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Vascular endothelial growth factor-A inhibits tumicamycin-induced endoplasmic reticulum stress and apoptosis in HK-2 cells

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Objectives: Vascular endothelial growth factor A (VEGF-A) is a potent angiogenic factor and this angiogenic factor induces proliferation and migration of vascular endothelial cells. It has been also demonstrated that VEGF is a renal tubular epithelial survival factor. The endoplasmic-reticulum (ER**)** stress response constitutes a cellular process that is initiated by many conditions that disturb folding of proteins in the ER. ER stress is involved in the pathogenesis of many diseases. However, there is no report about effect of VEGF-A on ER stress in kidney cells. The purpose of this study was to clarify the effect of VEGF-A and apoptosis in HK-2 cells under endoplasmic reticulum (ER) stress.

Methods: HK2 cells were treated with tunicamycin (TM) for 6 hours with or without VEGF-A (30 ng/mL). Apoptosis and ER stress expression were evaluated by Western blot analyses.

Results: ER stress markers, including glucose-regulated protein 78 (GRP78), CCAAT/enhancer-binding protein homologous protein (CHOP) was significantly upregulated at 6 hours after TM stimulation. VEGF-A decreased TM-induced expression of GRP78 and CHOP expression at same time. TM-induced protein kinase RNA-like ER kinase (PERK) expression was decreased at 6 hours by VEGF-A treatment. Furthermore, TM-induced cleaved-caspase-3 expression of HK-2 cells was suppressed by VEGF-A administration.

Conclusions: All of these results suggest that VEGF-A ameliorates TM-induced ER stress by regulating the expression of GRP78, CHOP and PERK and TU-inducde apoptosis.