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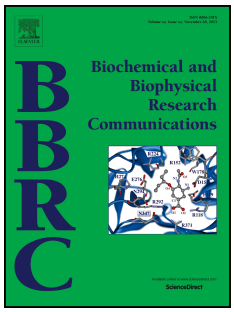

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Lingjun Meng, Wei Jin, Yuhui Wang, Huanwei Huang, Jia Li, Cai Zhang

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RIP3-dependent necrosis induced inflammation exacerbates atherosclerosis

Lingjun Meng[a,b,1], wei Jin[c], Yuhui Wang[d], Huanwei Huang[b], Jia Li[b], Cai Zhang[b],

[a]College of Biological Sciences, China Agricultural University, Beijing 100094, China; [b]National Institute of Biological Sciences, Beijing 102206, China; [c]Institute For Immunology, Tsinghua University, Beijing 100084, China; [d]Institute of Cardiovascular Sciences, Health Science Center, Peking University, Beijing 100191, China.

[1]To whom correspondence should be addressed. Email: menglingjun@nibs.ac.cn

Abstract

Atherothrombotic vascular disease is already the leading cause of mortality worldwide. Atherosclerosis shares features with diseases caused by chronic inflammation. More attention should concentrates 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-1α production. Injection of anti-IL-1α 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