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Gut-liver axis signaling in portal hypertension

Simbrunner B *et al.* Gut-liver axis signaling in portal hypertension

Abstract

Portal hypertension (PHT) in advanced chronic liver disease (ACLD) results from increased intrahepatic resistance caused by pathologic changes of tissue composition (structural component) and intrahepatic vasoconstriction (functional component). PHT is an important driver of hepatic decompensation such as development of ascites or variceal bleeding. Dysbiosis and an impaired intestinal barrier in ACLD facilitate translocation of bacteria and pathogen-associated molecular patterns that promote disease progression *via* immune system activation with subsequent induction of proinflammatory and profibrogenic pathways. One decisive pathophysiological mechanism linking PHT to increased intestinal permeability lies in the congestive portal venous blood flow: The intestinal barrier function is affected by impaired microcirculation, neoangiogenesis, and abnormal vascular and mucosal permeability. The close bidirectional relationship between the gut and the liver has been termed "gut-liver axis". Treatment strategies targeting the gut-liver axis *via* modulation of microbiota composition and function, intestinal barrier integrity, as well as amelioration of liver fibrosis and PHT, are supposed to exert beneficial effects. The activation of the farnesoid X receptor in the liver and the gut and associated fibroblast growth factor 19 signaling have shown promising results in animal experiments, however, further studies regarding efficacy and safety

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Portal hypertension

Portal hypertension is hypertension in the hepatic portal system – made up of the portal vein and its branches, that drain from most of the intestine to the liver. Portal hypertension is defined as a hepatic venous pressure gradient. Cirrhosis is the most common cause of portal hypertension; other, less frequent causes are therefore grouped as non-cirrhotic portal hypertension. When it becomes severe enough to cause symptoms or complications, treatment may be given to decrease portal hypertension itself or to manage its complications.

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