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[관심분야]

The role of monocytes and macrophages in atherosclerosis, Myeloid reprogramming in Diet-induced obesity, Maternal-to-fetal metabolic reprogramming

[논문]

1. Ahn YJ, Wang L, Kim S, Eber MR, Salerno AG, Asmis R. Macrophage-restricted overexpression of glutaredoxin 1 protects against atherosclerosis by preventing nutrient stress-induced macrophage dysfunction and reprogramming. Atherosclerosis. 2023 Dec;387:117383.
2. Ahn YJ, Wang L, Tavakoli S, Nguyen HN, Short JD, Asmis R. Glutaredoxin 1 Controls Monocyte Reprogramming During Nutrient Stress and Protects Mice Against Obesity and Atherosclerosis in a sex-specific manner. Nat. Commun. 2022 Feb 10;13(1):790.

Monocyte priming and macrophage reprogramming in atherosclerosis and diet-induced obesity

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Atherosclerosis begins in early life and develops subclinically for decades, and may cause cardiovascular and cerebrovascular diseases in adulthood. Chronic inflammation is a risk factor for later cardiovascular disease. Diet-induced obesity and the development of low-grade inflammation can accelerate the development of atherosclerosis. The nutrient stress-induced reprogramming of blood monocytes gives rise to dysregulated, obesogenic, proatherogenic monocyte-derived macrophages. The primed blood monocytes by a high-calorie diet increased chemotactic activity *in vitro* and *in vivo* in response to metabolic stress gradient. These monocytes and the recruitment of monocyte-derived macrophages into the site of inflammation play a key role in atherogenesis. MKP-1 is a key functional regular of monocyte and macrophage phenotypes and can be used as the biomarker of atherosclerosis. We found that dietary supplemental 23-hydroxy ursolic acid, a phytochemical, protects mice monocytes against nutrient-stressed induced weight gain, adipose tissue inflammation, and atherogenesis.

Oxidative stress plays key roles in monocyte and macrophage biology in atherogenesis, from the formation of early fatty streaks to the development of advanced plaques. Glutaredoxin 1 (Grx1) mediates the deglutathionylation of proteins induced by H2O2. Grx1 protects monocytes from nutrient-stress-induced inactivation of MKP-1, a master regulator of monocyte adhesion and chemotaxis, and macrophage functions and plasticity. This new macrophage-dependent mechanism may contribute to the well-established differences in cardiovascular risk between men and women and to the elevated obesity and cardiovascular risk among postmenopausal women.