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): March 31, 2014

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))

SECTION 8 – OTHER ITEMS

Item 8.01 Other Items.

On March 31, 2014, Galectin Therapeutics Inc. posted a presentation on its website that contains a summary of the results of the first cohort of patients in the Phase 1 clinical trial, which is attached as Exhibit 99.1, and issued the attached press release.

SECTION 9 – FINANCIAL STATEMENTS AND EXHIBITS

Item 9.01 Financial Statements and Exhibits.

(a) Financial Statements of Businesses Acquired.

Not applicable.

(b) Pro Forma Financial Information.

Not applicable.

(c) Shell Company Transactions.

Not applicable.

(d) Exhibits.

Exhibit Number	Description
99.1	Presentation on Phase 1 Clinical Trial: Results of First Cohort
99.2	Press Release dated March 31, 2014

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: March 31, 2014

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

Exhibit 99.1

Galectin Therapeutics

GT-020 Phase 1 Clinical Trial: Results of First Cohort

Release: March 31, 2014
Webcast: April 1, 2014

NASDAQ: GALT
www.galectintherapeutics.com

Forward-Looking Statement

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 which could cause actual results to differ materially from those described in the statements. These statements include those regarding potential therapeutic benefits of GR-MD-02 and expectations regarding the clinical trial, including the future enrollment of patients and the timing of results from the second cohort. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, that results from the first cohort of Phase 1 may differ materially from future results, and there is no guarantee that the current clinical trial will lead to positive outcomes or that GR-MD-02 will ever be approved by the FDA. We may experience delays in the current trial, and we may have difficulty enrolling patients and processing the resulting 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. Regardless of the results of current or future studies, we may be unsuccessful in developing partnerships with other companies or obtaining capital that would allow us to further develop and/or fund any studies or trials. To date, we have incurred operating losses since our inception, and our ability to successfully develop and market drugs 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, 2013, 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.

Our Pipeline Of Galectin-3 Inhibitors

Clinical Focus		Stage of Development				
Drug	**Indication**	**Discovery**	**Pre-clinical**	**Phase 1**	**Phase 2**	**Phase 3**
Fibrosis						
GR-MD-02	Fatty liver disease with advanced fibrosis					
	Lung fibrosis					
	Kidney fibrosis					
Cancer Immunotherapy						
GR-MD-02	Melanoma					
Galectin-3 Inhibitors						
GR-MD-03	Subcutaneous					
GR-MD-04	Oral					
G-XXX*	Oral					

Report on first cohort of Phase 1 Clinical Trial

Timely Reporting:
Last bloods: 3-7-14
Last visit: 3-21-14

*Galectin Sciences, LLC

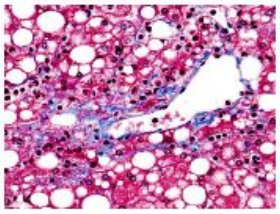
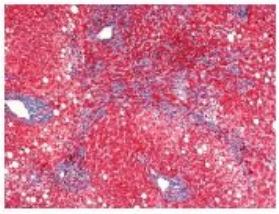
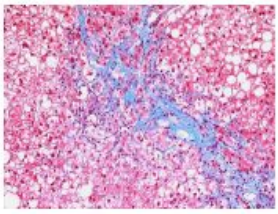
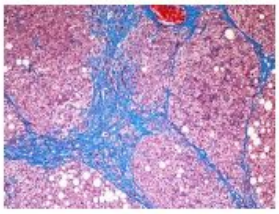
Summary of Findings

- GR-MD-02 was safe and well tolerated at 2 mg/kg (80 mg/m^2) with no drug-related adverse events
- Pharmacokinetics was consistent between individuals and after single and multiple doses; exposure was 40% of lowest dose used in NASH animal model; this was a therapeutic dose
- Key composite biomarkers of fibrosis improved after four doses of GR-MD-02
- Key inflammatory cytokines were decreased after four doses of GR-MD-02
- Patients with greater cellular injury as indicated by elevated ALT levels, had a marked decrease in CK-18, a cell death biomarker
- Galectin-3 blood levels do not correlate with disease activity and are not a biomarker of drug effect in patients with NASH with advanced fibrosis

In addition to being safe and well tolerated, GR-MD-02 improved biomarkers of fibrosis, inflammation and liver cell injury in patients with NASH with advanced fibrosis

All Chronic Liver Diseases Lead To Fibrosis
Example: Liver Fibrosis In Fatty Liver Disease (NASH)

Liver biopsy
Blue=fibrosis

Stage 1	Stage 2	Stage 3	Stage 4
			
Pericellular/Central (High Mag)	Portal/Central	Bridging Fibrosis	Cirrhosis

Patient ← Asymptomatic → Liver failure, Bleeding, Encephalopathy, Edema

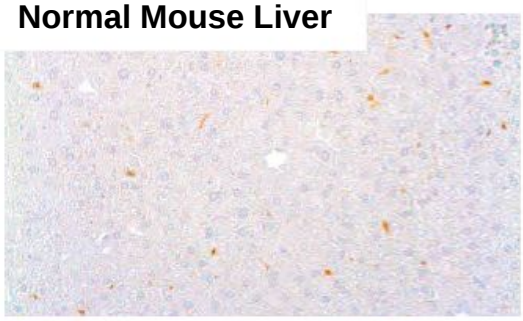
Occurs over decades

Only therapy for patients with cirrhosis is liver transplantation

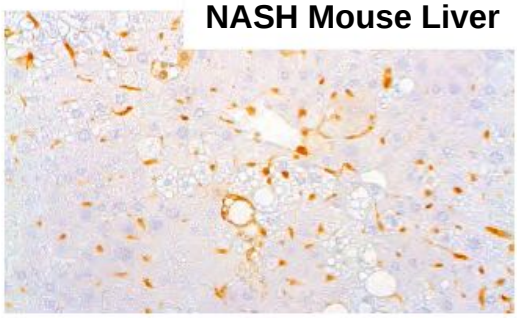
Galectin-3 is Expressed In Liver Macrophages And Is Markedly Increased In Human and Mouse NASH

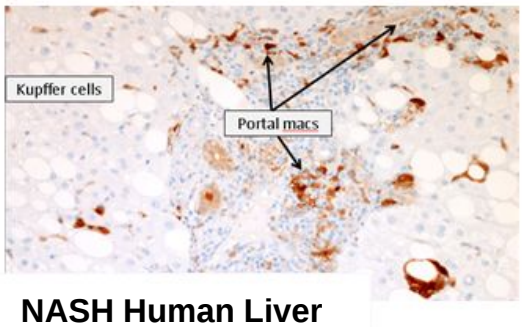
Immunohistochemistry for Gal-3 (brown pigment indicates gal-3)

