Murine model provides insight into mechanisms of reverse cardiac remodeling

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Sustained pathological remodeling of the heart after injury is associated with increased risk of heart failure and death. Several recent studies have shown that strategies to reduce heart failure are associated with a reversal of adverse remodeling. The mechanisms of this reverse remodeling are not fully understood and have potential to guide the development of future interventions.

In this issue of *JCI Insight*, Douglas Mann and colleagues at Washington University School of Medicine developed a murine model of dilated heart failure that exhibited normalization of left ventricle structure following suppression of pathogenesis. In these animals, normalization of left ventricular structure and function was accompanied by improved cardiac myocyte function and reduced expression of genes associated with incident heart failure. Moreover, mortality in animal models was increased when expression of heart failure-induced genes was sustained. A similar normalization of gene expression in the heart was also observed in patients with dilated cardiomyopathy with evidence of reversed remodeling following circulatory support.

These results indicate that evaluation of gene expression following cardiac injury can identify those at the highest risk of further cardiac deterioration.

More information: Veli K. Topkara et al. Functional significance of the discordance between transcriptional profile and left ventricular structure/function during reverse remodeling, *JCI Insight* (2016). DOI:

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