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miRagen Therapeutics, Inc. and Collaborators Publish Study Identifying microRNA Perturbations Following Acute Myocardial Infarction

Results identify miR-29 as a key regulator of cardiac fibrosis

BOULDER, Colo.--([BUSINESS WIRE](#))--miRagen Therapeutics, Inc., a biopharmaceutical company focused on the discovery and development of breakthrough therapies for cardiovascular and muscle disease based on microRNA biology, today announced the publication of novel research demonstrating that acute myocardial infarction (MI) in mice and humans results in the dysregulation of a specific set of microRNAs.

The microRNAs identified by this research may serve as novel targets for therapeutic intervention against acute myocardial infarction due to coronary artery occlusion, which represents a major cause of human morbidity and mortality.

The study was conducted in collaboration with researchers led by Eric N. Olson, Ph.D., at the University of Texas Southwestern Medical Center and appears in the *Proceedings of the National Academy of Sciences* (van Rooij E, et al. Dysregulation of microRNAs following myocardial infarction reveals a role of miR-29 in cardiac fibrosis. Proc Nat Acad Sci USA 105:13027-13032).

“Our results implicate specific microRNAs that are dynamically regulated in different areas of the heart after acute myocardial infarction. This provides us with the unique opportunity to intervene against the disease drivers and tailor therapies to address specific aspects of post-MI management,” said William S. Marshall, Ph.D., President and Chief Executive Officer of miRagen Therapeutics, Inc., and a co-author of the publication. “The identification and validation of miR-29 as a key regulator of cardiac fibrosis gives us the potential to target a single entity that controls a complex disease process and contributes to heart dysfunction.”

Among the MI regulated microRNAs, the investigators further characterized the importance of a family of microRNAs known as miR-29. The results suggest that the down-regulation of miR-29 contributes to cardiac fibrosis and that strategies to maintain miR-29 levels may be beneficial in reducing cardiac fibrosis. Cardiac fibrosis, which results in stiffening of the heart, diminished contractility and abnormalities in cardiac conductance, is a common consequence of numerous forms of heart disease. Thus, reversal of this process represents an important therapeutic target in post-MI management and heart failure. The results also suggest a broader role for miR-29 in the settings of fibrotic diseases in general.

“We previously showed that cardiac hypertrophy and heart failure are accompanied by characteristic changes in the expression of a collection of specific microRNAs. This work validates many of those observations and expands the number of potential targets for therapeutic intervention in heart disease,” said Eric N. Olson, Ph.D., Professor and Chairman of the Department of Molecular Biology at the University of Texas Southwestern Medical Center and Chief Scientific Advisor to miRagen.

About microRNAs: MicroRNAs have emerged as an important class of small RNAs encoded in the genome. They act in a combinatorial manner to control the expression of sets of genes and entire pathways and are thus thought of as master regulators of gene expression. Recent studies have demonstrated that mis-regulation of microRNA expression is correlated with many disease states. Because they are single molecular entities that dictate the expression of fundamental regulatory pathways, they represent potential therapeutic intervention targets of unprecedented power.

About miRagen Therapeutics: miRagen Therapeutics was founded in 2007 to exploit recent discoveries showing that specific perturbations in microRNA expression are correlated with various forms of cardiovascular and muscle disease. miRagen’s Founders and scientists combine a strong knowledge of cardiovascular medicine with deep understanding of microRNA biology and chemistry. The company controls an estate of intellectual property focused on the treatment of cardiovascular and muscle disease through microRNA manipulation.

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