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EVOLUTIONARY ANALYSIS OF LIFE SPAN, COMPETITION, AND ADAPTIVE RADIATION, MOTIVATED BY THE PACIFIC ROCKFISHES (*SEBASTES*)

Marc Mangel,[1,2] Holly K. Kindsvater,[3,4] and Michael B. Bonsall[5]

[1]*Center for Stock Assessment Research and Department of Applied Mathematics and Statistics, University of California, Santa Cruz, California 95064*

[2]*E-mail: msmangel@ucsc.edu*

[3]*Department of Zoology, University of Florida, Gainesville, Florida 32611, E-mail: hollyk@zoo.ufl.edu*

[5]*Department of Zoology, University of Oxford, South Parks Road, Oxford, United Kingdom, OX1 3PS, E-mail: michael.bonsall@zoo.ox.ac.uk*

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The Pacific rockfishes (*Sebastes* spp) are remarkable for both their diversity (on the order of 100 species) and range of maximum life span (∼10 years for Calico rockfish to ∼200 years for Rougheye rockfish). We describe the natural history and patterns of diversity and life span in these species and then use independent contrasts to explore correlates of these. When phylogenetic history is taken into account, maximum life span is explained by age at maturity, size at maturity, and the interaction of these two. We introduce a life-history model that allows insight into the origin of these correlations. We then describe a variety of mechanisms that may increase lifepans and diversity. These include fluctuating environments (in which organisms basically have to "wait out" bad periods to reproduce successfully), diversity, and longevity inspired by interspecific competition and physiological complexity in growth and accumulation of cellular damage. All of the results point toward the importance of flat or "indifferent" fitness surfaces as a key element in the evolution of diversity. We conclude that further development of the theory of flat or indifferent fitness surfaces as applied to diversity and life span is clearly warranted.

KEY WORDS: **competition, damage, diversity, life span, linear chains, phylogenetic contrasts, *Sebastes*.**

Rockfishes of the genus *Sebastes* are remarkably diverse (Love et al. 2002). This great diversity has apparently been achieved sympatrically in the northeast Pacific Ocean. Furthermore, some, but not all, members of the genus are exceptionally long-lived (Fig. 1). Indeed, the range of life span* in the rockfishes begs explanation: the life span of Calico rockfish (*Sebastes dalli*) is 12 years whereas that of Rougheye rockfish (*Sebastes aleutianus*) is estimated to be 205 years (We follow Wilmoth (1997): life span of an individual refers to that individual's age at death, life span of a species refers to the maximum potential length of life for the most robust members of the species, and mean life span is equivalent to life expectancy). There are other fish genera in which long-lived species exist (Beverton 1992). For example, spur dogfish (*Squalus acanthias*) lives up to 60 years at a size of 1 m; Beluga sturgeon (*Husa husa*) lives up to 118 years and achieves length of 5 m; white sturgeon (*Acipenser transmontanus*) may live up to 100 years; Icelandic-Norwegian herring (*Clupea harengus*) lives up

[4]Current address: Department of Ecology and Evolutionary Biology, Yale University, New Haven, CT 06511.

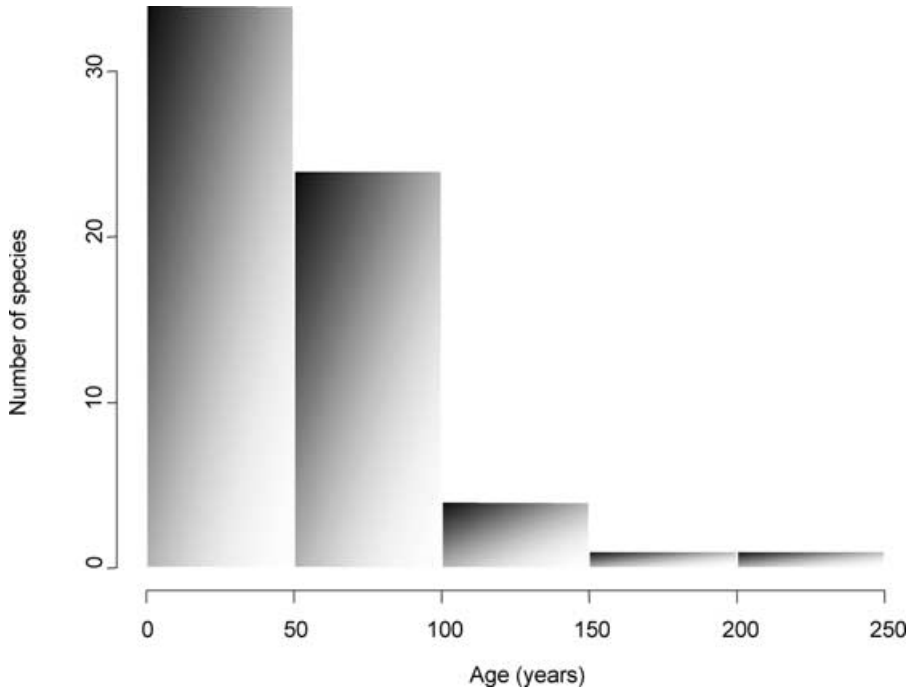

Figure 1. **Frequency distribution of age in the *Sebastes* (data from Love et al. 2002). In fish, life spans greater than 15 years are considered to be long-lived and greater than 50 years exceptionally long-lived (Beverton 1992).**

to 30 years, rarely exceeding 35 cm. It is well known that larger animals tend to live longer (e.g., Gavrilov and Gavrilova 1991; Ricklefs and Finch 1995). However, when compared with other Pacific species, rockfish live much longer than their length would predict from a simple interspecific comparison (Fig. 2): they are overage for their length.

Many species of *Sebastes* are very slow growing and have low rates of natural mortality (Ralston 2002). That is, if L(a) denotes size at age a and changes according to von Bertalanffy growth

$$\frac{dL}{da} = k(L_\infty - L), \tag{1}$$

where k is the rate of growth and L_∞ is asymptotic size, then k (measured in 1/yr) is much less than 1 (of the order of 0.05–0.15). If we denote the natural mortality rate (also measured in 1/yr) by μ so that survival of the next small interval of time Δa is approximately $1 - \mu\,\Delta a$ (see Mangel 2006), then survival to age a is $e^{-\mu a}$. For many rockfishes, $\mu \ll 1$ (of a similar order to k). Slow growth and low natural mortality have profound effects for the management of rockfish fisheries (Leaman and Beamish 1984; Leaman 1991).

