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RIP3-dependent necrosis induced inflammation exacerbates atherosclerosis

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Abstract

Atherothrombotic vascular disease is already the leading cause of mortality worldwide. Atherosclerosis shares features with diseases caused by chronic inflammation. More attention should concentrate on the innate immunity effect atherosclerosis progress. RIP3 (receptor-interacting protein kinase 3) act through the transcription factor named Nr4a3 (Nuclear orphan receptors) to regulate cytokine production. Deletion RIP3 decreases IL-1 α production. Injection of anti-IL-1 α antibody protects against the progress of atherosclerosis in ApoE ^{-/-} mice. RIP3 as a molecular switch in necrosis, controls macrophage necrotic death caused inflammation. Inhibiting necrosis will certainly reduce atherosclerosis through limit inflammation. Necrotic cell death caused systemic

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