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Effect of liquiritigenin on apoptotic beta-cell death by palmitate-induced lipotoxicity in *INS-1* cells

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

Methods: To examine e ect 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 μM) was treated for 24 h and amount of apoptotic cells were analyzed using a ow cytometer with annexin-V staining. Expression level of apoptotic proteins and endoplasmic reticulum (ER) stress markers were analyzed by western blot analysis a er LQ treatment. Tunicamycin and thapsigargin were used to ER stress inducer and AKT inhibitor (*AK -1/2*) was used to inhibit LQ-induced AKT phosphorylation at ser 473.

Results: Exposure of *I -1* to 5 μM of LQ signi cantly 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 signi cantly inhibited by LQ treatment. LQ treatment inhibited cell death by ER stress inducers and

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