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): December 5, 2017

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 December 5, 2017, Galectin Therapeutics Inc. (the "Company) posted to its website a presentation of top line 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 December 5, 2017, 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: December 5, 2017

By: /s/ Peter G. Traber
Peter G. Traber, M.D.
Chief Executive Officer

Exhibit 99.1

Galectin Therapeutics

NASH-CX Clinical Trial Top Line Results

December 5, 2017

NASDAQ: GALT
www.galectintherapeutics.com

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 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 which could cause actual results to differ materially from those described in the statements.

These statements include those regarding the potential therapeutic benefits of our drugs and specifically the results of our NASH-CX clinical trial. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others that:

- the data presented today represent a top line analysis, and there may be changes in the final clinical trial report due to further analysis of the full data set including additional statistical analysis;
- subsequent trials, if any, in whatever patient population chosen may fail to validate any positive results of our trial now concluded;
- future phases or future clinical studies could prove prohibitively time consuming and/or 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, personnel, and spending projections may change;
- due to the novel nature of our compounds, future phases of manufacturing scale-up and supporting chemical and physical characterizations for both trials and commercial purposes can be challenging and costly and there is no certainty this can be accomplished nor certainty it would acceptable to regulators;
- we may be unsuccessful in developing partnerships or other business relationships with other companies or obtaining capital that would allow us to further develop and/or fund any future studies or trials or sell or license our intellectual property; and, further,
- there is the uncertainty that any drug in development could obtain regulatory approval in any patient population.

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, 2016, 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.

Summary: NASH-CX Clinical Trial Results in Patients with NASH Cirrhosis

- A statistically significant and clinically meaningful effect of GR-MD-02 was observed on the primary endpoint measurement of HVPG[1] in the subgroup of NASH[2] cirrhosis patients without esophageal varices (81 patients or 50% of total group), regardless of the severity of their baseline portal hypertension
- There was a positive trend in the total group of patients, but the difference did not reach statistical significance for this primary endpoint because there was more variability in HVPG measurements for patients with esophageal varices
- On liver biopsy, the entire study group of GR-MD-02 treated patients had a statistically significant improvement in hepatocyte ballooning, a measure of cell death and an important factor of NASH activity
- A statistically significant clinical outcome effect of GR-MD-02 treatment was observed on reducing the development of esophageal varices in patients without varices at baseline
- We believe this is the first large, randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in portal hypertension or liver biopsy in patients with NASH cirrhosis

[1] HVPG = Hepatic Venous Pressure Gradient [2] NASH = Non-Alcoholic Steatohepatitis

Background: Drug Development Program with GR-MD-02

- GR-MD-02 is a galectin-3 inhibitor that reverses fibrosis, reduces cell death and inflammation, and decreases portal pressure of cirrhosis, in rodent models of NASH fibrosis[1] and toxin-induced liver cirrhosis[2]
- NASH[3] is a chronic disease with progressive fibrosis that may lead to cirrhosis with portal hypertension and its consequent complications, and liver transplant or ultimately death

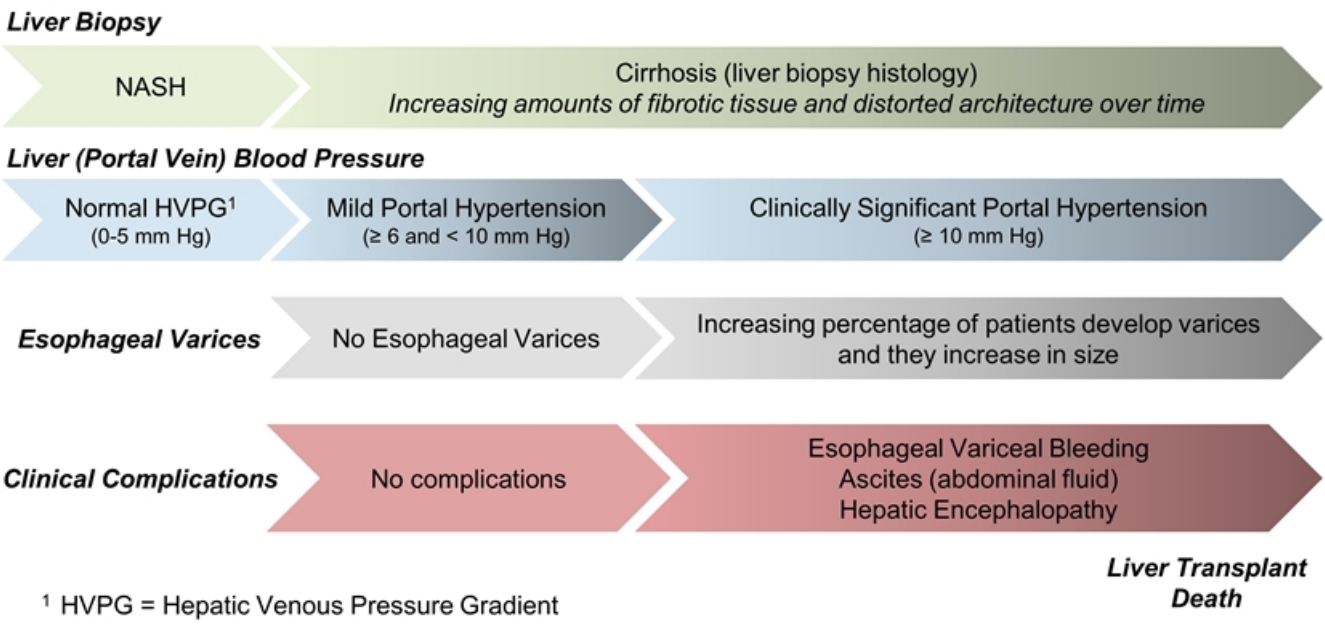
- The aim of the NASH-CX clinical trial was to evaluate the safety and efficacy of GR-MD-02 in patients with well-compensated NASH cirrhosis

