INITIAL CHARACTERIZATION OF VASOACTIVITY IN THE VENTRAL AORTA OF THE EEL (*ANGUILLA ROSTRATA*)

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In recent years, we have discovered that a variety of receptors for vasoactive hormones and paracrines are expressed in the ventral aorta of the shark, *Squalus acanthias* (e.g., Evans, J. Comp. Physiol. 162: 179-183, 1992; Evans et al., J. Exp. Zool. 265: 84-87, 1993; Evans et al., J. Comp. Physiol. 165: 659-664, 1996). Most recently, our data suggest that the endothelium-derived relaxing factor (EDRF) in the shark aorta is not nitric oxide (NO), but a prostaglandin (PG) (Evans and Gunderson, Am. J. Physiol., in press). This summer, we initiated a series of studies to extend these data to a teleost species, in a effort to determine the evolution of the NO/PG system of EDRFs.

American eels (*Anguilla rostrata*) were purchased from a local dealer and kept in running sea water. The ventral aorta was dissected from double-pithed eels, and tissue rings were mounted in 10 ml of teleost Ringer's in the tension recording system that has been previously described for isolated aortic rings from the shark (e.g., Evans, Op. Cit., 1990 and Evans et al., Op. Cit., 1996). The endothelium lining the rings was not removed, and the rings were stabilized at 100 mg tension before precontraction with 10^{-4} M acetylcholine (ACh) and subsequent addition of putative vasoactive agents. Data are expressed as mean ± se. On a molar basis, endothelin (ET-1) was 2000 X as constrictory as ACh in this preparation. 10^{-7} M ET-1 produced a contraction of 82.6 ± 26.9 mg (N=5) and 10^{-4} M ACh contracted the rings by 34.8 ± 4.6 mg (N=17). The nitric oxide donor, sodium nitroprusside (NaNP; 10^{-4} M) produced significant dilation (-17. 8 ± 5.2 mg; N = 9; p = 0.009), as did nitric oxide itself (4.2 x 10^{-6} M; -12.4 ± 1.7 mg, N = 10; p < 0.0001) and two E-type prostaglandins (10^{-6} M PGE_1: -21.8 ± 5.6 mg; N = 8; p = 0.006 and 10^{-6} M PGE_2: -41.3 ± 12.1; N = 9; p = 0.006). The dilation produced by NO was concentration-dependent, with an initial dilation produced at 2 x 10^{-7} M NO (data not shown).

As has been found in other fishes (e.g., Evans and Gunderson, Am. J. Physiol., submitted), ACh is constrictory when applied to eel aortic rings. In mammals, ACh usually produces dilation when applied to vascular rings with an intact endothelium (e.g., Furchgott and Zawadzki, Nature 288: 373-376, 1980). It is clear that ET-1 is an even more potent constrictory agent in the eel aorta. Our data corroborate earlier studies on the effect of ET-1 in the catfish (*Amiurus melas*; Poder et al., Can. J. Physiol. Pharmacol. 69: 215-217, 1991) and trout (*Oncorhynchus mykiss*; Olson et al., Am. J. Physiol. 260: H1214-H1223, 1991). Unlike the ventral aorta of *Squalus acanthias* (Evans and Gunderson, Op. Cit, submitted), the eel aorta dilates when either NaNP or NO is applied, suggesting that the NO-generated soluble guanylyl cyclase signaling system is expressed in eel vascular smooth muscle. Whether nitric oxide synthase (which generates NO from L-arginine) is expressed in the eel, or any other fish endothelium, remains to be seen. As we found with *S. acanthias*, PGEs relax the rings from the ventral aorta of the eel, suggesting that prostaglandins also may be an endothelium-derived relaxing factor in this species. Thus, our preliminary studies suggest that a variety of receptors for vasoactive substances are expressed in the ventral aorta of the eel, as is soluble guanylyl cyclase. More careful characterization of these systems is warranted. (Supported by NSF IBN-9604824)