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of hepatic bile salt transporter Na+ taurocholate cotransporting polypeptide (NTCP) prevents the dysregulated metabolism in Fxr-/- mice.

Methods: WT, Fxr-/-, Ntcp-/-, and Fxr-/- Ntcp-/- mice received a high-fat diet (60%) for 15 weeks or a single injection of DEN at age of 2 weeks and chow for 36 weeks.

Results: FXR deletion elevated toxicity markers ALT, AST, and ALP in plasma, lowered body weight and delayed glucose clearance during an oral glucose tolerance test. NTCP deletion attenuated ALT, AST, and ALP levels and fully prevented the reduction in body weight and the delayed glucose clearance. Carcinoma marker AFP was elevated in the DEN-treated Fxr-/- group, and this also normalized upon genetic NTCP deletion.

Discussion/Conclusion: These data indicate that dysregulated metabolism in Fxr-/- mice may be a consequence of hepatotoxicity, due to hepatic bile salt accumulation. We are currently investigating underlying mechanisms.

42. Difficulties of NAFLD treatment. How to improve it?

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Introduction: Treatment of NAFLD requires a decision of which patients need to be treated, because not all patients progress to steatohepatitis, fibrosis or cirrhosis. Aim of our study was to evaluate the role of UDCA and vitamin E in improvement of NAFLD evolution.

Methods: We included 60 patients with NAFLD evaluated by FibroMax at the beginning and in the end of the study and excluded the patients with others conditions associated with hepatic steatosis. We divided the patients into two groups matched by gender, weight and fibrosis degree: group A (31 patients treated with UDCA 10 mg/kg/day) and group B (29 patients with UDCA 10 mg/kg/day + 400 mg vitamin E per day). All patients were also placed on a 1200-calorie diet during the period 6 months. In all patients we calculated BMI, fatty Liver Index (FLI), NAFLD Liver Fat Score (NAFLD-LFS) before medication and after 6 months. In both groups we determined aminotransferases, GGT, lipid profile, albumin, glucose, at the beginning and at the end of the study. No significant differences between the two groups in ALT, AST, GGT, and lipid profile at the beginning.

Results: After 6 months' treatment aminotransferases and GGT levels diminished in UDCA group compared with the initial values, without statistical significant from the beginning and were normal in the combination group, with significant statistical difference between the start and the end of treatment (p < 0.001). FLI and NAFLD-LFS were significant better compared with the beginning in the group B but we also observed a better score in UDCA group. No improvement for lipid profile in both groups. No differences during the study period in BMI, presence of diabetes mellitus or metabolic syndrome in all patients. We noticed an improvement of fibrosis degree (evaluated by FibroMax) in the group B but without statistical significance compared with the start.

Discussion/Conclusion: UDCA in dose 10 mg/kg/day improves aminotransferases levels and fatty liver scores in patients with NAFLD but the combination of UDCA with vitamin E seems to be more efficient in normalizing aminotransferases and also in fibrosis progress during 6 months.

43. Generation of an improved murine model for progressive familial intrahepatic cholestasis type 3

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