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Connection between Portal Venous System and Hepatic Inflammation

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Bacterial endotoxin dysregulate metabolism including liver-releated diseases. Our body establish innate immune systems to protect against endotoxin-induced damages, but gut-derived endotoxin can overcome our defense systems. Generation of high density lipoprotein (HDL) requires Apolipoprotein A1 (ApoA1) and the cholesterol transporter ABCA1. Although the liver produces most HDL in circulation systems, HDL synthesis also occurs in the small intestine. However, distinct functions for intestinal HDL are unrevealed. Here, we showed that HDL in the portal vein, which connects intestine to liver, derived mainly from intestine via using photoconvertible GFP-tagged ApoA1 knockin mice. Intestine-derived HDL in portal vein was mainly composed of small-sized HDL3 and showed strong effects on neutralization of lipopolysaccharide (LPS) endotoxin. In a mouse model of short bowel syndrome which induces dramatic liver inflammation and fibrosis via TLR4, loss of intestine-derived HDL worsened liver injury, whereas liver pathology was improved by therapeutic challenge of low-dose oral LXR agonist that elevated and depended upon intestinal HDL production. Additionally, we found novel innate immunity system in portal vein to effectively remove bacteria translocation. Thus, we found that protection of the liver from injury in response to gut-derived signals like LPS is a major function of intestinally synthesized HDL and portal innate immunity.

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