GW23-e0182

GINSENOSIDE RE ENHANCES THE SURVIVAL OF H9C2 CARDIAC MUSCLE CELLS THROUGH REGULATION OF AUTOPHAGY

doi:10.1136/heartjnl-2012-302920a.131

Zi-Long Zhang, Yan Fan, Mei-Lin Liu. *Department of Geriatric, Peking University First Hospital*

Objectives To examine the effect of ginsenoside Re (G-Re) on autophagy in H9c2 cardiomyocyte cells under starvation.

Methods H9c2 cells were cultured in glucose-free and serum-free medium for several hours as the glucose deprivation (GD) stimulation. To measure the amount of autophagosome formation, we determined the membrane-bound form of autophagy-related protein LC3B-2 by immunoblotting. To evaluate the situation of autophagy flux, we added 10 nmol/l bafilomycin A_1 (BafA1) into the medium to block the fusion processes between autophagosomes and lysosomes. H9c2 cells under GD were treated with 100 μmol/l G-Re. LC3B-2 measurement and immunofluorescence were conducted to display the effect of G-Re on autophagy in these cells. Cell viability, ATP content, malondialdehyde level and superoxide dismutase activity in cultured medium were determined to appreciate the physiological relevance of autophagy changes due to G-Re treatment. We also assayed phosphorylated AMPKα and mTOR to explore the mechanisms underlying the effect of G-Re on autophagy in cells under GD.

Results In H9c2 cells under GD, LC3B-2 increased in a time-dependent manner in association with the decrease of cell viability and cellular ATP content. In H9c2 cell under GD and treated with 100 μmol/l G-Re, LC3B-2 expression decreased, accompanied by the rescue of cell death, the increase of cellular ATP content, and the relief of oxidative stress. The higher p-AMPKα in H9c2 cell under GD decreased when they were treated with 100 μmol/l G-Re, probably relating to the mechanisms underlying the inhibition of autophagosomal formation by G-Re.

Conclusions Starvation induced autophagy in H9c2 cells and led to cell injury. Treatment of 100 μmol/l G-Re inhibited autophagosomal formation in the cells, which may be beneficial to the cardiomyocytes under starvation.