
**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**

**December 19, 2011
Date of Report (Date of earliest event reported)**

GALECTIN THERAPEUTICS INC.

(Exact name of registrant as specified in its charter)

NEVADA
(State or other jurisdiction
of incorporation)

000-32877
(Commission
File Number)

04-3562325
(IRS Employer
Identification No.)

**7 WELLS AVENUE
NEWTON, MASSACHUSETTS
02459**

(Address of principal executive offices) (Zip Code)

(617) 559-0033
(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))
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Item 8.01 Other Events.

Press Release dated December 19, 2011: Galectin Therapeutics Provides Corporate Update.

The information in this Report is being furnished pursuant to this Item 8.01 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 dated December 19, 2011: Galectin Therapeutics Provides Corporate Update.

SIGNATURES

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

GALECTIN THERAPEUTICS INC.

By: /s/ Anthony D. Squeglia
Anthony D. Squeglia
Chief Financial Officer

Date: December 19, 2011

EXHIBIT INDEX

Exhibit No.:

99.1 Press Release dated December 19, 2011: Galectin Therapeutics Provides Corporate Update.



Galectin Therapeutics Provides Corporate Update

Newton, MA – December 19, 2011 – Galectin Therapeutics Inc. (OTC: GALT), the leader in developing carbohydrate-based therapeutic compounds to inhibit galectin proteins, today issued the following corporate update to its shareholders:

Dear Galectin Therapeutics Stockholder:

As 2011 draws to a close we are very pleased to report the progress that Galectin Therapeutics has made in its mission to create new therapeutics based on its pioneering work in the area of galectin inhibition. In particular, we are proud of the accomplishments we have made in the brief period since the mid-year implementation of a refined corporate strategy. This strategy is designed to efficiently maximize the value of our unique scientific and medical leadership in this field. In conjunction with that announcement, we also unveiled our new Company name which, of course, reflects our expertise. This strategy, briefly, is to leverage our galectin expertise in the two therapeutic areas where our drugs have the best scientific rationale and which have the largest medical needs and markets: cancer and fibrosis. In this letter we would like to reflect on these accomplishments but, more importantly, look forward to the objectives we intend to achieve under this strategy in the year to come.

The Bright Future of Galectin Inhibition in Cancer Therapy

The promise of galectin inhibition to play an important role in cancer remains very strong. However, to provide the highest value for our development investments and have achievable short term milestones, Galectin Therapeutics has shifted its focus in cancer from use with standard chemotherapies to use in combination with cancer vaccines.

We have elected not to expend further corporate resources toward a U.S. approval for GM-CT-01 (DAVANAT®) in colorectal cancer at this time based on a number of factors. These factors include: the extensive cost of clinical trials of GM-CT-01 necessitated by current regulatory requirements, the results of colorectal cancer trials would not be available for nearly four years, the decline in use of intravenous 5-FU (the drug with which GM-CT-01 was used in combination), and the highly competitive treatment landscape for colorectal cancer.

Another ancillary approach for the development of GM-CT-01 in colorectal cancer is our efforts to gain approval of GM-CT-01 in Colombia. The Company was encouraged by a key oncologist at Colombia's National Cancer Institute, the government, and a regional pharmaceutical company to seek approval for GM-CT-01 as an adjuvant to 5-FU because of the data that showed it may increase the efficacy of 5-FU and reduce its side effects. We are currently waiting for approval from the Colombian regulatory agency. While Colombian marketing is not a central component of our overall corporate strategy, this would represent a novel way to obtain revenue to support development programs, reduce the amount of future equity placement, and gain additional clinical experience with GM-CT-01. While we remain optimistic that this approach will be successful, with potential revenue in 2012, we remain conservative in our expectations based on the absence of an approval in a major region such as the U.S. or Europe.

The promise of our technology to treat cancer remains very strong however, based on the promise for galectin inhibition to play a key role in the burgeoning area of cancer immunotherapy. The role of galectins in cancer immunotherapy can be understood through the "Galectin Effect", a recent discovery of how tumors avoid the body's own immune system. Our current program to block the "Galectin Effect" is supported by multiple important data points. First, the "Galectin Effect" was discovered by a

world leading tumor immunologist at the Ludwig Institute of Cancer Research in Brussels, Belgium and published in top scientific journals. Based on these results, we have an important Key Opinion Leader (KOL), who will aid in the communication and acceptance of this approach, and we know the mechanism of action (MOA). Dr. Pierre van der Bruggen and his colleagues showed that galectin-3, which is produced by the vast majority of human cancers, binds to and blocks the actions of T-lymphocytes, the major immune cell in the body's defense against cancers. Therefore, in the presence of galectins, the body's immune cells are unable to attack and kill tumor cells. The most exciting finding was that in the presence of GM-CT-01, the galectins are blocked and the ability of the T-lymphocytes to kill tumor cells is restored.

This is a remarkable finding and holds great promise in cancer therapy. Many approaches, such as tumor vaccines, are being used to increase the number of T-lymphocytes that can attack cancer. According to market analysts, the tumor vaccine market is expected to exceed \$7 billion by 2015. However, if these newly generated T-lymphocytes are unable to kill cancer cells because of the "Galectin Effect" they will be ineffective. Our galectin inhibitors may act as immune enhancers for all tumor vaccines, representing tremendous therapeutic potential.

In addition to these exciting developments, there is also potential for a rapid pathway to showing proof of concept in human patients. In a significant milestone for this program, we recently initiated a phase I/II clinical trial of GM-CT-01 in combination with a tumor vaccine in patients with advanced melanoma, a deadly skin cancer. This trial is being conducted at three centers in Belgium and one in Luxembourg. We expect the first stage of this trial to be completed within a year and provide data that could deliver an indication of efficacy; which would be another critically important milestone for the Company. Further, positive results would demonstrate that this approach could be an enabling technology for therapy in many other tumors. Importantly for Galectin and its shareholders, our share of the cost for this trial and the results it generates will be less than \$1 million. This approach is more favorable for the Company than a very long-term result in colorectal cancer in combination with chemotherapy.

