Identification of a gene signature associated with dilated cardiomyopathy

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Dilated cardiomyopathy (DCM) is a progressive thinning of heart muscle that commonly results in heart failure. DCM is a known secondary complication of conditions such as alcohol abuse and infection and is also an inherited disorder. However, the molecular events that underlie DCM progression are not fully understood.

A study in this issue of *JCI Insight* identifies a gene signature that characterizes the transition from DCM to heart failure. Michael Burke of Emory University School of Medicine, Christine Seidman of Harvard Medical School, and their colleagues evaluated gene expression in a murine model of congenital DCM at various stages of disease. As the mice progressed from pre-DCM to heart failure, there was increased expression of fibrotic and inflammatory genes, an increase in heart muscle cell proliferation, and a shift in the metabolic profile of the heart muscle cells.

The authors then compared the DCM gene signature to the gene profile of mice with hypertrophic cardiomyopathy (HCM), which has features similar to DCM but does not usually progress to heart failure. While DCM and HCM had some overlap in their gene expression profiles, there were distinct differences in pro-fibrotic and metabolic networks that could distinguish these conditions.

Together, the results of this study provide new insight into the gene programs that drive cardiac remodeling.

More information: Michael A. Burke et al, Molecular profiling of dilated cardiomyopathy that progresses to heart failure, *JCI Insight* (2016). DOI: 10.1172/jci.insight.86898

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