

Some consequences of size variability in juvenile prickly sculpin, *Cottus asper*

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Synopsis

Variation in size among fishes can have important ecological consequences. The origin and persistence of variation in size among a cohort of juvenile prickly sculpin, *Cottus asper*, was explored experimentally by manipulating the size variability in juvenile fish and the presence of conspecific adult fishes in a factorial design. I found that adult fish significantly suppressed the growth and survivorship of juveniles, while the variance treatment alone had no effect on growth or survivorship. The presence of adults was also associated with a decrease in size variability in the high variance treatment. An analysis of juvenile growth patterns revealed no evidence for size dependent growth or temporal correlations in growth ('growth autocorrelation'), and are consistent with a lack of amplification of size variance through time. Juvenile *C. asper* appear to have been feeding on a relatively homogeneous resource and utilizing those resources similarly. Thus, patterns of individual growth and the presence of adult conspecifics may both serve to dampen size differences in this coastal fish species.

Introduction

Variation in size among individuals is ubiquitous (Uchmanski 1985, Lomnicki 1988), especially in fishes where individuals of the same age can often differ markedly in size (e.g. Thorpe 1977, Elliott 1994, Wiegmann et al. 1997). It is essential to describe differences among individuals and understand the underlying mechanisms that generate size patterns for a number of reasons. Variability among individuals might alter the competitive environment if intrapopulation variation in resource use reduces intraspecific competitive effects (e.g. Van Valen 1965, Roughgarden 1972). Variation in size can also alter the representation of cannibalism in a population (DeAngelis et al. 1979), the amount of predation (e.g. Crowder et al. 1992), and the eventual reproductive capacity of the population (DeAngelis et al. 1979). Additionally, variability among individuals may affect our estimation of population-level responses such as extinction risk (Uchmanski 2000, Kendall & Fox 2002).

Differences in size among individuals that are established early in the life history can persist or be amplified (e.g. 'growth depensation', Ricker 1958) if growth is positively size dependent and if there are positive correlations in growth through time among individuals. The latter phenomenon is referred to as 'growth autocorrelation' (Pfister & Stevens 2002) and is the pattern of persistent growth differences among individuals that is independent of size. Similarly, differences among individuals in size can dampen through time (e.g. 'growth compensation', Ricker 1958) if growth is negatively related or unrelated to size and there is no growth autocorrelation among individuals. The pattern of growth autocorrelation can be the result of several mechanisms, including factors that are intrinsic to the organisms, such as genetic or behavioral traits that confer performance differences among individuals (e.g. Fraser et al. 2001). Alternatively, factors extrinsic to organisms, such as environmental heterogeneity, can drive persistent differences among individuals. One such well-studied example is variation in the light environment for terrestrial plants, where some

individuals have abundant light energy while others are shaded (Schmitt et al. 1986). Finally, in addition to size-dependent growth and the phenomenon of growth autocorrelation, size-dependent survivorship can further alter the changes in size structure through time.

In many fishes, especially temperate species, the seasonal establishment of new individuals (or recruitment) can occur over a period of days or months, resulting in a cohort of juveniles that vary greatly in size (Keast & Eadie 1984, Pfister 1997, 1999). Furthermore, the survivorship of a cohort and the rate at which they reach adult size can depend upon interactions with already established adults (e.g. Persson 1983, Sweatman 1985, Jones 1987, Forrester 1990, Persson et al. 2000). Here I examine experimentally how size variation initiated during the juvenile phase of a coastal fish species is propagated through time. I manipulated experimentally the variance in size structure of a cohort of juvenile fish in the presence and absence of adult conspecifics capable of a competitive and predatory role. I specifically test whether size-dependent growth or survivorship and growth autocorrelation contribute to changes in individual variation throughout the experiment.

