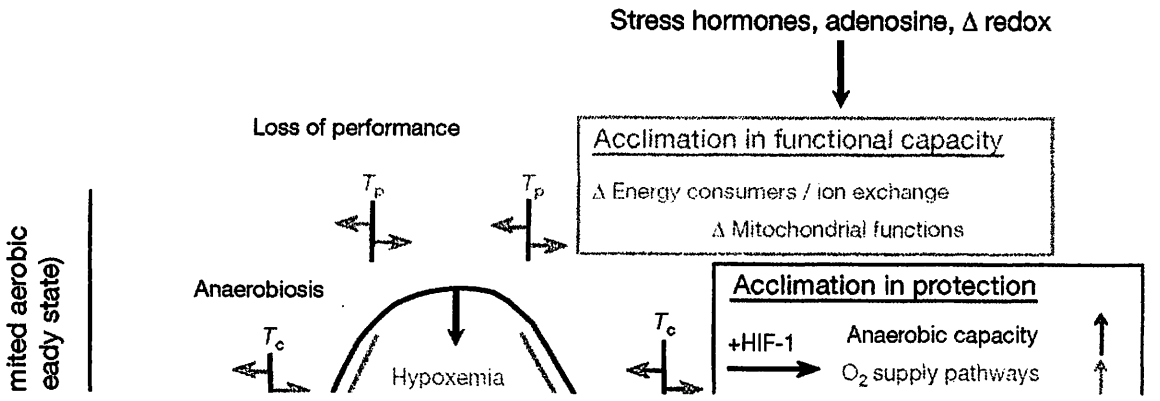
Stress hormones, adenosine, Δ redox
Loss of performance
Acclimation in functional capacity
Δ Energy consumers / ion exchange
Δ Mitochondrial functions
$T_P$
$T_P$
Anaerobiosis
$T_c$
Hypoxemia
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Acclimation in protection
+HIF-1
Anaerobic capacity
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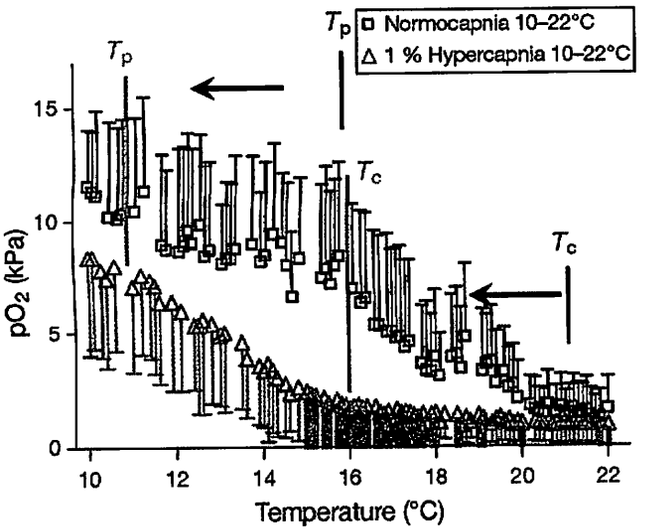


Fig. 4. Heat tolerance of the edible crab *Cancer pagurus* under normo- and hypercapnia (after Metzger et al. 2007). Discontinuities in the curve depicting arterial oxygen tensions ($pO_2$) under normocapnia were identified as indicators of thermal limits (upper pejus temperature, $T_p$, according to Frederich & Pörtner 2000) reflecting onset of a loss in ecologically relevant performance and fitness (Pörtner & Knust 2007). Highly elevated $CO_2$ levels (1% hypercapnia) cause heat tolerance to decrease dramatically by about 5°C. Similarly, the general lowering of haemolymph $pO_2$ under hypercapnia causes a downward shift of upper critical temperatures ($T_c$) by about 4.5°C. Assuming a symmetric thermal window the data reflect a high sensitivity to $CO_2$ and shrinkage of the thermal window by more than 80%. Temperature-dependent biogeographical ranges of marine animals may thus respond to even moderately elevated $CO_2$ levels (Pörtner et al. 2005)

This paper presents a set of hypotheses for a comprehensive mechanistic framework which brings the individual effects of the factors temperature, $CO_2$ and hypoxia together into an integrative picture of climate sensitivity at organismal level (Fig. 5). The mechanistic scheme illustrates how virtually all mechanisms relevant in setting and shifting thermal windows will be affected through the exacerbation of hypoxemia (hypoxia in body fluids) under the effects of ambient hypercapnia or hypoxia. Both factors cause a decreased pH regulation capacity and setpoint of acid–base regulation, and will likely do so to the largest extent where temperature extremes are already causing hypoxemia. Thermal windows and sensitivities differ between species co-existing in the same ecosystem. Through differences in sensitivities, some of these effects will cause changes in species interactions and thereby functional shifts observed in ecosystem level processes.

Comparable to thermal limitation (Pörtner 2002), efforts to understand sensitivity of marine animals to $CO_2$ should include studies at a high organisational level, especially with respect to the intact organism and the mechanisms involved. This includes studying the patterns of acid–base regulation and hypoxemia as well as the capacity to regulate extracellular acid–base status and mainly extracellular pH, at extreme temperatures for an analysis of the background of temperature-dependent $CO_2$ or hypoxia sensitivity and, vice versa, $CO_2$- and oxygen-dependent thermal sensitivity.

While larval and juvenile stages may be more sensitive when effects of hypercapnia are studied in isolation (Ishimatsu et al. 2004, 2005) these relationships may become more complicated when temperature effects are considered. The temperature signal is currently the strongest signal eliciting ecosystem change, due to physiological impacts and the limited thermal windows of individual species (e.g. Pörtner & Knust 2007). The available data indicate that (1) thermal extremes affect large individuals first and (2) a thermally variable environment favours species with smaller individuals including juveniles, due to their wider windows of thermal tolerance (e.g. Pörtner et al. 2008). If $CO_2$ exacerbates these relationships by narrowing thermal windows this would favour smaller body sizes (and their wider thermal windows) even more and further constrain the size range of a species. Constant $CO_2$ conditions may thus favour larger body sizes. The synergistic interactions between temperature and $CO_2$ thus have implications for how the sensitivity of a species to global change depends on body size (allometry). While sensitivity to $CO_2$ per se may be highest in early life stages of many organisms, thermal stress also impacts the largest individuals of a species. With their already constrained thermal windows, they may then also become more sensitive to the synergistic effects of $CO_2$. Once again, the regulation of extracellular acid–base status may be crucial in this context as efficient pH regulation and its temperature-dependent characteristics are limited to within the thermal window of a species (e.g. Sommer et al. 1997).

As a general conclusion, these relationships and their implications at an ecosystem level need to be investigated with a wide range of organisms from various habitats. With the currently available data it is unclear whether these relationships have already started to affect species and ecosystems, for example through a narrowing of biogeographical distribution ranges. It appears most likely that such integrative effects will be the first to be observed in the field and bring with them the need to then disentangle the contribution of $CO_2$, hypoxia and temperature as well as their synergistic interaction in causing those effects.