Scientific Paper:

The Journal of Experimental Biology 206, 3895-3903, 2003

Energy budget of hepatocytes from Antarctic fish (*Pachycara brachycephalum* and *Lepidonotothen kempi*) as a function of ambient CO_2: pH-dependent limitations of cellular protein biosynthesis?

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Abstract:

Scenarios of rising CO_2 concentration in surface waters due to atmospheric accumulation of anthropogenic CO_2, or in the deep sea due to anticipated industrial dumping of CO_2, suggest that hypercapnia (elevated partial pressure of CO_2) will become a general stress factor in aquatic environments, with largely unknown effects on species survival and well being, especially in cold and deep waters. For an analysis of CO_2 effects at the cellular level, isolated hepatocytes were prepared from two representatives of the Antarctic fish fauna, *Pachycara brachycephalum* and *Lepidonotothen kempi*. Correlated changes in energy and protein metabolism were investigated by determining the rates of oxygen consumption at various levels of P_{CO2}, of intra- and extracellular pH, and after inhibition of protein synthesis by cycloheximide. A decrease in extracellular pH (pHe) from control levels (pHe 7.90) to pHe 6.50 caused a reduction in aerobic metabolic rate of 34–37% under both normocapnic and hypercapnic conditions. Concomitantly, protein biosynthesis was inhibited by about 80% under conditions of severe acidosis in hepatocytes from both species. A parallel drop in intracellular pH probably mediates this effect. In conclusion, the present data indicate that elevated P_{CO2} may limit the functional integrity of the liver due to a pronounced depression in protein anabolism. This process may contribute to the limits of whole-animal tolerance to raised CO_2 levels.

Key-words: Hypercapnia, CO_2, antarctic fish, p*achycara brachycephalum*, *lepidonotothen kempi*, oxygen consumption, metabolic rate, respiratory acidosis, protein synthesis, hepatocyte