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Density-Dependent Energy Use Contributes to the Self-Thinning Relationship of Cohorts

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ABSTRACT: In resource-limited populations, an increase in average body size can occur only with a decline in abundance. This is known as self-thinning, and the decline in abundance in food-limited populations is considered proportional to the scaling of metabolism with body mass. This popular hypothesis may be inaccurate, because self-thinning populations can also experience density-dependent competition, which could alter their energy use beyond the predictions of metabolic scaling. This study tested whether density-dependent competition has an energetic role in self-thinning, by manipulating the abundance of the fish *Macquaria novemaculeata* and tank size to partition the effects of competition from metabolic scaling. We found that self-thinning can be density dependent and that changes in intraspecific competition may be more influential than metabolic scaling on self-thinning relationships. The energetic mechanism we propose is that density-dependent competition causes variation in the allocation of energy to growth, which alters the energetic efficiency of self-thinning cohorts. The implication is that food-limited cohorts and populations with competitive strategies that encourage fast-growing individuals will have less body mass at equilibrium and higher mortality rates. This finding sheds light on the processes structuring populations and can be used to explain inconsistencies in the mass-abundance scaling of assemblages and communities (the energetic-equivalence rule).

Keywords: intraspecific competition, food limitation, metabolic scaling, energetic equivalence, self-thinning.

Introduction

The relationship between abundance and body size is an important scaling attribute in ecology (White et al. 2007). In resource-limited animal populations, abundance is expected to decline in proportion to gains in average body size (Bohlin et al. 1994). This is called self-thinning (Begon et al. 1986), and much research has been devoted to quantifying the precise scaling of the relationship (Hughes and Griffiths 1988; Armstrong 1997). The self-thinning relationship (STR) predicts the trajectory of a thinning population and is used to determine the structure and regulation of intraspecific animal cohorts (Dunham et al. 2000), to estimate carrying capacity (Armstrong 2005), and to predict the dynamics of populations (McGarvey and Johnston 2011; Smith et al. 2012). The relationship is well studied in situations where space (Steingrímsson and Grant 1999; Fréchette et al. 2010) or food (Bohlin et al. 1994; Keeley 2003) is the limiting resource.

Self-thinning theory originated in plant ecology (Yoda et al. 1963; Westoby 1981) and was soon applied to animal populations (Begon et al. 1986), but its suitability for mobile fauna is uncertain. Populations of mobile animals often deviate from the expected STR (Elliott 1993; Dunham and Vinyard 1997; Steingrímsson and Grant 1999), even when the STR is species specific (Grant and Kramer 1990; Bohlin et al. 1994). This deviation is sometimes attributed to ontogenetic changes in resource use that are not encompassed by the self-thinning model (Armstrong 1997; Steingrímsson and Grant 1999). Animals can also alter their resource use in response to a declining density of competitors, however, and the contribution of this density-dependent resource use to self-thinning is unknown. Quantifying this contribution is an essential step in identifying the processes by which competition structures populations of mobile animals.

When food is the limiting resource, the shape of the STR is typically determined through the scaling of metabolism with body mass (Begon et al. 1986). Self-thinning theory states that because metabolism is proportional to body mass$^{\beta}$ (a relationship known as metabolic scaling),

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abundance (N) decreases with body mass (M) at the rate of $-\beta$ (Bohlin et al. 1994). Thus, in a food-limited animal population with a constant food supply, $N = aM^{-\beta}$ (where a is a constant). In mobile animals, β is often considered equal to 0.75—known as Kleiber's rule (Kleiber 1947). In experimental studies that manipulate abundance, body mass M should be the dependent variable, which gives $\log M = -\beta \log N + \log a$ (fig. 1). In this case, the reciprocal of 0.75, which equals 1.33, is used (β is also used to represent the reciprocal). If density-dependent competition is likely in self-thinning populations, then the scaling of energy use with density would cause β to deviate from the value expected from metabolic scaling.

Density-dependent energy use could alter β through numerous mechanisms. An assumption of self-thinning is that the amount of energy available is constant. Thus, it is not the amount of energy that a cohort receives that determines the STR but how the constant energy is allocated (among metabolism, activity, and growth) or how efficiently this energy is used. For example, a cohort that allocates a larger proportion of available energy to nongrowth components, such as activity, will become food limited at a smaller body mass. Alternatively, a cohort that uses energy more efficiently will reach a larger equilibrium body mass. One likely way in which density dependence could alter β is if a cohort changes the allocation of energy toward growth as it thins, because this can influence overall energetic efficiency (Arendt 1997).

Density dependence has been discussed in the context of self-thinning (Armstrong 1997; Dunham and Vinyard 1997), but only in reference to the density of animals sharing a resource and without conclusively demonstrating that mortality was caused by resource limitation (Lobón-Cerviá and Mortensen 2006). The effect of density-dependent competition can be discerned by examining two distinct metrics of abundance: "population size," the number of animals sharing a resource, and "population density," the number of animals per unit space. In mobile animals, population density can vary independently of population size, a rarely made distinction that may be crucial for determining the mechanisms behind self-thinning. STRs can be measured as the abundance-mass trajectory of a self-thinning population or cohort, or it can be estimated experimentally according to the trajectories of numerous populations of different abundance up to the point of food limitation, after which self-thinning occurs (fig. 1). In either case, a change in population size represents a proportional change in the population density. A change in the number of interactions between individuals per unit space or time ("competition") as density changes is the primary cause of density dependence. If density dependence is to be accounted for in the STR, then population size must be varied while a constant population density is maintained. In an experimental setting, this is achieved by varying the arena size.

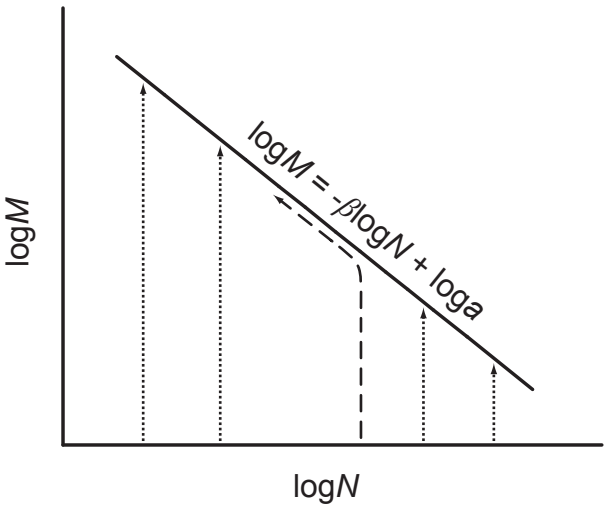

Figure 1: The self-thinning relationship (STR), where M is average body mass, N is abundance, and a is a constant. Traditional food-limited self-thinning states that β is proportional to the scaling of metabolism with body mass. The STR can be determined by following a cohort's trajectory as it becomes food-limited (dashed line), which is the process known as self-thinning. Alternatively, the STR can be estimated by following multiple cohorts until the initial equilibrium before self-thinning begins (dotted lines), which was the method used in this study. Body mass is considered the dependent variable in this study because abundance was the manipulated variable, but these axes are sometimes reversed (e.g., Bohlin et al. 1994; Keeley 2003).

