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Investigation into the interplay between incretin hormones (GLP-1 and GIP), glucose metabolism and sepsis

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Introduction: Incretin hormones include glucagon like peptide-1 (GLP-1) and glucose dependent insulinotropic polypeptide also called gastric inhibitory polypeptide (GIP). Both of them are gut hormones, GLP-1 secreted from L-cells which are located in the proximal part of caecum, while GIP is secreted from K cells, located mainly in the duodenum and jejunum. GLP-1 and GIP are produced in response to nutrient ingestion particularly glucose and some amino acids and fatty acids.

Aim: To confirm the presence of incretin hormone receptors (GLP-1R, GIPR) and insulin receptor (INSR) in hepatocytes and monocytes cell lines, and to study whether hyperglycaemia in sepsis results from the effects of endotoxin on glucose-modulating hormone receptors (incretins and insulin receptors), in addition to insulin resistance.

Materials & Methods: U937, a monocyte cell line and HUH7, hepatocyte cell line were cultured in Dulbecco's Modified Eagle Medium (DMEM) (Sigma) containing 10% fetal calf serum (FCS). Both U937 and HUH7 were cultured with different lipopolysaccharide (LPS) (Sigma) concentrations (0.2, 1 and 5 μg/ml) for 24 hr to simulate sepsis. From cells that have been cultured as indicated above, mRNA was obtained using TRI Reagent and revere transcribed by using Superscript II to synthesize the cDNA. Real-time RT-PCR quantitation of gene expression was used to compare the rates for relative expression (using β-actin) as a reference gene. This technique was performed and screened using iCycleriQ.

Results: There is a direct correlation between the LPS doses and expression of mRNA for GIPR, INSR in U937, which there was a significant decrease in expression of GIPR and INSR at 1 μg and 5 μg/ml LPS. No GLP-1R expression was detected in U937 cells. Results obtained from HUH7 cells indicated for the first time that GLP-1R and GIPR mRNA was expressed on HUH7 cells. Moreover, LPS stimulated significant decrease in expression of GLP-1R, GIPR and INSR at different LPS doses.

Conclusion: This study has indicated that sepsis can affect the expression of incretins and insulin receptors in a monocytes and hepatocytes.

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