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Adipose tissue-liver axis in alcoholic liver disease

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## Abstract

Alcoholic liver disease (ALD) remains an important health problem worldwide. The disease spectrum is featured by early steatosis, steatohepatitis (steatosis with inflammatory cells infiltration and necrosis), with some individuals ultimately progressing to fibrosis/cirrhosis. Although the disease progression is well characterized, no effective therapies are currently available for the treatment in humans. The mechanisms underlying the initiation and progression of ALD are multifactorial and complex. Emerging evidence supports that adipose tissue dysfunction contributes to the pathogenesis of ALD. In the first part of this review, we discuss the mechanisms whereby chronic alcohol exposure contributed to adipose tissue dysfunction, including cell death, inflammation and insulin resistance. It has been long known that aberrant hepatic methionine metabolism is a major metabolic abnormality induced by chronic alcohol exposure and plays an etiological role in the pathogenesis of ALD. The recent studies in our group documented the similar metabolic effect of chronic alcohol drinking on methionine in adipose tissue. In the second part of this review, we also

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