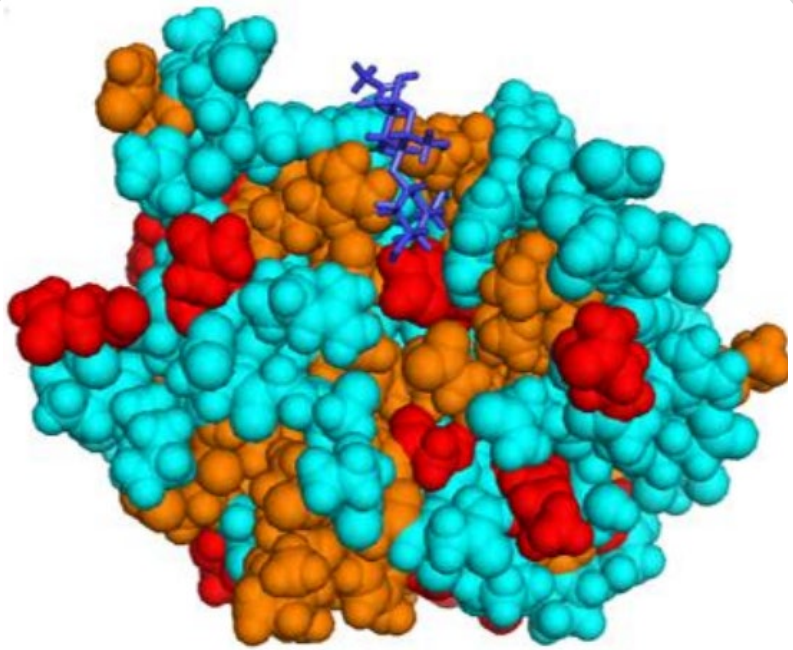
Galectin Therapeutics Corporate Overview

December 2025



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 those regarding potential therapeutic benefits of our drugs, expectations, plans and timelines related to our clinical trials, supporting activities, potential partnering opportunities and estimated spending for 2025 and beyond. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, full analysis of the NAVIGATE trial data 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, 2024, 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 galectin-based 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

Low toxicity as a carbohydrate-based molecule which is degraded by natural processes

Patent protection through 2032

MASH Cirrhosis

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

Significant efficacy observed in cirrhotic patients without varices

Promising NAVIGATE results at 18 month read out, ≥40% reduction in new varices vs placebo in ITT; significantly lower incidence of new varices in per protocol population

Oncology (Combination Therapy)

Encouraging clinical response in difficult-to-treat cancers in combination with checkpoint inhibitor

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

Highly Experienced Leadership Team

JOEL LEWIS
Chief Executive Officer & President

Financial executive with over 25 years of management experience in a taxation, restructuring, acquisition, and private equity ventures.

KHURRAM JAMIL, M.D.
Chief Medical Officer

Have two decades of expereince 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 32 years of public and private company experience including more than a decade of audit, tax and SEC registrant experience with a major accounting firm.

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

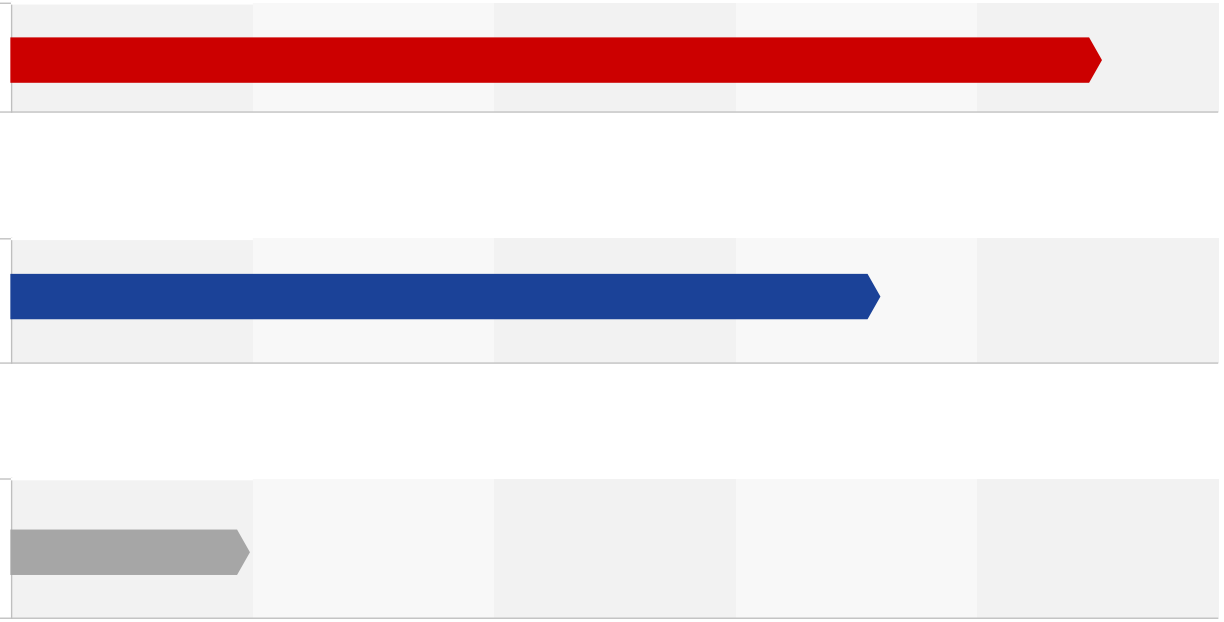
Over 30 years of experience in CMC and biopharmaceutical development. Previously served as Chief Technology Officer at Sensorin and Director of Technology Assessment at Genentech.

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
Fibrosis						
Belapectin	**MASH Cirrhosis and Portal Hypertension**					
Cancer Immunotherapy (Combination therapy)						
Belapectin + Keytruda	**Melanoma + Head / Neck Cancer**					
Oral Galectin-3 Inhibitors						
Discovery program to identify subcutaneous forms of carbohydrates and oral small molecules						



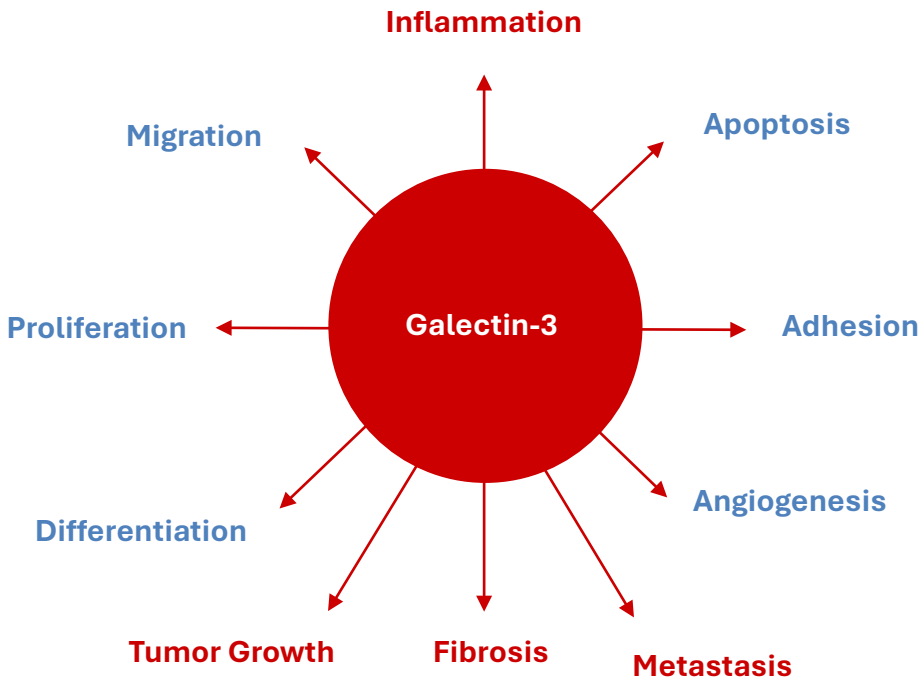
Galectin-3 is a Promising Therapeutic Target in Inflammatory and Fibrotic Diseases[1,2]

Galectin 3 is part of the galectin family of sugar-binding proteins that act as a “molecular glue”, it is:

- Predominantly produced by activated macrophages
- Involved in a wide number of biological and pathological processes

Galectin-3 recruits macrophages to injury sites and promotes chronic inflammation by activating proinflammatory pathways

Galectin-3 drives many pathophysiological process in fibrotic diseases and cancer



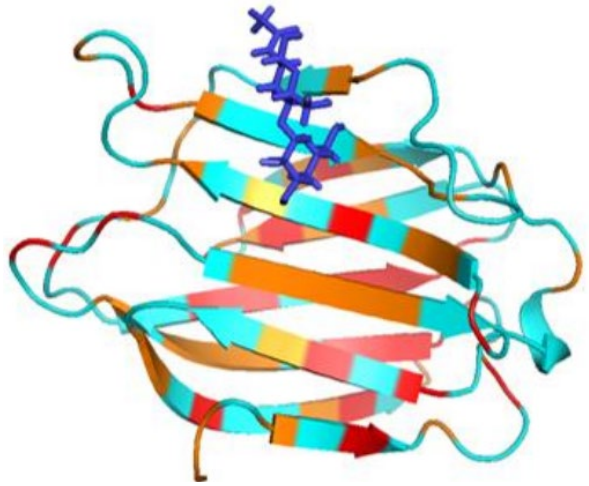
1. Marino KV, et al. *Nat Rev Drug Discov*. 2023;22(4):295-316. 2. Henderson NC, et al. *Proc Natl Acad Sci U S A*. 2006;103(13):5060-5.

Belapectin: a Proprietary Galectin-3 Inhibitor with Low Toxicity and Anti-fibrotic Activity

Belapectin Preclinical Data:

In 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



In toxicology studies, including monkeys, belapectin:

- Was 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

Galectin Therapeutics

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

MASH Cirrhosis

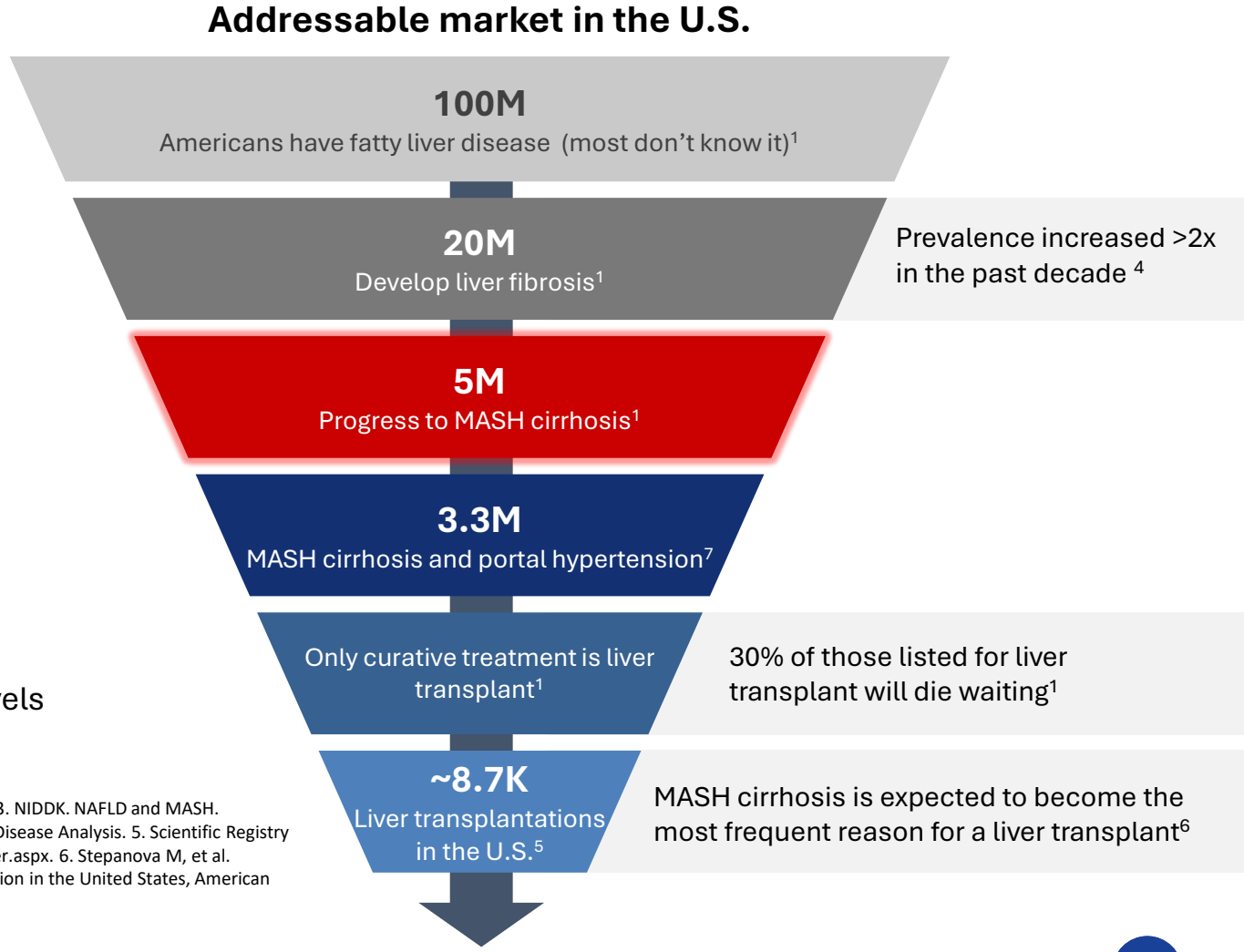
MASH Cirrhosis Represents a Significant Market Opportunity in the U.S. with No FDA-Approved Treatment

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 2021 Annual Data Report: Liver. https://srtr.transplant.hrsa.gov/annual_reports/2021/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 is a Novel Therapy with First- and Best-in-Class Potential in MASH Cirrhosis

United States Estimates[1]

5M Patients with compensated MASH cirrhosis in 2024

1.7M Patients with compensated cirrhosis and portal hypertension with no varices in 2024

$18B Peak belapectin sales in 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

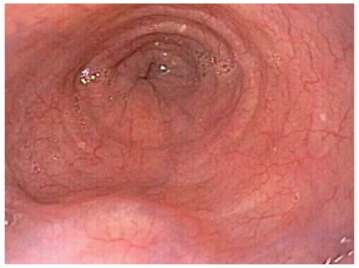
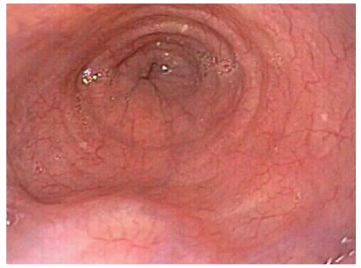
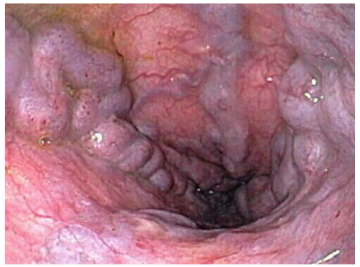
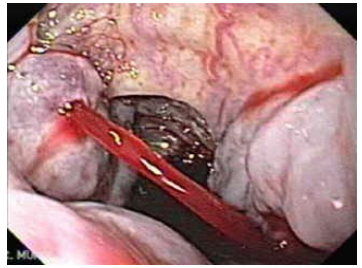
Payers believe in the high unmet need in MASH cirrhosis

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

Galectin Therapeutics

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

When to Intervene in Cirrhosis- before its too late!

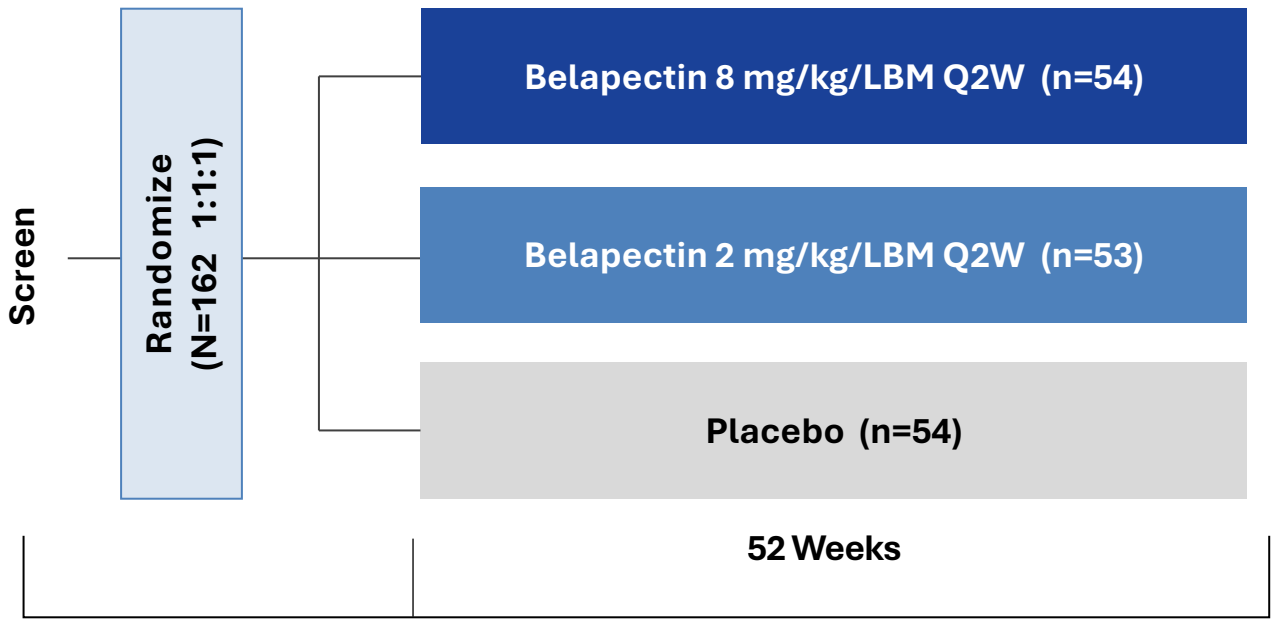
Compensated cirrhosis			Decompensated cirrhosis
No Portal Hypertension	Portal Hypertension		
No varices	*No varices*	*Varices, small to large*	Varices Bleeding, ascites, encephalopathy
			
≥6	HVPG[1] mm Hg	>10	
	One year mortality 1-3%		One year mortality ~50%

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

HPVG=hepatic venous pressure gradient.

