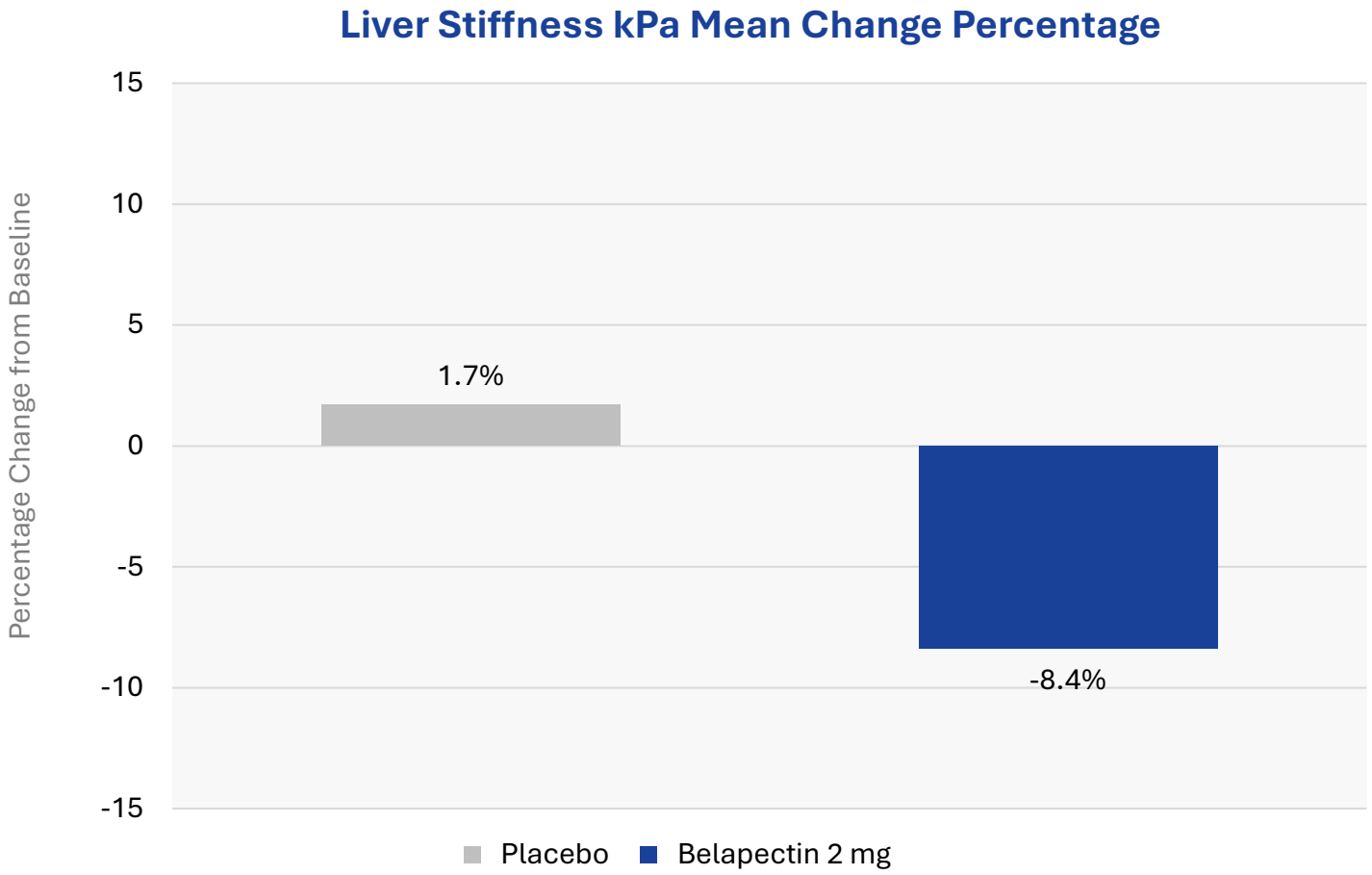
Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774.

NAVIGATE
Key Biomarker Results

Galectin Therapeutics

NAVIGATE: Belapectin Treatment Associated with Decreased Liver Stiffness

Per Protocol

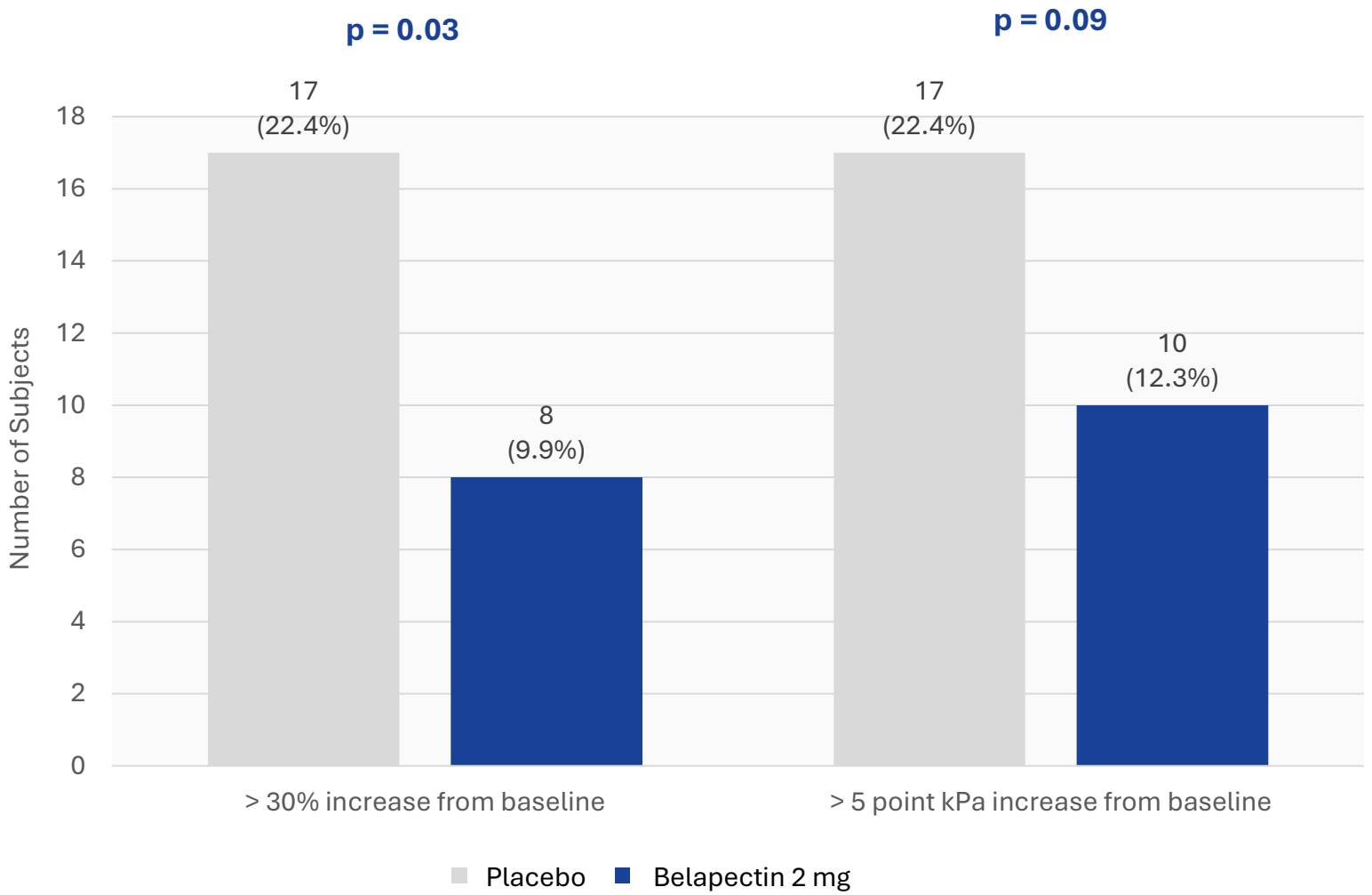


	Baseline	
	Placebo (n = 76)	**2 mg** (n = 81)
Mean	22.6	24.6
Standard Deviation	10.31	13.97
Median	22.4	21.8

Values based on Per Protocol Set for Subjects with all Phase 2b assessments

Significantly Less Progression of Liver Stiffness on Belapectin

Per Protocol

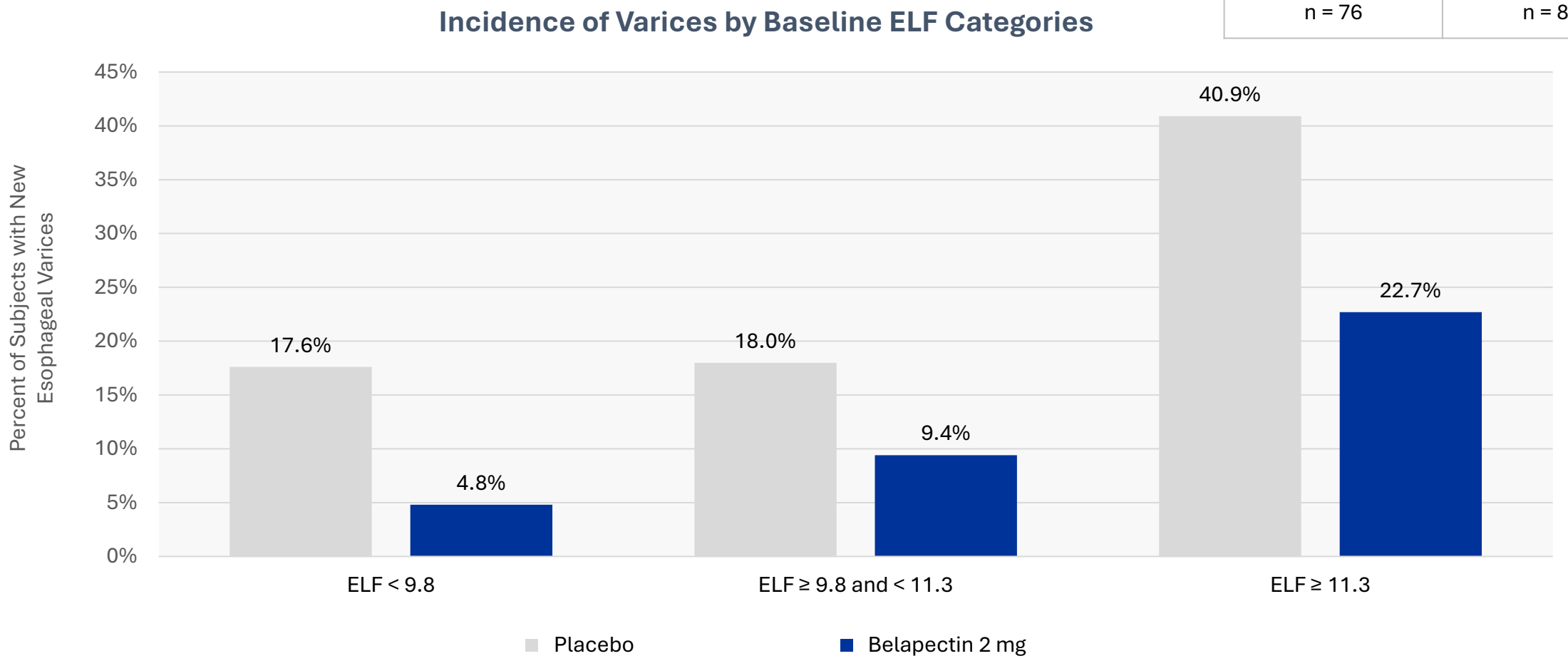


Placebo	2 mg
n = 76	n = 81

ELF Score Stratification Reveals Greater Variceal Benefit of Belapectin in Highest-Risk Patients

