

Study hints at experimental therapy for heart fibrosis

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Researchers seek treatments for adult and congenital diseases.



Singapore - Researchers report encouraging preclinical results as they pursue elusive therapeutic strategies to repair scarred and poorly functioning heart tissues after cardiac injury – describing an experimental molecular treatment for heart failure.

In a study posted online by Journal of the American College of Cardiology, researchers at the Cincinnati Children's Heart Institute inhibited a protein that helps regulate the heart's response to adrenaline. This alleviated the disease processes in mouse models of human heart failure, and in cardiac cells isolated from heart failure patients undergoing reparative surgery.

The experimental approach focuses on the role of the proteins $G_{\beta\gamma}$ and GRK2, which are involved in a signaling pathway activated by adrenaline stimulation. The adrenergic system plays a fundamental role in maintaining normal heart function, according to study authors. Their data show that chronic over stimulation of the system (which happens after a heart attack) prompts hypertrophy – a thickening and enlargement of the heart muscle. It also causes fibrosis, the formation of scar tissue.

"Regrettably, there are essentially no clinical interventions that effectively target these tissue-damaging cardiac fibroblasts. This work may provide evidence that shifts the way we think about treating heart failure," said Burns Blaxall, PhD, senior investigator and the heart institute's director of translational science. "Not only has our study identified the cardio-protective properties of pharmacological and fibroblast-specific $G_{\beta\gamma}$ -GRK2 inhibition in a clinically relevant mouse model, we also showed that inhibition reduced the activation of human heart failure cardiac fibroblasts. This is a key cell type responsible for the scarring of heart tissue."