[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*

[3] *NASH = Non-Alcoholic Steatohepatitis*

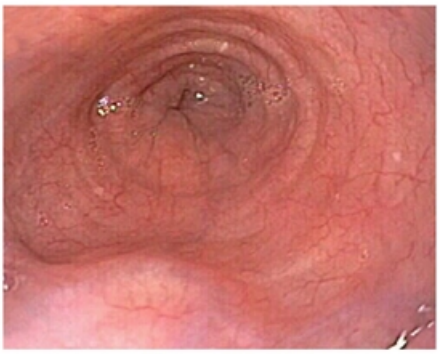
Liver (Portal Vein) Blood Pressure is Critical in Patients with NASH Cirrhosis



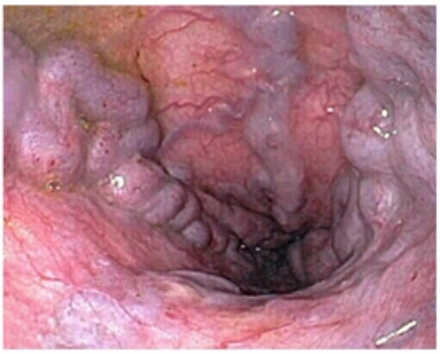
[1] HVPG = Hepatic Venous Pressure Gradient
(method for measuring the pressure in the portal vein)

Critical Importance of Esophageal Varices in NASH Cirrhosis

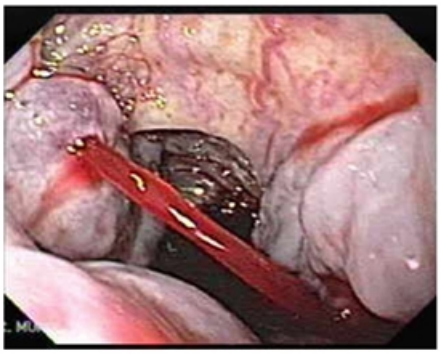
Esophagus: No Varices



Esophageal Varices



Bleeding Esophageal Varices



NASH-CX Clinical Trial Design[1]

Major Inclusion Criteria

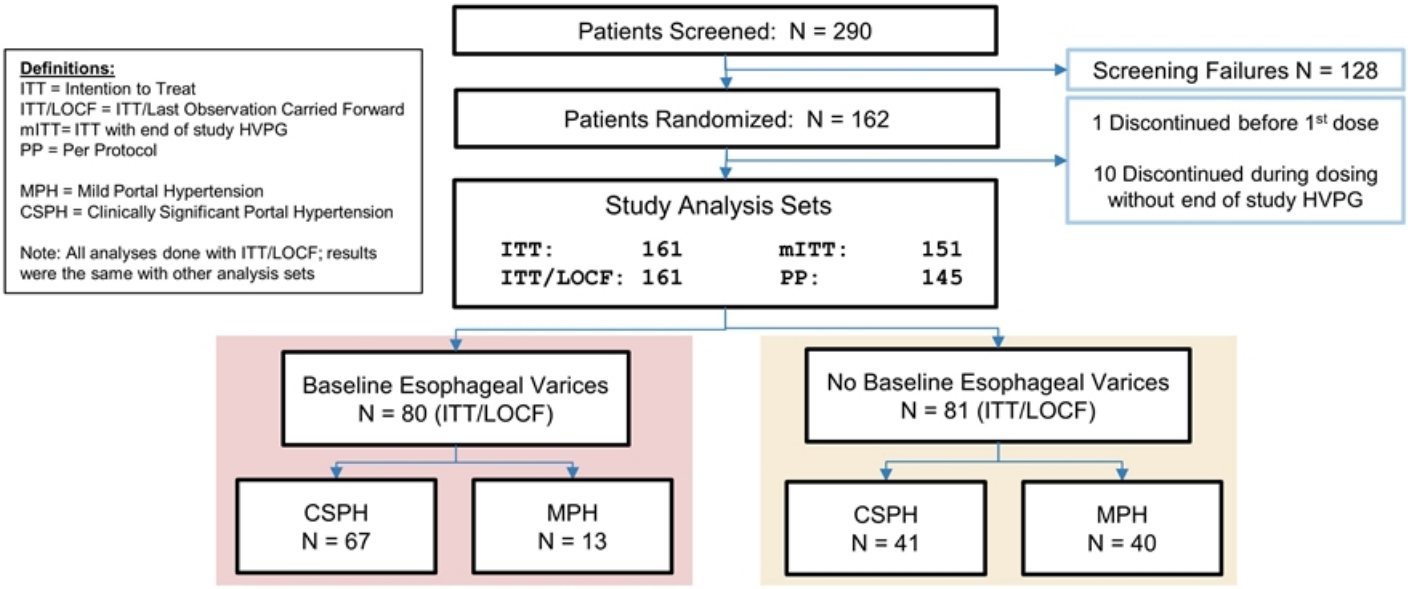
NASH cirrhosis (biopsy)
HVPG ≥ 6 mmHg
No cirrhosis complications
No or small varices

Every other week infusion X 26

		Baseline	*Week 26*	*Week 54*
Primary endpoint	HVPG[2]	X		X
Secondary endpoints	Liver Biopsy[3]	X		X
	FibroScan	X	X	X
	MBT[4]	X	X	X
	Complications[5]	X		X
	Endoscopy	X		X