Galectin Therapeutics

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 (HPVG) change from baseline to Week 54

Secondary endpoints at Week 54

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

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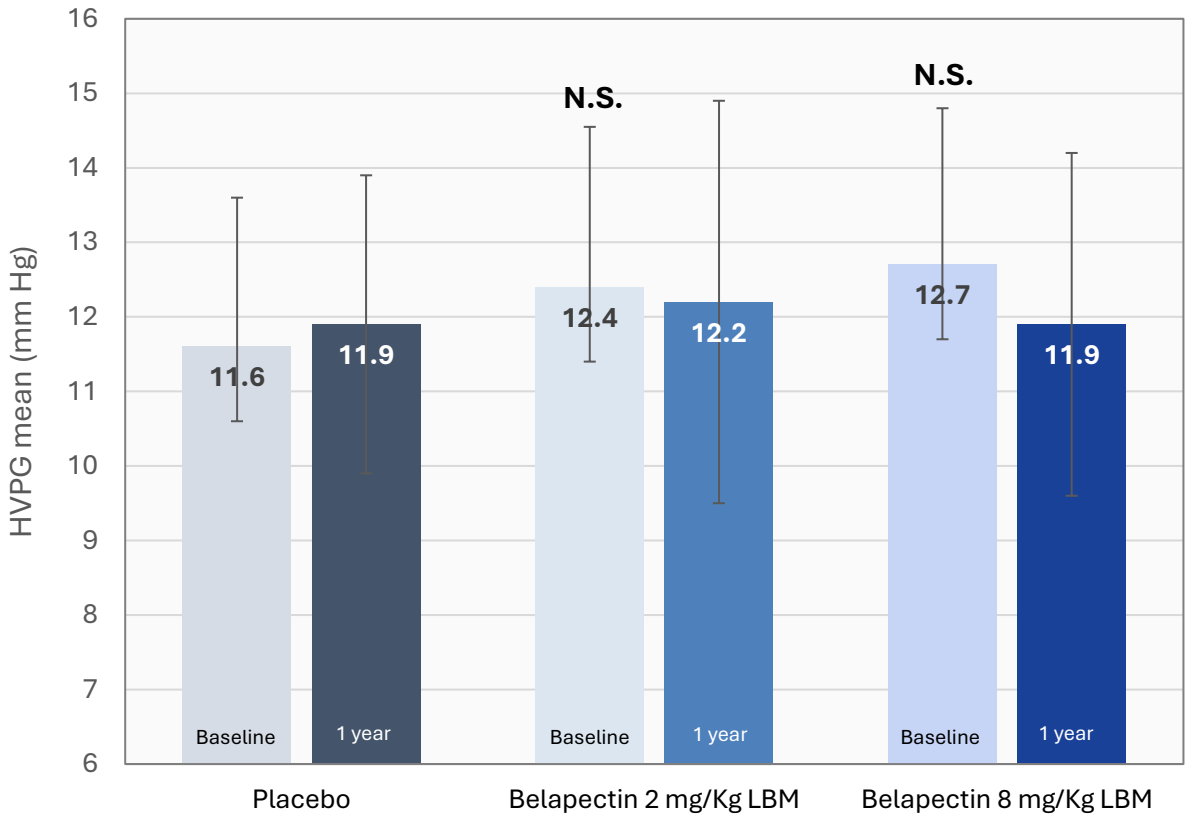
HVPG = Hepatic Venous Pressure Gradient; LBM=lean body mass.

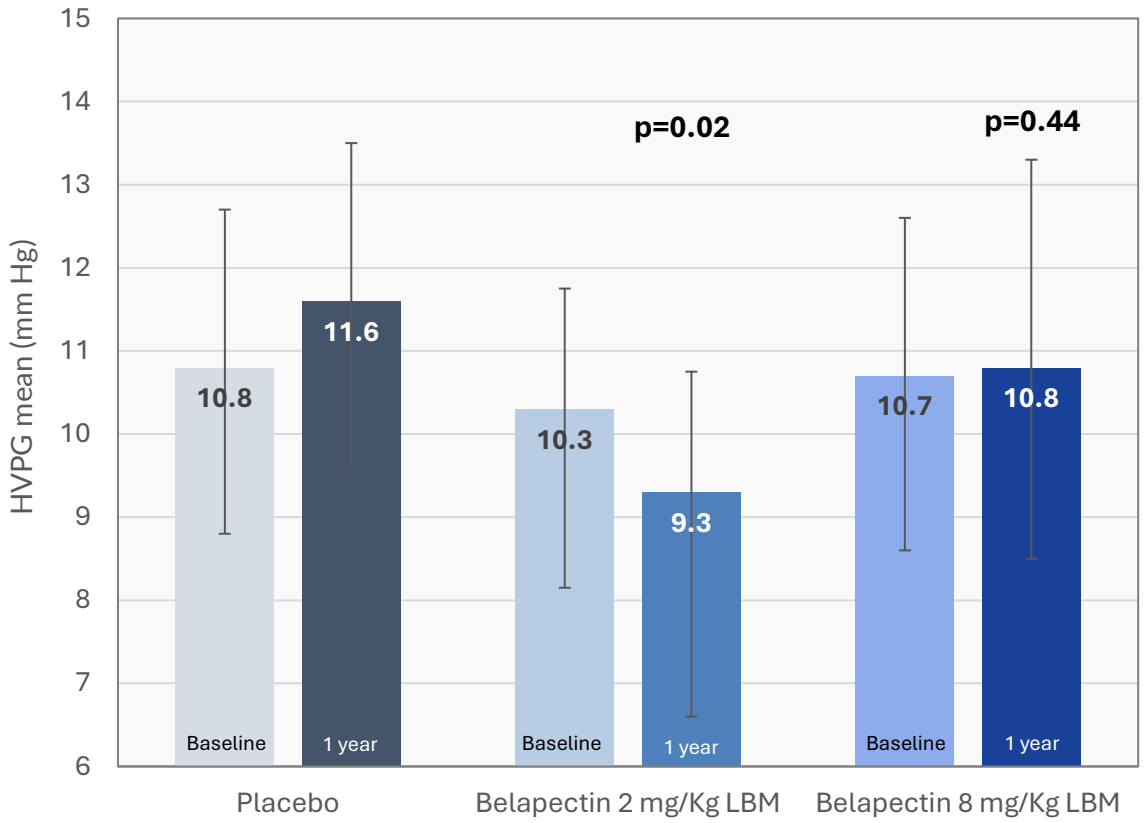
1. Chalasani N, et al. *Gastroentrol.* 2020;158:1334-45.

Belapectin Impact on HPVG at One Year[1,*]

ITT Population



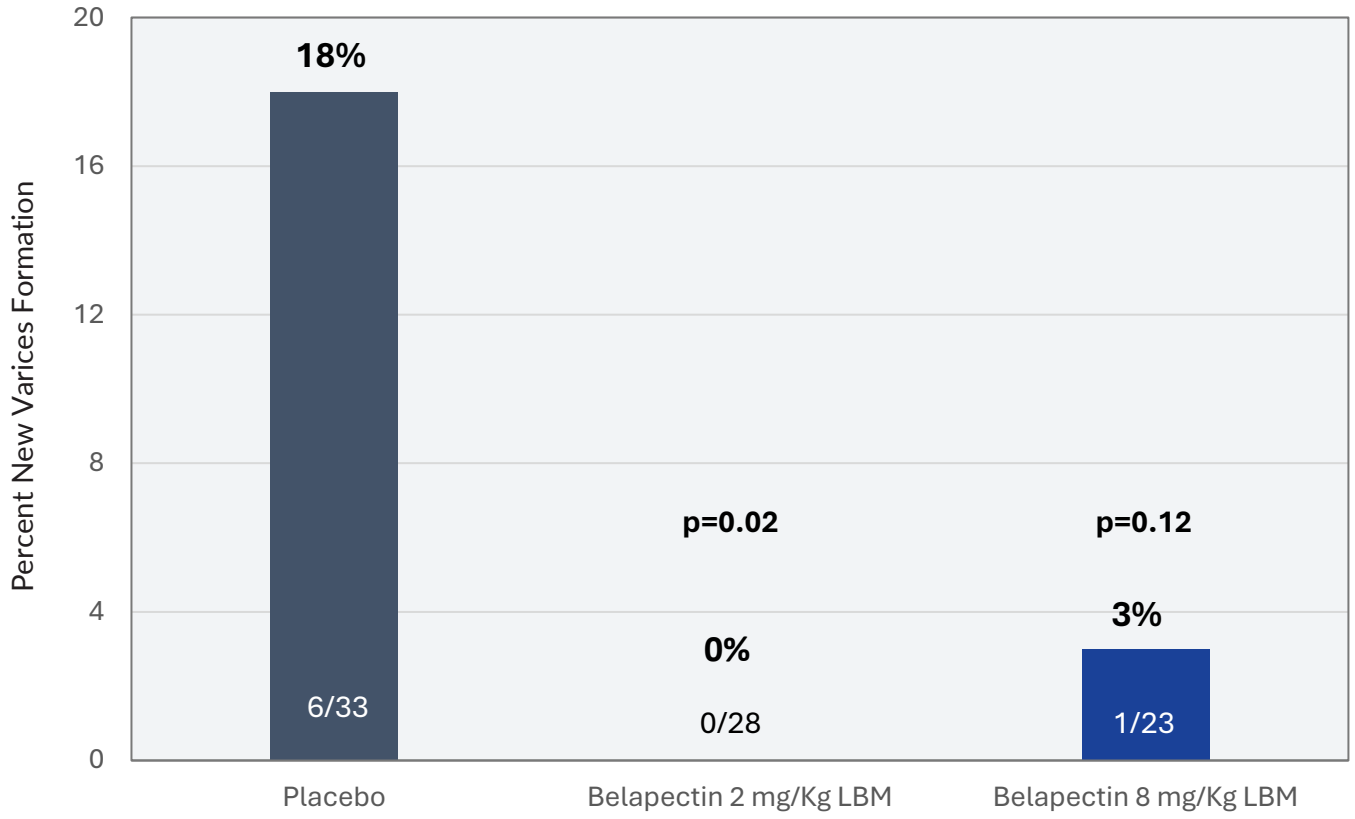
Subjects with no varices at baseline



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.

Belapectin Reduces Emergence of Varices in Patients with MASH Cirrhosis[1,*]



Significantly fewer new varices on belapectin vs placebo

No patients on 2 mg/kg/LBM developed new varices

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

LBM=lean body mass.
*Chi square
1. Chalasani N, et al. *Gastroentrol.* 2020;158:1334-45.

NAVIGATE Trial Design

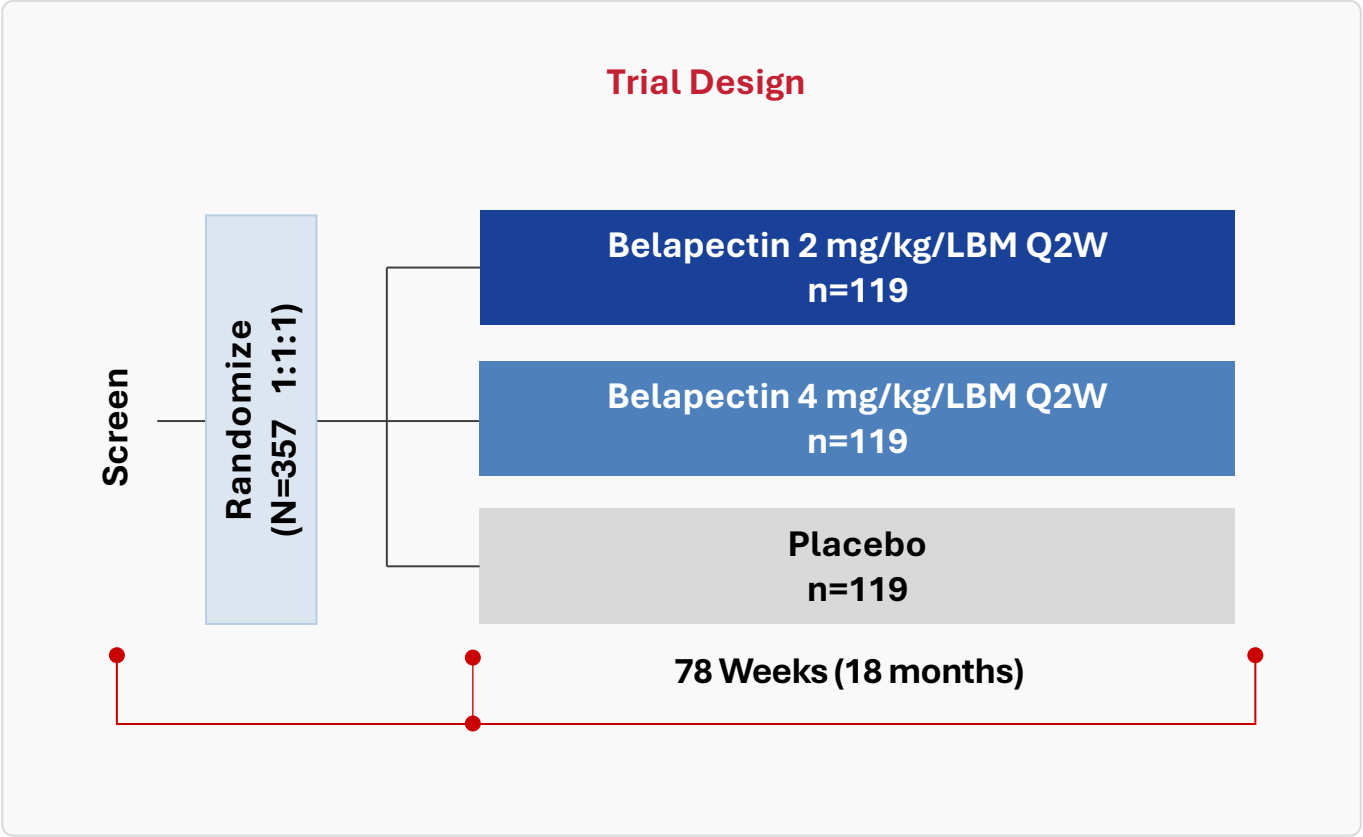
Patient Population

MASH cirrhosis based on Liver Forum Recommended Criteria for Clinical Trials[1]

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

No gastroesophageal varices by endoscopy at baseline

Assessment of Varices thru central adjudication of endoscopy videos by multiple blinded reviewers based on standardized protocol.



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

- AST/ALT > 1
- Spleen ≥ 14 cm
- Collaterals by imaging
- Stiffness ≥ 20 kPa

Primary endpoint

Development of new varices (composite strategy) in ITT population

Incidence of Varices in per protocol population (Completers)

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

Non-invasive biomarkers

Galectin GT Therapeutics

ALT=alanine aminotransferase ; AST=aspartate transaminase; CTP=Child-Turcotte-Pugh; EGD=Esophagogastroduodenoscopy; MELD=model for end-stage liver disease.
*Intercurrent events include; Liver related clinical events, any AE leading to discontinuation, TIPS Trans-jugular intrahepatic portosystemic shunt; ≥12-month use of GLP-1 or NSBB
ITT- Intent to Treat

Key Populations for Assessment of Varices Outcome

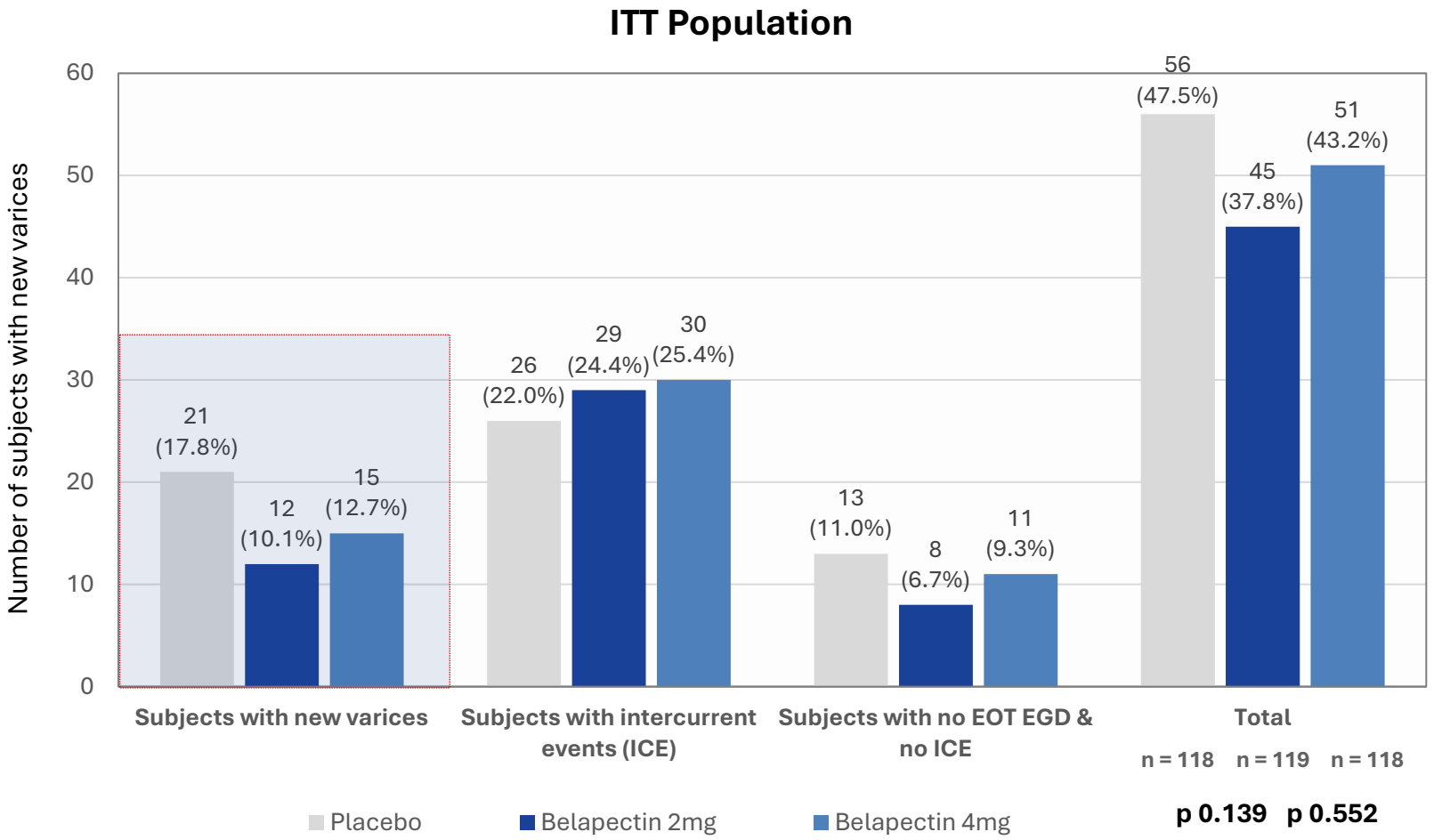
- **ITT population-** All randomized subjects minus two subjects who had varices at baseline;
- **Per-Protocol or completer population-** All subjects who completed 18 month of therapy and had an EGD at baseline and 18 months
    - Subject were required to complete the study even after development of varices unless subject dropped out for other reasons
- **Composite Primary end point:** 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

