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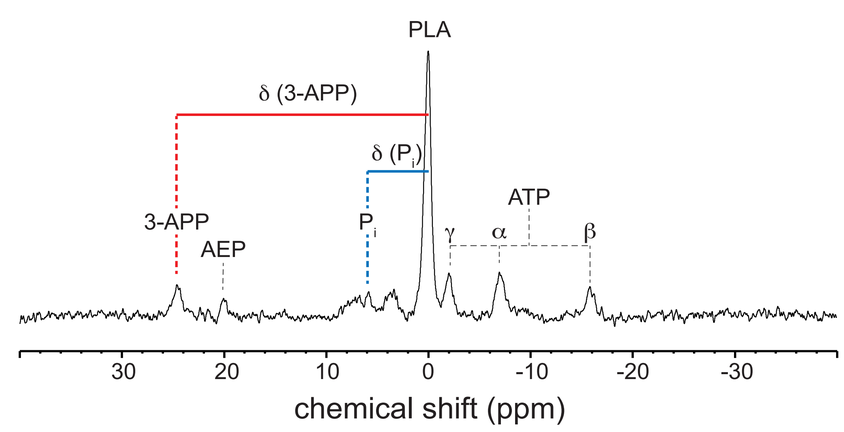
Seawater alkalinity modulates the response of *Carcinus maenas* to Ocean Acidification

Background

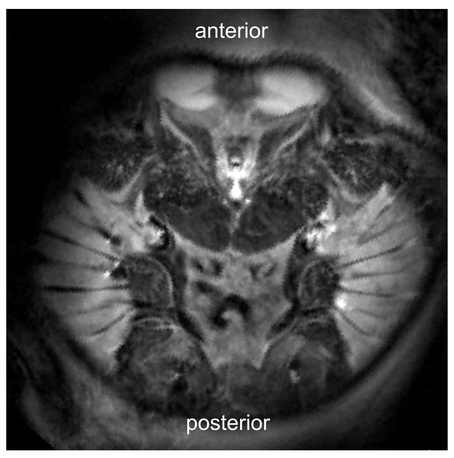
Decapod crustaceans are thought to compensate for an hypercapnic acidosis through net uptake of bicarbonate from sea water. Failing to maintain pH_e would induce metabolic depression[1;2]. We studied the capacity for acid-base regulation in *Carcinus maenas* under various conditions of water physicochemistry and responses of internal acid-base parameters and ion concentration, relating to potential feedbacks on metabolic rate and cardiovascular activity.

Methods

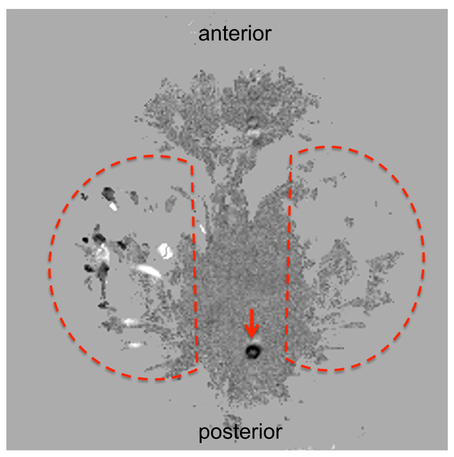
Shore crabs *C. maenas* were exposed for four weeks to: 1. control; 2. ocean acidification (1800 µatm); 3. low total alkalinity (TA = 1.1 mM); 4: OA + low TA. Metabolic rates were determined using intermittent flow respirometers[3]. Haemolymph CO_2 and ion concentrations were measured through gas-, and ion-chromatography, respectively. *In vivo* MRI determined heart rate and blood flow, while ^{31}P-NMR spectroscopy was used to measure pH_e and pH_i from the chemical shift of 3-aminopropylphosphonate (3-APP) and P_i signals, relative to PLA as an internal standard (below)[4].



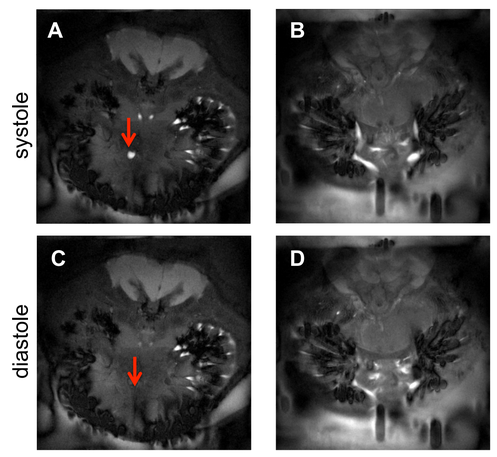
In vivo ^{31}P-NMR spectrum of *C. maenas*. Intra- and extracellular pH were calculated from chemical shifts of P_i and 3-APP signals, relative to PLA.



Anatomical MRI overview slice, including the heart of *C. maenas* (dorsal view). In the centre of the heart, the entrance to the *arteria sternalis* is visible. Single gill filaments are visible, lateral of the heart.

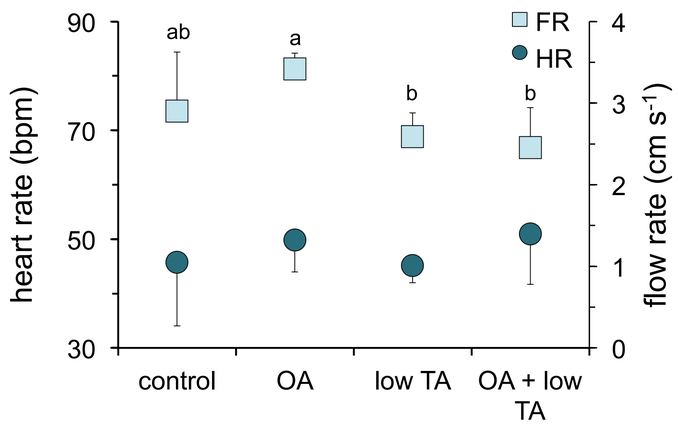


Phase contrasted MRI (dorsal view), depicting haemolymph velocity in *C. maenas*. Brightness indicates flow direction and -intensity. The red arrow highlights the *arteria sternalis*.

