Ecological Complexity 8 (2011) 320–325

Contents lists available at ScienceDirect

Ecological Complexity

journal homepage: www.elsevier.com/locate/ecocom

Warming alters density dependence, energetic fluxes, and population size in a model algae

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ARTICLE INFO

Article history:
Received 23 February 2011
Received in revised form 5 July 2011
Accepted 12 July 2011
Available online 4 August 2011

Keywords:
Density dependence
Scaling
Population energy use
Metabolism
Life history

ABSTRACT

A fundamental question in ecology is what determines the density of a population. Many populations may grow to a maximum density which is set by the interplay of resource availability and per capita resource requirements. Resource availability is determined in part by the kind of interactions occurring within a population, such that the total resource acquired by a population is a function of its size. Often taking the form of a power function with a scaling parameter <1, this scaling imposes density dependence on resource acquisition and metabolic rates of individuals. An increase in the scaling parameter signifies a reduction in density dependence that would allow a population to grow past its previous limits. We evaluated this possibility using laboratory populations of the algae *Chlamydomonas reinhardtii* grown at cold (20 °C) and warm (25 °C) temperatures in semi-continuous batch cultures. Warm populations rapidly developed larger cells and grew to a higher population density and total biovolume than the cold populations. Furthermore, the density dependence of metabolic rate was more severe in the cold, and for a given metabolic rate cells in the cold populations divided more quickly. These results suggest that a temperature-dependent life history set the strength of density dependence in these populations. Increasing temperature led to an increase in the resource scaling parameter and enabled the warm populations to escape the limits experienced by the cold populations.

1. Introduction

All populations are limited in some way. They may be limited intrinsically, by competition for resources within the population, or extrinsically, by a competitor, a predator, a disease, or an abiotic disturbance (Pearl, 1927; Turchin, 1999). Populations of many kinds of organisms, including algae, bacteria, insects, plants, and humans, occasionally escape their limitations and grow to a larger size, sometimes at a fast rate. Relaxation of both intrinsic and extrinsic regulating factors may stimulate such increases.

Intrinsically regulated populations reach maximum density when all of the available resource is used by the individuals in the population. We will define this maximum density, N_{max}, as the steady-state density of a population when limited by resources. If no extrinsic factors come into play, populations grow to N_{max}, and the density dependence is determined by how density affects access to resources. We can relate this to resource use by recognizing that the steady state (indicated by '^') level of resource use by a population at N_{max}, $\widehat{R}_{pop}$, divided by the steady state level of per capita resource use at N_{max}, $\widehat{R}_{pc}$, gives the value of N_{max}:

$$N_{max} = \frac{\widehat{R}_{pop}}{\widehat{R}_{pc}} \quad (1)$$

Eq. (1) suggests two ways in which N_{max} can be increased: increase the amount of resource available to the population or decrease the per capita use of the available resource. The former can be seen in cases where increasing limiting resources boosts population sizes (Taylor, 1977; Pettorelli et al., 2009). The latter is subject to a variety of interacting effects. For example, increasing environmental temperature might be expected to decrease N_{max} because it increases individual resource demands through the kinetic effect of temperature on metabolic rates (Brown et al., 2004; Marquet et al., 2004). Yet for some organisms, increasing temperature also may reduce body size (Atkinson, 1994), potentially counteracting the effect. Clearly, there are many factors that may alter $\widehat{R}_{pc}$ and change N_{max}.

A further complication is that resource uptake and density interact in a non-linear way. For a growing population, the total level of resources used by the population, R_{pop}, increases as individuals, N, are added to the population. However, R_{pop} may not increase at the same rate as the number of individuals, leading to a scaling of R_{pop} with N that can be described by a power function:

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doi:10.1016/j.ecocom.2011.07.001

$R_{pop} \propto N^{\beta}$ (DeLong and Hanson, 2009a,b). The exponent β is a measure of the strength of density dependence. A smaller β indicates more intense density dependence, and a higher β means less intense density dependence. Most importantly, for any level of N, a higher β means a higher R_{pop}, and a relaxation of the constraint in Eq. (1). A powerful way to increase N_{max}, then, is to increase β.