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)

NAVIGATE 18-Month Primary Analyses Result – ITT Population



Composite Primary Endpoint, ITT (All Randomized)

Key points

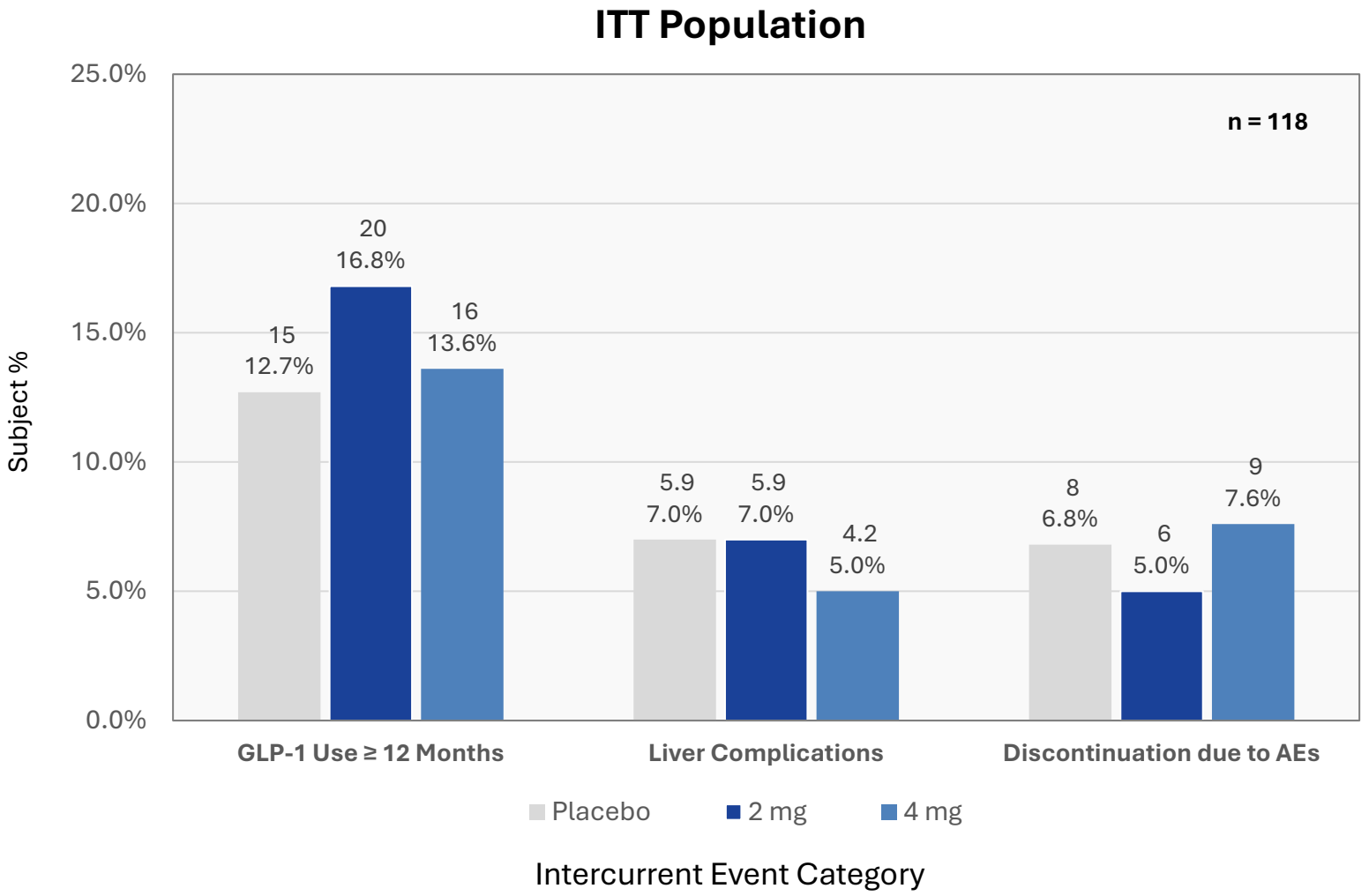
Intent to Treat (ITT) -All randomized subjects

Primary end point 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.

NAVIGATE: Intercurrent Events breakdown by category

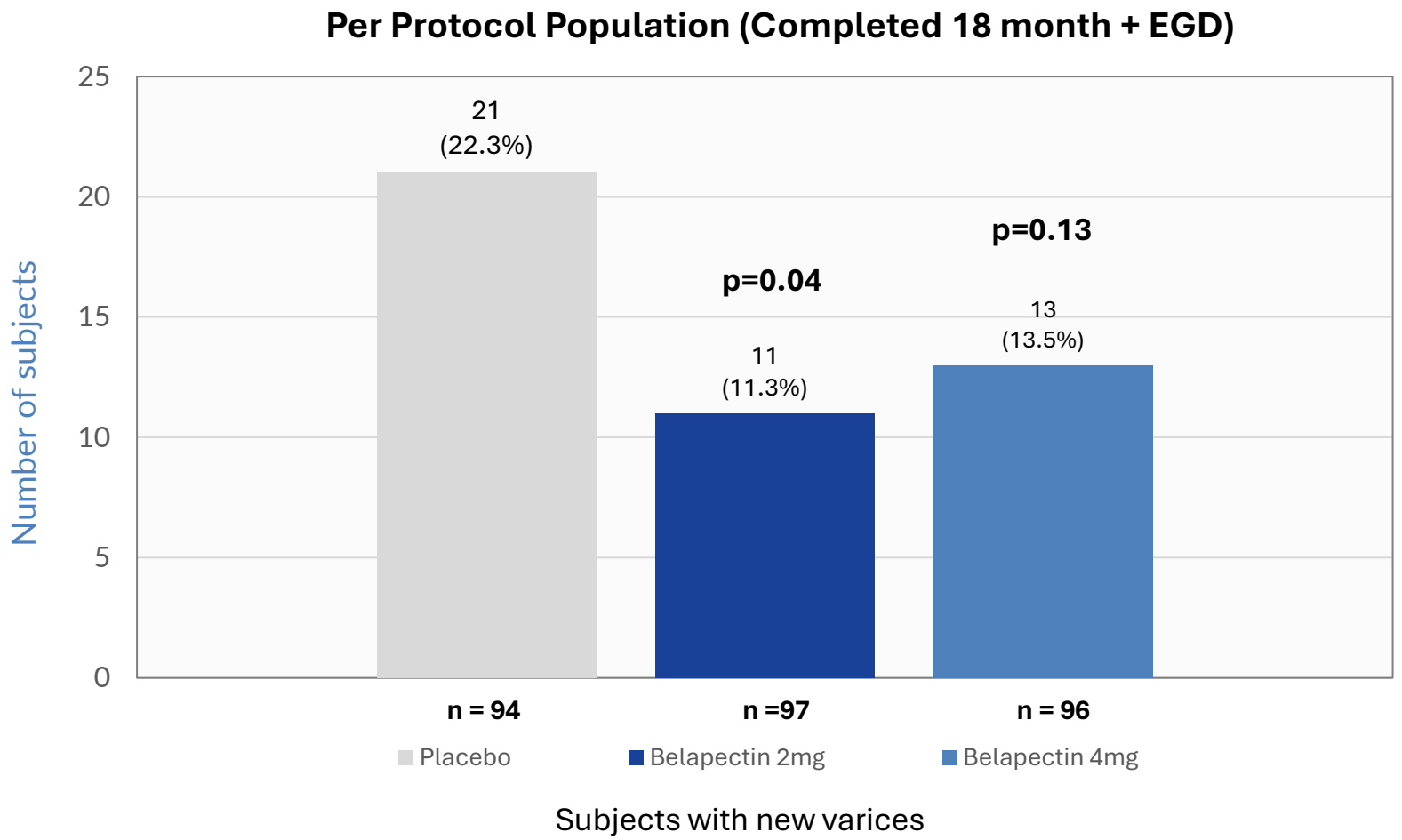


Key points

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

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

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



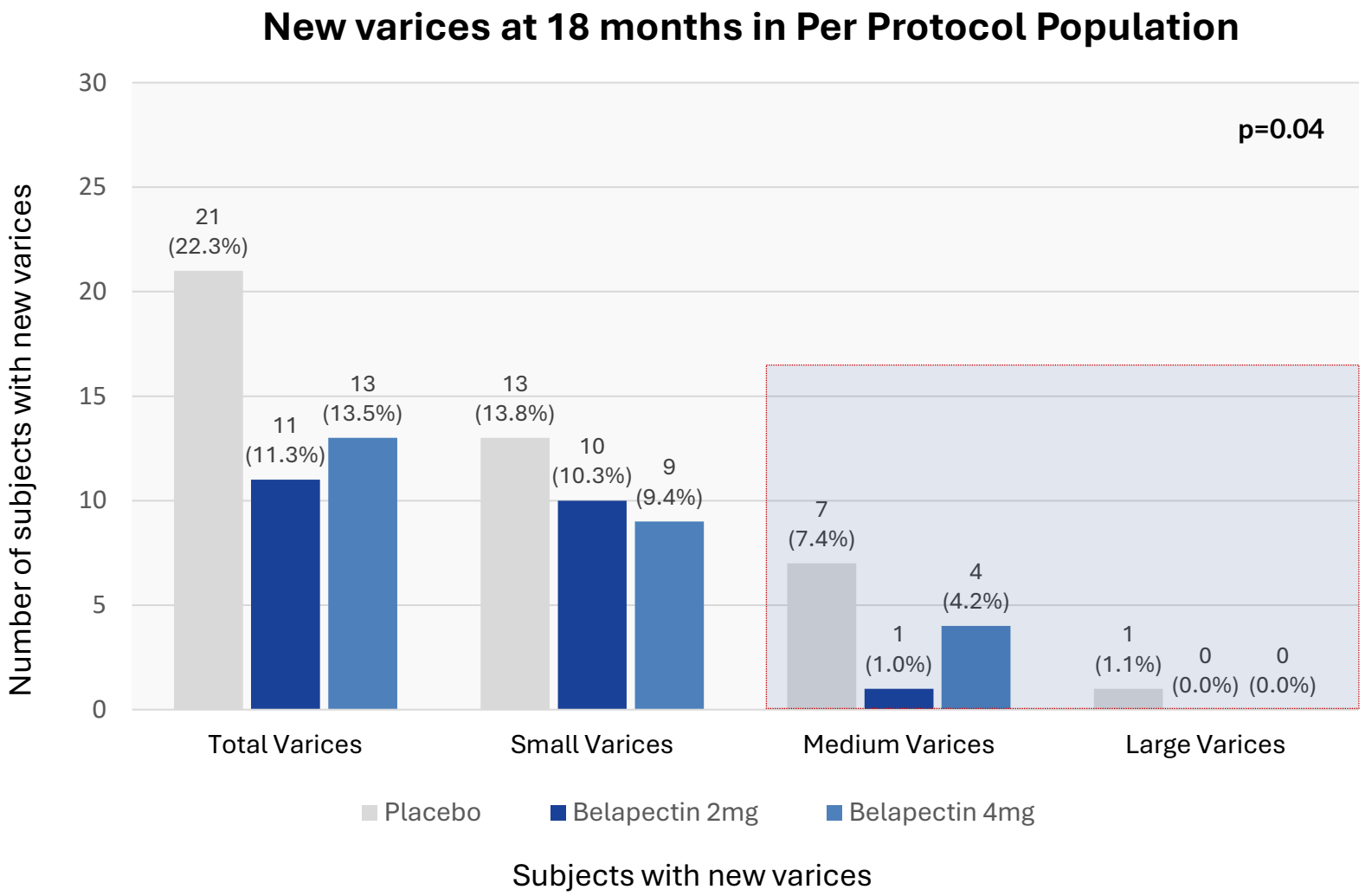
Key points

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.

NAVIGATE: Incidence of Varices by Size at 18-months



Key points

Placebo Treatment Group: N = 94

2mg/kg Belapectin Treatment Group: N = 97

4mg/kg Belapectin Treatment Group: N = 96

Varices grade definition

- Large > 5 mm in diameter, occupying more than 1/3 of esophageal lumen
- Medium >5 mm in diameter, occupying less than 1/3 of esophageal lumen
- Small <5 mm in diameter, minimally elevated above esophageal mucosa.

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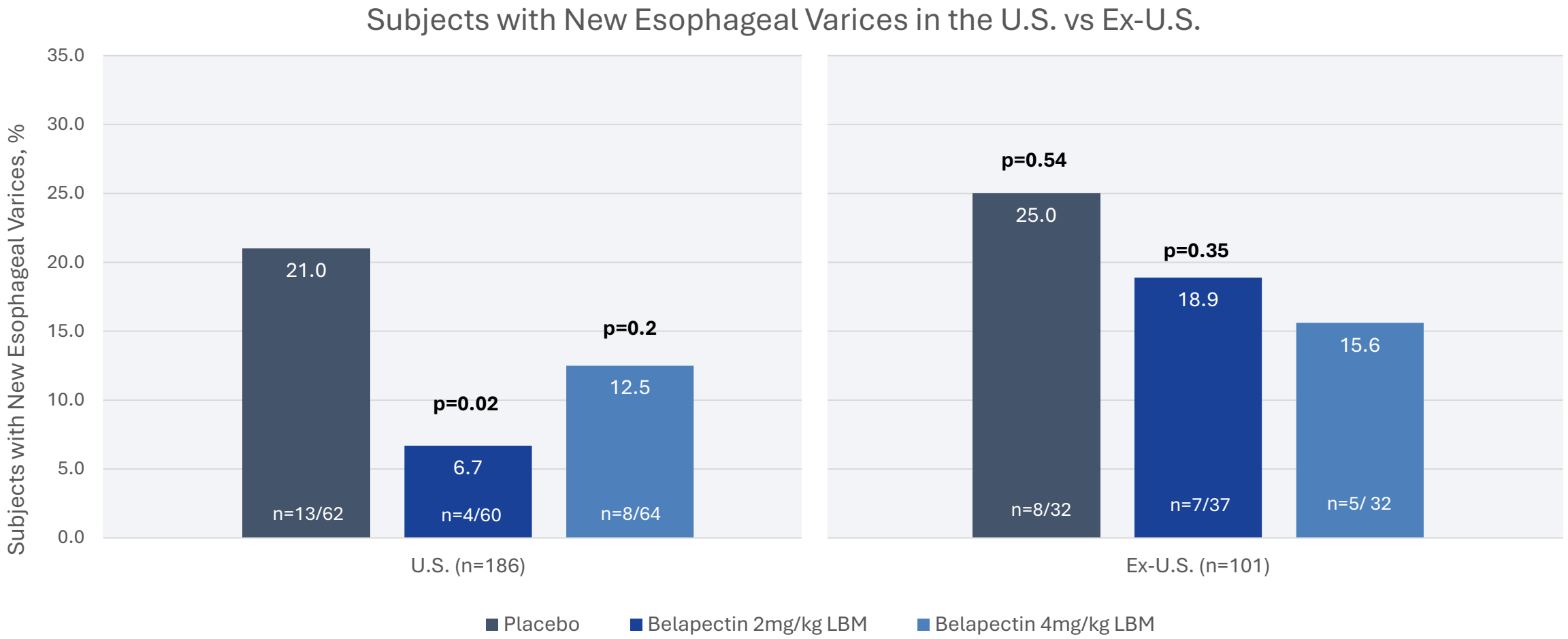
Incidence of New Varices was Significantly Lower in Patients in the U.S.

