DENSITY DEPENDENCE IN MARINE FISH POPULATIONS REVEALED AT SMALL AND LARGE SPATIAL SCALES

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Abstract. Experimental manipulation of population density has frequently been used to demonstrate demographic density dependence. However, such studies are usually small scale and typically provide evidence of spatial (within-generation) density dependence. It is often unclear whether small-scale, experimental tests of spatial density dependence will accurately describe temporal (between-generation) density dependence required for population regulation. Understanding the mechanisms generating density dependence may provide a link between spatial experiments and temporal regulation of populations. In this study, I manipulated the density of recently settled kelp rockfish (Sebastes atrovirens) in both the presence and absence of predators to test for density-dependent mortality and whether predation was the mechanism responsible. I also examined mortality of rockfish cohorts within kelp beds throughout central California to evaluate temporal (between-generation) density dependence in mortality. Experiments suggested that short-term behavioral responses of predators and/or a shortage of prey refuges caused spatial density dependence. Temporal density dependence in mortality was also detected at larger spatial scales for several species of rockfish. It is likely that short-term responses of predators generated both spatial and temporal density dependence in mortality. Spatial experiments that describe the causal mechanisms generating density dependence may therefore be valuable in describing temporal density dependence and population regulation.

Key words: density dependence; mortality; open populations; population regulation; predation; recruitment limitation; reef fish; rockfish; Sebastes.

INTRODUCTION

Populations often fluctuate in response to environmental variation (e.g., changes in climate, habitat, or the abundance of other species), and only through compensatory changes in demographic rates can populations counter such variability and persist over long periods of time (Murdoch 1994). Knowledge of both the mechanisms (e.g., predation, competition, disease) and conditions under which demographic rates change in response to population density (i.e., density dependence) is central to our understanding of population dynamics (Royama 1977, Hixon et al. 2002, Turchin 2003). Identifying the mechanisms driving demographic density dependence, and evaluating their effects at the scale of large populations has been difficult, especially for species with complex life histories. Nonetheless, our ability to manage and conserve species' populations relies heavily on our understanding of processes that contribute to their regulation and persistence.

The life history of many marine organisms, including most demersal (seafloor-oriented) fishes, involves a dispersive larval stage that is followed by juvenile and adult stages that exhibit more restricted movement in the benthic environment. This "bipartite" life history and potential for long-distance larval dispersal may result in the "open" (sensu Caswell 1978) spatial structure of many marine populations, in which local adult populations are typically replenished by young that are produced elsewhere and delivered by ocean currents. The addition of young to a local population (i.e., "recruitment") can be highly variable from one year to the next, and variation in larval supply is known to cause marked fluctuations in the size and structure of benthic populations (reviewed by Caley et al. 1996). In the face of such variable replenishment, mechanisms that contribute to density dependence in post-settlement growth, movement, and mortality are key to regulating variation in population size and, eventually, reproduction.

Population sizes of many demersal fish species are sensitive to rates of post-settlement mortality (Shulman and Ogden 1987, Hixon 1991, Doherty et al. 2004). Density-dependent mortality of juveniles can therefore have large effects on the regulation of population size (Myers and Cadigan 1993, Caley et al. 1996, Hixon and Webster 2002). A growing body of experimental studies suggests that post-settlement mortality of reef fishes is often, but not always, density dependent (reviews by Hixon and Webster 2002, Osenberg et al. 2002). In general, experimental studies of density de-
pendence are necessarily conducted at small scales (Harrison and Cappuccino 1995) and typically provide evidence of spatial (i.e., within-generation) density dependence (but see Webster 2003 for an example of temporal density dependence in fish). It is often unclear whether tests of spatial density dependence via small-scale experiments will be useful in describing temporal (between-generation) density dependence, which can act to regulate populations (Stewart-Oaten and Murdoch 1990). Conversely, for some demersal fishes, evidence for density-dependent mortality has been inferred from time series observations of population density (Myers and Cadigan 1993, Stenseth et al. 1999). However, in these cases no causal mechanisms have been established.