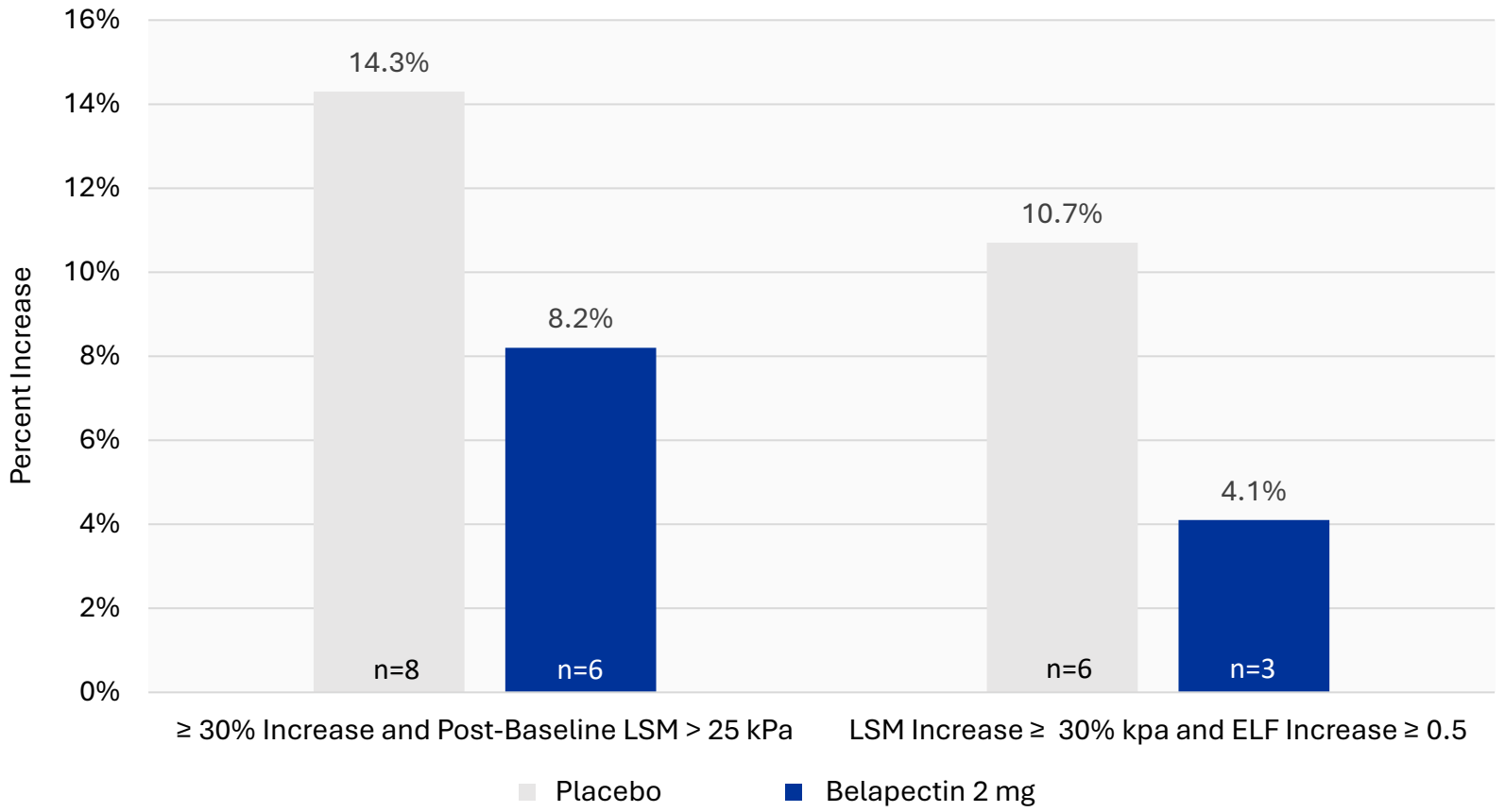
Per Protocol

Placebo	2 mg
n = 76	n = 81



Concordant Fibrosis Biomarkers Support Belapectin's Effect on Stopping Disease Progression

Per Protocol

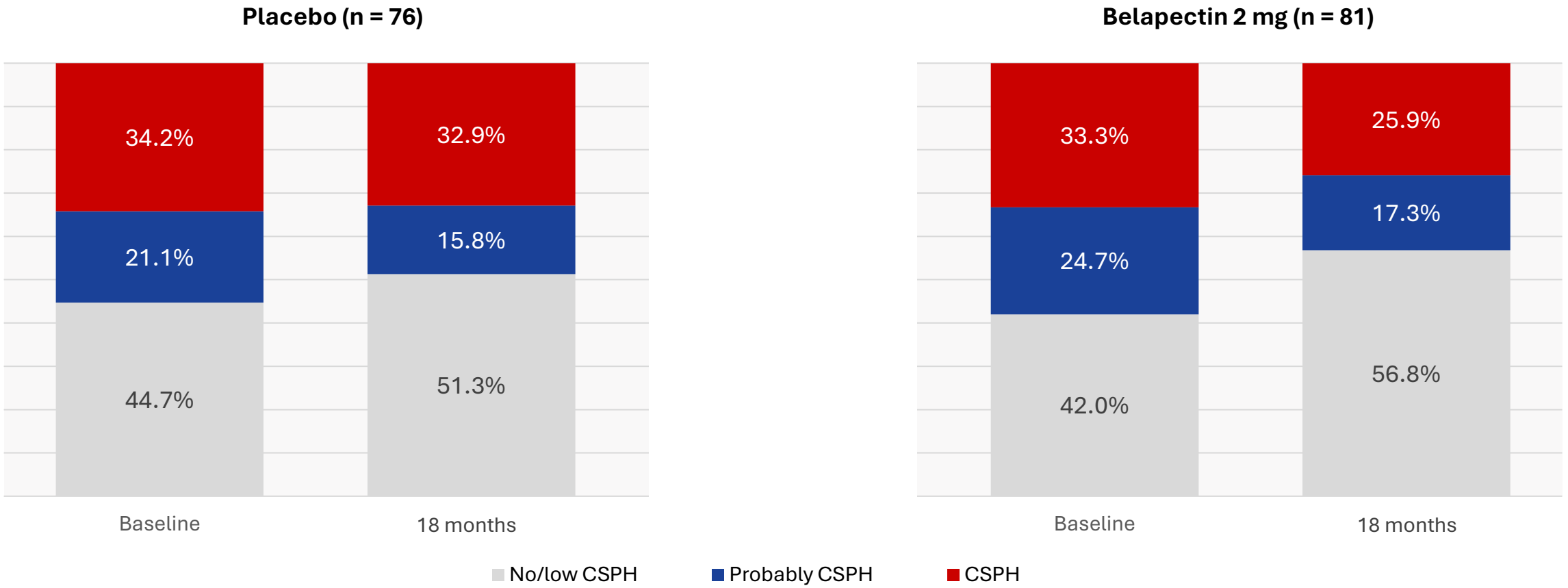


Placebo	2 mg
n = 56	n =73

Values based on Per Protocol Set, Subjects with all scheduled Phase 2b and ELF assessments

