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Discussions

Interactive comment on "The real limits to marine life: a further critique of the Respiration Index" *by* B. A. Seibel and J. J. Childress

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Climate change and its biological effects have emphasized the need to understand the limiting conditions of life. It would be very useful to have an indicator at hand that allows those limits to be quantified and that links biological principles and the physical environment. One such indicator recently suggested is the so-called respiration index that has been proposed to reflect thermodynamic limits to aerobic metabolism (Brewer and Peltzer, 2009). The concept proposes that with falling ambient oxygen and increasing CO2 partial pressures a threshold RI value indicates insufficient free energy change during oxidative metabolism of organic matter for the organism to be viable. This indicator has already been used as a quantitative indicator in modeling and projecting geographic patterns of limiting ocean conditions.

Previous criticism has already emphasized that such limits are beyond real limitations and found far beyond the actual conditions limiting animal life, as mitochondria, the sites of oxidative metabolism, normally see lower than ambient oxygen and higher than ambient CO2 partial pressures. In animals diffusion gradients from ambient to mitochondria and from mitochondria to ambient are modulated by convective blood oxygen and CO2 transport, resulting in species specific consequences and sensitivities to environmental oxygen shortages.

The present criticism goes to the core of the equation pointing to the exergonic nature of glucose oxidation and the relative irrelevance of realistic CO2 concentrations in influencing this driving force. This is a convincing argument backed further by discussing the transition to breathing air or the role of pH regulation in blood oxygen transport, or the classic example of squid being asphyxiated by elevated CO2 through its action on the blood pigment, not at the mitochondrial level. Here a ratio of gas partial pressures of zero certainly does not indicate a no flow equilibrium situation. When dissecting the equation one also wonders why partial pressure or fugacity are used instead of concentration, as concentrations and not partial pressures are relevant components of the equation. The Respiration Index thus does not reflect physiological and ecological realities as pointed out by the authors and others in this and in earlier criticisms.

The following points require revision:

p. 16526, l.25: Intracellular PO2 being brought to higher than ambient values sounds like a very unlikely and rare situation. That statement must be backed by data and a reference. Also to say that cellular PO2 is independent of ambient values is overstated and not backed by evidence.

p. 16527: That any predictions by use of the RI are inappropriate is clear but the word "dangerous" is not the right one to use here.

p. 16528: The authors rightly discuss the critical PO2 as the relevant oxygen threshold which is species and life stage specific. Here they should include further literature that goes beyond their own and broaden their statement to one valid for all animals. Pcrit being defined as "the oxygen partial pressure below which metabolism cannot be regulated independently of PO2" is the classical definition dating back to the pioneers of comparative physiology (this should be said) but this definition is only valid for oxyregulators and not encompassing oxyconformers among invertebrates. Progress made in the early nineties in the Grieshaber lab and elsewhere has shown in all cases tested that the Pcrit is also characterized by transition to anaerobic metabolism, in oxyconformers and oxyregulators. This transition is a more reliable component in quantifying limiting oxygen conditions. Metabolic depression may occur above Pcrit but is not per se indicative of an oxygen limitation threshold.

Grieshaber, M.K., I. Hardewig, U. Kreutzer, H.O. Pörtner (1994) Physiological and metabolic responses to hypoxia in invertebrates. Rev. Physiol. Biochem. Pharmacol. 125: 43-147.

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