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Myeloid-specific IL4 receptor alpha knockout increases adverse cardiac remodeling post myocardial infarction

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Myocardial infarction (MI) elicits inflammatory reactions that are orchestrated by a wide range of immune cells including macrophages. Reprogramming macrophages towards a resolving and reparative phenotype is a potential therapeutic approach for MI. However, how to modify macrophages in order to improve cardiac injury is not clear. Interleukin 4 receptor alpha (IL4Ra) activation is one of the major alternative activated macrophage (also names M2 macrophage) inducers, so IL4Ra is a potential macrophage modifier. Here, we knocked out IL4Ra from myeloid cells to determine how IL4Ra signaling is involved in cardiac remodeling post MI. Myeloid-specific IL4Ra knockout (MyIL4RaKO) did not show significant change in cardiac hypertrophy and interstitial fibrosis. There was decreased infarct size at one week post MI, but by three weeks there was no significant difference in infarct size. We saw significantly increased infarct thickness and perivascular fibrosis at three weeks, indicating the involvement of IL4Ra signaling in cardiac remodeling. Importantly, along with these significant changes in cardiac remodeling, MyIL4RaKO mice showed significantly decreased cardiac function at three weeks post MI. IL4Ra signaling was significantly inactivated in macrophages; however no change was shown in macrophage polarization in heart tissues post MI. In conclusion, IL4Ra signaling in myeloid cells is critical in maintaining proper cardiac remodeling and cardiac function post MI, which suggests the potential of modifying macrophages through enhancing IL4Ra signaling as a therapeutic strategy for MI.

Biography

Jianrui Song is a PhD candidate in Cell and Developmental Biology at University of Michigan. She has received her Master's Degree from China in 2009, where she worked on "The role of autophagy in hypoxic microenvironment and chemo- and radiotherapy in liver cancer". As a PhD candidate, she is pursuing training in the field of Cardiovascular Disease and Immunology. She is working on how myeloid cells participate in the response of cardiac ischemia, the formation of infarct and the post infarct response, and also in determining how altering myeloid (especially macrophage) phenotype by IL4Ra can affect these responses in order to identify processes that can be targeted for beneficial effect. She is supported by the Bradley Merrill Patten Research Fellowship and by an American Heart Association Pre-doctoral Fellowship.

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