Chromatin immunoprecipitation (ChIP) assay was used to screen and verify the GCLM target genes.

**Results** We found that glucose deprivation condition upregulated the expression of GCLM rather than hypoxia or H2O2. Compared to adjacent tumor tissues, the expression of GCLM was upregulated in CRC tumor tissues. The high expression of GCLM predicted poor prognosis in CRC patients. Inhibition of GCLM results in decreased proliferation rate, migration and invasion ability and sphere formation in HT-29 and DLD1 in vitro. Similarly, GCLM inhibition suppressed CRC tumorigenesis and metastasis in xenograft models and PDX models in vivo. The cancer cell stem cell markers were also downregulated after GCLM inhibition. Interestingly, we found that glucose starvation could rescue the anti-cancer phenotypes of GCLM inhibition and led to nucleus-translocation and accumulation of GCLM. ChIP assay showed GCLM could interact with octamer-binding transcription factor 4 (OCT4) promoter, a cancer stem cell marker, to increase its expression, and the target gene of OCT4 showed a positive correlation with GCLM expression in CRC patients.

**Conclusions** Our data showed that GCLM displayed nucleus accumulation in the condition of glucose deprivation which increased the transcription of OCT4. This study revealed an unorthodox oncogenic function of GCLM in colorectal carcinoma and suggested GCLM as a potential therapeutic target in CRC.

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**HSF2 PROMOTE THE MUCOSAL REPAIR IN ULCERATIVE COLITIS BY INHIBITING PRO-INFLAMMATORY CYTOKINE AND PROMOTING TGF-β EXPRESSION**

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**Background** In our previous study, we found that fecal heat shock transcription factor 2 (HSF2) concentration may be an evaluation index for predicting the mucosal healing of Ulcerative Colitis (UC). So this study is aiming to explore the mechanism of HSF2 in the mucosal repair of UC.

**Methods** The DAI, colon damage score and histopathology score were recorded in DSS induced colitis. The transcription and expression level of HSF2 and TGF-β in the mice colon tissue were detected by PCR and immunohistochemistry. HSF2 levels in HT-29 cells was manipulated by RNA interference and plasmids transfection. The concentrations of IL-1β, TNF-α and TGF-β in cells supernatant were detected by ELISA. The phosphorylation level of ERK, P38, JNK and Smad2/3 were detected by Western Blot.

**Results** The DAI, colon damage and pathology score of methylprednisolone(MP)treatment group were lower than DSS model group (figure 1). The transcription and expression of HSF2 and TGF-β in colon mucosal tissue were increased and decreased synchronously in DSS model group and MP treatment group(figure 2). The phosphorylation level of ERK, P38 and JNK increased significantly in HSF2 siRNA compared with the negative control group. The concentrations of IL-1β and TNF-α in the supernatant of HT-29 cells increased in HSF2 siRNA group. On the contrary, the phosphorylation level of ERK, P38 and JNK decreased significantly in HSF2-FLAG plasmid transfection compared with the Blank Vector group. The concentrations of IL-1β and TNF-α in the supernatant of HT-29 cells reduced (figure 3,figure 4).

The phosphorylation level of Smad2/3 decreased in HSF2 siRNA compared with the negative control group. The concentrations of TGF-β in the supernatant of HT-29 cells reduced in HSF2 siRNA group. On the contrary, the phosphorylation level of Smad2/3 increased significantly in HSF2-FLAG plasmid transfection compared with the Blank Vector group. The concentrations of TGF-β in the supernatant of HT-29 cells increased (Figure 4).

**Conclusions** HSF2 can promote the mucosal repair of ulcerative colitis by inhibiting the inflammatory response and promoting mucosal repair factor expression.