

P022 Changes of $[Ca^{2+}]_c$ in pancreatic mouse α -cells induce oscillatory translocations of PKC β II to the plasma membrane
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The role of protein kinase C (PKC) isoforms in controlling glucagon release is poorly understood. Here we assess the effect of regulators of glucagon secretion on the intracellular localization of PKC β II, a conventional PKC isoform stimulated by diacylglycerol and Ca^{2+} . After dispersal of mouse islets, α -cells were localized by adenovirus-mediated expression of red fluorescent protein (mRFP) under the control of the preproglucagon promoter. Cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$) was imaged with Fura2 and the distribution of PKC β II by total internal reflection fluorescence microscopy of adenovirally-expressed enhanced green fluorescent protein (EGFP)-tagged PKC β II. Large increases in $[Ca^{2+}]_c$ induced with 30mM K^+ , 10mM arginine, 5 μ M adrenaline or 100 μ M acetylcholine were closely followed by PKC β II translocation to the plasma membrane. Oscillations in $[Ca^{2+}]_c$ at low glucose concentrations (0.5mM) were either suppressed or decreased in frequency as glucose concentrations increased. No or few oscillatory translocations of PKC β II.EGFP to the cell surface were apparent at 10mM glucose, whereas a decrease in glucose concentration to 0.5mM induced either a stable or oscillatory increase in plasma membrane association. Oscillations in $[Ca^{2+}]_c$ induced by a decrease in glucose concentration or by receptor-coupled agonists are thus decoded as pulsatile translocations of PKC β II to the plasma membrane, which may be involved in stimulating glucagon release.