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

NORCROSS, Ga.--(BUSINESS WIRE)--Nov. 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.

Source: Galectin Therapeutics

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