Many rockfish species live in the California Current, which flows eastward from the Pacific toward North America and then southward along the coast. This environment is characterized by seasonal variability (Chelton et al. 1982; Lynn and Simpson 1987) and large-scale fluctuations (MacCall 1996; Watanabe and Nitta 1999) in which different environmental regimes persist for decades before an abrupt transition in environmental characteristics (particularly a switch between warm and cold water temperature and corresponding low and high productivity) occurs. Seasonally unpredictable environments are associated with high variation in spawning success. Murphy (1968) and Mann and Mills (1979) showed that there was a strong correlation between reproductive life span (and thus overall life span) and variation in spawning success (maximum observed reproductive success divided by minimum observed reproductive success).

Rockfishes have indeterminate growth (they grow after maturity) and internal fertilization (Yoklavich and Boehlert 1991; Love et al. 2002). It is likely that internal fertilization played a part in the radiation of the group, because internal fertilization promotes reproductive isolation in sympatry. In addition, many rockfish species have vivid and unique coloration patterns that are monomorphic among sexes, suggesting that coloration is important in species recognition (Love et al. 2002, p. 16). Rapid radiation of the species flock is supported by evidence of short genetic distances between species (Johns and Avise 1998; Rocha-Olivaires et al. 1999; Kai et al. 2003).

Rockfishes have a pelagic larval stage, ranging from one to three months, and subsequently recruit to adult habitat in which they acquire pigmentation. Juvenile stages may include several shifts in depth and diet, and thus in competitive environments (Love et al. 2002). Populations are putatively well mixed because of the dispersal phase although within-species genetic differences have been detected in Rougheye (*S. aleutianus)* and Rosethorn

rockfishes (*S. helvomaculatus*) from populations in the Gulf of Alaska and the coast south of Vancouver Island, B.C. (Love et al. 2002).

In general, as adults, rockfish are either demersal or midwater. Demersal species (e.g., *S. pinniger, S. nebulosus*) are sedentary bottom dwellers, associated with rocky reefs and pinnacles, and brightly colored, whereas midwater species (e.g., *S. entomelas, S. jordani*) are frequently in large schools above rocky reefs and pinnacles and usually gray, black, or brown in color. Sexual maturity occurs after three to 15 years. The increase in size after maturity can be substantial. For example, *S. melanops*, the Black rockfish, matures at 38 cm after six to 10 years, but eventually (maximum age 36 years) reaches 60 cm (Love et al. 2002). Ovary wet weight is a power function of length, with the exponent ranging between 2 and 5 (Gunderson 1997). Thus, larger fish produce greater numbers of offspring (Reznick et al. 2002), so that fitness may significantly increase as fish grow older. There is evidence that older individuals have an advantage in offspring quality as well as quantity. For example, older Black rockfish produce larvae with higher triglycerol lipid content than the offspring of younger females (Berkeley et al. 2004). Offspring of older mothers have dramatically higher growth rate and are more resistant to starvation. This increase in quality of offspring of older mothers further boosts fitness of older fish. There is no knowledge about reproductive senescence in the rockfishes, although skipped spawning (sensu Rideout et al. 2005) occurs. However, all indications are that there is an increasing contribution to the gene pool as fish become older.

Because the rockfishes are sympatric, we assume that there is (possibly intense) interspecific competition. Interspecific competition is a well-known ecological process that restricts diversity by preventing species occupying the same niche (Hardin 1960). It is also an important process in generating diversity through evolutionary responses (Bulmer 1974; Taper and Case 1985; Schluter 2000). Interspecific competition is the main driver of diversification through character displacement in evolution (Schluter 2000; Polechova and Barton 2005). However, the importance of the determinants of competition on coexistence requires careful consideration, and there is still disagreement about the range of conditions supporting sympatric speciation (e.g., Waxman and Gavrilets 2005, especially p. 1151 and following, Bolnick 2006).

In such a competitive environment, the growth rate of a population provides an appropriate and robust measure of fitness (Metz et al. 1992). Persistence requires that a species is able to increase whenever it is rare and coexistence requires that all species in an assemblage can resist exclusion (Pielou 1977).

Our hypothesis is that life span and competitive ability trade-off to affect diversity and diversification, in much the same way that dispersal and competitive ability trade-off in insect parasitoids

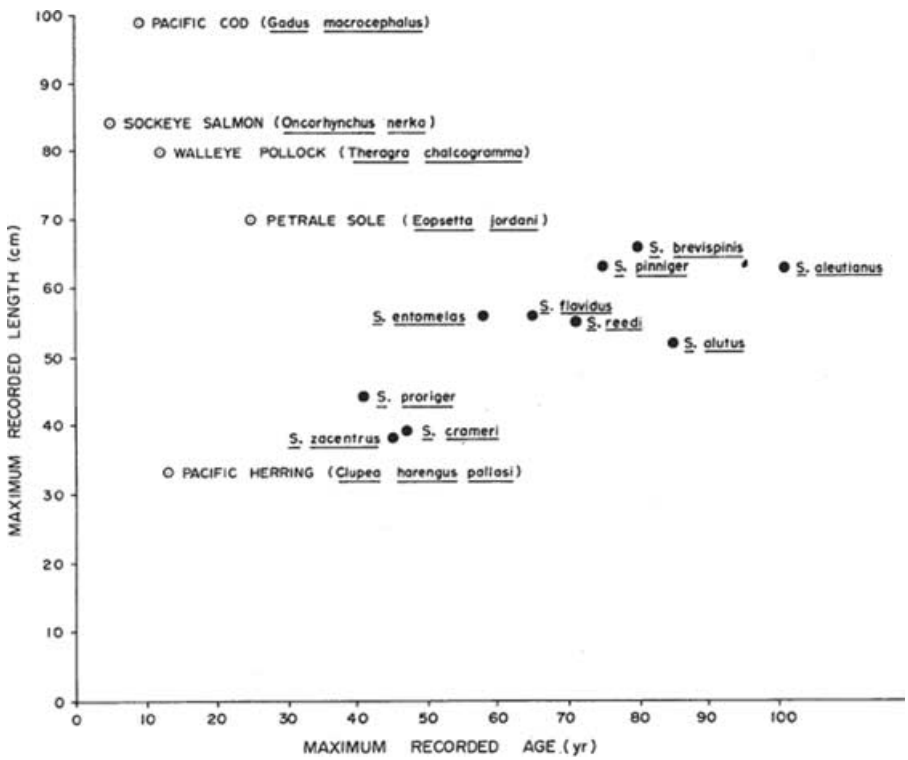

Figure 2. **An interspecific plot of size versus life span for Pacific fishes shows that *Sebastes* are overage for their length, or underlength for their ages. (Reproduced from Archibald et al. 1981).**

(Bonsall et al. 2004). To explore how competition-life span tradeoffs could lead to diversification, we consider a stylized interaction among rockfish competing for a limiting resource. There is no *a priori* partitioning of this resource, and individuals interact through competition mediated by life-history trait differences: species with similar life spans compete intensely, species with different life spans experience weaker competition because of less overlap of similar life-history stages.