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

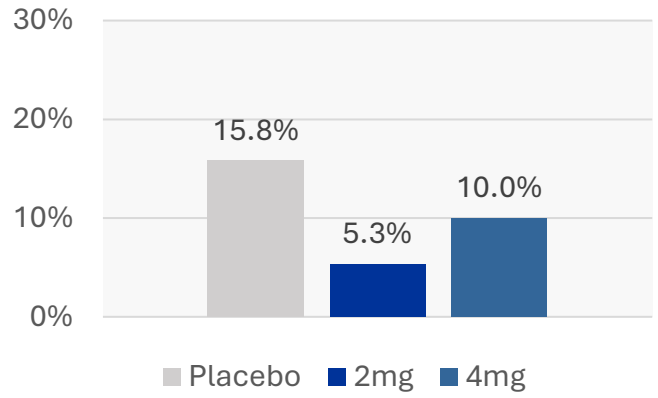
		Treatment Group			
		Placebo	Belapectin 2mg/kg LBM	Belapectin 4mg/kg LBM	Total
U.S.		(N=62)	(N=60)	(N=64)	(N=186)
	Concomitant Use of GLP-1 n (%)	**28 (45.2%)**	**22 (36.7%)**	**18 (28.1%)**	**68 (36.6%)**
	Concomitant Use of NSBBs n (%)	5 (7.9%)	3 (5.0%)	3 (4.6%)	11 (5.9%)
	Concomitant Use of Statins 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%)
EX-U.S.		(N=32)	(N=37)	(N=32)	(N=101)
	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

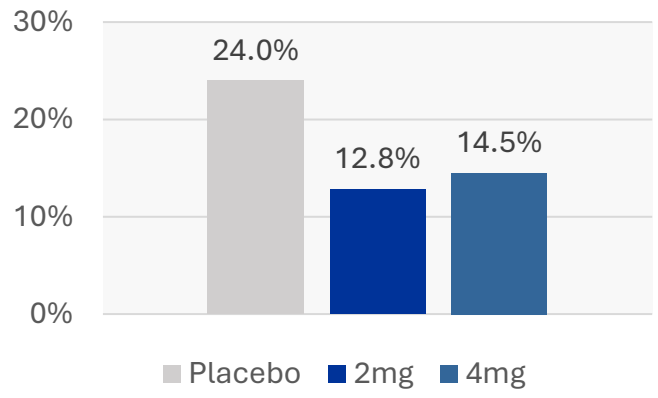
	Placebo	2mg/kg LBM	4mg/kg LBM	Total
Yes	18	19	20	**57**
No	75	78	76	**229**

New Varices at 18 months

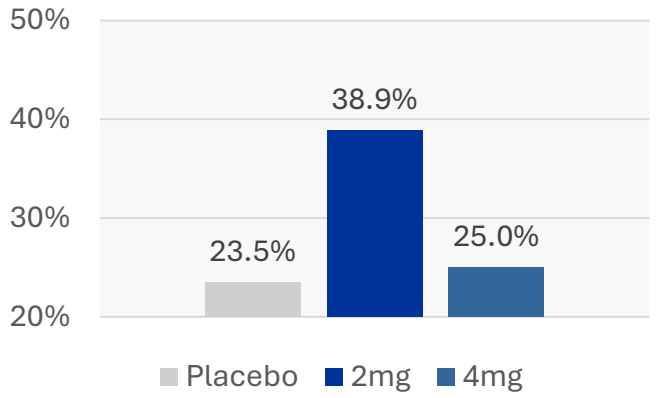
YES (n=57)

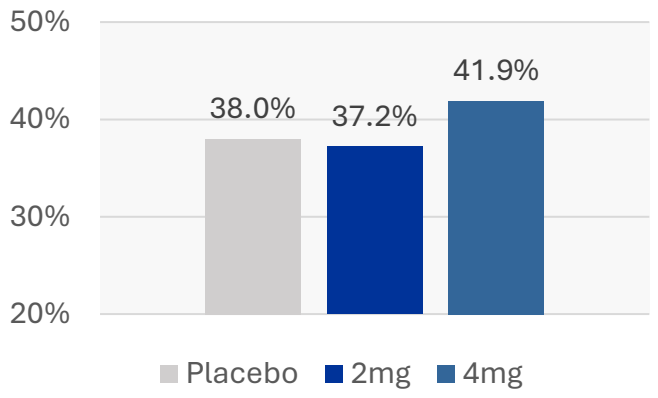


NO (n=229)

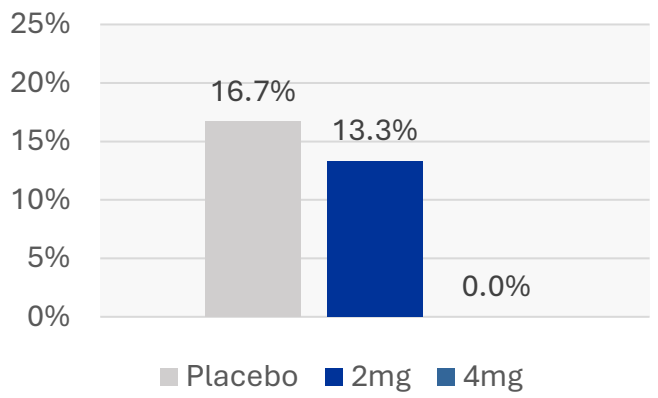


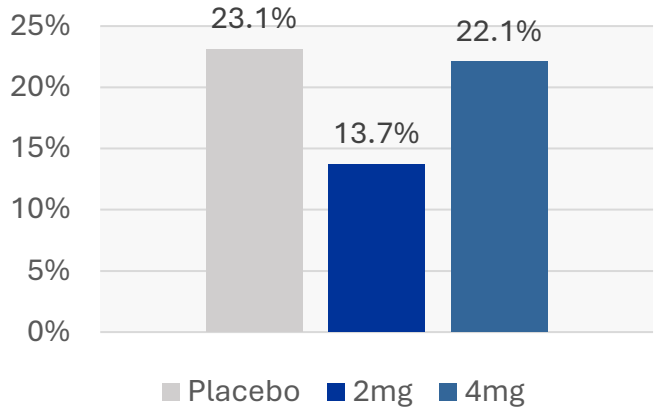
≥ 0.5pt ELF Increase





≥ 5 kPa LSM Increase





Per-Protocol (n = 287)

Liver Related Outcomes/MACE at 18 months

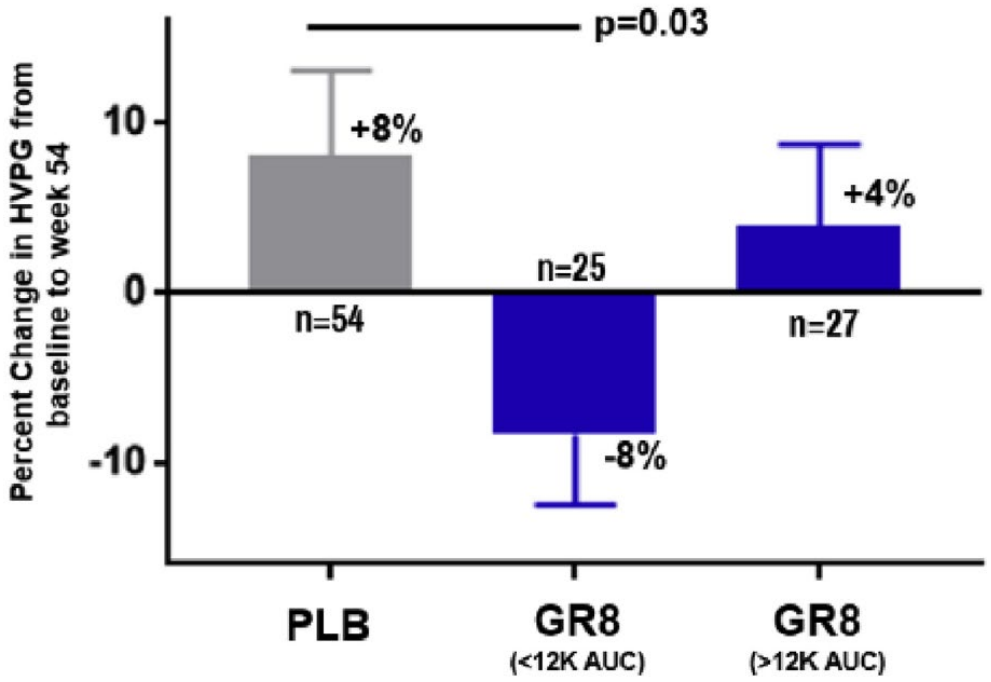
Per Protocol population

		Belapectin	
	Placebo (N = 95) n (%)	2mg/kg LBM (N = 97) n (%)	4mg/kg LBM (N = 98) n (%)
Subjects with Composite Clinical Outcomes, n (%)	**4 (4.2)**	**3 (3.1)**	**7 (7.1)**
Varices (Esophageal or Gastric) Requiring Treatment	3 (3.2)	3 (3.1)	3 (3.1)
Variceal Bleed Requiring Hospitalization	0	0	0
Clinically Significant Ascites Requiring Hospitalization	0	0	0
Spontaneous Bacterial Peritonitis	0	0	0
Overt Hepatic Encephalopathy (West Haven Score ≥2 and Requiring Hospitalization)	0	0	1 (1.0)
Liver Transplant	0	0	0
Model End Stage Liver Disease (MELD) Score ≥ 15	0	0	1 (1.0)
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

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 pharmacodynamic (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



Chalasani et al. Gastroenterology 2020; 158: 1334-1345

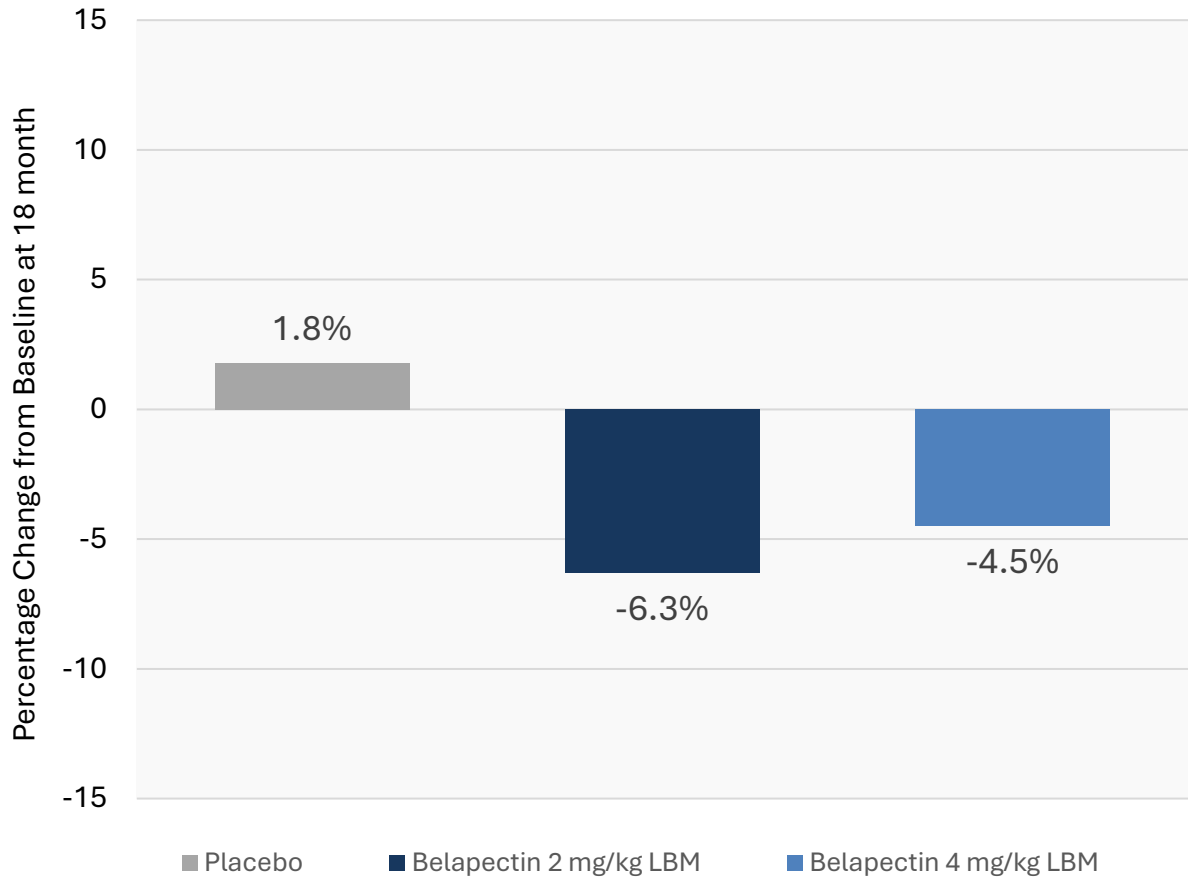
Reference Guide: Biomarkers and Non-Invasive Scores in MASH Cirrhosis

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

NAVIGATE: Improvement in LSM - Baseline to 18 months Full Analysis Set-FAS

ITT Population (n: 315)

		Belapectin	
	Placebo (N=102)	2mg/kg LBM (N=107)	4mg/kg LBM (N=106)
Baseline LSM Value (kPA)			
Mean (SD)	23.6 (11.44)	25.1 (15.04)	25.8 (12.91)
Median	22.5	21.8	23.6
18-month LSM Value (kPa)			
Mean (SD)	22.7 (13.71)	21.1 (12.88)	22.9 (13.40)
Change from Baseline in LSM Value (kPa) @ 18 months			
Mean (SD)	-0.6 (11.38)	-2.9 (11.61)	-2.2 (10.54)
% Change from Baseline @ 18 months LSM Value (kPa) *			
Mean %	1.8 (47.26)	-6.3 (39.13)	-4.5 (37.30)



Liver Stiffness kPa mean change %

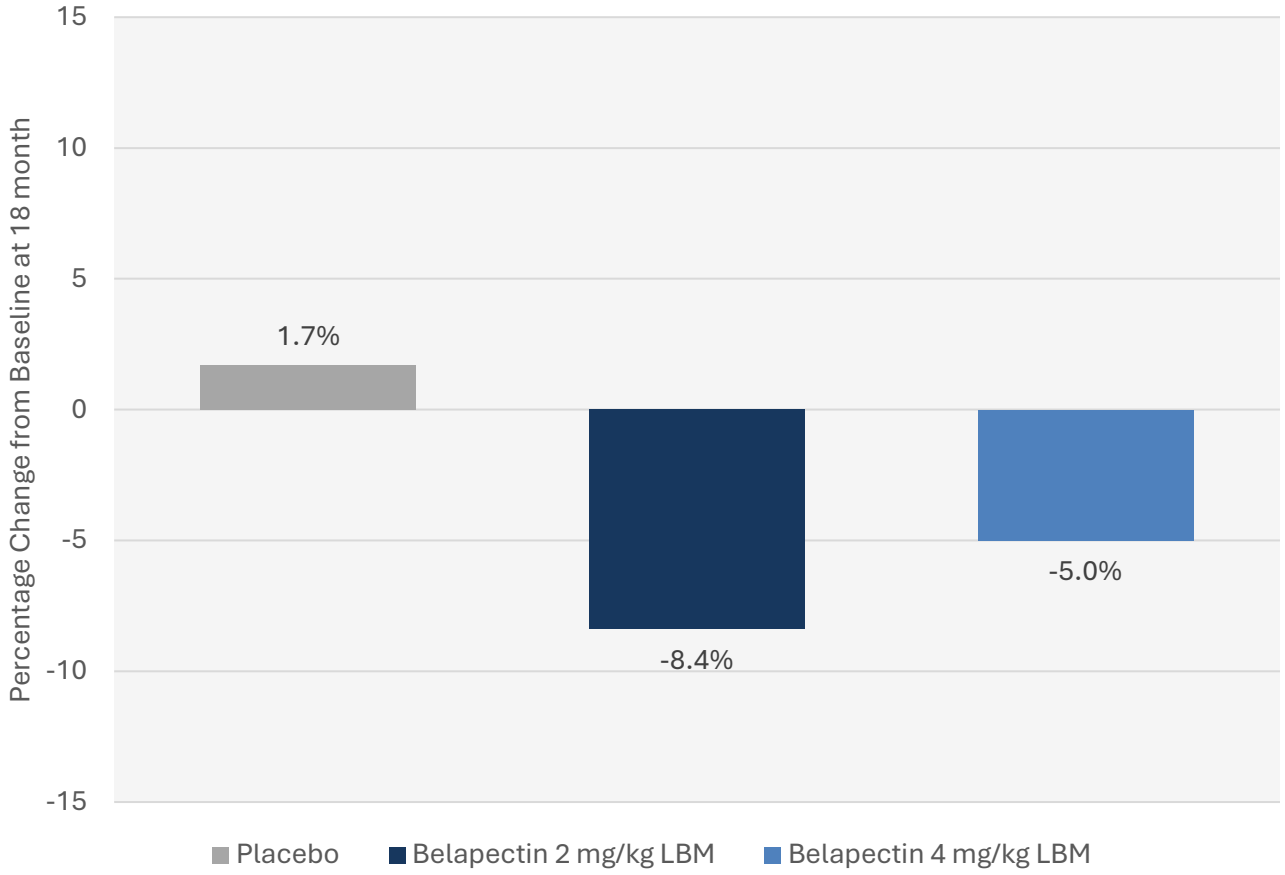
Galectin Therapeutics

* Calculated percentage change for each subject

NAVIGATE: Improvement in LSM - Baseline to 18 months
Per-Protocol– *Subjects with all available valid LSM*

Per-Protocol (Completers n: 234)

		Belapectin	
	Placebo (N=76)	2mg/kg LBM (N=81)	4mg/kg LBM (N=77)
Baseline LSM Value (kPA)			
Mean (SD)	22.6 (10.31)	24.6 (13.71)	25.7 (12.26)
Median	22.4	21.8	23.4
18-month LSM Value (kPa)			
Mean (SD)	21.9 (12.54)	21.4 (13.32)	23.4 (13.85)
Change from Baseline in LSM Value (kPa) @ 18 months			
Mean (SD)	-0.7 (10.71)	-3.2 (12.05)	-2.4 (10.90)
% Change from Baseline @ 18 months LSM Value (kPa) *			
Mean %	1.7 (46.78)	-8.4 (38.33)	-5.0 (38.05)

