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Poster Sessions

Disruption of steroidogenic acute regulatory protein-related lipid transfer domain containing 10 (STARD10) prevents the development of nonalcoholic steatohepatitis (NASH)

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Nonalcoholic fatty liver disease (NAFLD) is associated with lipid accumulation in hepatocytes and advances into NASH. A recent study implicated that *STARD10* gene expression is epigenetically regulated by methylation in the liver of NAFLD patients. We found that STARD10 is highly expressed in the liver and STARD10 knockout mice (*Stard10*^{-/-}) are resistant to develop fatty liver when fed high fat diet. Therefore we hypothesized that *Stard10*^{-/-} is resistant to develop NASH. Aimig at elucidating the hypothesis, we induced NASH model by choline deficient diet and compared the lipid accumulation in the liver between wild type (WT) and *Stard10*^{-/-} mice. Total lipid droplet area of *Stard10*^{-/-} mice was significantly smaller than that of WT mice. Gene expression levels of proinflammatory cytokines and fibrosis marker genes were significantly lower in the liver of *Stard10*^{-/-} mice compared with WT mice. It is well known that cytokines released from Kupffer cells promote NASH. However, *Stard10* was not expressed in Kupffer cells. We also confirmed that there was no difference in the lipopolysaccharide-induced gene expression of proinflammatory cytokines in the Kupffer cells or macrophages between WT and *Stard10*^{-/-} mice. In conclusion, the disruption of STARD10 prevented the liver from developing NASH through the reduced accumulation of lipids.