Suppressed androgen receptor expression promotes M2 macrophage reprogramming through the STAT3/SOCS3 pathway

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Abstract

Macrophages are important mediators of inflammatory cardiovascular diseases, and various macrophage phenotypes exert opposite effects during inflammation. In our previous study, we proved that suppressed androgen receptor (AR) alleviated inflammation during experimental autoimmune myocarditis (EAM). As anti-inflammatory cells, whether M2 macrophages are involved in this process remains unclear. Here, we showed that anti-inflammatory cytokines and M2 macrophages were elevated when AR was suppressed during EAM. In IL-4 stimulation-induced M2 macrophages, impaired AR with ASC-J9 increased the expression of M2 macrophage-related factors. Moreover, suppressed AR expression resulted in macrophage M2 polarization by reducing SOCS3 production and enhancing STAT3 activation. Taken together, our data suggest that AR plays a critical role in macrophage polarization and suppressed redundant AR expression promotes anti-inflammatory M2 macrophages reprogramming. This study suggests a potential therapeutic agent for inflammatory cardiomyopathy through the use of ASC-J9.

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