Physiological structure and the evolution of demographic traits are inexorably linked. For instance, early experiments on guppies showed that life span can be influenced by the pattern of feeding (Comfort 1963). Continuous or intermittent feeding had a different effect on mortality trajectories (see Carey [2003] for a similar story about Mediterranean fruit fly). Guppy life spans were extended by 600 days under the intermittent feeding regime. Thus, phenotypically plastic responses to environmental processes are predicted to affect species diversity.

Here, we describe a number of analyses that alone or in combination may explain the remarkable diversity and life spans of the *Sebastes*. In doing so, we emphasize the importance of nearly flat (Bagnoli and Bezzi 1997) or "indifferent" (Orzack 1997) fitness surfaces for speciation and the need to link physiology and fitness in the construction of the adaptive landscape.

Our view is that aging is a process of the accumulation of damage from multiple sources (Harman 1956; Dobzhansky 1962; Finkel and Holbrook 2000; Gavrilov and Gavrilova 2002; Gems and McElwee 2005; Yin and Chen 2005). A major source of damage is reactive oxygen species (ROS) associated with environmental factors such as UVB and the processes of life itself (Rollo 2002; Metcalfe and Monaghan 2003), for which antioxidant defenses (AOD) are perforce not 100% effective (Barja 2002, 2004) because ROS are needed for signaling and immune response. Thus damage accumulates and causes aging (Rollo et al. 1996). However, the relationship between ROS production, AOD, and longevity is complicated (Perez-Campo et al. 1998; de Grey 2000; Barja 2002b).

Our philosophy in the modeling is to introduce the simplest model that will do that job at hand. For example, to begin we assume that mortality is characterized by a single rate, as shown below equation (1).We then consider the case in which mortality is a distributed function that represents the molecular, cellular, and physical damage accumulated through physiological processes (Mangel and Bonsall 2004). This distributed mortality can be described in a standard way through a linear chain of ordinary differential equations (MacDonald 1978; Mangel and Bonsall 2004).

In Methods, we first describe the statistical analysis of life-history correlates of life span. We then describe a very simple extension of the von Bertalanffy growth model (1) that can be used to explain the statistical results. That model can be expanded by the method of linear chains to model more explicitly growth in size and accumulation of damage that determines mortality. We then extend Beverton's (1992) theory of growth–maturity–longevity (GML) for populations in fluctuating environments, to investigate how a fluctuating environment may select for diversity in species and life spans. We then move from single-species models without density-dependent effects to multispecies models that show how competition may lead to diversity in species and life spans. We use the method of linear chains (MacDonald 1978) to add structure to the intensity of competition and particularly explore the importance of the strength of competition for the evolution of diversity. We provide a variety of insights about the importance of understanding flat or indifferent fitness surfaces for diversification.

Methods

STATISTICAL ANALYSIS OF THE CORRELATES OF LIFE SPAN

We first consider the physiological explanation that life span is determined by the basal metabolic rate (BMR) of a species, which is in turn affected by the physical characteristics of the habitat in which the species lives. A fish living in deep water survives with no light (and consequently less food) at colder temperatures and lower oxygen levels, all of which may contribute to reduced BMR and extended life span.

A second explanation is that selection on other life-history traits acts on life span as a correlated trait. In general, mortality in the marine environment scales inversely with size (Lorenzen 1996, 2005). Early age at maturity will slow growth and have additional potential costs so that smaller size at maturity will generally lead to smaller asymptotic size (Stamps et al. 1998). Thus, we expect high risk of extrinsic mortality to correspond with early age at maturity or small size at maturity.

We examined the correlation of physical and life-history variables with life span of *Sebastes* species using independent contrasts (Purvis and Rambaut 1995), which allowed us to control for lineage effects and to treat each species as an independent data point (Felsenstein 1985; Harvey and Pagel 1991). We used a published maximum parsimony tree based on cytochrome-*b* data for 55 species (Rocha Olivares et al. 1999) and assumed equal branch lengths. We calculated contrasts using CAIC (Purvis and Rambaut 1995). We chose maximum depth, temperature, and dissolved oxygen concentration at that depth, age at maturity, size at maturity, and body size as explanatory variables, with maximum age as the dependent variable. We treat age at maturity (the age at which there is a 50% probability of maturity) and size at maturity as independent variables across species because the solution to the von Bertalanffy growth equation (1) involves asymptotic size, growth rate, and a constant of integration. Thus, the same age at maturity may correspond to different sizes at maturity and vice versa.

We obtained data from oceanographic buoy data from the live access server of the Pacific Fisheries Environmental Laboratory (NOAA, 2003) and from published life-history values for each species (Pikanowski et al. 1999; Love et al. 2002). We computed average values of temperature and dissolved oxygen concentration over the Northeast Pacific region (latitude 26–63°N, longitude 112–170°W) from winter 2001. Depth, temperature, and dissolved oxygen concentration are highly correlated variables, so we used the first principal component of a PCA for these variables, which we called *physical environment.* We logarithmically transformed variables (Freckleton 2000; Quader et al. 2004).

We also analyzed the raw values in a conventional analysis, with the caveat that these results are indistinguishable from lineage effects. Both sets of data were analyzed in a multiple regression, with simplification of the model by stepwise removal of nonsignificant explanatory variables ($P \leq 0.05$).

A LIFE-HISTORY MODEL FOR THE CORRELATES OF LIFE SPAN

We adapt the work of Stamps et al. (1998) and Mangel and Stamps (2001). In particular, we rewrite equation (1) and assume that if a_m is age at maturity then for $a < a_m$

$$\frac{dL}{da} = q - kL, \tag{2}$$

so that if the organism never matured asymptotic size would be $L_\infty = q/k$. Here q is the maximum growth rate (i.e., growth rate at small size).

