RENAL FUNCTION CHANGES IN SQUALUS ACANTHIAS DURING ACUTE SEAWATER DILUTION: THE IMPORTANCE OF HEMODYNAMIC CHANGES

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Acute dilution of the seawater environment is followed by a rapid increase in renal excretion of chloride (Solomon et al, Bull MDIBL 27:18-21, 1987). This chloruretic response is believed to be a consequence of expansion of extracellular volume which attends environmental dilution. Expansion of the extracellular volume could influence renal function through changes in renal hemodynamics and/or the release of diuretic hormones, such as atriopeptin. The purpose of the present study was to investigate the mechanisms underlying this chloruretic response to environmental dilution and to assess the possible role of atriopeptin in this response.

Female sharks weighing 2-3 kg were gently restrained in running 100% seawater tanks with the urinary papilla and the ventral surface of the tail exposed above the seawater. Fish had received 7 ml of 15% inulin 12-15 hours beforehand. A catheter was placed in the bladder and secured with a suture through the papilla. Urine was collected by gravity drainage and suction at the end of each 30 minute period. A balloon tipped catheter (FR#2) was inserted into the dorsal aorta such that the balloon was proximal to the renal arteries. A second catheter (PE90) was inserted distal to this first catheter and attached to a Micron pressure transducer for the continuous monitoring of dorsal aortic pressure.

The seawater environment was acutely diluted by the addition of running tap water at a rate calculated to reduce the seawater to 75% within 30 minutes. Glomerular filtration rate was determined from inulin clearance. Inulin was measured in urine and blood by the resorcinol method. Urine and serum chloride were determined by amperometric analysis and serum osmolality by freezing point depression. All data are expressed as mean + SEM.

Following the acute dilution of the seawater environment to 76±3%, serum osmolality fell progressively over 3 hours from 977±19 to 951±18. Glomerular filtration rate, urine volume, and urine chloride excretion all increased within 30 minutes, the earliest collection period (Table 1). These changes in renal function were associated with a slight rise in dorsal aortic pressure (27.0±2.6 to 28.8±2.1 mmHg, P=NS). There was no increase in the fractional clearance of chloride.

Table 1: The effect of acute dilution of the seawater environment on renal function in Squalus acanthias

TIME (minutes)	100%SW 0	75%SW 60	75%SW 120
GLOMERULAR FILTRATION RATE (ml/h/kg)			
balloon deflated	1.77±.63	2.98±.66	3.35±.73
balloon inflated	1.57±.41	0.94±.38	1.10±.42
CHLORIDE EXCRETION (ueq/min/kg)			
balloon deflated	1.10±.38	2.46±1.30	1.92±.47
balloon inflated	1.10±.34	0.88±0.54	0.46±.10
FRACTIONAL CHLORIDE EXCRETION			
balloon deflated	0.19±.09	0.15±.03	0.12±.02
balloon deflated	0.14±.05	0.29±.20	0.17±.09

deflated n=5 inflated n=4 Data are mean ± SEM

When the balloon tipped catheter was inflated simultaneously with dilution of the environment, dorsal aortic pressure fell (22.5±2.6 to 17.3±3.9, mmHg P=NS) and glomerular filtration rate did not increase. No increase in chloride secretion was observed.

Atriopeptin was infused for a period of three hours at a rate of 55/ng/min following a 500 ng bolus. This infusion failed to alter dorsal aortic blood pressure, glomerular filtration rate, or urinary chloride excretion.

We have previously hypothesized that atriopeptin released in response to volume expansion played a role in the regulation of extracellular volume and sodium/chloride balance. Volume expansion of the intact animal via intraarterial infusion of shark Ringer's solution stimulated both rectal gland and renal excretion of electrolyes and also increased circulating levels of shark atriopeptin (Epstein et al, Bull MDIBL 27:72-73, 1987). However, administration of an intraarterial bolus of rat atriopeptin stimulated chloride secretion by the rectal gland but had no effect on the renal excretion of salt and water (Solomon et al, Am J physiol 249:R348-354, 1985). This suggested that the renal response to a volume load was not mediated by atriopeptin. On the other hand, we and others have observed that in a dilute seawater environment, non-homologous atriopeptin did increase GFR (Benyajati and Yokota, Bull MDIBL 27:56-58,1987) and renal chloride excretion (Solomon et al 1987, loc. cit.)

Following exposure to acute environmental dilution, serum osmolality fell reflecting extracellular volume dilution. We have previously reported that during this same time period serum chloride concentation and hematocrit also fell slightly supporting the notion that extracellular volume was being increased by the entry of hypotonic fluid (Solomon et al 1987, loc. cit.).

We can now describe the hemodynamic changes which occur with such expansion. Although dorsal aortic pressure changes very little over the first few hours, there is a twofold increase in glomerular filtration rate and chloride excretion. This increase in filtration rate can be eliminated by inflation of a balloon catheter placed proximal to the renal arteries which produces a slight but non significant fall in the systemic pressure seen by the kidneys. In no case did renal volume and electrolyte excretion increase when the catheter was inflated in contrast to the increase in these parameters in the cases with a deflated balloon. This suggests that the increase in chloride excretion with acute dilution results from an increase in the filtered load of chloride perhaps attending a volume dependent increase in cardiac output. The lack of change in the fractional excretion of chloride during the periods of increased chloride excretion supports this view.

These results indicate that renal salt excretion in Squalus acanthias is primarily regulated by hemodynamic factors. We were unable to affect renal function in 100% SW adapted animals using a constant infusion of human atriopeptin which should have substantially increased serum levels. Taken together with the effects of atriopeptin in animals adapted to a dilute environment, these observations are consistent with similar findings in mammals suggesting that the volume/hemodynamic status of the animals modulates the effects of atriopeptin on renal function. A more physiologic understanding of the possible role of atriopeptin in the shark must await the availability of specific inhibitors and the homologous form of atriopeptin.

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