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Methylglyoxal-induced apoptosis is dependent on the generation of reactive oxygen species through the down-regulation of c-FLIPL expression in HUVECs

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Methylglyoxal (MGO) is a reactive dicarbonyl metabolite of glucose and its levels are elevated in diabetic patient's plasma. Some studies have demonstrated that MGO combined with amino and sulfhydryl groups of proteins to form stable advanced glycation end products (AGE), which was associated with vascular endothelial cells (ECs) injury and was responsible for atherosclerosis. In the present study, we found that MGO induced apoptosis in a dose-dependent manner in HUVECs, which was attenuated by pretreatment with z-VAD, pancaspase inhibitor. Treatments with MGO dose-dependently down-regulated c-FLIPL expression. MGO-induced increase in ROS levels preceded the down-regulation of c-FLIPL, which was abrogated by the ROS scavengers NAC, indicating that MGO acts through ROS generation to repress c-FLIPL expression. The forced expression of c-FLIPL attenuated the MGO-mediated apoptosis in HUVECs. We also confirmed that MGO induced apoptosis in endothelium derived from mice. Collectively, this study demonstrates that MGO-induced apoptosis is dependent on the generation of ROS through the down-regulation of c-FLIP expression via repressing NF–κB pathways in HUVECs.

Biography

Tae Jin Lee is an Associate Professor of Yeungnam University, Department of Anatomy, College of Medicine from 2008. He obtained his PhD degree from the Department of Microbiology of Kyungpook National University, South Korea in 2000. He had a Post-doctoral Training at Louisiana State University Health Sciences Center, Stanley S Scott Cancer Center. After relocation to Korea in 2005, he worked on mechanisms of novel TRAIL sensitizers, and role of c-FLIP in apoptotic pathways. Currently, he studies on the molecular mechanisms underlying regulating apoptosis-related genes by miRNA in cancer and human umbilical vein endothelial cells (HUVECs).

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