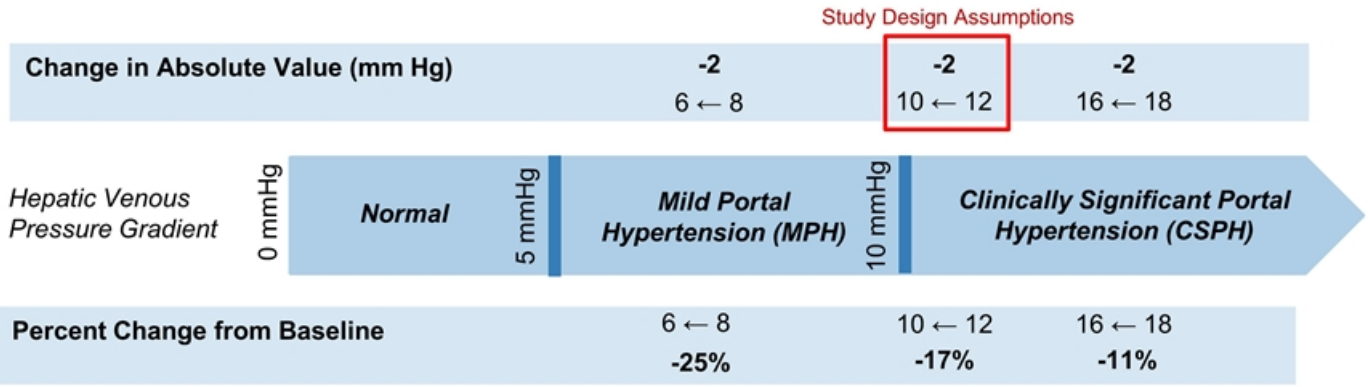
[1] All subjects were enrolled across 36 sites in the US (Appendix 1)
[2] HVPG = Hepatic Venous Pressure Gradient
[3] Histologic staging & quantitative morphometry for collagen
[4] MBT = ^{13}C Methacetin Breath Test
[5] Liver-related complications (varices/bleeding, ascites, hepatic encephalopathy, liver-related death, or transplant)

Patient Populations



Demographic characteristics (age, gender, BMI, nationality, diabetes) and baseline HVPG measurements were balanced across the three treatment groups in study analysis sets (Appendices 3 & 4)

HVPG Data Expressed as Change in Absolute Value and Percent Change From Baseline



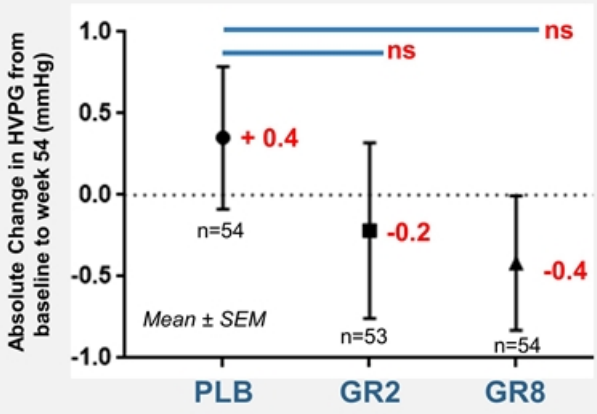
Many relevant clinical observations have been made based on percent change

- *Clinical trials have shown that the lower threshold for a clinically significant change in HVPG, which has an effect on patient outcomes, ranges between 10% and 20%*

HVPG Primary Endpoint: Total Patient Population[1] (Mean baseline HVPG 12.2 mmHg)

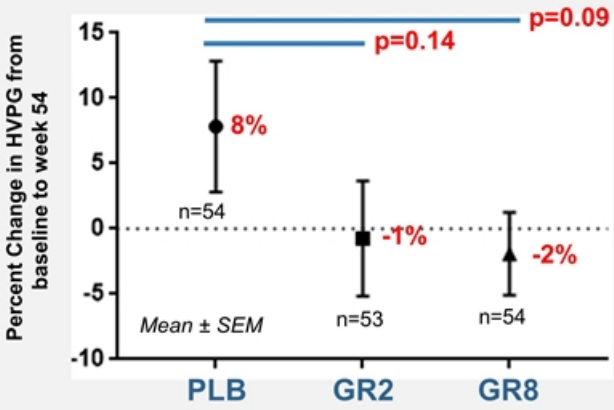
There was a 0.8 mmHg and 10% difference between placebo and the GR8 treatment group, but the differences did not reach statistical significance

Absolute change in HVPG (mm Hg)



