ficial taxa relative RNA and DNA abundances are consistent, which can predict hospitalizations similarly.

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Different intestinal microbiota profile in alcoholic pancreatitis as compared to alcoholic hepatitis

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Objective Chronic excessive alcohol consumption may cause alcoholic liver disease (ALD) or alcoholic pancreatitis (AP) in only a subset of patients. We have shown that individual susceptibility to ALD is substantially driven by intestinal microbiota (IM). However, factors related to tissue predilection, liver or pancreas, to alcohol toxicity are unknown. We aimed to characterize the IM profile in alcoholic patients according to the presence and the nature of the complication ie severe alcoholic hepatitis (sAH) or AP. **Design** 82 alcoholic patients were included into 3 groups according to their complications: AP (N=24), sAH (N=13) and no complication despite a similar amount of alcohol consumption (alcoholic controls, N=45). IM was analyzed using high-throughput sequencing of the 16S Ribosomal RNA (16S RNA) gene. **Results** Patients with AP had a reduced bacterial diversity (p=0.001) and a different global microbial composition as compared to alcoholic controls (p=0.001). 17 taxa at the genus level were different between the 2 groups; among them, 8 were increased in AP (*Klebsiella, Enterococcus, Aquabacterium and Sphingomonas*). When compared to sAH there was no difference in bacterial diversity between the 2 groups. However, 16 taxa were increased in sAH and 10 in AP. After adjusting for confounding factors (age, sex, BMI, alcohol intake, diabetes and proton-pump inhibitors) there was a marked increase in *Haemophilus* in sAH patients. **Conclusion** Patients with AP have a specific dysbiosis as compared to alcoholic controls. Specific microbiome signatures are associated with AP and sAH.

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Intestinal Microbiota Transplantation From HFD-fed and Quercetin Treated Donors Results in a Complex Metabolic Phenotype Transfer that Modulates Obesity-Related NAFLD in Germ Free Mice

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Background: Intestinal microbiota imbalance and related gut-liver axis activation have been identified as key mechanisms in nonalcoholic fatty liver disease (NAFLD) development. Modulation of intestinal microbiota, through administration of prebiotics or faecal microbiota transplantation, is a promising therapeutic approach for obesity associated diseases including NAFLD. The aim of the present study is to evaluate the benefits of gut microbiota transplantation from donors to germ free mice (GFm) following an experimental treatment with the flavonoid quercetin in a high fat diet (HFD)-based NAFLD model. **Methods:** GFm were colonised with gut microbiota from donors and fed with control or HFD for 16 weeks. Gut bacterial communities were identified pyrosequencing the 16S-rRNA from caecal samples of donors and GFm. Caecal microbiota donors were selected from control (dC), HFD-fed (dHFD- and dHFD+, as non-responder and responder to the HFD, respectively) and control and HFD supplemented with quercetin (dCQ and dHFDQ) groups, according to metabolic parameters. **Results:** dHFD- and dHFDQ-receiver groups fed with HFD showed reduced body weight gain, NAFLD activity score, HOMA-IR, and endotoxemia, with respect to other receivers. dHFD+ phenotype transfer was associated with increased NAS index and hepatic markers alteration in control diet-fed mice. The microbial composition at phylum level in donor mice showed an increase in *Firmicutes* and *Verrucomicrobia* in dHFD- and dHFDQ, respectively, in comparison to the other donors. At the genus level, a higher detection of *Helicobacter* was observed in dHFD+ vs dHFD-, while *Oscillospira*, *Lactobacillus* and *Alkaliphilus* exhibited an opposite pattern. Interestingly, a dramatically increase of *Akkermansia* was detected in dHFDQ with respect the other donors. In GFm dC, dCQ, dHFD- and dHFDQ-receiver groups fed with HFD a notable increase in *Verrucomicrobia* was observed, which was undetectable in dHFD+-receiver groups independently of the diet. *Akkermansia* genus was increased in HFD-fed dC, dCQ, dHFD- and dHFDQ-receivers and undetected in all dHFD+-receiver groups. Differences in microbiota composition were accompanied by gut-liver axis disturbance and inflammasome activation in dHFD+-receiver mice independently of the diet. **Conclusions:** This different microbiota composition could be associated with the transfer of a complex metabolic phenotype with specific functionality in the receivers. Our data sustain the suitability of intestinal microbiota transplantation as a therapeutic approach for obesity-associated NAFLD. Supported by BFU2013-48141-R, LE063U16 (JCyL and FEDER) and GRS 1428/A/16. CIBERehd is funded by ISCIII