Belapectin Drives Meaningful Improvement in Portal Hypertension Risk Category

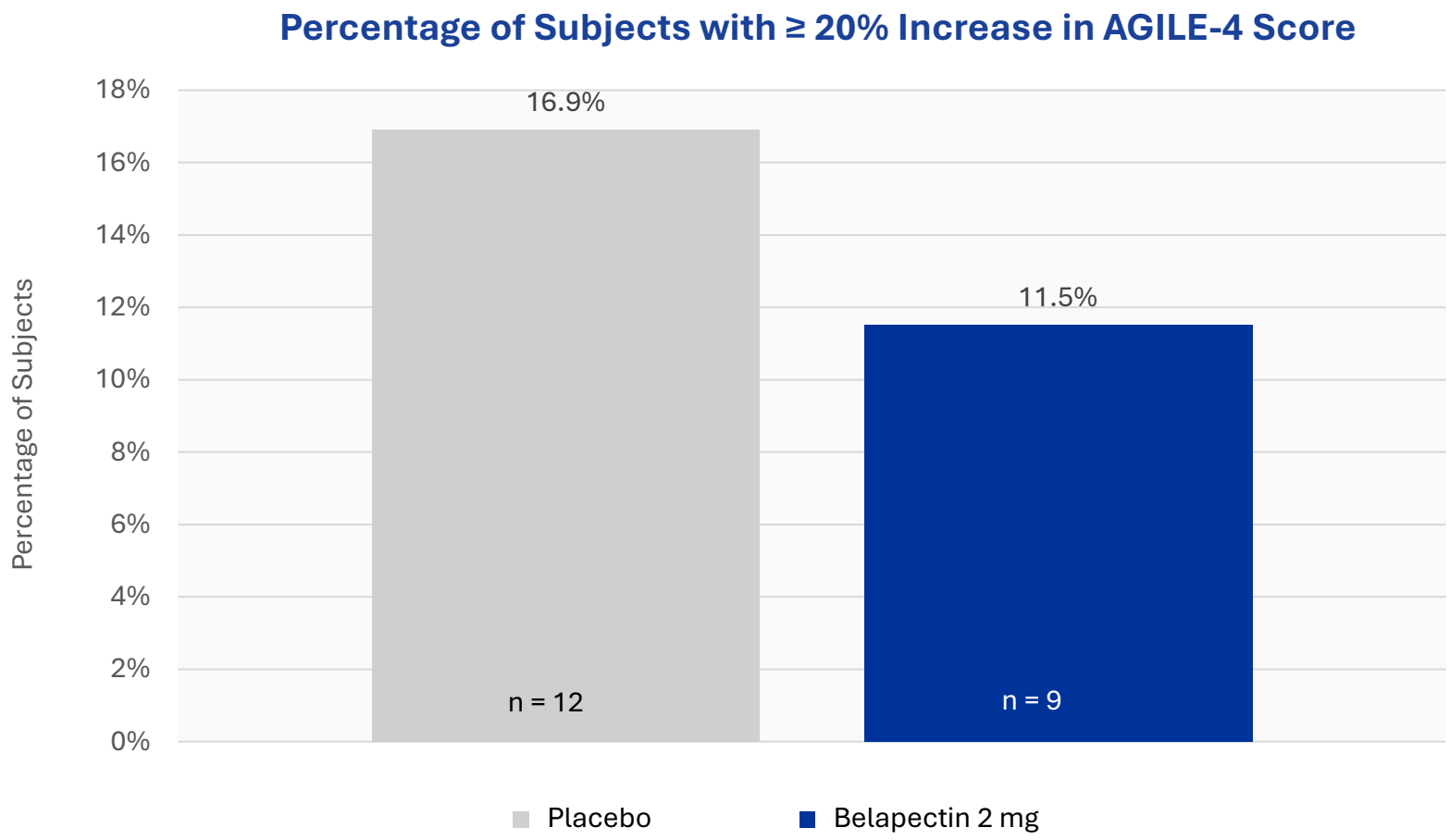
Per Protocol



CSPH LSM ≥ 25 kPa; Probable CSPH LSM ≥20 & platelet <150 or LSM 15-20 & platelet <110; no/low CSPH LSM≤ 15 and platelet≥ 150;
Baveno VII Guidelines de Franchis 2022 Jhep
Values based on Per Protocol Set, Subjects with all scheduled Phase 2b and ELF assessments

Belapectin Reduces Worsening in AGILE-4 Score Across Treated Patients

Per Protocol



Baseline

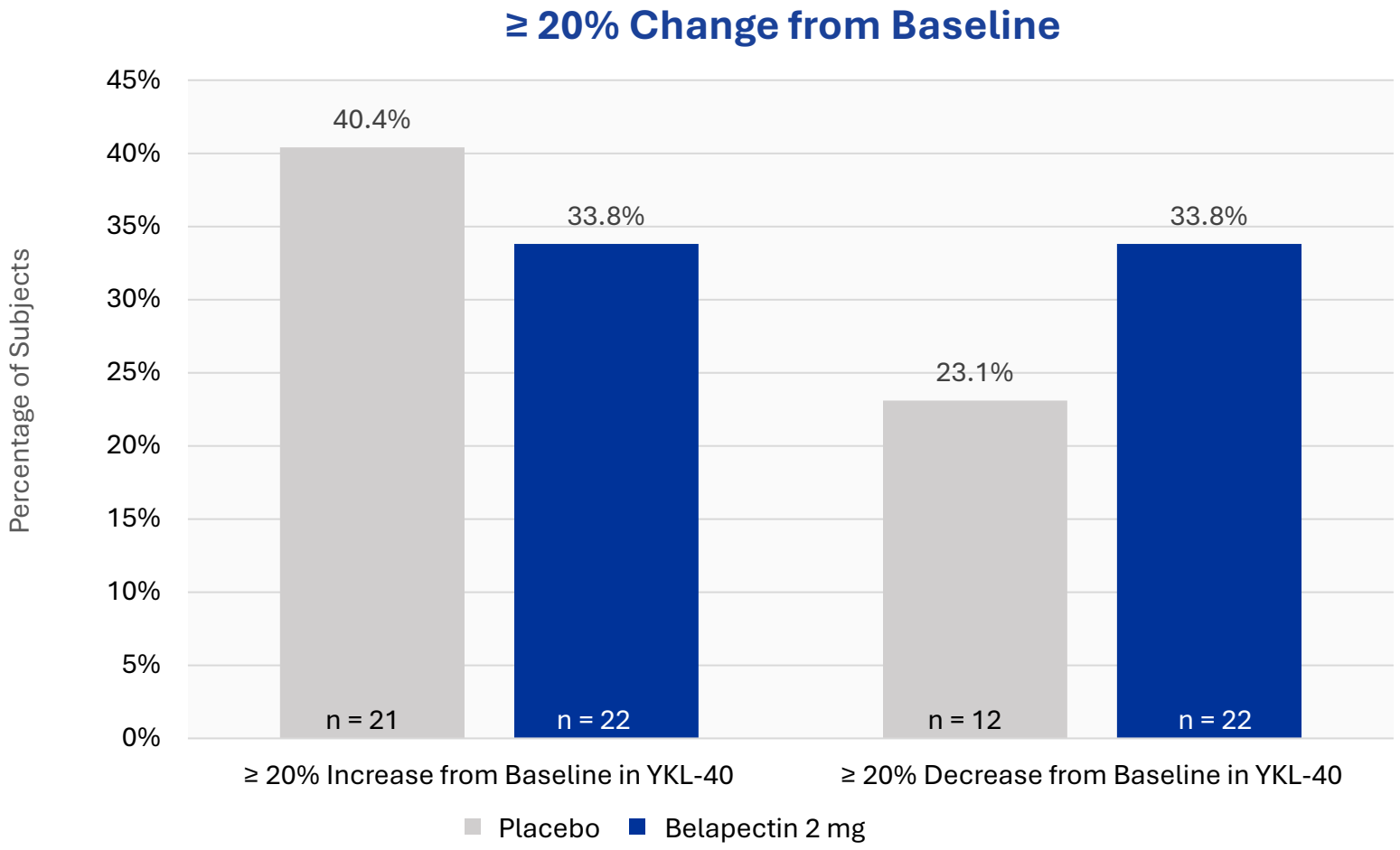
	Placebo n = 71	2 mg n = 78
Mean	0.66	0.68
Standard Deviation	0.24	0.23

AGILE-4 incorporates LSM, Age, Sex, AST/ALT, Platelet, Diabetes status
Data values for populations where subjects had all 4 Phase 2b assessments.

Belapectin Treatment Associated with Meaningful Changes in YKL-40

YKL-40 is a circulating marker of inflammation, also part of NIS-4

Per Protocol



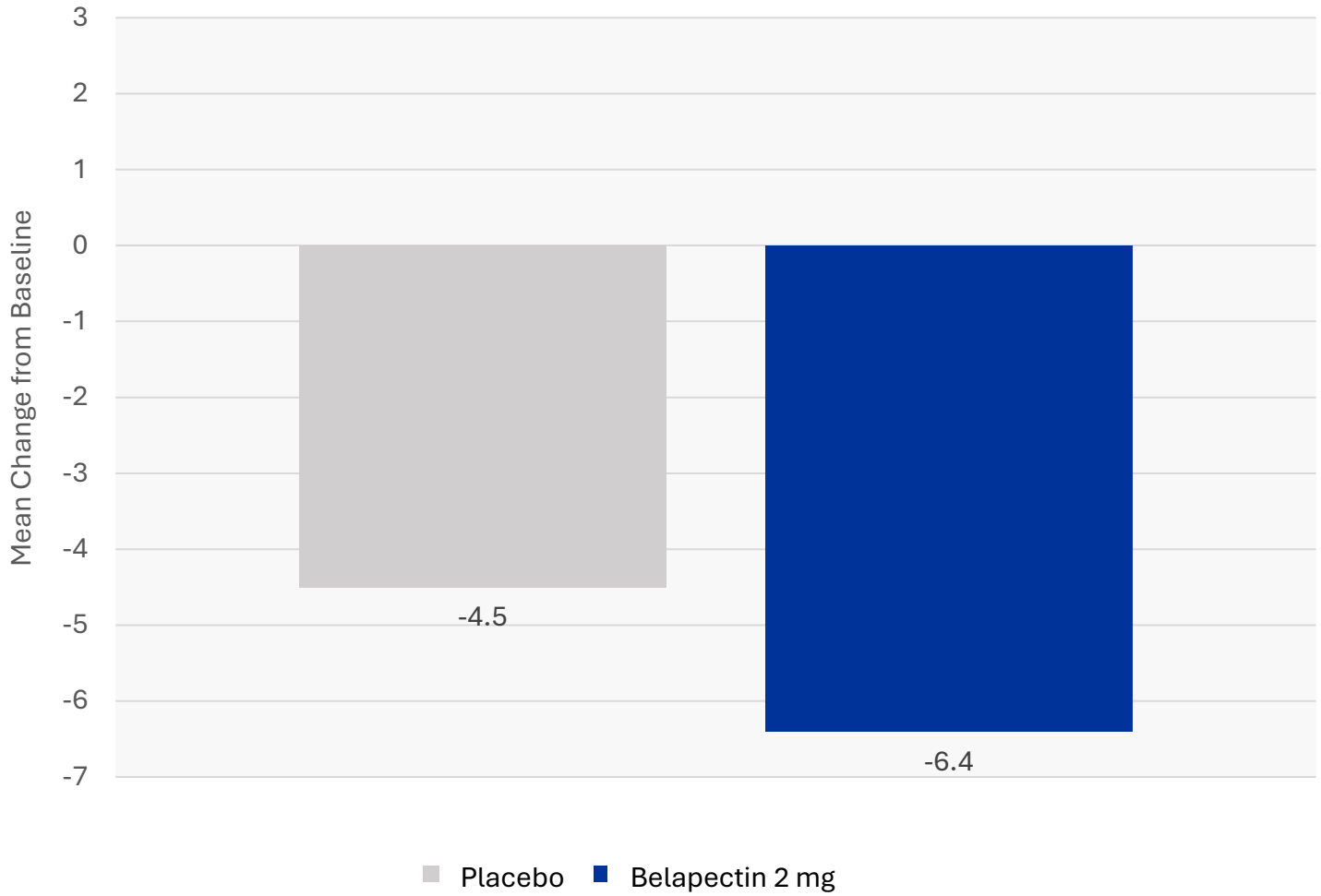
	Baseline	
	Placebo (n = 52)	**2 mg** (n = 65)
Mean	5.11	4.95
Standard Deviation	0.945	0.837

Chart values for subjects with all Phase2b YKL-40 assessments; table presents log transformed values

