Gastrointestinal response of the gulf toadfish (*Opsanus beta*) to respiratory acidosis induced by imminent projected oceanic carbon dioxide concentrations

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Oceanic uptake of elevated atmospheric CO_2 has decreased ocean pH by 0.1 at unprecedented rates since pre-industrial levels, and is predicted to decrease by 0.77 units over the next two centuries. Contrary to the breadth of studies showing effects of ocean acidification on calcifying organisms, there have been few studies addressing acid-base compensation of marine teleost fish exposed to imminent CO_2 levels. In the present study, intestinal and rectal contents were collected from toadfish (*Opansus beta*) exposed to 380(control) and 1900ppm CO_2 for 72 hours. Fluids were analyzed for pH, osmotic pressure, ionic composition, and total CO_2. Solid precipitated $CaCO_3$ samples were analyzed for determination of titratable alkalinity, Mg^{2+} and Ca^{2+}. Fish exposed to 1900ppm exhibited higher rectal HCO_3^- excretion rates, higher rectal fluid HCO_3^- and lower rectal fluid Cl^- concentrations, suggesting increased intestinal anion exchange. These results were not unexpected considering PCO_2 and HCO_3^- are known substrates for intestinal base secretion, a vital part of marine fish osmoregulation. This study verifies that elevation of plasma HCO_3^- during hypercapnia exposure results in greater loss via the intestine and provides evidence for a contribution of the gastrointestinal tract acting against the compensatory mechanism for CO_2 induced acidosis.