Abstract IDDF2019-ABS-0343 Figure 1

Conclusions MGP promotes proliferation and inhibits apoptosis of GC cells via Jak2/Stat5 pathway and may serve as a potential prognostic biomarker in GC patients.

Basic Hepatology

IDDF2019-ABS-0026

METADOXINE PREVENTS DIET-INDUCED NON-ALCOHOLIC STEATOHEPATITIS IN MICE

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Background Non-alcoholic fatty liver disease (NAFLD) is one of the most common chronic liver disorders worldwide. Metadoxine appears to be an effective strategy to manage alcoholic steatohepatitis. However, its role during non-alcoholic steatohepatitis (NASH) remains poorly defined. The study aimed to assess the therapeutic efficacy and mechanisms of metadoxine in NASH.

Methods Male C57BL/6J mice were randomly divided into three groups of six animals. The treatments were as follows: 1): Control group: standard diet. 2) NASH group: 42% fat 'high fat' diet (HFD) ad libitum for 16 weeks. 3) Metadoxine group: HFD and a single oral dose of metadoxine (200 mg/kg). Mice body weight, liver weight, fat mass was measured. Sera were collected for the analysis of biochemical markers and livers were obtained for further histological staining and

gene expression analysis. Transmission electron microscope (TEM) was used to observe the cell ultrastructure. The expression of inflammation genes, lipogenesis genes, and oxidative stress genes were assessed by real-time PCR and western blot.

Results After the dietary intervention, metadoxine decreased body weight and liver weight compared to the HFD group. Liver sections showed that HFD mice developed marked macro- and microvesicular steatosis, as well as multifocal necrosis compared to the controls. However, metadoxine treatment abolished steatosis. Less lipid droplets were

Abstract IDDF2019-ABS-0026 Figure 1

Abstract IDDF2019-ABS-0026 Figure 2

Abstract IDDF2019-ABS-0026 Figure 3

observed in the metadoxine treated animals in Oil Red O-stained sections. Metadoxine group showed lower serum concentrations of ALT, AST and TC, LDL-C than HFD mice. Moreover, we found mRNA levels of TNF- α , IL-1 β , NF- κ B were higher in HFD-fed mice than the control group. However, metadoxine treatment could decrease these genes and protein expression. In addition, metadoxine significantly increase the expression of lipogenesis genes (PPAR- α/γ , SREBP1c, FASN and ACO). Hepatic mRNA levels of oxidative stress genes were increased in HFD group, and metadoxine treatment further enhanced the expression of oxidative stress factors, such as NRF2, SOD1, NQO1. (figure 1,2,3)

Conclusions Our data established a therapeutic role of metadoxine in NASH. Metadoxine has a protective effect on NASH and its mechanism may be related to decrease the lipid accumulation, inhibit the oxidative stress and ultimately deduce inflammation.

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A META-ANALYSIS ON THE EFFECTIVENESS OF NANOPARTICLE-MEDIATED HYPERTHERMIA AGAINST HEPATOCELLULAR CARCINOMA

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Background Hepatocellular carcinoma (HCC) is one of the leading causes of cancer-related mortality worldwide. HCC treatment remains a challenge; wherein tumor size limits immediate surgical interventions. Moreover, the success of radiofrequency ablation-based treatment is likewise restricted by the number and size of tumor, operator dependent imaging techniques, and collateral damage to the normal cells surrounding the tumor. A promising experimental treatment modality for tumors that can potentially overcome some of these limitations is nanoparticle - mediated hyperthermia (NMH). Currently at the developmental stage, NMH usually utilizes gold or iron oxide nanoparticles to convert the radio-frequency or alternating magnetic field into heat that causes targeted cell death. This meta-analysis therefore aims to assess