Trend Toward Reduced Pro-C3 with Belapectin

Per Protocol

Pro-C3 is a marker fibrosis or scar tissue build up



Baseline

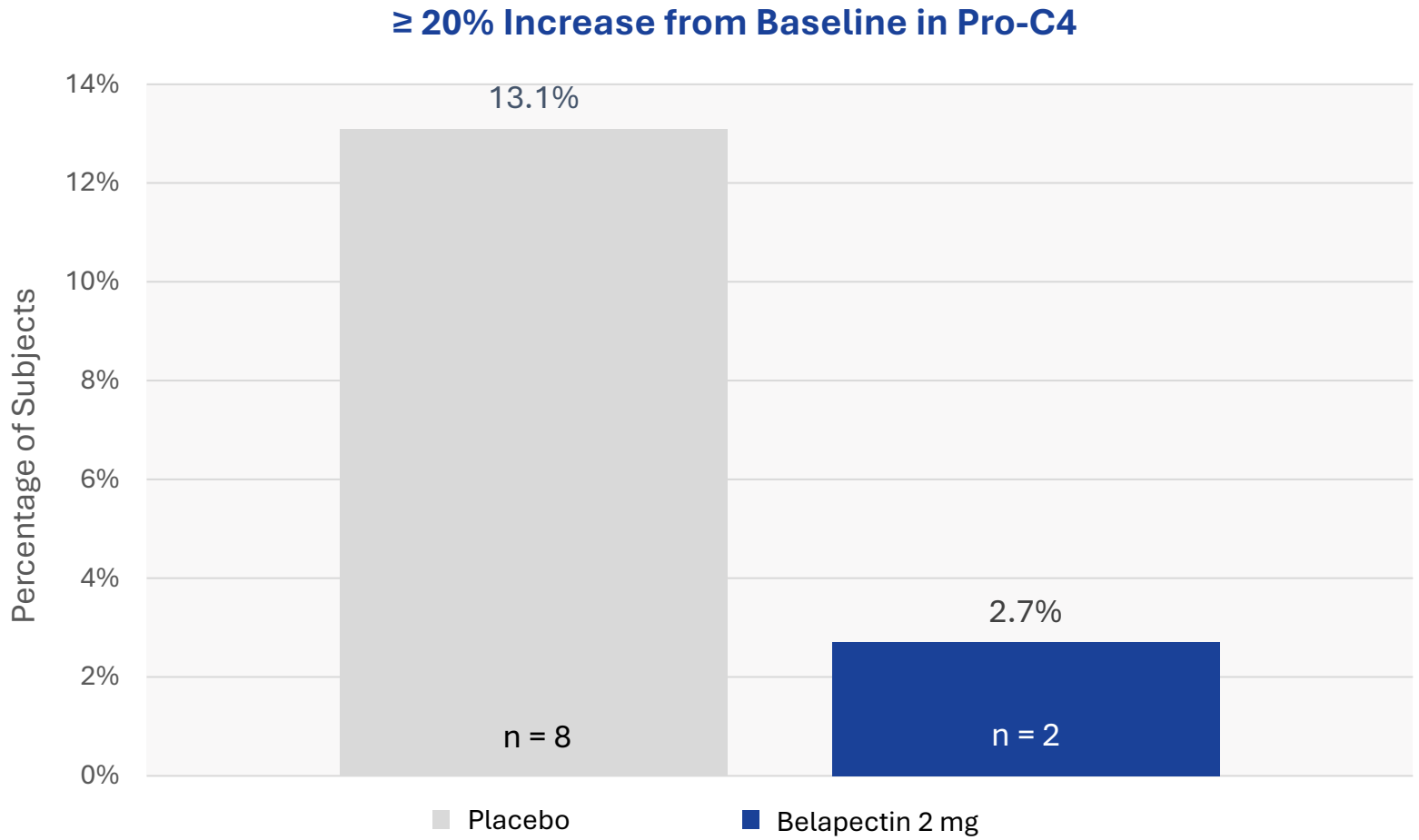
	Placebo n = 79	**2 mg** n = 81
Mean	50.19	45.91
Standard Deviation	38.45	31.58

Chart values for subjects with all Phase2b YKL-40 assessments; ; table presents raw values; Pro-C3 unit ng/mL

Lower Rate of Pro-C4 Worsening with Belapectin Treatment

Per Protocol

Pro-C4 is a marker of type-IV collagen build-up indicates increase in fibrosis and disease progression



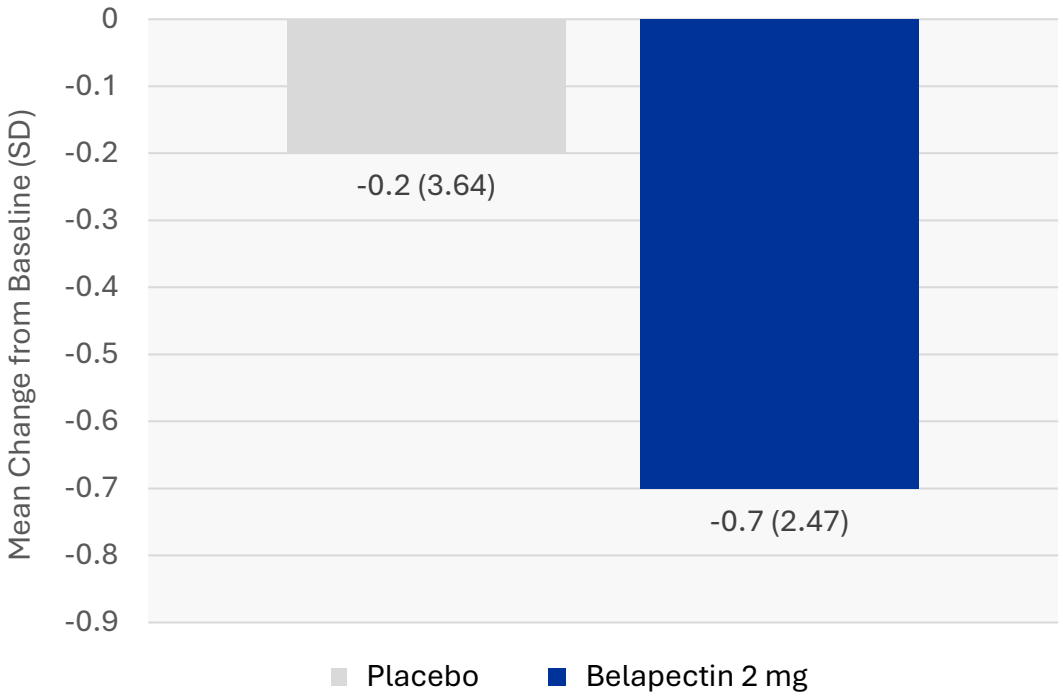
Baseline

	Placebo n = 61	**2 mg** n = 73
Mean	8.61	8.62
Standard Deviation	0.207	0.212

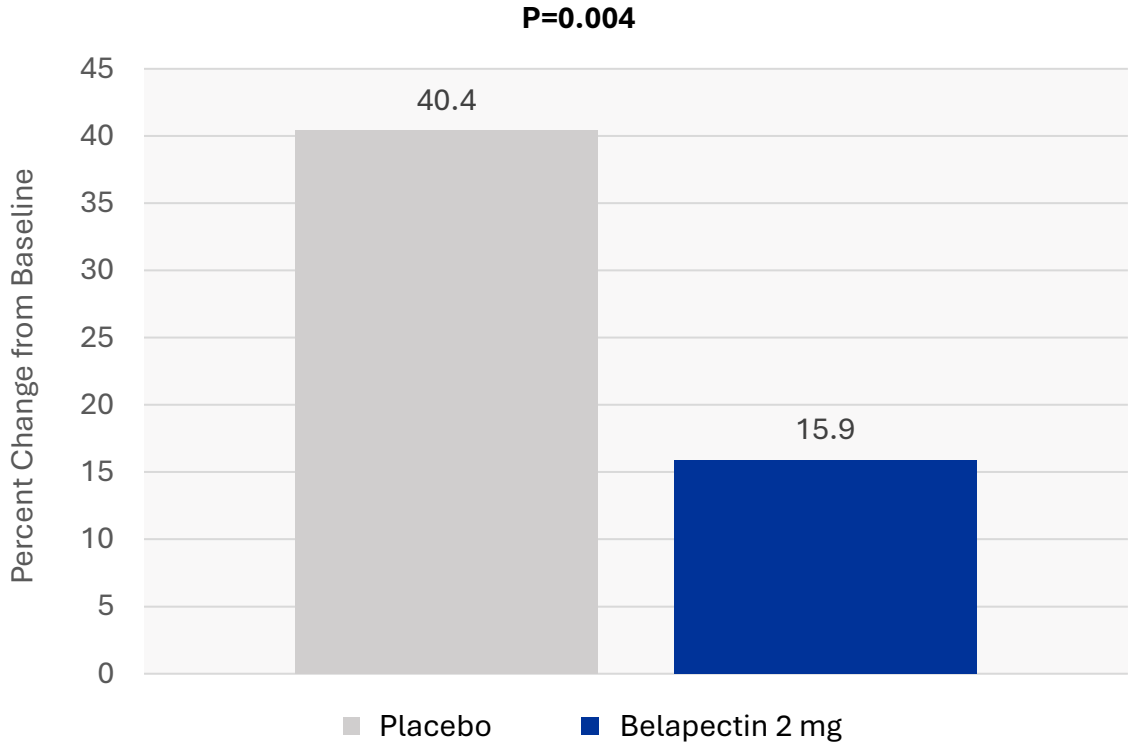
Table presents log transformed values for subjects with baseline and week 78 assessments