Normal Mouse Liver



NASH Mouse Liver



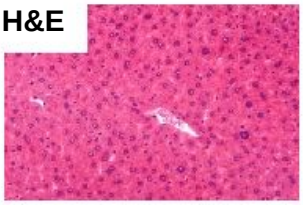
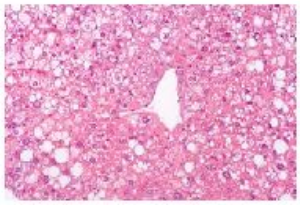
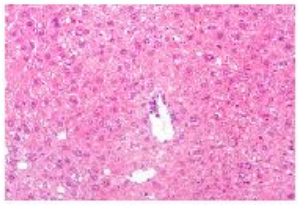
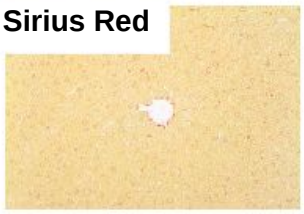
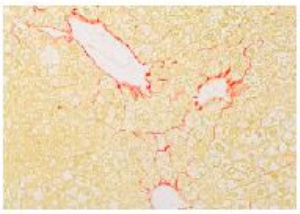
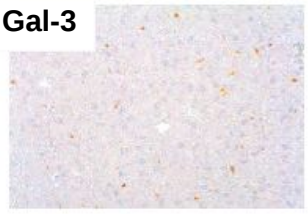
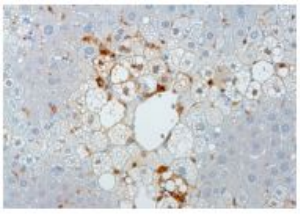
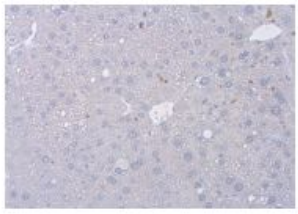


NASH Human Liver

- **Kupffer cells= liver resident macrophages**
- **Portal macs=macrophages located in portal regions**

GR-MD-02, A Galectin-3 Inhibitor, Has Therapeutic Effect On NASH With Fibrosis In Mouse Model

Improvement is linked to decreased tissue Galectin-3

	Normal	NASH:Control	NASH:GR-MD-02	GR-MD-02 Effects
H&E				↓ NAFLD Activity Score • Fat • Cell death • Inflammation
Sirius Red				↓ Collagen (Fibrosis)
Gal-3				↓ Galectin-3 Protein

GR-MD-02 Is A Galectin-3 Inhibitor That Reduces Collagen Synthesis And Increases Collagen Degradation In Pre-Clinical Models

Liver Fibrotic Tissue Homeostasis

In the normal liver, collagen and matrix protein synthesis matches degradation to provide appropriate amount of extracellular matrix.

Normal

Collagen Synthesis = Collagen Degradation

Fibrosis results from increased collagen and other matrix protein synthesis with little to no change in collagen degradation.

Fibrosis

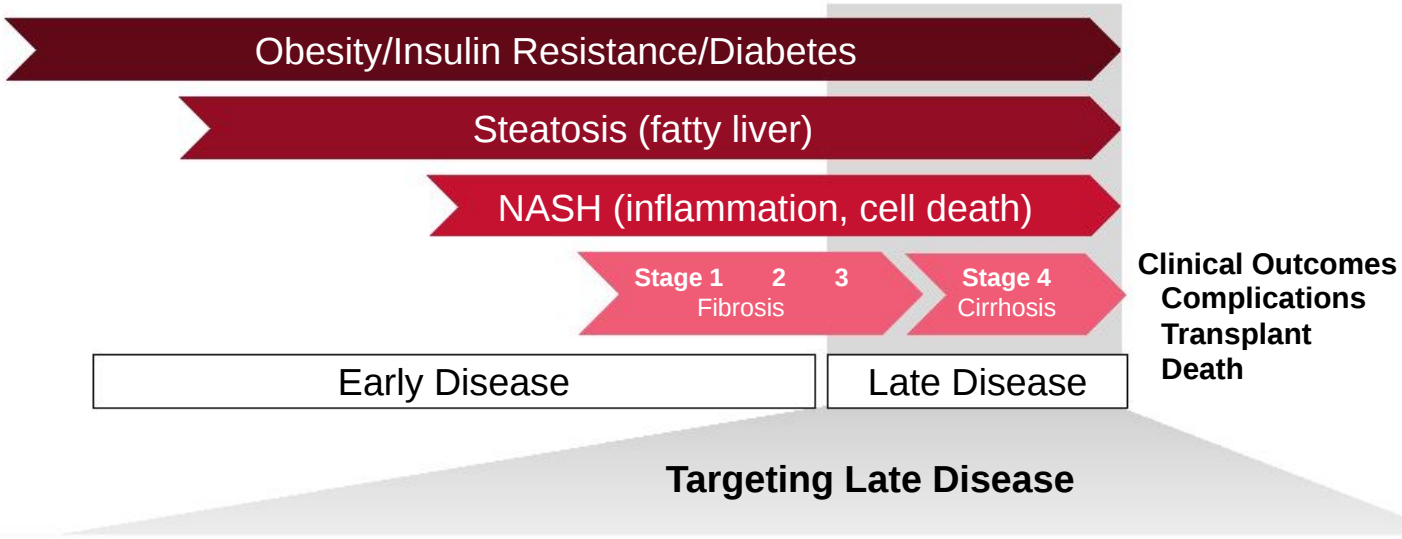
↑ Collagen Synthesis + ↔ Collagen Degradation

Fibrosis can resolve either by a reduction in collagen synthesis or an increase in degradation. The combination would increase rate of resolution.

Restoration to Normal

↓ Collagen Synthesis +/- ↑ Collagen Degradation

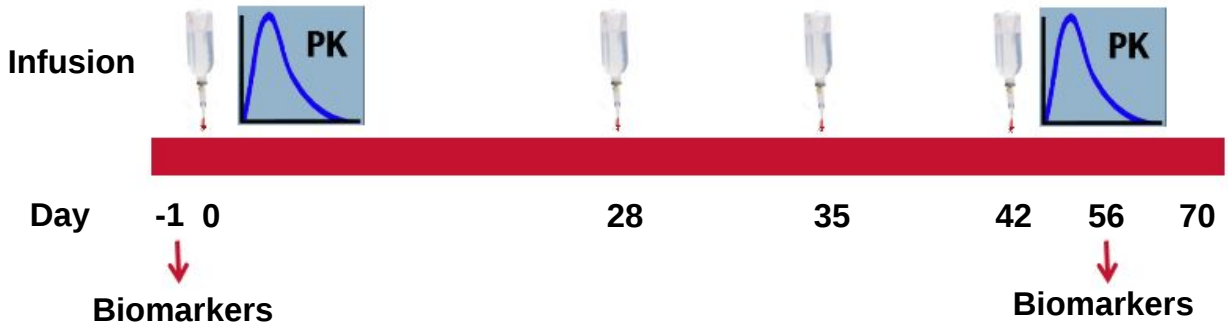
GR-MD-02 Is Being Developed For The Indication Of NASH With Advanced Fibrosis (Stage 3 and 4)



- **No certainty of progression from early to late disease in an individual**
- **Late disease much closer to clinical outcomes**
- **Surrogates of clinical outcomes are better developed for late disease**
- **GR-MD-02 reduces inflammation, ballooning and fat in NASH and reduces existing fibrosis and reverses cirrhosis in animal models**

Phase 1 Clinical Trial Of GR-MD-02 In NASH With Advanced Fibrosis: Report On Cohort 1

Patient inclusion: Biopsy proven NASH with advanced fibrosis (stage 3)
Design: Cohort has 8 patients (6 active, 2 placebo, blinded)
Dose: Starting dose of 2 mg/kg lean body weight (equivalent to 80 mg/m ^{2}); Infusions at days 0, 28, 35 and 42.