This study tested whether density-dependent competition contributes to self-thinning, by manipulating the abundance in cohorts of fish (Australian bass *Macquaria novemaculeata*) in tanks. Tank size was manipulated in one experiment to maintain a constant density of fish per liter (while still varying cohort size), to measure a density-independent food-limited STR as predicted by metabolic scaling. Non-food-limited cohorts were maintained alongside the self-thinning cohorts to develop density dependence relationships (DDRs). This was done to examine how the pattern of density-dependent intraspecific competition (slope b of the DDR) influenced the slope (β) of the corresponding STR (fig. 2). Swimming speed and foraging activity were measured during the experiments to provide a mechanistic explanation of how density-dependent competition affected energy use. This study had two main predictions: (1) that the STR has a density-dependent component and that a change in β will occur if density is experimentally controlled and (2) that density-dependent competition drives this difference, which will be observed by a relationship between the β of the STR and the strength of density-dependent competition (the b of the DDR).

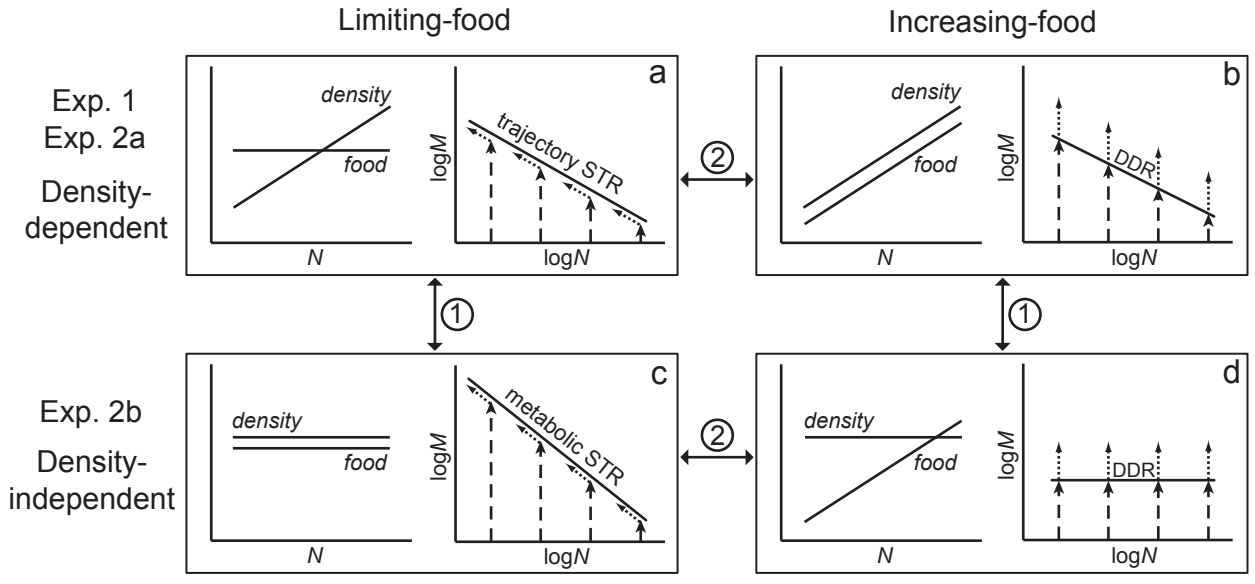

Figure 2: The four treatments manipulating density and food ration and the expected output from each treatment (STR = self-thinning relationship; DDR = density dependence relationship). The trajectories of fish populations (dashed lines) were measured until the STR and DDR could be established. The trajectories expected if the experiments continued are displayed (dotted lines). Comparisons between experiments and feeding treatments (limiting-food, increasing-food) needed to test this study's predictions are numbered. To test whether removing density-dependent competition alters the STR, the feeding treatments were compared between experiments 2a and 2b (prediction 1). To test for a relationship between the strength of density-dependent competition and the STR, the feeding treatments were compared within each of the three experiments (prediction 2). Tank size was consistent within experiments 1 and 2a, so that cohort density increased with cohort size (*N*; *a*, *b*), but was manipulated in experiment 2b to create a constant starting cohort density (*c*, *d*). Food was limiting and static in the limiting-food treatment (*a*, *c*) and nonlimiting and increasing at a constant rate in the increasing-food treatment (*b*, *d*). It was expected that making density constant would prevent density-dependent growth (cf. *d* and *b*), allowing the density-independent (or "metabolic") STR to be measured (cf. *c* and *a*).

Methods

Experimental Design

To test the predictions of this study, two feeding treatments were required in each experiment (table 1): (1) a food ration that remained constant during the experiment and would become limiting as fish accumulated body mass (to examine the self-thinning relationship [STR]; hereafter the "limiting-food" treatment) and (2) a food ration that increased during the experiment as a consistent percentage of the average fish body mass (to examine the general pattern of density dependence without food limitation; hereafter the "increasing-food" treatment). Experiments 1 and 2a produced "trajectory" STRs (in which cohort size and density simultaneously decline) and provided a comparison of the β value of the STR with the b value of the density dependence relationship (DDR; fig. 2). Experiment 2b was performed with the same objectives but with tank size varied to test the same cohort sizes (fish tank^{-1}) as experiment 2a with a constant cohort density (fish L^{-1}). Experiment 2b was designed to remove density-dependent intraspecific competition and thus allow the estimation of the density-independent "metabolic" STR (the product of metabolic scaling only). The increasing-food treatment was replicated in experiment 2b to test whether the design successfully removed density-dependent growth. The treatments and the comparisons between treatments and experiments needed to test the study's predictions are detailed in figure 2.

