Bacteroides caccae alleviates hepatic fibrosis by suppressing inflammation

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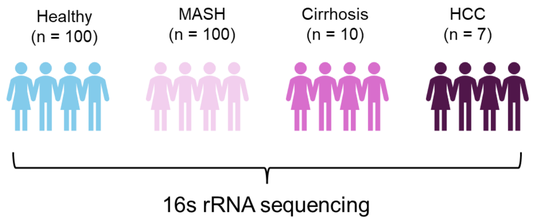
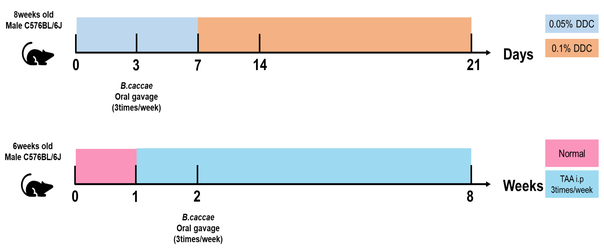
Introduction

Hepatic fibrosis represent a key pathological change in the progression of chronic liver disease (CLD). Various forms of CLD, including metabolic-associated steatohepatitis (MASH), viral hepatitis, alcohol-related liver disease (ALD), and autoimmune liver disease, can progress to cirrhosis, which serves as a major risk factor for hepatocellular carcinoma (HCC). Recent reports suggest that certain microbiotas may help inhibit or ameliorate the progression of MASH-associated cirrhosis. This study aimed to characterize the role of *Bacteroides caccae (B. caccae)* in the gut microbiome of patients with CLD. Furthermore, using a mouse model of MASH-associated cirrhosis, we evaluated the effects of *B. caccae* on the gut microbiota and investigated the associated molecular mechanisms.

Experimental design





Acknowledgments

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Result

Fig1. Comparison of relative abundances of gut microbiome between healthy control and patients.

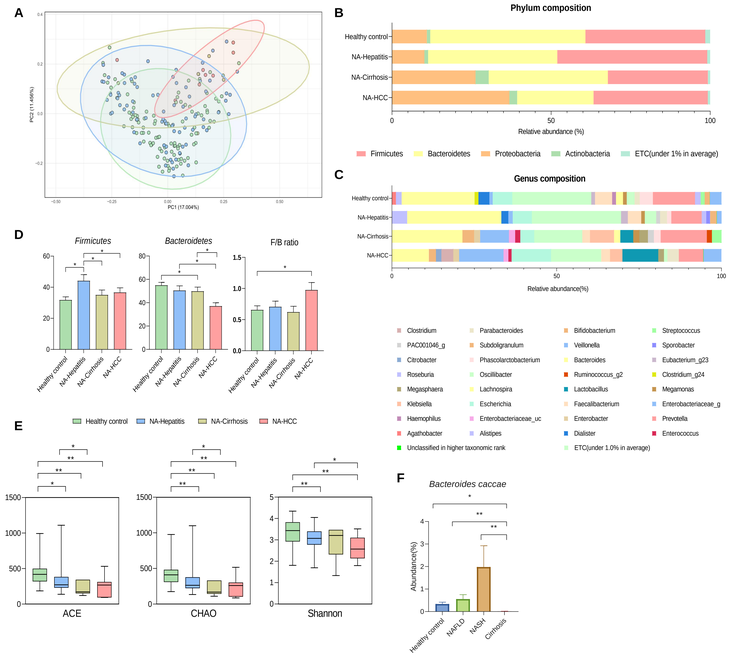


Figure 1. Comparisons of gut microbiome composition and functional biomarkers between healthy controls and NA patients.

(A) Principal coordinate analysis (PCoA) showing beta diversity.

(B–C) Phylum- and genus-level composition of fecal microbiota from healthy controls, NA-Hepatitis, NA-Cirrhosis, and NA-HCC patients.

(D) Relative abundances of Firmicutes, Bacteroidetes, and the Firmicutes/Bacteroidetes (F/B) ratio..

(E) Alpha diversity indices including ACE and Chao1 (species richness), and Shannon index (species evenness).

(F) Relative abundance of *Bacteroides caccae* among patient groups.

Fig2. *B. caccae* improves DDC-induced liver injury by suppressing inflammation and fibrosis.

