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

FORM 8-K

CURRENT REPORT
PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): April 16, 2018

GALECTIN THERAPEUTICS INC.
(Exact name of registrant as specified in its charter)

Nevada	**001-31791**	**04-3562325**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

4960 PEACHTREE INDUSTRIAL BOULEVARD, Ste 240
NORCROSS, GA 30071
(Address of principal executive office) (zip code)

Registrant's telephone number, including area code: (678) 620-3186

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

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

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

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

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

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).

Emerging growth company ☐

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

SECTION 7 – REGULATION FD

Item 7.01 Regulation FD Disclosure.

On April 16, 2018, Galectin Therapeutics Inc. (the "Company) posted to its website a presentation of results of its NASH-CX clinical trial attached hereto as Exhibit 99.1.

The information in this report is being furnished pursuant to this Item 7.01 and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, and it shall not be deemed incorporated by reference in any filing under the Securities Act of 1933 or under the Exchange Act, whether made before or after the date hereof, except as expressly set forth by specific reference in such filing to this report.

SECTION 8 – OTHER ITEMS

Item 8.01 Other Items.

On April 16, 2018, the Company issued the press release attached hereto as Exhibit 99.2.

SECTION 9 – FINANCIAL STATEMENTS AND EXHIBITS

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number	Description
99.1	Presentation
99.2	Press release

SIGNATURES

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

Galectin Therapeutics Inc.

Date: April 16, 2018

By: /s/ Jack W. Callicutt
Jack W. Callicutt
Chief Financial Officer

A multicenter, randomized, double-blind, placebo-controlled trial of Galectin-3 inhibitor (GR-MD-02) for one year in patients with NASH cirrhosis and portal hypertension

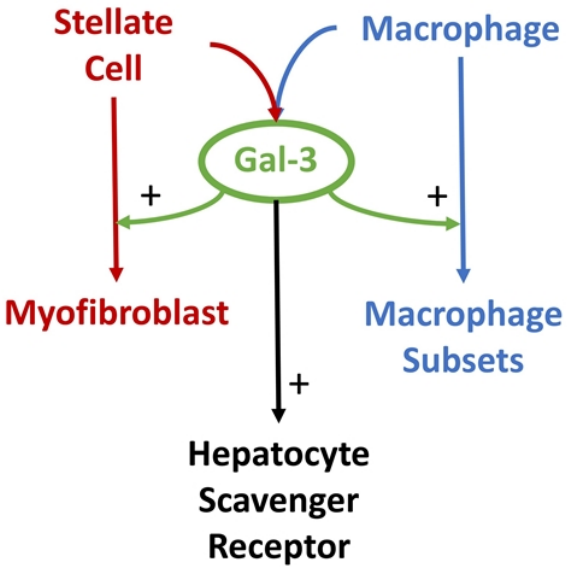
The NASH-CX Trial

[1]Naga Chalasani, [2]Guadalupe Garcia-Tsao, [3]Zachary Goodman, [4]Eric Lawitz, [5]Manal Abdelmalek, [6]Mary Rinella, [7]Michael Ryan, [8]Mazen Noureddin, [9]Christopher Jue, [1]Maxmillan Pyko, [10]Adam Allgood, [10]Harold Shlevin, [10]Rex Horton, [10]Eliezer Zomer, [10]Peter G. Traber, [11]Rohit Loomba, [12]Brent Neuschwander-Tetri, [13]Arun Sanyal, [14]Stephen A Harrison

[1]Indiana University, Indianapolis, IN, [2]Yale University, New Haven, CT, [3]INOVA Fairfax Hospital, Fairfax, VA, [4]Texas Liver Institute, San Antonio, TX, [5]Duke University, Durham, NC, [6]Northwestern University, Chicago, IL, [7]Digestive and Liver Disease Specialists, Norfolk, VA, [8]Cedars Sinai Medical Center, Los Angeles, CA, [9]Digestive Health Specialists, Winston-Salem, NC, [10]Galectin Therapeutics, Norcross, GA, [11]UCSD, San Diego, CA, [12]St. Louis University, St. Louis, MO, [13]VCU, Richmond, VA, [14]Pinnacle Clinical Research, San Antonio, TX

Rationale for Galectin-3 Inhibition in NASH

- Gal-3 is a lectin protein that binds to galactose residues on glycoproteins and is increased in NASH and liver fibrosis/cirrhosis
- Gal-3 null mice are resistant to NASH and fibrosis
- Gal-3 involved in multiple pathophysiologic processes in NASH and liver fibrosis
- GR-MD-02 is a complex carbohydrate drug that inhibits gal-3 and improves pathology of NASH and reverses fibrosis/cirrhosis in animal models [1,2]
- Safe and well tolerated in normal and NASH patients with advanced fibrosis in Phase 1 studies



