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

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**FORM 8-K**

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

**Date of Report (Date of earliest event reported): January 21, 2014**

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**GALECTIN THERAPEUTICS INC.**

(Exact name of registrant as specified in its charter)

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**Nevada**  
(State or Other Jurisdiction  
of Incorporation)

**001-31791**  
(Commission  
File Number)

**04-3562325**  
(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)

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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))
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**SECTION 7 – REGULATION FD**

**Item 7.01 Regulation FD Disclosure.**

On January 21, 2014, Galectin Therapeutics Inc. (the “Company”) issued the attached press release. The press release, which is being furnished and not filed, and is 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 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.

<u>Exhibit Number</u>	<u>Description</u>
99.1	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: January 21, 2014

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



### Preclinical Study Demonstrates Effect of Galectin Inhibitor on Serum Biomarker in Fatty Liver Disease with Fibrosis

Norcross, GA (January 21, 2014) – Galectin Therapeutics Inc. (NASDAQ: GALT), the leading developer of therapeutics that target galectin proteins to treat fibrosis and cancer, today announced that data from a preclinical study show its leading galectin-inhibiting drug GR-MD-02 demonstrates an effect on a blood biomarker in an animal model of nonalcoholic steatohepatitis (NASH, or fatty liver disease) with fibrosis. Hyaluronic acid, a well investigated marker of liver fibrosis, was significantly reduced by approximately 33 percent when untreated animals were compared with those treated with GR-MD-02.

In the study, NASH-induced mice were treated with once weekly doses of GR-MD-02 at four different doses for a total of six weeks of treatment. Results revealed that treatment with GR-MD-02 at doses of 10, 30, and 60 mg/kg body weight significantly reduced the plasma levels of hyaluronic acid in the NASH mice. Other biomarkers examined did not change (MIG (monokine induced by interferon gamma) and TIMP-1 (tissue inhibitor of metalloproteinase)) or were not detectable (IP-10 (interferon inducible protein), KC (keratinocyte-derived chemokine), MIP-1a (macrophage inflammatory protein), and MCP-1 (monocyte chemo-attractant protein). Importantly, plasma levels of galectin-3 were measurable and did not change with therapy, indicating that changes in tissue galectin-3 and improvement in NASH histology do not correlate with blood levels of galectin-3 in this model.

“These results in this preclinical model of NASH show that improvement in NASH and fibrosis with GR-MD-02 treatment appear to correlate with plasma levels of hyaluronic acid, a biomarker that has been shown in multiple human studies to correlate with liver fibrosis,” said Peter G. Traber, M.D., Chief Executive Officer, President and Chief Medical Officer, Galectin Therapeutics. “We are examining the levels of hyaluronic acid as well as multiple other markers of inflammation, cell death and fibrosis in our current Phase 1 clinical trial of GR-MD-02 in NASH patients with advanced fibrosis.”

As previously reported and published in *PLOS ONE*, the same study showed that GR-MD-02 improved all components of NASH in mice, including fibrosis [<http://dx.plos.org/10.1371/journal.pone.0083481>].

GR-MD-02 is a proprietary molecule that binds to and inhibits galectin proteins, predominantly galectin-3. Patient enrollment is complete in cohort 1 of a blinded Phase 1 clinical trial of GR-MD-02 for patients with NASH with advanced fibrosis. No serious adverse events have been reported. The Phase 1 first-in-man study is evaluating the safety, tolerability, pharmacokinetics and exploratory biomarkers for efficacy for single and multiple doses of GR-MD-02 when administered to patients with fatty liver disease with advanced fibrosis. Following the 70 day study period and analysis of the data, the Company anticipates that initial safety and tolerability results, as well as biomarkers to evaluate for potential disease effect, from the first cohort will be available around the end of the first quarter of this year.

## **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](http://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 preclinical data and the potential role for GR-MD-02 and GM-CT-01 in the treatment of liver fibrosis and cirrhosis in humans. Factors that could cause our actual performance to differ materially from those discussed in the forward-looking statements include, among others, that our plans, expectations and goals regarding any preclinical data and potential therapeutic uses and benefits of our drugs and any future pre-clinical or clinical studies are subject to factors beyond our control. 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, 2012, 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: Galectin Therapeutics Inc.  
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