depression were clearly and independently associated with the overall IBS-QOL score.

Conclusions Anxiety and depression were frequently observed in school going adolescent in Jaipur city IBS patients and were related to the severity of their symptoms and the impairment of the patient's QOL. Our data suggest that assessing anxiety and depression is important when evaluating IBS patients. There is a need for early and effective identification of anxiety, depression that can prevent many psychiatric disorders at their nascent stage with irritable bowel syndrome.

Basic Hepatology

**O-GlcNAcylation on Rab3A attenuates its effects on mitochondrial oxidative phosphorylation and metastasis in hepatocellular carcinoma**

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Background Metabolic reprogramming is widely observed in different cancers including Hepatocellular carcinoma (HCC). Reprogrammed metabolism results in hyper-O-GlcNAcylation in tumour cells, which can regulate transcription factors or tumour suppressors to modulate cancer metabolic reprogramming. Rab3A has been reported as an oncogenic factor in some cancers. However, the functions of Rab3A in HCC are never determined. Here we investigated the potential roles of Rab3A in HCC progression and determined how hyper-O-GlcNAcylation regulates the function of Rab3A.

Methods Western blot, qPCR, and immunohistochemistry assays were performed to quantify the relative expression of Rab3A in HCC. The functions of Rab3A in tumour progression were evaluated in HCC cell lines and nude mice. The interaction between Rab3A and OGT was determined by IP and GST pull-down. The GTP-binding affinity was observed in GTP-binding assays. The mitochondrial respiratory capacity was determined by XF cell Mito stress analysis, lactate assays, ROS-Glo H2O2 assays, and MitoSOX assays.

Results Both the mRNA and protein levels of Rab3A were elevated in HCC. However, decreasing Rab3A in HCC cells had no significant effects on tumour progression, and the upregulation of Rab3A in HCC patients conferred no correlations with metastasis or overall survival. We determined that Rab3A is modified with O-GlcNAcylation in HCC, which attenuated the Rab3A-mediated inhibition on HCC metastasis. Further analysis proved that Rab3A and its O-GlcNAcylation also played opposite roles in mitochondria oxidative phosphorylation (mtOXPHOS), and their functions on HCC metastasis partially depended on their effects on metabolic reprogramming.

Conclusions This study evaluated Rab3A as a tumour suppressor in HCC and revealed that the functions of Rab3A in both metastasis and metabolic reprogramming were attenuated by its modification of O-GlcNAcylation in HCC. Mechanistically, Rab3A elevated the expression of some mtOXPHOS-related genes, and O-GlcNAcylation decreased the GTP-binding affinity of Rab3A.

**CircScd1 promotes fatty liver disease via the Janus kinase 2/signal transducer and activator of transcription 5 pathway**

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Background Non-alcoholic fatty liver disease (NAFLD) is one of the most common liver diseases in affluent countries. Recent studies have reported that circular RNAs (circRNAs) are important regulators of hepatic steatosis. However, the role and mechanism of circRNA in NAFLD are poorly understood.

Methods Through NAFLD-related circRNA microarrays, we used real-time quantitative reverse transcription-polymerase chain reaction to screen circScd1 levels in control and test mice of mice fed a high-fat diet. RNA interference and overexpression plasmid vectors were used to manipulate the expression of circScd1, and the biological effects were evaluated by oil red staining, triglyceride detection, and western blot analysis.

Results CircScd1 expression was significantly lower in NAFLD tissues than in control tissues. Moreover, overexpression of circScd1 significantly inhibited the formation of lipid droplets. Western blot analyses showed that the protein levels of Janus kinase 2 (JAK2) and signal transducer and activator of transcription 5 (STAT5) were significantly increased. However, knockdown of circScd1 significantly promoted the degree of hepatocellular lipidosis and reduced the expression levels of JAK2 and STAT5.

Conclusions Aberrant expression of circScd1 affects the extent of hepatocellular lipidosis in NAFLD and promotes fatty liver disease via the JAK2/STAT5 pathway.

**Analysis of clinical signification KRAS and SCN5A gene expression in pancreatic cancer by TCGA datasets**

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Background To investigate the prognostic significance of KRAS (Kirsten rat sarcoma viral oncogene) and SCN5A Gene mutation in pancreatic carcinoma by TCGA (the cancer genome atlas) datasets.

Methods The TCGA dataset for liver cancer, gastric cancer, and pancreatic cancer in the TCGA official network, the top 100 genes affected by the number of people affected. Do Wayne map to get genes that do not coincide with the other two diseases in pancreatic cancer, and to find the genetic mutation that has a significant impact on the survival of pancreatic cancer.

Results There are 13 329 mutations in pancreatic cancer, and there are 16 348 and 19 288 mutant genes in the liver and gastric cancer, respectively. The first 100 mutations were analysed from 3 data sets, and the Wayne diagram showed that there are 25 mutations in the mutant gene of pancreatic cancer, the liver cancer and gastric cancer. There are respectively 3 and 11 genes for the coincidence of pancreatic cancer with