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## Methylglyoxal stimulates autophagy in vascular endothelial cells through regulating the PI3K/Akt/mTOR and endoplasmic reticulum stress/MAPKs signaling pathways

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Methylglyoxal (MGO) is formed naturally in our body as byproduct of glycolysis from the degradation of glyceraldehyde-3-phosphate. One is as a glycation agent and another as a molecule with some beneficial functions. Long term diabetic hyperglycemia leads to high serum MGO levels in our body which can then contribute to Advanced Glycation End Products (AGEs). On the other side as having a beneficial function, MGO might induce autophagy and apoptosis to clear up or recycle damaged cells. Autophagy is essential in cell development, differentiation, repair, immunology and maintenance of proteins. Therefore, MGO-induced autophagy and apoptosis agent expression in human aortic endothelial cells (HAoEC) have been hypothesized. We investigated autophagy related expression of LC3-1/LC3-II, PI3K/Akt/mTOR, ER stress and MAPKs signaling pathway. Also, to check MGO effect on apoptosis, Bcl-2 (Anti-apoptotic), Bax (Pro-apoptotic) and cleaved caspase-3 expression levels were experimented. To investigate the autophagic vacuoles and to monitor autophagic flux in fixing cells, Cyto-ID<sup>®</sup> autophagy detection kit and confocal microscope was used. We have found that MGO effectively induced autophagy and apoptosis by inhibiting PI3K/Akt/mTOR signaling pathway and by activation of LC3 via ER stress and MAPKs signaling pathway. In conclusion, MGO-induced autophagy and type-II autophagic cell death (autosis) may effectively treat cardiovascular diseases with vascular endothelial cells via inhibition of PI3K/Akt/mTOR signaling pathway and ER stress/MAPKs signaling pathway.

### Biography

Jae Hyuk Lee has completed his MS degree in the Department of Pharmacognosy and Pharmacology at Gachon University and is currently a PhD student. He has been investigating the underlying mechanism of the development of diabetes complications.

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