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Gut microbiota regulates bile acid metabolism

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A new study presented today at the International Liver Congress 2012 demonstrates that the gut microbiota has a profound systemic effect on bile acid metabolism.

Bile acids are synthesised from cholesterol in the liver and further metabolised by the gut microbiota into secondary bile acids. The main function of bile acid is to promote processing of dietary fat. In addition, hepatic synthesis of bile acids is a major mechanism of cholesterol breakdown in the body. Farnesoid-x-receptor (FXR) is known to play a key role in the regulation of bile acid synthesis and homeostasis.

The authors propose that the gut microbiota modulates bile acid synthesis by changing the bile acid pool composition and, hence, reducing FXR inhibition in the small intestine.

The study found that germ-free mice (without any gut microbiota) had elevated muricholic bile acid levels, in particular TβMCA (tauroconjugated β-muricholic acid), while conventionally housed mice (with gut microbiota) had reduced levels of muricholic acids. TβMCA blocks the activation of FXR.

The results demonstrate the gut microbiota's suppression of biosynthetic genes in the liver cholesterol-7α-hydroxylase (CYP7A1) was consistent with increased FXR-dependent activation of FGF15 (ileal fibroblast growth factor-15) in the small intestine, due to reduced TβMCA mediated inhibition of FXR.

The gut microbiota performs unique digestive functions that cannot be performed by a germ-free intestinal tract. The cells constituting the microbial organ form metabolic and signalling networks with each other and their host.

More information: Islam S et al., Gut microbiota regulates bile acid metabolism by reducing the levels of tauro-betamuricholic acid, a naturally occurring FXR antagonist. Abstract presented at The International Liver Congress 2012.

Provided by European Association for the Study of the Liver