Belapectin Reduces Fibrogenesis in Highest-Risk Patients

Change in Pro-C3/CTX-III ratio in Subjects with baseline ELF≥ 11.3



Percentage Change in Pro-C3:CTX Ratio



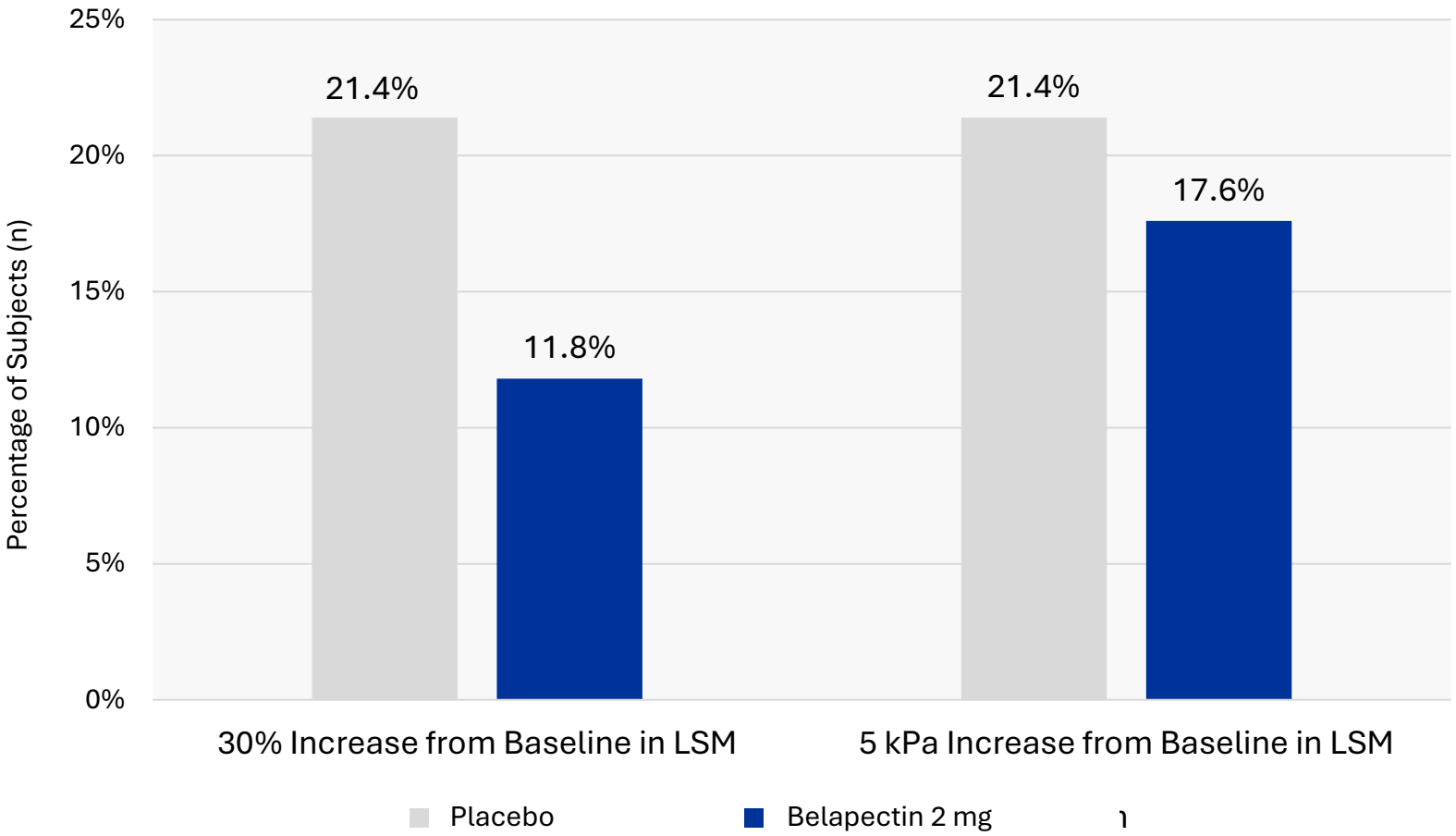
Placebo	2 mg
n = 21	n = 28

*Subjects with **ELF Increase ≥ 0.5***

P-value calculated utilizing Kruskal-Wallis Test

Fewer Subjects Worsening in LSM in High-Risk Patients

Baseline ELF ≥ 11.3



	Placebo	2 mg
n	14	17

NAVIGATE Phase 2b Trial Results Summary

Belapectin reduced varices incidence at 18 months

Belapectin 2 mg vs placebo reduced varices incidence by **43.2% (p=0.13) in the ITT** population and by **48.9% (unadjusted p=0.04) in the PPP***

In U.S. patients, results suggest synergistic benefit with GLP-1 therapy, highlighting Belapectin's potential as both monotherapy and in combination regimens

Concordant changes in key markers of liver fibrosis increase confidence in the clinical meaningfulness of new varices prevention

Belapectin was well tolerated with no safety signals

Favorable safety profile with comparable adverse events, serious adverse events, and discontinuation rates verses placebo

Probable reasons for not reaching statistical significance:

- Fewer recorded varices than expected; mid-study sample size re-estimation based on composite endpoint, not varices
- Shorter treatment duration
- Higher dropout rate than expected mostly due to the COVID pandemic

*Per-protocol population definition (18-month treatment + EGD) parallels completer biopsy definition in MASH trials.
Chalasani N, et al. *Hepatology*. 2026; DOI: 10.1097/HEP.0000000000001774.

Planned Phase 3 Trial in Patients with Compensated MASH Cirrhosis and Portal Hypertension

Primary endpoint

Composite liver outcome, including prevention of large esophageal varices (>5 mm) - a clinically meaningful indicator of disease progression

Central Endoscopy Review Methodology

FDA accepted the blinded central review process for endoscopic assessment of esophageal varices, a process Galectin pioneered and successfully implemented in the NAVIGATE trial[1]

Dosing

A single 2 mg dose of belapectin will be tested based on the consistent efficacy observed in two prior clinical studies[2,3]

Clear regulatory path toward a broad indication of prevention of progression of MASH cirrhosis

Phase 3 protocol is being finalized per FDA feedback received in June 2026 with anticipated submission in Q3 2026

1. Alkhouri N, et al. *Gastro Hep Adv*. 2026;5(8):101004. 2. Chalasani N, et al. Hepatology. 2026; DOI: 10.1097/HEP.0000000000001774. 3. Chalasani N, et al. *Gastroentrol*. 2020;158:1334-45.

Key Takeaways

01 Belapectin 2 mg significantly reduced the incidence of new esophageal varices at 18 months in patients with MASH cirrhosis and portal hypertension

02 Biomarkers results provide supportive evidence of the clinically meaningful results

03 These findings validate prior favorable observations from the GT-026 trial[1]

04 Favorable safety profile with adverse events, serious adverse events, and discontinuation rates comparable to placebo.

05 Belapectin has the potential to address the critical unmet need of patients with MASH cirrhosis and portal hypertension

06 Phase 3 registration trial in planning; actively pursuing strategic/partnership opportunities

1. Chalasani N, et al. Gastroentrol. 2020;158:1334-45.

Thank you!



Galectin Therapeutics

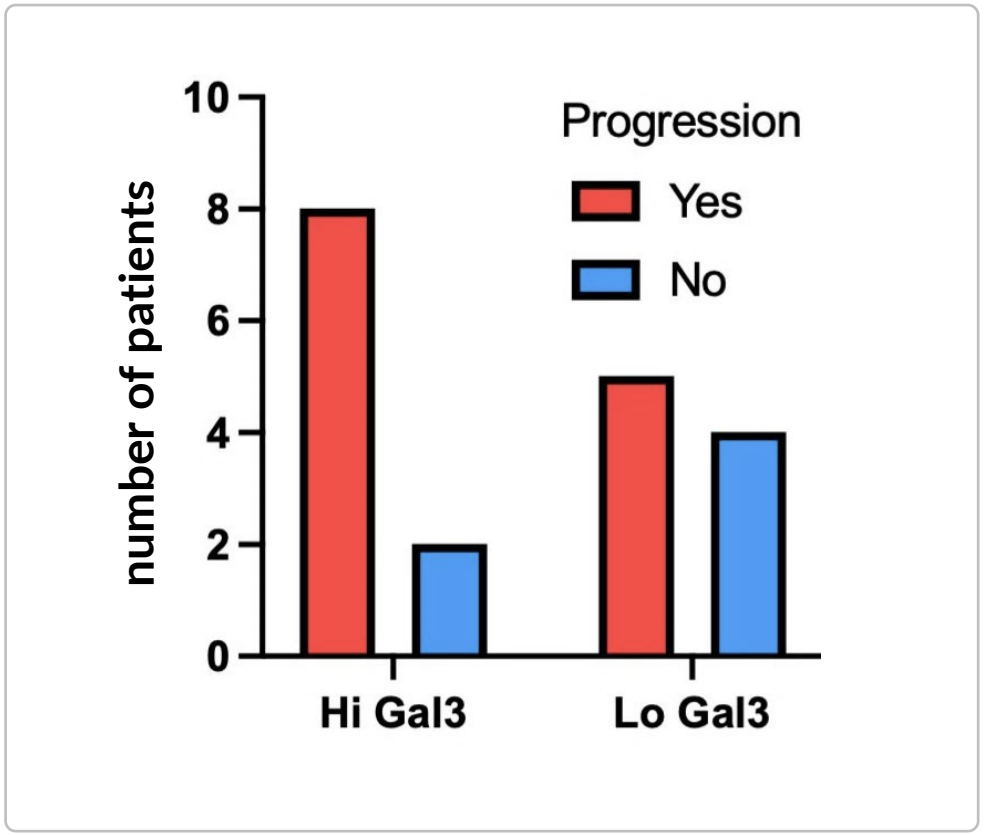
Cancer Immunotherapy Program (Belapectin + checkpoint inhibitor)

