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Myeloid-specific deletion of SIRT1 impairs obesity and ageing-associated endothelial dysfunction

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SIRT1 is a class III, NAD-dependent histone that plays a key role in curbing inflammatory responses in macrophages. However, whether SIRT1 controls macrophage polarization and its implication in vascular diseases is unclear. Here, we aim to clarify whether SIRT1 deficiency biases macrophage polarization and contributed to endothelial dysfunction. Wild type (WT) and myeloid-specific SIRT1 knockout mice (MKO) were fed with standard chow or westernized diet for 12 weeks. Endothelium-dependent relaxation (EDR) in aortic rings was assessed by wire myograph. Macrophages were differentiated in vitro from bone marrow cells. Compared to WT mice, aortic EDR was significantly impaired in MKO mice under both obese and ageing conditions. In accompany, MKO mice were more susceptible to atherosclerosis in apoE-/- background. Macrophage were resistant to interleukin 4 (IL4)-induced polarization to M2 subtype. Moreover, endothelial cells co-cultured with MKO-derived macrophages exhibited higher inflammation and lower eNOS expression. SIRT1 plays a pivotal role in controlling macrophage polarization, which in turn causes impaired endothelial function.

Biography

Ping Gu has completed her PhD from the Second Military Medicine University of China in 2007 and Postdoctoral studies from the Hongkong University. She has published more than 20 papers in international journals and has been serving as Associate Chief Physician in Jinling Hospital.

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