Given size at maturity $L_m = L(a_m)$, growth after maturity follows the dynamics (Stamps et al. 1998; Mangel and Stamps 2001)

$$\frac{dL}{da} = q - (k + \frac{b}{L_m})L, \tag{3}$$

so that asymptotic size depends on the size at maturity $L_\infty = \frac{q}{k+\frac{b}{L_m}}$. Here b captures the growth cost of maturity. These equations are easily solved analytically (Mangel 2006).

We assume that mortality is size-dependent so that if survival to age a is $S(a)$ then (numerical coefficients from Lorenzen 1996, 2005)

$$\frac{1}{S}\frac{dS}{da} = -3L(a)^{-0.864}. \tag{4}$$

We define life span to be the age at which survival falls below 0.01%.

PHYSIOLOGICAL COMPLEXITY TO GROWTH AND MORTALITY

We add structure to our life-history models through the use of delay differential equations. These have the advantages of parameterizations of instantaneous rates of mortality, reproduction, and development and direct connection to obvious biological variables. Most of the difficulties in the use of such equations come from the numerical solution of them (see Gurney et al. 1983; Nisbet and Gurney 1986; Caswell et al. 1997; Nisbet 1997 for more details). More particularly, here we focus on the dynamics of changes of the length or mass of individuals and on the dynamics of changes of populations (which create the environment that individuals experience; see de Roos [1997] for a more general treatment and Bonsall and Mangel [2004] and Mangel and Bonsall [2004] for specific applications to aging and life span).

We replace equation (1) by a growth chain (MacDonald 1978; Bonsall and Mangel 2004; Mangel and Bonsall 2004)

$$\begin{aligned}
\frac{dL}{da} &= k(w_0 - L), \\
\frac{dw_i}{da} &= \beta\,(w_{i+1} - w_i)\,, \\
\frac{dw_q}{da} &= \beta(L_\infty - w_q),
\end{aligned} \tag{5}$$

which is characterized by two additional parameters: the length q and the rate parameter β of the chain. The interpretation of equation (5) is that food in the environment is not immediately converted into a change in size but must be converted through a series of irreversible steps, with intermediates characterized by the w_i, into a change in size. As $\beta \to \infty$ we recover the usual von Bertalanffy growth equation. Equation (5) is supplemented by initial conditions $L(0) = w_0(0) = l_0$ and $w_i(0) = 0$ for $i = 1, \ldots, q$.

Next we assume that survival is determined by accumulated molecular and cellular damage at age a, $D(a)$. For the computations reported here we used

$$S(a) = \frac{1}{1 + \left(\frac{D(a)}{D_{50}}\right)^\gamma}, \tag{6}$$

where γ and D_{50} are parameters. Qualitatively similar results are obtained for other functional relationships between damage and survival. A chain with length p and rate parameter α characterizes the accumulation of damage

$$\begin{aligned}
\frac{dD}{da} &= v_0, \\
\frac{dv_i}{da} &= \alpha\,(v_{i+1} - v_i)\,, \\
\frac{dv_p}{da} &= \alpha\left(\mu_0 + \mu_1 L(t)^b + \mu_2 \frac{1}{L(a)}\frac{dL}{da} - v_p\right).
\end{aligned} \tag{7}$$

The terms on the right-hand side of the last equation account for exogenously caused damage, damage due to metabolism (which scales allometrically with length) and damage due to growth. The initial conditions are $D(0) = d_0$ and $v_i(0) = 0$ for $i = 1, \ldots$ p.

We assume that fecundity is an allometric function of length (so that fecundity is AL^B) and that density-dependent processes

can be ignored. Thus, the solution of the Euler–Lotka equation, which gives the natural rate of increase, provides a measure of fitness (Fisher 1930/1999; Charlesworth 1994). Explicitly noting that survival to age a, $S(a, \alpha, p)$, depends on the parameters of the damage chain and that length at age a, $L(a, \beta, q)$, depends on the parameters of the length chain, the Euler–Lotka equation is

$$1 = \sum_{a=0}^{\infty} e^{-rt} S(a, \alpha, p)\{AL(a, \beta, q)\}^B. \quad (8)$$

Fitness $r = r(p, q, \alpha, \beta)$ thus depends on all four parameters.

Without further constraints, a Darwinian demon emerges. That demon is characterized by $\beta -> \infty, q -> 0$ in the length chain and $\alpha -> 0$, $p -> \infty$ in the damage chain. That is, food is converted into growth as rapidly as possible and with the shortest number of intermediary steps and sources of mortality are expressed as slowly as possible and through as many intermediary steps as possible. We thus constrain the chains by $\beta = \kappa\,\alpha$, $\kappa < 1$ and $q = p + \Delta p$, $\Delta p > 1$: the rate of the length chain is slower than the rate of the damage chain, and the number of intermediary steps in the length chain is greater than the number of intermediary steps in the damage chain.

SINGLE-SPECIES POPULATION GROWTH IN FLUCTUATING ENVIRONMENTS

We extend Beverton's GML theory of life span in fish (Beverton 1963, 1987, 1992; Beverton and Holt 1959) to include (i) varying environments and (ii) a proximate mechanism for reproduction. The key aspect of Beverton's theory is the relationships between k, μ, and maximum life span ($A_{\max}$). Beverton (1992, Table III) reports a range of 6.5–13.5 for $kA_{\max}$ and a range of 0.2–0.4 for μ/k. For computations, we use

$$kA_{\max} = 10.0$$

and

$$\mu = 0.3k. \quad (9)$$

Thus the rates of growth and maximum life span are inversely related (e.g., Pearl 1928), and mortality is proportional to the rate of growth (as would happen in the case of foraging under predation risk, growth leading to damage, or both).

We assume that weight and length are related by an allometric relationship $W(a) = AL(a)^B$, where A and B are fixed parameters (Table 1) and where length is the solution of equation (1) in the form

$$L(a) = L_\infty(1 - e^{-k(a-a_0)}), \quad (10)$$

where $a_0 < 0$ captures the early life history of the fish in the sense that initial size is $L(0) = L_\infty(1 - e^{ka_0})$. The parameters we use are consistent with those of Gunderson (1997).