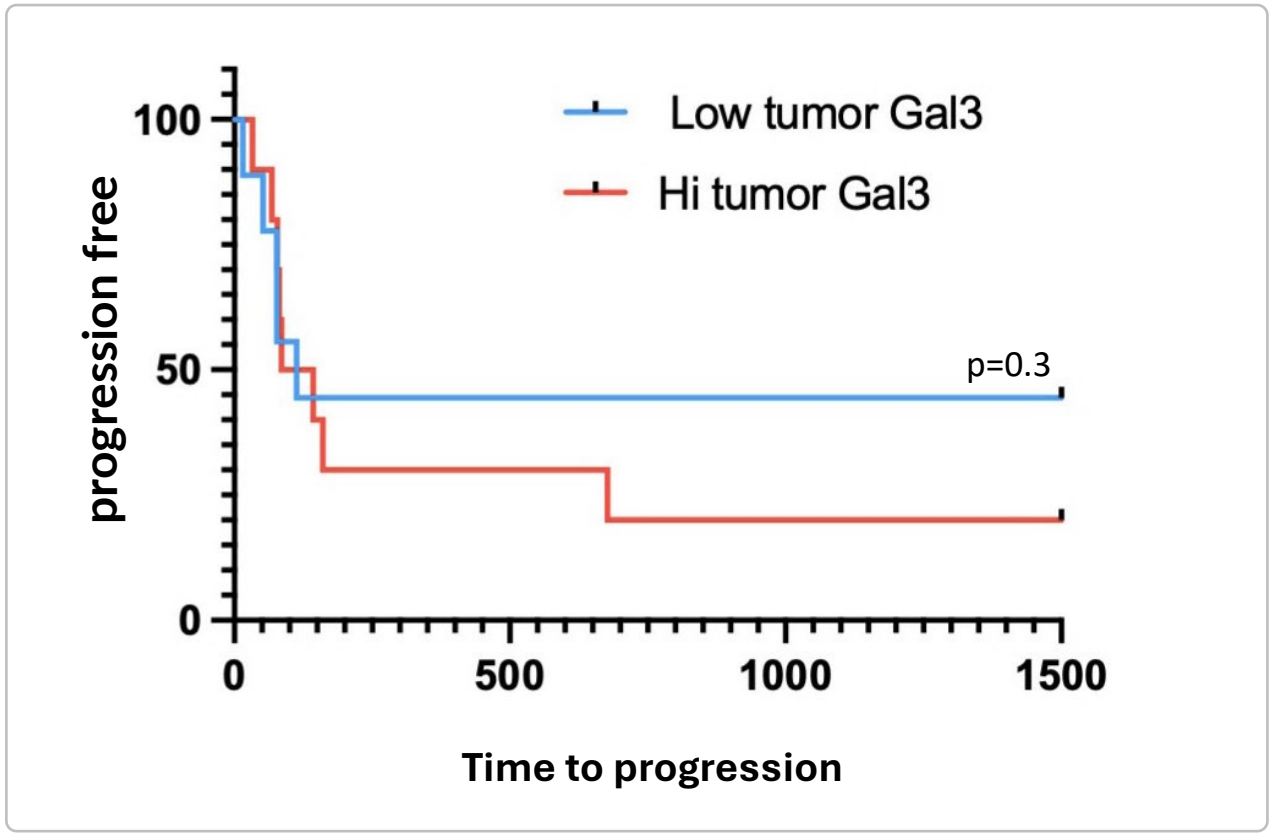


Galectin Therapeutics

Higher Galectin-3 Tumor Levels are Associated with Metastatic Melanoma Progression

Number of patients with or without progressive disease in hi/lo Galectin-3 expression in metastatic melanoma





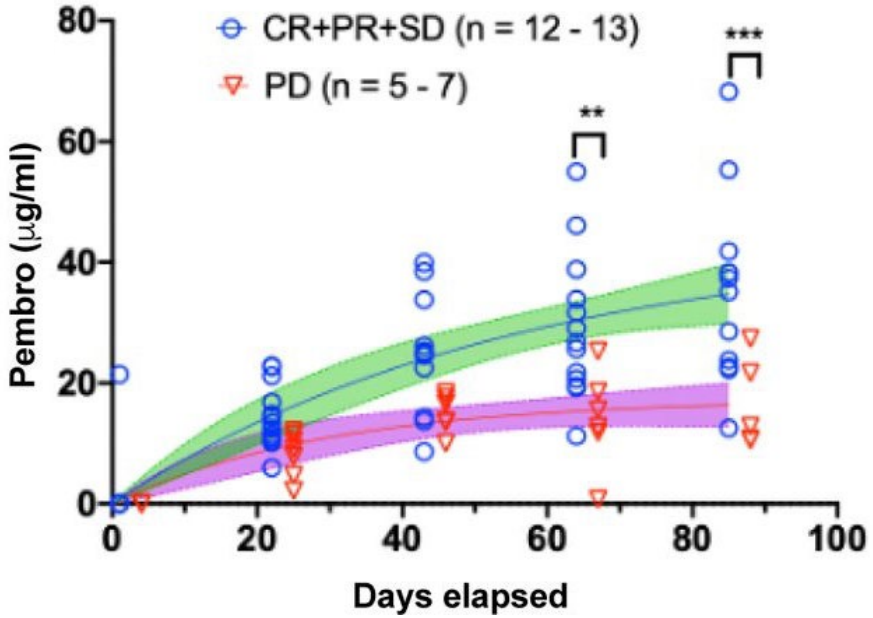
**p<0.01, ***p<0.001.

CR=complete response; HNSCC=head and neck squamous cell carcinoma; MM=metastatic melanoma; PD=progressive disease; PR=partial response; SD=stable disease.

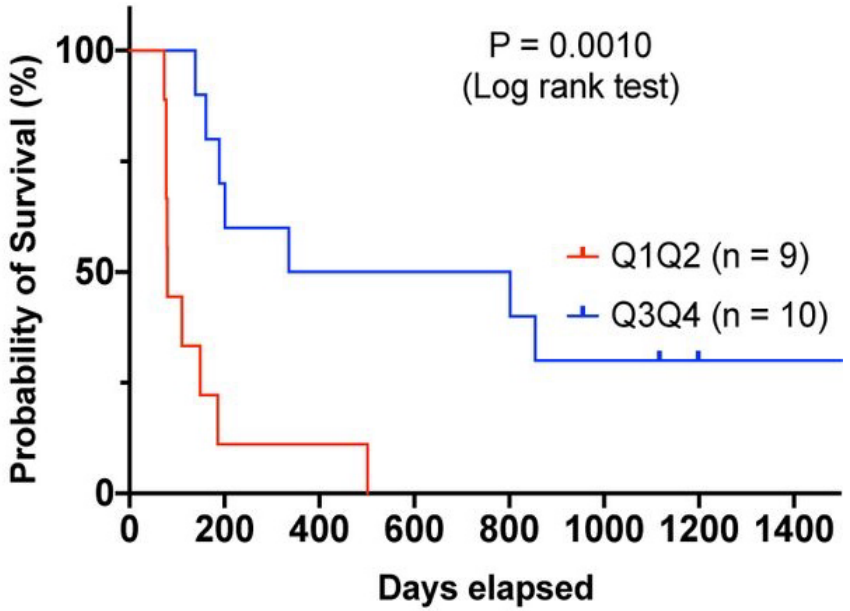
1. Greisen SR, et al. *J Immunother Cancer*. 2024;12(10):e009952.

Reduced PD-1 Clearance Correlates with Better Survival in Patients with MM and HNSCC

Serum trough levels of pembrolizumab in patients with disease control or progressive disease[1]



Increased progression-free survival in patients with higher trough level of pembrolizum[1,*]



Increased trough levels of belapectin and pembrolizumab correlated with better clinical outcome including progression free survival in patients with MM and HNSCC

*Patients were grouped based on the trough levels of pembrolizumab at day 43: Q1Q2 (below population mean) and Q3Q4 (above population mean).
**p<0.01, ***p<0.001.
CR=complete response; HNSCC=head and neck squamous cell carcinoma; MM=metastatic melanoma; PD=progressive disease; PR=partial response; SD=stable disease.
1. Curti B. *J Immunother Cancer.* 2021;9:e002371.

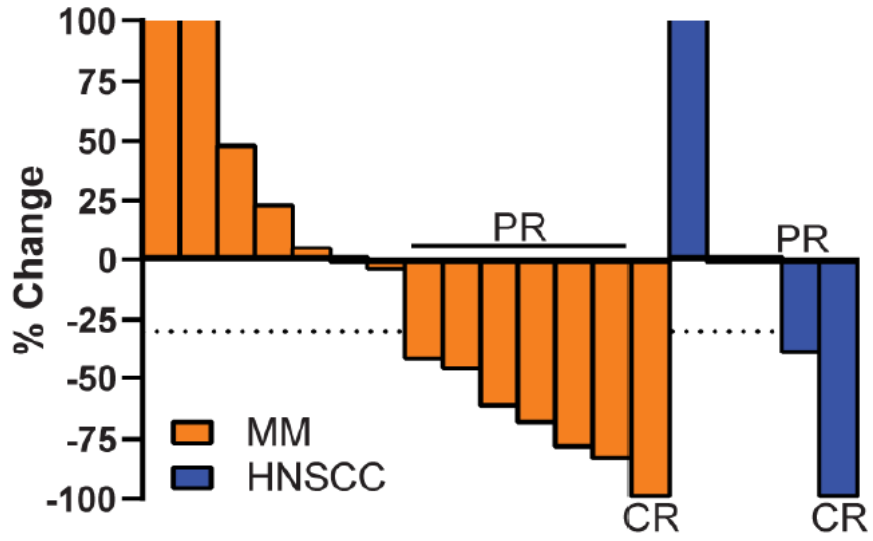
Belapectin in Combination with Pembrolizumab Showed Clinical Efficacy and Safety in Phase II

Phase 1 (Investigator-Initiated) of belapectin + pembrolizumab (Keytruda®)

- Objective response observed in 50% of MM (7/14) and 33% of HNSCC (2/6) patients
- Extension in more advanced patients showed stable disease in 56% MM (5/9) and 40% in HNSCC (2/5)
- Combination treatment was well tolerated with no dose-limiting toxicity observed
- Fewer immune adverse events than expected
- Increased baseline expression of Gal3^{+} tumor cells, periphery PD-1^{+}CD8^{+} T cells and reduced clearance of pembrolizumab correlated with clinical response

IND filed and approval to proceed received from FDA (Head and Neck cancer)

Objective response to belapectin+pembrolizumab therapy at Day 85



HNSCC=head and neck squamous cell carcinoma; MM=metastatic melanoma.

