Glucose-induced membrane fluidification in endocrine pancreatic cells.

M. Deleers, J.M. Ruysschaert and W.J. Malaisse

Laboratories of Experimental Medicine and Physical Chemistry of Macromolecules, Brussels University, Brussels, Belgium.

The process of glucose-induced insulin release coincides with changes in a number of biochemical and biophysical variables in the pancreatic B-cell (1). We have now investigated the influence of glucose upon membrane viscosity in isolated endocrine pancreatic cells.

Isolated islets obtained by collagenase digestion of pancreases removed from fed albino rats were disrupted into isolated cells by mechanical agitation in a medium deprived of Ca²+ and containing collagenase. The isolated cells were preincubated for 240 min in a culture medium containing glucose (8.3 mM) and then exposed, in the absence of glucose, to the lipophilic fluorescent probe 1,6-diphenyl-1,3,5-hexatriene(DPH). After about 20-30 min, when the fluorescence intensity had reached a close-to-equilibrium value, the cell suspension was examined for fluorescence polarization in an Elscint MVIa microviscosimeter (2). The glucose concentration of medium was increased in a step-wise manner from zero to 5.6, 11.1 and 16.7 mM.

Under steady-state conditions of exposure to glucose, the degree of fluorescence polarization at a temperature of 37°C was calculated from the records obtained, at each glucose concentration, by a progressive change in temperature from 30 to 38°C over a period of approximately 10 min. The basal viscosity measured in the absence of glucose averaged 2,010 \pm 120 mP. By paired comparison, the viscosity was decreased below basal value by 67 \pm 20, 192 \pm 47 and 301 \pm 50 mP (n= 5 to 10 pairs; P < 0.02 or less) in the presence of glucose 5.6, 11.1 and 16.7 mM, respectively. At a fixed temperature (37°C) a glucose-induced decrease in fluorescence polarization was detected within 1 min after increasing the glucose concentration of the incubation medium.

In conclusion, glucose augments membrane fluidity in isolated endocrine pancreatic cells. The dose-action relationship and time course for such an effect is well suited to play a role in the functional response of the cells to glucose stimulation.

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