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Pacific oysters do not compensate growth retardation following extreme acidification events

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Abstract :

Ocean acidification caused by anthropogenic carbon dioxide emissions alters the growth of marine calcifiers. Although the immediate effects of acidification from global ocean models have been well studied on calcifiers, their recovery capacity over a wide range of pH has never been evaluated. This aspect is crucial because acidification events that arise in coastal areas can far exceed global ocean predictions. However, such acidification events could occur transiently, allowing for recovery periods during which the effects on growth would be compensated, maintained or amplified. Here we evaluated the recovery capacity of a model calcifier, the Pacific oyster Crassostrea gigas. We exposed juveniles to 15 pH conditions between 6.4 and 7.8 for 14 days. Oyster growth was retarded below pH 7.1 while shells were corroded at pH 6.5. We then placed the oysters under ambient pH > 7.8 for 42 days. Growth retardation persisted at pH levels below pH 7.1 even after the stress was removed. However, despite persistent retardation, growth has resumed rapidly suggesting that the oysters can recover from extreme acidification. Yet we found that the differences in individual weight between pH conditions below 7.1 increased over time, and thus the growth retardation cannot be compensated and may affect the fitness of the bivalves.

Keywords : bivalve, ocean acidification, compensatory growth, phenotypic plasticity, tipping point, recovery

1. Introduction

In the Anthropocene era, increasing anthropogenic carbon dioxide emissions are causing global ocean acidification (OA) at a pace unprecedented in the last 300 million years [1]. OA induces a reduction in seawater pH and a major shift in carbonate system equilibrium [2]. Such rapid changes will challenge the adaptability of marine organisms, especially calcifying species that precipitate calcium carbonate to make their exoskeleton and shell [3,4]. To date, the effects of near-future OA as predicted in the global surface ocean, *i.e.* a reduction of 0.16/0.44 pH unit under the low and high-emission scenario respectively [5], are overall neutral, often negative, but rarely positive on the physiology and shell/exoskeleton integrity of marine calcifiers [6].

Many calcifying species live in coastal areas where marked fluctuations in pH and carbonate chemistry are already occurring naturally due to local metabolism, freshwater inputs, eutrophication and upwelling, and often exceeding levels expected by the end of the century (e.g. [7–9]). These acidification events are however transient, varying from nychthemeral to seasonal [10]. It therefore provides an opportunity for these organisms to recover from exposure to suboptimal conditions. However, the intensity, frequency and duration of these events are expected to increase with climate change [11,12], which will challenge recovery capacity.

During stress events, marine organisms often experience growth depression by diverting energy from somatic growth toward the maintenance of homeostasis, ultimately resulting in growth retardation [13–15]. Compensatory growth, defined as the acceleration of growth of individuals due to the return of optimal living conditions after a period of stress [13], is probably the most studied recovery response in aquatic organisms [14,15]. It represents an evolutionary advantage in variable environments for species whose fitness is related to size and growth rate [16,17]. This physiological mechanism has been studied mainly in fish and crustaceans exposed to food restriction or deprivation [14], and more occasionally in marine calcifiers facing starvation [18,19], hypoxia [20] and ocean acidification [21–25]. Research on nutritional physiology

reveals that compensatory growth can be partial, *i.e.* when individuals exhibit accelerated growth without reaching the weight/size of unstressed ones, or complete, *aka* individuals' catch-up growth [13], or no longer possible, beyond a certain stress intensity or duration threshold [14,18]. Here we investigate if such a threshold of irreversibility exists in the context of ocean acidification.

Our study focuses on the recovery capacity of the juvenile oyster, *Crassostrea gigas*, a keystone calcifying species exploited worldwide, in response to ocean acidification. We specifically investigated compensatory growth because growth impacts oyster vulnerability to predation [26], disease resistance [27,28] and thus aquaculture yields. This experiment is a continuation of that of [29] where we studied the effects of 15 pH conditions ranging from 7.8 to 6.4 for 23 days of exposure and showed that oyster juveniles exhibited a pH tipping point at 7.1 below which growth was retarded. Here, we assess the reversibility of these effects by placing pre-exposed organisms back in ambient pH condition. We modeled weight gain as a function of time and exposure pH, and we test whether growth retardation is reversible, compensated or persistent.

