

12th International Conference on

ENDOCRINOLOGY, DIABETES AND METABOLISM

October 01-02, 2018 Osaka, Japan

Effect of liquiritigenin on apoptotic beta-cell death by palmitate-induced lipotoxicity in *INS-1* cellsGong Deuk Bae¹, Hee-Sook Jun¹, Yoon Sin Oh²¹Lee Gil Ya Cancer and Diabetes Institute, Department of Molecular Medicine, Incheon, Republic of Korea²Department of Food and Nutrition, Eulji University, Seongnam, Republic of Korea

Objective: Activation of estrogen receptor signaling plays an important role to preserve functional beta-cell mass in treatment of diabetes. Liquiritigenin (LQ), a flavonoid isolated from *Glycyrrhiza uralensis*, is an estrogenic compound which acts as an agonist for the estrogen receptor β . In this study, we investigated protective effect of LQ on palmitate (PA)-induced apoptosis in *INS-1* cells.

Methods: To examine effect of LQ on beta cells, glucose stimulated insulin secretion (GSIS) by enzyme immunoassay (EIA) method and cell viability by MTT were measured in rat beta-cell line *INS-1* cells. To induce lipotoxicity, PA (400 μ M) was treated for 24 h and amount of apoptotic cells were analyzed using a flow cytometer with annexin-V staining. Expression level of apoptotic proteins and endoplasmic reticulum (ER) stress markers were analyzed by western blot analysis after LQ treatment. Tunicamycin and thapsigargin were used to ER stress inducer and AKT inhibitor (*AKTi-1/2*) was used to inhibit LQ-induced AKT phosphorylation at ser 473.

Results: Exposure of *INS-1* cells to 5 μ M of LQ significantly increased GSIS as well as cell viability. PA treatment increased annexin-V stained cells and apoptotic proteins such as cleaved caspase-3, cleaved poly (ADP-ribose) polymerase and bax, but these increases were significantly inhibited by LQ treatment. LQ treatment inhibited cell death by ER stress inducers and PA induced ER stress marker proteins such as CHOP and phosphorylated forms of PERK and eIF2 α was also significantly downregulated in LQ treated cells. LQ phosphorylated AKT at ser 473 via estrogen receptor element dependent pathway and blocking AKT signaling inhibited LQ induced decrease in level of phosphorylated PERK, consequently cell viability was not recovered.

Conclusion: Our data demonstrated that LQ has anti-apoptotic effect against PA induced lipotoxicity and AKT mediated ER stress inhibition was involved in the anti-apoptotic effect of LQ.

Biography

Gong Deuk Bae is a PhD course student in Gachon university. His research topic is The effect of natural products on the prevention and treatment of type 2 diabetes focusing on pancreatic beta-cells.

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