IS THE SHARK (*SQUALUS ACANTHIAS*) RECTAL GLAND CONTRACTILE?

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The importance of the rectal gland in shark osmoregulation is clear: it secretes a plasma-hypertonic fluid that plays a major, if not vital (e.g., Evans, D. H., A. Oikari, G. A. Kormanik, and L. Mansberger. *J. Exp. Biol.* 101: 295-305, 1982) role in balancing the net diffusional (and possibly oral) influx of salt (e.g., Shuttleworth, T. J. In: *Physiology of Elasmobranch Fishes*, ed. T. J. Shuttleworth. Berlin: Springer-Verlag, 1988, p. 171-199; Evans, D. H. Osmotic and Ionic Regulation. In: *The Physiology of Fishes*, edited D. H. Evans. Boca Raton: CRC Press, p. 315-341, 1993. The gland is innervated and highly vascularized and therefore potentially under the control of a variety of neurotransmitters, hormones, or paracrines that may have direct effects on the transporting cells or control intra-gland blood flow and perfusion of the transporting cells. In an effort to determine if contraction of the rectal gland itself may play a role in secretion, we mounted 2-3 mm, cross-sectional rings cut from rectal gland from the dogfish shark, *Squalus acanthias*, in thermally-jacketed tissue chambers in order to monitor changes in the diameter of the rings after the application of putative agents, chosen because they have been shown to be vasoactive in the ventral aorta of this species (Evans and Gunderson, *Am. J. Physiol.* 274: R1050-R1057, 1998). Data are expressed as mg change; mean±S.E. (N). The rings were mounted with wires in the central lumen and stabilized at 150-200 mg tension before agents were added. All responses were significantly different from zero ($p < 0.05$) unless indicated

Agonist	ET-1* 0.1 uM	ACh 0.1 mM	NO 10 uM Initial	NO Final	pCNP 0.1 uM	Carb 1 uM	PGE_1 1 uM
Tension Change	70.4±10.9 (11)	11.5±2.7 (9)	-7.3±1.3 (11)	22.3±2.7 (11)	-5.9±1.9 (11)	8.1±4.9 (6) NS	1.9±3.4 (11) NS

*ET-1 = endothelin; ACh = acetylcholine; NO = nitric oxide, pCNP = porcine C-type natriuretic peptide; Carb = carbaprostacyclin; PGE_1 = prostaglandin E_1

The responses of the rectal gland rings were much smaller than we have described for vascular rings maintained at only slightly higher tensions, presumably because of the mechanical buffering produced by the tissue between the central lumen and the gland capsule. Attempts to place the mounting wires just below the capsule, to avoid this potential complication, were unsuccessful. Both ET-1 and ACh produced significant contractions; ET-1 was more stimulatory, as has been described for a variety of tissues. Nitric oxide produced a biphasic response with an initial, slight dilation, followed by a more significant contraction. This is the first time that we have seen any NO-mediated dilation in a shark tissue; the usual response is contraction (e.g., Evans, D.H. and Gunderson, M.P.,*Am. J. Physiol.* 274: R1050-R1057, 1998). CNP, which dilates the shark ventral aorta (Evans, D. H., T. Toop, J. Donald, and J. N. Forrest, Jr., *J Exp Zool* 265: 84-7, 1993) also relaxes the rectal gland rings. Neither prostaglandin (carbaprostacyclin and PGE1) produced a significant response, contrary to what we have seen in the aorta. A potential role for contractile elements in the rectal gland has not been discussed in the recent literature (e.g., Shuttleworth, Op. Cit. 1988), but Bulger (Anat. Rec., 147: 95-127, 1963) described an inner muscle layer below the peripheral connective tissue layer. It is possible that the effector agents produced cellular swelling or shrinkage, which would have appeared to be changes in ring tension; however, it is clear that more structural data are needed. (Supported by NSF IBN-9604824 and REU NSF BIR 9531348).