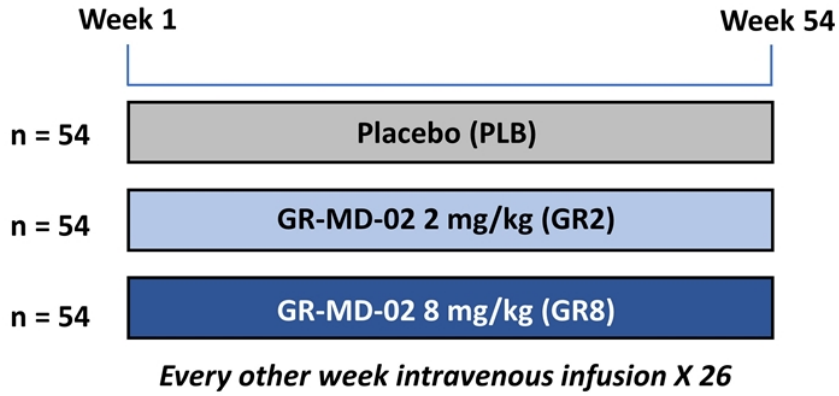
[1] Traber PG and Zomer E.PLOS ONE 2013;8:e83481

[2] Traber PG, Chou H, Zomer E, Hong F, Klyosov A Fiel M-I, Friedman, SL. PLOS ONE 2013;8:e75361.

NASH-CX Clinical Trial Design

AIM: Evaluate Safety and Efficacy of GR-MD-02 in Compensated NASH Cirrhosis

Major Inclusion Criteria		
	NASH cirrhosis (biopsy) HVPG ≥ 6 mmHg	No decompensating event No or small varices



Every other week intravenous infusion X 26

Study Endpoints & Assessment Methods

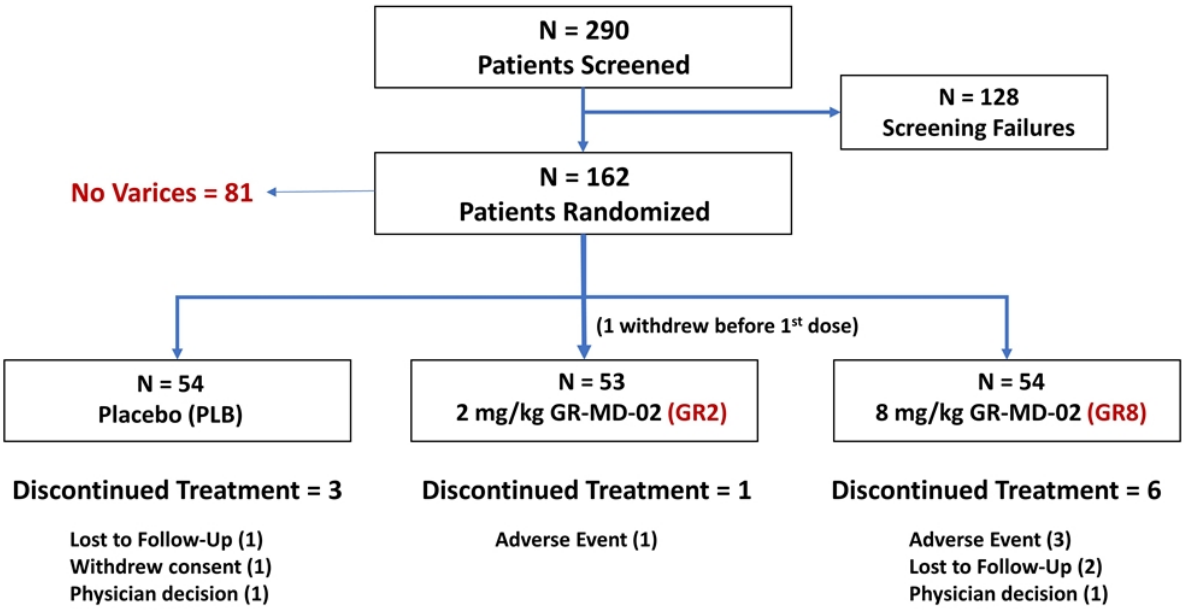
➢ Primary Endpoint

- **Change in Hepatic Venous Pressure Gradient (HVPG)**
  - Baseline and Week 54
  - Standardized Procedure and Central Blinded Reading

➢ Secondary Endpoints

- **Change in Liver Histology**
  - NAFLD Activity Score and Fibrosis Staging
  - Quantitative Morphometry for Collagen
  - Baseline and week 54
  - Central Blinded Reading
- **Endoscopy to Evaluate for Varices**
- **Complications of Cirrhosis**

Study Disposition (36 US Sites)



Discontinued Treatment = 3

Lost to Follow-Up (1)
Withdrew consent (1)
Physician decision (1)

Discontinued Treatment = 1

Adverse Event (1)

