LACTOCOCCUS GARVIEAE AGGRAVATES CHOLESTATIC LIVER DISEASE THROUGH PROMOTING INTESTINAL PERMEABILITY AND ENHANCING BILE ACID REABSORPTION

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UEG Week Copenhagen 2023 | Poster

Hepatobiliary

October 17, 2023

Lactococcus garvieae, a bacterium detected in PBC patients but not controls, exacerbates experimental cholestatic liver disease in mice by disrupting intestinal barrier function and enhancing bile acid reabsorption.

KEY POINTS

- Lactococcus garvieae was isolated from livers and gallbladders of cholestatic mice and detected in PBC patients but not in control subjects with hepatic cysts.
- Mice inoculated with L. garvieae exhibited increased susceptibility to experimental cholestatic liver disease, while those inoculated with Ligilactobacillus murinus did not.
- L. garvieae disrupted intestinal barrier function through downregulation of tight junction proteins, increasing intestinal bacterial translocation and liver inflammatory responses.
- L. garvieae decreased apical sodium-dependent bile acid transporter expression in the terminal ileum, increased serum taurine-conjugated bile acids, reduced bile salt hydrolase activity and faecal bile acid excretion, and enhanced hepatic bile acid accumulation and liver fibrosis.
- The authors conclude that L. garvieae contributes to bile acid-induced hepatobiliary injury and fibrosis through promoting intestinal permeability and enhancing bile acid reabsorption in this murine model.

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