[1]ITT with LOCF ANCOVA with Bonferroni

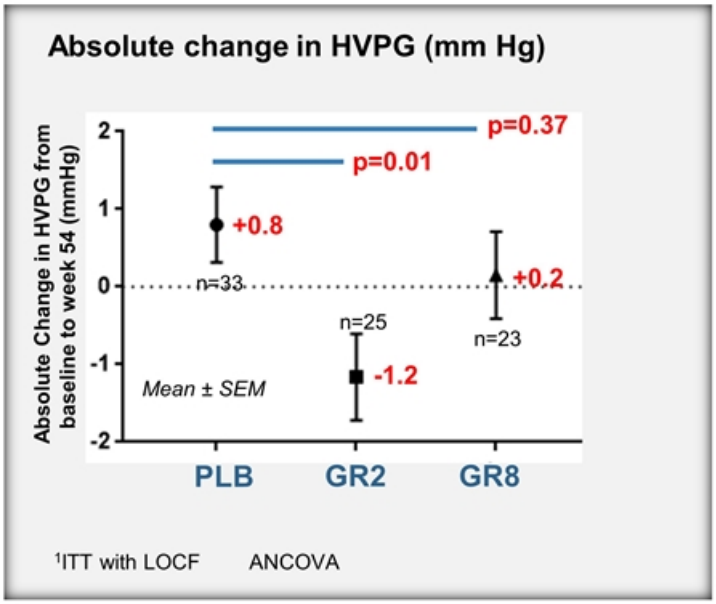
Percent change from baseline HVPG

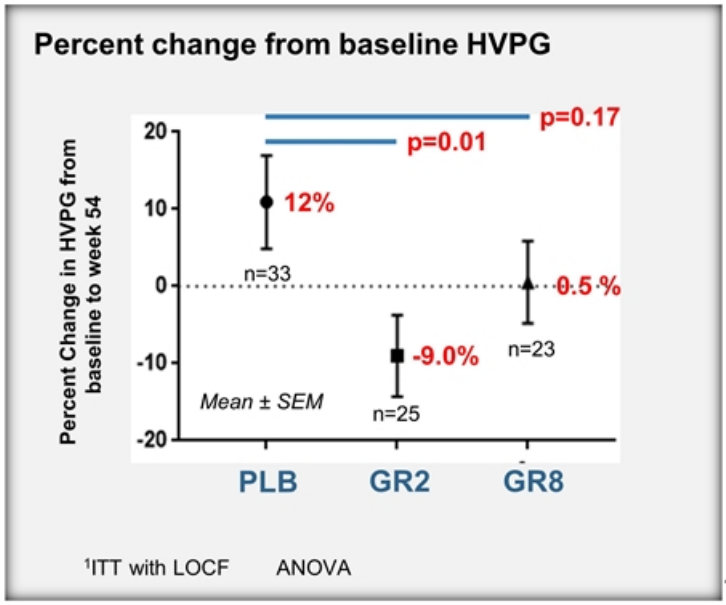


[1]ITT with LOCF ANOVA

HVPG in NASH Cirrhosis Without Varices at Baseline[1] (Mean baseline HVPG 10.6 mmHg)

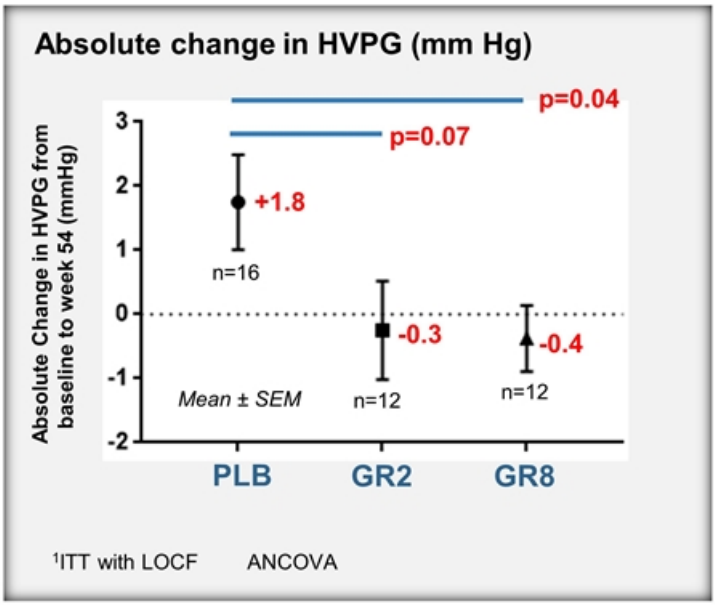
The drug effect was significantly dependent on the presence of varices at baseline (p<0.02). Analysis in the absence of baseline varices showed a statistically significant effect of GR2.

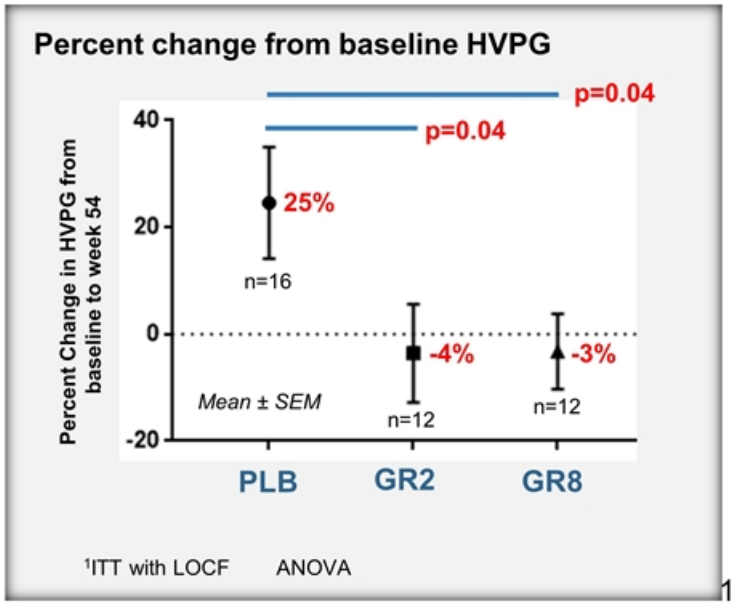




Mild Portal Hypertension Without Varices at Baseline[1] (Mean baseline HVPG 7.8 mmHg)

In the absence of varices and with mild portal hypertension, there was statistically significant treatment effect on HVPG in both dose groups