Table 1. Variables and parameters for the model relating fluctuating environment and life span

Symbol	Interpretation	Value
E(t)	Environment in year t	1 or 2 (index)
L_∞ [$E(t)$]	Asymptotic size when the environment in year t is E(t)	50 cm [$E(t) = 1$] 25 cm [E(t) = 2]
L_0	Initial size	0.4 cm
w_0	Initial mass	Determined allometrically
A	Allometric scaling constant relating mass and length	0.05
B	Allometric exponent relating mass and length	2.8
P	Period of the regimes in the environment	30 years
ε	Scaling factor for the reproductive cost of somatic growth	0.05
$k_{\max}$	Growth rate at which reproduction drops to 0	0.35
G_{thr}	Threshold level of gonads needed for reproduction	143
$s[E(t)]$	Offspring survival when environment is E(t)	0.0009 [E(t) = 1] 0.00045 [E(t) = 2]

Asymptotic size is a function of the environment. In particular, the environment in year t, $E(t)$, is either good [$E(t) = 1$] or bad [$E(t) = 2$] and is a two state Markov process. Thus

$$\Pr[E(t+1) = j | E(t) = i] = p_{ij}, \quad (11)$$

where $i, j = 1$ (good) or 2 (bad). For simplicity, we assume that

$$p_{ii} = \frac{P-1}{P}, \quad (12)$$

where P is approximately the period of the environmental regime Starting with $E(0) = 1$, equations (11 and 12) are used to simulate a sequence of environmental states [$E(1), E(2), E(3), \ldots$].

Because the environment fluctuates, length is a function of both age and time, which we denote by $L(a,t)$. The von Bertalanffy dynamics written as a discrete time iteration become

$$L(a+1, t+1) = L(a, t+1)e^{-k} + L_\infty[E(t)](1 - e^{-k}) \quad (13)$$

to which we add the proviso that fish are not allowed to shrink. Individuals could thus lose condition, since mass will decline even if length does not.

In this model, fish accumulate gonads and reproduce when the accumulated gonads cross a genetically determined threshold (sensu Thorpe et al. 1998). If a fish either grows or maintains length, then we assume that its gonads at age a and year t, $G(a, t)$, are incremented according to the rule

$$G(a+1,t+1) = G(a,t) + 0.03W(a)\left(1 - \varepsilon\frac{k}{k_{\max}}\right), \quad (14)$$

so that gonadal accumulation follows the same allometry as mass, and there is a cost for faster growth where $k_{\max}$ is the rate of somatic growth at which the accumulation of gonads drops to 0. Reproduction occurs in a year when accumulated gonads exceed a threshold G_{thr}.

We let

$$N(a,t) = \text{Number of individuals of age a at the start of year } t. \quad (15)$$

Then for all age classes except the birth age class

$$N(a+1,t+1) = N(a,t)e^{-\mu}, \quad (16)$$

where μ is the rate of mortality. The size of the birth class is given by

$$N(0,\ t+1) = s[E(t)]\sum_{a=1}\frac{G(a,t)}{w_0}I_{G(a,t)-G_{thr}}N(a,t), \quad (17)$$

where $s[E(t)]$ is survival from larvae to age class 0 when the environment is $E(t)$, w_0 is birth mass, determined by an allometric relationship using birth length and I_z is an indicator function that is 1 if $z > 0$ and 0 otherwise. The total population in year t is given by

$$N_T(t) = \sum_{a=0} N(a,t). \quad (18)$$

The geometric mean growth rate (Tuljapurkar 1989; Easterling and Ellner 2000; McNamara 2000) characterizes the fitness of the population. We ran the model for 200 years, to eliminate transients, and used that as the initial population distribution for another 1000 years to estimate the averages needed to compute fitness. Once we determine maximum age, other life-history parameters are fixed and we are able to compute geometric mean growth rate (fitness) associated with that life span.

COMPETITION INSPIRED DIVERSITY AND LIFE SPAN

We study diversity and life span driven by competition using structured population models and delay differential equations. To do this, we separate mortality rate μ and maximum life span τ. Clearly the latter is a convenient fiction, since no reasonable person would expect that an individual could live to age τ but not to $\tau + \tau_0$, where τ_0 is arbitrarily small (see Wilmoth [1997] and references therein). However, as will be seen things are complicated enough with a fixed maximum life span. Roughly, one may think that mortality rate is driven by external factors (e.g., predators) and maximum life span by internal factors (e.g., the accumulation of molecular and cellular damage).

Changes in mass m_i of the i^{th} species follow (Bonsall and Mangel 2004)

$$\frac{dm_i}{dt} = \frac{a_i m_i^{3/4}}{1 + \sum_j \gamma_{ij} m_j^{3/4} N_j} - b_i m_i, \quad (19)$$

where γ_{ij} is the strength of competition between strategies i and j, N_j is the size of the j^{th} population, and a_i and b_i are the rate of uptake of resource and rate of resource utilization for the i^{th} strategy. The presence of both conspecifics and competitors slows growth. Note that if $\gamma_{ij} = 0$ for all j, then the dynamics are simply $\frac{dm_i}{dt} = a_i m_i^{3/4} - b_i m_i$ and the transformation $H_i = m_i^4$ leads one to a linear equation similar to equation (1) for $H(t)$ (see Mangel and Bonsall 2004 for further details).

The population dynamics of the i^{th} species follows

$$\frac{dN_i}{dt} = r_i N_i - \left[\mu_i + \sum_j \gamma_{ij} m_j^{3/4} N_j\right] N_i - S_i(t,\tau_i), \quad (20)$$

where r_i is the intrinsic rate of increase, μ_i is the natural mortality rate, and $S_i(t, \tau_i)$ is the rate of removal of individuals surviving to the end of life

$$S_i(t,\tau_i) = r_i N_i(t-\tau_i)\exp\left(-\int_{t-\tau_i}^{t}\left[\mu_i + \sum_j \gamma_{ij} m_j^{3/4}(x)N_j(x)\right]dx\right). \quad (21)$$

Note that mortality has both density-independent and density-dependent components, with the latter determined by the biomass of competitors and the strength of competition, determined by the closeness of traits. We assume μ_i and τ_i covary and determine the strength of competition

$$\gamma_{ij} = \gamma_{jj}\ \exp\left(-\frac{1}{2(1-\rho^2)}\left[\left(\frac{\mu_i - \mu_j}{\sigma_\mu}\right)^2 - 2\rho\frac{\mu_i - \mu_j}{\sigma_\mu}\frac{\tau_i - \tau_j}{\sigma_\tau} + \left(\frac{\tau_i - \tau_j}{\sigma_\tau}\right)^2\right]\right), \quad (22)$$

where ρ is the correlation between the traits, and σ_μ and σ_τ govern the width of the competition window. The fitness of an initially rare mutant invading a resistant assemblage is

$$R_i = \frac{1}{N_i}\frac{dN_i}{dt} = r_i - \left[\mu_i + \sum_j \gamma_{ij} m_j^{3/4} N_j\right] - r_i \exp\left[-(\mu_i + \sum_j \gamma_{ij} m_j^{3/4} N_j)\tau\right] + o(\tau), \quad (23)$$

where $o(\tau)$ denotes higher order powers of τ.

