**ROLE OF CD4+ T-CELLS AFTER MYOCARDIAL INFARCTION**

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The role of innate immunity has been studied for long time whereas adaptive immunity quite recently came into focus in the context of myocardial disease. There is accumulating evidence that CD4+ T-cells contribute to myocardial ischemia-reperfusion injury. We showed that absence of CD4+ T-cells improves microvascular perfusion in a mouse myocardial ischemia- reperfusion model. After completion of infarction, an exaggerated or persistent inflammatory activation after myocardial infarction leads to maladaptive healing and subsequent remodeling of the left ventricle. Monocyte derived cells/ macrophages constitute the central cellular component in the innate immune response to myocardial injury. Meanwhile their role in myocardial healing is well established. CD4+Foxp3+ regulatory T cells (Treg cells) contribute to inflammation resolution. We showed that both conventional T-cells and Treg cells become activated in an experimental MI mouse model most likely due to recognition of autoantigens. Treg cells are especially recruited to the infarcted myocardium during the early healing phase. They drive the switch in macrophage polarization from a pro-inflammatory to a pro-healing phenotype. This cellular interaction especially promotes the formation of a stable scar. Experimentally, Treg cells stimulation after MI is able to improve survival and left ventricular remodeling. Therefore, the interaction of T-cells with monocyte-derived cells/ macrophages constitutes a promising field for development of future therapeutic approaches aiming at modulation of inflammation in myocardial disease.