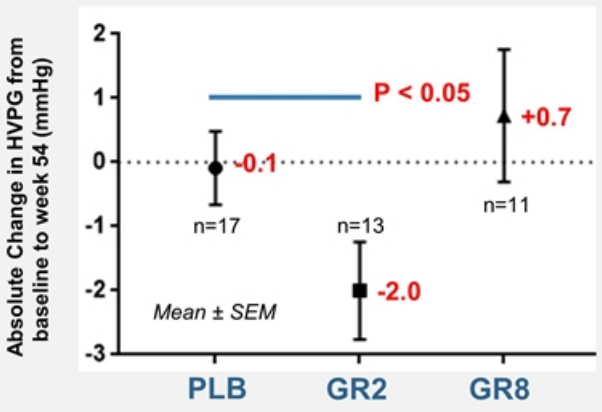




Clinically Significant Portal Hypertension Without Varices at Baseline[1] (Mean baseline HVPG 13.4 mmHg)

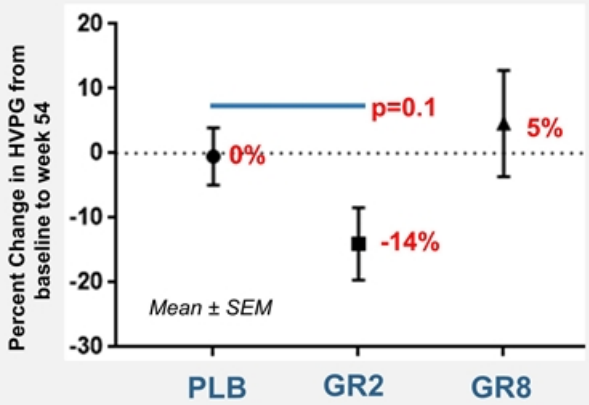
In the absence of varices and with clinically significant portal hypertension, there was a statistically significant treatment effect on HVPG in the GR2 group

Absolute change in HVPG (mm Hg)



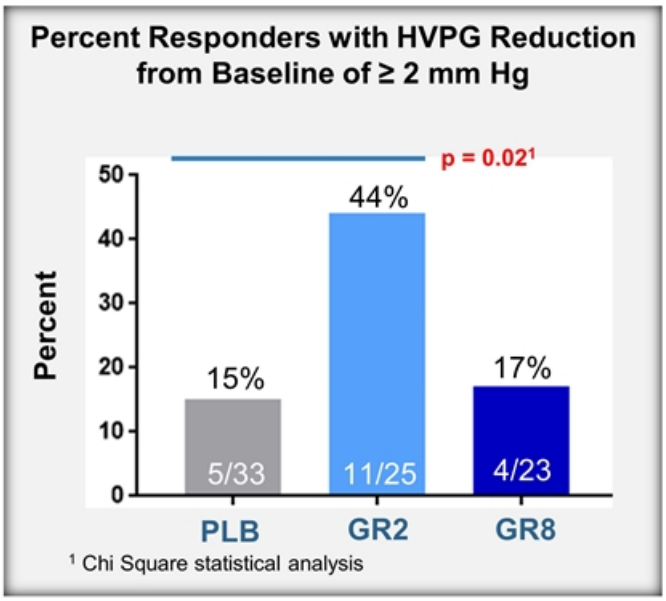
[1]ITT with LOCF ANCOVA

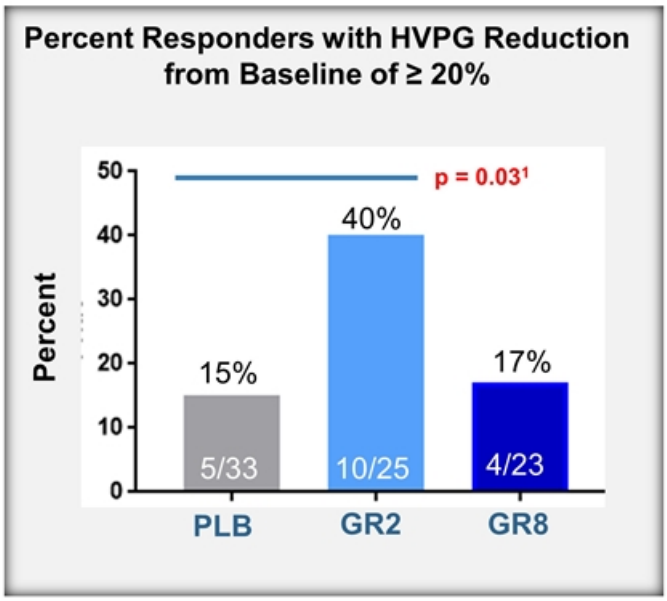
Percent change from baseline HVPG



[1]ITT with LOCF ANOVA

Responder Analysis: Percentage of Patients Without Varices at Baseline who have Clinically Significant Reductions in HVPG

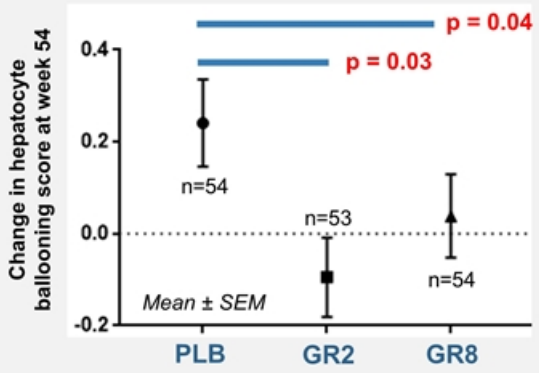




Statistically Significant Improvement of Hepatocyte Ballooning on Liver Biopsy

- The activity NASH is assessed by the NAFLD[1] Activity Score (Ballooning hepatocytes, inflammation and fat)
- We observed a trend reduction in NAS in treatment groups as compared to placebo (Appendix 4)
- A critical component of NAS is hepatocyte ballooning, an indicator of dying liver cells which is a critical in driving the progression of NASH, inflammation and fibrosis
- There was a statistically significant improvement in ballooning in both treatment groups compared to placebo
- The reduction in ballooning hepatocytes with GR-MD-02 correlates with what was seen in NASH animal models[2]

