

**OSMOREGULATION IN AMERICAN SHAD; READJUSTING
EXPECTATIONS OF MIGRATORY PHYSIOLOGY**

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The study of migratory fish physiology has a rich history that has often focused on the development of salmonids through their downstream migratory period. The parr-smolt transformation has been well characterized and its study stands as a most impressive example of the interface of behavioral, physiological and ecological approaches to biology. For salmonids, the development of seawater tolerance, changes in morphology and migratory behavior are largely coordinated. As such, there is a temptation to transpose these patterns on other species. The purpose of this work is to explore the migratory physiology of American shad, *Alosa sapidissima*. This prominent anadromous fish deviates widely from the salmonid model.

Adult shad spawn in fresh water in the spring and young shad develop the ability to enter into full strength seawater at the larval-juvenile transition, months prior to downstream migration (Zydlewski and McCormick, 1997a). This development is coincident with gill development, increased gill Na⁺,K⁺-ATPase activity and differentiation of gill chloride cells (Zydlewski and McCormick, 2001). While juvenile shad are physiologically competent to enter seawater in the summer of their fresh water residence, most remain in fresh water until autumn.

While shad maintain seawater tolerance through the autumnal period of seaward migration they gradually lose the ability to regulate their ions in fresh water. This is evidenced by decreased plasma chloride, decreased plasma osmolality, increased muscle moisture and high mortality if prevented from entering

seawater (Zydlewski and McCormick, 1997b; unpublished data). Associated with impaired hyperosmoregulatory ability is an increase in gill Na^+, K^+ -ATPase activity which is the result of an intense proliferation and enlargement of Na^+, K^+ -ATPase-rich chloride cells on both the primary filament and secondary lamellae of the gill (Zydlewski and McCormick, 2001). The disappearance of chloride cells on the secondary lamellae upon seawater entry indicates that the function of these cells is likely to be in ion uptake. The nature of this developmental loss of hyperosmoregulatory ability is not understood, but the clear parallel acceleration of physiological changes under declining temperature through autumn implies a direct relationship between osmoregulatory ability in fresh water and downstream migratory behavior.

In nature, migration is protracted over several months and over a considerable temperature range. There is no apparent physiological disadvantage to migrating early while river temperatures are warm but the impending osmoregulatory changes associated with declining temperature in fresh water may impact the migratory success of late migrants. In captivity, a cessation of feeding below 10 °C in juvenile shad has been observed and other sub-lethal and lethal effects occur at lower temperatures in fresh water. During the period of migration, juvenile shad also exhibit a heightened responsiveness to acute handling and confinement stress (Shrimpton et al, 2001). These effects are likely due to osmotic stress.

These behavioral and physiological effects are likely to impair downstream migratory success through osmotic perturbation upon seawater entry. Indeed, juveniles iso-thermally transferred to 24 ppt seawater in September (24°C; "early") and in November (10°C; "late") demonstrated markedly different abilities to acclimate to seawater. Early acclimation resulted in a modest osmotic perturbation that recovered rapidly. Hematocrit declined 14% at 24h, recovering within 48 h. Plasma osmolality increased 6% at 4h, recovering within 8 h. Early acclimation caused a 2-fold increase in gill Na^+, K^+ -ATPase activity by 24 h and a 4-fold increase by 4 d. The number of chloride cells on the primary gill filament increased 2-fold by 4 d. Chloride cells on the secondary lamellae rapidly decreased from 22 cells/mm to less than 2 cells/mm within 4 d. Late acclimation resulted in a severe and protracted osmotic perturbation. Hematocrit levels declined 23% by 4 d, recovering by 14 d. Plasma osmolality increased 36 % by 48 h, recovering by 4 d. Initial gill Na^+, K^+ -ATPase activity was 2-fold greater than in early fish and did not change during acclimation. Initial number of chloride cells on the primary filament was 2-fold greater than early fish and did not increase during acclimation. Initial

number of chloride cells on the secondary lamellae was 5-fold greater than early fish (116 vs 22 cells/mm) declining to negligible numbers over 14 d.

These data indicate a unique pattern of behavior and physiology in juvenile American shad. Not only does the physiological ability to enter into seawater greatly precede migration, but migrant fish exhibit impaired osmoregulatory ability in fresh water. In fact, late migrants face an even greater physiological challenge during seawater acclimation. Physiological performance, together with ecological factors (such as predation and food availability) apparently stabilizes the timing of autumnal migration.

References

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