Primary endpoints: Safety
Pharmacokinetics

Secondary endpoints: Disease-related serum biomarkers to assess for potential treatment effect

Patient Characteristics & Safety

Patient Characteristics

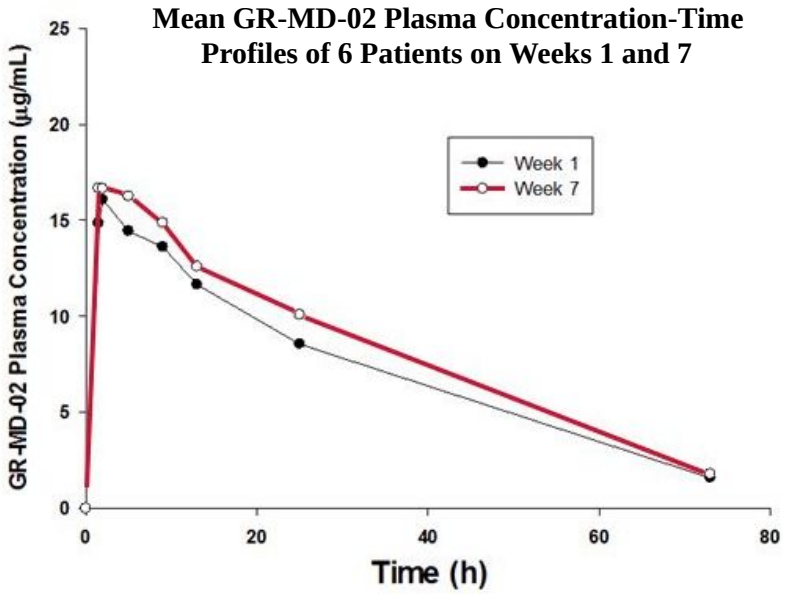
- 6 women and 2 men
- Ages 40-64 (mean=54)
- Mean body mass index (BMI)=39 (obese >30)
- Diabetes Mellitus in 6 patients
- Patients had a liver biopsy within one year of enrollment
  - All patients had definitive pathological diagnosis of NASH
  - 7 patients had stage 3 fibrosis (bridging); 1 patient had stage 4 fibrosis
- All patients enrolled completed full protocol through final follow-up visit at day 70.
- Last subject, last blood draw was 3-7-14; Last subject, last visit was 3-21-14

Patient Safety

- There were no Serious Adverse Events
- There were no Treatment Emergent Adverse Events in patients receiving GR-MD-02 that were attributed to the drug
- One patient receiving GR-MD-02 had several mild AE's that were judged by investigator to be unrelated to drug
- Two patients receiving placebo had mild AE's that were judged by investigator as possibly related
- There were no treatment emergent laboratory or ECG findings

GR-MD-02 at a dose of 2 mg/kg (80 mg/m^2) was safe and well tolerated

Pharmacokinetics: GR-MD-02 Blood Levels Were Consistent Between Individuals And Not Significantly Different After Single Or Multiple Infusions



First Infusion (week 1)

C_{max} = 16.3 µg/mL
$T_{1/2}$ = 19.9 h
AUC = 572.6 h*µg/mL
V_{ss} = 5.2 L
Variability ≤ 15%

Fourth Infusion (week 7)

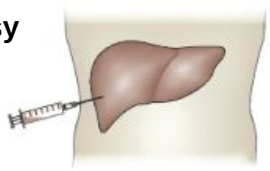
C_{max} = 17.7 µg/mL
$T_{1/2}$ = 20.5 h
AUC = 645.4 h*µg/mL
V_{ss} = 4.7 L
Variability ≤ 24%

Accumulation ratio ~1.16
(95% CI 0.85 to 1.47)

The AUC in humans given 2 mg/kg was approximately 40% of the AUC of the lowest therapeutic dose in the mouse NASH model

Assessment Methods for Liver Fibrosis

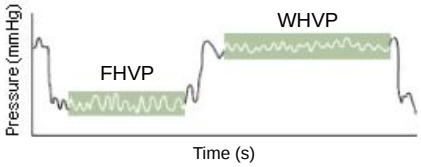
Liver biopsy



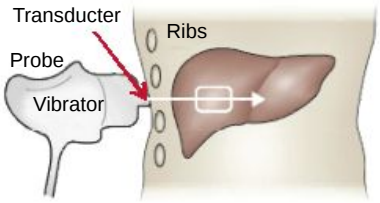
- Potential serious complications
- 1/50,000th of liver
- High sampling variability
- 41% discordance of 1 fibrosis stage in NASH*

Serum markers

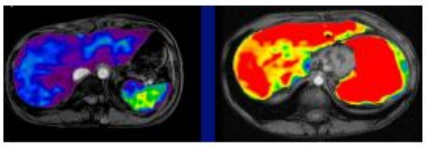
Hepatic venous pressure gradient (HVPG)



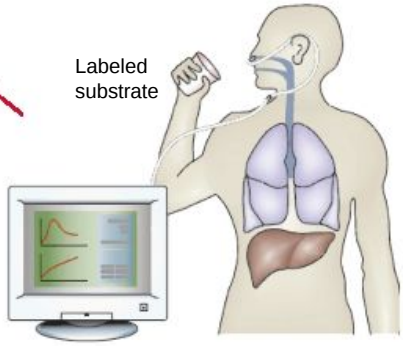
Transient Elastography (FibroScan™)



MR-Elastography



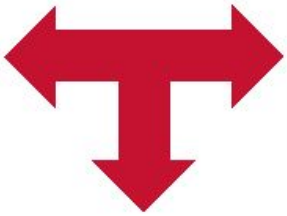
Functional metabolic and shunt tests (eg. HepQuant™)



Source: Nat Rev Gastroenterol Hepatol. 2010 Aug;7(8):425-36.Epub 2010 Jun 29. *Ratziu, et al. Gastro. 2005

Major Pathological Processes in NASH

Steato-Hepatitis (NASH Activity)
- Ballooning of liver cells (cell death/apoptosis) key hallmark
- Fat in liver cells (steatosis)
- Immune cell infiltration (inflammation)



Fibrosis/Cirrhosis
- Increase in collagen/matrix
- Disruption of architecture
- Liver cell nodules

Do Not Always Correlate in Same Patient

- Can have high NASH activity score with minimal fibrosis
- Can have advanced fibrosis/cirrhosis with minimal NASH activity

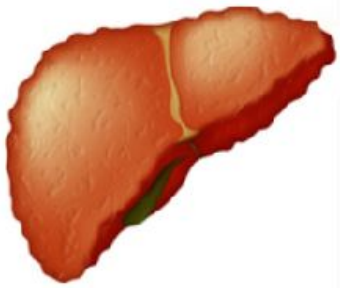
We measured biomarkers of both major pathological processes

