



HOKKAIDO UNIVERSITY

Title	STUDIES ON THE ACETYLCHOLINE-INDUCED EXOCRINE RESPONSES IN THE ISOLATED AND PERFUSED RAT PANCREAS
Author(s)	HABARA, Yoshiaki
Citation	Japanese Journal of Veterinary Research, 26(1-2), 26-26
Issue Date	1978-04
Doc URL	https://hdl.handle.net/2115/2130
Type	departmental bulletin paper
File Information	KJ00003407844.pdf



**STUDIES ON THE ACETYLCHOLINE-INDUCED
EXOCRINE RESPONSES IN THE ISOLATED
AND PERFUSED RAT PANCREAS**

Yoshiaki HABARA

*Department of Veterinary Physiology
Faculty of Veterinary Medicine
Hokkaido University, Sapporo 060, Japan*

Continuous stimulation with acetylcholine (ACh, 3×10^{-7} M) induced 3 phases of amylase release: transient maximum release in the first phase; continuous release in the second phase lasting about 40 min; and declining release in the third phase. These 3 phases of amylase release were uniformly nullified when 5×10^{-6} M atropine was added to the perfusing solution 10 min prior to the initiation of continuous ACh stimulation. In a Ca-deficient environment, the amount of amylase release was inhibited, and the release decayed rapidly. The ACh-induced amylase release was little affected by the removal of HCO_3^- . The amount of amylase release rose when the concentration of ACh was increased. A dose-response relation was found between the amount of amylase release and the concentration of ACh over the range from 10^{-8} M to 3×10^{-7} M. The addition of a low dose of cholecystinin-pancreozymin (CCK-PZ, 1.0 m-U/ml) shifted the dose-response relation to the left. The addition of a low dose of secretin (Sc, 1.0 m-U/ml) also shifted the dose-response relation to the left. The increase in amylase release during continuous stimulation with ACh was usually accompanied by a concomitant increase in the pancreatic juice flow. These results were analysed using Michaelis-Menten kinetics. These results may be explained by postulating a receptor complex composed of receptor subunits and an ionophore subunit. The receptor subunits may be activated by either one of these secretagogues, and, in turn, may activate the ionophore subunit.