Heminic receptor in situ suggests the pancreatic acini have two binding sites, whereas there is only a lower affinity binding site for the muscarinic receptor, recognized only by I-125-QNB, in addition to the high affinity binding site recognized by both I-125-QNB and H-3-NMS.

The analysis of muscarinic receptors on rat pancreatic acini was carried out using I-125-quinochloridine-n-yl benzilate (QNB) and H-3-N-methyl scopolamine (NMS). I-125-QNB and H-3-NMS were used to analyse the muscarinic receptors on dispersed pancreatic acini. The binding of both tracers to rat pancreatic acini reached a maximum after 30 min of incubation at 37°C and were specific and reversible. The binding of H-3-NMS to rat pancreatic acini was completely inhibited by various muscarinic receptor agonists and antagonists. On the other hand, the binding of I-125-QNB to rat pancreatic acini was completely inhibited by QNB, atropine, and muscarine, but only partially inhibited by acetylcholine and carbacol. According to Scatchard analysis of the binding inhibition studies, the binding of NMS to rat pancreatic acini indicated a single binding site, whereas there appeared to be at least two binding sites for QNB. These results suggest the possibility that there is a lower affinity binding site for the muscarinic receptor, recognized only by I-125-QNB, in addition to the high affinity binding site recognized by both I-125-QNB and H-3-NMS.

Clinical study of reconstructive technique in stomach cancer using biliary tract scintigram.

To characterize CCK receptors on rat pancreatic acinar cell and its membrane fraction, we prepared I-125-Bolton Hunter reagent and radioiodinated porcine CCK-33. Specific activity of I-125-BH-CCK was 169 μCi/μg. I-125-BH-CCK stored at -20°C was stable until 5 weeks. The specific binding of I-125-BH-CCK to acinar cell at 37°C was rapid and reversible, being half-maximal at 8 min and maximal at 45 min. I-125-BH-CCK binding to acinar cell and its membrane fraction were competitively inhibited by increasing concentration of CCK-8 but not by insulin and VIP. On acinar cell, scatchard analysis of binding was compatible with two classes of binding sites: a high affinity site (Kd=74.7pM) and a lower affinity site (Kd=6.8nM). On membrane fraction, however, only a lower affinity site was detected (Kd=2.1nM).