**LRH-1 deficiency accelerates the development of NAFLD by inhibiting the production of hydrogen sulfate in the liver**

**Seung-Soon Im, PhD**

Department of Physiology, Keimyung University School of Medicine, Daegu, Korea

Liver receptor homolog-1 (LRH-1) is a member of the nuclear receptor NR5A subfamily. It plays a crucial role in bile acid synthesis and cholesterol reverse transport in the liver and pancreas. However, the function of LRH-1 has been not well addressed in liver diseases. In this study, we investigated to identify new target of LRH-1 in CDA-HFD induced NAFLD model, and found that cystathione -layse (CTH) may be a putative target of LRH-1. CTH is an enzyme among regulatory enzymes of hydrogen sulfide production like cystathione -layse (CTH), cystathione -synthase, and 3-mecaptopyruvate sulfur transferase. CTH gene expression is decreased in LRH-1 KO mice livers. In addition, we investigate how LHR-1 controls CTH expression and the impact of hydrogen sulfide on the hepatic triglyceride accumulation in the liver. Also, it was confirmed that hydrogen sulfide production was significantly reduced in LRH-1 LKO mice than in WT mice. In conclusion, this study supports that CTH is a putative target of LRH-1 and that LRH-1 deficiency leads to reduced hydrogen sulfide production by downregulating CTH expression, indicating that LRH-1 KO stimulates hapatic steatosis in NAFLD.