A combination of small-scale experiments and large-scale observations over time can determine both the mechanisms causing density-dependent mortality and how these processes are likely to affect population regulation. This information is especially important for demersal fishes that are targeted by fisheries (e.g., rockfish, cod, grouper), as evaluating density-dependent effects can improve predictive fisheries models and management strategies (Rose et al. 2001). In this study, I conducted field experiments where I manipulated both the density of recently settled kelp rockfish (Sebastes atrovirens) and the presence of predators to test the hypothesis that early post-settlement mortality is density dependent and caused by predation. I also used annual counts of rockfish within nearshore habitat (kelp beds of ∼500 × 70 m in size) to examine evidence for temporal density dependence in input to rockfish populations.

METHODS

Study area and species

Fieldwork was conducted in Carmel Bay, California (36°33.6′ N, 121°56.3′ W). Shallow, subtidal habitat in this area mainly consists of rocky reefs that support stands of giant kelp (Macrocystis pyrifera). The young of many rockfish species inhabit the canopy of the kelp bed (Anderson 1983, Carr 1991). I conducted experiments on kelp rockfish (Sebastes atrovirens), a common species with a life history that is representative of many nearshore rockfishes (Love et al. 2002). Adult females release larvae that remain in the plankton for 1–2 mo prior to settling to the kelp canopy, which they occupy for another 1–2 mo before descending and dispersing across the reef. Juvenile kelp rockfish are amenable to density manipulations, have a high affinity for kelp cover, and show strong site fidelity (Carr 1991, Nelson 2001). Kelp rockfish generally aggregate shortly after settlement, and during the peak recruitment season, group size can be >300 individuals per large kelp plant (personal observation).

Observational analysis of density-dependent mortality within kelp beds focused on several common rockfish species: the blue rockfish (Sebastes mystinus) and the “KGB” species complex, which includes kelp, gopher, and black-and-yellow rockfish (Sebastes atrovirens, S. carnatus, and S. chrysomelas, respectively). Although juvenile kelp rockfish can be identified to species upon close inspection (Anderson 1983), species within the KGB complex were pooled for analysis because they school together, are very similar in behavior and appearance, and as juveniles, cannot be reliably identified to species on visual transect surveys. Recent settlers of the KGB species complex are small (1.5–2.5 cm total length [TL]); weak swimmers that inhabit the kelp canopy as juveniles and rely on kelp as a source of cover and refuge (Carr 1991). Blue rockfish settle at a size of 3.5–4 cm TL, are adept swimmers as juveniles, associate with reef structure, and are found near the bottom (Love et al. 2002).

Experimental test of density-dependent predation

To evaluate whether predation upon juvenile rockfish was density dependent, I manipulated the density of recently settled kelp rockfish (<3 cm TL; i.e., “recruits”) on isolated plots of kelp canopy and measured loss of fish from the plots over time. I established seven initial densities of fish (10, 20, 30, 40, 50, 75, and 90 fish/plot) with each density represented on one predator-exposed and one predator-exclusion plot. Kelp plots were designed to mimic the upper portion and canopy area of large kelp plants, features of the habitat that juvenile rockfish tend to aggregate around in large numbers. Kelp plots consisted of a standardized amount of kelp (total of 13 fronds, arranged in three bundles; each frond measured 3 m long, with 40–50 cm long blades separated by 10–16 cm) attached to a floating, rectangular PVC frame (2.5 × 1.5 × 1 m) that was anchored to the sea floor. Fish were tagged with subcutaneous injections of elastomer (Northwest Marine Technology, Shaw Island, Washington, USA), color-coded to identify which treatment each fish was transplanted to. To control for effects of handling and tagging, I stocked each plot with tagged fish and began tracking the loss of fish from each plot only after the required density of fish remained for 24 h. After this period I revisited plots every 1–2 d, identifying and counting the number of fish remaining, and removing any new recruits or immigrants as needed. Loss was measured from each plot for a period of 27 d. Although experimental treatments began at separate times, all treatments began within a 10-d period, and the initiation of the experiment was not confounded with density level or predator exposure.

