**IL-17 is dispensible for inflammatory myocarditis but essential for the progression to dilated cardiomyopathy**

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Myocarditis and dilated cardiomyopathy (DCM) are significant causes of sudden death in young adults. Whereas CD4+ helper lymphocytes are essential for myocarditis induction, the subset(s) and effector function(s) mediating inflammation in the heart are not well defined. We sought to determine the role of Th17 CD4+ T cells in the induction of myocarditis, and the progression to DCM using a murine model of myocarditis (EAM). Gene knockout studies revealed that IL-12p40, but not IL-12p35, is essential for myocarditis induction, underscoring a critical role for IL-23 dependent Th17 cells in the initiation of myocarditis. However, deficiency in IL-17A, a major cytokine secreted by Th17 cells, did not abrogate heart inflammation, nor did it abolish the production of myosin-specific autoantibodies. Remarkably, echocardiography analysis revealed that IL-17A deficient mice were completely protected from DCM, which correlated with decreased deposition of collagen in the myocardium. Thus, IL-17 is dispensable for myocarditis inflammation but is a critical mediator of tissue fibrosis and cardiac remodeling leading to post-inflammatory heart dysfunction.