The starting density chosen in experiment 2b was 0.4 fish L^{-1} (table 1). This was an arbitrary value, but it was chosen to meet the assumption that the cohort sizes (fish tank^{-1}) were functionally equivalent between experiments 2a and 2b. Cohort size is defined as the number of fish sharing a resource; therefore, if cohort sizes were to be comparable between the two experiments, the tank volumes had to be similar enough to meet the assumption that fish cohorts of equal sizes had access to the entire food resource regardless of tank volume. Tank volume for 5– and 10–fish tank^{-1} cohort sizes was reduced and the tank volume for 40–fish tank^{-1} was increased in experiment 2b (table 1). The tanks with 20–fish tank^{-1} cohorts were used in experiments 2a and 2b, as they intentionally met the requirements of both experiments.

Table 1: Experimental design, showing the food rations provided as a percentage of average body mass to examine density dependence (increasing-food treatment) and the fixed food rations provided to examine self-thinning (limiting-food treatment)

Experiment: conditions; food	Food ration: Increasing-food treatment (% body mass d^{-1})	Food ration: Limiting-food treatment (g $tank^{-1}$ d^{-1})	Tank volume (L)	Cohort size (fish $tank^{-1}$)	Cohort density (fish L^{-1})
Experiment 1:					
constant volume, variable density; *Artemia*	30	.5	100	100	1
	30	.5	100	50	.5
	30	.5	100	20	.25
	30	.5	100	10	.1
Experiment 2a:					
constant volume, variable density; pellets	4	.6	50	40	.8
	4	.6	50	20	.4
	4	.6	50	10	.2
	4	.6	50	5	.1
Experiment 2b:					
variable volume, constant density; pellets	4	.6	100	40	.4
	4	.6	50	20	.4
	4	.6	25	10	.4
	4	.6	12.5	5	.4

Note: Various tank volumes were used, and the number of fish is expressed as both cohort size and cohort density. Each combination of daily food ration and cohort size was replicated three times, for a total of 24 tanks used per experiment. The same six 50-L tanks with 20 fish were used for experiments 2a and 2b. The cohorts of 5 fish per tank did not reach equilibrium (limiting-food treatment only) within the time frame of experiment 2.

Experiments 1 and 2 were performed in separate tank systems and with differently sized hatchery-reared Australian bass. Experiment 1 was performed in 2009 with Australian bass fry (initially 0.025 ± 0.006 g) in a flow-through aquarium system at the Port Stephens Fisheries Institute, Taylors Beach, New South Wales, Australia. Experiments 2a and 2b were performed simultaneously in 2010 with Australian bass fingerlings (initially 0.298 ± 0.060 g) in a recirculating aquarium system using variously sized tanks (table 1) at the University of New South Wales, Sydney, Australia. Both systems were held at a constant 22°–23°C.

The duration of the experiments was dependent on the limiting-food treatment, because these tanks had to reach a food-limited equilibrium of body mass indicating that the cohorts had reached the STR. Equilibrium was determined to occur when the average body mass showed no increase between two sampling weeks. In experiment 1, all cohorts in the limiting-food treatment reached equilibrium after approximately the same duration (5 weeks), at which point both the limiting-food and increasing-food treatments were stopped. Experiments 2a and 2b ended after different durations for each cohort size in the limiting-food treatment: the 5–fish $tank^{-1}$ cohort did not reach equilibrium after 14 weeks; the 10–fish $tank^{-1}$ cohort reached equilibrium in week 14, the 20–fish $tank^{-1}$ cohort in week 10, and the 40–fish $tank^{-1}$ cohort in week 6. The increasing-food treatment in experiment 2 were ended after 6 weeks for all cohort sizes, as this was deemed a sufficient period to observe the pattern of density-dependent growth.

Feeding

In experiment 1, Australian bass fry were fed enriched *Artemia* sp. (Great Salt Lake *Artemia*, AA-grade; INVE Aquaculture, Salt Lake City, UT; enrichment diet Algamac 3050, Bio-Marine, Hawthorne, CA). The ration for the increasing-food treatment was 30% of average body mass per fish per day (table 1), provided in two equal feedings at 9 a.m. and 4 p.m. The food ration for each tank was calculated each day, using the average live body mass (calculated weekly) and the number of fish per tank (calculated daily). The 30% dosage was considered appropriate from a pilot study, which showed that this ration provided good scope for growth but without complete satiation. A lack of satiation was theorized to induce competitive behavior between individuals, which was necessary if the results of density-dependent competition on growth and survival were to be observed. The ration for the limiting-food treatment was a fixed 0.5 g $tank^{-1}$ day^{-1}, provided in two equal feedings. This ration was chosen to allow some initial growth in the largest cohort size treatment and an eventual limitation of food for all treatments within the experiment's time frame.

A wet weight : volume ratio of *Artemia* was calculated

before each feeding by syringing a known volume of well-mixed *Artemia* solution through a sieve and weighing after removing the excess water. This ratio was used to convert the required feeding rations into volumes, which were added to the tanks with a measuring cylinder or syringe. In experiments 2a and 2b, Australian bass fingerlings were fed with manufactured pellets (Otohime C1: Marubeni Nisshin Feed, Tokyo). The ration for the increasing-food treatment was 4% of the average body mass per fish per day (table 1), provided in two equal feedings, and was calculated daily as in experiment 1. The ration for the limiting-food treatment was 0.6 g $tank^{-1}$ day^{-1}, provided in two equal feedings. The suitability of both rations was judged as in experiment 1. Water flow to tanks was stopped for 1 h during feeding in all experiments, so that food would not be washed away.

Measuring Growth and Mortality

At the beginning of experiments 1 and 2, 100 fish were weighed to calculate the average initial body mass for the population. Fish were then randomly assigned to cohort size treatments. During the experiments, all fish (for 5 or 10 fish $tank^{-1}$) or a sample of 10–15 fish (for ≥20 fish $tank^{-1}$) from each tank were weighed (± 0.001 g) every 7 days. Live weights were measured by netting a fish, absorbing the excess water with paper towel, and adding the fish to a weighed container of water.

Mortality in the experiment was addressed with a "quasi-mortality" protocol, which reduced the suffering of fish due to starvation and was necessary to comply with Animal Care and Ethics legislation, which prohibits death as an experimental endpoint. This protocol required the identification of poorer competitors that showed a high probability of dying and the removal of these fish for euthanasia. Specific signs used in these experiments were (1) a lack of feeding behavior, (2) poor swimming ability, and (3) a change in coloration. If a fish exhibited any two of these signs, it was removed from the tank and euthanized in an ice slurry (Barker et al. 2002). This protocol was employed at all times, and tanks were examined at least twice a day for weak fish. To test the assumption that removed fish would not recover, 10 fish removed in experiment 1 were selected for rehabilitation. These fish were placed alone in a separate tank with food and observed for 12 h, but none improved or showed signs of feeding and were euthanized. Fish removed according to this protocol are considered deaths in all results.

