Synergistic impacts of increasing temperature and CO_2 levels on the physiology of *Mytilus edulis* from the White Sea

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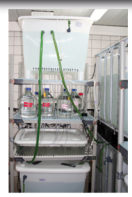
INTRODUCTION

With global climate change ocean warming and acidification occur concomitantly. Environmental change develops rapidly and may leave insufficient time for evolutionary adaptation. Thus, the survival and distribution of species will depend on their existing ability to exploit their physiological plasticity. Investigating the synergistic effects of warming and CO_2 accumulation is important to predict the future state of matrine ecosystems.

An existing concept places emphasis on a central role for extracellular pH and the capacity of acid-base regulation in shaping sensitivity to ocean acidification. pH regulation and the energetically costly processes involved appear crucial to sustain the performance of marine organisms. Especially under rising temperatures, small pH disturbances in body fluids might already exert critical impact on physiological processes. We therefore studied the effects of ocean acidification on thermal tolerance, energy metabolism and acid-base regulation capacity of the White Sea (sub-Arctic) population of the blue mussel *Mytilus edulis*.

MATERIAL AND METHODS



Mytilus edulis

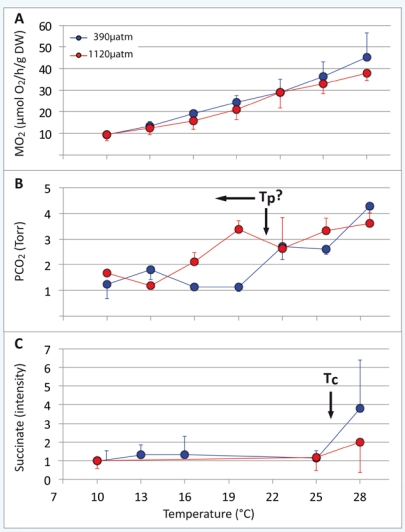
Wild type adult mussels (3-8cm shell length) were collected from the subtidal zone of the White Sea, Russia, transfered to the AWI, divided in two groups and acclimated for at least one month at 10°C. After pre-acclimation mussels were exposed to normocapnia (390µatm) and hypercapnia (1120µatm), respectively, in a recirculating seawater system with pre-equilibrated header tanks (see above).

Experimental design

Two weeks after CO_2-exposure mussels were challenged by an acute temperature rise from 10-28°C (3°C/night). Respiration rate was recorded online using microoptodes (PreSens). At each temperature step samples of haemolymph were taken anaerobically close to the posterior adductor muscle. Samples of mantle tissue were frozen in liquid N_2. Haemolymph acid-base and oxygen status were determined with a blood gas analyser (PO_2, PCO_2, pH) and a gas chromatograph (CCO_2). Additional acid-base parameters (e.g. HCO_3^-) were calculated according to Heisler (1986). Intracellular pH of tissue was determined by the homogenate method developed by Pörtner et al. (1990). Onset of anaerobiosis (succinate level) were measured in deproteinized tissue extracts using high resolution 1H-NMR spectroscopy in a 400 WB Avance NMR spectro meter (BrukerBioSpin).

RESULTS

Thermal tolerance window & energy metabolism

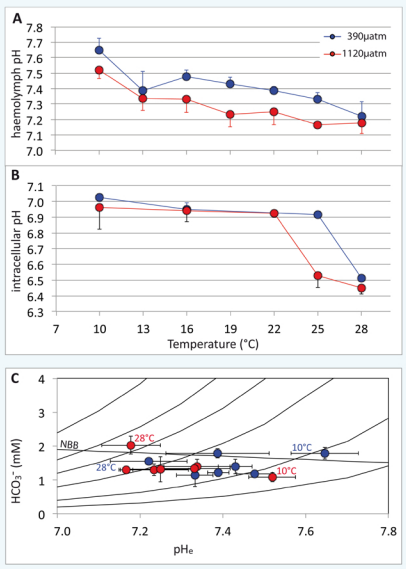


N=5-8

The temperature induced increase in oxygen consumption (MO_2) led to Q_{10}-values (10-28°C) of 2.4 (390µatm) and 2.2 (1120µatm) indicating a mild limitation of aerobic metabolism in CO_2 exposed animals (Fig A). Haemolymph PO_2 was unaffected by CO_2 (data not shown). In contrast, the temperature induced increase of haemolymph PCO_2 (Fig B) occurred at a lower temperature (19°C) in CO_2 exposed animals than in controls (22°C). The difference might be due to reducted ventilation activity under CO_2 which might indicate a downward shift of the upper pejus-temperature (Tp). Anaerobic metabolites (succinate) accumulated above 25°C indicating an upper critical temperature (Tc) independent of CO_2 albeit anaerobic metabolic rate was lower under hypercapnia (Fig C).

The data set indicates that Tp is shifted downwards whereas Tc seems to be unaffected by ocean acidification. However, the lower anaerobic (and aerobic) metaboic rate indicates an enhanced passive heat temperature tolerance under hypercapnia.

Acid-base regulation capacity



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The decrease in haemolymph acid-base status during warming followed alphastat expectations (Fig A) involving a respiratory acidosis largely along the non-bicarbonate buffer line (NBB, Fig C). Hypercapnia led to a continually stronger acidosis of the internal milieau. Intracellular pH (pH_i, Fig B) was initially maintained under warming before a sudden acidification set in. The drop in pH_i occurred earlier under hypercapnia, indicating reduced energy investigation for pH_i regulation.

Reduced energy-dependent processes under hypercapnia may support short term passive tolerance but hamper long term aerobic performance in the warmth.

CONCLUSIONS

- Combined hypercapnia and heat exposure lead to an early intracellular acidosis indicating reduced energy allocation to intracellular pH regulation.
- Reduction of energy-dependent processes by hypercapnia may enhance passive tolerance to temperature extremes, however, at the expense of aerobic performance
- CO_2 exposure may affect pejus limits more than critical
- Permanent hypercapnia may only be sustained at the expense of organismic performance, especially at the limits of the thermal performance window.

LITERATURE

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