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Energy budget of hepatocytes from Antarctic fish (Pachycara brachycephalum and Lepidonotothen kempi) as a function of ambient CO₂: pH-dependent limitations of cellular protein biosynthesis?

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Abstract:

Scenarios of rising CO₂ concentration in surface waters due to atmospheric accumulation of anthropogenic CO₂, or in the deep sea due to anticipated industrial dumping of CO₂, suggest that hypercapnia (elevated partial pressure of CO₂) 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 CO₂ 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 Pₐ₄, 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 Pₐ₄ 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 CO₂ levels.

Key-words: Hypercapnia, CO₂, antarctic fish, pachycara brachycephalum, lepidonotothen kempi, oxygen consumption, metabolic rate, respiratory acidosis, protein synthesis, hepatocyte