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EFFECT OF LONG AND SHORT-TERM CORTISOL TREATMENT ON GILL SODIUM EFFLUX AND NA-K-ATPase DURING SALTWATER ADAPTATION IN THE AMERICAN EEL (Anguilla rostrata)

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We previously reported a correlation between gill Na^+ efflux and Na-K-ATPase during saltwater adaptation in the American eel (Bull, MDIBL 10:17, 1970). Prolonged pretreatment (10-14 days) with cortisol before transfer to saltwater induced a rise in gill Na-K-ATPase activity and stimulated gill Na^+ efflux during adaptation so that rates characteristic of full adaptation (9-14 days) were achieved by 4-5 days. To determine if pretransfer cortisol induced elevation in Na-K-ATPase was necessary for this accelerated Na^+ efflux, further studies were performed on normal, long-term cortisol (10-14 days) and short-term cortisol (2 days) treated eels during adaptation to salt water. Treated eels received hydrocortisone, 4 mg/Kg/day.

The top graph of Figure 1 shows Na^+ efflux plotted against days of salt water adaptation in the three groups. Unlike the European eel (Anguilla anguilla) in which full adaptation is reported to occur in 2-3 days, the American eel (solid line, solid dots) has a progressive increase in efflux with full adaptation reached by 9-14 days. During early adaptation (middle graph) there is an abrupt rise in plasma Na^+ (mean 228 mEq/L on day 2) which falls gradually as Na^+ efflux increases. The bottom graph (dots, solid lines) shows an increase in gill Na-K-ATPase in normal eels which parallels the increase in sodium efflux during saltwater adaptation (correlation coefficient for 49 pairs of Na-K-ATPase levels and fluxes, $r = 0.628$, $p < .001$).

In animals with long-term cortisol pretreatment (open circles, dashed line), full efflux adaptation and a normal plasma Na^+ are reached by 3-4 days with activity of Na-K-ATPase remaining elevated throughout. Animals receiving short-term cortisol pretreatment (open circles, dotted line) had statistically significant increases in Na^+ efflux at days 2, 3, and 4, and had a plasma Na^+ intermediate between long-term cortisol and control eels by day 4. In contrast to long-term cortisol eels, gill Na^+-K^+-ATPase activity in this group was not elevated above control values during days 2-4 when Na^+ efflux was increased. Gill Mg-ATPase was unchanged by saltwater adaptation or cortisol treatment.

These studies indicate that (1) in normal eels adapting to saltwater, there is a progressive increase in gill Na-K-ATPase activity which correlates with the gradual rise in gill Na^+ efflux; (2) long-term cortisol treatment before adaptation increases gill Na-K-ATPase and permits a marked acceleration of Na^+ efflux adaptation; (3) brief cortisol treatment, while insufficient to induce an increase in Na-K-ATPase activity, exerts a permissive effect on Na^+ transport, permitting an accelerated Na^+ efflux when the eel is exposed to saltwater. Milne (J. Endocr. 49:177, 1971) has recently shown that the reduction in eel gill Na-K-ATPase activity following hypophysectomy can be restored by ACTH injections. Each of these observations lends support to the hypothesis that cortisol is the mineralocorticoid involved in regulation of both Na-K-ATPase activity and sodium transport in this species.

EFFECT OF CORTISOL TREATMENT ON GILL Na^+ EFFLUX, PLASMA Na^+ AND GILL Na-K ATPase DURING SW ADAPTATION IN THE EEL (ANGUILLA ROSTRATA)