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Rg3 inhibits puromycin induced apoptosis in human podocyte through suppression of ER stress.

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Objectives : Podocyte is a key player in glomerular disease. Recent studies have shown that podocyte endoplasmic reticulum (ER) stress leads to severe proteinuria. However there is little information about therapeutic strategies to overcome ER stress in podocyte. In this study, we investigated the effect of Ginsenoside Rg3 on puromycin induced apoptosis and ER stress in podocytes.

Methods : We used human podocyte cell lines. RG3 toxicity and therapeutic concentrations was examined by XTT test. Puromycin induced podocyte injury is known as a cellular stress model of focal segmental glomerular sclerosis. We stimulated podocyte with puromycin and induced apoptosis in this cell. The protein expression of ER stress markers such as glucose-regulated protein 78 (GRP78) and C/EBP homologous protein (CHOP) were evaluated by western blot.

Results : In human podocyte, puromycin increased apoptotic protein, cleaved PARP in a dose-depend manner at 48 hours. ER stress marker, C/EBP homologous protein (CHOP) was also increased by puromycin stimulation. Rg3 pretreatment prevented puromycin induced apoptosis and decreased expression of CHOP on western blotting.

Conclusions : Our results suggest Ginsenoside Rg3 prevents puromycin induced apoptosis in human podocyte through suppression of CHOP pathway. Rg3 might be a potential therapeutic option for glomerular disease

Keywords : Podocyte, ER stress