Discontinued Treatment = 6

Adverse Event (3)
Lost to Follow-Up (2)
Physician decision (1)

Study Demographics & Baseline Assessments

	Total (n=162)	Placebo (n=54)	GR2 (n=54)	GR8 (n=54)
Age, years; median (IQR)	59 (52, 65)	59 (53, 64)	60 (53, 65)	58 (51, 63)
Female, n (%)	113 (70)	36 (67)	34 (63)	43 (79)
Non-Hispanic White, n (%)	132 (81)	46 (85)	46 (85)	40 (74)
BMI, kg/m^2; median (IQR)	34 (31, 39)	34 (30, 38)	36 (31, 41)	35 (31, 38)
Diabetes, n (%)	100 (62)	32 (59)	32 (59)	36 (67)
AST (U/L) mean ± SD	49.8 ± 33.8	51.9 ± 48.2	48.3 ± 23.0	49.3 ± 24.8
ALT (U/L) mean ± SD	47.1 ± 34.1	48.1 ± 38.1	42.4 ± 21.0	50.9 ± 40.1
ELF Score mean ± SD	10.7 ± 1.2	10.8 ± 1.1	10.7 ± 1.2	10.7 ± 1.2
NAFLD Activity Score	4.2 ± 1.6	4.2 ± 1.5	4.3 ±1.3	4.2 ± 1.6
Ishak Stage (5/6)	48/123	13/41	20/43	15/39
Collagen (%) mean ± SD	10.5 ± 6.1	10.8 ±6.5	9.7 ± 5.9	11.0 ± 6.1

IQR=interquartile range; BMI=body mass index; AST=aspartate transaminase; ALT=alanine transaminase; ELF=enhanced liver fibrosis; NAFLD=non-alcoholic fatty liver disease

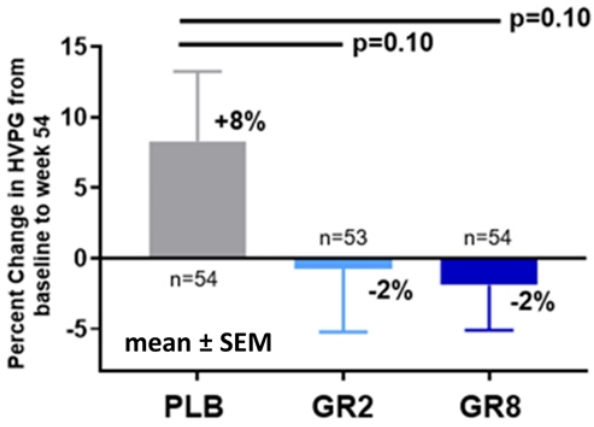
Baseline HVPG (mmHg) in Patient Groups

	Total Mean ± SD (n)	Placebo Mean ± SD (n)	GR2 Mean ± SD (n)	GR8 Mean ± SD (n)
Full Analysis Set	12.2 ± 4.2 (162)	11.6 ± 4.0 (54)	12.4 ± 4.3 (54)	12.7 ± 4.2 (54)
CSPH Sub-group	14.3 ± 3.4 (108)	14 ± 3.1 (33)	14.2 ± 3.9 (37)	14.8 ± 3.1 (38)
MPH Sub-Group	7.9 ± 1.2 (53)	7.8 ± 1.3 (21)	8.2 ± 1.0 (16)	7.8 ± 1.3 (16)
No Varices Sub-Group	10.6 ± 3.5 (81)	10.8 ± 3.8 (33)	10.3 ± 2.9 (25)	10.7 ± 3.8 (23)
With Varices Sub-Group	13.9 ± 4.2 (80)	12.9 ± 4.1 (21)	14.2 ± 4.6 (28)	14.2 ± 3.9 (31)

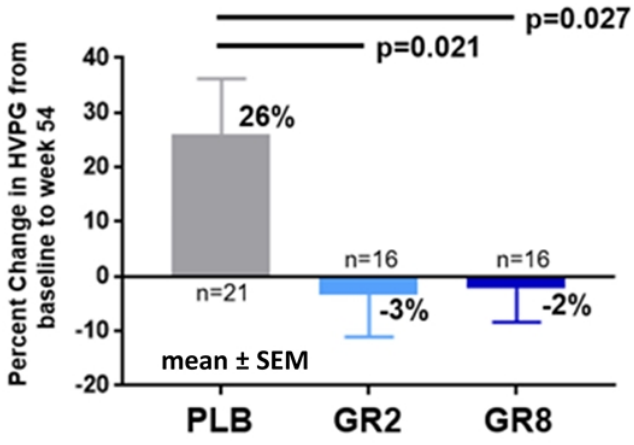
There were no statistical differences between the three treatment groups for any of the measures.
CSPH=clinically significant portal hypertension (≥ 10 mm Hg).
MPH=mild portal hypertension (≥ 6 and < 10 mm Hg).

