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Metabolism of glycerophospholipid and bile acid are disturbed in human, mice and rats with liver diseases

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Chronic hepatitis B virus (HBV) infection is a huge burden to public health. An estimate 375 million people are chronically HBV infected worldwide. And the number of HBV infected people continues to increase with 4.5 million every year. Patients with HBV infection are apt to develop advanced liver disease such as cirrhosis and hepatocellular carcinoma. But there is a noteworthy disparity of HBV treatment in current clinical practice which is largely attributed to the poor understanding on the pathogenesis of HBV-mediated liver injury. Serum contains a large variety of small-molecular metabolites which are closely related to physiological states. Comprehensive analysis of these metabolites can hence facilitate the understanding of mechanism underlying the disease. Therefore, to decipher the mechanism underlying HBV-mediated liver injury, mass spectrometry-based metabolomic analysis was carried out in patients with chronic hepatitis B (CHB), liver failure (LF) and healthy controls. As a result, significantly decreasing of lysophosphatidylcholines accompanied with increasing of bile acids were found in patients with CHB and LF. In addition, to mimic the clinical symptoms of the injury, two animal models were replicated, including mice model with immune-mediated liver injury induced by concanavalin A and rats model with chemical liver injury induced by CCl4. Metabolic profiling analysis of animal models also showed a similar alteration in lysophosphatidylcholines and bile acids. Therefore, our cross-species analysis of serum provides evidence for a common architecture in liver injury and present disorders of glycerophospholipid and bile acid metabolism are the key players in pathogenesis of the liver injury.

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The role of apolipoprotein E in uptake of atovaquone into the brain in murine acute and reactivated toxoplasmosis

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We investigated whether coating of atovaquone nanosuspensions (ANSs) with apolipoprotein E (apoE) peptides improves the uptake of atovaquone into the brain. The passage across the blood-brain barrier (BBB) of ANSs stabilized by polysorbate 80 (Tween® 80), poloxamer 184 (P184), or poloxamer 338 (P338) and the same formulations coated with apoE peptides were analyzed *in vitro* and *in vivo*. Passage through a rat coculture model of the BBB did not differ between individual atovaquone formulations, and the addition of apoE peptides did not enhance the transport. Following the induction of toxoplasmic encephalitis (TE) in mice, treatment with all atovaquone formulations reduced the number of parasites and inflammatory foci compared with untreated mice. Uptake of atovaquone into the brain did not depend on coating with apoE. Finally, incubation of apoE peptide–coated ANSs with brain endothelial cells for 30 min did result in the accumulation of nanoparticles on the cell surface but not in their uptake into the cells. In conclusion, ANSs coated with Tween® 80 or poloxamers showed therapeutic efficacy in murine toxoplasmosis. ApoE- and apoE-derived peptides do not induce the uptake of ANSs into the brain. Alternative mechanisms seem to be in operation, thereby mediating the passage of atovaquone across the BBB.

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