New insights into nitrogen excretion at the gulf toadfish gill: The role of Rh glycoproteins

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The gulf toadfish, *Opsanus beta*, is facultatively ureotelic. The transition from ammonotely to ureotely can be triggered by crowding or confinement stress and involves the cortisol-mediated activation of urea synthesis and excretion pathways. At the same time, branchial ammonia excretion falls. Because models of ammonia excretion in teleost fish recently have been reshaped by the discovery of Rhesus (Rh) glycoproteins, a family of proteins that facilitate the movement of ammonia across cell membranes, ammonia excretion by the toadfish was revisited. Real-time RT-PCR was used to assess branchial mRNA expression of four Rh glycoprotein isoforms in relation to ammonia and urea excretion during the transition to ureotely and in response to manipulation of cortisol and/or ammonia levels. The data suggest that a reduction in ammonia transport capacity contributes to the transition to ureotely, and that cortisol regulates branchial Rh mRNA expression and ammonia excretion. Thus, cortisol appears to play a key role in coordinating and controlling nitrogen excretion pathways in the toadfish through its dual effects in activating urea synthesis and excretion pathways while inhibiting ammonia excretion pathways. This research was funded by NSF (MDM) and NSERC of Canada (KMG and PJW).