**GINSENOSIDE RE ENHANCES THE SURVIVAL OF H9c2 CARDIAC MUSCLE CELLS THROUGH REGULATION OF AUTOPHAGY**

Z-L. Zhang, Y. Fan, **M-L. Liu**

Dept. ofGeriatric, Peking University First Hospital,Beijing, China

Aim: To examine ginsenoside Re (G-Re) on autophagy in H9c2 cardiomyocyte cells under starvation.

Methods:H9c2 cells were cultured in glucose-free medium as the glucose deprivation (GD) stimulation. To measure autophagosome formation, we determined autophagy-related protein LC3B-2 by immunoblotting. To evaluate the situation of autophagy flux, we added 10 nmol/L bafilomycin A1 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 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 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. 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 treated with 100 μmol/L G-Re, probably relating to the mechanisms underlying the inhibition of autophagosomal formation by G-Re.

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