Serum Biomarkers Of Fibrosis In NASH

Composite Scores

FibroTest™ (FibroSURE™)
- Indirect biomarker of fibrosis
- Age and gender, Alpha-2-macroglobulin, Haptoglobin, Apolipoprotein A1, GGTP, Total bilirubin

ELF (Enhanced Liver Fibrosis) Score
- Direct biomarker of fibrosis
- Hyaluronic acid
- TIMP1 (tissue inhibitor of metalloproteinase-1)
- P3NP (amino terminal propeptide of type III pro-collagen)



Individual Markers

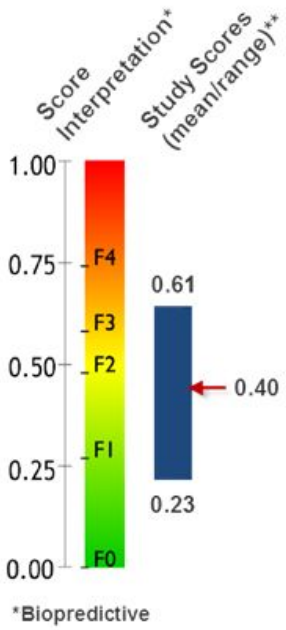
Hyaluronic Acid
- Matrix polysaccharide
- Direct marker
- Correlates to fibrosis

Exploratory*
- TGF-β
- Lumican
- Osteopontin
- Matrix Metalloproteinases

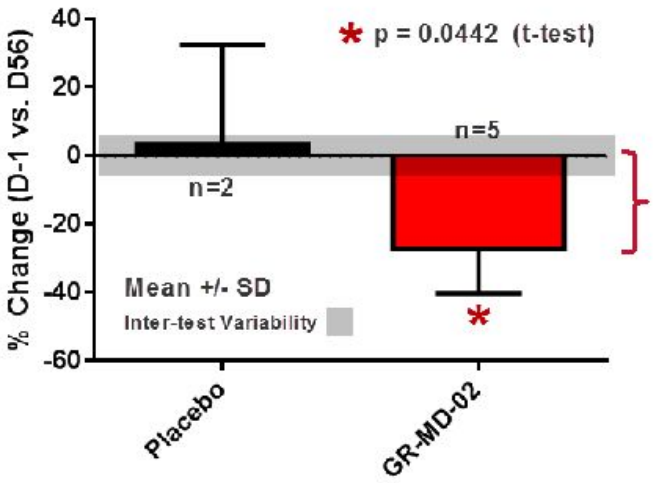
* Indicates that there is some evidence that suggests they are increased in fibrosis, but not confirmed in sufficient number of patients or studies

For more information and references on biomarkers: http://bit.ly/1jzFK50

FibroTest™ (FibroSURE™) Scores Significantly Decreased In GR-MD-02 Treated Patients



**One patient on GR-MD-02 had scores < 0.08 which was highly discordant with biopsy (stage 3). Patient had high haptoglobin which is known for false negative test.

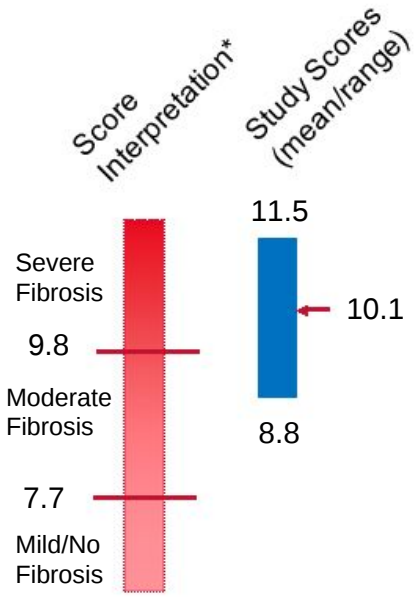


FibroTest™ has been shown to:

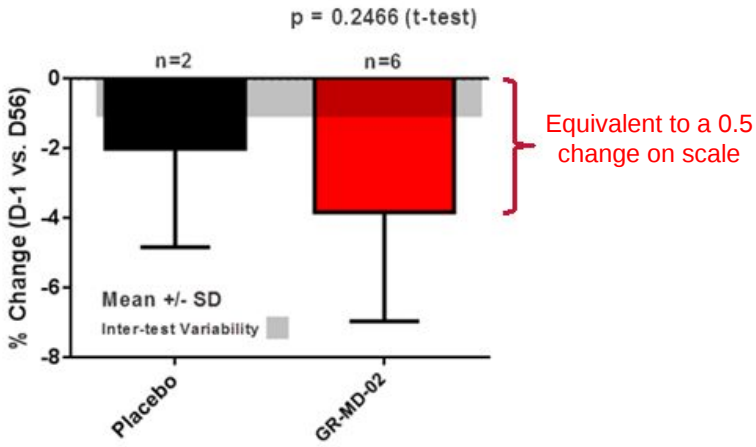
- Correlate with stage of fibrosis
- Assess fibrosis regression
- Assess fibrosis progression
- Predict liver-related mortality

Note: While the numbers are small, exploratory statistics have been performed to evaluate differences using a one-sided t-test and confirmed using a non-parametric test, Mann-Whitney

ELF Score Tended To Decrease In GR-MD-02 Treated Patients



*Siemens Diagnostics



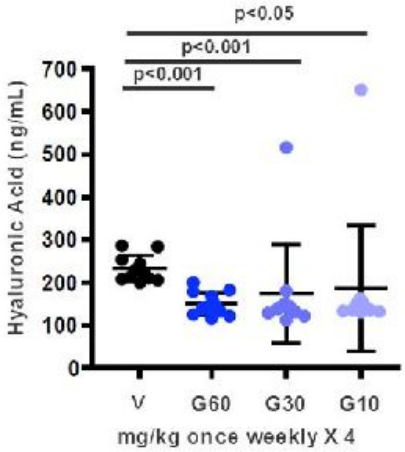
ELF score has been shown to:

- Correlate with stage of fibrosis
- Assess progression (0.032 increase/yr in PBC)
- Predict mortality (a one unit change in score correlates to a 2-fold change in liver related mortality)

Hyaluronic Acid (HA) Levels Were Decreased In A Subset Of Patients On GR-MD-02

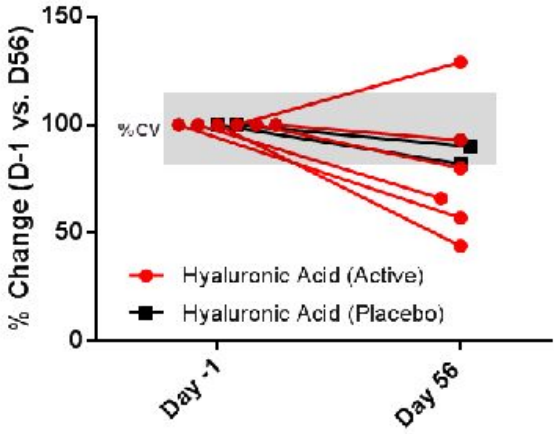
Animal Study

- HA levels measured in NASH mice treated with GR-MD-02
- HA levels decreased at all three doses compared to vehicle-treated controls
- Some animals had variable levels