Activity

An indicator of activity was measured in each experiment to help interpret the patterns of density-dependent growth in terms of energy allocation. In experiment 1, swimming speeds were analyzed from video footage of the tanks recorded during weeks 1, 3, and 4. A video camera was positioned directly over each tank, and 5 min of footage was recorded between 1 and 3 h after feeding. Five fish were followed for 10–20 s each, so that at least 1 min of swimming was analyzed per tank. The paths of fish were traced from a computer screen onto transparency film, which was scanned into a computer, and the distances were measured with ImageJ software (National Institutes of Health, Bethesda, MD). The average body length of each fish was calculated for the duration it was followed, to estimate the average swimming speed (body lengths s^{-1}). In experiments 2a and 2b, activity was estimated with two measures of foraging activity: the number of feeding "hits" fish made at the surface in the first minute after feeding and the time (seconds) it took for the first hit to occur. Surface activity during this early period was considered a suitable indicator because pellets initially floated. This measurement was repeated three times for each cohort size in the limiting-food treatment during week 6.

Statistical Analysis

The relationship between body mass and the number of fish per tank was the focus of the limiting-food and increasing-food treatments and was termed the self-thinning relationship (STR) or the density dependence relationship (DDR), respectively (fig. 2). Like STRs, DDRs are usually expressed as a power curve (Imre et al. 2005; Amundsen et al. 2007; Lobón-Cerviá 2007) that becomes linear when the variables are log transformed. The slope of the log-transformed STR represents $-\beta$, and the slope of the log-transformed DDR (b) was interpreted as the pattern of density-dependent intraspecific competition. Least squares regression statistics and significance of slopes were calculated on $\log_{10}$-transformed data for all STRs and DDRs.

To examine the relationship between density-dependent competition and self-thinning, the b values derived from DDRs were compared with the β values derived from STRs for each of the three experiments (fig. 2). The resulting relationship between b and β indicates the effect of density-dependent intraspecific competition on the STR. The slopes β of the STR treatment in experiments 2a (variable fish L^{-1}) and 2b (constant fish L^{-1}) were not statistically compared. Making a statistical comparison between experiments 2a and 2b assumes that the cohort sizes replicated between the two experiments were functionally identical (i.e., that all fish had equal access to the resources regardless of tank size). This remains an assumption in this experiment (given that tank sizes in 2b were arbitrary),

making a statistical test of the specific β values in experiments 2a and 2b illogical.

Average swimming speeds and foraging-activity rates were plotted against cohort size, but only foraging activity was analyzed. An effect of cohort size on the two measures of foraging activity was tested within experiments with ANOVA on square root–transformed data. Tukey's HSD test was used to examine differences between cohort size treatments. All parametric tests were performed in JMP (7.0, SAS Institute, Cary, NC). Normality was assessed with a normal probability plot, and residuals were examined for homogeneity of variance.

Results

The Self-Thinning Relationship (STR)

The STR estimated in experiments 1, 2a, and 2b had β values of 0.802 ± 0.071 (95% confidence interval), 1.325 ± 0.078, and 1.177 ± 0.059, respectively (fig. 3*a*, 3*c*, and 3*e*, respectively), which were significantly different from 0 when $\log_{10}$ transformed and tested with a general linear model (table 2). The trajectories followed by the various cohort sizes provide evidence that the fitted relationships represent STRs, because the average body mass stopped increasing in all treatments (fig. A1, available online). One cohort size (5 fish tank^{-1}) in experiments 2a and 2b did not reach equilibrium before the experiment concluded and was not included in the analysis of the STR. Mortality increased with abundance and was higher in experiment 1 (fig. A1), possibly because of the smaller size of fish in this experiment.

The Density-Independent STR

Experiments 2a and 2b were performed to test the prediction that the STR can have a density-dependent component. Tank size was varied in experiment 2b to maintain a constant cohort density (fish L^{-1}) while examining the same cohort sizes (fish tank^{-1}) as in experiment 2a. The increasing-food treatment was used to determine the pattern of density-dependent intraspecific competition in each experiment, and it revealed that density-dependent growth was greatly reduced when cohort density was constant (experiment 2b) and that the slope b was nonsignificant when three cohort sizes were analyzed (fig. 3*f*; table 2). This nonsignificance suggests that the corresponding limiting-food treatment in experiment 2b provided a close approximation of the density-independent STR, that is, when $\beta = 1.177$ (fig. 3*e*; table 2). When cohort density varies with cohort size (experiment 2a), intraspecific competition is more strongly density dependent, as observed in the increasing-food treatment. The corresponding STR is steeper and represents the STR with a density-dependent component, that is, when $\beta = 1.325$ (fig. 3*c*; table 2).

The Association of b *and* β

The increasing-food treatment was used to determine the pattern of intraspecific competition in each experiment for comparison with the corresponding STRs, to determine whether density-dependent competition has a predictable influence on self-thinning. The density dependence relationships (DDRs) for cohorts in the three experiments had different slopes (-0.311, 0.103, and -0.051 for experiments 1, 2a, and 2b, respectively) that were all significantly different from 0 (table 2; fig. 3). This significance shows that growth was density dependent in all experiments, showing a negative trend in experiments 1 and 2b and a positive trend in experiment 2a. The weakest relationship was observed in experiment 2b when density (fish L^{-1}) was constant, and the slope was nonsignificant ($P > .05$) when the 5–fish tank^{-1} cohort was excluded (table 2). There was evidence of a negative linear relationship ($-\beta = -1.274b - 1.212$; $r^2 = 0.999$) between the pattern of density-dependent intraspecific competition (the DDR slope, b) and the self-thinning relationship (the STR slope, $-\beta$; fig. A2, available online) for the three experiments.

Density-Dependent Activity Rates

Indicators of activity rates were measured to provide evidence that density-dependent competition contributes to self-thinning by altering energy use. Swimming speeds did not show a conclusive relationship with cohort size (fish tank^{-1}) in either the increasing-food or the limiting-food cohort in experiment 1 (fig. 4). Swimming speeds averaged around 4–6 body lengths s^{-1} in the increasing-food cohorts, except at cohort sizes around 80 fish tank^{-1}, which showed increased swimming speeds on two occasions (fig. 4*a*). Swimming speeds were also around 4–6 body lengths s^{-1} in the limiting-food cohorts and showed the most variation for the smallest cohort size (fig. 4*b*).