Population growth is the outcome of birth and death processes occurring at the individual level. Thus, understanding density dependence requires investigating how individuals in the population are affected by β and asking how demographic and life history traits respond to the limitations imposed by the population. Rearranging Eq. (1) and defining α as $\beta - 1$, we see that average per capita resource availability is density dependent in a similar way:

$$\bar{R}_{pc} \propto \frac{N^{\beta}}{N} \propto N^{\alpha} \quad (2)$$

The exponent α defines the rate at which the average per capita resource availability, $\bar{R}_{pc}$, changes with density. The resources afforded to individuals in the population drive the metabolism of those individuals such that

$$\bar{B}_{pc} = cN^{\alpha} \quad (3)$$

where $\bar{B}_{pc}$ is the average per capita energy use, or metabolic rate, of an individual and c is a constant converting resources to metabolism. This type of non-linear density dependence has been found previously for protozoa, algae, and small aquatic invertebrates in experimental cultures, with α ranging between 0 and -1 (Hoshi, 1957; Nässberger and Monti, 1984; DeLong and Hanson, 2009a,b). This constraint on energy use will drive the changes in birth and death rates that alter population growth rate, because energy must be allocated to reproduction and survival and any constraint on total energy use will constrain these processes as well (DeLong and Hanson, 2009b). Thus, the change in N_{max} associated with a change in α stems from the effects of the scaling of resource acquisition on the energy budgets of individuals. This is one way in which density dependence may change, but the process has not been investigated empirically.

In this study, we attempt to understand whether temperature can alter the density dependence of a population via some effect on the scaling parameters α and β. We grew replicate microcosm populations of wild-type *Chlamydomonas reinhardtii* at two temperatures (cold, 20 °C and warm, 25 °C) and measured the energetic, demographic, and life history responses to the temperature change. The data illustrate the direct and indirect effects of temperature on algal population growth, and clearly illustrate that changes in density dependence can result in a major increase in population size.

2. Materials and methods

We grew wild-type *C. reinhardtii* populations (from UTEX: http://web.biosci.utexas.edu/utex/) in axenic, semi-continuous laboratory microcosms. *Chlamydomonas* can show phenotypic responses in metabolism to changes in environmental conditions and is therefore a good model for investigating this hypothesis (Collins and Bell, 2004). There were four replicate populations at each temperature. Microcosms were 10 mL cultures of TAP medium (Harris, 1989) in 16 mL vials sealed with septum tops. Micro-filtered air (using a 0.2 μm filter) was bubbled through the microcosms via vial-length needles, and bent needles vented the headspace. We initiated replicate populations in two stages. First, we inoculated a single test tube containing TAP medium directly from plated algae cultures and allowed cells to grow and acclimatize for one week. Second, we sub-cultured from this population to a fresh tube and allowed the culture to grow for one day. At that time, we began the experimental cultures with 1 mL of this new culture added to 9 mL of fresh TAP medium. Experiments lasted six days in the warm treatment (25 °C) and seven days in the cold treatment (20 °C). We removed 0.5 mL of culture daily and replaced it with 0.5 mL of fresh TAP medium. Any evaporated water was replaced with filtered, autoclaved water and allowed to mix prior to extraction of samples. All additions to the microcosms were made through 0.2 μm filters to maintain sterility. Microcosms were grown under 24-h light conditions, at an approximate average irradiance of 100 μmol s^{-1} m^{-2} within the microcosm vials. There was no indication of bacteriological contamination of the cultures.

We used a fluorescent oxygen needle probe (FOXY-18G with overcoat, Ocean Optics Inc., Dunedin, FL, USA) to measure metabolic rates as oxygen consumption (see DeLong and Hanson, 2009a, for more details). In short, the probes detect the decline in molar fraction of dissolved oxygen due to metabolic activity in the sample. We transferred ~0.3 mL of the daily 0.5-mL sample into a 0.3-mL micro-centrifuge tube modified with a small opening at the tip for insertion of the needle probe. After inserting the oxygen probe, the needle was sealed to the sample tube by pressing a piece of tacky rubber around the opening. Measurements were made in the dark, with aluminum foil wrapped around the tube.