As juveniles, kelp rockfish move very little (Carr 1991, Nelson 2001). Studies of a closely related species, the gopher rockfish (S. carnatus) indicated that over a 36-d period most of the tagged and resighted individuals moved fewer than 2–5 m from their original capture site (63.5% were found within 2 m and 84.1% within 5 m; Hoelzer 1988). In my experiment I further
discouraged emigration by setting kelp plots 15 m apart from each other and at least 20 m from adjacent reefs. Plots were placed above sandy substrate where rockfish were not found. I arranged the plots so that predator-exposed and predator-exclusion plots for each density treatment were adjacent to each other, and so that high and low density treatments were alternately represented along the onshore–offshore gradient. Predators were excluded by enclosing the kelp and PVC frame within a nylon mesh (1.3 cm) net. This mesh size was large enough to allow free movement of juvenile fishes, but small enough to exclude predatory fish (as small as 7 cm TL).

For each treatment, I calculated the instantaneous rate of loss as the negative of the change in the logarithm of the number of fish remaining on each plot over time. This estimate of per capita loss was calculated between each census and averaged over the 27-d experiment. I used an analysis of covariance to examine the relationship between per capita loss and initial density and to test for a significant difference in density-dependent loss between the predator treatments. To detect predator-induced mortality, I compared the predator-exposed treatments (all sources of loss combined) to the predator-exclusion treatments (all sources of loss minus predation, i.e., primarily loss due to emigration). I attributed the difference in loss rates between the two treatments to predation for two reasons: (1) predatory species were frequently observed foraging within the experimental array and several observations of predation events were made on predator-exposed treatments; and (2) observations suggested no predator-induced migration: in over 100 person hours of predator–prey observations, no observations were made of juvenile rockfish attempting to escape predators by long distance (>2 m) swimming through open water. Since there appeared to be a strong difference between the treatments (see Results), I analyzed each treatment separately with linear regression to test for a relationship between loss and density.

Over the duration of the experiment, some of the tagged and translocated fish migrated to other plots within the experimental array. When identified, these migrant fish were removed. Since the migrants could be traced to their original treatments, I used the number of recovered migrants as an additional measure of migration rate within my experiment. I used these data and linear regression to analyze proportional emigration from plots as a function of initial density.

**Predator abundance and activity**

Although predators regularly foraged within the experimental array, observations of predation events were rare. To quantify potential predation of recruits during the 27-d experiment, I estimated both the abundance of predators within the experimental array and their activity when hunting juvenile KGB rockfish. I then used these measures to generate a rough estimate of the total number of juvenile fish within my experiments that could be expected to be lost to predation.

Within this system, predatory fishes were most active during twilight. In general, predatory fishes began foraging within kelp beds and made forays to the periphery of the kelp beds (and into the experimental array) as light level decreased. To record predator activity, I periodically monitored the experimental plots at twilight, for a total of 10 censuses over the 27-d experiment. During each census, I identified and counted predators within 2 m of each plot. Predators were frequently observed striking at prey on experimental plots, and over the course of the experiment I observed several predation events from predator-exposed plots with certainty. However, a rigorous assessment of natural behavioral responses (e.g., aggregative or functional) was impossible since censuses were infrequent and predators within the experimental array often had to be detected and identified by flashlight. Instead, I quantified predator foraging by conducting timed observations of predator behavior within the kelp canopy of natural reefs, where predators tended to be active earlier in the day. I chose haphazardly encountered, predatory fish that were hunting and would observe each individual for 10 min, recording the total number of strikes and number of prey captured as predators hunted juvenile KGB rockfish. Prey capture was easy to determine with certainty since predators typically had to manipulate prey for several minutes before ingestion. Strike efficiency was calculated for each individual as the total number of observed prey captures/total number of strikes observed. I spent a total of 11.8 h conducting timed observations of predators.

I estimated the number of fish within my experimental array that could be lost to predation by multiplying the mean number of predatory fish present by the amount of time predators were likely to be active by the estimated strike rate and efficiency. I used all-predator averages for rates of activity and efficiency and assumed that fish observed near plots were foraging at the same rate as when foraging within the kelp canopy. Because I was unable to make observations at dawn, I conservatively estimated that predatory fishes were present and active for 45 min each day (one twilight period). These estimates did not consider density dependence in mortality rates; however, calculation of potential predation rates was useful in determining whether the number of predators encountered on these isolated, artificial habitats was enough to potentially account for the number of fish that were lost on the predator-exposed plots.