Foraging movements were measured in experiment 2 as an alternative to swimming speed. The time for a fish to take a pellet from the water's surface was significantly longer for smaller cohort sizes in experiment 2a (ANOVA: $F_{3,35} = 35.48$, $P < .001$; white bars in fig. 4*c*). When cohort density (fish L^{-1}) was constant (experiment 2b), this hesitancy in smaller cohort sizes was removed, and all cohort sizes had an individual take a pellet within 2 s (ANOVA: $F_{3,32} = 1.69$, $P = .192$; gray bars in fig. 4*c*). Feeding rate also showed a relationship with cohort size. When tanks were all the same size (experiment 2a), we saw a significantly reduced feeding rate for the smallest and largest

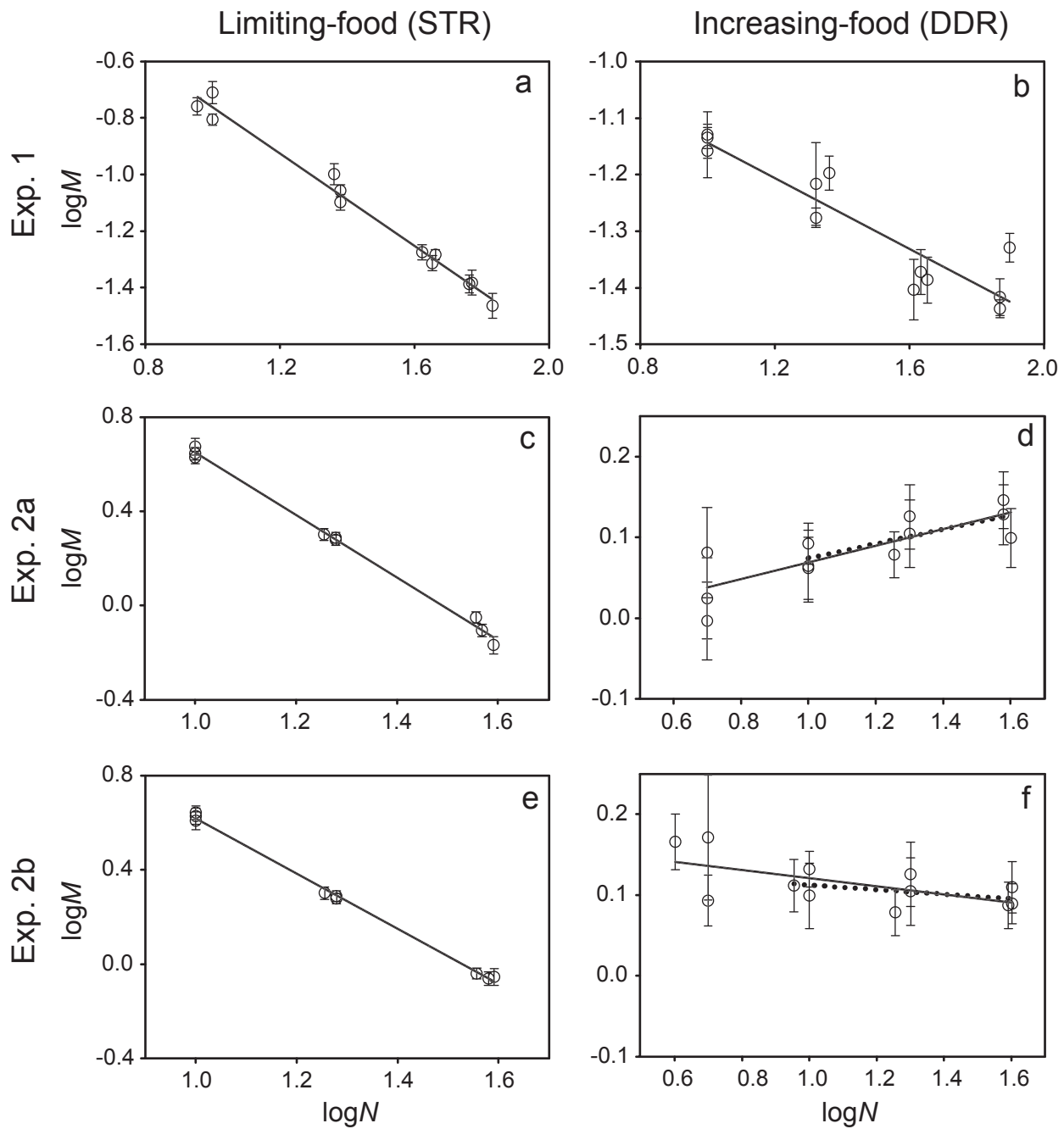

Figure 3: Self-thinning (STR) and density dependence (DDR) relationships for the three experiments. Body mass (M) in grams and cohort size (N), the number of fish per tank, are $\log_{10}$ transformed. The statistics of the least squares regressions (lines) are given in table 2. Two DDRs were tested in experiment 2, one testing all four densities (solid line) and one testing the same three cohort sizes used in the STRs (dotted line).

cohort size treatments ($F_{3,35} = 10.44$, $P < .001$) but still a relatively slow feeding rate of 1–2 surface hits fish^{-1} for all treatments (white bars in fig. 4*d*). In experiment 2b, feeding rates were significantly higher for the two smallest cohort size treatments ($F_{3,32} = 45.64$, $P < .001$; gray bars in fig. 4*d*). Thus, both hesitancy to feed and feeding rate are mechanistically related to cohort density (fish L^{-1}) rather than to cohort size (fish tank^{-1}).

Table 2: Results of the least squares regression for all experiments, as illustrated in figure 3

Experiment, relationship	b (95% CI)	$\log_{10}a$	r^2	n	t ratio	P
Experiment 1:						
DDR	−.311 (.091)	−.824	.853	12	−7.61	<.001
STR	−.802 (.071)	.042	.985	12	−25.22	<.001
Experiment 2a:						
DDR^4	.103 (.051)	−.034	.674	12	4.55	.001
DDR^3	.089 (.065)	−.015	.597	9	3.22	.015
STR	−1.325 (.078)	1.982	.996	9	−40.34	<.001
Experiment 2b:						
DDR^4	−.051 (.045)	.171	.379	12	−2.47	.033
DDR^3	−.028 (.054)	.141	.182	9	−1.25	.252
STR	−1.177 (.059)	1.806	.997	9	−46.65	<.001

Note: Regression: $\log_{10} M = b\log_{10} N + \log a$. DDR and STR represent the density dependence and self-thinning relationships, respectively. For an STR, $\beta = -b$. The t ratio and P value evaluate the null hypothesis that $b = 0$. Two regressions were calculated for the DDRs in experiments 2a and 2b: one with all four density treatments (DDR^4) and one with the same three density treatments used in the STR (DDR^3; see table 1; fig. 3). CI = confidence interval.