HVPG Primary Endpoint (Pre-Specified Analyses)

Total Patient Population



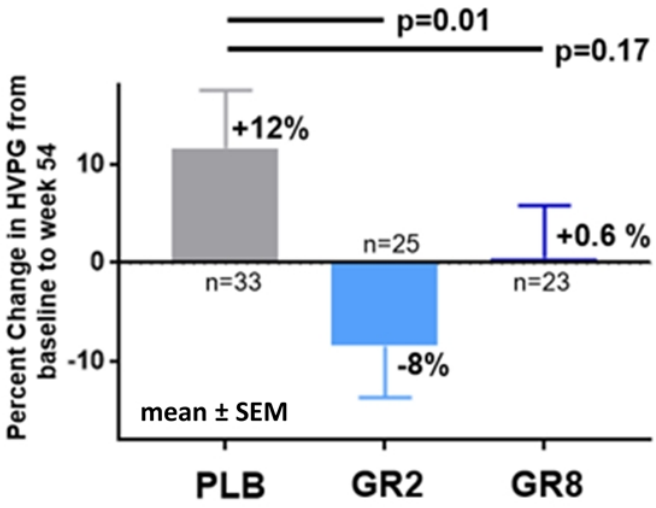
Mild Portal Hypertension



ITT with LOCF (last observation carried forward); ANOVA with LSD (least squared difference)

No Esophageal Varices at Baseline (Post Hoc Analysis)

50% of patients (81) did not have varices at baseline

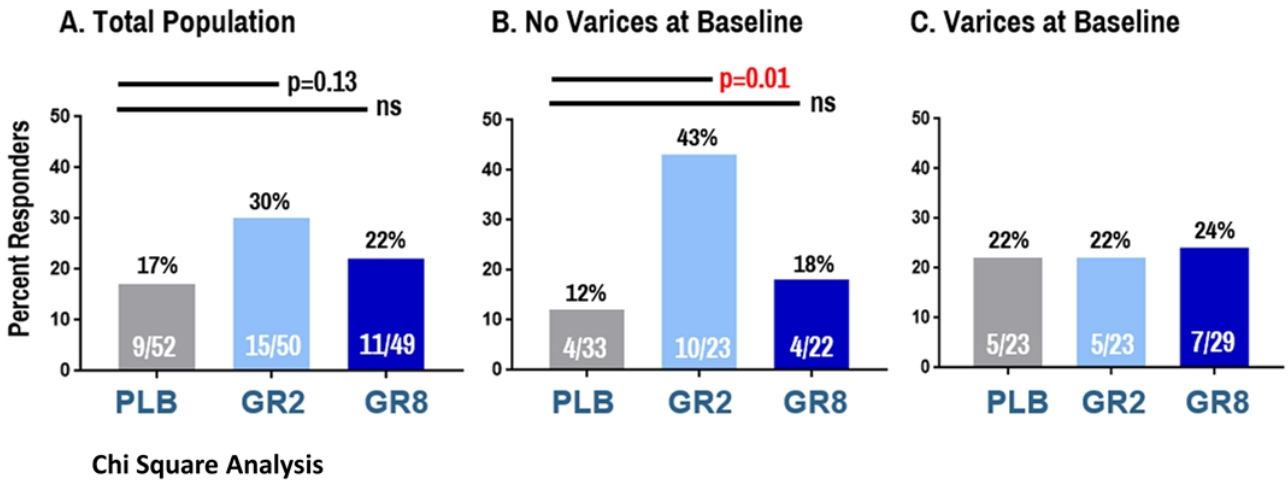


ITT with LOCF; ANOVA with LSD

Responder Analysis (Post Hoc Analysis)

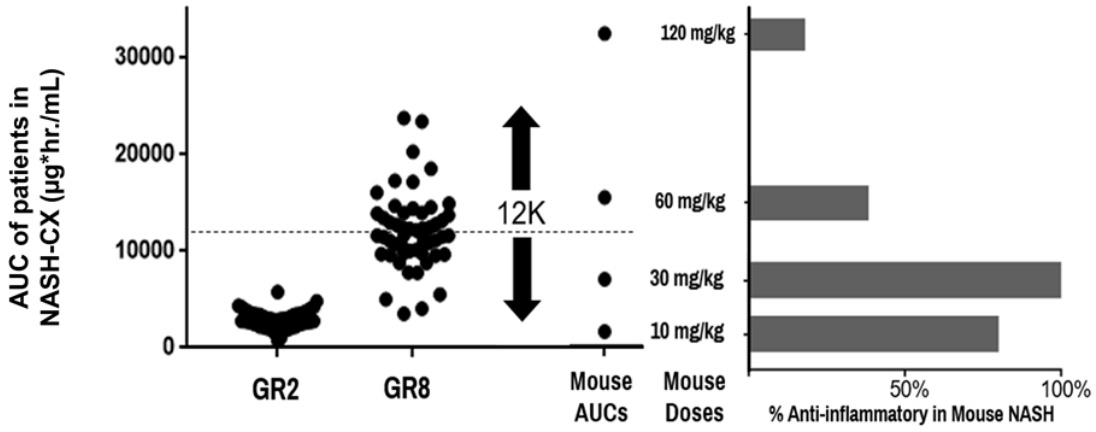
Percentage of Patients Who Had a Clinically Relevant Reduction in HVPG With:

