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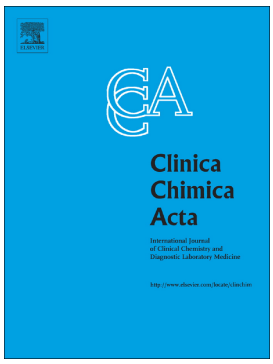

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Atherogenic postprandial remnant lipoproteins；VLDL remnants as a causal factor in atherosclerosis

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Abstract

Oxidized LDL (Ox-LDL) and chylomicron (CM) remnants have been suggested to be the most atherogenic lipoproteins that initiate and exacerbate coronary atherosclerosis. In this review, we propose a hypothesis of the causal lipoproteins in atherosclerosis based on our recent findings on postprandial remnant lipoproteins (RLP). Plasma RLP-C and RLP-TG increased significantly after food intake, especially a fat load. More than 80% of the TG increase after the fat load consisted of the TG in RLP, which contained significantly greater apoB100 than apoB48 particles as VLDL remnants. The majority of the LPL in non-heparin plasma was found in RLP as an RLP-LPL complex and released into the circulation after hydrolysis. Plasma LPL did not increase after food intake, which may have caused the partial hydrolysis of CM and VLDL as well as the significant increase of RLP-TG in the postprandial plasma. LPL was inversely correlated with the RLP particle size after food intake. We showed that VLDL remnants are the major atherogenc lipoproteins in the postprandial plasma associated with insufficient LPL activity and a causal factor in the initiation and progression of atherosclerosis. We also propose “LPL bound TG-rich lipoproteins” as a new definition of remnant lipoproteins based on the findings of the RLP-LPL complex in the non-heparin plasma.