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A pathway controlling inflammatory responses aids recovery after heart attack

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After a heart attack, or myocardial infarction, a patient's long-term prognosis depends on the ability of the heart tissue to heal and remodel. Immune system activation and inflammatory responses that occur in the aftermath of a myocardial infarction can be detrimental to healing, so better understanding of the pathways that contribute to these processes may improve treatments in heart attack patients.

A study published this week in the *JCI* has identified a signaling pathway in cardiac tissue that suppresses inflammatory responses after cardiac injury.

Work led by Jonathan Epstein at the University of Pennsylvania showed that in heart muscle, activation of the Hippo pathway leads to the recruitment of T regulatory cells, which in turn help control inflammation.

Mice lacking the Hippo pathway components YAP and TAZ displayed increased cardiac fibrosis as well as exacerbated inflammation around the heart. These observations were associated with reduced numbers of T regulatory cells and lower expression of the immune system signaling protein, IFN-γ.

When IFN-γ was delivered directly to the injured heart muscle, T regulatory cell levels were restored and fibrosis was ameliorated in these mice.

Together, these findings identify the Hippo signaling pathway and IFN-γ production as crucial control points for the immune response to heart injury and recovery.

More information: Vimal Ramjee et al, Epicardial YAP/TAZ orchestrate an immunosuppressive response following myocardial infarction, *Journal of Clinical Investigation* (2017). DOI: 10.1172/JCI88759

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