- **≥ 2 mmHg Decrease From Baseline AND**
- **≥ 20% Decrease From Baseline**



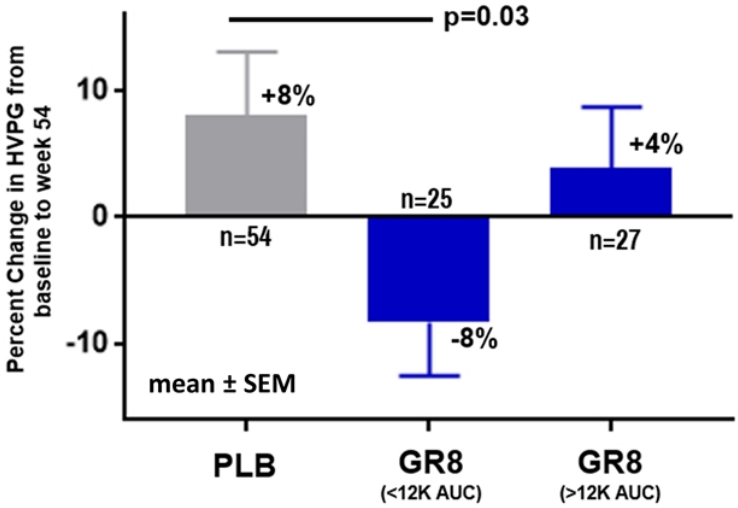
Chi Square Analysis

PK-PD Correlation Between Human and Mouse Data



[1]Traber, P.G. and E. Zomer, *Therapy of experimental NASH and fibrosis with galectin inhibitors.* PLoS. One, 2013. 8(12): p. e83481.

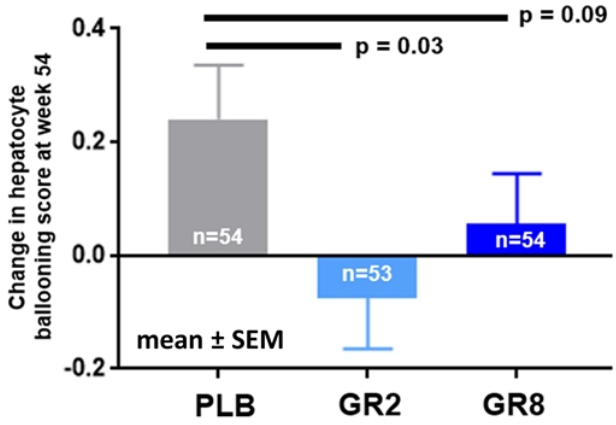
Change in HVPG Using PK Range Groups for GR8



ITT; ANOVA with LSD; AUC=area under concentration curve (µg*hr./mL)

Changes in Liver Histology in Total Patient Population

- Trend towards improvement in NAS that did not reach significance
- No differences in steatosis across the treatment groups
- Statistically significant difference between GR2 and placebo for inflammation scores in the patients without baseline varices
- There was no effect on fibrosis staging or percent collagen on morphometry
- Statistically significant improvement in hepatocyte ballooning in GR2 group and trend in GR8 group



ITT Analysis Set; Ordinal logistic regression analysis

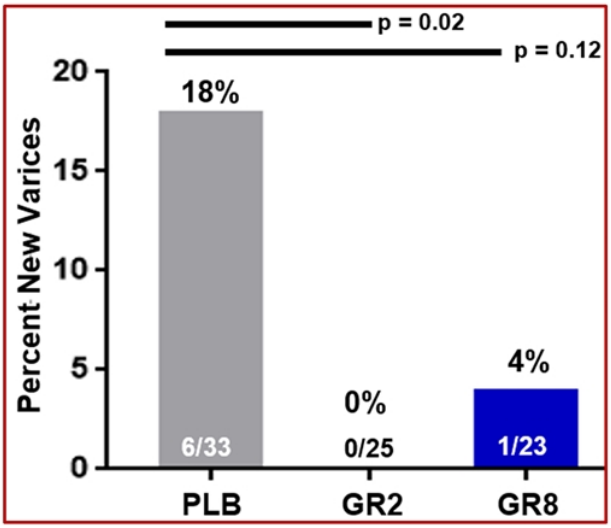
Correlation of Liver Biopsy Findings in HVPG Responders

Total Patient Population

	GR2[1]	GR8[1]
Hepatocyte Ballooning	0.04	0.05
NAFLD Activity Score	0.19	0.28
Ishak Stage	0.20	0.59

[1]p value compared to placebo
Ordinal logistic regression analysis was used to compare groups. ITT analysis set.

