Excretion of a Sodium Chloride Load by the Marine Dogfish, Squalus Acanthias

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In the marine dogfish, exogenous sodium bicarbonate is excreted by the gills. It has been postulated that this involves an ion exchange mechanism using either cations (Na^+ for H^+) or anions (HCO_3^- for Cl^-).

Intravenous administration of sodium chloride (as much as 20 ml of a 5N NaCl solution in a 3.8 kg fish) causes: 1) increased renal excretion of sodium without a proportionate increase in chloride excretion, and 2) a disproportionate increase in plasma sodium concentration as compared to chloride.

These observations indicate that the sodium of the administered sodium chloride load is preferentially excreted by the kidneys, possibly in combination with $H_2PO_4^-$. An extrarenal site of chloride excretion must be postulated to account for the rapid decrease in plasma chloride concentration which occurred even though chloride excretion by the kidney increased less than sodium excretion. It is conceivable that chloride is excreted via the gills, possibly by an ion exchange mechanism linked to bicarbonate.

Active Transport of Endogenous Bases in the Aglomerular Kidney of Lophius

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Creatine and trimethylamine oxide (TMAO) are the chief nitrogenous constituents of normal *Lophius* urine, and both of these organic bases have characteristically high urine/plasma concentration ratios. Competition studies involving various organic bases indicate that creatine and TMAO are excreted independently by separate transport mechanisms. TMAO excretion is inhibited competitively by tetraethylammonium ion (TEA) and the Cyanine dye #863 — compounds previously shown to be transferred actively by an organic base secreting mechanism which is of general occurrence among vertebrates.

TEA does not inhibit competitively the active tubular reabsorption of TMAO in the dogfish, *Squalus,* with doses which markedly depress its tubular excretion in *Lophius.*

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