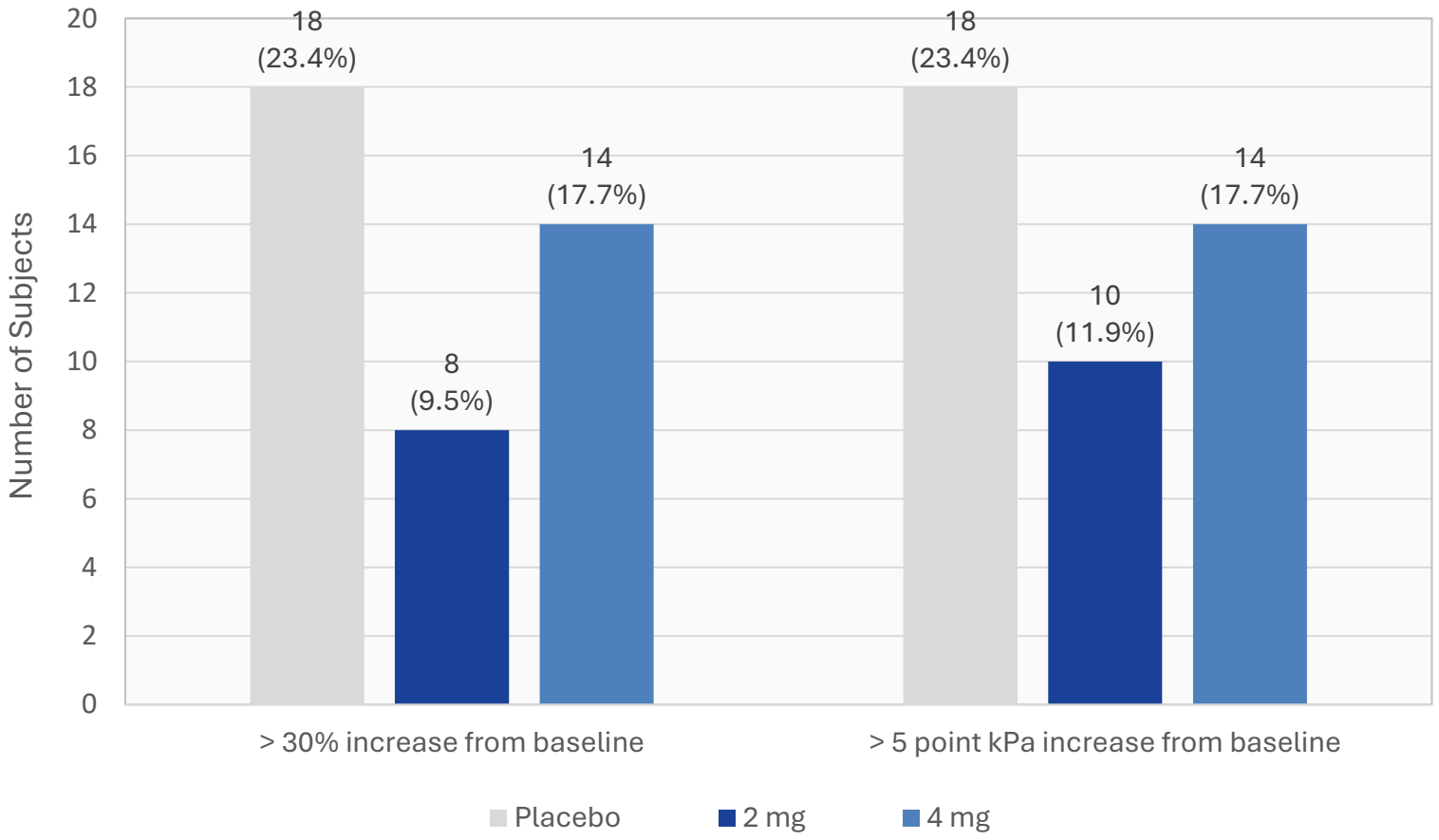


Liver Stiffness kPa mean change %

* Calculated percentage change for each subject

Fewer Subjects Showed Worsening in Liver Stiffness Measure - LSM (kPa)

FAS with all available data (n = 240)



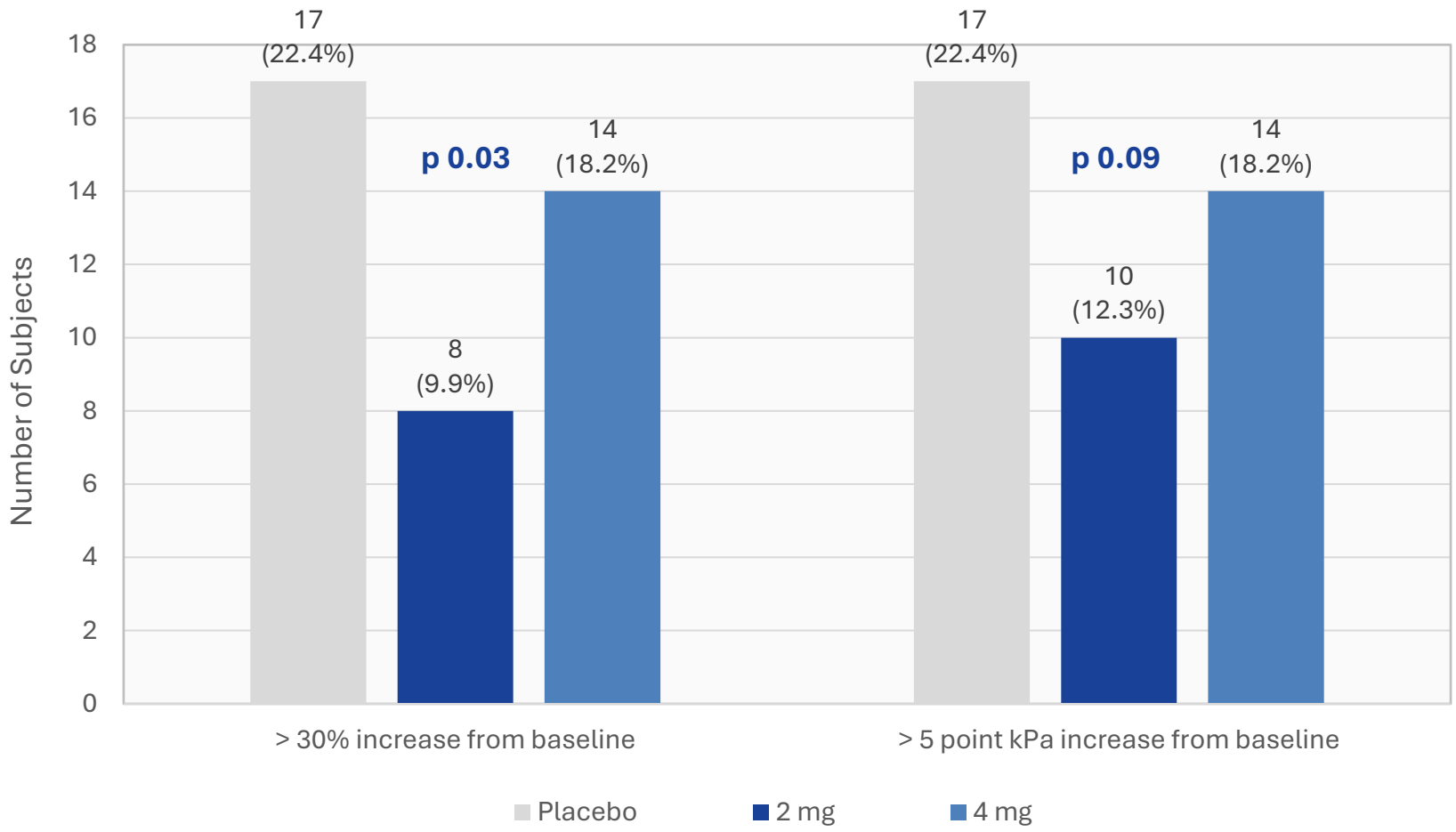
Belapectin

N	Placebo	2mg/kg LBM	4mg/kg LBM	Total
	77	84	79	**240**

Gawrieh S. et al. JHep 2024:81.600-608

Fewer Subjects Showed Worsening in Liver Stiffness Measure - LSM (kPa)

Per-Protocol with all LSM (n = 234)



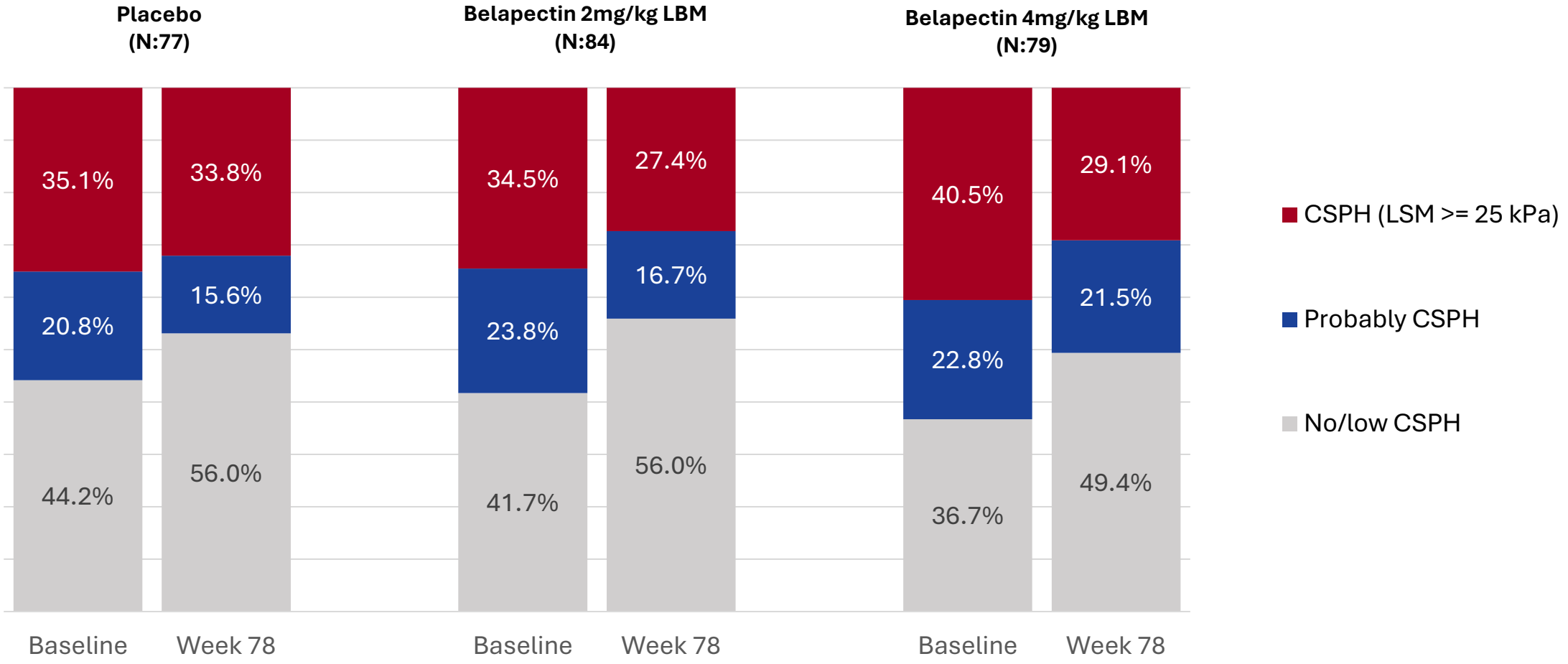
Belapectin

	Placebo	2mg/kg LBM	4mg/kg LBM	Total
N	76	81	77	**234**

Gawrieh S. et al. JHep 2024:81.600-608

Belapectin Led to Improvement in Portal Hypertension Risk Category

Full Analysis Set- **FAS** (n :240)

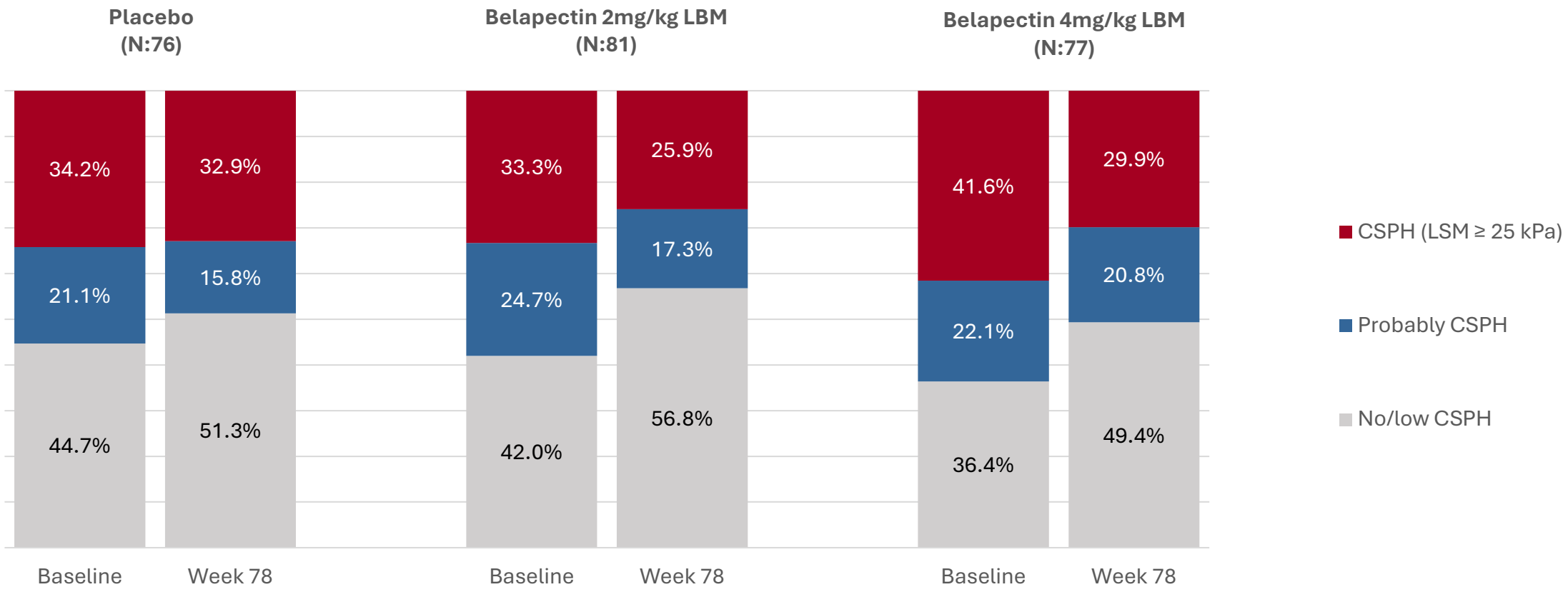


Probable CSPH LSM ≥20 & platelet <150 or LSM 15-20 & platelet <110; no/low CSPH LSM≤ 15 and platelet≥ 150; Baveno VII Guidelines de Franchis 2022 Jhep

FAS with all available data

Belapectin Led to Improvement in Portal Hypertension Risk Category in Completer Population

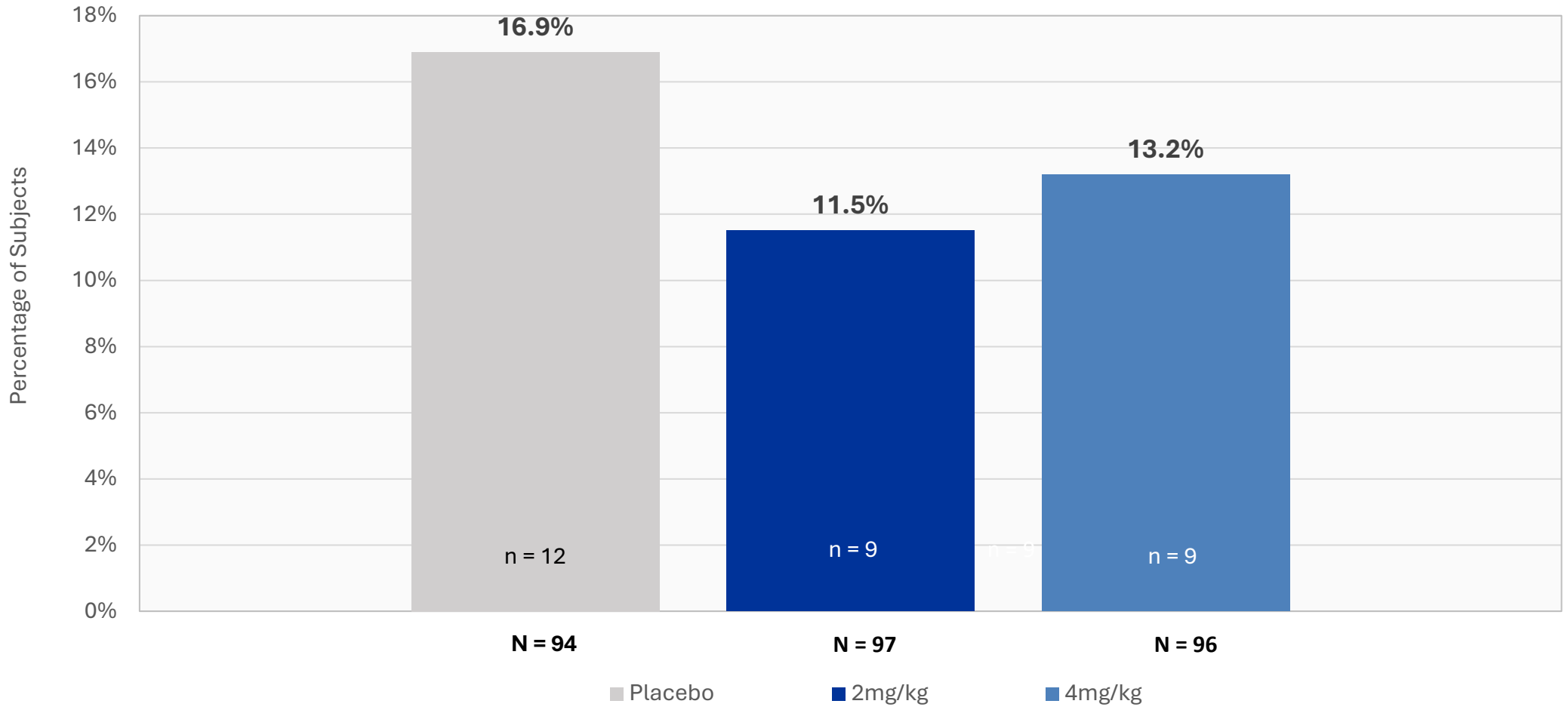
Per Protocol Population (n :234)



