of Apc\textsuperscript{min/+} mice to determine the causality between HFD-induced dysbiosis and carcinogenesis.

**Results** Participants with HFD were more likely to develop AN, especially invasive carcinoma. The expression of MCP-1/CCR2 and CD163 in CRC patients with HFD was significantly higher. In the Apc\textsuperscript{min/+} mice, the total number of intestinal adenoma of HFD group was significantly increased. Pathological analysis confirmed intestinal carcinogenesis in small intestine and colon in HFD group, while there were only adenomas in the control group. This was accompanied by promoting tumor cell proliferation and decreasing apoptosis. Moreover, HFD administration altered gut microbiota, with increased opportunistic pathogens and decreased SCFA producing bacteria. The dysbiosis up-regulated the expression of MCP-1 and CCR2. And an increased M2 TAMs was observed in HFD group. And the effects of intestinal carcinogenesis can be restored by antibiotics cocktail. The transfer of fecal microbiota from HFD-fed mice also increased the tumor multiplicity and promoted carcinogenesis, while the MCP-1/CCR2 axis was also activated.

**Conclusions** HFD-induced dysbiosis promoted the intestinal carcinogenesis through activating the MCP-1/CCR2 axis.

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**HOMEBOXC6 PROMOTES METASTASIS OF COLORECTAL CANCER BY ACTIVATING WNT/B-CATENIN AND INDUCING EMT**

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**Background** HomeoboxC6 (HOXC6) belongs to the homeobox family, members of which encode a highly conserved family of transcription factors that play an important role in morphogenesis in all multicellular organisms. It was found to be upregulated in multiple-cancers, such as breast cancer, prostate cancer, liver cancer, colorectal cancer and so on. Upregulation of HOXC6 also contributed to poor prognosis in colorectal cancer. However, there were very few researches about HOXC6 in colorectal cancer and it was still unknown how it plays its role in tumor cell proliferation and metastasis in colorectal cancer.

**Methods**
1. Cell culture and HOXC6 overexpressed HCT116 cell line construction
   HCT116 was cultured with 1640 medium added with 10% FBS and 1% Penicillin-Streptomycin Solution with general culture condition.
2. Clinical patient tumor samples
   7 patients including 4 left-sided patients and 3 right-sided colorectal patients were collected for HOXC6 expression analysis compared to their para-tumor samples
3. Total protein and plasma & nuclear protein extraction and Western blot analysis
4. CCK8 and transwell with or without matrigel analysis
5. RNASeq and Co-IP analysis
6. Total mRNA extraction and qPCR analysis
7. Survival analysis of gene expression in UALCAN database

**Results**
1. HOXC6 was upregulated in right-sided colorectal cancer (figure 1A)
2. Upregulation of HOXC6 contributed to migration and invasion but not in proliferation (figure 1D, E)
3. Wnt/b-catenin and P53 pathway were activated by HOXC6 upregulation (figure 1F, G, H)
4. DKK1 was upregulated by HOXC6 and could combined to HOXC6 (figure 1I)
5. EMT was induced by HOXC6 upregulation (figure 1J)
6. Upregulation of HOXC6 contributed to poor prognosis in colorectal cancer (figure 1K).

Conclusions: HOXC6 could upregulate DKK1, Wnt/b-catenin pathways and induce EMT which contributed to metastasis in colorectal cancer. Further, we will explore the mechanisms of how upregulation of DKK1 could activate Wnt/b-catenin pathway.

EVALUATION OF BOWEL CANCER AWARENESS AMONG UNDERGRADUATE PHARMACY STUDENTS IN MALAYSIA AND PAKISTAN: A CROSS-SECTIONAL STUDY

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Background: As a pharmacist-wannabes, the final year undergraduate pharmacy students must have adequate foundational cancer knowledge and training to educate the public regarding the identification of warning signs and risk factors of bowel cancer. This study was conducted to assess and compare the levels of bowel cancer awareness among pharmacy students in Malaysia and Pakistan.

Methods: In this multi-centre cross-sectional study, a total of 174 final year undergraduate pharmacy students were enrolled from two Malaysian and two Pakistani universities. Post-approval from Cancer Research UK, a pre-validated Bowel Cancer Awareness Measure (CAM) was used. Bowel CAM consists of eight questions with a total of 26 items focusing on warning signs (10 items), delay in seeking medical help (1 item), bowel age (1 item), risk factors (11 items), bowel cancer screening programme (2 items), knowledge (1 item), age of first invitation (1 item), and confidence in detecting bowel symptom (1 item). The extracted data from the completed questionnaires were analysed descriptively and inferentially using the Statistical Package for the Social Sciences (SPSS(R)), version 22.

Results: The mean age of enrolled students was 22.78, SD=1.77, and the majority of them were females (n=107, 61.5%), and Malaysians (n=111, 63.8%). The enrolled students possessed high and moderate levels of awareness regarding bowel cancer warning signs (n=129, 74.1%), and risk factors (n=148, 85.1%), respectively. The findings of an independent-samples t-test suggested that there was a statistically significant difference in warning signs awareness score in Malaysian (M=24.13, SD=2.67), and Pakistani students [M=22.57, SD=3.43; t(172)=3.332, p=0.001]; whereas, this difference was insignificant for the scores of risk factors (M=32.59, SD=4.20), [M=31.30, SD=4.65; t(172)=1.875, p=0.062]. Moreover, a significant positive correlation was found between scores of warning signs and risk factors (r=0.364, p=0.001).

Conclusions: Malaysian students showed a better score for Bowel CAM. Specialised periodic training sessions can be helpful to enhance awareness among these future pharmacists.

INTRANUCLEAR MGP PROMOTES TUMOR PROGRESSION BY REGULATING JAK2/STAT5 PATHWAY AND INDICATES A POOR PROGNOSIS IN GASTRIC CANCER

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Background: Matrix Gla protein (MGP) has been reported as an extracellular matrix protein with dysregulation in various types of malignancies. However, the regulatory function of MGP inside the nucleus of gastric cancer (GC) cells remains largely unknown. In this context, we aimed to investigate the intranuclear molecular mechanisms of MGP contributing to GC development and progression.

Methods: MGP expression in GC was evaluated by immunohistochemistry and was further confirmed by two independent GEO datasets. Also, analysis of TCGA datasets illustrated its prognostic potential. Effects of MGP on GC cell proliferation, apoptosis, migration and invasion were all estimated. Luciferase reporter assays, immunoprecipitation, chromatin immunoprecipitation, et al were performed to figure out the underlying signaling pathways related to oncogenic effects of MGP.

Results: We demonstrated a higher expression level of MGP in GC compared to the adjacent normal tissues, leading to a worse prognosis of GC patients (figure 1. A-C). MGP can promote proliferation, migration and invasion of GC cells while inhibit apoptosis (figure 1. D-E) by interacting with phosphorylated-Stat5 inside the nuclear (figure 1. F-I) and consequently activating Jak2/Stat5 signaling pathway (figure 1. J-K). Rescue assays confirmed that Jak2/Stat5 pathway is critical for the protumorigenic characterization of MGP to facilitate GC cell proliferation and inhibit apoptosis.