ALCOHOL AND IRON METABOLISM

Summary

Consumption of alcohol is known to be associated with misregulation of iron metabolism. Patients with alcoholic liver disease frequently exhibit increased hepatic iron content, which is caused by the increased iron absorption in duodenum. Within the past 12 years an enormous progress has been made in understanding molecular basis of mammalian iron homeostasis. In particular, the discovery of liver-derived peptide, hepcidin, and its role in the concerted regulation of iron release from absorptive enterocytes and macrophages through interaction with ferroportin, the sole cellular iron exporter known in mammalian cells, has proved to be fundamental in the understanding of iron circulation in the body. The binding of hepcidin to ferroportin expressed at the surface of enterocytes induces its internalization and degradation, which in turn inhibits iron absorption from the diet. The molecular mechanisms underlying alcohol-induced iron accumulation in the body involves suppression of hepcidin expression in hepatocytes, which in consequence leads to increased duodenal iron transport. Exacerbation of alcohol-induced oxidative stress in the liver by iron overload is responsible for liver injury observed in the alcoholic liver disease.