The cell density of each sample was determined by repeated counts of cells in a hemocytometer, with dilutions if necessary. Subsamples from the daily 0.5-mL sample were used for counts. Growth rates were calculated as the change in density divided by the change in time, and converted to per capita rates by dividing by the initial density in the time frame.

The number of mitotic cell divisions occurring during all counts for a sample was recorded and divided by the total number of counted cells to give the proportion of cells dividing. Evidence of a division included late-division mitosis (dividing cells still connected), but in *Chlamydomonas*, cells may divide more than once before bursting out of cell walls. Thus, we observed sets of dividing cells, and counted one division for every two cells within the cell wall. This division index can be scaled to the population growth rate to produce the division rate of the population. The rescaling is necessary because there is no *a priori* time frame for the division index, and rates require a unit of time. We made the conversion by calculating the actual growth rate between time steps during the growth phase of the time series, and fitting an exponential curve to the declining growth rates. Then we plotted the growth rate curve over the actual division index data, and rescaled the index data (adjusted the intercept) until it matched the magnitude of the growth rates. This works because in the early growth phase of these populations, death rates are negligible, and the changes in population size are due primarily to division. All results and discussion refer to the rescaled division rates, not the original index.

We measured the sizes of cells by photographing them with a digital microscope and measuring the cell diameters with the microscope's software application. Volume was calculated using the formula for a sphere.

We analyzed the relationships between time and density, between density and metabolic rates, and between metabolic rate and division rate using linear mixed models. Models were implemented using the *nlme* library (Pinheiro and Bates, 2000) in R version 2.11.1 (R Development Core Team, 2010). We controlled for autocorrelation within experimental units by including a random effect in the models for the replicate-treatment combination. The effect of temperature on the growth, metabolic, and division rates of the populations is indicated by the interaction term between temperature treatment and the main factor. We used log transformations on density and metabolic rate data but used untransformed data for the division index.

3. Results

Algae populations grew to higher densities in the warm than in the cold temperatures (Fig. 1a). The slope of the increase in growth was significantly greater in the warm cultures than in the cold cultures ($t = 4.24$, d.f. = 22, $p < 0.001$), and the warm populations achieved densities that were about twice those of the cold populations at their highest points. The magnitude of the increase was more apparent in terms of biovolume than in terms of cell number (Fig. 1b), with the warm population achieving a 10-fold increase in biovolume at their highest points. This was because warm cells were bigger than cold cells ($t = 19.7$, d.f. = 13, $p < 0.01$; Fig. 2), leading to a larger difference between the populations in total biovolume than in density. Densities and biovolumes were higher at all times in the warmer cultures than in the colder cultures, except at the initial sampling. Population growth was sub-exponential in both the warm and the cold treatments, as indicated by declining per capita growth rates through time (Fig. 3). Mean growth rates were higher in the warm population than in the cold population early in the experiment, but rates converged as the experiments progressed and populations in both treatments stopped growing and began to decline.

Both warm and cold populations showed density dependent metabolic rates, with suppression of metabolic rates up to about two orders of magnitude (Fig. 4; $t = -7.05$, d.f. = 24, $p < 0.001$).