Study Results

- 3 of 6 patients treated with GR-MD-02 had significant reductions in HA
- No change in placebo patients
- Multiple clinical studies have shown that HA levels correlate with liver fibrosis



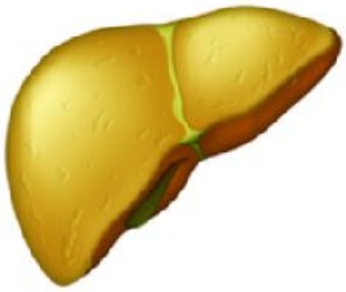
No consistent elevation and/or changes in Osteopontin, TGF-β or MMPs; Lumican presented in later slides

Serum Biomarkers of NASH Inflammation and Injury

Inflammatory Cytokines

Key cytokines*
- IL-6
- IL-8
- TNF-α

Exploratory**
- INF-γ
- Endothelin-1
- IP-10
- VEGF
- CD40-ligand



* Evidence of association with human NASH and importance in pathogenesis, particularly as products of macrophages

** Some evidence of association with human and/or animal NASH in at least one publication

Cellular Injury

Serum Transaminases
- ALT and AST
- Enzymes released from liver cells
- 2/3 of NASH patients have normal levels at any given time
- Entire spectrum of disease can be seen with normal levels

Cell Death (Apoptosis)

Cytokeratin 18
- A circulating biomarker of cell death
- Predictive of NASH severity

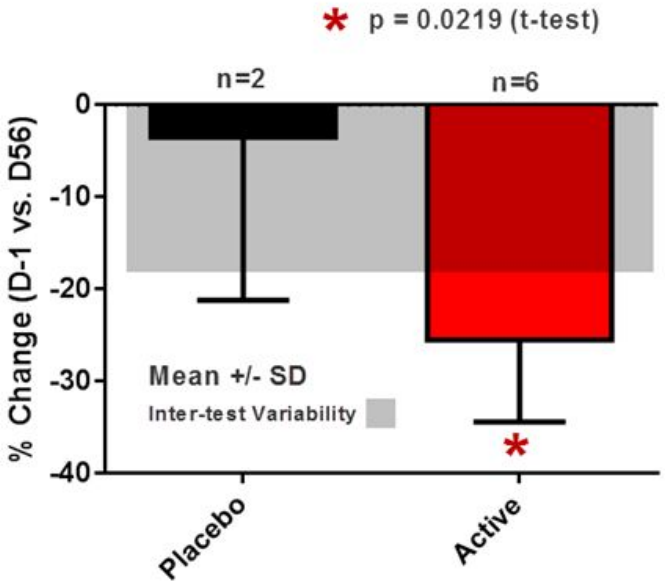
For more information and references on biomarkers: http://bit.ly/1jzFK50

Interleukin-8 Levels Were Significantly Reduced In GR-MD-02 Treated Patients

- Pro-inflammatory cytokine expressed in macrophages
- Elevated serum levels in NASH
- Study patients had elevated serum levels
- GR-MD-02 treated patients had significant reduction when compared to placebo

	Study Cohort*	NAFLD**	Obese Controls**
IL-8 pg/mL	28.0 ± 8.6	24.1 ± 38.5	7.8 ± 3.6

*Baseline levels
**Jarrar, et al. Aliment. Pharmacol. Ther. 2007

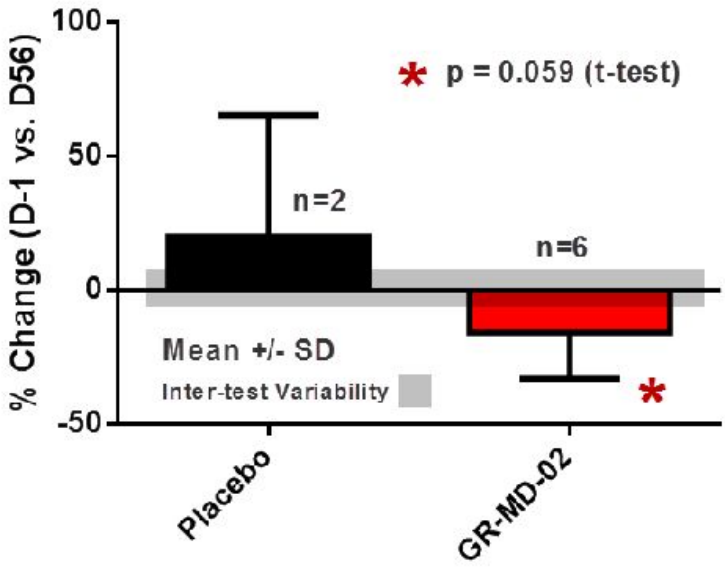


TNF-α Levels Were Significantly Reduced In GR-MD-02 Treated Patients

- Pro-Inflammatory cytokine and promotes lipid accumulation
- Elevated serum levels in NASH
- Study patients had elevated serum levels
- GR-MD-02 treated patients had significant reduction when compared to placebo

	Study Cohort*	NAFLD**	Obese Controls**
TNF-α pg/mL	23 ± 5.8	6.0 ± 16.6	1.9 ± 0.3

*Baseline levels
**Jarrar, et al. Aliment. Pharmacol. Ther. 2007

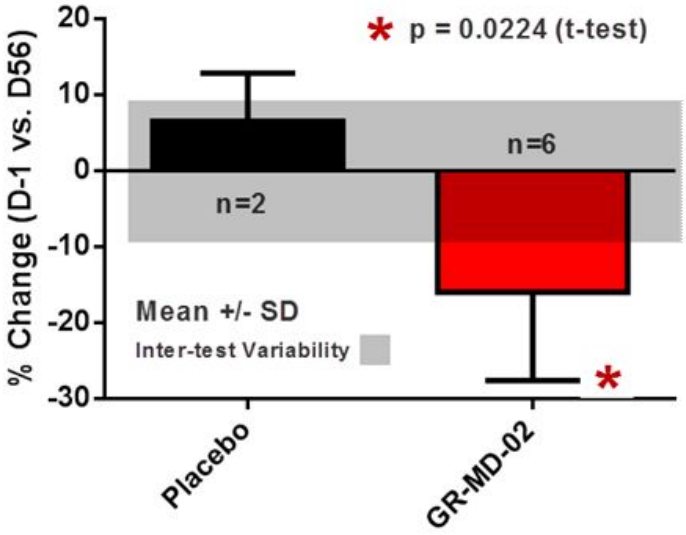


Interleukin-6 Levels Were Significantly Reduced In GR-MD-02 Treated Patients

- Pro-Inflammatory cytokine secreted by T cells and macrophages.
- Increased serum levels in NASH
- Levels not increased in patients
- GR-MD-02 treated patients had significant reduction when compared to placebo