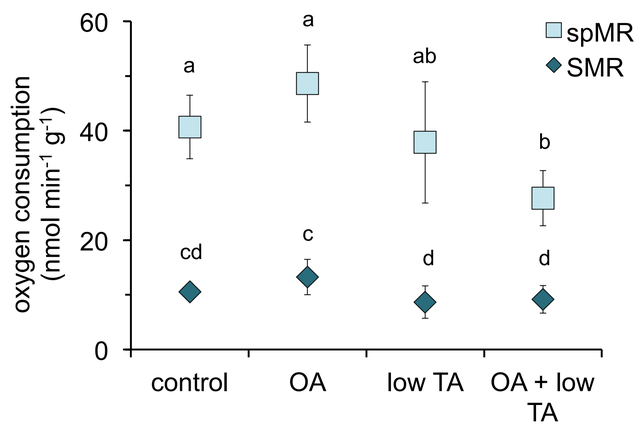


Self-gated, flow-weighed MR images of systole (A, B) and diastole (C, D) of *arteria sternalis* and heart of *C. maenas*. Bruker IntraGate© software detects heart rate non-invasively.

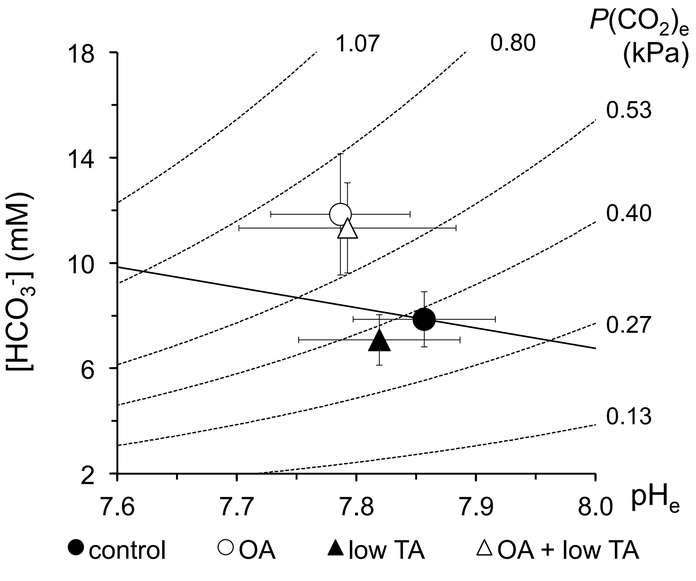
Results



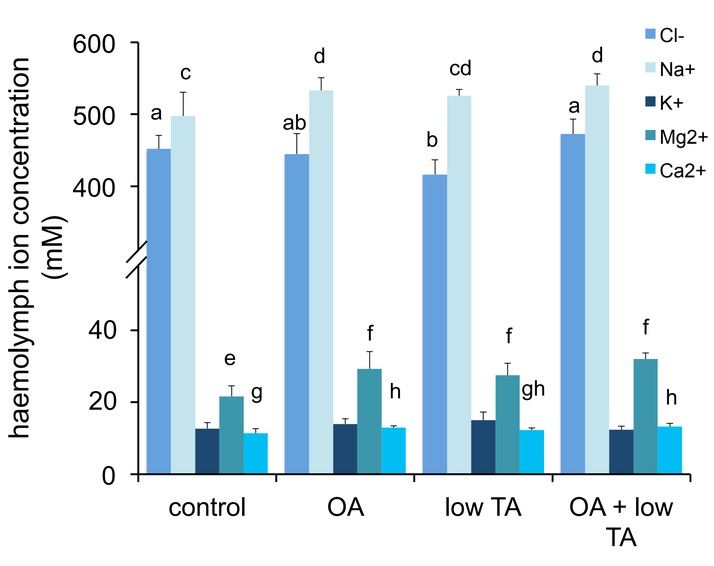
Heart rates were unaffected by changes in water chemistry, while blood flow in the arteria sternalis under OA was significantly depressed at low TA.



Standard metabolic rates and oxygen consumption rates during spontaneous activity were both significantly depressed under OA, when TA was reduced.



C. maenas was able to maintain pH_e during exposure to different acidified water conditions, through actively elevating haemolymph bicarbonate concentrations.



While treatments caused elevated haemolymph levels of cations, $[Cl^-]$ were depressed under normocapnic low TA. The increase in [cations] at mostly constant [anions] reflects a higher strong ion difference under acidified water conditions.

Conclusions

Hypercapnia led to elevated haemolymph PCO_2, but changes in intra- and extracellular pH were compensated for through increased bicarbonate levels, irrespective of ambient alkalinity. pH regulation caused an increasing strong ion difference[5], possibly elicited by Na^+/H^+ exchange or related to NH_4^+ excretion as acid-base regulatory mechanisms.

Total alkalinity simultaneously affects the response of cardiovascular activity and whole-animal energy demand under OA. High oxygen consumption rates and blood flow under OA were significantly depressed under hypercapnic low TA.

The seawater carbonate system does not affect pH regulation, but hypercapnic low alkalinity depresses cardiac- and whole-animal activity

References
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4) Gillies, R. J., Liu, Z., Bhujwalla, Z. (1994): ^{31}P-MRS measurements of extracellular pH of tumors using 3-aminopropylphosphonate. *American Journal of Physiology.* 267: C195-C203.
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