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Letter to the Editor

Apolipoprotein A-IV may inhibit inflammation via regulating NLRP3/IL-1 path to anti-atherosclerosis

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Dear editor:

Recently, S. Bleda et al. has reported that NLRP1 inflammasome is the key in the shift to proinflammatory state on endothelial cells in peripheral arterial disease [1], but the NLRP3 inflammasome has also gained lots of attraction on involving in inflammation of atherosclerosis via pattern recognition receptor-associated signaling pathway. Furthermore, the Canakinumab Anti-Inflammatory Thrombosis Outcomes Study has demonstrated the exact role of inflammation in atherosclerosis [2] and NLRP3/IL-1 activation contributes to the vascular inflammatory response driving atherosclerosis development and progression [3]. And, NLRP3/IL-1 is activated by various endogenous danger signals [3], similarly, there are also many mediators in the body that block or regulate inflammatory pathways and inhibit atherosclerosis. Apolipoprotein A-IV (ApoA-IV) is a 46-kDa lipid-binding glycoprotein, which has regulation of lipid and glucose, anti-oxidation, anti-inflammatory and anti-atherosclerosis properties [4]. It has been confirmed that apoA-IV knockout mice presented a significantly greater inflammatory, reversely, the exogenous administration of apoA-IV or the overexpression of apoA-IV in apoE knockout mice reduces the susceptibility to atherogenesis by decreasing the secretion of proinflammatory cytokines and diminishing atherosclerotic lesions [5].

ApoA-IV can reverse the inflammatory response during atherosclerosis [5], and we assume that apoA-IV may inhibit inflammation via regulating NLRP3/IL-1 path to anti-atherosclerosis, but its mechanism is unknown and needs further study.

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Conflicts of interest

XL and JP conceived the idea; JP collected and read the literature and wrote the manuscript; XL read through and corrected the manuscript. All authors read and approved the final manuscript. All authors report no conflict of interest.

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