Background In our study, the influence of gender, diet, race, living habits, areas, sample types, sequencing methods and other factors on gut microbiome were included in the criteria of sample collection as far as possible, to fill in the deficiencies of the current research in which many factors are mixed to bias the results, and analyze the structure and function of gut microbiome of UC patients and their healthy spouses, speculating the possible pathogenic mechanism during the development of UC.

Methods The feces and intestinal mucosa of 112 patients with first attack untreated UC and mild-moderate UC and their healthy spouses were collected, including 112 males and 112 females. 112 in urban and 112 in rural areas. The race, dietary structure, living environment and lifestyle of UC patients and their healthy spouses were the same. The V3-V4 region of 16S rRNA was sequenced by Illumina sequencer. Among the 112 pairs of UC patients and their healthy spouses, feces of 28 pairs of UC patients and their healthy spouses and mucosa of 56 pairs of UC patients and their healthy spouses were randomly selected and sequenced by Metagenomic Sequencing.

Results Alpha diversity of intestinal microflora was significantly different between UC and HC, and also between Mucosa-associated microbiota(MAM) and Lumen-associated microbiota(LAM). Compared with healthy spouses, the ulcerative colitis, oxidative stress, flagellum assembly, peptidoglycan, lipopolysaccharide and other metabolic pathways of intestinal microflora function in UC patients is significantly enriched in. Metabolic pathways involved in carbohydrate, amino acid, cofactor synthesis and nucleotide for nutrient transport and intake are significantly downregulated.

Conclusions Significant gut microbiome structural and functional disorder were found in UC patients. Compared with LAM, MAM can more precisely reflect the imbalance of gut microbiome of UC. The imbalance of gut microbiome in UC patients leads to increased abundance of oxidative stress pathway and decreased abundance of nutrient delivery and uptake pathway. Enriched bacteria in further UC may be involved in the synthesis of peptidoglycan and lipopolysaccharide, triggering an abnormal immune response. Ultimately, it resulted in persistent, excessive and irreversible immune damage, leading to dysfunction of the intestinal mucosal barrier.
aim to identify the fecal fungal secretome in patients with CRC-stricken gut.

Methods Fecal samples from 26 clinically-diagnosed patients with CRC and 20 non-CRC control individuals were collected, homogenized and filtered followed by protein extraction and profiling by quantitative label-free proteomics using Nano-Liquid Chromatography TripleTOF Mass Spectrometry. The mass spectra datasets were searched using MaxQuant against the fungi’s Uniprot Fasta databases. Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) version 22. Fungal taxa associated with clinical parameters were identified using multivariate association with linear models.

Results We identified 570 fungal proteins secreted into the human gut. A distinct alteration in the diversity and composition of the fungal microbiota was observed in the CRC, with tremendous reductions of the overall fungal proteins, the diversity and composition of fungal microbiota as compared to the control (p<0.05). Interestingly, the proportion of candida was increased in the CRC, indicating a modified gut microenvironment that favours the fungi. However, the discriminative distributions of the fungi secretome proteins in both CRC and non-CRC were observed with great individual variation.

Conclusions The distinct alteration of fungal proteins observed in CRC may suggest a possible CRC-specific gut microenvironment for fungi. This knowledge may be exploited for new therapeutic approaches for gut-related issues.

Basic Hepatology

IDDF2019-ABS-0174 TARGETING MONOCYTE-INTRINSIC ENHANCER REPROGRAMMING IMPROVES IMMUNOTHERAPY EFFICACY IN HEPATOCELLULAR CARCINOMA

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Background Hepatocellular carcinoma (HCC), mostly developed in fibrotic/cirrhotic liver, exhibits relatively low responsiveness to immune-checkpoint blockade (ICB) therapy. As