1. Curti B. J Immunother Cancer. 2021;9:e002371.

Appendix

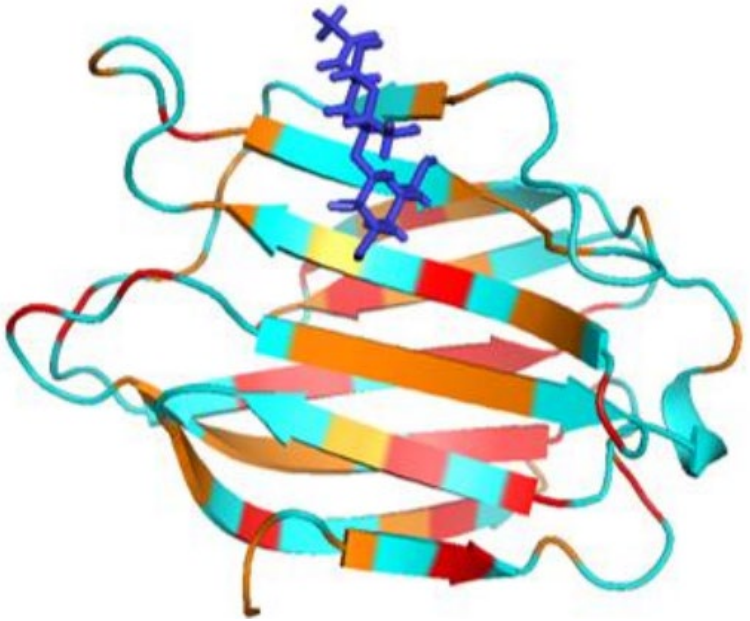
Galectin Therapeutics

Belapectin for MASH Cirrhosis and Portal Hypertension

Belapectin Preclinical Data:

Multiple animal models of MASH (streptozotocin High-Fat Diet mice[1]) and cirrhosis (thioacetamide treated rats[2]) belapectin was associated with decreased:

- Galectin-3 staining and galectin-3 expression in macrophages
- NAFLD Activity Scores
- Collagen-1 expression
- Hepatic collagen deposition
- Hepatic fibrosis
- Portal pressure



Toxicology Data

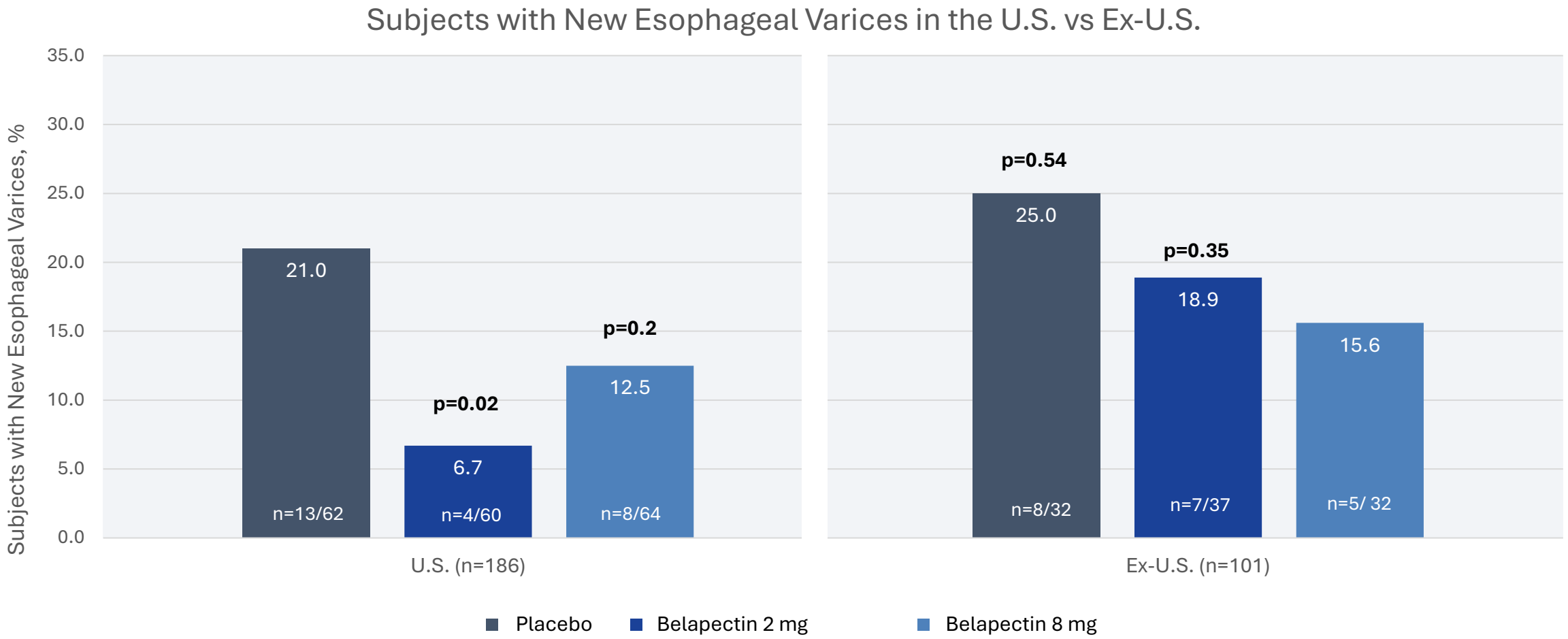
- Well-tolerated even at high doses
- Accumulated in macrophages with a residence time longer than in plasma

Belapectin is a polysaccharide polymer comprising galacturonic acid, galactose, arabinose, rhamnose and smaller amounts of other sugars. Suite of oral galectin-3 inhibitor molecules as part of life cycle management plan.

1. Traber PG, et al. *PLoS One*. 2013;8(12):e83481. 2. Traber PG, et al. *PLoS One*. 2013;8(10):e75361.

Incidence of New Varices was Significantly Lower in Patients in the U.S.

NAVIGATE 18-month; Per protocol population (n=287)



Use of GLP-1 and Statin was Higher in Patients in the U.S.

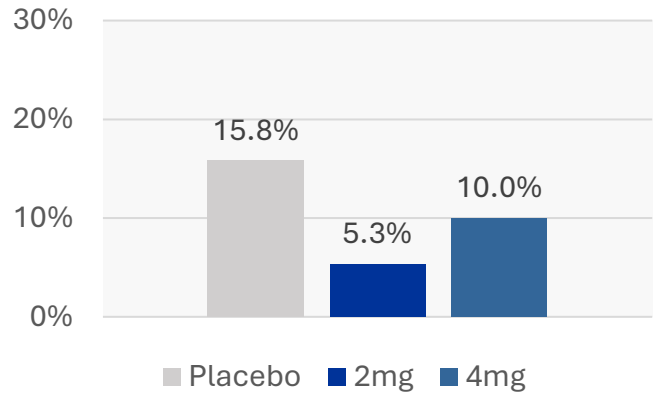
Concomitant medication Use U.S. vs Ex- U.S.- Per Protocol

		Treatment Group			
		Placebo	Belapectin 2 mg	Belapectin 4 mg	Total
		(N=62)	(N=60)	(N=64)	(N=186)
U.S.	**Concomitant Use of GLP-1 n (%)**	**28 (45.2%)**	**22 (36.7%)**	**18 (28.1%)**	**68 (36.6%)**
	Concomitant Use of NSBBs n (%)	5 (7.9%)	3 (5.0%)	3 (4.6%)	11 (5.9%)
	Concomitant Use of Statins n (%)	**34 (54.8%)**	**31 (51.7%)**	**26 (40.6%)**	**93 (48.9%)**
	Concomitant Use of ACE Inhibitors n (%)	15 (23.8%)	17 (28.3%)	18 (27.7%)	50 (26.6%)
		(N=32)	(N=37)	(N=32)	(N=101)
EX-U.S.	**Concomitant Use of GLP-1 n (%)**	**5 (15.6%)**	**8 (21.6%)**	**12 (37.5%)**	**25 (24.5%)**
	Concomitant Use of NSBBs n (%)	2 (6.3%)	2 (5.4%)	3 (9.4%)	7 (6.9%)
	Concomitant Use of Statins n (%)	**8 (25.0%)**	**14 (37.8%)**	**16 (50%)**	**38 (37.6%)**
	Concomitant Use of ACE Inhibitors n (%)	4 (12.5%)	12 (32.4%)	11 (34.4%)	28 (27.5%)

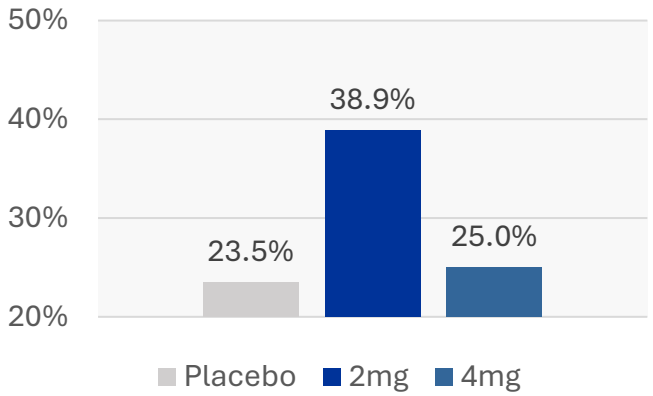
Impact of Concomitant Use of GLP-1 and Statins

	Placebo	2 mg	4 mg	Total
Yes	18	19	20	**57**
No	75	78	76	**229**

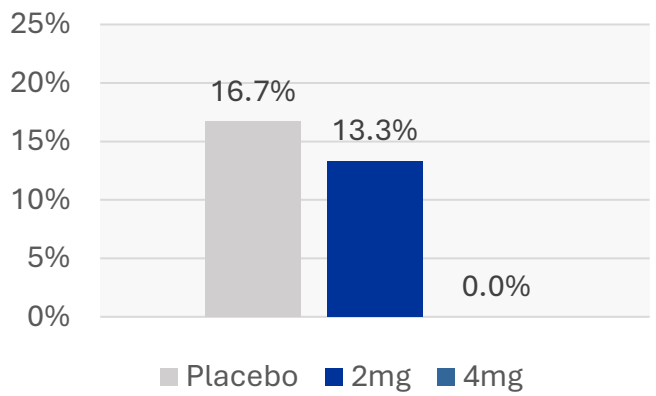
New Varices at 18 months



≥ 0.5pt ELF Increase

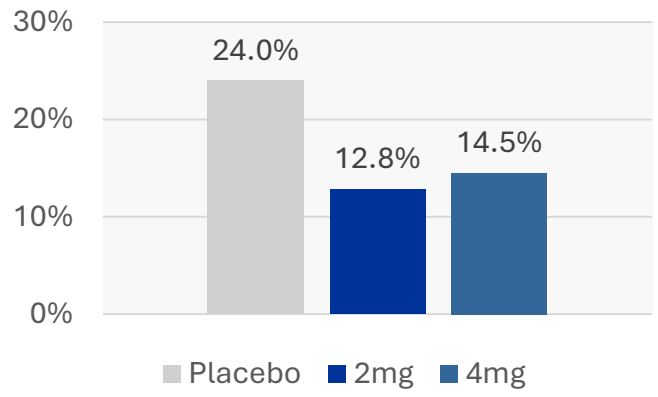


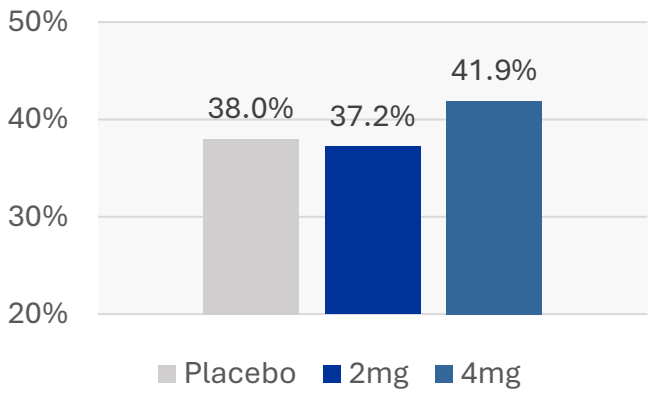
≥ 5 kPa LSM Increase

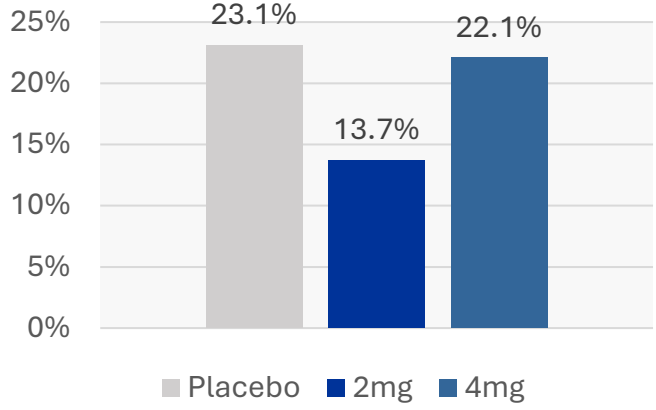


YES
(n=57)

