Macrophage population activates repair in murine heart attack model

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Following a heart attack, successful repair of damaged tissue can prevent cardiac rupture and other adverse outcomes. The ability to repair myocardial tissue depends on the activation of fibroblasts, which stimulate the formation of connective tissue.

In this month's issue of the *JCI*, a team led by Ken Suzuki at the William Harvey Research Institute determined that tissue reparation after a heart attack depends on the production of a type of white blood cell called M2 macrophages.

They examined myocardial repair in tribbles homolog-1-deficient (Trib1-deficient) mice, which do not form M2 macrophages after myocardial infarction. After an induced myocardial infarction, Trib1-deficient mice showed less fibroblast activation than control mice, which led to an increased risk of cardiac rupture.

Supplementing Trib1-deficient mice with M2 macrophages restored their ability to repair myocardial tissue. Moreover, treating these mice with the cytokine IL-4 increased macrophage production and decreased the risk of cardiac rupture.

This study highlights the importance of M2 macrophages in myocardial tissue repair and indicates that IL-4 treatment may have important therapeutic potential during recovery from a heart attack.

More information: Manabu Shiraishi et al, Alternatively activated

macrophages determine repair of the infarcted adult murine heart, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI85782