**Temporal density dependence in mortality**

I used annual counts of rockfish to estimate mortality rates of juveniles during their first year on the reef. I compared estimates of the mean density of recruits (<9 cm TL for blue rockfish; <7 cm TL for KGB) at each site to the mean density of 1-yr-olds (10–14 cm TL for
blue rockfish; 8–15 cm TL for KGB) estimated at that site in the following year. At scales of entire kelp beds, loss of rockfish is likely to be due to mortality and not migration because juvenile rockfish suffer high rates of predation (Hallacher and Roberts 1985, Adams and Howard 1996, Hobson et al. 2001) and exhibit little to no long distance movement (review by Love et al. 1991). Data were collected over a period of five years at six sites (each 500 × 70 m) along the coast of central California. Sites were located between northern Monterey Bay and northern Big Sur coast and encompassed a range of ~85 km of coastline. Each year, surveys were conducted during late summer, shortly after most KGB rockfish have recruited. Within each site, divers identified, counted, and visually estimated the total length of fish (to the nearest cm) encountered on a total of 24 120 m² transects that were stratified by four depth zones and two regions to ensure representative coverage of the whole kelp bed.

The instantaneous mortality rate for each cohort over its first year was calculated as the negative of the difference between the natural logarithm (x + 0.01) of 1-yr-olds and the natural logarithm (x + 0.01) of recruits. To detect temporal density dependence, I analyzed instantaneous mortality as a function of initial density (e.g., Dennis and Taper 1994), using observations in time as replicates. I used mixed-effects linear regression with site treated as a random factor and initial density as a fixed factor. This approach allowed me to conduct an overall test for temporal density dependence in mortality while explicitly accounting for site-specific variation in mortality (Pinheiro and Bates 2000). In my initial analyses, I also included the square of density as a fixed factor term to test for any significant non-linearity in overall patterns of density dependence. If nonlinear patterns were found in the data, then Akaike’s information criterion (AIC) was used to compare the fit of a linear regression of mortality on density to the fit of a model where mortality is linearly dependent on the log of density (a commonly used model that describes direct, yet decelerating density dependence; Dennis and Taper 1994). Since the presence or intensity of density-dependent mortality could vary among sites, I used a likelihood ratio test to determine whether a full model (including a site × density interaction) or a reduced model (common slope) was sufficient to explain site-specific variation in mortality.

RESULTS

Experimental test of density-dependent predation

Changes in the logarithm of the number of recruits remaining in each treatment yielded estimates of daily, per capita loss rates. Analysis of covariance indicated a strong difference in density-dependent loss (density × cage interaction, P = 0.014) between predator-exclusion and predator-exposed treatments, so each was analyzed separately. When predators were excluded, per capita loss was independent of density (Fig. 1) with an average loss of 40% over the duration of the experiment. In sharp contrast, in the predator-exposed treatments, per capita loss strongly increased with the initial density of recruits (Fig. 1), up to ~70% at the highest density treatments.

Over the 27-d experiment, there was a moderate amount of emigration from experimental plots. Tagged fish were occasionally recovered on nearby plots. Of the 134 fish lost from predator-exclusion plots, 24 (18%) were recovered on plots other than those to which they were stocked. Of these 24 emigrants, similar proportions (number of emigrants from each plot/initial number on that plot) came from each of the density levels, suggesting that emigration was not density dependent (P = 0.28). On the predator-exposed plots, 200 fish were lost, however only 13 (6.5%) were recovered. Again, the proportion of recovered emigrants was independent of initial density (P = 0.604).

The number of recruits lost to predation can be estimated by subtracting the estimated number lost to other causes from the total number missing from predator-exposed plots. If the presence of predators does not affect either the relative loss to emigration vs. other non-predation causes, or the percentage of emigrants recaptured elsewhere, then the approximate number of recruits lost to other causes from predator-exposed plots can be estimated as (number of recruits lost in predator-excluded plots) × (number of emigrants from predator-exposed plots)/(number of emigrants from predator-excluded plots) = 134 × 13/24 = 72.6. The approximate number of recruits killed by predators in predator-exposed plots is then 200 − 72.6 = 127.4.

