Carbon tetrachloride mediated liver fibrosis is alleviated in α7 nicotinic acetylcholine receptor knockout mice

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Background: Cirrhosis is a condition come from excessive liver fibrosis and followed by serious secondary diseases, but there is no effective therapeutic medicine. α7 nicotinic acetylcholine receptor (α7nAChR), initially found as a receptor related to neurotransmission on neural cells. This receptor also expresses on immune cells to do anti-inflammatory action. However, there is few reports showing the relationship between α7nAChR and fibrosis.
Aim: We investigated whether α7nAChR has any effects on liver fibrosis and what is the mechanism.
Methods: Liver fibrosis model mice were established with CCl_4. The pro-fibrotic mRNA expressions and collagen content in livers were measured at 1.5 and 4 weeks. Moreover, we performed immunohistochemical staining and RT-PCR to determine which cells were involved in the mechanism.
Results: α7nAChR KO mice treated with CCl_4 showed significant decrease in pro-fibrotic mRNA expressions at 1.5 weeks and liver fibrosis at 4 weeks compared to WT mice. Furthermore, hepatocytes around fibrosis area expressed ACh transferase and activated hepatic stellate cells expressed α7nAChR.
Conclusion: The severity of fibrosis was significantly decreased in α7nAChR KO mice. Moreover, it is suggested that ACh produced by hepatocytes might stimulate hepatic stellate cells to promote collagen production.