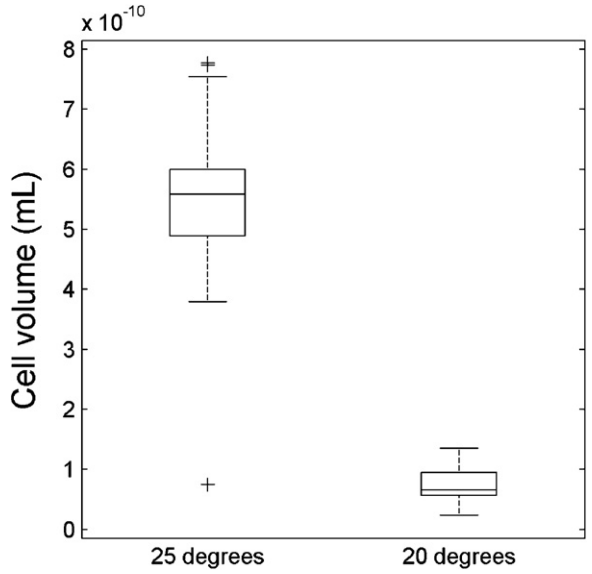

Fig. 2. Individual *Chlamydomonas reinhardtii* cell volume was five times larger in the warm populations than in the cold populations. Boxes show median and 25th and 75th percentiles.

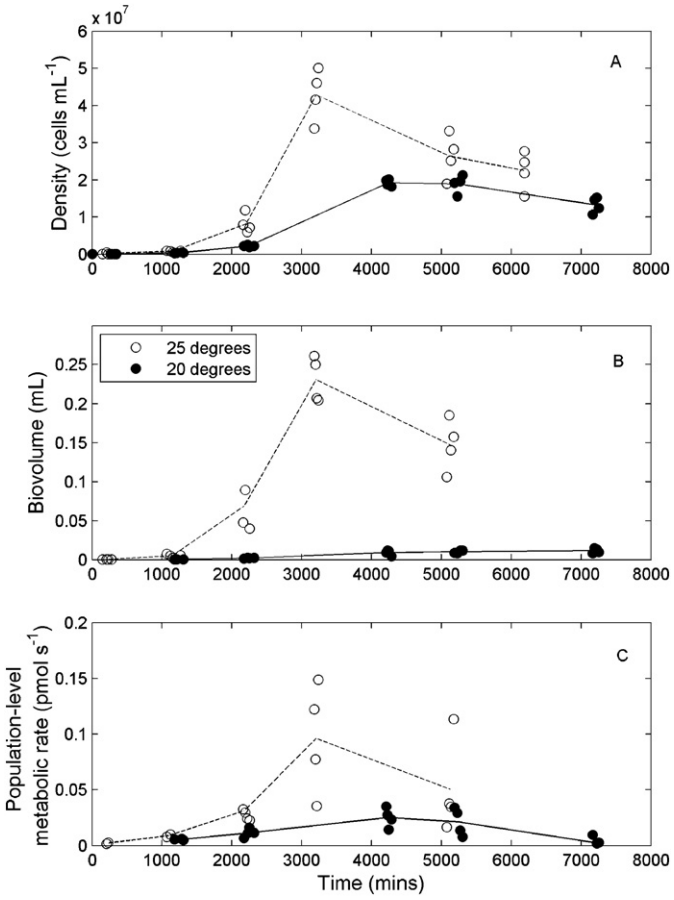

Fig. 1. Population density (a) and biovolume (b) changes of four replicate microcosm populations of *Chlamydomonas reinhardtii* grown at 20 and 25 °C. Both populations grew sub-exponentially to a peak and declined exponentially to the end of the experiment. (c) Population-level metabolism for warm and cold treatments through time, showing that warm populations reached higher overall levels of metabolism than the cold populations. Lines connect means across sampling days.

Metabolic rates were somewhat lower in the cold populations ($t = -2.33$, d.f. = 6, $p = 0.059$), but more importantly, the relationship between density and metabolic rate was steeper for cold-temperature populations than warm-temperature populations ($t = 2.64$, d.f. = 21, $p = 0.014$). Estimates of the scaling parameter α in Eq. (3) derived from the linear mixed-models were −0.40 for the warm population and −0.79 for the cold population. This significant difference in the density dependence of metabolic rate allowed warm populations to maintain higher per capita metabolic rates than cold populations over most density levels. At low densities, cells in both populations had similar metabolic rates, but as densities increased, the metabolic rate of cold cells decreased more rapidly than those of warm cells, indicating stronger density dependence in the cold cultures.