Fewer Patients in GR Groups Developed New Varices



Chi Square Analysis

Development of Cirrhosis Complications[1]

	Total	PLB	GR2	GR8
FAS Population	n=161	n=54	n=53	n=54
• Complications – n(%)	21 (13)	9 (17)	5 (9)	7 (13)
No-Varices Population	n=81	n=33	n=25	n=23
• Complications – n(%)	12 (15)	7 (21)	3 (12)	2 (9)
MPH Population	n=53	n=21	n=16	n=16
• Complications – n(%)	4 (8)	3 (14)	1 (6)	0

[1] Development of new varices
Variceal hemorrhage
Clinically significant ascites
Overt hepatic encephalopathy
↑ CTP score ≥ 2
↑ MELD to ≥ 15
Liver transplantation or death

Adverse Events

	Total (n=161)	PLB (n=54)	GR2 (n=53)	GR8 (n=54)
Treatment Emergent (TE) AEs	1323	431	509	383
Patients with at least ≥ grade 3 AE (%)	33 (20.5)	11 (20.4)	11 (20.8)	11 (20.4)
Patients with at least 1 TE SAE[1] (total)	25 (34)	8 (10)	5 (10)	12 (14)
Study drug discontinued due to AE	3	0	0	3[2]
Death	1	0	1[3]	0

[1] Two treatment emergent SAEs were rated by PI as possibly related to study drug (transient ischemic attack and worsening of hyponatremia, both GR8) but were rated by sponsor as unrelated; All other SAEs were unrelated to study drug

[2] *Probably related to drug*: spasmodic cough (1); *Unrelated to study drug:* esophageal variceal bleeding (2).

[3] Pulmonary embolism following hernia repair surgery, judged to be unrelated to study drug

Conclusions

- **Change in HVPG associated with GR treatment was not significant in total patient population, but statistically significant in the pre-specified group of mild portal hypertension**
- **In patients without varices at baseline, there was a statistically significant difference in the GR2 group in the change in HVPG, percentage of responders, and development of new varices**
- **GR treatment improved hepatocyte ballooning in the total, which correlated with an improvement in HVPG**
- **Less pronounced effects of GR8 may be explained by its variable pharmacokinetics**
- **GR 2 and GR 8 treatment was well-tolerated with no safety signals**
- **These results warrant further trials with GR-MD-02 in compensated NASH cirrhotic patients without esophageal varices or those with mild portal hypertension**

Acknowledgements

We extend our thanks to the patients, their families and all participating investigators

Indiana University School of Medicine-Dr. Chalasani
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Pinnacle Clinical Research, PLLC-Dr. Harrison
Digestive and Liver Disease Specialists-Dr. Ryan
Cedars Sinai Medical Center-Dr. Noureddin
Digestive Health Specialists, PA-Dr. Jue
Medical University of South Carolina-Dr. Rockey
Thomas Jefferson University-Dr. Halegoua-De Marzio
Texas Clinical Research Institute LLC-Dr. Ghalib
Virginia Commonwealth University-Dr. Sanyal
University of Mississippi Medical Center-Dr. Borg
Bon Secours Richmond Health System-Dr. Shiffman
University of Colorado Denver-Dr. Wieland
Columbia University Medical Center-Dr. Wattacheril
University of Michigan-Dr. Conjeevaram
Mcguire Veterans Affairs Medical Center-Dr. Fuchs
Baylor College of Medicine-Dr. Vierling
Piedmont Hospital-Dr. Rubin
Mary Immaculate Hospital-Dr. Shiffman
Saint Louis University-Dr. Tetri
Mercy Medical Center-Dr. Thuluvath
Swedish Medical Center-Dr. Kowdley
UH Cleveland Medical Center-Dr. Gholam
International Medical Investigations Center-Dr. Rodriguez
Intermountain Medical Center-Dr. Charlton
Tulane University Health Sciences Center-Dr. Balart
Vanderbilt University Medical Center-Dr. Scanga
Walter Reed National Military Medical Center-Dr. Torres
Tampa General Medical Group-Dr. Kemmer
University of California San Diego Medical Center-Dr. Loomba
Beth Israel Deaconess Medical Center-Dr. Lai
University Gastroenterology-Dr. Sepe
Minnesota Gastroenterology PA-Dr. Zogg
Brooke Army Medical Center-Dr. Paredes
HVPG
Yale University School of Medicine-Dr. Garcia-Tsao
Liver Biopsy
Inova Fairfax Hospital-Dr. Goodman

This study was funded by Galectin Therapeutics, Inc.

