Abstract IDDF2021-ABS-0165 Figure 1

features (i.e., *Streptococcus*, *Erysipelotrichaceae* UCG-003, *Sutterella* and *Parabacteroides*) were found to be positively associated with severe distress, of which *Erysipelotrichaceae* UCG-003 was more abundant in both distressed and stressed donors (DESeq2, $p < 0.05$; IDDF2021-ABS-0165 Figure C. Bar plot showing differentially abundant genera identified between individuals falling under (i) severe (red) vs. well (blue) category of distress and (ii) high (pink) vs. low (light blue - not detected) stress)). Besides, the abundance of *Megamonas* in the gut was negatively correlated with PSQI after covariates (i.e., age and BMI) adjustment (IDDF2021-ABS-0165 Figure D. Correlation matrix showing the association between differentially abundant bacterial genera and K10, PSS and PSQI categories after covariates adjustment (based on Pearson's correlation; $p < 0.05$)), suggesting the lowered abundance of *Megamonas* observed in distressed individuals may be related to their sleep quality.

Conclusions One-third of the participants exhibited high stress/distress levels and almost half poor sleep. In this study, we identified several bacterial genera that are significantly altered in the gut microbiome of stressed individuals, with *Erysipelotrichaceae* UCG-003 being consistently increased across people with high stress and severe distress levels. *Erysipelotrichaceae* UCG-003 was previously reported to be elevated in individuals with sleep deprivation. Our findings also suggest the need to factor for stress and sleep quality when studying the gut microbiome of healthy individuals.

IDDF2021-ABS-0166 TRANSPLANTATION OF STOOL FROM OBESE PATIENTS PROMOTES INTESTINAL TUMORIGENESIS IN MICE

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Background Epidemiological evidence supports a relationship between adiposity and elevated risk of colorectal cancer (CRC). Nevertheless, the role of gut microbiota in mediating the carcinogenic effect of adiposity is unknown. In this study, we transplanted stool from obese patients to mice to evaluate its effects and mechanisms on intestinal carcinogenesis.

Methods Azoxymethane (AOM)-treated mice, *Apc*^{Min/+} and germ-free mice were gavaged with feces from obese patients and control subjects respectively. The colonic tumor load and number were recorded at endpoint. The gut microbiota composition was assessed by metagenomic sequencing and colonic transcriptome was assessed by total RNA sequencing.

Results The colonic tumor load and number were significantly higher in mice receiving feces from obese patients (OB), compared with those from control subjects (NB, normal BMI), in both AOM-induced and *Apc*^{Min/+} mice (IDDF2021-ABS-0166 Figure 1A-C). Histological assessments showed increased dysplasia proportions and Ki-67 positive cells (IDDF2021-ABS-0166 Figure 1D) in mice receiving feces from obese patients in both murine cancer models. Metagenomic analysis showed an altered microbiota composition (IDDF2021-ABS-0166 Figure 2A), with a lower alpha diversity (IDDF2021-ABS-0166 Figure 2B), decreased abundance of *Faecalibaculum*, *Bifidobacterium* and *Lactobacillus*, as well as increased abundance of *Akkermansia* in recipients of feces from obese patients (IDDF2021-ABS-0166 Figure 2C). Transcriptomic analysis on colon tissue showed upregulation of oncogenic (Wnt signaling pathway, MAPK signaling pathway and Rap1 signaling pathway) and pro-inflammatory pathways (TNF signaling pathway and chemokine signaling pathway). Mice received feces from obese patients showed impaired murine barrier function evidenced by decreased mucus layer thickness, loosened tight junction and over-expression of epithelial leaky protein Claudin-2 in the colonic crypt.

Conclusions Feces from obese patients promoted colorectal carcinogenesis in mice. Such action was associated with the

Abstract IDDF2021-ABS-0166 Figure 1

Abstract IDDF2021-ABS-0166 Figure 2

modulation of the gut microbiota composition, induction of oncogenic and pro-inflammatory factors and impairment of epithelial barrier. This study provides new insight into obesity-promoting colorectal carcinogenesis, at least in part by causing gut microbiome dysbiosis.

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ASSOCIATION OF COFFEE INTAKE WITH REDUCED CANCER-RELATED MORTALITY AMONG DIAGNOSED PATIENTS WITH COLORECTAL CANCER: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background Colorectal cancer (CRC) is the third most common cancer and the fourth most common cause of cancer mortality globally. Recent advances have in diagnostic and treatment options have reduced CRC-related mortality. Coffee consumption has been studied to decrease mortality in colon, liver and pancreatic cancer.