	Study Cohort*	NAFLD**	Obese Controls**
IL-6 pg/mL	6.1 ± 2.5	23.1±72.9	7.6±6.3

*Baseline levels
**Jarrar, et al. Aliment. Pharmacol. Ther, 2007

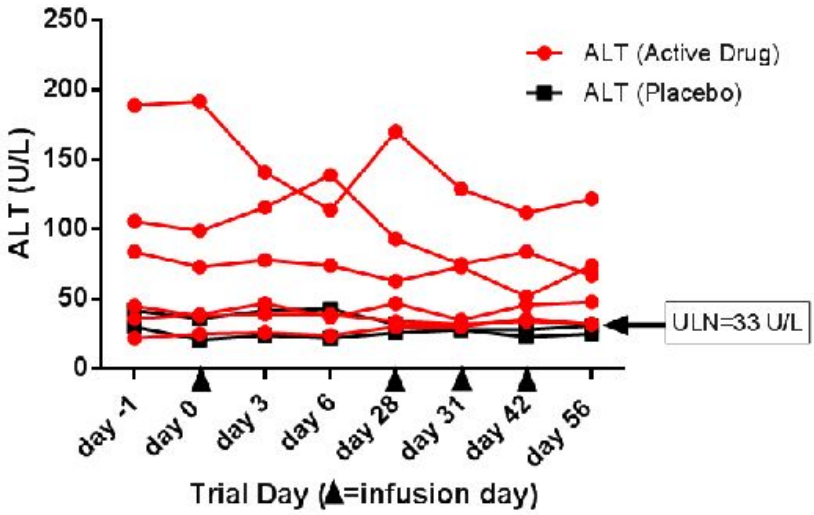


Exploratory cytokines were not elevated and/or did not change including INF-γ, Endothelin-1, IP-10, VEGF, CD40-ligand

Markedly Elevated Alanine Aminotransferase (ALT) Levels Decreased With GR-MD-02 Treatment

- Typical for NASH patients, there was a broad range of baseline ALT levels
- Those with ALT levels below 50 U/L had no change with therapy
- Two patients with ALT above 100 U/L, both of whom received active drug, had reductions of 39 U/L and 67 U/L
- One patient with ALT between 50 and 100 had minimal reduction of 10 U/L

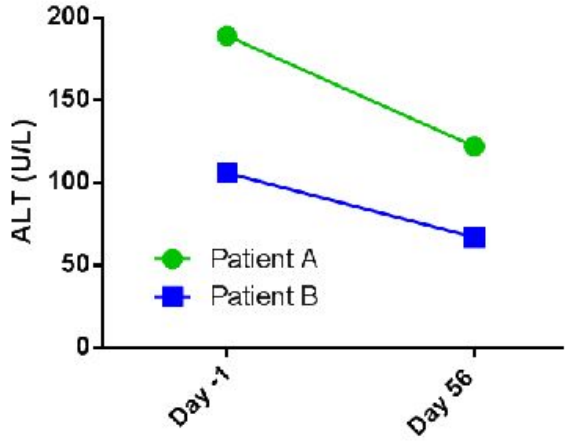
100-200 (2 patients)
50-100 (1 patient)
0-50 (5 patients)



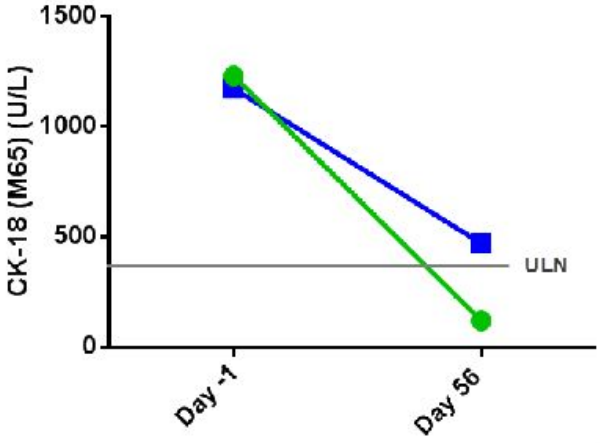
Cell Death Biomarker CK18 Was Reduced In Two Patients With Highest ALT Levels

- **CK-18, a biomarker of cell death of hepatocytes, was markedly reduced in the two patients with ALT greater than 100 U/L**

ALT

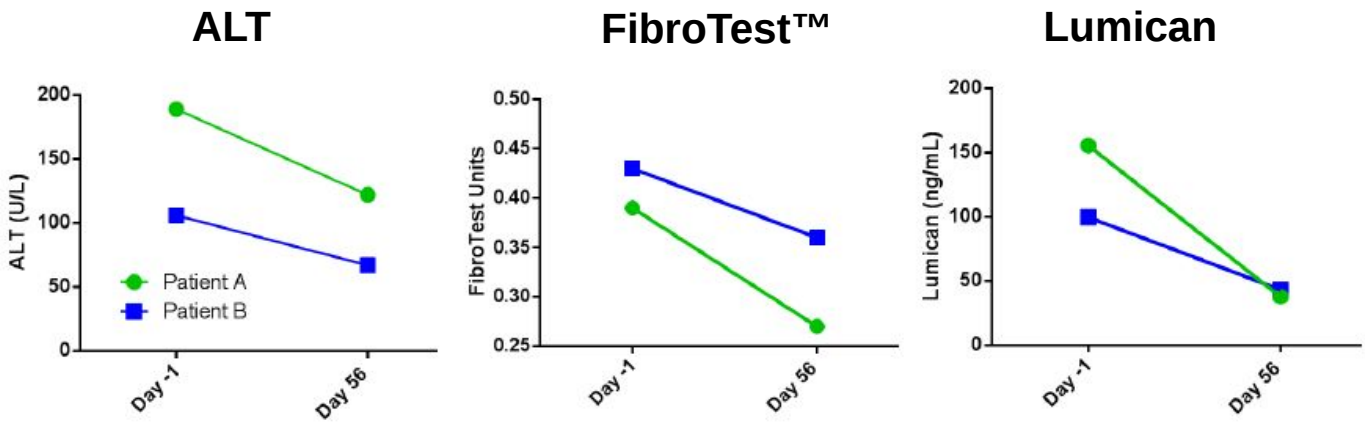


CK-18 (M65)



Fibrosis Biomarkers Were Reduced In The Two Patients Receiving GR-MD-02 With Highest ALT*

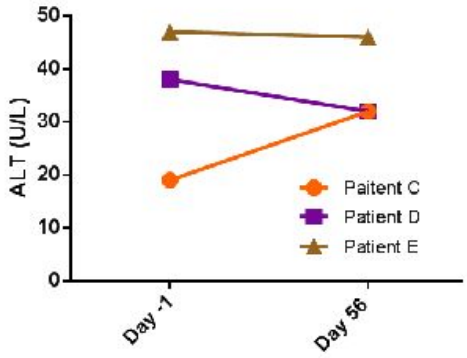
- FibroTest™ scores were markedly decreased in the high ALT patients after treatment with GR-MD-02
- Lumican, a matrix protein that is involved in fibrogenesis in the liver, was elevated in all patients, but was highest and had the greatest decrease with treatment in the two patients with high ALT levels



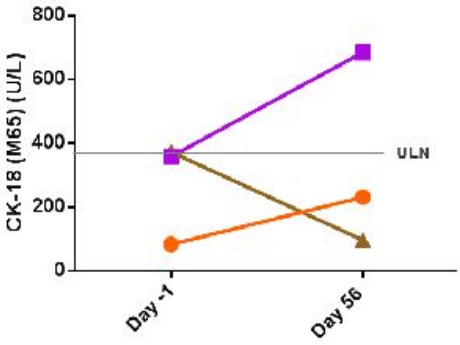
*Patient with intermediate ALT not included in analysis because of false negative FibroTest™ score