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
PP with all available data

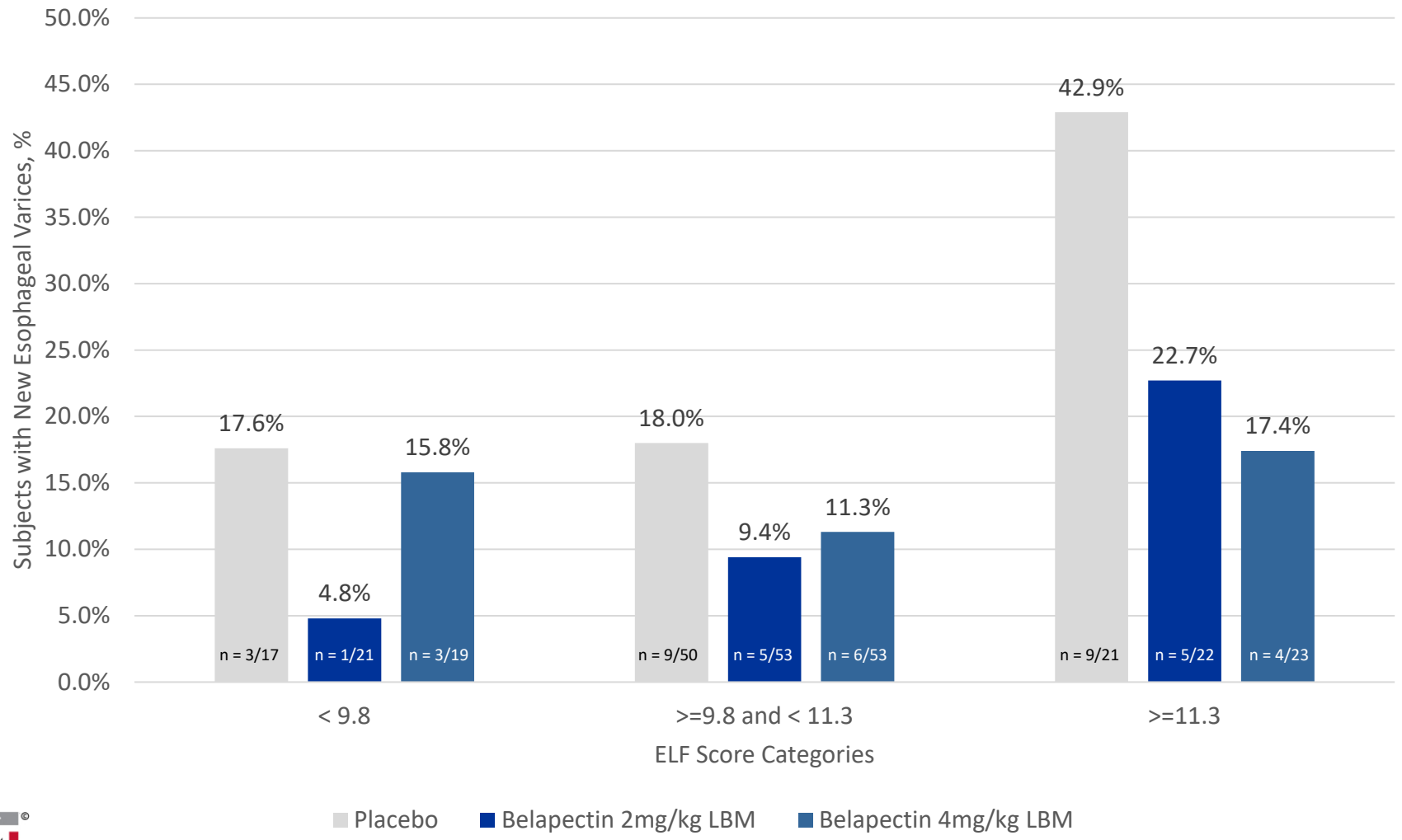
Increase in AGILE-4 score ≥ 20%

Per protocol population n= 287



Incidence of Varices at 18 Months by Baseline ELF Categories

Per protocol population (n = 279)



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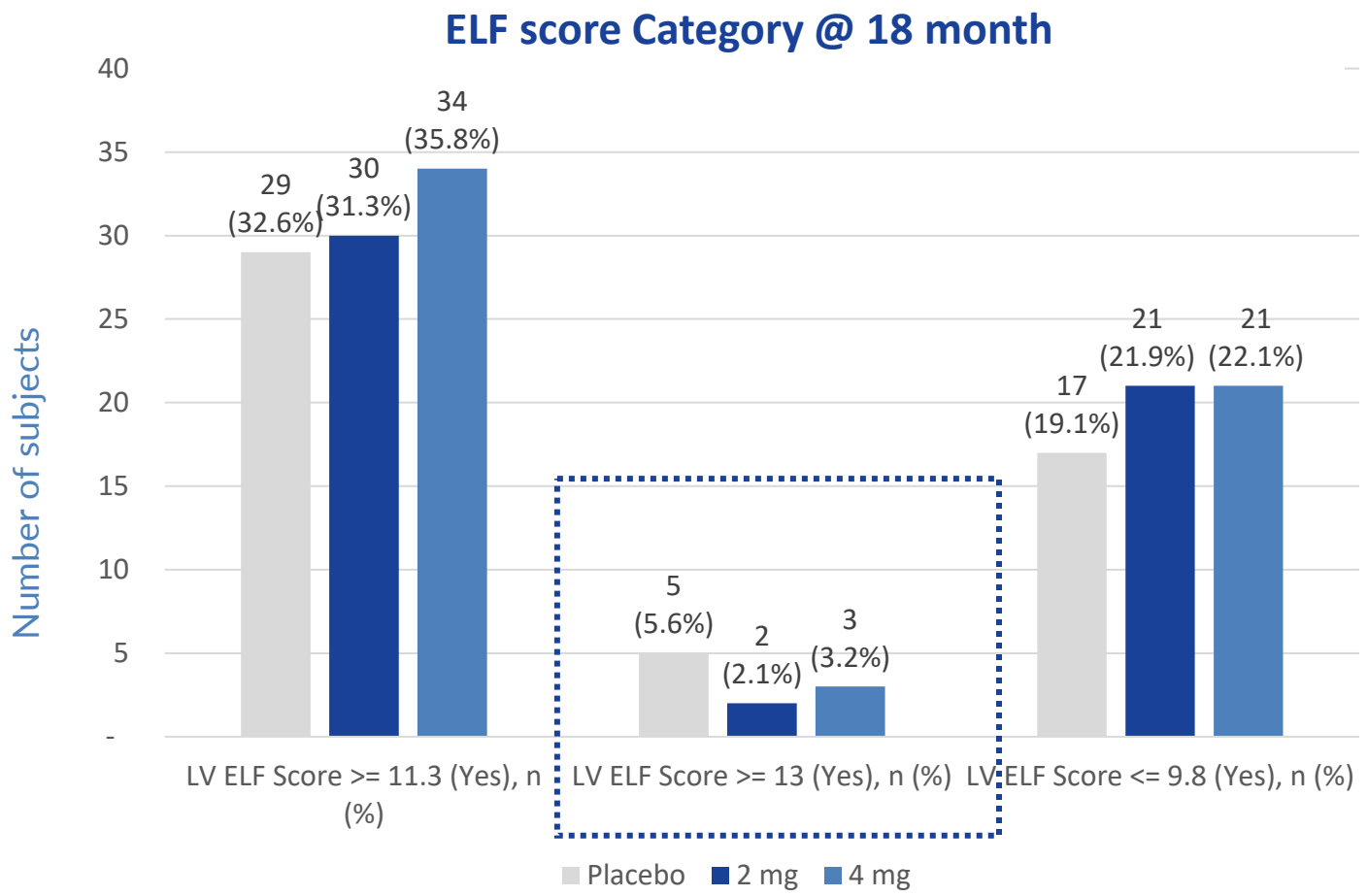
Fewer Subjects Progressed to High-Risk ELF Category (ELF ≥13)

Per-Protocol (n=280)



Key points

Baseline ELF Value	Placebo	Belapectin 2mg/kg LBM	Belapectin 4mg/kg LBM
N	**89**	**96**	**95**
Mean	10.67	10.54	10.59
SD	1.16	0.96	1.04

ELF Enhanced Liver Fibrosis Score- combined for HA, PIIINP and TIMP-1

ELF: Risk of disease progression. < 9.8 Low risk, ≥11.3 mid risk, highest risk ≥13

Baseline to 18 months Per-Protocol (n=280)

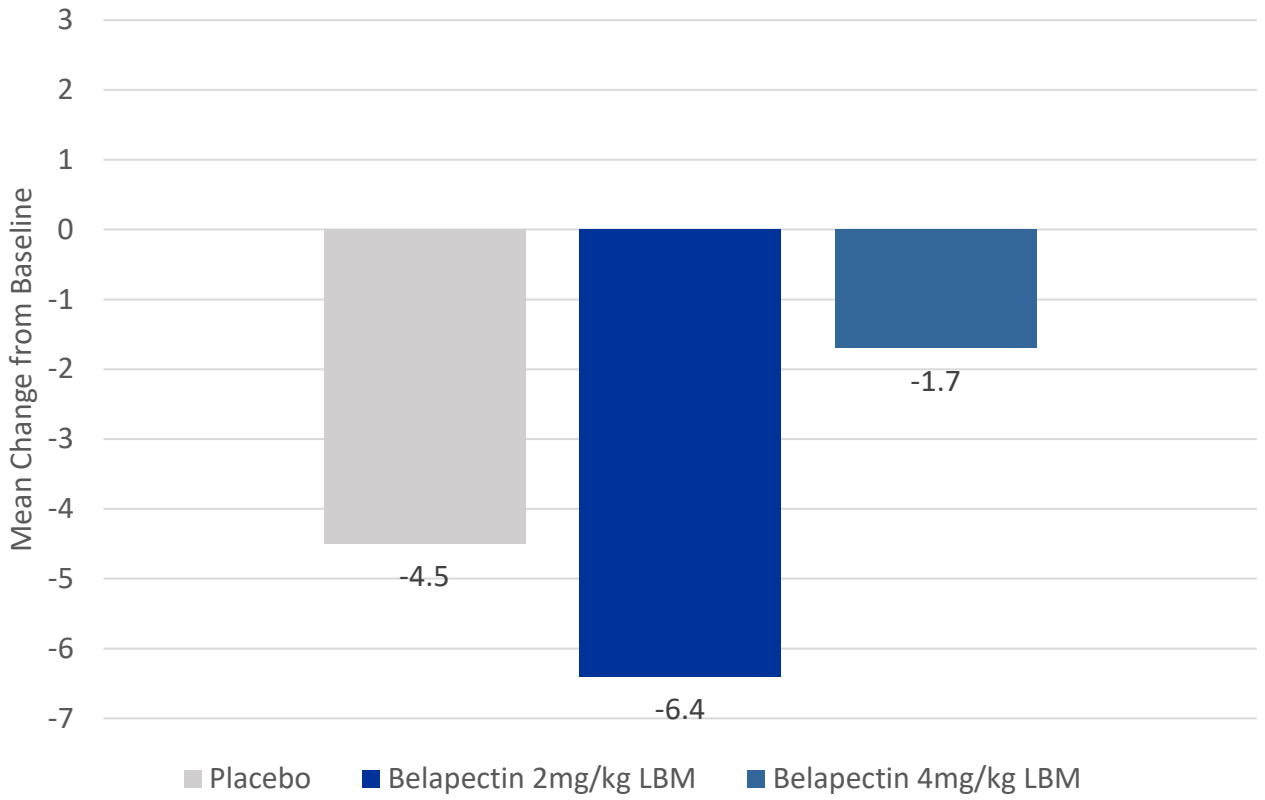
Galectin Therapeutics

Enhanced liver fibrosis (ELF) score predicts hepatic decompensation and mortality. Pearson M, et al JHEP Rep. 2024 Mar 11;6(6):101062.

NAVIGATE- Pro-C3 change from Baseline to 18 months

Per Protocol Population (n: 243)

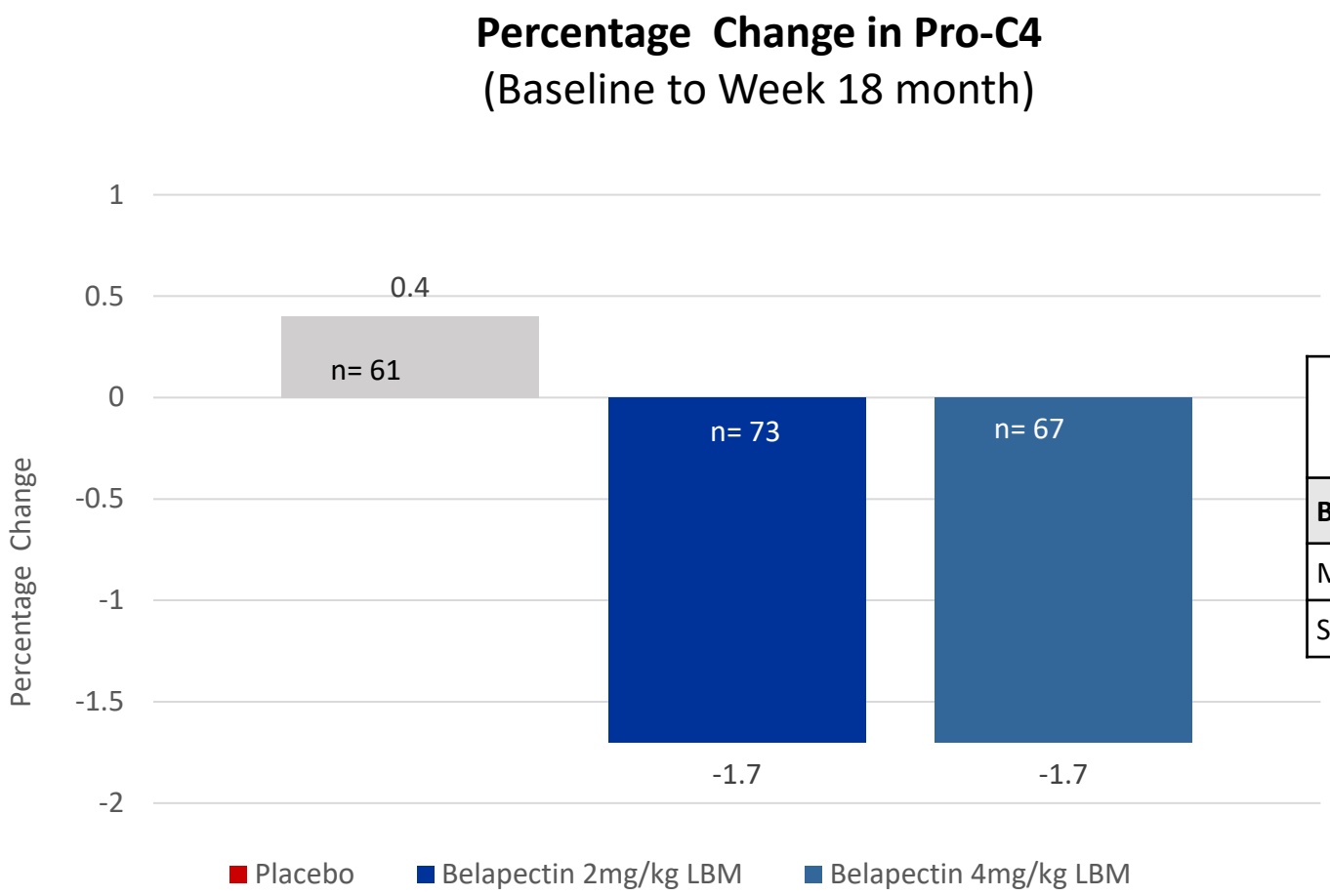
		Belapectin	
	Placebo (N=79)	2mg/kg LBM (N=81)	4mg/kg LBM (N=83)
Baseline			
Mean	50.19	45.91	43.39
Standard Deviation	38.45	31.58	17.42



