Journal of Lipids

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Special Issue on

# Regulation of LDL and ox-LDL Accumulation: Novel Findings in Atherosclerosis and Cardiovascular Diseases

CALL FOR PAPERS

Low density lipoprotein (LDL) accumulation within arteries is the initial process of atherosclerosis. Containing large amount of unsaturated fatty acids, LDL is prone to be converted to oxidized LDL (ox-LDL), which is known to promote atherogenesis through foam cell formation and inflammatory responses. Besides shear stress mediated mass transport, LDL accumulation was determined by LDL receptor (LDLr), while ox-LDL accumulation was regulated by lectin-like LDL receptor-1 (LOX-1), cluster differentiating 36 (CD36), scavenger receptor class A (SRA), and possibly other receptors. Beyond oxidized LDL, many other factors also play important roles in the atherosclerosis and its related cardiovascular diseases, such as inflammation, blood pressure, and blood glucose. Recently, proprotein convertase subtilisin/kexin type 9 (PCSK9) was proven to indirectly regulate LDL uptake by inducing LDLR degradation. Thus, PCSK9 inhibition has emerged as a potential novel drug therapy to treat hypercholesterolemia and associated disease states.

This special issue is intended to solicit high quality, original research articles as well as review articles focused on the role of LDL and ox-LDL accumulation regulation in atherosclerosis and cardiovascular diseases, which could open new therapeutic targets for the management and the prevention of atherosclerosis.

Potential topics include but are not limited to the following:

- The possible mechanism in the conversion of LDL to ox-LDL, such as reactive oxygen species (ROS), superoxide dismutase (SOD), glutathione peroxidase, and catalase
- The crosstalk between PCSK9, LOX-1, CD36, and SRA in vascular cells and macrophages
- Effect of hemodynamics (such as swirling, disturbed, oscillatory, and steady flow) on the expression of PCSK9, LOX-1, CD36, and SRA
- Numerical simulation of LDL/ox-LDL particles mass transport in arteries and the role of PCSK9, LOX-1, CD36, and SRA in this process
- Basic or clinical studies on LOX-1, CD36, SRA, and PCSK9 inhibitors treatment and clinical studies on the role of antioxidants in regulation of LDL/ox-LDL accumulation, such as HOPE trials and the CARET study
- Studies using new methodological paradigms that challenge current thinking in basic or clinical research for atherosclerosis

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/jl/ldox/.

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