2. Material and methods

(a) Exposure to 15 acidification conditions

The pH manipulation protocol and the origin of the oysters are fully described in [29]. Briefly, 15 batches of oysters, each containing 292 ± 20 five-month-old individuals, were exposed to pH levels ranging from 7.8 to 6.4 (on the total scale; -0.1 pH unit difference between each levels), without replicates. The exposure was carried out in 15 independent flow-through experimental units where seawater was acidified by pure CO_2 bubbling. Oysters were fed *ad libitum* with live phytoplankton (mix of *Isochrysis affinis galbana* and *Chaetoceros gracilis;* 1:1 in dry weight), photoperiod was 10h light:14h dark and temperature was 22°C. On January

18, 2019, each oyster batch was randomly assigned to one tank and held at ambient pH for 3 d. Then, pH was progressively decreased (-0.2 pH unit d^{-1}) in each tank. The decrease in pH lasted for 7 days in the lowest condition. Experimental pH conditions were all reached on January 27, 2019, corresponding to the first day of the exposure period. Oysters were exposed to these constant pH levels until day 14 while holes appeared in the shell at the lowest pH conditions (pH 6.4-6.5). The physiological damages were then evident, and we chose to return the oysters to ambient pH after 14 d of exposure to test their recovery capacities.

(b) Return to ambient pH after exposure period

At the end of the exposure period, 46 ± 1 individuals per pH condition were transferred into a 500L-tank in "common garden" for 42 days where they were kept separated in tagged baskets. The seawater circulated in open flow and was completely renewed every 80 minutes. pH was ambient depending on the seawater inflow while temperature, photoperiod and phytoplankton diet remained controlled as previously described. Airlift ensured oxygenation and mixing of seawater. Each basket was randomly moved twice a week to avoid any "position-effect".
No mortality was recorded during both experimental periods.

(c) Seawater carbonate chemistry

The complete protocols for determining seawater physico-chemical parameters are described in [29] and followed standard procedures in OA research field [30]. Temperature, salinity, oxygen saturation and pH (on the total scale) were measured twice a day and every 2 days during the exposure and ambient pH periods respectively. For the titration of total alkalinity, seawater samples were collected 2 times (days 2 and 10) and 4 times (days 7, 18, 30 and 39) during the exposure and ambient pH periods respectively. Carbonate chemistry parameters were

111 then calculated from pH, total alkalinity, salinity and temperature using *seacarb* package on R
112 software v4 0.3 (Table 1).
113
114 **(d) Biometry**
115 The oysters from each pH condition were briefly dried with absorbent paper and the total wet
116 body weight was measured with a Mettler precision balance (Mettler-Toledo) during the
117 exposure period on days 6, 10 and 14, and then during the ambient pH period on days 4, 7, 9,
118 11, 14, 18, 22, 28, 36 and 42. The mean individual body weight, thereafter designed as "weight",
119 was calculated as:
120 $W = \frac{TW}{n}$,
121 where W is the mean individual body weight (g), TW is the total weight of the batch (g) and n
122 is the number of individuals in the batch.
123
124 **(e) Statistical analyses**
125 The procedure of statistical analysis is fully detailed in [29]. The R software v4.0.3 was used
126 and the threshold of statistical significance was set at 0.05. The relationship between the
127 dependent variables (weight, weight difference) and the time or exposure pH was computed
128 using linear or piecewise regression models that were compared using information criteria and
129 verified for assumptions.
130 First, we used piecewise linear regressions of total body weight *vs.* exposure pH to estimate a
131 tipping point for each day of measurement (Fig. 1, top panel), defined as the value of the factor
132 (pH) where the dependent variable tipped. We considered the first day of the ambient pH period
133 as day 0 and therefore the experiment ran from day -14 to day 42. The significance of each
134 slope was tested using Student's *t* test (File S1). All regression models exhibited a pH tipping
135 point above which slopes (factor > tipping point) were not significant.

Thus, we assessed compensatory growth only below the tipping point (factor < tipping point) by computing weight differences across pH levels (g pH^{-1}, dependent variable), given by the slopes, as a function of time using linear regression models (File S1; Fig. 1, lower panel). We expected that (1) the weight differences below the tipping point would gradually fade after returning to ambient pH showing signs of compensatory growth (Fig. 1A), or that (2) weight differences would increase indicating persistent growth retardation (Fig. 1B). Theoretical models corresponding to these hypotheses were constructed from the data of [29].

3. Results

For each day of measurement, the regression models exhibit a tipping point at pH 7.12 ± 0.01 (average ± standard error). At pH levels above the tipping point, the slopes are not significantly different from zero, whereas below the tipping point, the slopes are significant and the average weight of oysters decreases sharply as the pH drops (Fig. 1C, top panel; File S1). After returning to ambient pH, the average weight of oysters exposed to conditions below the tipping point increases with time, *i.e.* from day 0 to 42 (Fig. 1C, top panel). However, the growth trajectories do not converge with the slopes becoming steeper with time. This is shown by the weight differences between individuals (g pH^{-1}) that increase linearly over time during both the exposure and common garden periods (Fig. 1C, lower panel). Therefore, there is no evidence of compensatory growth and our results (Fig. 1C) fit with the "irreversible growth retardation" theoretical model (Fig. 1B).