Patients With Low ALT Levels Receiving GR-MD-02 Had Improvement In Fibrosis Markers But Not Cell Death Markers

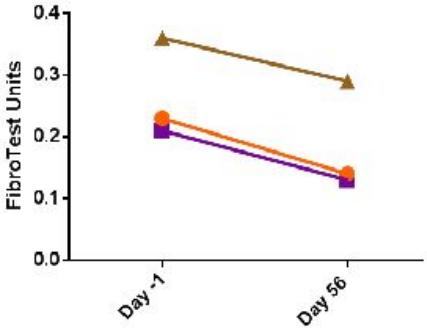
ALT



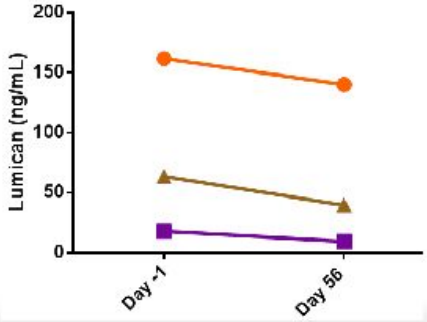
CK-18 (M65)



FibroTest™



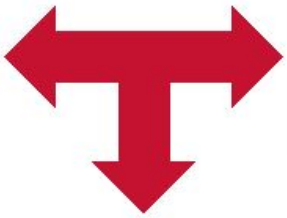
Lumican



- The three GR-MD-02 treated patients with low ALT levels did not have changes in ALT
- These three patients had lower CK-18 levels which did not decrease with therapy
- Fibrosis markers of FibroTest™ and Lumican did improve with treatment

GR-MD-02 Treatment Appears To Improve Both Major Pathological Processes In NASH

Steato-Hepatitis (NASH Activity)
- Ballooning of liver cells (cell death/apoptosis) key hallmark
- Fat in liver cells (steatosis)
- Immune cell infiltration (inflammation)



Fibrosis/Cirrhosis
- Increase in collagen/matrix
- Disruption of architecture
- Liver cell nodules

- Improvement in Fibrosis Biomarkers: There was a statistically significant reduction in Fibrotest™ and trends towards a reduction in ELF score and hyaluronic acid
- Improvement in Inflammation Biomarkers: There were statistically significant reductions in IL-6, IL-8 and TNF-a, all important cytokines in NASH
- Improvement in Cell Death Biomarkers: A patient subset with high ALT levels indicative of more cellular injury had improvement in CK-18

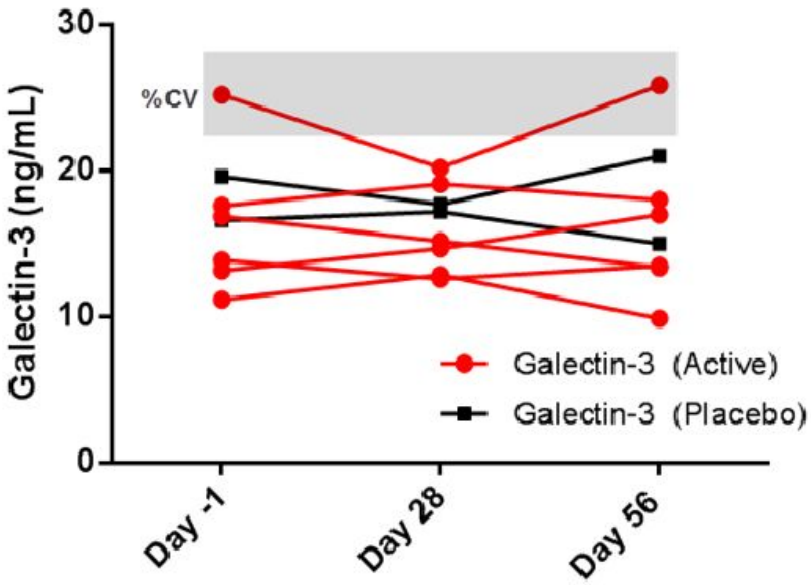
Patients Had A Normal Range Of Blood Galectin-3 Levels At Baseline And No Change With Treatment

	Cohort Range*	Normal Range**
Gal-3 (ng/mL)	**13.2 to 25.2**	**5.4 to 26.2**

*Baseline values
**Range of values in a non-diseased population published by manufacturer (BG Medicine)

Timing of gal-3 levels

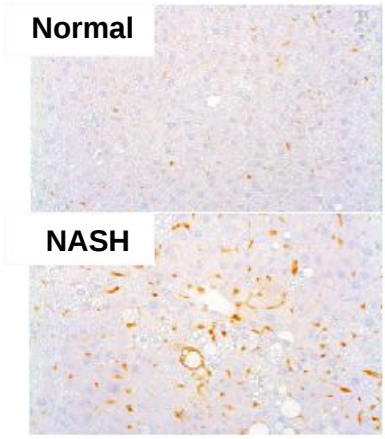
- Before 1 st infusion
- 28 day after 1 st infusion
- 14 days after 4 th infusion



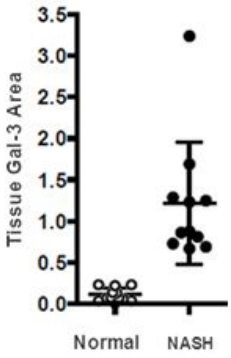
Blood and Tissue Levels Of Galectin-3 Are Not Correlated In Mouse NASH Model Nor Human NASH

Marked changes in expression of galectin-3 in liver macrophages are not reflected in changes in blood galectin-3

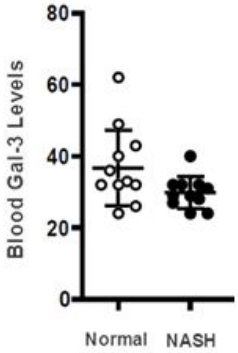
Liver Gal-3 Staining



Liver Tissue Level



Blood Level



- No evidence for correlation between blood galectin-3 levels and disease activity or fibrosis stage in patients with NASH[1]
- Blood galectin-3 levels in humans are correlated with obesity[1,2] and diabetes[2]

[1]Yilmax, et al. Clinical Biochemistry 2011 [2]Weigert, et al. J Clin Endocrinol Metab, 2010

Summary of Findings

- GR-MD-02 was safe and well tolerated at 2 mg/kg (80 mg/m^2) with no drug-related adverse events
- Pharmacokinetics was consistent between individuals and after single and multiple doses; exposure was 40% of lowest dose used in NASH animal model; this was a therapeutic dose
- Key composite biomarkers of fibrosis improved after four doses of GR-MD-02
- Key inflammatory cytokines were decreased after four doses of GR-MD-02
- Patients with greater cellular injury as indicated by elevated ALT levels, had a marked decrease in CK-18, a cell death biomarker
- Galectin-3 blood levels do not correlate with disease activity and are not a biomarker of drug effect in patients with NASH with advanced fibrosis

In addition to being safe and well tolerated, GR-MD-02 improved biomarkers of fibrosis, inflammation and liver cell injury in patients with NASH with advanced fibrosis

Next Steps: Continuation of Phase 1 Trial

- The dose of GR-MD-02 will be increased to 4 mg/kg (160 mg/m^2) in the second cohort of 8-10 patients
- Eight clinical sites in the US are now active to facilitate rapid enrollment of cohort 2
- FibroScan™, a FDA-approved ultrasonic measure of liver tissue elasticity, has been added to the protocol for cohort 2. FibroScan™ will be performed at baseline and after the four doses in as many patients as possible to gain experience with this method of fibrosis assessment.
- Results from Cohort 2 are expected to be reported in July-August 2014 time frame.
- Planning for phase 2 clinical trials is ongoing. The results of the first cohort suggest that 2 mg/kg is a safe, well-tolerated dose that has indication of anti-fibrotic and anti-inflammatory effect. Therefore, this defines at least one potential dose level for phase 2 clinical trials.

