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Galectin Therapeutics Announces Positive Preclinical Results on the Role of Galectin-3 in Mediating Vascular Remodeling in Pulmonary Arterial Hypertension

Researchers at Augusta University show galectin-3 inhibitors improve PAH in animal models

NORCROSS, Ga., June 02, 2016 (GLOBE NEWSWIRE) -- **Galectin Therapeutics, Inc.** (NASDAQ:GALT), the leading developer of therapeutics that target galectin proteins to treat fibrosis and cancer, reports that GR-MD-02, its development-stage galectin-3 (gal-3) inhibitor, has shown a positive effect on vascular remodeling in an animal model of pulmonary arterial hypertension (PAH). The results were presented at the American Thoracic Society (ATS) 2016 International Conference by investigators from the Vascular Biology Center and the Department of Pharmacology and Toxicology at Augusta University (formerly known as Georgia Regents University) (see poster presentation).

PAH results in a progressive narrowing of the pulmonary arteries, which leads to increased vascular resistance and the elevation of pulmonary blood pressure and, ultimately, right ventricular heart failure. It is believed that the primary cause of the permanently elevated pulmonary vascular resistance that occurs in PAH is due to physical obstruction from vascular remodeling. There are a number of vasodilator drugs that are indicated for the treatment of PAH, but additional therapies are desirable to address the structural narrowing of pulmonary arteries.

The results of this study demonstrated that rat models of PAH have significantly increased right ventricular systolic pressure, right ventricular hypertrophy and pulmonary arterial smooth muscle proliferation and fibrosis, as well as increased right ventricular wall thickness and lower pulmonary blood flow. Further, in rats and humans with PAH, robust induction of gal-3 expression was observed in the smooth muscle layer. Treatment with the gal-3 inhibitors GR-MD-02 and GM-CT-01 resulted in marked improvement in the functional heart characteristics and the vascular remodeling (smooth muscle proliferation and fibrosis) associated with PAH. A more complete description of the findings can be found in a companion CEO Perspective blog post in addition to the poster presentation.

"The alterations in cardiopulmonary function and vascular proliferation, as well as in fibrosis were significantly attenuated by *in vivo* treatment with specific gal-3 inhibitors," said David Fulton, Ph.D., Director of the Vascular Biology Center at Augusta University. "These findings indicate that gal-3 signaling contributes to the compromised pulmonary vascular function and prominent pulmonary arterial remodeling that occurs in PAH, which suggests that inhibition of gal-3 is a strategic and viable target for treatment of PAH and other related pulmonary vascular diseases."

"We are intrigued by the results of this study, which suggest another important indication for therapy with our gal-3 inhibitors," said Peter G. Traber, M.D., chief executive officer and chief medical officer of Galectin Therapeutics and one of the study's authors. "The severity of PAH is such that over half of patients die of the disease within seven years of initial diagnosis. A new therapeutic approach that addresses vascular remodeling would be a welcome addition to the vasoactive drugs currently on the market."

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

Forward Looking Statements
This press release contains 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 management's current expectations and are subject to factors and uncertainties that could cause actual results to differ materially from those described in the statements. These statements include those regarding the hope that its lead compounds will be successful in treating fibrosis and cancer and that those compounds may be effective in the treatment of pulmonary arterial hypertension. Regardless of the results of any of its development programs, Galectin may be unsuccessful in developing partnerships with other companies or raising additional capital that would allow it to further develop and/or fund any studies or trials. For a discussion of additional factors impacting Galectin's business, see the Company's Annual Report on Form 10-K for the year ended December 31, 2015, and subsequent filings with the SEC. You should not place undue reliance on forward-looking statements. Although

subsequent events may cause its views to change, management disclaims any obligation to update forward-looking statements.

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