It is unlikely that experimental densities (and associated loss rates) were affected by recruitment during the course of the experiment because recruitment was

![Fig. 1. Instantaneous per capita mortality of juvenile kelp rockfish from experimental units of habitat. Per capita mortality was density independent for the predator-exclusion treatments (dashed line and circles); linear regression: r² = 0.039, P = 0.67. When plots were exposed to predators, mortality increased with density (solid line, triangles); linear regression: r² = 0.949, P < 0.001.](image-url)
low (an average of 0.41 recruits per plot per day) and new recruits were removed within 1–2 d. Low replication was also unlikely to be a problem. Post hoc power analyses suggested that even weakly density-dependent trends in the predator-exclusion treatment would have been detectable: power to detect a density-dependent slope 0.5 and 0.25 times as strong as for the predator-exposed treatments was 0.999 and 0.754, respectively.

**Predator abundance and activity**

Within the experimental array, the most frequently observed predators were other, larger juvenile rockfishes including young-of-the-year (YOY) bocaccio, olive, and yellowtail rockfishes (*Sebastes paucispinus*, *S. serranoides*, and *S. flavidus*, respectively). Predators were present within the experimental array during each of 10 visual surveys conducted at twilight. The average number of predators visiting each plot varied slightly between days (0.49 ± 0.39 individuals/m³, mean ± SD), but no long-term trend in abundance was evident. Mean density of predatory rockfish on plots within my experimental array during twilight was slightly greater than density estimates from surveys of natural reefs (mean densities ranged from 0.005 to 0.26 individuals/m³). However, densities of predators on natural reefs were estimated on 30 × 2 × 2 m transects that were much larger than the experimental plots. Predators were often aggregated around structures such as large kelp plants and may be found in local densities similar to those on experimental plots.

Predatory species observed on experimental kelp plots were frequently observed preying upon KGB rockfish on natural reefs. Strike rates and strike efficiency for juvenile bocaccio were estimated as 0.48 ± 0.81 strikes/min and 0.054 ± 0.02 prey captured/strike (mean ± SE). Estimates of strike rate and efficiency for juvenile olive/yellowtail rockfish were slightly lower: 0.33 ± 0.52 strikes/min and 0.036 ± 0.023 prey captured/strike. Based on predator abundance and activity, a rough estimate of the number of fish that predators could be expected to consume over the duration of the experiment was calculated as: 7 predator-exposed plots × an average density of 1.8 predators/plot × 0.3 strikes/predator × minute × 0.03 prey captured/strike × 45 min of predator activity/day × 27 d, and totaled 137. This estimate, while only a rough approximation, was close to the estimates of predatory loss based on treatment differences and migration rates (127.4).

**Temporal density dependence in mortality**

Initial analyses indicated significant deviations from linearity in overall patterns of density-dependent mortality for KGB rockfish (*P* = 0.0277), but not for blue rockfish (*P* = 0.7295, Appendix). Subsequent analysis indicated that mortality of KGB rockfish was best described as a linear function of log-transformed density (AIC = 97.8), rather than density (AIC = 108.7). Although absolute rates of mortality varied somewhat among six large-scale sites, the patterns of density dependence appeared to be similar among sites for all species and an overall density-dependent increase in mortality was evident (Fig. 2). Likelihood ratio tests suggested that for both blue and KGB rockfish, a common slope model was sufficient to account for site-specific variation in mortality (*P* = 0.72 and *P* = 0.61, respectively). Analyses with linear, mixed-effects regression indicated that both types of rockfish experienced significant compensatory and temporally density-dependent mortality (*P* = 0.0027 for blue rockfish, *P* = 0.001 for KGB rockfish, Appendix).