Division rates were positively dependent on the metabolic rate of the cells (Fig. 5; $t = 4.14$, d.f. = 24, $p < 0.001$). This relationship did not differ between the groups, as indicated by a non-significant

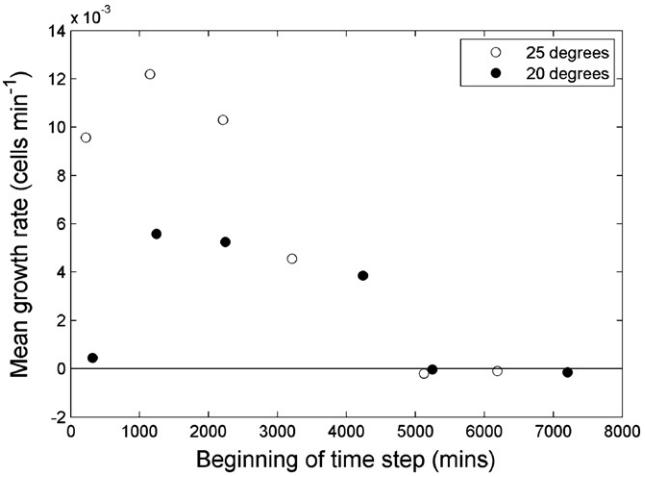

Fig. 3. Mean growth rates declined through the course of the experiment. Growth rates were higher in the warm population than in the cold population, especially in the beginning of the experiment, but converged as time went on.

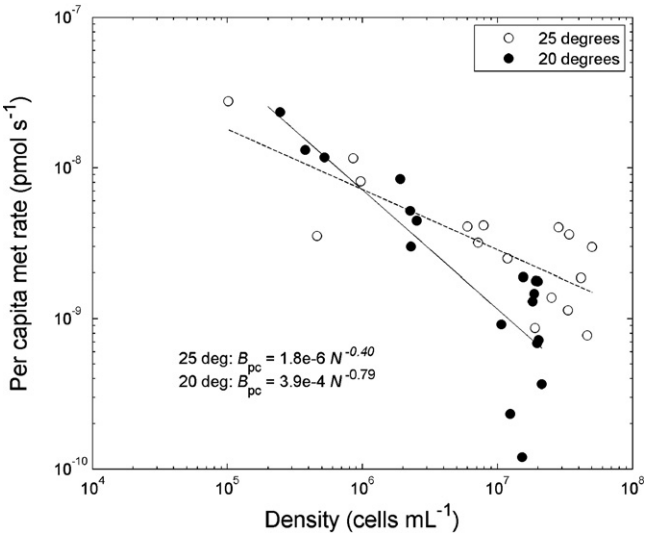

Fig. 4. Per capita metabolic rates were density dependent in both warm and cold populations. The magnitude of the density dependence was lower in the warm populations. Cells in the warm populations were able to maintain higher levels of energy flux even as the density climbed to twice that in the cold populations.

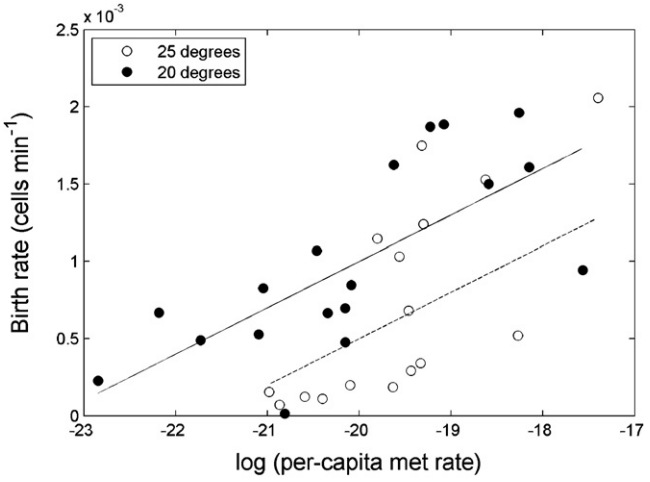

Fig. 5. Division (birth) rates were positively related to metabolic rates in both populations. The slopes were not different between the treatments, but cold populations had higher division rates for a given level of metabolism. This difference illustrates the life history trade-off in the cells.

interaction between metabolic rate and group (t = 1.13, d.f. = 24, p = 0.27).