Discussion

Self-thinning is a fundamental concept in population ecology, but questions remain about how it applies to mobile fauna. One such question is whether the declining density of competitors in a thinning population can also scale energy use and thereby confound the standard metabolic scaling model. We have shown that density-dependent competition can have a large effect on self-thinning and that density-driven changes in energy use can confound the influence of metabolic scaling. The difficulty of partitioning density dependence in the self-thinning of wild populations and cohorts might explain the lack of research addressing this question. There may also be an assumption that individuals in real self-thinning populations are too sparse to be affected by density dependence. While it remains uncertain whether the cohort sizes and densities tested in this study are realistic for wild populations, the primary cause of density-dependent self-thinning appears to be density-dependent competition, which is frequently observed in wild populations (Jenkins et al. 1999; Lorenzen and Enberg 2002; Utz and Hartman 2009). It therefore seems likely that density-dependent self-thinning can occur in wild populations, and this should be evaluated before the concept is applied to mobile animals.

Evidence for Density-Dependent Self-Thinning

Two types of food-limited STR were constructed in this study. Experiments 1 and 2a created "trajectory" STRs, which simulate a trajectory of a typical self-thinning cohort that simultaneously declines in size and density. Experiment 2b created a "metabolic" STR, which simulates a cohort that declines in size but maintains a constant density (and therefore eliminates density-dependent energy use). Comparing the β values from the STRs in experiments 2a and 2b (which had the same cohort sizes) highlights the density-dependent component of self-thinning. The difference between the trajectory β (1.325 ± 0.078) and the metabolic β (1.177 ± 0.059) confirms that self-thinning can be density dependent.

The metabolic β (1.177; fig. 3*e*) in this study does not support the default β (1.333) proposed for typical food-limited self-thinning (Begon et al. 1986). The existence of a metabolic scaling β may still be supported, however, provided that β (1.177) is not significantly different from the true scaling of metabolism in Australian bass. This is possible, given that the scaling of metabolism with body mass can vary between taxa (Latto 1994); for example, β is around 1.15 for Atlantic salmon (Steingrímsson and Grant 1999). The difference observed between the β values of the metabolic and trajectory STRs, however, shows that metabolic scaling cannot accurately predict the STR of cohorts that experience density-dependent competition. The limitation of metabolic scaling for describing self-thinning cohorts is further demonstrated by the β value in experiment 1 (0.802 ± 0.071), which should be similar to the trajectory β from experiment 2a (1.325) if metabolic scaling is the only determinant of the STR. Thus, the suggestion that β should be calculated for the species of interest because of taxa-specific differences in metabolic scaling (Latto 1994) should be extended to include taxon- and environment-specific differences in density-dependent energy use.

Estimates of β for intraspecific cohorts of mobile aquatic animals are variable. The value of β can depend on whether a population is limited by food or by space (Steingrímsson and Grant 1999; Keeley 2003). Even so, there are numerous examples within studies and within species where β values

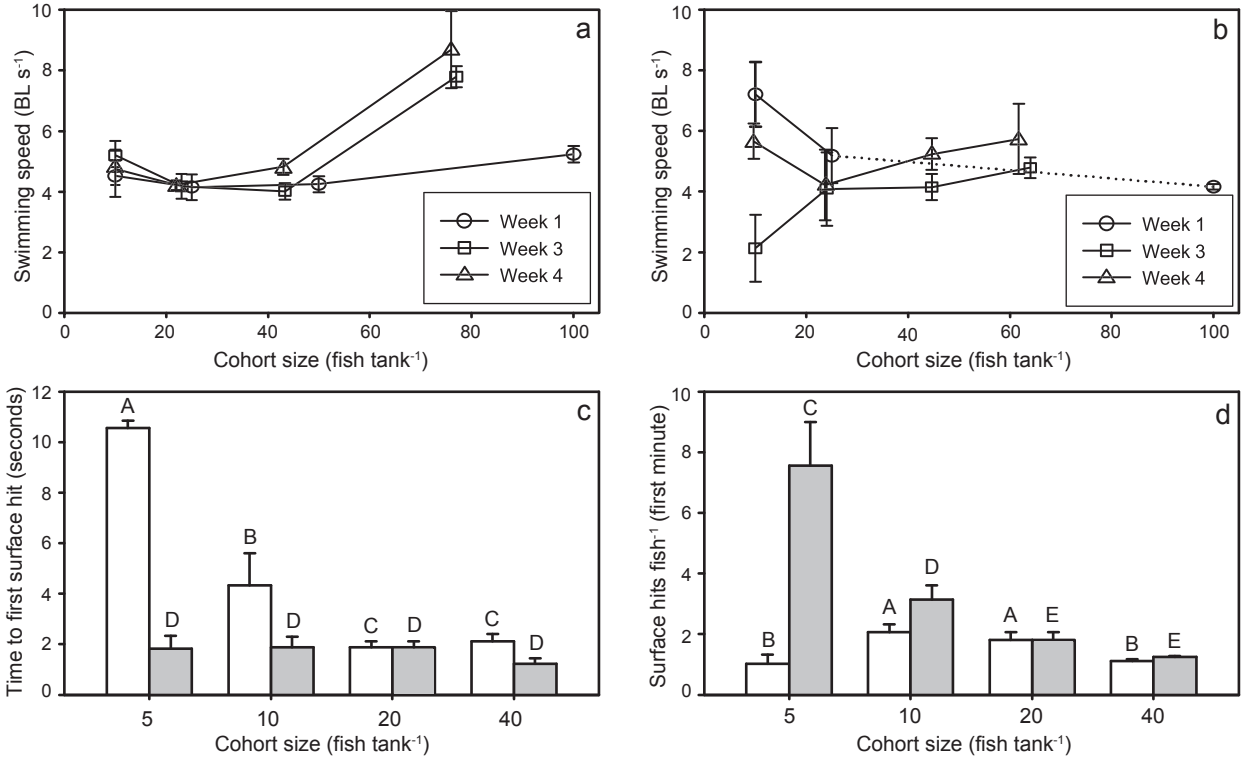

Figure 4: Swimming speeds and foraging movements. In experiment 1, swimming speeds (body lengths per second [BL s^{-1}]) were measured in three different weeks and were averaged for each cohort size in both the increasing-food (*a*) and limiting-food (*b*) treatments. Swimming speed was not measured for 50 fish $tank^{-1}$ in the limiting-food treatment (dotted line in *b*). In experiment 2, activity was measured as feeding intensity. The average number of surface "hits" for food in the first minute of feeding is shown (*c*), as is the time taken for the first surface "hit" to occur (*d*). Feeding intensity was measured only in experiment 2a (variable cohort density; white bars) and experiment 2b (constant cohort density; gray bars) in the limiting-food treatment. The result of Tukey's HSD test are given in capital letters (*c*, *d*; bars within an experiment not sharing a letter are significantly different). The 20–fish $tank^{-1}$ population size is duplicated to illustrate the results of Tukey's test. Error bars represent standard error ($n = 3$).

have shown large variation (Elliott 1993; Dunham and Vinyard 1997; Steingrímsson and Grant 1999). These β values may be variable because most were estimated for wild populations and therefore incorporate sampling errors and environmental variability (Dunham and Vinyard 1997; Keeley 2003), or they may be variable because of variation in metabolic scaling (Isaac et al. 2011). In our study, however, all three experiments had different β values, which suggests that an explanation for variation in β other than taxon-specific differences and environmental variation is required. Density-dependent competition and its effect on resource use can be used to explain this variation.