ADDITIONAL STRUCTURE IN COMPETITION

To investigate how additional structure in competition may affect diversity and life span, we replace equations (20 and 21) by a

chain

$$\frac{dN_i}{dt} = r_i N_i - \left[V_0(t) + \sum_j \gamma_{ij} m_j^{3/4} N_j \right] N_i - S_i(t, \tau_i), \quad (24)$$

$$S_i(t, \tau_i) = r_i N_i(t - \tau_i) \exp\left(- \int_{t-\tau_i}^{t} \left[V_0(x) + \sum_j \gamma_{ij} m_j^{3/4}(x) N_j(x) \right] dx \right), \quad (25)$$

and

$$\begin{aligned} \frac{dV_z}{dt} &= \lambda_z (V_{z+1} - V_z); z = 0, 1, 2, \ldots p-1 \\ \frac{dV_p}{dt} &= \lambda_z (\mu_i - V_p). \end{aligned} \quad (26)$$

Here, λ_z is the rate and p the length of the chain characterizing mortality. A change in mortality (μ_i) has to propagate through the chain to influence a change in cohort size. Larger values of p imply longer time lags between a change in baseline mortality and a change in cohort size, representing a more complex physiology. Increases in λ_z imply a faster response by the cohort to change in the underlying mortality representing changes in cellular biochemistry, metabolism, or function. The evolutionary dynamics are analyzed using invasion and replacement analysis (Bonsall and Mangel 2004).

THE IMPORTANCE OF THE STRENGTH OF COMPETITION

In equation (22), the strength of competition diminishes as the difference between the life-history traits (e.g., $|\mu_j - \mu_i|$) grows. Other things being equal, larger σ_μ means broader (in trait space) and more intense competition, smaller σ_μ implies narrower and less intense competition (also see Bolnick 2006). We first ask how does variation in σ_μ affect the likelihood of strategies with different (but fixed) life spans invading and co-occurring. Second, we explore how changes in the correlation ρ between life-history traits (μ and τ) affect the co-occurrence of alternative strategies.

To leading order in τ, an alternative strategy invades if

$$r_i - \left[\mu_i + \sum_j \gamma_{ij} m_j^{*3/4} N_j^* \right] - r_i \exp\left[-(\mu_i + \sum_j \gamma_{ij} m_j^{*3/4} N_j^*)\tau \right] > 0. \quad (27)$$

The response of fitness to changes in the nature or width of the competition window depends on the selection differential. For a fixed magnitude of difference in life-history traits, this is given by

$$\begin{aligned} \frac{dR_i}{d\sigma_\mu} = & -\frac{1}{\sigma_\mu^3}(\mu_i - \mu_j)^2 \gamma_{jj} m_j^{*3/4} N_j^* \\ & \bullet \exp\left[-\exp\left(-\frac{1}{2}\left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right)^2 \right) \gamma_{jj} m_j^{*3/4} N_j^* \tau_i \right. \\ & \left. - \mu_i \tau_i - \frac{1}{2}\left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right)^2 \right] \\ & \bullet \left(\exp\left[\tau_i \exp\left(-\frac{1}{2}\left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right)^2 \right) \right. \right. \\ & \left. \left. \times \gamma_{jj} m_j^{*3/4} N_j^* + \mu_i \right] + r_i \tau_i \right), \end{aligned} \quad (28)$$

where $m_j^{*3/4}$ and N_j^* are the mass and abundance of the resident j^{th} strategy at equilibrium.

The response in fitness to changes in ρ is evaluated from the selection differential

$$\begin{aligned} \frac{dR_i}{d\rho} = & -\exp\left(-\frac{\eta}{2(1-\rho^2)} \right) m_j^{*3/4} N_j^* \vartheta \\ & + \exp\left[-\exp\left(-\frac{\eta}{2(1-\rho^2)} \right) \gamma_{jj} m_j^{*3/4} N_j^* \tau_i \right. \\ & \left. - \mu_i \tau_i - \frac{\eta}{2(1-\rho^2)} \right] \gamma_{jj} m_j^{*3/4} N_j^* r_i \tau_i \vartheta, \end{aligned} \quad (29)$$

where

$$\eta = \left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right)^2 - 2\rho \frac{\mu_i - \mu_j}{\sigma_\mu} \frac{\tau_i - \tau_j}{\sigma_\tau} + \left(\frac{\tau_i - \tau_j}{\sigma_\tau} \right)^2 \quad (30)$$

and

$$\vartheta = -\frac{\rho \eta}{(1-\rho^2)^2} + \frac{1}{1-\rho^2} \left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right) \left(\frac{\tau_i - \tau_j}{\sigma_\tau} \right). \quad (31)$$

Results

STATISTICAL ANALYSIS OF THE CORRELATES OF LIFE SPAN

The final model for the independent contrast values had three significant variables: age at maturity, size at maturity, and the interaction with age at maturity and body size (Fig. 3A). The final model for the raw values had two significant variables: environment and body size (Fig. 3B), suggesting that selection on life span is determined by both physical environment and predation. Furthermore, although the rate of living explanation is confounded by common descent, it may be an important factor in explaining life span in ancestral *Sebastes*.