First Cohort Results in Galectin Therapeutics' Phase 1 Trial Reveal Biomarker Evidence of Therapeutic Effect on Fibrosis and Inflammation in NASH with Advanced Fibrosis

- ***GR-MD-02 was safe and well tolerated in patients***
- ***Galectin Therapeutics to host webcast, April 1, 8:30 a.m. EDT to discuss first cohort findings***
- ***Second cohort of Phase 1 trial to begin enrollment in April***

Norcross, Ga., March 31, 2014 – Galectin Therapeutics (Nasdaq:GALT), the leading developer of therapeutics that target galectin proteins to treat fibrosis and cancer, today announced that results from the first cohort of its Phase 1 trial show that GR-MD-02 had an effect on biomarkers that suggest a therapeutic effect on fibrosis, inflammation, and cellular injury. The first-in-man study, which enrolled eight patients in the first cohort, is evaluating the safety, tolerability, and exploratory biomarkers for efficacy for single and multiple doses of its galectin-inhibiting drug GR-MD-02 when administered to patients with fatty liver disease (NASH) with advanced fibrosis.

First cohort results indicate that GR-MD-02 was safe and well tolerated following four doses of 2 mg/kg (80 mg/m^2) and there were no serious adverse events. The pharmacokinetics were consistent between individuals and after single and multiple doses with no drug accumulation after multiple doses. In assessing secondary endpoints, it was found that multiple biomarkers of fibrosis and inflammation showed improvement after four doses of GR-MD-02. Additionally, patients with greater evidence of liver cell injury, as indicated by elevated transaminase enzyme levels, had a marked decrease in CK-18, a clinically validated biomarker of cell death. Galectin-3 blood levels, which do not correlate with tissue levels in NASH, were not changed with treatment.

Details of the findings will be discussed by the Company on a webcast and conference call on Tuesday, April 1 at 8:30 a.m. Eastern Daylight Time. The webcast can be accessed at the following link: http://w.on24.com/r.htm?e=773833&s=1&k=A95A6017BF0762550B5252D80F9A24FF. Audio only can be accessed using the following call-in number: 866-219-3563, conference ID 19710441. The presentation is now posted on the Company's website (www.galectintherapeutics.com) for review before the webcast.

"We are extremely pleased with the positive results of the first cohort of our Phase 1 trial, which suggest a role for GR-MD-02 in the treatment of patients with fatty liver disease with advanced fibrosis," said Peter G. Traber, M.D., Chief Executive Officer, President and Chief Medical Officer of Galectin Therapeutics. "Fatty liver disease, characterized by the presence of fat in the liver along with inflammation, over time can develop into fibrosis, or scarring of the liver, which is estimated to affect millions of Americans. Intervention with the intent of reversing the fibrosis is a potentially important therapeutic approach in fatty liver disease, a condition with significant unmet medical need."

The Phase 1 multi-center, blinded (to healthcare providers and patients) clinical trial is being conducted in patients with NASH with advanced fibrosis (Brunt Stage 3) who receive four doses of GR-MD-02 over a 42-day period. Each of the three planned cohorts consists of eight patients, six randomized to receive active drug and two randomized to receive placebo. Eight U.S. clinical sites with extensive experience in clinical trials in liver disease are now active to facilitate rapid enrollment of the second cohort. Trial design details can be found at http://clinicaltrials.gov/ct2/show/NCT01899859?term=gt-020&rank=1.

The second cohort, which will begin enrollment in April, will include an increased dose of GR-MD-02 to 4 mg/kg (160 mg/m^2). FibroScan™, an ultrasonic measure of liver tissue elasticity approved by the U.S. Food and Drug Administration, was added to the trial's protocol to provide another assessment of liver fibrosis. For those sites with access to FibroScan™, measurements will be performed at baseline and after the four doses. It is estimated results from the second cohort will be reported in late summer 2014.

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 scaring of organs including fibrotic disorders of the liver, lung, kidney, heart and vascular system. The drug binds to galectin proteins and disrupts their function. Preclinical data has shown that GR-MD-02 has robust treatment effects in reversing liver fibrosis and cirrhosis.

About Fatty Liver Disease with Advanced Fibrosis

Non-alcoholic steatohepatitis (NASH), also known as fatty liver disease, has become a common disease of the liver with the rise in obesity rates, estimated to affect nine to 15 million people, including children, in the U.S. Fatty liver disease is characterized by the presence of fat in the liver along with inflammation and damage in people who drink little or no alcohol. Over time, patients with fatty liver disease can develop fibrosis, or scarring of the liver, and it is estimated that as many as three million individuals will develop cirrhosis, a severe liver disease where liver transplantation is the only current treatment available. Approximately 6,300 liver transplants are done on an annual basis in the U.S. There are no drug therapies approved for the treatment of liver fibrosis.

About Galectin Therapeutics

Galectin Therapeutics (NASDAQ: GALT) is developing promising carbohydrate-based therapies for the treatment of fibrotic liver disease and cancer based on the Company's unique understanding of galectin proteins, key mediators of biologic function. We are leveraging extensive scientific and development expertise as well as established relationships with external sources to achieve cost effective and efficient development. We are pursuing a clear development pathway to clinical enhancement and commercialization for our lead compounds in liver fibrosis and cancer. Additional information is available at www.galectintherapeutics.com.

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 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 which could cause actual results to differ materially from those described in the statements. These statements include those regarding the clinical trial, including the expected timing of results for the second cohort, and potential benefits and therapeutic effects of GR-MD-02. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, that we may not be successful in developing effective treatments and/or obtaining the requisite approvals for the use of GR-MD-02 or any of our other drugs in development. Our current clinical trial and any future clinical studies may not produce positive results in a timely fashion, if at all, and could prove time consuming and costly. We may have difficulty enrolling new patients, which could impact timing and costs. Results from the first cohort of Phase 1 are not necessarily indicative of future results in the clinical trial. 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. Regardless of the results of any of our development programs, we may be unsuccessful in developing partnerships with other companies that would allow us to further develop and/or fund any studies or trials. To date, we have incurred operating losses since our inception, and our ability to successfully develop and market drugs 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, 2013, 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.

Contact

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