Galectin Therapeutics Late-Breaker Presentation at The International Liver Congress Reinforces and Extends the Positive Effects of GR-MD-02 in Patients With NASH Cirrhosis

NORCROSS, Ga. (April 16, 2018) – Galectin Therapeutics Inc. (NASDAQ: GALT), the leading developer of therapeutics that target galectin proteins, today provided highlights from a late-breaker oral presentation at The International Liver Congress™ 2018, European Association for the Study of the Liver (EASL) in Paris, France on Saturday, April 14, 2018.

Naga P. Chalasani, M.D., Associate Dean for Clinical Research; Director, Division of Gastroenterology and Hepatology at Indiana University School of Medicine; and co-lead principal investigator on Galectin Therapeutics' recent Phase 2b NASH-CX trial, delivered a late-breaker presentation entitled "A multicenter, randomized, double-blind, PLB-controlled trial of Galectin-3 inhibitor (GR-MD-02) in patients with NASH cirrhosis and portal hypertension" (click for presentation). The session focused on the Company's recent Phase 2b NASH-CX trial results and the innovative work the Company is doing for patients with non-alcoholic steatohepatitis (NASH) cirrhosis and portal hypertension.

Dr. Chalasani's late-breaker presentation highlighted and extended the study's primary findings in patients without esophageal varices and those with mild portal hypertension. Most importantly, in patients with NASH cirrhosis without esophageal varices, Galectin Therapeutics' lead compound, GR-MD-02, demonstrated a statistically significant improvement in portal pressure, an improvement in liver cell death (hepatocyte ballooning) on biopsy for the total population, and a reduction in the development of new esophageal varices at the end of the one-year study. This subgroup is large and commercially relevant as it comprises about half of all patients with NASH cirrhosis.

Since reporting these initial findings in December 2017, continued analysis of the data has led to two additional findings that reinforce the positive effects of GR-MD-02. First, a statistically significant ($p=0.04$) correlation was identified between the decrease in portal pressure (HVPG, or hepatic venous pressure gradient) and the improvement in hepatocyte ballooning (viz., representing a decrease in liver cell death) upon treatment with GR-MD-02 at 2 mg/Kg. This suggests an important pathophysiological link between the improvement in liver biopsy and reductions in HVPG. To our knowledge, this is the first time that such a correlation has been demonstrated in a human clinical trial in patients with NASH cirrhosis.

Secondly, an evaluation of GR-MD-02 blood levels provided an explanation for the reduced efficacy response observed in the higher GR8 (8 mg/Kg) dose group. In the GR2 (2 mg/Kg) dose group, the blood levels (or total exposure to the drug as measured by area under the concentration-time curve) were tightly grouped. In contrast, there was a broad distribution of higher drug exposures in the GR8 group. Approximately half of the patients who received GR8 had GR-MD-02 blood concentration levels that had risen to a range where a reduced efficacy effect in the liver had been noted at very high doses in the NASH animal models.

When the GR8 group was divided, based on pharmacokinetic analysis of drug levels, into separate low (<12K µg*hr./mL) and high (>12K µg*hr./mL**)** drug exposure ranges, a statistically significant effect (p=0.03) of GR8 on both HVPG and hepatocyte ballooning was observed in those patients with drug levels in the lower drug exposure range. There was no corresponding statistically significant effect in the higher drug exposure range group of patients receiving GR8 in analogy to what was observed in the NASH animal studies. Therefore, the GR8 dose, in cirrhotic patients, seems to be at the upper range of efficacy. Importantly, this not only provides an explanation of the dose ranging results, but also more clearly defines the upper range of human drug dosing for GR-MD-02 in patients with NASH cirrhosis. Further, these results suggest it might be useful to explore intermediate doses between 2mg/kg and 8mg/Kg in future clinical studies.

“The findings presented by Dr. Chalasani reinforce how the NASH-CX trial has demonstrated clinically meaningful improvement for those patients with NASH cirrhosis without esophageal varices with a drug that was well tolerated over one year of therapy,” said Dr. Peter Traber, CEO and Chief Medical Officer of Galectin Therapeutics. “Since about 50 percent of the total population of patients with NASH cirrhosis do not have esophageal varices and endoscopy to evaluate for varices is part of the standard of care for patients with NASH cirrhosis, there is a large and easily identifiable population of patients that might benefit from GR-MD-02. We look forward to presenting our findings to the FDA next month and to continuing advanced clinical studies to progress GR-MD-02 toward approval for the treatment of NASH cirrhosis in an appropriate patient group.”