Total Study Patient Population[3]



[3]ITT with LOCF (161) Ordinal Logistic Regression Analysis

- There was a trend of decreased collagen in treated groups, but the differences were not statistically significant
- There were no significant differences between treatment groups in FibroScan or methacetin breath test

[1] *NAFLD = Non-Alcoholic Fatty Liver Disease* [2] *Traber PG and Zomer E. PLOS ONE 2013;8:e83481*

Cirrhosis Complications[1]

In patients without varices, there was a statistically significant reduction in the number of new varices that developed in patients treated with GR-MD-02 versus placebo

	Patients with at least one complication			
	PLB	**GR2**	**GR8**	***Comments***
All Patients: Intention to Treat (n=161)	**7**	**7**	**6**	No difference between groups
No Baseline Esophageal Varices (n=81)	**5**	**3**	**1**	No difference between groups
New Esophageal Varices	3	0	0	**p = 0.03[2], PLB vs GR2 + GR8**
Clinically Significant Ascites	1	2	0	No difference between groups
Hepatic Encephalopathy	1	1	1*	* BL HVPG 17.5 mm Hg

[1] **Complications Include:**
Esophageal Varices
- Development of New Varices
- Progression to Large Varices
- Variceal hemorrhage

Development of Clinically Significant Ascites
Development of Hepatic Encephalopathy

[2] Chi Square statistical analysis

Safety Results

	Total (n=162)	PLB (n=54)	GR2 (n=54)	GR8 (n=54)
All adverse events	1422	464	541	417
Grade 3-4 (patients (total events))	31 (69)	10 (19)	10 (22)	11 (28)
SAE[1] (patients (total events))	25 (39)	9 (13)	5 (10)	11 (16)
Rx stopped due to AE	5	0	0	5[2]
Death	1	0	1[3]	0
Grade 3/4 lab (patients (total events))	8 (15)	3 (3)	2 (2)	3 (10)

[1] Two SAEs were determined by the PI to be possibly related to study drug (transient ischemic attack and worsening of hyponatremia, both GR8); All others SAEs were felt to be unrelated to study drug

[2] *Possibly related to drug*: spasmodic cough (1); *Unrelated to study drug:* esophageal variceal bleeding (2), sepsis (1), pancreatitis (1)

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

Major Conclusions from NASH-CX Clinical Trial Results

- This trial demonstrated that GR-MD-02 had a statistically significant and clinically meaningful effect in reducing the primary endpoint measurement of HVPG in the subset of patients with NASH cirrhosis who did not have baseline esophageal varices (50% of total patient population); this effect was seen regardless of the severity of the patient's baseline portal hypertension
- There was an important drug effect in the total study population on liver biopsy, with a statistically significant improvement in hepatocyte ballooning (cell death) with both doses of GR-MD-02
- There was a statistically significant reduction in the development of varices in drug-treated patients compared to placebo; prevention of the development of varices is a clinically critical goal in NASH cirrhosis.
- While there was a drug effect in both dosage groups on liver biopsy and in the mild portal hypertension group, there was a consistently greater and statistically significant effect of the 2 mg/kg dose
- GR-MD-02 appears to be safe and well tolerated in this one year, phase 2 clinical trial
- We believe this is the first large, randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in portal hypertension or liver biopsy in patients with NASH cirrhosis without varices

Discussion of Key Questions Raised by the Study Results

- **Why is there a differential effect of GR-MD-02 therapy in patients with and without esophageal varices?**
  - Liver biopsy showed an effect in all patients, so GR-MD-02 had a therapeutic benefit regardless of varices
  - The sensitivity and variability of the HVPG measurement to detect an improvement may be different in the presence of varices
- **How would the improvement in hepatocyte ballooning translate to an effect on portal hypertension?**
  - The death of liver cells triggers wide range of biochemical changes in the liver
  - This cascade of events from liver cell death might increase the resistance to blood flow through the liver
- **What is the reason for the more efficacious effect of the lower GR2 dose versus the higher GR8 dose?**
  - The sum of the data shows that there is clearly an effect of both the GR2 and GR8 doses
  - The GR2 dose had a more robust effect, which is most evident in the responder analysis which is the most rigorous assessment of efficacy because it requires a clinically significant *improvement* in HVPG from baseline
  - In an animal model of NASH, there was a similar effect of increasing drug doses on the NAFLD activity score[1]
  - These data suggest that higher doses of GR-MD-02 would not likely be more efficacious, and in future studies it may be logical to increase the duration of therapy to get a greater effect rather than increasing the dose

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

NASH-CX Trial: Next Steps

- **The trial results identify a significant patient population who may benefit from treatment with GR-MD-02**
  - Patients with well-compensated NASH cirrhosis without esophageal varices
  - Patients are readily identifiable since upper endoscopy for varices is recommended for all those with cirrhosis
- **The results suggest endpoints that may be employed in a phase 3 program**
  - Change in HVPG has been suggested by the FDA as a possible acceptable surrogate for outcomes in clinical trials
  - Change in HVPG could be used as an absolute or percentage change or as a responder analysis, as we performed
  - The development of varices in patients without varices at baseline may be considered a clinical outcome measure
- **We will explore the design of a phase 3 program for NASH cirrhosis without varices with a variety of stakeholders including the Regulatory Agencies, key opinion leaders, and pharmaceutical companies**
- **We currently have fast track designation for this program, and believe these clinical data will allow us to expedite development under the FDA's "breakthrough therapy" designation, for which we will apply**
- **These data will be submitted as a late-breaking abstract for presentation at the International Liver Congress in Paris, France in April 2018**

