Protective effects against and potential mechanisms underlying the effect of magnesium isoglycyrrhizinate in hypoxia-reoxygenation injury in rat liver cells

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ABSTRACT. We examined the protective effects of magnesium isoglycyrrhizinate (MgIG) on hypoxia-reoxygenation injury in rat liver cells. Rat liver cells in the logarithmic growth phase were divided into the hypoxia-reoxygenation injury model group and MgIG pretreatment group (0.01, 0.1, 1, 10, 100 mg/mL). After 24-h pretreatment, we detected the effects of MgIG on liver cell viability using the methyl thiazolyl tetrazolium (MTT) assay at 6-h hypoxia and 4-h reoxygenation. After 24-h pretreatment, liver cells were randomly divided into the hypoxia-reoxygenation injury model group and low-, moderate-, and high-MgIG-concentration groups (0.1, 1, 10 mg/mL, respectively), and hypoxia and reoxygenation were simulated for 6 and 4 h, respectively. Cell morphology was observed by light microscopy. Nuclear factor-kB gene expression was analyzed by quantitative reverse transcription-polymerase chain reaction. MTT results showed that MgIG (0.1, 1, 10 mg/mL) improved the A-value of anoxia-reoxygenation injury in liver cells ($P < 0.01$) compared with that of the model group. Cells did not survive when the MgIG concentration was 100 mg/mL. At an MgIG concentration

lower than 0.01 mg/mL, the A-value of the MTT group was higher than that of the model group (P > 0.05). Nuclear factor-kB mRNA expression (0.597 ± 0.062, 0.248 ± 0.067, 0.141 ± 0.029) in the low-, moderate-, and high-concentration groups was lower than that in the model group (P < 0.01). MgIG reduced hypoxia-reoxygenation injury of liver cells, indicating that it improved hepatic cell activity, inhibited lipid peroxidation and inflammatory reactions, and decreased nuclear factor-kB mRNA expression.

Key words: Hypoxia; Liver cell; Magnesium isoglycyrrhizinate; Reoxygenation