Possible Energetic Mechanisms

Evidence that density-dependent competition drives the deviation in β values in this study is the linear association between the exponents of the STR (β) and the density dependence relationship (DDR; *b*). An increase in the strength of negative density dependence ($b < 0$) corresponds to a decrease in β. This association between β and *b* is conditional on the pattern of density-dependent growth for the experimental cohort sizes being independent of feeding treatment and does not necessarily demonstrate a direct link. It does provide evidence, however, for a common mechanism that causes density-dependent growth and influences self-thinning.

The mechanisms causing density-dependent growth can be numerous and difficult to determine (Keeley 2001) and are often broadly defined as "competition." When density-dependent competition causes growth rate to decline with increasing density, a negative power curve is the result, which is the most common pattern and is usually attributed to exploitative competition (Post et al. 1999; Imre et al. 2005). Growth rate can also be suppressed at low densities (Jørgensen et al. 1993) by interference competition (Jobling and Wandsvik 1983) or reduced feeding rate (this

study). A positive power curve can result from this latter situation, although it would probably become negative at very high densities. Both positive and negative density-dependent growth was observed in this study, and there is some evidence that density-dependent activity contributed to these patterns.

The strong negative density-dependent growth observed in the increasing-food treatment in experiment 1 could be due to an increase in competition-induced activity at high densities (Marchand and Boisclair 1998), and increased activity was observed in the swimming speeds for 80 fish $tank^{-1}$ in these cohorts. A reduction in stress at low densities could also have contributed to the negative density-dependent growth (Fagerlund et al. 1981). Positive density-dependent growth was observed in experiment 2a, and there was evidence that this was due to reduced feeding activity at low densities. Fish were reluctant to leave the group and move to the surface to feed, which indicates a reduction in energy acquisition due to reduced foraging activity. Boldness increased for small group sizes in experiment 2b, such that all cohort sizes took food from the surface after equal durations when cohort density was constant. The number of surface hits in the first minute did not equalize when density was made constant, although the feeding rate increased for small cohort sizes when density was increased. Thus, the patterns of growth in the increasing-food cohorts were probably caused by reduced energy acquisition at low densities (positive density dependence) and reduced energy allocation for competition (or stress) at low densities (negative density dependence). What remains to be explained is how these patterns of growth can explain the variation in self-thinning relationships.

Interpreting the patterns of density-dependent growth (b) in the context of the food-limited self-thinning relationships (β) is not straightforward. The observed association between b and β can be described as inverse; that is, an increase in negative density-dependent growth causes a shallower STR (figs. 5, A2), noting that "shallower" is relative to the orientation of the M and N axes. The energetic mechanism that could cause this pattern is likely to be variation in the amount of energy allocated to activity or metabolism or the efficiency with which energy is used. Energy acquisition can explain density-dependent growth but cannot explain density-dependent self-thinning, because the amount of energy acquired in self-thinning cohorts is assumed to be constant (Westoby 1984). It is also apparent that if a decline in activity is the cause of negative density-dependent growth (fig. 5*c*), then a cohort should have more energy for growth as it thins and have a steeper STR, but the opposite was observed (fig. 5*d*). This leaves energetic efficiency and the allocation of energy to metabolism as possible mechanisms. In fact, it seems likely

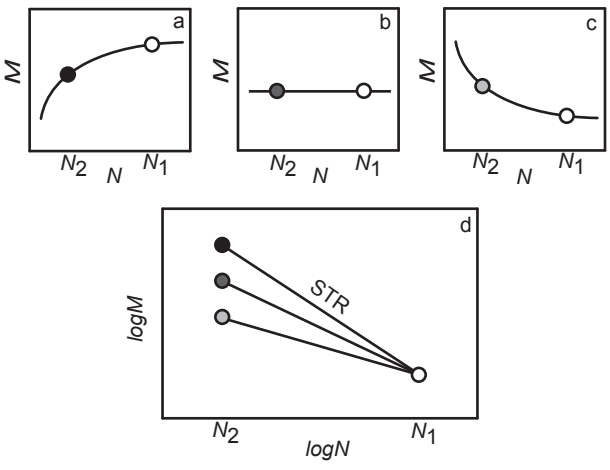

Figure 5: Summary of the relationship between density-dependent competition and the self-thinning relationship (STR) observed in this study (N = abundance; M = body mass). Populations can show positive density-dependent growth (*a*), density-independent growth (*b*), or negative density-dependent growth (*c*) in response to competition. This study suggests that the trajectory of a self-thinning population or cohort (*d*) is influenced by the pattern of density-dependent competition. A cohort that declines in abundance (from N_1 to N_2) and does not experience density-dependent competition (*b*) will have an STR approximated by metabolic scaling (dark gray circle in *d*). A cohort that experiences positive density dependence (*a*) will achieve a larger food-limited body mass as it thins and will have a steeper STR (black circle in *d*). A cohort that experiences negative density dependence (*c*) will achieve a smaller food-limited body mass as it thins and will have a shallower STR (light gray circle in *d*). One explanation for this pattern is that growth rate is proportional to energetic efficiency. A cohort that allocates more energy to growth with a decline in density (*c*) must also allocate more energy to the costs of maintaining fast growth and will achieve a lower food-limited body mass (light gray circle in *d*). The opposite is true of cohorts that decline in growth rate as density decreases, which are more likely to use limiting energy more efficiently (black circle in *d*).

that these mechanisms are related, such that the pattern of energy allocation toward growth in these cohorts is what determines their metabolic costs.