**DISCUSSION**

This study provides evidence that mortality of juvenile rockfish is density dependent and that predation is the mechanism responsible. Field experiments demonstrated density dependence in loss rates only when
predators had access to plots of kelp. Loss from predator-exclusion plots was density independent, indicating no density-dependent loss due to emigration or intraspecific competition during the first month after settlement. Recovery of tagged migrants also suggested that there was no density dependence in emigration from both predator-exclusion and predator-exposed treatments. Behavioral observations strongly suggested that predation, rather than predator-induced migration was the cause of density-dependent loss. Predators that consume juvenile KGB rockfish at a high rate regularly foraged within the experimental array, estimates of expected predation during the experiment were similar to observed loss rates, and several observations were made of predators eating juvenile rockfish on experimental plots. Additionally, over 100 person hours of predator–prey observations suggest that the natural reaction of juvenile kelp rockfish when being pursued by predators is to immediately take cover in kelp, rather than escaping the predator in open water.

Although predation generated density-dependent mortality in this study, the exact mechanistic response of predators was unclear. Density-dependent mortality of prey can be caused by short-term behavioral responses of predators, such as an aggregative response, or an accelerating functional response (type III) via switching or some other mechanism (Murdoch and Oaten 1975). Additionally, there may have been a shortage of refuge space (Jeffries and Lawton 1984), with resulting competition among prey contributing to density-dependent predation. While I was unable to identify specific predator responses, some can be ruled out. In my experiments, only one prey type was present, precluding switching of preferred prey types as the cause of density-dependent mortality. For some reef fishes, a shortage of refuge space has been linked to density-dependent mortality (Holbrook and Schmitt 2002, Forrester and Steele 2004). It seems likely that refuge provided by kelp or reef structure can affect density-dependent mortality of rockfish. Aggregative responses of predatory fish have also been demonstrated (Hixon and Carr 1997, Anderson 2001), although separating effects of aggregative and functional responses on prey mortality has been difficult (but see Anderson 2001). While I did not assess aggregative responses, it appears that the major predator of KGB rockfish in this study, juvenile bocaccio, can exhibit a type III functional response (unpublished data).

Although the results of the small-scale experiments and field observations described here do not provide direct evidence that the temporally density-dependent mortality observed at large scales was driven by predation, I believe that predation is the most likely explanation. Density-dependent migration seems unlikely since juveniles of the species examined here exhibit very little, if any movement among kelp beds (Miller and Geibel 1973, Hoelzer 1988). Similarly, other potential causes of density dependence (disease, starvation) appear to have no appreciable effects on mortality of juvenile rockfish (Love et al. 1991). In contrast, many studies have documented heavy predation on juvenile rockfish (Hallacher and Roberts 1985, Adams and Howard 1996, Hobson et al. 2001). Some of these studies provide evidence that rockfish predators switch prey preference to feed on abundant prey (Hallacher and Roberts 1985, Hobson et al. 2001); however, it is unclear whether this increase in consumption was disproportionate enough to generate density-dependent mortality of prey (i.e., a type III functional response).

In this study, temporal density dependence in mortality was observed at a time scale consistent with short-term behavioral responses (i.e., over the first year in benthic habitat). If predation is responsible for the observed temporal density dependence in mortality, then it would likely be caused by predatory mechanisms that are short term (e.g., a type III functional response or a shortage of refuge space) rather than long term (e.g., an increase in number or biomass of predators). In this case, mechanisms generating temporal density dependence may be similar to those generating spatial density dependence. Spatial tests, which can isolate and describe causal mechanisms, may therefore provide important information about temporal density dependence, and thus population regulation. Understanding variation in causal mechanisms is crucial for understanding both spatial and temporal variation in population dynamics. Density dependence is often difficult to model and detect because of random variation (reviewed by Hixon et al. 2002, Turchin 2003). However, much of what appears to be random, or stochastic variation may actually be due to natural variation in the intensity of the causal mechanism. For example, the intensity of density-dependent mortality of fishes may vary with characteristics of the habitat (Wilson and Osenberg 2001, Shima and Osenberg 2003) and has been linked to predation and variation in the abundance of prey refuges (Forrester and Steele 2004). In extreme cases, density dependence may only be detectable and interpretable if causal mechanisms are known and variation in these mechanisms is well understood. In any case, a better understanding of causal mechanisms of density dependence will allow us to account for such variation, and thereby provide a clearer picture of density-dependent regulation.

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LITERATURE CITED


APPENDIX

A table summarizing the results of linear, mixed-effects regressions used to test for overall presence of temporal density dependence in mortality of juvenile rockfish (Ecological Archives E087-017-A1).