

**THE DEVELOPMENT OF ION REGULATION
IN THE RAINBOW TROUT**

Gordon McDonald
Department of Biology, McMaster University
1280 Main Street West, Hamilton, Ontario Canada L8S4K1
(905) 525-9140, X 24266, mcdonald@mcmaster.ca

C.M. Misiaszek, K.J. Barrett, P.J.S. Smith[†] and M.J O'Donnell
[†]Biocurrents Research Center, MBL, Woods Hole MA, USA

EXTENDED ABSTRACT ONLY -- DO NOT CITE

These studies (the MSc thesis projects of K. Barrett and C. Misiaszek) examined the development of iono-regulatory mechanisms in rainbow trout from the eyed-egg stage (~28 days before hatching at 7° C) to the end of yolk sac absorption (~36 days after hatching) and up to ~25 days past the point of first feeding. Measurements of ion uptake (Na^+ , Cl^- and Ca^{2+}) from the external medium, changes in tissue ion concentrations, and in specific ion activities (NH_4^+ , H^+ , Na^+ and Ca^{2+}) at the surfaces of the gills, skin and yolk sac were used to localize and characterize the ion transport mechanisms.

Na^+ regulation develops early relative to Ca^{2+} or Cl^- regulation. A saturable Na^+ transporter is clearly present by the eyed-egg stage and is responsible thereafter for the net accretion of Na^+ over the remaining period of embryonic development and throughout larval development. The transport mechanism continues to mature well into larval development with increasing affinity and capacity. In contrast, evidence for the net accretion of Cl^- and Ca^{2+} is ambiguous until the beginning of larval development. There appears to be only minimal changes to the Cl^- transport mechanism through larval development and the Ca^{2+} transporter shows a decrease in affinity with development. Both the chorion of the embryo and the surface of the larvae have a high binding capacity for Ca^{2+} which complicates the interpretation of findings regarding the Ca^{2+} transport mechanism.

Sampling the near surface environment of the larval trout with a Na^+ specific electrode showed that the gills are, from early in larval development, the main site of Na^+ loss, and therefore, by inference, the main site of Na^+ uptake. Moreover, Na^+ permeability increases in parallel with increases in Na^+ uptake through development, indicating a much larger self-exchange for Na^+ than for other ions. In contrast, Ca^{2+} loss appears to be non-specifically distributed to all larval surfaces. However, the apparent Ca^{2+} loss is more likely evidence of pronounced surface binding of Ca^{2+} . The presence of mitochondrial rich cells (MRCs) in the yolk sac that show no evidence of Na^+/K^+ ATPase activity suggests that these cells might be specialized for Ca^{2+} transport while MRCs found in the gills are specialized for Na^+ (and Cl^-) transport. Measurements of boundary layer acidification suggest that the whole body surface appears to contribute to CO_2 excretion with the gills playing a minor role. In contrast, ammonia excretion appears to be confined to the gills. However, we found that acute elevation of external Na^+ did not stimulate ammonia excretion, a finding that argues against the presence of a $\text{Na}^+/\text{NH}_4^+$ antiport in the gills.

The loss of the yolk sac and the onset of feeding prompts a further suite of changes in the iono-regulatory machinery particularly with respect to Ca^{2+} ; a dramatic change in the surface binding properties for Ca^{2+} , a down-regulation of trans-epithelial Ca^{2+} transport and an increasing reliance on dietary Ca^{2+} to meet the demands of skeletal mineralization. This finding goes against the conventional wisdom that teleosts meet their Ca^{2+} requirements primarily through absorption across the gills.

We conclude that the regulatory mechanisms for each ion are qualitatively and quantitatively quite distinct from one another and may well be distributed to different MRC sub-types. The early development of the Na^+ transport mechanism and the fact that Na^+ uptake substantially exceeds that required for Na^+ balance suggests a prominent role for Na^+ in acid-base regulation (i.e. Na^+/H^+ exchange). The early development of an acid excretion mechanism may be necessary to clear acidic metabolites accumulated through embryonic and early larval development.

