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Poster Sessions

Involvement of MARCKS in amylase release in GLP-1-stimulated pancreatic acini

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It is well known that glucagon-like peptide 1 (GLP-1) can bind to the GLP-1 receptor of pancreatic islet to enhance insulin secretion through a cAMP-dependent pathway. However, little is known about the effects of GLP-1 on the pancreatic exocrine gland. In the gland, a signal transduction of amylase release is evoked mainly by an increase in intracellular Ca^{2+} levels and activation of PKC. Myristoylated alanine-rich C kinase substrate (MARCKS) is known as a major substrate for PKC. We previously demonstrated that MARCKS is involved in pancreatic amylase release through the Ca^{2+} -dependent pathway. Here, we studied the effects of GLP-1 on MARCKS phosphorylation and amylase release through the cAMP-dependent pathway in rat pancreatic acini. By the organ bath technique, GLP-1 did not induce amylase release in the intact pancreas. In contrast, it induced amylase release and MARCKS phosphorylation in isolated pancreatic acini. An inhibitor of PKA suppressed those effects. Furthermore, a MARCKS-related peptide inhibited the GLP-1-induced amylase release. These findings suggest that GLP-1 induces amylase release through MARCKS phosphorylation via activation of PKA in the isolated acini, but not in the intact pancreas.