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Short Communication

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Clinical Relevance of Bile Acid and their Conjugates in Health and Disease

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1. Short Communication

Bile Acids (BA) are synthesized by the oxidation of cholesterol in the liver and secreted into the duodenum. BAs were circulated from the duodenum back to the liver by enterohepatic recirculation. BAs regulate many physiological functions, including the expression of genes involved cholesterol, glucose, and their homeostasis. BAs have several pathologic effects, including carcinogenicity, liver toxicity and many others.

In hepatobiliary diseases, urinary excretion of BAs increases more than 100 times. Parallel to the situation under normal condition, a large proportion of BAs in urine is eliminated in the sulphate conjugated form under cholestatic condition [1]. The excretion of BA-sulphate in the urine to be used as specific biomarker for the diagnosis of intrahepatic cholestasis in pregnant women [2]. Other studies, nonetheless, have revealed that the percentage of sulfated BAs in serum in hepatobiliary diseases rises of total BAs. Sulfated BAs in bile remains low or further decrease under cholestatic condition (Fischer et al., 1996). This may be illustrated to the impairment of the liver's capability to sulfate BAs as results of the worsened liver function under these pathologic conditions [3, 4]. It is also likely that sulfation of BAs is increased as a compensatory mechanism in early stages of cholestasis, but with further damage at advanced stages, the liver function to sulfate BAs is diminished [5].

Based on the genetic expression, Bile acid-CoA: amino acid N-acyltransferase (BAAT) catalyzed bile acid conjugation, the last step in BA synthesis. BAAT gene mutation in humans' results in hypercholanemia, growth retardation, and fat-soluble vitamin insufficiency [6]. A single homozygous mutation was first described in Amish individuals with familial hypercholanemia, who characterized an almost absence of conjugated BA in the serum [7]. A detailed clinical and biochemical feature demonstrated by conjugated BA deficiency caused by homozygous BAAT mutation [8].

We have developed and validated the BA-liver disease complication model based on BA indices using logistic regression model to predict the prognosis of cholestatic liver disease, including ascites based on BA and sulfated BA as biomarker [9]. Also other evidence in preclinical models of Inflammatory Bowel Disease (IBD) suggests reduced sulfation causes barrier dysfunction, inflammation or carcinogenesis [10]. The characterization of the detailed BAs and their conjugates profile in healthy subjects in different biological metrices will facilitate better understanding towards human health and disease.

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