4. Discussion

Intertidal organisms face frequent environmental fluctuations, and are therefore interesting models for studying environment-induced plasticity in response to global change [31,32]. In particular, sessile bivalves must cope with the surrounding environmental conditions and therefore be able to respond and recover adequately from suboptimal conditions. Recently, we

have shown that *C. gigas* juveniles are tolerant of a wide range of acidification levels, exhibiting a growth tipping point as low as pH 7.1 [29]. Here, we decipher the plasticity of oysters' growth when they return to ambient pH conditions and assess both the persistence of effects and their capacity for compensatory growth.

Specifically, we previously identified that *C. gigas* growth was retarded below pH 7.1 after 23 days of exposure and stopped completely at pH 6.5, while shells were corroded and metabolic rates decreased, coinciding with a major rearrangement of membrane lipids and transcriptome [29]. This likely represents growth depression that involves sparing energy from somatic growth and redirecting it toward maintaining homeostasis under stressful conditions [13,33]. Here, we show that the growth retardation below the tipping point at pH 7.1 is demonstrated after just 14 days of exposure and persists for subsequent 42 days after the oysters return to ambient pH seawater. These results are the first evidence that short-term exposure to OA has lasting effects on the growth of juvenile oysters. Within the distribution range of the Pacific oyster, animals live in coastal areas where the pH varies naturally between 7.8 and 8.3 throughout the year (*e.g.* high-frequency monitoring of pH on the French coasts [34]), but the pH can drop abruptly and transiently with a minimum value of 7.15 recorded at the northern edge of the range, *e.g.* Kiel bay [8,35]. However, the average pH of the surface ocean is projected to decrease by -0.16/-0.44 unit by 2100 [5], and episodic acidification events are expected to be even more frequent and pronounced in coastal environments [11,12]. It can therefore be expected that OA will, at least transiently, exceed the pH tipping point of the species in some areas and sustainably impede the growth of juvenile oysters. However, the lack of projections available for ocean acidification in coastal areas limits the interpretation of our results [31,32].

Interestingly, the results also show that the return to ambient pH allows the oysters, previously exposed to pH levels < 7.1, to resume growth suggesting that they can recover from an extreme

186 acidification episode. The end of growth depression is expected when stress is removed, as
187 energy trade-offs are no longer required to maintain homeostasis, as shown in fish [13].
188 However, the weight differences of oysters recorded between all pH levels amplified over time,
189 including after cessation of acidification exposure. This indicates that the growth retardation is
190 not compensated and even increases during the experimental duration. Overall, compensatory
191 growth capacity is known to be species-specific [14,15], although it has been little studied in
192 the context of OA. For instance, the geoduck, *Panopea generosa*, exhibits compensatory
193 growth, or even overcompensation in shell length/area relative to the controls, at the larval and
194 juvenile stages when returned to ambient pH conditions after exposure of several days to pH
195 7.0-7.3 [23–25]. In contrast, there is no catch-up growth in Olympia oyster, *Ostrea lurida*, post-
196 larvae after being exposed during the entire larval cycle to pH 7.8 [21], or in hard clam
197 (*Mercenaria mercenaria*) and bay scallop (*Argopecten irradians*) larvae after short-term
198 exposure to moderate OA [22]. It is noteworthy to mention that the inability of compensatory
199 growth we observed in *C. gigas* has already been highlighted in larvae and juveniles after
200 dietary restrictions [36,37]. This lack of compensatory growth could result in major fitness loss
201 by increasing the vulnerability of oysters to predation [26] and disease associated with *Ostreid*
202 *herpesvirus* [27,28]. As a result, fitness loss and the increased time to reach marketable size
203 will have direct economic consequences for shellfish farmers in the future.
204 Finally, the difficulty in achieving growth compensation in *C. gigas* echoes studies showing
205 that beyond a certain duration/severity of food restriction, the induction of compensatory
206 growth is no longer possible [18,38]. The authors have shown that this point of no return is
207 reached when organisms show excessive growth retardation or alterations in digestive
208 functions, such as hyperphagia and feed conversion efficiency, which are growth accelerating
209 drivers [14,38]. In general, compensatory growth comes at a significant cost to the organism,
210 whose energy expenditure to recover from physiological damage might primarily be directed

toward maintaining immediate survival rather than catching up on growth [14,38]. Indeed, we observed shell drilling in oyster juveniles under the most extreme pH levels tested here (pH < 6.6), which completely disappeared after bathing in seawater at ambient pH (personal observation). This could suggest an active repair of the shell at the expense of growth. This trade-off hypothesis should be tested by quantifying the recovery of other physiological parameters, such as the net calcification rate.