Materials and methods

Site description

In coastal lagoons in the western U.S., the prickly sculpin, *Cottus asper*, reaches high abundances in the mid to late summer with the recruitment of young-of-the-year individuals. All experiments with *C. asper* were done in Cottoneva Creek, Mendocino County, California (39°44', 123°50') which forms a brackish-water lagoon area before emptying into the northeast Pacific Ocean. The abundance of young-of-the-year *C. asper* in the population could be so great that a 15 cm dip net swept along the bottom could routinely yield 5–30 juveniles in a couple of seconds. Perhaps due to variation in time of settlement (personal observation) as well as variation in time of hatching and growth conditions, young-of-the-year varied as much as 10 to 30 mm in length in August and September (personal observation). Based on gut content analyses, *C. asper* diet was predominantly midge larvae and adult midges that alight on the water surface, although larger individuals were cannibalistic. Other than *C. asper*, the only other fish species were the three-spine stickleback (*Gasterosteus aculeatus*) and steelhead trout (*Oncorhynchus mykiss*). Although *C. aleuticus* was

present in nearby coastal estuaries, I never observed any in Cottoneva Creek.

Experimental design

I tested how the presence of adults and the initial amount of variability in juvenile size affected juvenile performance and subsequent size variability in a 2 × 2 factorial experimental design where individual fish were contained in stream enclosures. The square enclosures were 0.50 m on each side and 0.21 m in height. They were constructed of 3 mm galvanized hardware cloth and held together with light gauge telephone wire. All enclosures were filled with 5–8 cm of estuarine gravel, the natural substrate from the study site. Enclosures were roofed and completely submerged in 1.0–1.25 m of water. I manipulated both the presence or absence of 2 large adult fish and the amount of variation in juvenile size with 8 juveniles present. The density of adult fish stocked in the enclosure was comparable to the density of adults I estimated snorkeling (4.2 adults m²), which was almost certainly a conservative estimate of density due to the cryptic nature of these fish.

In the low variation treatment all 8 juveniles were either 19 or 20 mm SL, while in the high variation treatment the 8 juveniles ranged in size from 17 to 25 mm SL. Thus, all enclosures had 8 juveniles and half of the enclosures had 2 adults (between 57 and 78 mm SL). Each treatment was replicated 6 times and all fish within each enclosure were individually marked using subcutaneous paint marks (Ceracoat™ acrylic paints) on the ventral side of fishes with a 26 G 1/2 needle and syringe. Prior to stocking the enclosures, each individual was measured to the nearest mm with a 15 mm plastic ruler and marked with a unique color code. All enclosures were stocked from 26–28 July 1995 and censused on 30 August and 2 September 1995, having been in the stream for 34–36 days total. All adults in enclosures were present and healthy for the entire interval, with 23 of 24 growing measurably in length.

The differential stocking of treatments led to the planned differences in initial juvenile size variation. In the low variance treatment, variability in standard length was estimated as the coefficient of variation (CV, or the standard deviation as a percentage of the mean) and ranged from 1.8 to 2.7, whereas variance in the mean (s²) ranged from 0.13 to 0.29. In contrast, the high variance treatment CVs ranged between 10.1 and 13.5

and s^2 was 4.1–7.9. Thus, both estimators of variability indicated an order of magnitude greater variation in size in the high variance treatment.

I used 2 factor ANOVA to test for effects of experimental manipulations on individual performance estimates such as size, growth, and survivorship. Survivorship data were arcsin square-root transformed for analysis. I also tested whether size variability estimates for juveniles differed from the beginning to the end of the experiment with an ANOVA on final CV and t-tests comparing the change in CV in each treatment.

To understand how different factors contributed to changes in size variance, I estimated whether growth and survivorship was size dependent in the juveniles, where growth was estimated from changes in SL. A linear regression was used to estimate the size and growth relationship, while a logistic regression was used to test whether survivorship was a function of size. I explored whether adults changed the effect of juvenile size on survivorship with a chi-square analysis of juveniles that survived or disappeared in the presence or absence of adults.

In order to estimate whether growth autocorrelations were present in this species, I established separate enclosures where individuals could be repeatedly measured. I stocked 3 enclosures with 24 individually marked juveniles (equivalent to amount of biomass in 2 adults and 8 juveniles) on 31 August 1994. The growth of these juveniles (ranging in size from 24 to 32 mm SL) was estimated in 2 subsequent monthly censuses. I estimated the strength of growth autocorrelation with a regression of growth in the second month against growth in the first month, in accordance with the methods in Pfister & Stevens (2002).