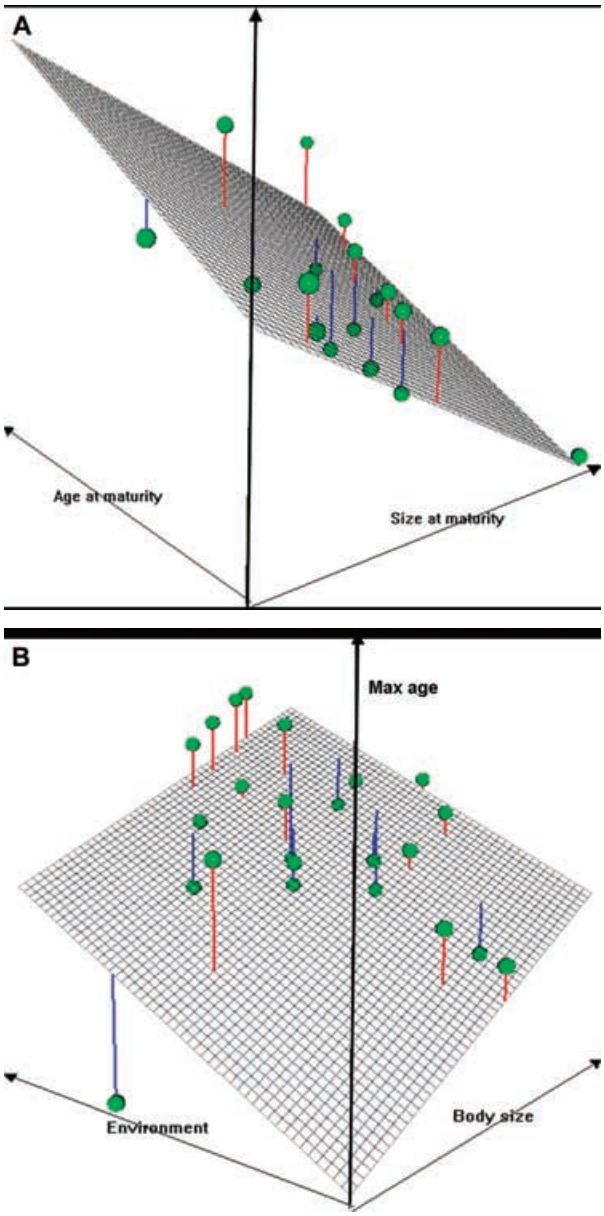

Figure 3. **(A) The final model for the independent contrast values has three significant variables: age at maturity, size at maturity, and the interaction between age at maturity and body size. Because there are only two significant predictor variables, we can draw the predicted model as a plane, showing the linear regression model (determined by multiple regression with stepwise removal). The orientation of the plane is set for convenience of viewing. The points, either above or below the prediction, represent the predicted traits of the ancestor of a group of species. (B) The final model for the raw values had two significant variables: environment and body size. The interpretation is the same as in panel (A), except that now each point represents a species.**

A LIFE-HISTORY MODEL FOR THE CORRELATES OF LIFE SPAN

Equations (2–4) allow us to determine life span as a function of age at maturity or size at maturity and show that life span is positively correlated with size and/or age at maturity (Fig. 4). Were we to draw L_∞, a_0, or k from distributions (as might occur for different species), we would introduce both scatter and correlation into these results.

PHYSIOLOGICAL COMPLEXITY TO GROWTH AND MORTALITY

With κ and Δp fixed, fitness is a function of α and p. In Figure 5A, we show the combinations of rate of the damage chain and length

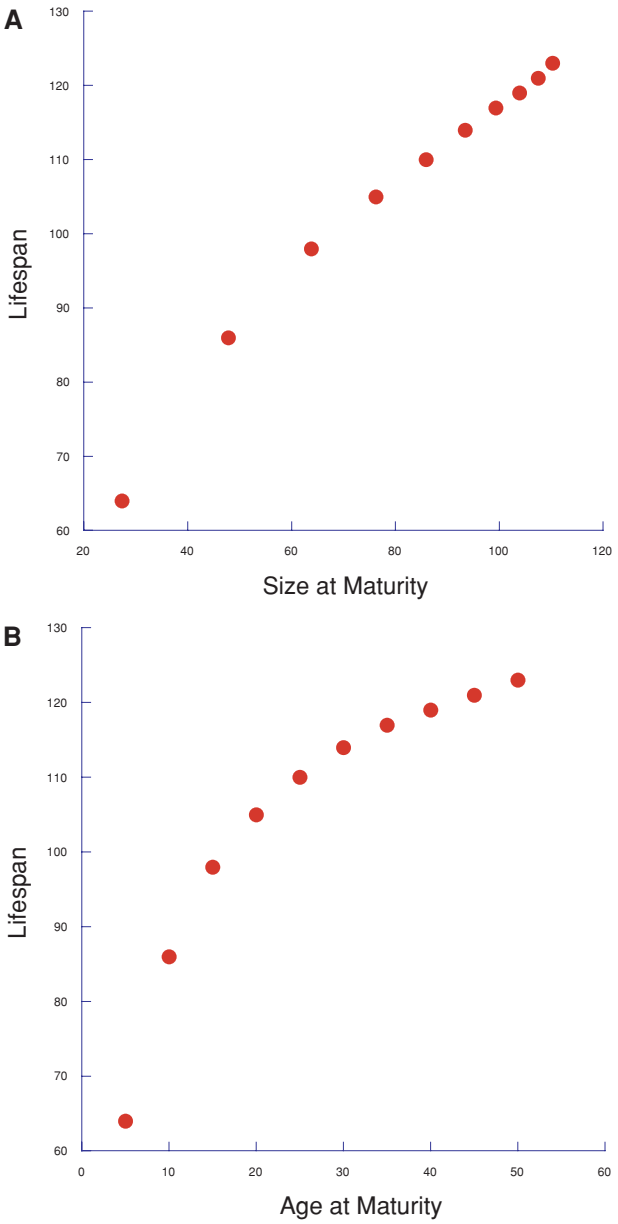

Figure 4. **If the growth cost of maturity depends on the size of maturity (as in eq. 3) and mortality is size-dependent (as in eq. 4), we predict a positive correlation between life span (defined as the age at which survival drops to less than 1%) and size at maturity (A) or age at maturity (B). For these calculations, we used $L(0) = 1.0$, $k = 0.05$, $q = 6$ (so that in the absence of maturity, asymptotic size is 120), and $b = 2.5$.**

of the damage chain that lead to values of fitness that are at least 99% of the optimal (indicated by the $*$ in the figure). A wide variety of combinations of rate and length of the chain lead to very high levels of fitness—the fitness surface is very flat and thus indifferent (sensu Orzack 1997) to values of the parameters. The different damage chains with very high levels of fitness lead to different growth trajectories (Fig. 5B). In generating this figure, for a given value of the rate parameter of the damage chain we used the smallest length chain that gives fitness at least 99% of optimal. Associated with these growth trajectories are survival curves. As a measure of life span, we use the age at which survival drops to 1% (Fig. 5C); the parameters giving indifference generate a nearly threefold variation in age to 1% survival.