In conclusion, we highlight that short-term exposure to extreme pH levels has lasting impacts on the growth performances of *C. gigas* juveniles, even after the stress is removed. Despite the apparent robustness of this species to withstand a drop in pH to 7.1, the inability of compensatory growth suggests a low resilience of *C. gigas* to severe acidification episodes. Our results are of concern because extreme OA events may exceed this pH tipping point and become more frequent in intertidal habitats with climate change. Ultimately, ongoing changes in ocean pH and carbonate chemistry will challenge the adaptability of marine species [39] and lead to both irremediable ecological changes and adaptation strategies for aquaculture.

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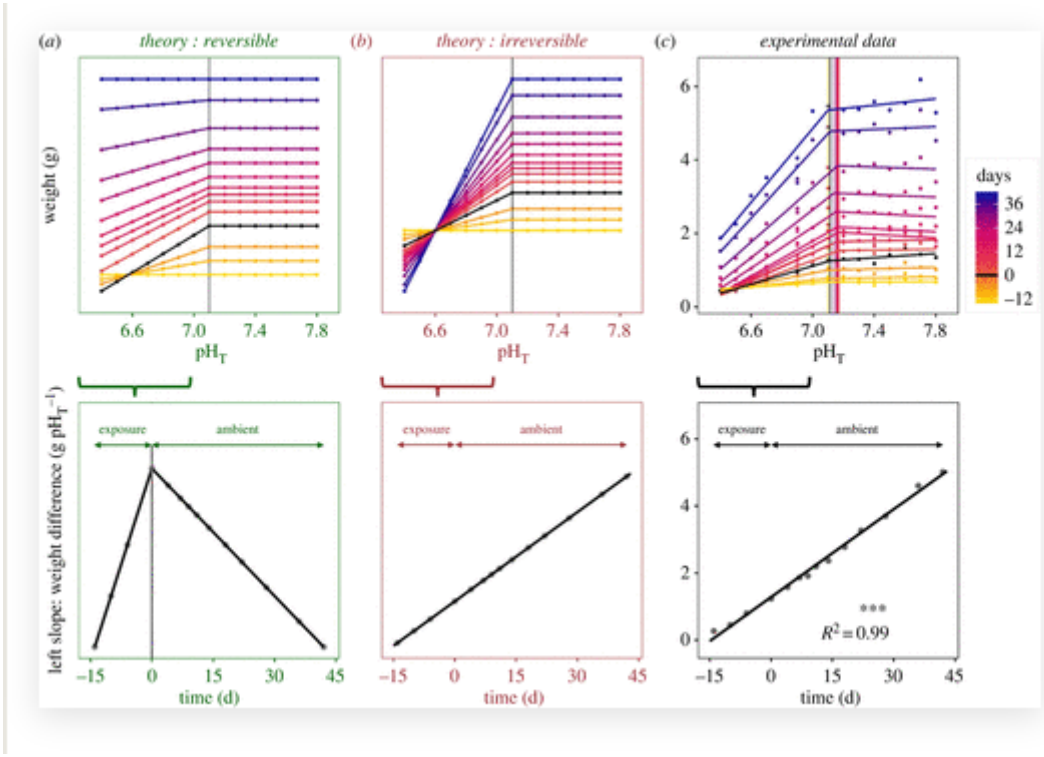
352 SEANOE https://10.17882/95774

Figure caption

Figure 1. (A-B) Theoretical models whether (A) growth retardation is reversible and full compensatory growth occurs by the end of the respite period, (B) growth retardation is irreversible and no compensatory growth occurs, and (C) experimental data. (Higher panel) Piecewise linear regressions of body weight as a function of exposure pH. The colour gradient represents the time, before and after day 0 of the return to ambient pH, which is highlighted in black. Vertical full lines represent tipping points for the different models. All the slopes above tipping point does not significantly differs from zero according to student t tests. (Lower Panel) The slopes of the piecewise linear regression models of body weight as a function of exposure pH represent the weight differences between individuals that were exposed to pH lower than tipping point, and are displayed as a function of time. Tipping points are indicated by a vertical line when they exist. For experimental data, the R squared and the significance levels of the slopes are provided ($P < 0.001$ ***).

Figure 1


(a)
theory : reversible
(b)
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(c)
experimental data
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$R^2 = 0.99$