Per protocol population with all available Pro-C3 data

NAVIGATE- Pro-C4 change at 18 month

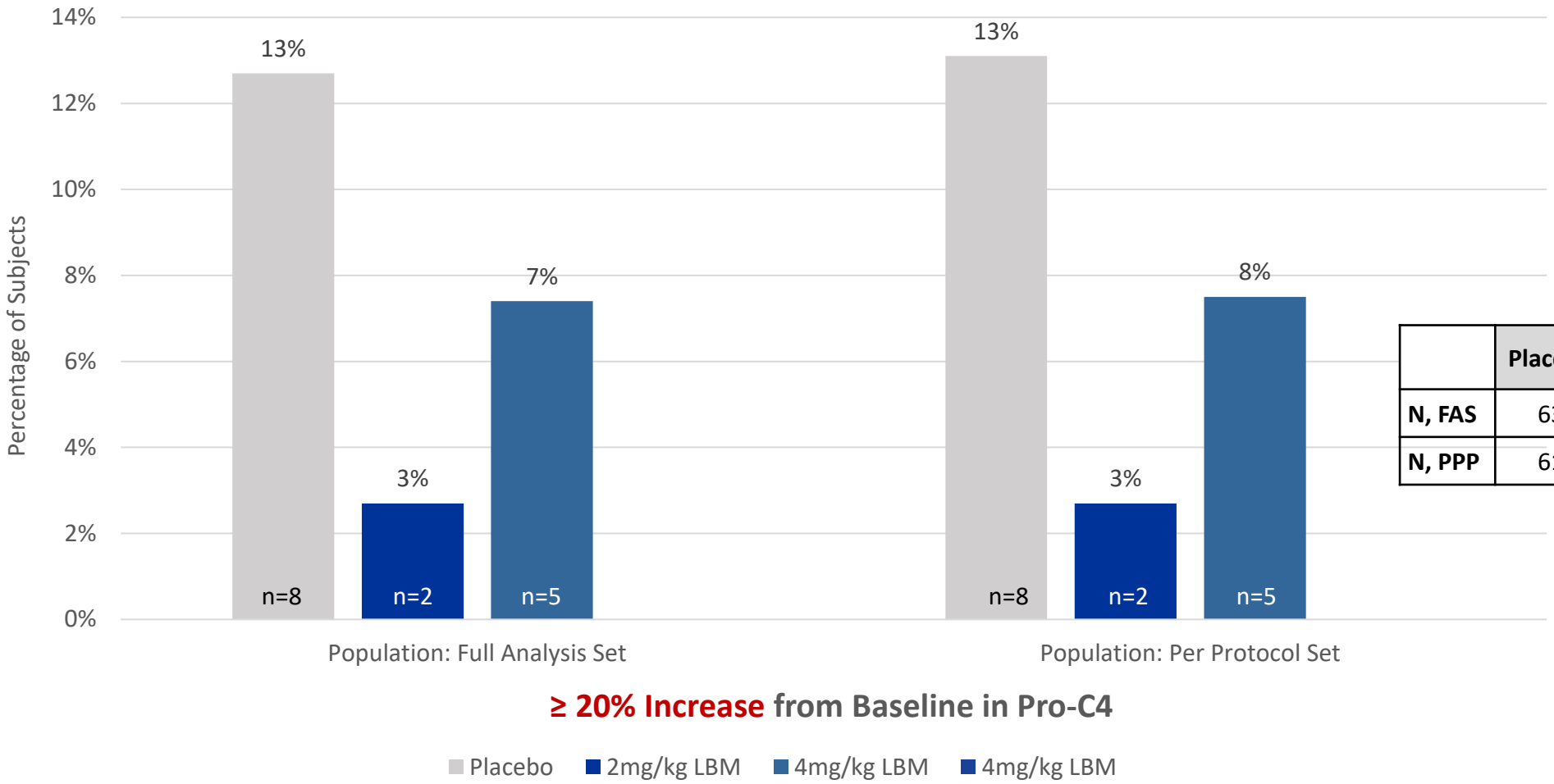
Per Protocol Set (n: 201)



	Placebo (N=61)	Belapectin 2mg/kg LBM (N=73)	Belapectin 4mg/kg LBM (N=67)
Baseline			
Mean	8.6	8.6	8.6
Standard Deviation	0.21	0.20	0.23

NAVIGATE: Change in Pro-C4 at 18 Month

Full Analysis Population (n :204) | Per Protocol Population (n :201)



Belapectin

	Placebo	2mg/kg LBM	4mg/kg LBM	Total
N, FAS	63	73	68	**204**
N, PPP	61	73	67	**201**

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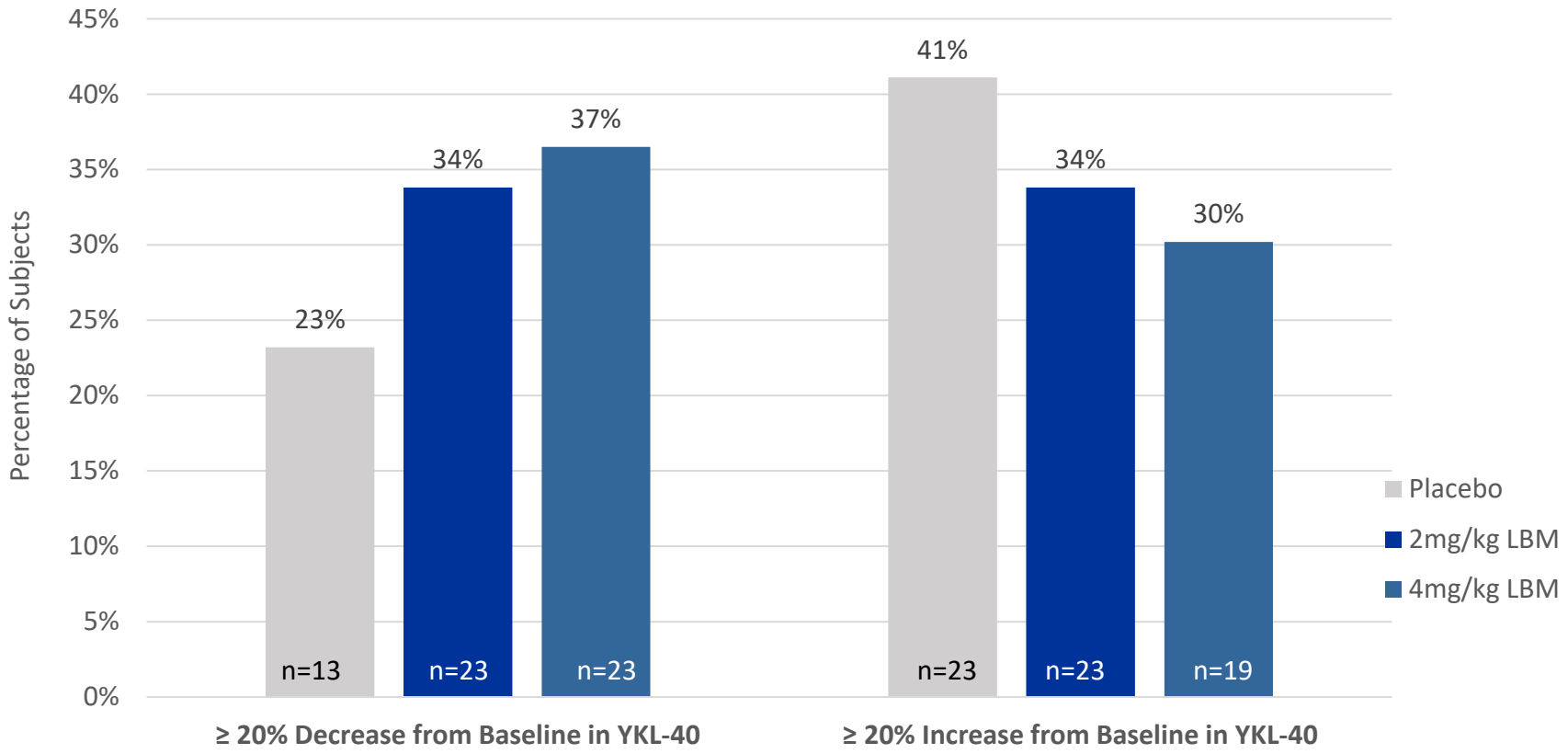
Percentage Change from Baseline in YKL-40

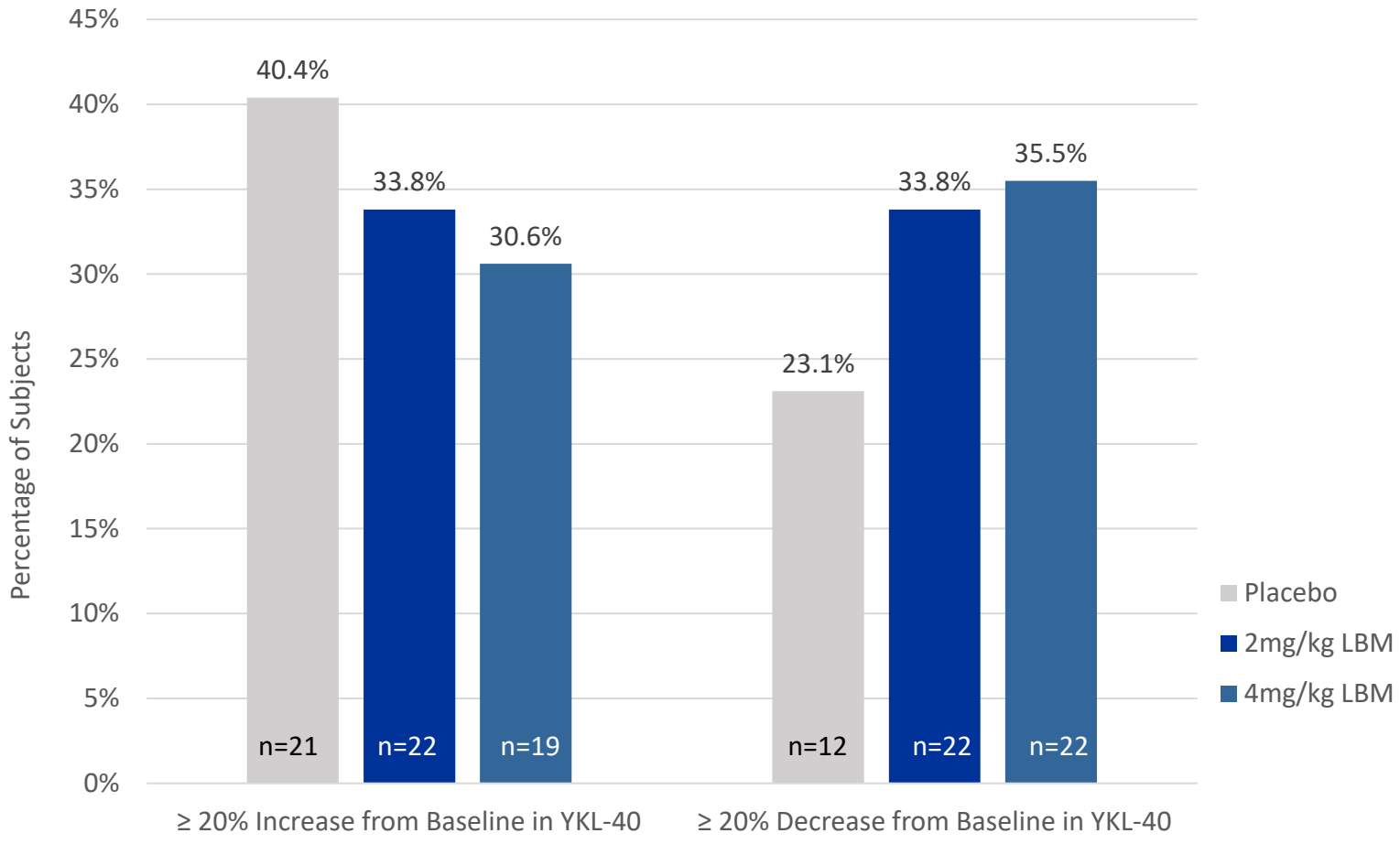
Full Analysis Set (n = 187)



		Belapectin	
	Placebo	2mg/kg LBM	4mg/kg LBM
YKL-40 at Baseline	56.0	68.0	63.0
Mean	5.14	5.02	5.00
Standard Deviation	0.92	0.82	0.78

Percentage Change from Baseline in YKL-40

Per Protocol Set (n = 179)

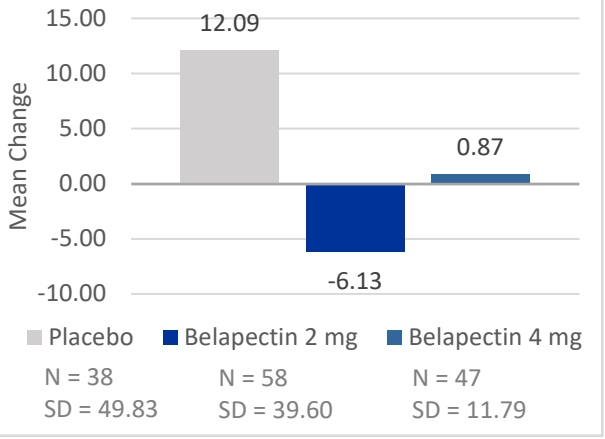


		Belapectin	
	Placebo	2mg/kg LBM	4mg/kg LBM
YKL-40 at Baseline	52.0	65.0	62.0
Mean	5.11	4.95	4.92
Standard Deviation	0.95	0.94	0.74

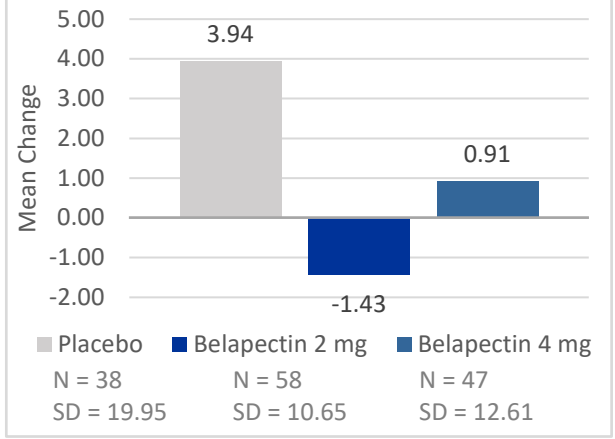
Belapectin consistently reduced Pro-Inflammatory Markers at 18 months

Pro-Inflammatory

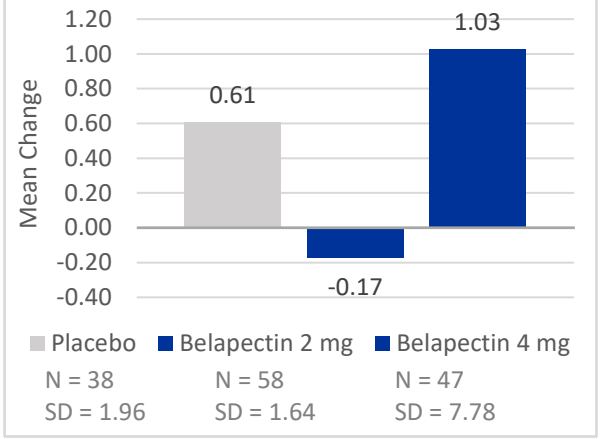
TNF-alpha



IL-1b

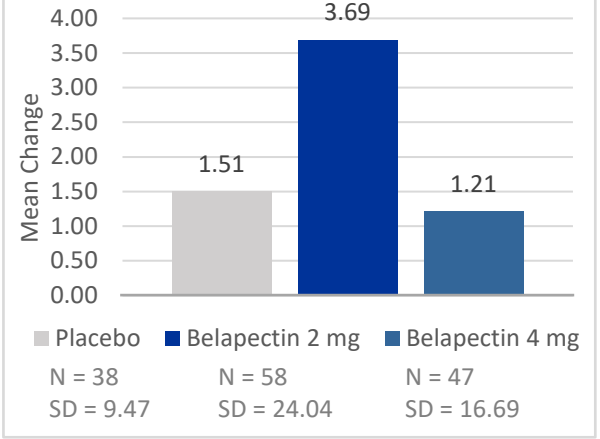


IL-6

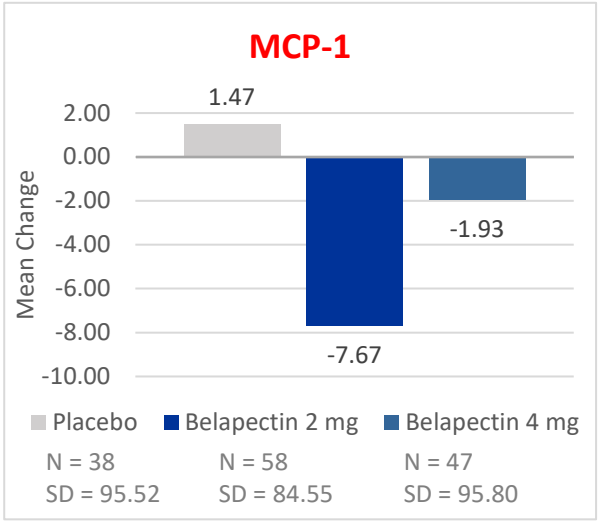


Anti-inflammatory

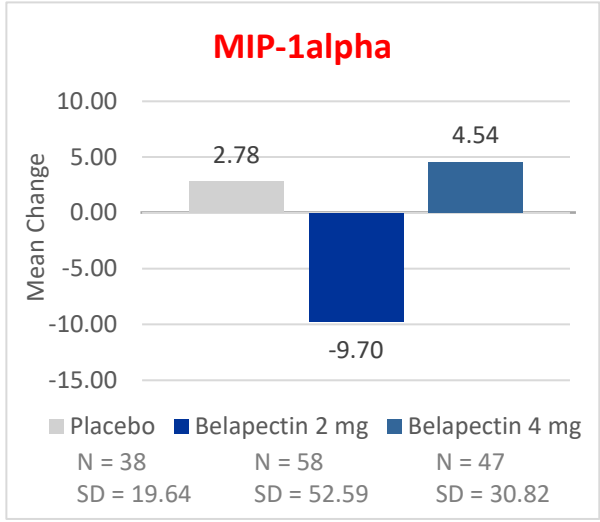
IL-10



MCP-1



MIP-1alpha



FAS population N: 143

Galectin Therapeutics

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

Subjects Clinical Outcomes or MACE at 18 months

Per Protocol population	Placebo (N = 95) n (%)	Belapectin 2mg/kg LBM (N = 97) n (%)	Belapectin 4mg/kg LBM (N = 98) n (%)
Subjects with Composite Clinical Outcomes, n (%)	**4 (4.2)**	**3 (3.1)**	**7 (7.1)**
Varices (Esophageal or Gastric) Requiring Treatment	3 (3.2)	3 (3.1)	3 (3.1)
Variceal Bleed Requiring Hospitalization	0	0	0
Clinically Significant Ascites Requiring Hospitalization	0	0	0
Spontaneous Bacterial Peritonitis	0	0	0
Overt Hepatic Encephalopathy (West Haven Score ≥2 and Requiring Hospitalization)	0	0	1 (1.0)
Liver Transplant	0	0	0
Model End Stage Liver Disease (MELD) Score ≥ 15	0	0	1 (1.0)
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

Safety Summary

Adverse Events

- **Discontinuation of the study due to Adverse Events** was similar across 3 cohorts:
  - 7 (5.9%) in the Pbo
  - 5 (4.2%) in 2 mg/kg Belapectin
  - 8 (6.7%) in 4 mg/kg 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 Pbo
  - 116 (97.5%) in 2 mg/kg Belapectin
  - 116 (96.7%) in 4 mg/kg Belapectin

Treatment-Emergent Serious Adverse Events (TESAEs)

- Similar proportion of subjects reported **Treatment-Emergent Serious Adverse Events (TESAEs)** across 3 cohorts:
  - 23 (19.5%) in Pbo
  - 27 (22.7%) in 2 mg/kg Belapectin
  - 25 (20.8%) in 4 mg/kg Belapectin

Assessment of Results

- Belapectin 2 mg reduced varices incidence by 43.2% compared to placebo in the overall population(ITT); results were not statistically significant.