Galectin Therapeutics

Appendix

NASDAQ: GALT
www.galectintherapeutics.com

Appendix 1: Deep Gratitude to Patient Volunteers and Clinical Study Sites

Indiana University School of Medicine-Dr. Chalasani
The Texas Liver Institute-Dr. Lawitz
Duke University Medical Center-Dr. Abdelmalek
Feinberg School of Medicine - Northwestern University-Dr. Rinella
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. Rocky
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 Biosy

Inova Fairfax Hospital-Dr. Goodman

Appendix 2: Study Demographics[1]

	Total (FAS[2]) (162)	PLB[3] (n=54)	GR2[3] (n=54)	GR8[3] (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)
White, n (%)	132 (81)	46 (85)	46 (85)	40 (74)
Hispanic/Latino, n (%)	28 (17)	8 (15)	7 (13)	13 (24)
Asian, n	1	0	1	0
Native Hawaiian, n	1	0	0	1
BMI, kg/m^2; Median (IQR)	34 (31, 39)	34 (30, 39)	36 (31, 41)	35 (31, 38)
Diabetes, n (%)	105 (65)	35 (65)	33 (61)	37 (69)

1 All subjects were enrolled across 36 sites in the United States
2 FAS = full analysis set, all subjects randomized
3 PLB = Placebo; GR2 = GR-MD-02 (2 mg/kg); GR8 = GR-MD-02 (8 mg/kg)

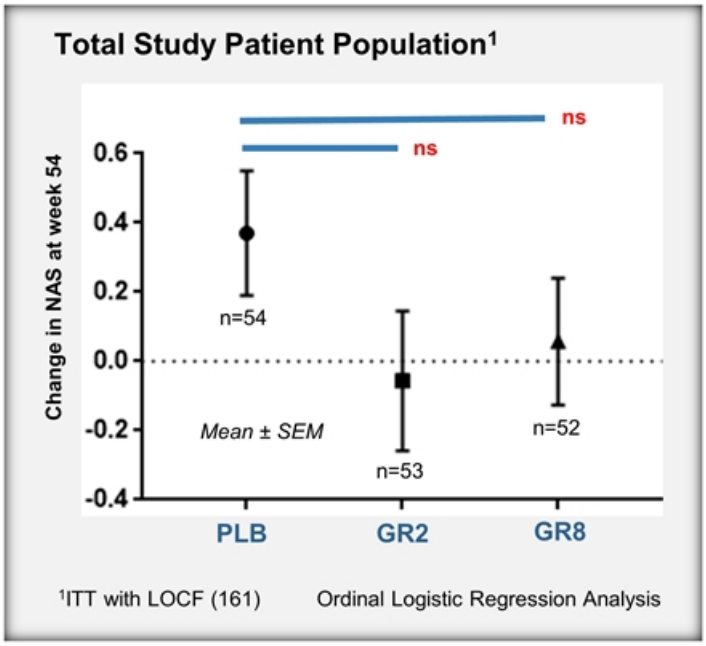
Appendix 3: HVPG at Baseline are Comparable Between Treatment Groups[1]

	Mean ± SD (n)	Total	PLB	GR2	GR8
Hepatic Venous Pressure Gradient	HVPG (mm Hg)	**12.2 ± 4.1 (162)**	**11.6 ± 3.9 (54)**	**12.3 ± 4.3 (54)**	**12.7 ± 4.2 (54)**
	CSPH[2] (mm Hg)	**14.3 ± 3.4 (109)**	**13.8 ± 3.1 (34)**	**14.2 ± 3.9 (37)**	**14.8 ± 3.1 (38)**
	MPH[3] (mm Hg)	**7.9 ± 1.2 (53)**	**7.8 ± 1.4 (20)**	**8.0 ± 3.3 (17)**	**7.6 ± 2.2 (16)**
	Neg Varices (mm Hg)	**10.6 ± 3.5 (81)**	**10.8 ± 3.8 (33)**	**10.4 ± 2.9 (25)**	**10.7 ± 3.8 (23)**
	Pos Varices (mm Hg)	**13.8 ± 4.2 (80)**	**12.7 ± 4.0 (21)**	**14.1 ± 4.6 (28)**	**14.2 ± 3.9 (31)**

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

Appendix 4: Effect of Drug Treatment on NAFLD[1] Activity Score

- The NAFLD[1] Activity Score (NAS) is the widely accepted way to evaluate liver biopsies for the severity NASH disease activity, absent an evaluation of liver fibrosis
- NAS is comprised of three components that are scored independently and then summed:
  1) Hepatocyte ballooning
  2) Inflammation
  3) Fat
- The activity of NASH, and hence the NAS, tends to decrease in NASH cirrhosis, in part because fat tends to be reduced
- The NAS was reduced in comparison to placebo in patients treated with GR-MD-02, which did not reach statistical significance



[1] *NAFLD = Non-Alcoholic Fatty Liver Disease*

Exhibit 99.2

Galectin Therapeutics Announces Results from Phase 2b NASH-CX Trial

Statistically significant and clinically meaningful effects observed in NASH cirrhosis patients without esophageal varices treated with GR-MD-02

Conference Call at 8:30 A.M. ET to Present Top Line Trial Results

NORCROSS, Ga., December 5, 2017 (GLOBE NEWSWIRE) — **Galectin Therapeutics Inc.** (NASDAQ:GALT), the leading developer of therapeutics that target galectin proteins, announced today that its Phase 2b NASH-CX trial of its proprietary compound GR-MD-02 showed statistically significant and clinically meaningful results in reducing the primary endpoint measurement of HVPG (hepatic venous pressure gradient) in comparison to placebo in NASH cirrhosis patients without esophageal varices, which represented 50 percent of the patients enrolled in the clinical trial. There was a positive trend in the total group of patients (both with and without varices), but the difference did not reach statistical significance for this primary endpoint because there was more variability in HVPG measurements for patients with esophageal varices.