Results

Despite the large differences among treatments in the variability of juvenile size at the beginning of the experiment, the variance (as either CV or s^2) associated with those size distributions converged and was indistinguishable at the end of the experiment (Figure 1a and Table 1a). Despite this congruence in CV at the end of the experiment, there were significant differences among treatments in the CV when estimates at the beginning of the experiment were contrasted to those at the end. Not surprisingly, low variance treatments increased in CV (e.g. individuals differed by more than 1 mm at the end) regardless of whether adults were present ($t = 8.590$, $df = 6$, $p < 0.001$) or absent

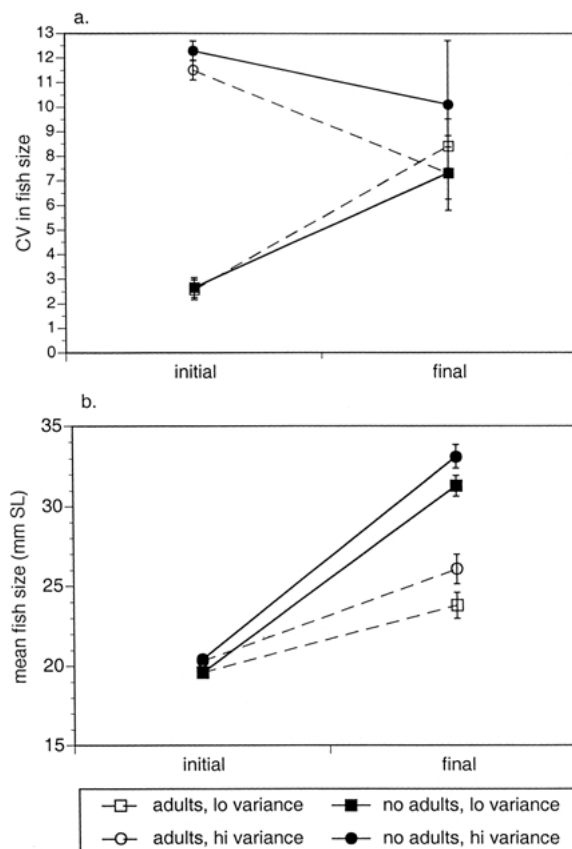


Figure 1. The mean CV in juvenile size (a) and the mean size of juveniles (b) at the start and end of the experiment in each of the four treatments. There was a significant effect of both adult presence and variance in juvenile size on mean size (Table 1b), while the CV in juvenile size was statistically indistinguishable among treatments at the end of the experiment (Table 1a). Because the number of individuals remaining within cages could be small, I used a CV estimate corrected for bias due to small sample size (Sokal & Rohlf 1995). Standard errors are shown; error bars are smaller than the symbols for the initial mean size measures.

($t = 2.727$, $df = 10$, $p = 0.021$). The high variance treatment, however, showed significant declines in CV when adults were present ($t = 4.176$, $df = 6$, $p = 0.006$), but showed no change in the absence of adults ($t = 0.939$, $df = 9$, $p = 0.372$).

Although variance converged in the enclosures, mean juvenile size diverged greatly among treatments (Figure 1b and Table 1b); juveniles in enclosures without adults were 29.1% larger, a result of juvenile growth rates that more than doubled when adults were absent (Figure 2a and Table 1c). ANOVA also indicated that the final difference in size between low and

Table 1. Summary of 2-way ANOVA for responses of marked *C. asper* juveniles to the presence or absence of adults and two different levels of variance in initial juvenile size. Bold values are significant. The figures corresponding to the data are referenced for each ANOVA.