- In the per-protocol population/completer (18-month treatment + end-of-treatment EGD), the reduction was **48.9%.**
  - Initial sample size assumed **52.5**% lower varices incidence with Belapectin vs. placebo.
  - Per-protocol population definition (18-month treatment + EGD) parallels completer biopsy definition in MASH trials.

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

- Less worsening in key markers of liver fibrosis marker provide further confidence in reduction in varices endpoint

- Belapectin maintained a clean safety profile with low discontinuation rates and no drug-related serious adverse events.
- Likely reasons for not achieving statistical significance in ITT;

  - Fewer recorded varices than expected; mid-study sample size re-estimation based on composite endpoint, not varices.
  - Shorter treatment duration; primary analysis at 18 months instead of 36 months.
  - Higher dropout rate (18.3% observed vs. 10% expected), mostly during COVID and first 4 months.

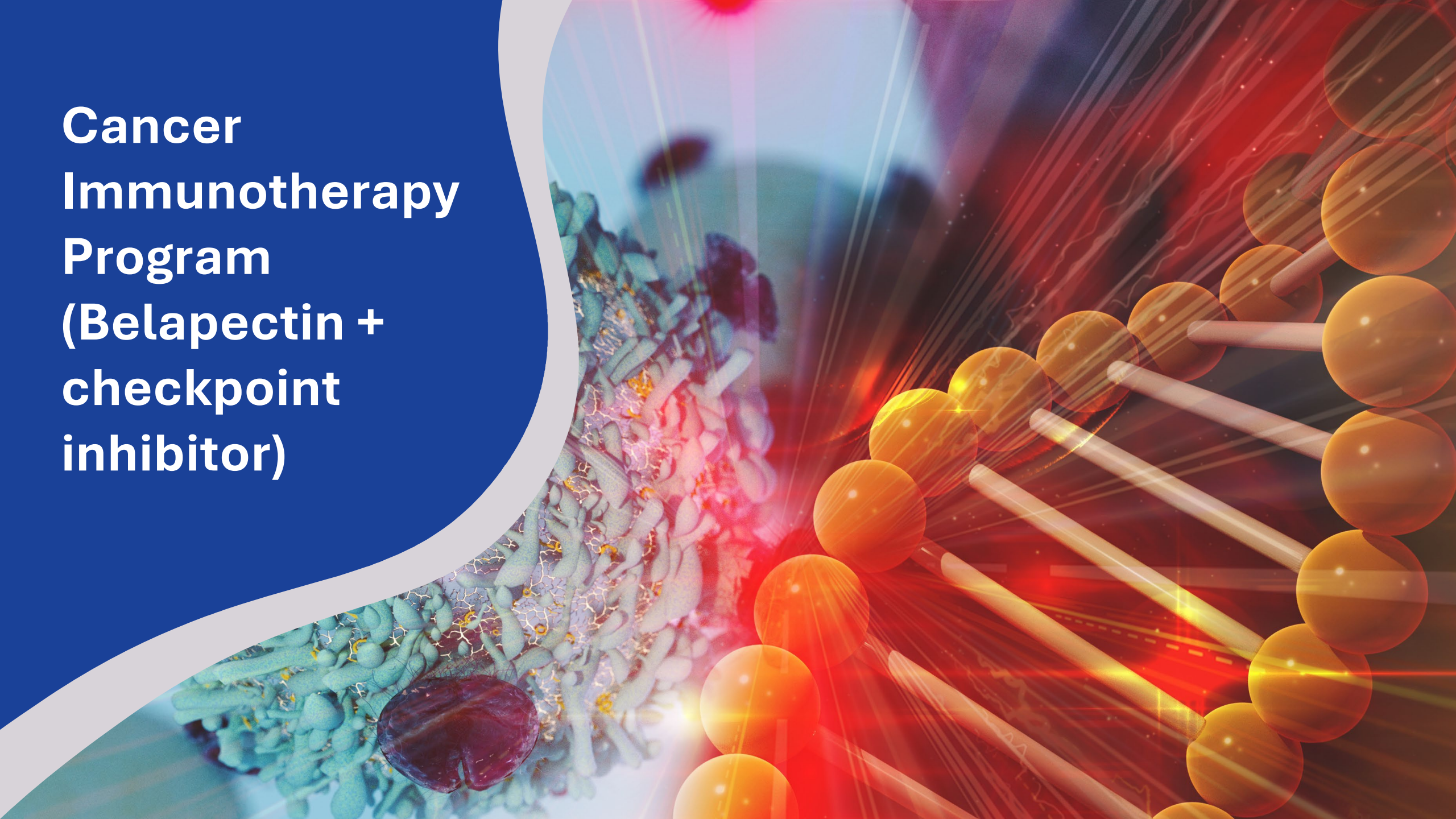
Key Takeaways and Next Steps

- NAVIGATE enrolled the most advanced patients of recent MASH trials, requiring both MASH cirrhosis and portal hypertension.
- A robust reduction in new varices was observed in both ITT and completer populations after 18 months of treatment.
- Non-invasive biomarkers results, including LSM and ELF, aligned with clinical outcomes and provide pharmacodynamic proof of effect.
- The 2 mg dose of Belapectin has demonstrated consistent, clinically meaningful effects across multiple trials*.
- The unique mechanism of Galectin-3 inhibition positions Belapectin as a differentiated and complementary candidate for MASH cirrhosis therapy.
- FDA feedback on NAVIGATE results planned by the year-end.
- Partnership opportunities are being actively pursued.

Galectin Therapeutics

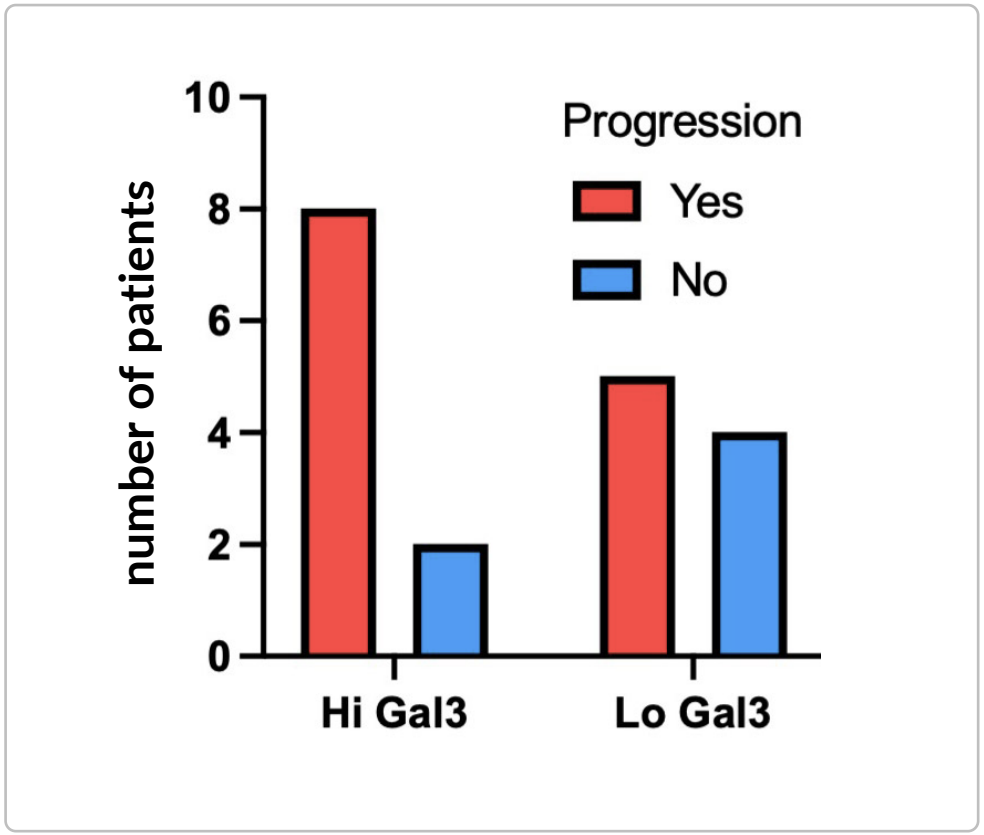
*Chalasani N, et al. *Gastroentrol.* 2020;158:1334-45

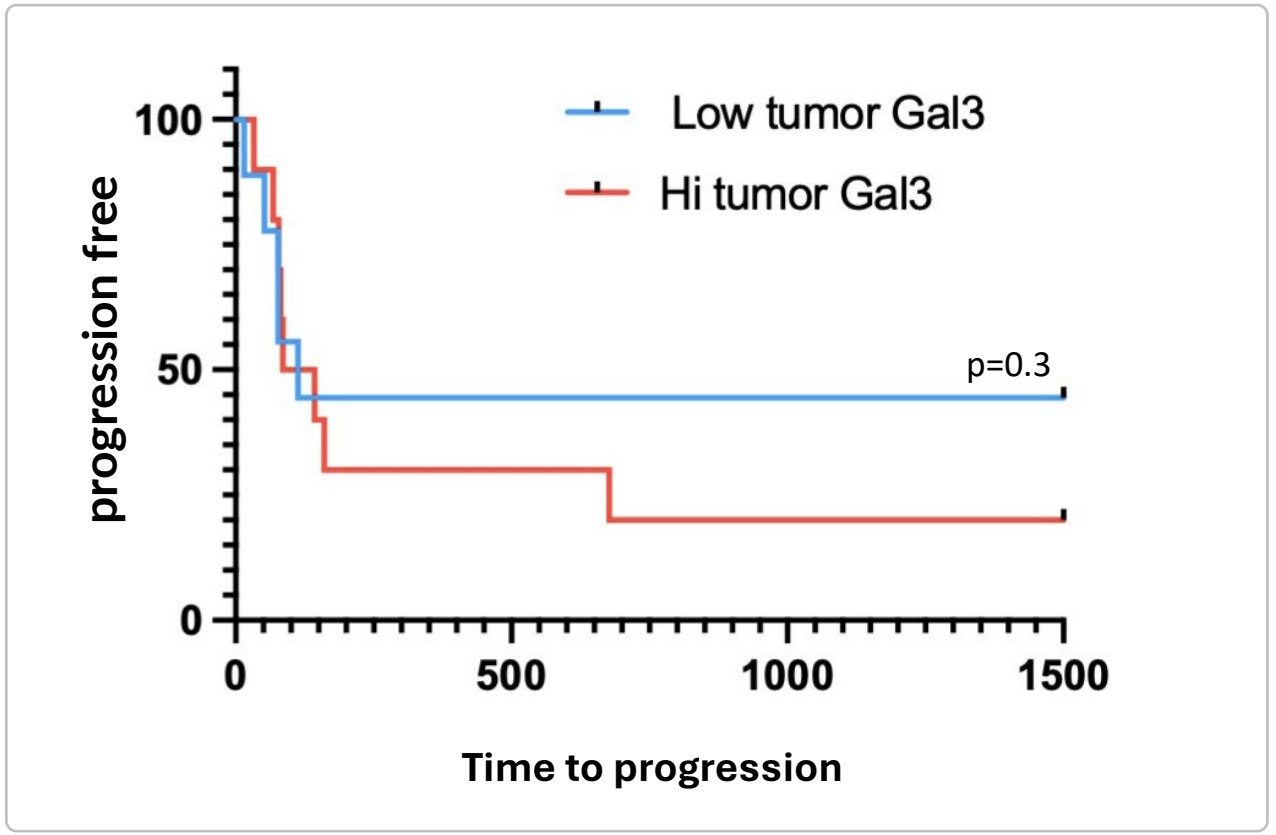
Cancer Immunotherapy Program (Belapectin + checkpoint inhibitor)



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

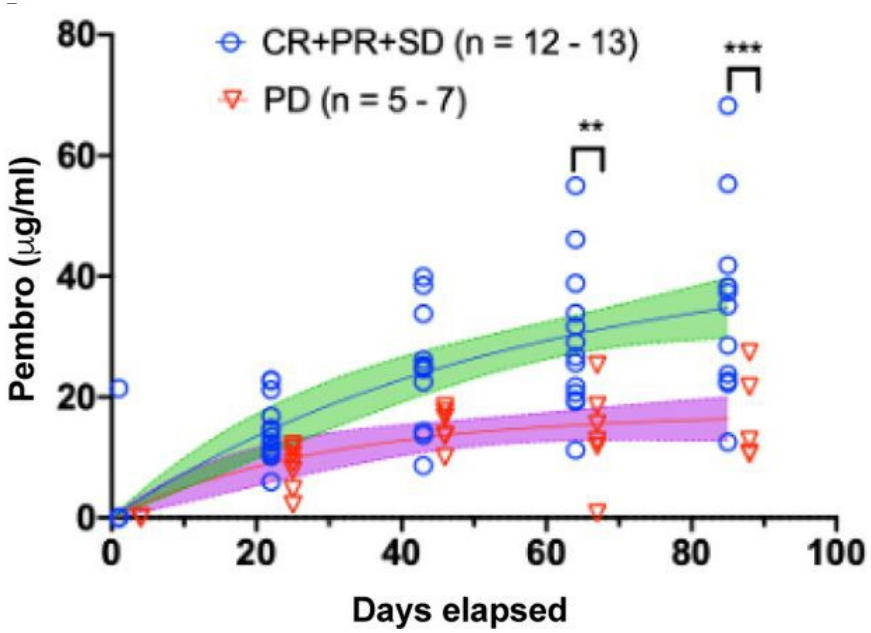




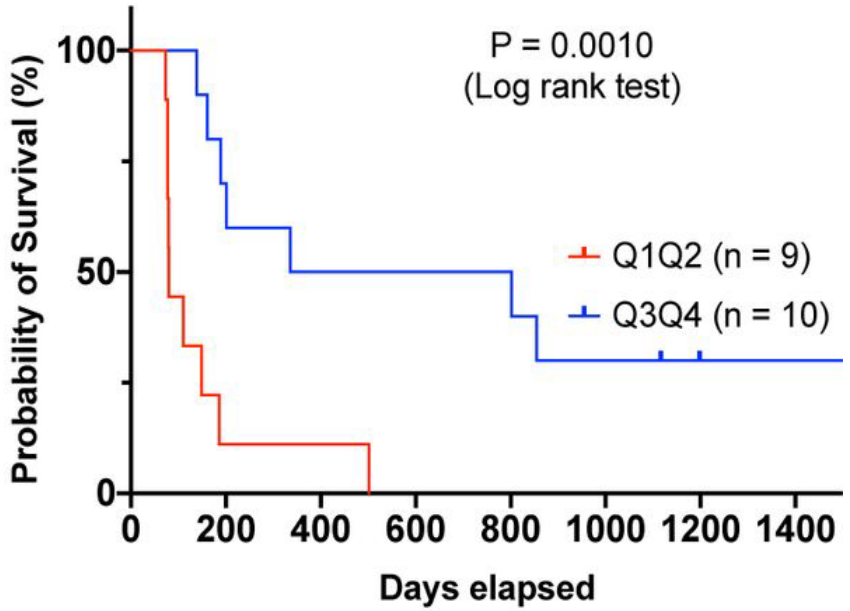
**p<0.01, ***p<0.001.
CR=complete response; HNSCC=head and neck squamous cell carcinoma; MM=metastatic melanoma; PD=progressive disease; PR=partial response; SD=stable disease.
1. Greisen SR, et al. *J Immunother Cancer*. 2024;12(10):e009952.

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.

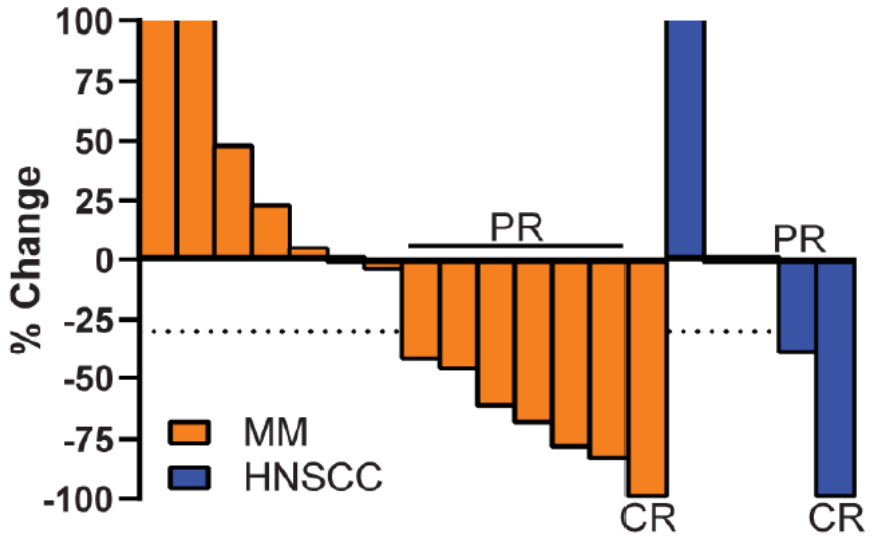
Belapectin in Combination with Pembrolizumab Showed Clinical Efficacy and Safety in Phase 11

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 $PD\text{-}1^{+}CD8^{+}$ 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.

Investment Highlights

Developing galectin-based 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

Low toxicity as a carbohydrate-based molecule which is degraded by natural processes

Patent protection through 2032

MASH Cirrhosis

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

Significant efficacy observed in cirrhotic patients without varices

Promising NAVIGATE results at 18 month read out, ≥40% reduction in new varices vs placebo in ITT; significantly lower incidence of new varices in per protocol population

Oncology (Combination Therapy)

Encouraging clinical response in difficult-to-treat cancers in combination with checkpoint inhibitor

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

Thank You!

Galectin Therapeutics