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

November 12, 2012
Date of Report (Date of earliest event reported)

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 Blvd., Suite 240
Norcross, GA 30071
(Address of principal executive offices) (Zip Code)

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

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))

Item 7.01. Regulation FD Disclosure.

On November 12, 2012, Galectin Therapeutics Inc. (the "Company) issued a press release (the "Release") containing information presented by Company executives at the American Association for the Study of Liver Disease (AASLD) Annual Meeting in Boston, MA on November 12, 2012 regarding new preclinical data on the mechanism of action of GR-MD-02. Exhibit 99.1 and Exhibit 99.2, which are being furnished and not filed herewith, contain the text of the Release and the Presentation.

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.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1 Press Release Issued November 12, 2012

99.2 Presentation November 12, 2012

SIGNATURES

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

GALECTIN THERAPEUTICS INC.

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

Date: November 13, 2012

Exhibit 99.1

FOR IMMEDIATE RELEASE

Galectin Therapeutics Presents New Data on the Treatment of Fatty Liver Disease and Fibrosis at AASLD 2012

Norcross, GA, November 12, 2012 – Galectin Therapeutics (NASDAQ: GALT), the leading developer of therapeutics that target galectin proteins to treat fibrosis and cancer, today presented new preclinical data on the mechanism of action of GR-MD-02 at the American Association for the Study of Liver Disease (AASLD) Annual Meeting in Boston, MA. GR-MD-02 is the Company's lead galectin inhibitor in development for the treatment of non-alcoholic steatohepatitis (NASH), or fatty liver disease. These new data help to further explain the mechanism of action of GR-MD-02, showing that this galectin inhibitor affects multiple pathways involved with both the prevention and reversal of fibrosis in NASH pathology.

"The data presented at AASLD further elucidate the mechanism of how galectin inhibition affects liver fibrosis in preclinical models of disease, resulting in the prevention and reversal of fibrosis," said Peter G. Traber, MD, President, Chief Executive Officer and Chief Medical Officer, Galectin Therapeutics. "As we continue to advance GR-MD-02 for the treatment of NASH with advanced fibrosis, we are hopeful that galectin inhibition could provide patients with a novel treatment option, where liver transplantation is currently the only therapy available. GR-MD-02 is expected to enter the clinic in the first quarter of 2013."

The presentation, entitled "Galectin-3 targeting drugs inhibit multiple pathological pathways leading to improvement of non-alcoholic steatohepatitis (NASH)", was authored by Peter G. Traber and Eliezer Zomer. As previously demonstrated, GR-MD-02 treatment in a mouse model of NASH resulted in marked improvement in liver histology with significant reduction in steatosis, ballooning and inflammation, as well as fibrosis, determined by Sirius red staining. This disease improvement upon treatment with GR-MD-02 was seen when animals were treated early in disease (disease prevention) or after fibrosis had been established (disease reversal).

The new data show that Galectin-3 protein expression was markedly increased in animals with NASH, and those levels were dramatically reduced to barely detectable levels following treatment with GR-MD-02. Elevated expression of iNOS, an important inflammatory mediator, and CD36, a scavenger receptor involved in the pathogenesis of NASH, were markedly reduced following treatment with GR-MD-02. Alpha-smooth muscle actin, a marker used to identify activated cells that cause liver fibrosis, showed increased numbers of cells in control livers, which was markedly reduced in livers treated with GR-MD-02. Together, these data suggest that GR-MD-02 works to prevent or reverse fibrosis in NASH by reducing galectin-3, which is associated with multiple pathogenic effects.

About NASH

NASH is a common disease of the liver, affecting 9 to 15 million people in the United States. NASH 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 NASH can develop fibrosis, or scarring of the liver, that can lead to cirrhosis, a severe liver disease where transplantation is the only current treatment available.

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. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others: incurrence of operating losses since our inception, uncertainty as to adequate financing of our operations, extensive and costly regulatory oversight that could restrict or prevent product commercialization, inability to achieve commercial product acceptance, inability to protect our intellectual property, dependence on strategic partnerships, product competition, and others stated in risk factors contained in our SEC filings. We cannot assure that we have identified all risks or that others may emerge which we do not anticipate. 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

Galectin Therapeutics Inc.
Peter G. Traber, MD, 678-620-3186
President, CEO, & CMO
ir@galectintherapeutics.com

Exhibit 99.2

Galectin-3 Targeting Drugs Inhibit Multiple Pathological Pathways Leading to Improvement of Non-Alcoholic Steatohepatitis (NASH)

Peter G. Traber, MD and Eliezer Zomer, PhD *Galectin Therapeutics Inc.*
4960 Peachtree Industrial Boulevard, Norcross, GA USA

Galectin Therapeutics

www.galectintherapeutics.com

Introduction:

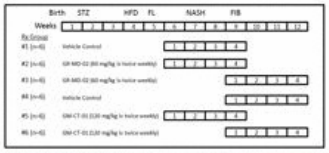
- Steatohepatitis, or NASH (non-alcoholic steatohepatitis), consists of fat accumulation, hepatocellular degeneration and necrosis, lobular inflammation, and fibrosis which can lead to cirrhosis.
- NASH affects up to 5% of the U.S. population and there is currently no accepted medical treatment for NASH or fibrosis.
- The galactose binding protein galectin-3 has been implicated in the pathogenesis of NASH.