Population-level metabolism climbed to higher levels in the warm populations than in the cold populations (Fig. 1c). This is what we expect from a difference in α and β, and greater access to resources afforded by the reduced density dependence should translate into larger population size and more biovolume. In this study, the warm population had both more individuals and more biovolume.

4. Discussion

One of the most fundamental problems in ecology is determining the nature of density dependence and how it influences population regulation in nature. Our results clearly illustrate that environmental changes can alter the magnitude of density dependence and lead to large increases in population size, independent of the many extrinsic regulating factors (such as predators or abiotic stressors) that also may change.

In this study, we compared energetic and demographic features of populations grown at two temperatures. Warm populations had an α that was roughly half that in the cold populations, indicating a substantial reduction in the rate at which cells were affected by their conspecifics (Fig. 4). This reduction in density dependence allowed cells in the warm population to maintain metabolic rates at higher levels even as density increased, sheltering individual cells from the effects of intra-specific competition until higher densities were reached. All else being equal, the reduction in α (or equivalently, the increase in β) facilitated an increase in N_{max}. Although increased temperatures would have increased metabolic rates (Gillooly et al., 2001), there was no overall difference between treatments, and standardizing to the same temperature involves only linear shifts not shifts in exponents. Therefore, this result does not stem from changes in the kinetic effect of temperature. In addition, for a given metabolic rate, cold populations had faster-dividing cells than warm populations (Fig. 5), indicating that warming did not simply speed up the production of new individuals.

We hypothesize that the reduction in density dependence in the warm populations relative to the cold populations was caused by a temperature-dependent life history change. One major life history decision is when to stop growing and reproduce. In this study, warming appears to have caused cells to grow larger than cold cells before dividing (Fig. 2). It is possible that the warmer temperature relaxed some regulating step in the acquisition or utilization of energy, allowing cells to grow larger yet still maintain their growth rates. And either size itself or a change in which nutrient was actually limiting may have altered the density dependence. Classically, we think of life history strategies as balancing components of fitness given a constraint on total allocation (Stearns, 1992). Here, it appears that cells were able to increase their size at a reduced cost to their rate of division, suggesting that the constraint on total energy availability was relaxed. This is indeed the case, as the warm population was able to use more total energy than the cold population, throughout the time series (again, a higher β, Fig. 1c). One might argue that this is merely a case of 25 °C being more optimal than 20 °C, but if that is true, then our results suggest that decreasing the strength of density dependence is part of what makes a particular temperature optimal.

Previous work has shown that nutrient limitations are involved in cell size and growth rate regulation in *Chlamydomonas*. Cell size in *Chlamydomonas* has been shown to increase with P limitation, suggesting the possibility that in the warm treatments, cells became P-limited and grew to a larger size (Dean et al., 2008). This effect is unlikely to explain the increase in cell size that we observed, because sizes increased by the end of the first day of the experiment (Fig. 2), not late in the experiment when nutrient limitations were likely to be stronger. Also, warm-population cells had greater metabolic rates per cell as density increased (Fig. 4), suggesting it was rather the cold populations that were more limited, either by energy (light) or nutrients. Similarly, diatom metabolic rates may be influenced by N availability, depending on its form (Goldman and Dennett, 1983), suggesting the possible role of N in facilitating the different levels of metabolism as mediated by population density. *Chlamydomonas* also alters its nutrient allocation patterns through the growth cycle (Healey and Hendzel, 1979), suggesting that temperature may have influenced these nutrient allocation patterns. Future work might ascertain the link between temperature and cell-level processes that would indicate how the change in density dependence arose via its effect on access to and utilization of limiting nutrients.