Response	Source	df	Sum of squares	F	P
a. CV of mean Final size n = 15, $r^2 = 0.062$ (Figure 1a)	Adults	1	2.28	0.11	0.751
	Size variance	1	2.11	0.10	0.760
	Interaction	1	11.11	0.52	0.487
	Error	11	236.50		
b. Final size n = 18, $r^2 = 0.874$ (Figure 1b)	Adults	1	222.25	86.97	<0.001
	Size variance	1	17.58	6.88	0.020
	Interaction	1	<0.21	0.08	0.781
	Error	20	35.78		
c. Growth rate n = 18, $r^2 = 0.902$ (Figure 2a)	Adults	1	214.67	123.92	<0.001
	Size variance	1	1.28	0.74	0.404
	Interaction	1	1.01	0.58	0.457
	Error	14	14.25		
d. Survivorship n = 24, $r^2 = 0.322$ (Figure 2b)	Adults	1	1.01	9.45	0.006
	Size variance	1	<0.01	0.01	0.920
	Interaction	1	<0.01	0.05	0.824
	Error	20	2.14		
e. Number of immigrants n = 24, $r^2 = 0.579$	Adults	1	5985.04	27.27	<0.001
	Size variance	1	30.38	0.14	0.714
	Interaction	1	30.38	0.14	0.714
	Error	20	4389.17		
f. Total final Mass n = 24, $r^2 = 0.074$	Adults	1	34.95	1.51	0.233
	Size variance	1	1.20	0.05	0.822
	Interaction	1	0.85	0.04	0.850
	Error	20	462.80		

high variance treatments was significant, a result of an initial difference of 4.0% that persisted throughout the experiment.

Unchanging or decreasing size variability in the treatments with high initial variability could occur if: (1) growth was negatively size dependent such that as individuals grew larger, their length increments decreased, allowing smaller individuals to 'catch up' with larger individuals, (2) growth autocorrelations were weak or zero (Pfister & Stevens 2002), or (3) survivorship was size-selective.

There was no relationship between the size of juveniles at the beginning of the experiment and their growth rate during the experiment ($p = 0.179$, $F_{1,30} = 1.894$, $r^2 = 0.059$). Additionally, there was no evidence that growth rates were positively correlated among juveniles in any of the enclosures where juveniles were measured consecutively. Previous growth never explained a significant amount of the variation in subsequent growth (enclosure 1: $p = 0.890$, $F_{1,7} = 0.02$, $r^2 = 0.003$, enclosure 2: $p = 0.556$,

$F_{1,20} = 0.359$, $r^2 = 0.018$, enclosure 3: $p = 0.508$, $F_{1,10} = 0.483$, $r^2 = 0.046$, all enclosures combined: $p = 0.599$, $F_{1,41} = 0.281$, $r^2 = 0.007$). On average, individuals grew 1.8 mm in the first month ($SD = 1.6$) and 2.5 mm in the second month ($SD = 1.3$). As in the experiment described above, there was no evidence that the growth of juveniles was size dependent in the growth-monitoring enclosures ($p = 0.179$, $F_{1,42} = 1.870$, $r^2 = 0.043$).

Survivorship of juveniles declined by nearly a factor of 3 when adult fish were present (Figure 2b and Table 1c). In 5 out of the 12 enclosures with adults, no juveniles survived. In contrast, variability in initial juvenile size had no effect on survivorship, nor was there any interaction between the two factors. When all individuals from the high variance treatments were combined, logistic regression showed that survivorship was greater when juveniles were larger. However, this result was dependent upon the presence of adults. When adults were present, there was no effect of size on survivorship ($\chi^2_1 = 1.86$, $p = 0.172$), whereas