Objective:

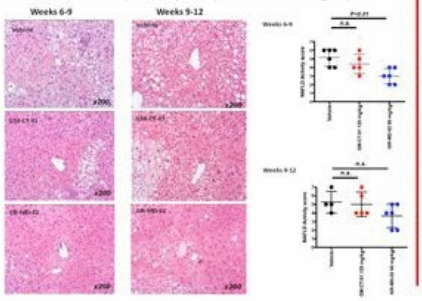
- To evaluate the efficacy and mechanism of novel complex carbohydrate drugs that inhibit galectin proteins in the treatment of NASH.

Methods:

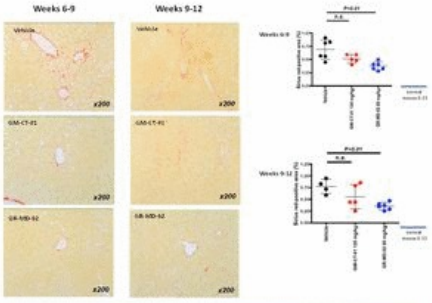
- NASH was induced in mice by making them diabetic and feed a high fat diet, which reproducibly caused steatohepatitis with fibrosis (Stelic Institute & Co., Tokyo, Japan).
- NASH mice were treated with either vehicle as a control or various concentrations of GM-CT-01 (galactomannan) or GR-MD-02 (arabinogalacto-rhamnogalacturonan), both of which bind galectin-3.

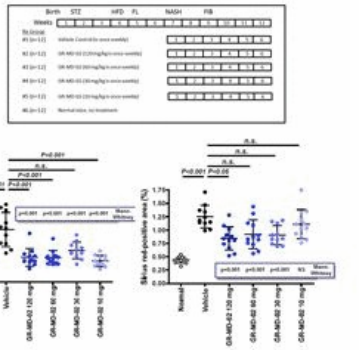


Treatment with GR-MD-02 markedly improved the NAFLD Activity Score (fat deposition, hepatocellular ballooning degeneration and necrosis, inflammatory infiltrate) in early and late treatment groups.

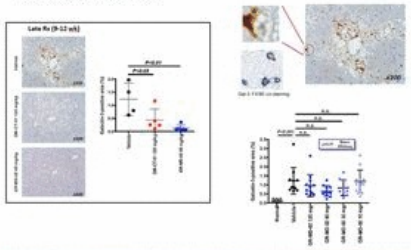


Collagen deposition was evaluated by digital morphometric analysis following Sirius Red staining. Treatment with GR-MD-02 reduced collagen deposition to normal levels whereas GM-CT-01 had a less marked effect.

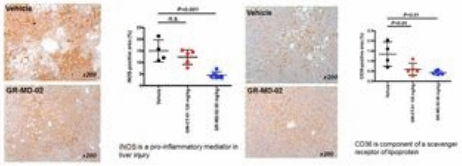




GR-MD-02 appears to reduce the number of galectin-3 expressing macrophages in NASH mice



GR-MD-02 markedly reduces expression of iNOS and CD36 in the livers of NASH mice



Conclusions:

- The model of NASH in mice developed robust histologic findings of NASH with fibrosis.
- Treatment with galectin inhibitors GM-CT-01 and GR-MD-02 had no effect on blood glucose levels, body weight, or general condition of the animals.
- Treatment with GR-MD-02 had approximately four fold greater effect on NASH pathology and fibrosis than GM-CT-01.
- GR-MD-02 ameliorated NASH pathology and reduced or eliminated fibrosis when administered as a single weekly dose in a relatively dose dependent fashion down to doses of 30 mg/kg.
- GR-MD-02 reduced the number of galectin-3 expressing macrophages in the liver while not reducing the absolute number of macrophages.
- GR-MD-02 markedly reduced hepatocellular expression of iNOS in NASH mice livers, a potent pro-inflammatory mediator.
- GR-MD-02 reduced the expression of CD36 which is a component of a scavenger receptor for lipoprotein.
- GR-MD-02 is an inhibitor of galectin-3 which is efficacious in a mouse model of NASH and appears to act through multiple pathways.