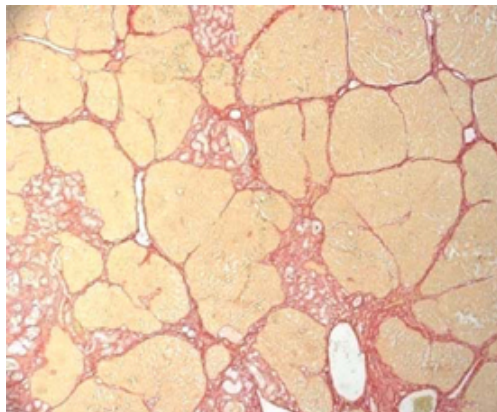
Liver Fibrosis: Bold New Strategy in an Unmet Medical Need

One of the most exciting areas for the application of galectin inhibition, and the second main initiative in our development strategy, is in the area of liver fibrosis. The potential of this program is enormous because there are no therapies on the market for liver fibrosis, a condition that leads to cirrhosis. Currently, nearly 500,000 patients have cirrhosis with nearly 50,000 losing their lives yearly in the United States; while only 6,200 were saved by liver transplantation at a cost of \$350,000 per transplantation.

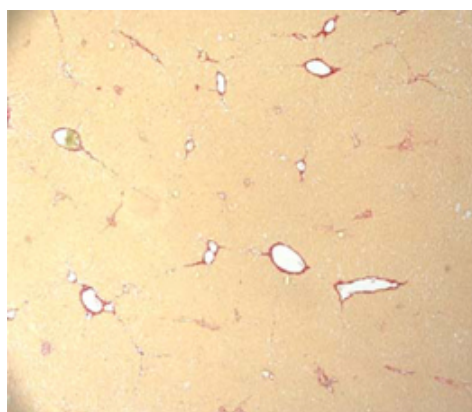
The driving factor for our commitment to galectin inhibition for fibrosis is the strong scientific evidence that galectin-3 is essential for the development of liver fibrosis in animals. Published data shows that mice lacking the galectin-3 gene are incapable of developing liver fibrosis in response to toxin insult to the liver. Moreover, the mice that have no galectin-3 gene are resistant to lung and kidney fibrosis.

Therefore, we evaluated the ability of Galectin Therapeutics' carbohydrate drug candidates to block galectin-3 in animal models of liver fibrosis. The results were clear and exciting. In the figures below are selected pictures of the liver from those experiments. The figure on the left shows a microscopic section of a rat liver that was treated with a chemical toxin for eight weeks, which induced liver fibrosis, and then given a placebo for four weeks. One can see the reddish scars that marbled the tissue, indicative of severe fibrosis. In contrast, the figure on the right shows a microscopic section of a rat liver that was treated with the same chemical toxin for eight weeks, and then given four weeks of one of our carbohydrate drugs called GR-MD-02. One can see that there is virtually no scar tissue on the figure on the right. This indicates that treatment with GR-MD-02 is able to **reverse and prevent the development of scar tissue** in the liver. These experiments, along with several others that included human liver cells, clearly identify the mechanism of action for the creation of fibrotic scar tissue in the liver and for the first time, millions of people with cirrhosis will have hope for a treatment.

Liver Fibrosis, induced by injection of chemical toxin for 8 weeks



Regression of Fibrosis after 4 weeks of treatment with GR-MD-02



Recently, we generated additional data that adds to the enthusiasm over GR-MD-02 as a treatment candidate for liver disease and fibrosis. Dr. Traber presented compelling preclinical data at the European Association for the Study of the Liver in Lisbon, Portugal. These data demonstrate that GR-MD-02 reversed NASH-induced fibrosis in the liver of the animals in the trial. NASH, or non-alcoholic steatohepatitis, aka fatty liver disease, is a liver disease characterized by the accumulation of fat in the liver with associated inflammation and fibrosis that can lead to end-stage cirrhosis requiring a liver transplantation. The National Institute of Health (NIH) estimates that 9 to 15 million Americans suffer from NASH, and that the number is growing due to obesity and diabetes. The NIH forecasts that NASH is an epidemic and will become the leading cause of liver cirrhosis and liver transplantation.

Similar to our cancer immunotherapy program, the strength of our science has attracted the interest of leaders in the study of liver diseases. Specifically, our program for galectin inhibition to treat liver fibrosis has attracted Key Opinion Leaders (KOLs) from such prominent institutions such as Harvard, Mount Sinai, Penn, Emory, Michigan, and Wisconsin. These experts will be critical in disseminating information about our therapy, designing the best clinical trials, communicating with the FDA, and enrolling patients in clinical trials.

Summary

In the short period of time since we began executing on this strategy we have made presentations of positive data at multiple significant medical meetings, initiated the cancer immunotherapy trial with the Ludwig Institute, and defined our promising development program in fibrosis. These achievements are reflected in the initiation of research coverage by two independent Wall Street analysts.

We hope and expect to continue this excellent progress in 2012 and very much look forward to keeping you informed throughout the year.

Sincerely,



Peter G. Traber, M.D.
Chief Executive Officer, President



James C. Czirr
Executive Chairman

About Galectin Therapeutics

Galectin Therapeutics (OTC: GALT) is developing promising carbohydrate-based therapies for 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: Anthony D. Squeglia, Chief Financial Officer, 617.559.0033, squeglia@galectintherapeutics.com