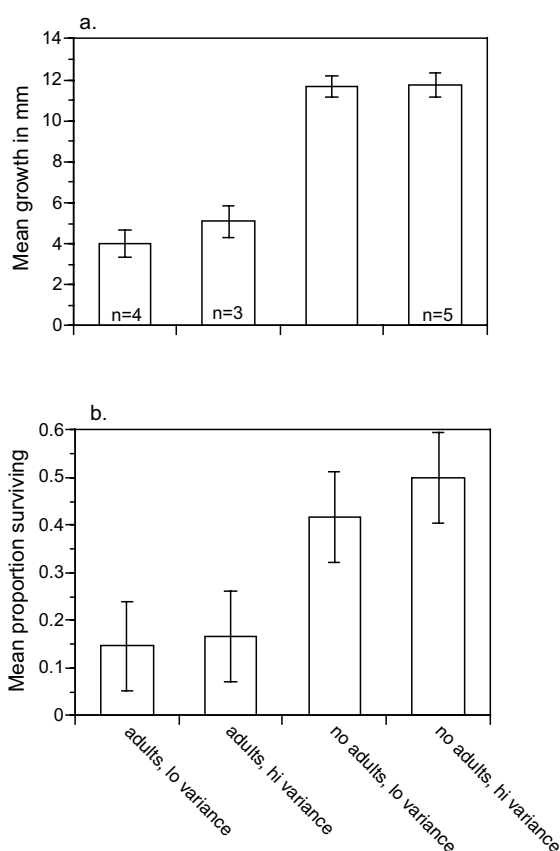


Figure 2. The mean juvenile growth rate (a), and the proportion of juveniles surviving (b) in each treatment. Error bars are standard error. For all variables, there was a statistically significant effect of adult presence. Corresponding statistical analyses for these figures are in Table 1c and d. As in Figure 1, corrected CV estimates were used. Unless indicated otherwise, each estimate is based on 6 replicate enclosures.

larger individuals had a survivorship advantage in the absence of adults ($\chi^2_1 = 8.17$, $p = 0.004$).

At the time the experiment was set-up, there were many immediately post-larval fish (approximately 8–10 mm SL) still recruiting. Some of these fish migrated into my enclosures and then grew to a size where they could not exit through the mesh (≥ 14 mm SL). Immigrant abundance was unrelated to the manipulation of variation in juvenile size, but was greatly affected by the presence of adults (Table 1e). Thus, immigrants were not responsible for the convergence in variance among treatments. When adults were absent, immigrants were more than an order of magnitude more abundant, with a mean of 33.8 in enclosures without adults, and only 2.2 in enclosures with adults. Although

immigrants were significantly smaller than marked fish ($x \pm se = 20.7 \pm 0.4$ mm SL), their high abundance in enclosures without adults increased the biomass in those cages such that the final mass in enclosures did not differ among treatments (Table 1f). Despite this, there were no significant correlations between mean growth or survivorship and the total biomass in an enclosure at the end of the experiment ($r = 0.198$, $n = 18$, $p > 0.10$ and $r = 0.145$, $n = 24$, $p > 0.10$, respectively). Therefore, immigrants were unlikely to be responsible for the treatment effects of adult fish on marked juveniles.

Discussion

Variation and juvenile performance

The presence of adults consistently explained more of the variation in juvenile performance than variability in the initial size of juveniles. Adults could negatively affect juveniles in a number of ways in Cottoneva Creek. First, adults can have a direct negative effect via cannibalism. Even if adults do not directly consume all juveniles, the risk of predation can be enough to decrease foraging by juveniles (e.g. Werner et al. 1983). A third mechanism by which adults can negatively affect juveniles is by being superior competitors. Larger fish might be capable of consuming a larger size range of prey and this increased niche width may give them an advantage over juveniles. Alternatively, social inhibition might have resulted from hierarchical interactions within an enclosure (e.g. Forrester 1990). The potential superiority of adults as competitors is suggested by the fact that immigrants to enclosures had no effect on marked juveniles, despite the equivalence of final fish biomass among enclosures (Table 1f). Thus, adult fish had strong effects on the growth and survivorship of juveniles in this system. Although some juvenile fish in reef environments have been shown to have relatively weak interactions with adult conspecifics (e.g. Steele et al. 1998), the strong effects of adult fish reported here are similar to those documented for other fish species (Persson 1983, Sweatman 1985, Jones 1987, Forrester 1990, Persson et al. 2000) and may be exacerbated by the high local abundances *C. asper* can achieve.

