Conclusions The result of this study shows that most of GI symptoms due to GI cancer were in mild to moderate category and can be managed with prior knowledge of their occurrence. Managing GI symptoms will not ameliorate the disease process but certainly will improve the daily activities and add to the improved quality of life.

IDDF2018-ABS-0183 P53-INDUCED MIR-1249 SUPPRESSES TUMOUR PROGRESSION BY TARGETING VEGFA AND HMGA2 IN COLORECTAL CANCER

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Background MicroRNAs (miRNAs) are an important class of functional regulators involved in human cancers development, including colorectal cancer (CRC). Exploring aberrantly expressed miRNAs may provide us with new insights into the initiation and development of CRC by functioning as oncogenes or tumour suppressors. The aim of our study is to discover the expression pattern of miR-1249 in CRC and investigate its clinical significance as well as a biological role in CRC progression.

Methods QRT-PCR was used to detect the relative expression of miR-1249 in CRC tissues and cell lines. EdU, CCK-8, Wound healing, transwell and HUVECs tube formation assays were performed to assess the effect of miR-1249 on CRC cell proliferation, migration, invasion and angiogenesis *in vitro*, nude mouse xenograft, tail vein injection and chicken chorioallantoic membrane(CAM) model were used to observe CRC growth, metastasis and angiogenesis *in vivo*. Luciferase reporter assay, western blot, immunohistochemistry(IHC) and

immunofluorescence(IF) staining were fulfilled to explore the molecular mechanism of miR-1249 in CRC.

Results MiR-1249 was found to be markedly downregulated in CRC tissues and cell lines. The expression of miR-1249 was negatively related to pN stage, pM stage, TNM stage and overall survival(OS). Moreover, we found that miR-1249 was a direct transcriptional target of P53 and revealed that P53-induced miR-1249 inhibited tumour growth, metastasis and angiogenesis *in vitro* and *vivo*. Additionally, we verified that miR-1249 suppressed CRC proliferation and angiogenesis by targeting VEGFA as well as inhibited CRC metastasis by targeting VEGFA and HMGA2. Further studying showed that miR-1249 suppressed CRC cell proliferation, migration, invasion and angiogenesis via VEGFA-mediated Akt/mTOR pathway as well as inhibited EMT process of CRC cells by targeting VEGFA and HMGA2.

Conclusions Our study demonstrated that P53-induced miR-1249 might suppress CRC growth, metastasis and angiogenesis by targeting VEGFA and HMGA2, as well as regulate Akt/mTOR pathway and EMT process in the initiation and development of CRC. miR-1249 might be a novel the therapeutic candidate target in CRC treatment.

IDDF2018-ABS-0184 LNCRNA AGPG REGULATES ANABOLISM REMODELLING THROUGH AFFECTING PFKFB3 STABILITY IN ESCC

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Abstract IDDF2018-ABS-0183 Figure 1