

VASOACTIVE INTESTINAL PEPTIDE (VIP) STIMULATION OF CHLORIDE SECRETION
IN SHARK RECTAL GLAND (*SQUALUS ACANTHIAS*) IS NOT INHIBITED BY
CYTOCHALASIN D AND PROCAINE.

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Cytochalasin D, which disrupts the formation of actin filaments, markedly inhibits the secretion of chloride by the perfused rectal gland, when the gland is stimulated by C-type natriuretic peptide (CNP). Stimulation by VIP is not inhibited by cytochalasin. Procaine (10^{-2} M) prevents the release of VIP from intrinsic nerves when CNP is given, and in the presence of procaine the inhibition of CNP stimulated chloride secretion by cytochalasin is virtually complete. Procaine (10^{-2} M) does not prevent the stimulatory effect of VIP. The present experiments were undertaken to see if procaine itself might confer susceptibility to inhibition by cytochalasin, in a way unrelated to the known effect of procaine to block neurotransmitter release. To examine this we stimulated chloride secretion with VIP in the presence of procaine, which should have no effect on VIP stimulation, and added cytochalasin D to see if the latter inhibited the effect of VIP in the presence of procaine.

The rectal gland of spiny dogfish sharks (*Squalus acanthias*) were perfused as described by Silva et al. (Methods in Enzymol. 192:754-66, 1990). In all experiments with cytochalasin the glands were perfused with procaine 10^{-2} M. After three initial periods of 10 minutes each, the secretion of chloride was stimulated with VIP (Sigma V3628) given as a bolus over one minute at the dose calculated to expose the gland to a concentration of 10^{-7} M VIP. In six experiments cytochalasin D 10^{-6} M was added to the perfusate at the beginning of the experiment; in 7 experiments cytochalasin was omitted. Collections were continued at 10 minute intervals for the next 30 minutes. The volume secreted by the rectal gland in each period was measured by weighing and its chloride concentration was measured by amperometric titration using a Buchler-Cotlove chloridometer.

The peak chloride excretion stimulated by VIP in the presence of procaine and cytochalasin D was 1321 ± 358 (mean \pm SEM, $n=6$) μ Eq/h/g, and the increment produced by VIP was 1167 ± 385 . These values are not significantly different from the stimulation by VIP in glands perfused with procaine but without cytochalasin D (1008 ± 149 , peak, and 856 ± 162 , increment, $n=7$). Stimulation of chloride secretion by VIP in the presence of procaine was comparable to that measured in 33 additional experiments in which the same dose of VIP was administered in the same way, but no procaine was added to the perfusate (1085 ± 459).

These results bolster the validity of the use of procaine to block VIP release by perfused rectal glands in order to investigate the diverse pathways leading to secretion.

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