Although there are a number of mechanisms by which the variance treatment could have affected survivorship and growth, I found no evidence for them here. For example, the variance treatment could alter

the intensity of interspecific competition if individuals with a greater range of size ameliorated competitive effects relative to populations with individuals close in size (e.g. Van Valen 1965, Roughgarden 1972). There are several possible explanations for why variation in size did not result in changes in juvenile performance indicative of reduced intraspecific competition. First, competition among juveniles did not occur at the density I stocked cages. Second, intraspecific competition is possible but the size range that I used was not great enough to result in different resource use patterns among individuals. Third, the effects of the variance treatments were not persistent due to the convergence of size variance at the end of the experiment (Figure 1a). Although it is impossible to distinguish among these 3 reasons at this time, the latter 2 reasons may be the most likely, given that the diet of individuals at these small sizes overlap greatly and are almost exclusively midge larvae (personal observation), and given my initial variance treatments did not persist through time. The role that individual variability plays in ameliorating competition has conflicting empirical support. Two experimental studies using salamanders also found no link between individual variation and intraspecific competition. Despite size-based differences in prey resource use, Smith (1990) determined that variability in larval salamander size did not reduce intraspecific competition, and Brunkow & Collins (1996) found that density had far greater effects on larval performance than size variability. Perhaps only pronounced ontogenetic niche shifts can reduce competitive interactions within fish populations (Werner & Gilliam 1984, Osenberg et al. 1988, Olson 1996).

How is variation transmitted through time?

Although the experimental populations differed in size variation by greater than a factor of 4 (as CV) in the beginning of the experiment, they were statistically indistinguishable at the end (Figure 1a). It is not surprising that the variation in size increased in the low variance treatment, because any variability in growth should cause the size of individuals to increase beyond 1 mm of each other. For the enclosures with high initial variation, increases in variability can only occur if there is positive size dependent growth and positive correlations in growth through time (Pfister & Stevens 2002); neither mechanism was supported by the patterns of growth in *C. asper* and the resultant size patterns were consistent with growth compensation (Ricker 1958).

Thus, decreasing or unchanging variation through time is expected with *C. asper* and suggests that individuals shared similar traits related to foraging and were feeding on a relatively homogeneous resource. Size selective mortality is another mechanism by which size variability could change. For *C. asper* in the high variance treatment and isolated from adults, larger individuals were more likely to survive. Despite this slight advantage for larger individuals, CV patterns showed no change through time. When adults were present, there was no size-selective mortality and CV declined significantly through time. Thus, in the field, where adults are common, size variation in juveniles should not change due to survivorship patterns.

This study contributes to a relatively limited pool of knowledge about the patterns of individual variation among organisms and their underlying mechanisms, although there have been several studies that have documented the changes in size variance through time. Adults of the coral reef fish, *Pomacentrus amboinensis*, depressed juvenile growth and decreased overall variation in size, without affecting juvenile mortality (Jones 1987). Similarly, variation in individual size of a cohort of brown trout did not increase through time (Elliott 1994). Not surprisingly, *C. asper* shares patterns of individual variation with another coastal sculpin species, *Oligocottus maculosus*, which also shows no evidence of persistent correlations in growth (Pfister & Stevens 2002). In contrast, studies with other fish species suggest that growth correlations may be present. For example, otolith data for *Pomatomus saltatrix* suggest that there may be correlations in growth through time (Hare & Cowen 1997), although it is impossible to be certain until size effects are accounted for. Additionally the size bimodality that develops in some fish populations also suggests the presence of growth correlations (Thorpe 1997, De Angelis & Coutant 1982). The absence of growth autocorrelation does suggest that members of the population are encountering relatively homogeneous resources or are using available resources similarly. It may be possible that growth autocorrelation and the resultant amplification of size variability are enhanced in species where competitive hierarchies develop (e.g. Rubenstein 1981) or food is distributed unevenly (Magnuson 1962, Ryer & Olla 1996). Growth autocorrelation may also be more likely in species where ontogenetic prey switching facilitates continued faster growth of some individuals. For example, a switch to cannibalism was associated with faster growth rates in largemouth bass (De Angelis et al. 1979).

Because the nature of individual variation greatly affects population model choice (DeAngelis et al. 1993), the extinction risk of a population (Uchmanski 2000, Kendall & Fox 2002), and the genesis of size structure (Pfister & Stevens 2002), we need a better understanding of the origin of variability patterns among different taxa. Only further demographic and experimental studies among disparate taxa will reveal patterns of individual variation and any consequences to population trajectories.

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