NO
(n=229)







Per-Protocol (n = 287)

Reference Guide: Biomarkers and Non-Invasive Scores in MASH Cirrhosis

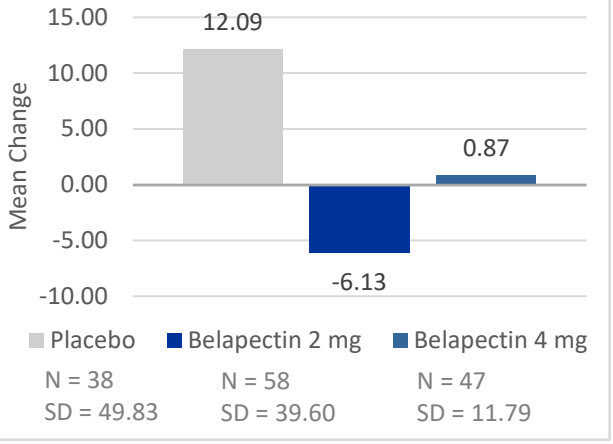
Biomarker	Target	Biological Process
LSM by VCTE	Measures fibrosis via transient elastography (ultrasound-based)	Non-invasive quantification of liver stiffness reflecting fibrosis severity; used for diagnosis, prognosis, and treatment monitoring
ELF Test	Composite score of three extracellular matrix markers (HA, PIIINP, TIMP-1)	Reflects active fibrogenesis and matrix turnover; prognostic for fibrosis progression and clinical outcomes
PRO-C3	Formation of type III collagen	Marker of **active fibrogenesis** reflecting stellate cell collagen synthesis and matrix deposition
PRO-C4	Formation of type IV collagen	Reflects **basement membrane remodeling** and endothelial-matrix interface turnover in fibrosis
YKL-40	Chitinase-like glycoprotein secreted by macrophages and stellate cells	Marker of **inflammation, macrophage activation, and extracellular matrix remodeling**
AGILE-4	Integrates LSM, Age, Sex, AST/ALT, Platelet, Diabetes status	Diagnostic and prognostic tool in MASH and MASH cirrhosis
Baveno Criteria	Expert consensus criteria to define portal hypertension using LSM and Platelet Count	Risk stratification, treatment decision support

Belapectin Consistently Reduced Pro-Inflammatory Markers at 18 months

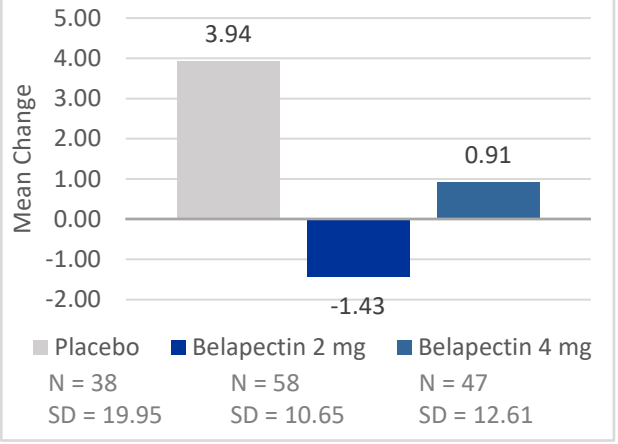
Pro-Inflammatory

Anti-inflammatory

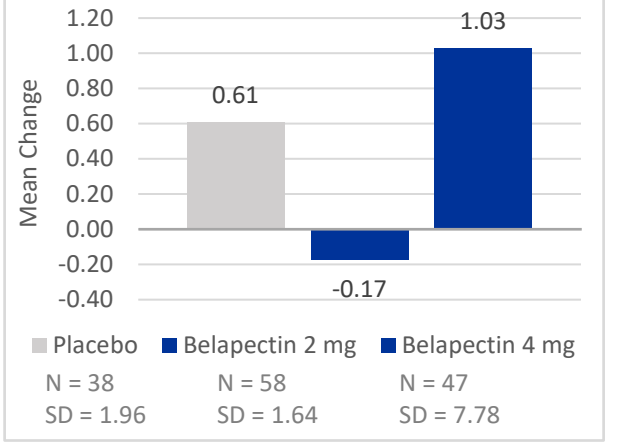
TNF-alpha



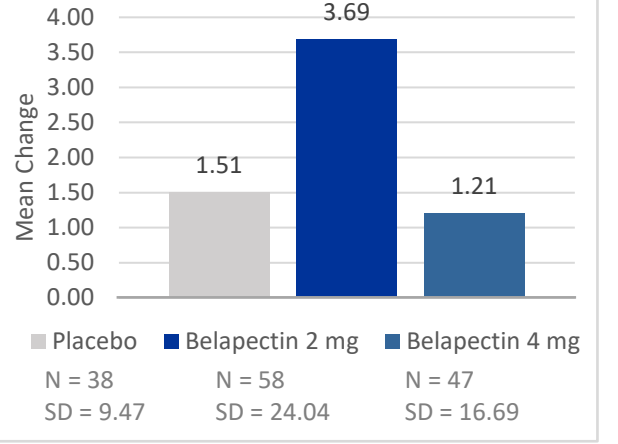
IL-1b



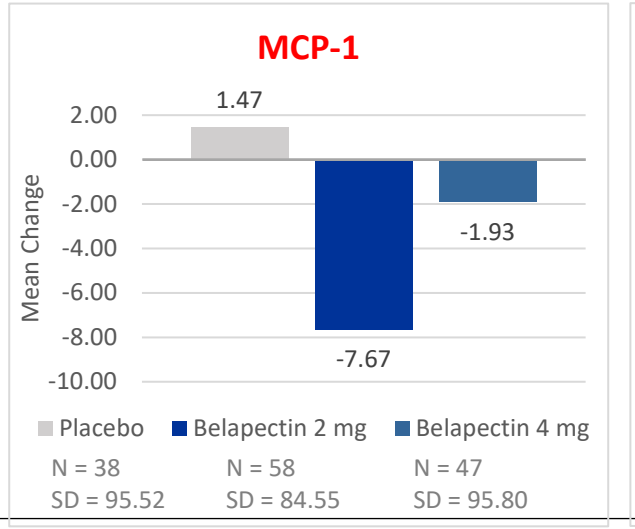
IL-6



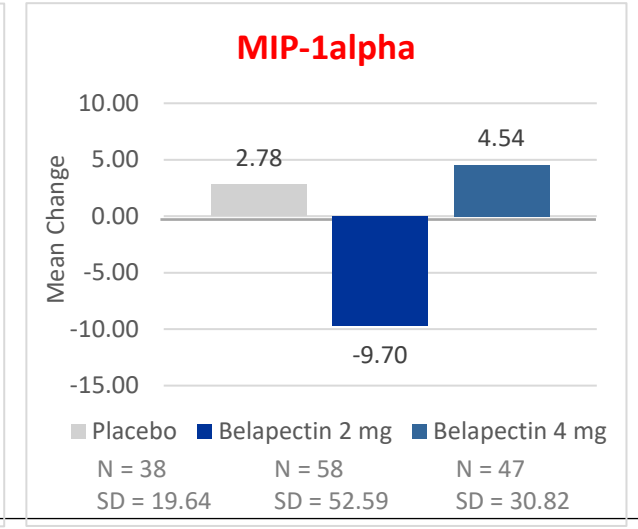
IL-10



MCP-1



MIP-1alpha



ITT population n=143

Inflammatory Biomarkers: Role and Function

Biomarker	Primary Cellular Source(s)	Pathophysiologic Role in MASH / Cirrhosis	Fibrosis Association	Expected Modulation with Gal-3 Inhibition
IL-1β (Pro-inflammatory)	Kupffer cells, macrophages, stellate cells	Activates inflammasome, promotes hepatocyte death and HSC activation; initiates inflammatory cascade	↑ Strongly correlated with necroinflammation and fibrosis	↓ **Decrease** — reduced macrophage activation and inflammasome signaling
IL-6 (Pro-inflammatory / regenerative)	Kupffer cells, hepatocytes	Induces acute-phase response and insulin resistance; sustains chronic inflammation	↑ Elevated in progressive MASH and cirrhosis	↓ **Decrease** — attenuation of inflammatory drive and acute-phase signaling
MCP-1 / CCL2 (Pro-inflammatory chemokine)	Kupffer cells, stellate cells, endothelial cells	Recruits monocytes and macrophages; key mediator of macrophage accumulation in fibrotic liver	↑ Strong correlation with fibrosis stage	↓ **Decrease** — reduced monocyte infiltration under Gal-3 blockade
MIP-1α / CCL3 (Pro-inflammatory chemokine)	Macrophages, stellate cells, endothelial cells	Amplifies local cytokine cascade and immune cell recruitment	↑ Elevated in fibrogenic and portal inflammatory zones	↓ **Decrease** — dampened chemokine amplification loop
TNF-α (Pro-inflammatory)	Kupffer cells, infiltrating macrophages, adipocytes	Drives hepatocyte apoptosis, metabolic dysfunction, and stellate cell activation	↑ Strongly associated with inflammation grade and fibrosis severity	↓ **Decrease** — suppression of TNF-α cascade via macrophage deactivation
IL-10 (Anti-inflammatory)	Kupffer cells, regulatory T cells, monocytes	Suppresses TNF-α, IL-1β, IL-6; limits inflammation and fibrosis progression	↓ Often reduced in active MASH; compensatory rise in cirrhosis	↑ **Increase** — restoration of anti-inflammatory signaling balance