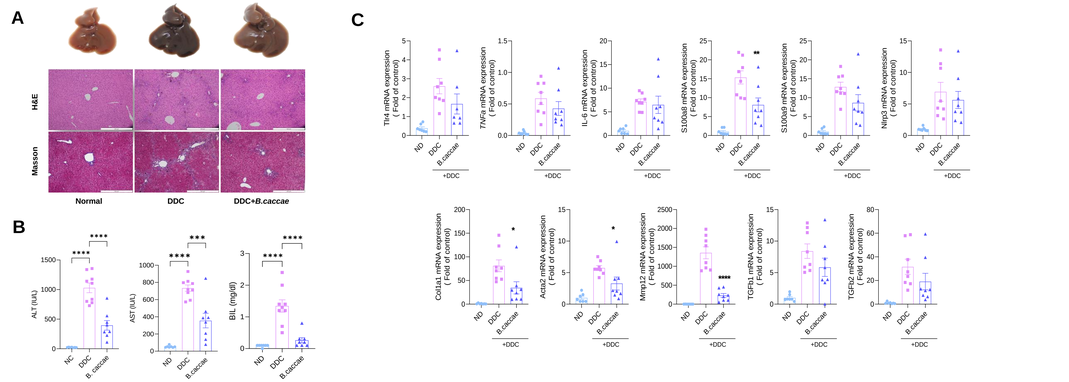


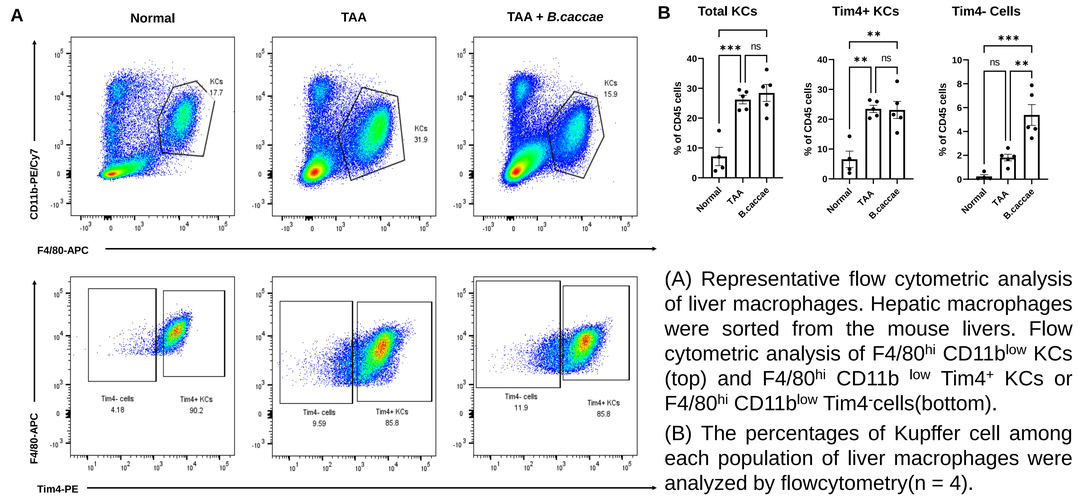
Figure2. *B. caccae* improves DDC-induced liver injury by suppressing inflammation and fibrosis.

(A) Representative liver (top) and H&E staining (middle) and Masson staining (bottom) of liver sections.

(B) Plasma ALT,AST, BIL activities.

(C)The relative transcription levels of measured by qRT-PCR (n=7-8).

Fig3. *B. caccae* reprograms Kupffer cell responses in TAA-induced liver injury.



(A) Representative flow cytometric analysis of liver macrophages. Hepatic macrophages were sorted from the mouse livers. Flow cytometric analysis of F4/80^{hi} CD11b^{low} KCs (top) and F4/80^{hi} CD11b ^{low} Tim4^{+} KCs or F4/80^{hi} CD11b^{low} Tim4^{-}cells(bottom).

(B) The percentages of Kupffer cell among each population of liver macrophages were analyzed by flowcytometry(n = 4).

Conclusion

Bacteroides caccae alleviates hepatic inflammation and fibrosis in MASH-associated cirrhosis by reprogramming Kupffer cell responses and modulating the gut–liver immune axis. These findings highlight the therapeutic potential of gut microbiota modulation in chronic liver disease and underscore the role of B. caccae in reprogramming hepatic immune responses.