About NASH Cirrhosis

NASH cirrhosis is the final stage in the progression of non-alcoholic steatohepatitis (NASH), a disease of the liver which affects millions of people in the U.S. and worldwide. The liver cell death and inflammation seen in NASH eventually causes progressive scarring of the liver, that eventually can result in liver cirrhosis. While the early stages of NASH can be treated by changes in lifestyle, such as losing weight and exercising, once the disease progresses to NASH cirrhosis there is no treatment available short of a liver transplant. Of the total number of individuals in the world felt to presently have NASH, it is predicted that NASH cirrhosis will eventually kill 20 million of those people.

One of the results of NASH cirrhosis is an increase in blood pressure in the portal vein that brings blood and nutrients from the digestive tract through the liver and then out to the rest of the body. As the scarring effect of cirrhosis on the liver progresses, blood flow through the liver becomes more difficult,

increasing the blood pressure in the portal vein, creating varying degrees of portal hypertension. Eventually, this increase in blood pressure causes the veins connected to the liver to dilate and form esophageal varices, in which are dilated veins that divert blood through the esophagus, bypassing flow through the liver. These dilated veins in the esophagus are prone to bleeding, which is a major cause of morbidity and mortality in patients with NASH cirrhosis.

About the NASH-CX Trial

The NASH-CX trial was a randomized, double-blind, placebo-controlled Phase 2b clinical trial which enrolled 162 NASH cirrhosis patients; NASH-cirrhosis was confirmed both by liver biopsy and by confirmation of an elevated hepatic venous pressure gradient (HVPG). Enrolled patients received either 8 mg/kg or 2 mg/kg of GR-MD-02 or placebo every other week for 52 weeks, for a total of 26 doses. The aim of the NASH-CX clinical trial was to evaluate the safety and efficacy of GR-MD-02 in patients with well-compensated NASH cirrhosis. The primary study endpoint was a reduction in HVPG. Patients treated with GR-MD-02 were evaluated to determine the change in HVPG as compared to patients treated with placebo. Secondary end-points include NASH fibrosis stage and percent of fibrotic tissue based on liver biopsy and other non-invasive measures (see: www.clinicaltrials.gov for further details).

About GR-MD-02

GR-MD-02 is a non-biologic complex carbohydrate drug that targets galectin-3, a critical protein in the pathogenesis of fatty liver disease and fibrosis. Galectin-3 plays a major role in diseases that involve scarring of organs including fibrotic disorders of the liver, lung, kidney, heart and vascular system. The drug binds to galectin-3 proteins and disrupts its function. Preclinical data in animals have shown that GR-MD-02 has robust treatment effects in reversing liver fibrosis and cirrhosis.

About Galectin Therapeutics

Galectin Therapeutics is dedicated to developing novel therapies to improve the lives of patients with chronic liver and skin diseases and cancer. Galectin’s lead drug (GR-MD-02) is a carbohydrate-based drug that inhibits the galectin-3 protein that is directly involved in multiple inflammatory, fibrotic, and malignant diseases. The lead development program is in non-alcoholic steatohepatitis (NASH) with cirrhosis, the most advanced form of NASH related fibrosis. This is the most common liver disease and one of the largest drug development opportunities available today. Additional development programs are for treatment of severe atopic dermatitis, moderate-to-severe plaque psoriasis, and in combination immunotherapy for advanced melanoma and other malignancies. Galectin seeks to leverage extensive scientific and development expertise as well as established relationships with external sources to achieve cost-effective and efficient development. Additional information is available at www.galectintherapeutics.com.

Forward looking statements

This press release contains 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

management's 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 the hope that Galectin's development program for GR-MD-02 will lead to the first therapy for the treatment of NASH with cirrhosis, and those regarding the hope that our lead compounds will be successful in connection with the treatment of skin disease and cancer immunotherapy. Factors that could cause actual performance to differ materially from those discussed in the forward-looking statements include, among others, that Galectin may not be successful in developing effective treatments and/or obtaining the requisite approvals for the use of GR-MD-02 or any of its other drugs in development; the Company's future clinical studies may not 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 Galectin's drugs are subject to change at any time based on the changing needs of the Company as determined by management and regulatory agencies; the Company may find that its patents does not offer the protection anticipated, and regardless of the results of any of its development programs, Galectin may be unsuccessful in developing partnerships with other companies or raising additional capital that would allow it to further develop and/or fund any studies or trials. Galectin has incurred operating losses since inception, and its ability to successfully develop and market drugs may be impacted by its ability to manage costs and finance continuing operations. For a discussion of additional factors impacting Galectin's business, see the Company's Annual Report on Form 10-K for the year ended December 31, 2017, and subsequent filings with the SEC. You should not place undue reliance on forward-looking statements. Although subsequent events may cause its views to change, management disclaims any obligation to update forward-looking statements.

Investor Contact:

Galectin Therapeutics, Inc.

Jack Callicutt, Chief Financial Officer
(678) 620-3186
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