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# Therapeutic Potentials for ALD by Targeting ER Stress Pathways †

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**Abstract:** The accumulation of misfolded proteins or depletion of calcium in the Endoplasmic reticulum is the major cause of ER stress. One of the major chronic diseases of the liver is AlcoholicLiver Disease (ALD). ALD is associated with simple steatosis, which may progress to more severe steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma. In many experimental ALD disease models and patients affected with ALD, it has been observed that ER stress and unfolded protein response (UPR) pathways are activated. UPR genes, such as BIP and CHOP have been found to be highly expressed in mice that are alcohol-fed intragastrically. Disturbances in lipid regulation and homeostasis in liver cells cause a build-up of lipids. This hepatic lipid accumulation activates ER stress, which in turn leads to UPR. This review intends to survey the potential therapeutic implications of ALD about ER stress and UPR pathways. It is anticipated that the IRE1α/XBP1 pathway can regulate ALD-induced hepatic steatosis; its role will also be discussed.

**Keywords:** endoplasmic reticulum stress (ER); alcoholic liver disease(ALD); unfolded protein response (UPR); BIP; CHOP; IRE1α.



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## Conflicts of Interest

The authors declare no conflict of interest.