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Mechanism of Action of Pancreozymin and Acetylcholine on Pancreatic Acinar Cells

In a number of secretory tissues, such as the adrenal medulla[1], the pancreatic islets[2], the pancreatic acinar cells[3] and the salivary acinar cells[4], the first step in the activation of the secretory process caused by the physiological stimulant has been suggested to be an increase in membrane permeability resulting in Na^+ and/or Ca^{2+} influx. Only in the salivary acinar cells, however, has it been demonstrated directly that the effective membrane resistance decreases during stimulation[5] and it has been reported that acetylcholine (ACh) and pancreozymin markedly increase the membrane resistance in rat pancreatic acinar cells[6].

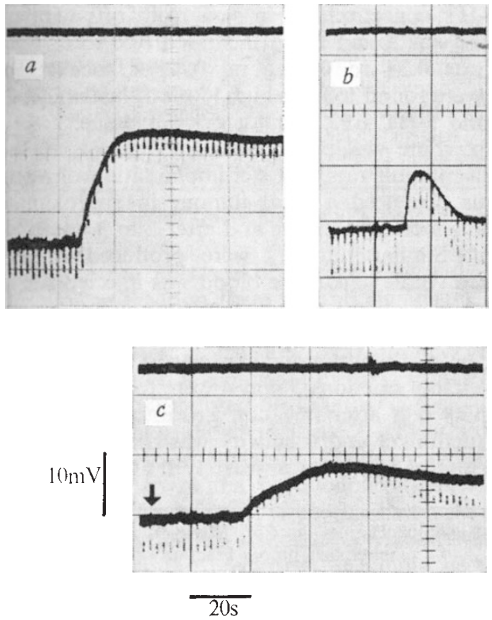

Fig. 1 Effect of ACh and pancreozymin on membrane potential and resistance. The distance between the two heavy lines corresponds to the membrane potential. The short vertical bars are membrane potential displacements caused by hyperpolarizing current pulses of constant strength (10^{-9} A) and 70 ms duration sent through the recording electrode. The arrows indicate addition of stimulant to the tissue bath. The volume of the bath was 25 ml and the flow of Krebs-Henseleit solution through the bath was 6 ml min^{-1}. In (*a*) 100 μg ACh was given. In (*b*) 10 μg ACh was added to the bath. In (*c*) 4 units (Crick) of pancreozymin (Sigma) was given.

Membrane potentials were measured in pancreatic acinar cells from mice with the help of K-citrate filled microelectrodes (50–90 MΩ) as previously described[3,7]. The resistance between the cell interior and the external medium was determined by applying hyper or depolarizing current pulses (10^{-9} A, 70 ms) through the recording micro-electrode, the tip resistance of which had been carefully balanced out by a bridge arrangement. (W-P, M4 microprobe system), and measuring the resulting change in membrane potential (Fig. 1). In all three tracings it is seen that addition of stimulant (ACh or pancreozymin) to the tissue bath resulted in depolarization and a concomitant fall in membrane resistance. In tracing *b* it can be seen that the effect on both potential and resistance was reversible. In the unstimulated state the mean value of the membrane resistance was 5.0 MΩ ± 0.3 ($n = 19$). The membrane resistance during stimulation with ACh at the time of maximal depolarization was 2.4 MΩ ± 0.3 ($n = 19$). This value is significantly ($P < 0.001$) lower than the resting value. Similarly the effect of pancreozymin was to reduce membrane resistance significantly ($P < 0.01$) from 4.6 MΩ ± 0.2 ($n = 5$) to 2.0 MΩ ± 0.5 ($n = 5$).

These results disagree with the recent report on rat pancreas[6] stating that stimulation causes a marked increase in membrane resistance (from about 50 MΩ to more than 100 MΩ). My results seem more reliable, however, as they are based on changes actually measured in individual cells in contrast to the statistical multiple impalement type of approach used in the investigation of Kanno[6]. That ACh and pancreozymin cause a marked fall in membrane resistance is consistent with the general concept of stimulus-secretion coupling[1] and with the finding that the acetylcholine-induced depolarization in pancreatic acinar cells varies with the extracellular Na^+ concentration[3].

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Basophilic Leukaemia in the Albino Rat and a Demonstration of the Basopoietin

Mast cell tumours in mice arising in subcutaneous tissues have been described which were not associated with a leukaemic blood picture and did not show symptoms due to the release of heparin, histamine, or 5-hydroxytryptamine (5-HT)[1,2].

The potent leukaemogenic properties of a β-chlorethylamine [2-(α-chlor-β-isopropylamine)ethylnaphthalene hydrochloride] were shown when 90% of Wistar rats fed a diet containing 0.1% of this compound developed a myeloid or lymphatic leukaemia[3]. Each type of leukaemia can be successively transplanted into newborn rats but not into adult rats.