For the major secondary endpoint assessment of liver biopsy, analysis of the total study population (161 patients) showed a statistically significant effect of drug treatment for improving hepatocyte ballooning (liver cell death), which is a key factor in the underlying disease process in NASH. Importantly, analysis of the secondary endpoint of complications of cirrhosis showed there was a statistically significant reduction in the development of new esophageal varices in patients without varices at baseline.

We also performed a rigorous assessment of the response to therapy by evaluating the percent of patients who had a reduction of HVPG from baseline (Responder Analysis). Responders were defined as having reductions of HVPG from baseline that have been shown to be clinically significant, an absolute reduction of ³ 2 mmHg of HVPG from baseline or a ³ 20 percent reduction of HVPG from baseline. Based on reduction in absolute HVPG, patients without varices who received a 2 mg/kg dosage of GR-MD-02 showed a statistically significant greater percentage of responders than those without varices in the placebo group (44 percent versus 15 percent, p=0.02). The same statistically significant results were seen when responders were analyzed based on a ³ 20 percent reduction from baseline HVPG (40 percent versus 15 percent, p=0.03).

“There is no current therapy for patients with NASH cirrhosis — and a therapy such as GR-MD-02 that could improve portal hypertension and potentially prevent the development of esophageal varices in NASH cirrhosis and subsequent complications — would be clinically valuable,” said Stephen A. Harrison, M.D., one of lead investigators of the NASH-CX trial, medical director of Pinnacle Clinical Research in San Antonio, Texas, and visiting professor of medicine at the University of Oxford, United Kingdom. “An indication of NASH cirrhosis without varices would be clinically meaningful to physicians, because it is standard of care for all patients with cirrhosis to have an upper endoscopy to assess for the presence of esophageal varices.”

“We believe this is the first large, randomized clinical trial of any drug to demonstrate a clinically meaningful improvement in portal hypertension or liver biopsy in patients with NASH cirrhosis without varices,” said Dr. Peter G. Traber, M.D., CEO and CMO of Galectin Therapeutics. “Furthermore, we believe that the results stratify a large and easily identifiable group of patients. The results also suggest several potential registration endpoints that may be employed in a phase 3 program, including absolute or percent changes in HVPG, the percentage of patients who respond with a clinically relevant reduction in HVPG (Responder Analysis), and the development of esophageal varices, which may be considered a clinical outcome by regulatory agencies. Additionally, we are gratified to note that the drug was also well tolerated, and no safety concerns were detected.

“We would like to express our gratitude to the NASH patients who participated in this trial and to their physicians. Their unwavering commitment, over the year-long course of therapy, allowed this trial to be completed in a timely manner, and their dedication to helping others find a treatment for NASH is most laudable.”

Galectin Therapeutics will hold a conference call at 8:30 a.m. Eastern today to discuss the trial results. Peter G. Traber, M.D., the company’s CEO and CMO, and Stephen A. Harrison, M.D., one of lead investigators of the NASH-CX trial, medical director of Pinnacle Clinical Research in San Antonio, Texas, and visiting professor of medicine at the University of Oxford, United Kingdom, will present the results in the webcast.

Dial-in information and webcast details are listed below:

Tuesday, December 5, 2017, 8:30 a.m. Eastern Time
Participant Toll Free Dial-In Number: 888-317-6003
Participant International Dial-In Number: 412-317-6061
Conference ID: 3493752
Webcast URL:
https://services.choruscall.com/links/galt171205.html

Dial-in information and webcast details for replay access are listed below. The dial-in replay will be available until Tuesday, Dec 12, 2017. The webcast replay will be archived for one year.

Replay Toll Free Dial-In Number: 877-344-7529
Replay International Dial-In Number: 412-317-0088
Replay Access Code: 10114808
Webcast Replay:
https://services.choruscall.com/links/galt171205.html

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, which 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 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.

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Forward Looking Statements

This press release 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 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 which could cause actual results to differ materially from those described in the statements.

These statements include those regarding the potential therapeutic benefits of our drugs and specifically the results of our NASH-CX clinical trial. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others that:

- the data presented today represent a top line analysis, and there may be changes in the final clinical trial report due to further analysis of the full data set including additional statistical analysis;
- subsequent trials, if any, in whatever patient population chosen may fail to validate any positive results of our trial now concluded;
- future phases or future clinical studies could prove prohibitively time consuming and/or 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, personnel, and spending projections may change;
- due to the novel nature of our compounds, future phases of manufacturing scale-up and supporting chemical and physical characterizations for both trials and commercial purposes can be challenging and costly and there is no certainty this can be accomplished nor certainty it would acceptable to regulators;
- we may be unsuccessful in developing partnerships or other business relationships with other companies or obtaining capital that would allow us to further develop and/or fund any future studies or trials or sell or license our intellectual property; and, further,
- there is the uncertainty that any drug in development could obtain regulatory approval in any patient population.

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, 2016, 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.

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