



August 16, 2016

## **GlycoMimetics Announces Publication of Preclinical Data Showing Drug Candidate GMI-1271 Reduces Inflammatory Responses After Heart Attack**

*Research shows effectiveness in stabilizing atherosclerotic plaques, reducing risk of further injury due to reduced blood flow*

ROCKVILLE, Md.--(BUSINESS WIRE)-- GlycoMimetics, Inc. (NASDAQ: GLYC) today announced the publication of results from a preclinical study that showed its drug candidate GMI-1271 reduced the cellular interactions that often lead to a buildup in inflammatory response and unstable atherosclerotic plaque formation after a heart attack. The study, entitled "E-Selectin Inhibition Mitigates Splenic HSC Activation and Myelopoiesis in Hypercholesterolemic Mice With Myocardial Infarction," was published in the September issue of [Arteriosclerosis, Thrombosis, and Vascular Biology](#).

In the study, researchers demonstrated a wider range of potential clinical applications of the E-selectin antagonist GMI-1271, which is currently being evaluated as a potential treatment for acute myelogenous leukemia (AML) in a Phase 1/2 clinical trial.

"The results in this animal model of myocardial infarction and atherosclerosis demonstrate both the biological activity of GMI-1271 and the possible broader uses of an E-selectin antagonist. While GlycoMimetics is currently focused on developing GMI-1271 for treatment of AML, this drug candidate has shown activity in pre-clinical models of a number of diseases where E-selectin plays a key functional role," said [John L. Magnani](#), Ph.D., Vice President and Chief Scientific Officer of GlycoMimetics.

Myocardial infarctions are often triggered by unstable atherosclerotic plaque material, the growth of which is initiated by the production and infiltration of inflammatory cells through the action of E-selectin. The study, conducted at Harvard Medical School and Massachusetts General Hospital, found that after a myocardial infarction (MI), GMI-1271 not only reduced the production of inflammatory cells and their hematopoietic stem and progenitor cells, but also their infiltration into atherosclerotic plaques, thereby stabilizing existing plaques and decreasing the risk of a further ischemic injury which can lead to a second MI. The study showed that GMI-1271-induced E-selectin inhibition significantly reduced the numbers of stem and progenitor cells leading to reduced numbers of inflammatory monocytes, and neutrophils in the blood. It also inhibited their infiltration into existing plaques leading to the stabilization of atherosclerotic plaques (smaller plaque size, reduced necrotic core area, and thicker fibrous cap) after an MI in animal models.

### **About GlycoMimetics, Inc.**

GlycoMimetics is a clinical-stage biotechnology company focused on cancer and sickle cell disease. GlycoMimetics' most advanced drug candidate, rivipectin, a pan-selectin antagonist, is being developed for the treatment of vaso-occlusive crisis in sickle cell disease and is being evaluated in a Phase 3 clinical trial being conducted by its strategic collaborator, Pfizer. GlycoMimetics' wholly-owned drug candidate, GMI-1271, an E-selectin antagonist, is being evaluated in an ongoing Phase 1/2 clinical trial as a potential treatment for AML. GlycoMimetics is located in Rockville, MD in the BioHealth Capital Region. Learn more at [www.glycomimetics.com](http://www.glycomimetics.com).

### **Forward-Looking Statements**

This press release contains forward-looking statements regarding GlycoMimetics' planned activities with respect to the clinical development of its drug candidate GMI-1271. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including the availability and timing of data from ongoing clinical trials, the uncertainties inherent in the initiation of future clinical trials, whether interim results from a clinical trial will be predictive of the final results of the trial or results of early clinical trials will be indicative of the results of future trials, expectations for regulatory approvals, availability of funding sufficient for GlycoMimetics' foreseeable and unforeseeable operating expenses and capital expenditure requirements, other matters that could affect the availability or commercial potential of GlycoMimetics' drug candidates and other factors discussed in the "Risk Factors" section of GlycoMimetics' Annual Report on Form 10-K that was filed with the U.S. Securities and Exchange Commission on February 29, 2016, and other filings GlycoMimetics makes with the Securities and Exchange Commission from time to time. In addition, the forward-looking statements included in this press release represent GlycoMimetics' views as of the date hereof. GlycoMimetics anticipates that subsequent events and developments may cause its views to change. However, while GlycoMimetics may elect to update these forward-looking statements at some point in the future, GlycoMimetics specifically disclaims any

obligation to do so, except as may be required by law. These forward-looking statements should not be relied upon as representing GlycoMimetics' views as of any date subsequent to the date hereof.

View source version on [businesswire.com](http://www.businesswire.com/news/home/20160816005628/en/): <http://www.businesswire.com/news/home/20160816005628/en/>

GlycoMimetics

**Investor Contact:**

Shari Annes, 650-888-0902

[sannes@annesassociates.com](mailto:sannes@annesassociates.com)

or

**Media Contact:**

Jamie Lacey-Moreira, 410-299-3310

[jamielacey@presscommpr.com](mailto:jamielacey@presscommpr.com)

Source: GlycoMimetics

News Provided by Acquire Media