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Probucol protects endothelial progenitor cells against oxidized low-density lipoprotein via suppression of reactive oxygen species formation *in vivo*

Yuqi Cui

Ohio State University, USA

Oxidized low-density lipoprotein (Ox-LDL) is a major component of hyperlipidemia and contributes to atherosclerosis. Endothelial progenitor cells (EPCs) play an important role in preventing atherosclerosis and notably decreased in hyperlipidemia. Ox-LDL and Ox-LDL-related reactive oxygen species (ROS) have deleterious effects on EPCs. Probucol as an antioxidant and anti-inflammatory drug reduces ROS production. The present study was to determine if probucol could protect EPCs from ox-LDL *in vivo* and to investigate the potential mechanisms. Ox-LDL was injected into male C57BL/6 mice for 3 days with or without probucol treatment with PBS as control. Bone marrow (BM) fluid, serum, circulating mononuclear cells (MNCs) and EPCs were collected for analysis. The increased extracellular ROS in BM, serum and blood intracellular ROS production in the mice with Ox-LDL treatment in association with a significant reduction of circulating MNCs and EPCs were restored with probucol treatment. A significant increase in the serum Ox-LDL and C-reactive protein and decrease in superoxide dismutase and circulating MNCs and EPCs were observed in hyperlipidemic patients that were effectively reversed with probucol treatment. These data suggested that probucol could protect EPCs from Ox-LDL through inhibition of ROS production *in vivo*.

burningmoon2442@hotmail.com