S52-5 Cross-talk between statins and high-density lipoprotein in anti-atherogenic and anti-inflammatory actions

◯Fumikazu OKAJIMA[1]

[1]Inst. for Mol. Cell. Reg., Gunma Univ.

The reverse cholesterol transport mediated by high-density lipoprotein (HDL) is an important mechanism for maintaining body cholesterol at normal levels and, hence, the critical anti-atherogenic action of the lipoprotein. Recent studies, however, showed that HDL exerts a variety of anti-inflammatory and anti-atherogenic actions independently of the cholesterol metabolism. On the other hand, statins, inhibitors of HMG-CoA reductase, were initially developed to lower low-density lipoprotein cholesterol in plasma, and they are now recognized to exert a variety of pleiotropic or beneficial actions. Thus, although the mechanisms are different, both endogenous HDL and exogenous statins regulate cholesterol balance in a negative manner and exert a variety of beneficial actions independently of their cholesterol-lowering activity. These results raise the possibility that statins act in part through modulating the plasma levels of HDL and/or its actions. Here, we reviewed the cross-talk mechanism between statins and HDL in anti-inflammatory and anti-atherogenic actions, with a focus on scavenger receptor class B type I, one of main players involved in the cholesterol metabolism-independent HDL actions, and its downstream signaling pathway, leading to the activation of endothelial nitric oxide synthase and the inhibition of adhesion molecule expression in endothelial cells.