There is a positive association between growth rate and metabolic costs (Jobling 1981; Arendt 1997), and being a fast grower is a disadvantage when food becomes scarce (Derting 1989). Evidence for this in our study is seen by comparing experiments 2a and 2b, which showed that fish grew faster in the increasing-food treatment when density was increased but attained a smaller asymptotic body mass in the limiting-food treatment (fig. A3, available online). This pattern can be explained by the "food-limitation hypothesis for slow growth," which states that slow growth is beneficial when food is limiting because energetic efficiency is increased (Arendt 1997). This hypothesis is normally used to explain the existence of different growth rate

strategies between individuals (Arendt 1997), but it could equally explain differences between populations or even those between stages of a cohort's self-thinning trajectory on the basis of the combined behavior of the individuals within it. The model of "density-dependent self-thinning" that we propose is that animals in a self-thinning cohort alter their allocation of energy to growth in response to a decline in density (fig. 5*a*–5*c*), which alters the energetic efficiency of the cohort, which determines its body mass (fig. 5*d*). In terms of individual interactions, the change in energy allocation might be due to a shift from predominantly contest competition to exploitative competition (Ward et al. 2006) as cohort density decreases.

A consequence of relating growth rate to self-thinning is that mortality rate can be included in the density-dependent self-thinning model. That is, the stronger the negative density dependence in a cohort, the shallower the STR but also the faster mortality (or emigration) occurs. This pattern has been acknowledged for plant assemblages (Westoby 1984) but could not be tested in this study. Variation in mortality rate would be observed in density-dependent self-thinning both within cohorts (the mortality rate will change during the trajectory) and between cohorts (cohorts with more fast growers will have higher mortality rates as well as shallower STRs). Another consequence of the density-dependent self-thinning model is that β will vary most at low competitor densities. Density-dependent growth is frequently demonstrated to be most detectable at low densities (Jenkins et al. 1999; Imre et al. 2005; Lobón-Cerviá 2007) and may be an empirical result of density dependence being defined as a power curve (Amundsen et al. 2007). If a power curve is used (e.g., fig. 5*c*), a reduction in absolute density will alter competitive interactions more strongly for smaller densities. Given this, a power curve may be inappropriate to describe density-dependent self-thinning, and it may be more accurate to measure abundance as a function of average estimated resource use rather than body mass (White et al. 2007).

Implications for Energetic Equivalence

Metabolic scaling is not restricted to self-thinning theory and is used to describe the general structure of communities and food webs (e.g., Cyr et al. 1997; Ernest et al. 2003; Reuman et al. 2009). Metabolic scaling in these studies is rebranded "energetic equivalence," which states that the difference in abundance between species (whether they share energy or not) can be explained by the difference in their mass-specific metabolic rates (Nee et al. 1991; White et al. 2007). The metabolic scaling β used in energetic equivalence is generally the default used in self-thinning: $\beta = 0.75$ (Damuth 1981; Nee et al. 1991). Self-thinning in mobile animals is usually measured for single-species populations but can be used for multispecies sessile communities (Westoby 1984; Hughes and Griffiths 1988; Enquist et al. 1998) and mobile animal assemblages (Bohlin et al. 1994). In these multiple-species studies, the energetic-equivalence and self-thinning concepts are difficult to distinguish. The complication of linking the two concepts is illustrated by the choice of the dependent variable, which reflects the conflicting assumptions of constant biomass accumulation (self-thinning) and determinate body mass (energetic equivalence). It may also be that the accuracy of the default metabolic scaling β depends on the range of masses analyzed. Multiple-taxa studies average a wider range of masses than cohort or single-species studies (Moran and Wells 2007), so the relevance of density-dependent energy use may also depend on the diversity of the assemblage.

Two ways in which density-dependent competition could relate to energetic equivalence are describing within-species variation in mass-abundance relationships and explaining size-specific inconsistencies between the absolute densities of predators and prey. Within-species variation could be explained with the absolute density of discrete populations, as large variations in density are likely to cause changes in energy use. Size-based interactions with the food resource are thought to complicate the energetic-equivalence rule (Carbone et al. 2007; Isaac et al. 2011), and competition between species has been identified as a determinant of population density (Carbone and Gittleman 2002). Relating density-dependent competition to these studies is best done by using mortality rate, because they consider a species' body mass fixed at equilibrium.

In density-dependent self-thinning, an increasing growth rate as density declines increases the mortality rate. In studies using energetic equivalence, it has been observed that the largest consumers use more energy (Isaac et al. 2011) and are more sparse (Carbone et al. 2011) than expected. These two patterns may share a cause: that populations with competitive strategies that include fast growth are less energetically efficient than predicted by metabolic scaling. If body mass is fixed, then a decline in density would be expected (Carbone et al. 2011). Given density-dependent energy use, it also follows that the abundance of a consumer depends on the absolute abundance of the consumer-prey assemblage. Further investigation into the known links between self-thinning and community dynamics (Westoby 1984) is necessary to test these ideas and to discern the common energetic mechanisms structuring populations and communities.

Confounding Factors

It is important to note that density and body mass covary and are difficult to separate and that some potential ar-

tifacts were introduced as a result of the experimental design. First, this study makes an assumption that the pattern of density-dependent competition in the increasing-food treatment (b) is an appropriate indicator of the energy allocation in the limiting-food treatment (β). While the strength of competition is likely to vary with per capita food ration (and would therefore change during the limiting-food treatment), the manipulations of food type (experiments 1 and 2), cohort size (experiments 1 and 2), and cohort density (experiments 2a and 2b) are probably robust to differences imposed by feeding treatment, although this remains an assumption. Second, varying tank size in experiment 2b to manipulate the density of fish also changed the density of food. The distribution of food can be an important determinant of growth rate (Walters and Post 1993). Thus, the difference in density-dependent growth between experiments 2a and 2b could be due to an alteration of energy use driven by an interaction with the food supply rather than to interactions with the changing density of competitors. Replicating the density-dependent treatments across more tank sizes could test this.

Concluding Remarks

Self-thinning via metabolic scaling depends on energy use being proportional to metabolic rate, but there is evidence that this may not be the case. The allocation of energy to activity or growth can be density dependent in mobile animals, which contributes to when a food-resource becomes limiting. The efficiency of energy use may also change in a self-thinning cohort, because the metabolic requirements increase beyond the predictions of metabolic scaling when growth rate is increased. The precise energetic pathway causing this pattern could not be resolved, but the pattern of density-dependent competition (slope b) shows promise as a surrogate of a predictive energetic model. Finally, research on self-thinning and size-density relationships in general may benefit from distinguishing between population size and population density, because comparison of these distinct metrics could be used to discover the role of space in food competition and to identify the mechanisms driving mass-abundance scaling.

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