A recent hypothesis about density dependent metabolic rates is that it is driven both by resource levels that drop due to

exploitation competition and by interference competition where density reduces resource uptake independent of resource levels (DeLong and Vasseur, 2011). Many small aquatic organisms show interference competition, and it is typically seen as a decline in resource acquisition with increasing density, following a power law in form with scaling exponents between 0 and −1. Because resource acquisition declines in this fashion with density due to interference competition, it is logical that metabolic rates also would decline in this fashion, because resources fuel metabolism. This density dependence has been observed both when density is manipulated and when it changes via population growth, suggesting that exploitation and interference effects may both contribute to density dependent metabolic rates (DeLong and Hanson, 2009a,b; Nässberger and Monti, 1984). Many workers think of interference competition only in terms of aggressive interactions between individuals that occur at high density (Schoener, 1983), but interference often takes a passive form with large effects even at low density (Skalski and Gilliam, 2001; DeLong and Vasseur, 2011). Whether interference competition is actually operating in this algal system remains unknown.

This study provides a test of the predictions made by Brown et al. (2004) and Marquet et al. (2004) that increased temperature should decrease steady state population size because of the increase in metabolic demands for resources (Eq. (1)). Here we have shown that the theoretical framework for the prediction must be expanded to include resource-density scaling – the non-linear relationship between total resource amount accessed by a population and the size of the population. This relationship depends on scaling parameters which reflect the nature of the interaction among individuals. Without including resource-density scaling, our results would seem to be a violation of the conservation laws of mass and energy – how can per capita demand and population size both increase without increasing population-level access to resources? With resource-density scaling, it is revealed that there is a change in density dependence that facilitates an increase in access to resources.

Our results may have implications for understanding the temperature-size rule. The temperature-size rule is a pattern of decreasing size with increasing rearing temperature, which is widespread in many ectothermic organisms, including phytoplankton (Atkinson, 1994). Our finding of an increase in size with increasing temperature is one of many exceptions to the rule (Atkinson, 1995), and therefore may shed additional light on the phenomenon. The two main hypotheses suggested by (Atkinson et al., 2003) that small size is favorable at warmer temperatures because it aids in acquiring oxygen, which declines in solubility with increasing water temperature (see also Atkinson et al., 2006), or that it promotes Darwinian fitness by increasing representation of early-dividing cells in subsequent generations, seem to be not supported by our data. The amount of oxygen in our populations would not have increased with warming temperatures given the constant bubbling with air, but the cells got larger anyway. In addition, the warm population grew faster even though the warmer cells allocated a smaller fraction of their metabolism to division, indicating that they were not trading higher growth rates for smaller size. Rather, it seems plausible that a warmer temperature had a relaxing effect on other constraints, such as access to nutrients or light, which then altered the ontogeny of the cells and the decision to grow large before dividing. We suggest that the plasticity of the effect of temperature on size merits further attention, for our data indicate that there are population consequences of such life history shifts.

Temperature-induced shifts in population behavior may play a role in the response of food webs to climate change. O'Connor et al. (2009) showed that the effect of temperature on food web structure depends on resource availability. Increasing nutrient supplies allowed greater heterotrophic control of experimental pelagic food webs, through the differential scaling of autotrophic and heterotrophic metabolism with temperature. Our results show that temperature may alter density, biovolume, and metabolic fluxes without a change in environmentally supplied nutrient levels but rather with a change in how much of the resource could be accessed. Temperature changed the density dependence of *Chlamydomonas*, which had a large effect on population behavior that added to the direct effects of temperature on metabolism. Future work on food web responses to climate change might look for changes in life histories and altered patterns of density dependence.

This study highlights the need to understand the energetics of population phenomenon and points to unexplored potential interactions between organisms and their environment. Although not a widely appreciated phenomenon, the density dependence of metabolic rate played a large role in determining how these populations behaved. The difference between the cold and warm populations may have arisen from the temperature-dependence of algae life history, suggesting an interesting connection between environmental conditions, individual behavior, and population dynamics. With expected increases in global mean temperatures of a few degrees, the effect we describe could have wide ramifications for ecological processes.

Acknowledgements

This work benefited from discussions with James Brown and David Vasseur and several anonymous reviewers. Funds for this work came from UNM GRAC, SRAC, and a Grove Summer Scholarship to JPD. Funding was also provided by UNM research allocation committee Grant #05-L-07 and NMTRC funding to DTH.

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