seroconversion, a key stage to T1D, the conventional analysis found some microbiota with changed abundance after the occurrence of seroconversion (figure 1a). Meanwhile, our analysis further show that a lot of microbiota actually have great abundance variance change before seroconversion (figure 1b), which can even be efficient features to classify the healthy and seroconversion individuals with about 80% accuracy (figure 1c). More importantly, this microbiota with critical abundance variance as key species is also associated with T1D clinical antibody or even some new (figure 1d).

Conclusions Dissimilar to common biomarkers like a clinical antibody, the individual specific signatures, e.g. variance of gut microbiota abundance, would be an alternative approach for personalised pre-disease or early-disease diagnosis.

**GUT MICROBIOME ACROSS STAGES OF HBV INFECTION**

Zhangran Chen*, Yuru Xie, Chuanxing Xiao, Xiang Zhang, Jianlin Ren. Institute for Microbial Ecology, Medical College of Xiamen University, China

Background Increasing evidence suggests that the gut microbiota has evolved as a new important player in the pathogenesis of hepatitis B virus-induced chronic liver disease, including chronic hepatitis, cirrhosis. However, the composition and structure alteration of the gut microbiota associated with the stage and progression of HBV infection remains unknown. Hence, the aim of the study is to elucidate the microbial influence which contributes to the microbial shift of HBV patients in different stages.

Methods All subjects enrolled had clinic test performed in the Zhongshan Hospital, Xiamen University. A total of 62 subjects were finally selected for inclusion in this study, including 7 healthy individuals, 13 HBeAg negative chronic HBV infection patients (HNCHB), 6 HBeAg negative chronic hepatitis B patients (HNCHB), 5 HBeAg positive chronic hepatitis B patients (HPCHB) and 31 decompensated cirrhosis after hepatitis B patients (DCAHB) matched in age, gender and body mass index (BMI). Stools from all subjects were collected to extract DNA, then for 16S rDNA high-throughput sequencing analysis further show that a lot of microbiota actually have great abundance variance change before seroconversion (figure 1a). Meanwhile, our analysis further show that a lot of microbiota actually have great abundance variance change before seroconversion (figure 1b), which can even be efficient features to classify the healthy and seroconversion individuals with about 80% accuracy (figure 1c). More importantly, this microbiota with critical abundance variance as key species is also associated with T1D clinical antibody or even some new (figure 1d).

Conclusions Dissimilar to common biomarkers like a clinical antibody, the individual specific signatures, e.g. variance of gut microbiota abundance, would be an alternative approach for personalised pre-disease or early-disease diagnosis.

**ALTERED GUT MICROBIOTA IN EXPERIMENTAL MOUSE MODELS OF NON-ALCOHOLIC FATTY LIVER DISEASE**

Bangzhou Zhang*, Lina Fan, Chuanxing Xiao, Jianlin Ren. Department of Gastroenterology, Zhongshan Hospital Affiliated to Xiamen University, China

Background Non-alcoholic fatty liver disease (NAFLD) has become the most common liver disease worldwide and is thought to be strongly associated with gut microbiota. Several diet models were therefore built in mice to try to clarify the molecular mechanisms. However, how and to what extent these diet models alter the composition of the gut microbiota have not yet been clearly elucidated.

Methods In this study, we developed three mouse models of NAFLD using methionine-choline-deficient (MCD) diet, high-fat (HF) diet, and choline-deficient–high-fat (CD-HF) diet, evaluated the severity of steatohepatitis and sequenced the faecal bacteria by targeting 16S V4-V6 regions on Illumina MiSeq using PE 300 reagents.

**ALCOHOLIC FATTY LIVER DISEASE**

Abstract IDDF2018-ABS-0162 Figure 1 PCA ordination of the gut microbiota communities, fitted with serum and liver tissue biochemistry assays (**P<0.01, ***P<0.001)

Results Histological scores showed that MCD induced the severest steatohepatitis, followed by HF and CD-HF diets. Based on operational taxonomic units (OTUs) at cutoff of 97% similarity, there were significant (PERMANOVA, p=0.001) differences in overall gut bacterial communities among MCD, HF, CD-HFD, and the Control, forming three major clusters in PCA ordination with HF and CD-HF groups more similar (figure 1). Furthermore, α-diversity of HF and CD-HF groups, including observed OTU numbers, Shannon index, and Pielou evenness were significantly (ANOVA, p<0.05) higher than the Control and MCD group. Overall,