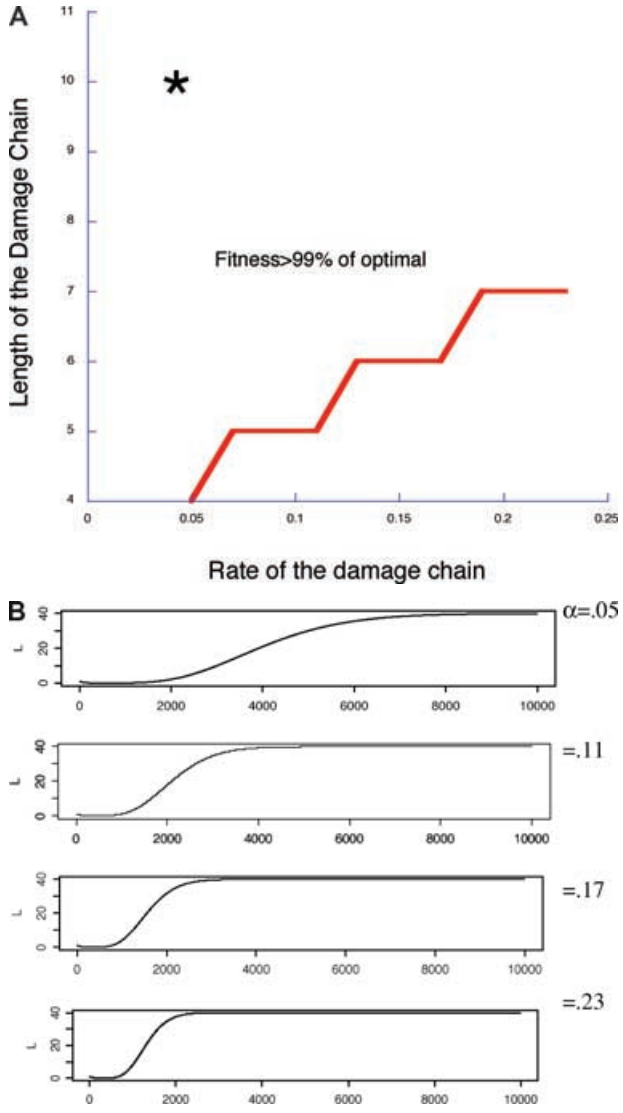

Figure 5. Linked linear chains that describe size and damage lead to fitness surfaces that are flat. (A) The values of α and p that maximize fitness (marked by $*$) and the boundary in (α, p) space for which fitness is at least 99% of optimal. (B) The growth trajectories for values of α and the smallest value of p that produces fitness at least 99% of optimal. (C) This method generates longevities that vary by a factor of nearly 3. Parameters are $\gamma = 0.5$, $\kappa = 0.7$, $\Delta p = 3$, $L_\infty = 40$, $L(0) = 1$, $\mu = 0.1$, $A = 0.045$, $B = 2.85$, $\mu_1 = 20\mu/(L_\infty)^B$, $\mu_2 = 20\mu L(0)/(BkL_\infty)$, $D(0) = \mu/35$. We integrated the equations using a step of $dt = 0.05$.

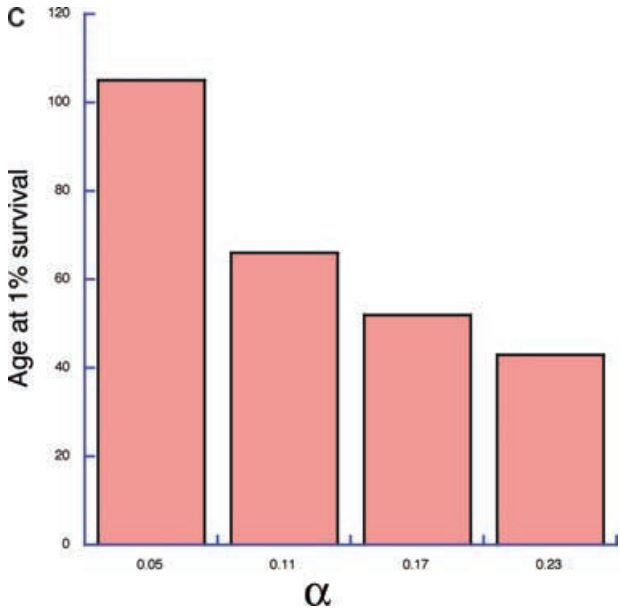

Figure 5. Continued.

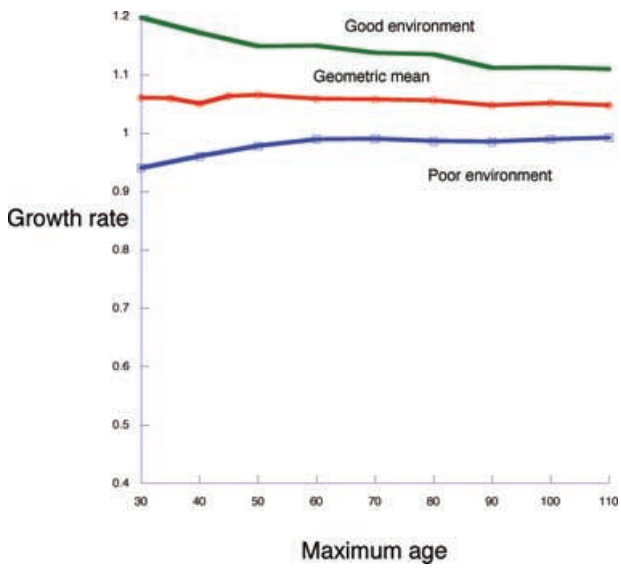

Figure 6. A fluctuating environment and a life-history invariant in which life span is inversely related to the von Bertalanffy growth rate lead to a broad and relatively flat region in which geometric growth rate is greater than 1 as long as maximum age is sufficiently large. When maximum age falls below 30 years (the period of the approximate period of the environment), the geometric mean of λ drops rapidly. For example, it is 0.42 when maximum age is 20 years.

SINGLE-SPECIES POPULATION GROWTH IN FLUCTUATING ENVIRONMENTS

The key result is shown in Figure 6. In a constant but bad environment, there is essentially no choice of life span that allows persistence of the population because all population growth rates are less than 1—although longer life spans with slower growth and lower mortality creep up toward $\lambda = 1$. In a constant but good environment, short-lived species have a selective advantage, in the sense that λ declines as maximum age increases (and thus growth rate and generation time decline). As noted by Hamilton (1966), there is an advantage of early reproduction, when such reproduction is more or less guaranteed, so that early senescence is more strongly favored in populations in which growth rate is positive. However, the geometric mean is remarkably flat, fluctuating between about 1.045 and 1.06 as maximum age ranges from 30 to 110 years. One interpretation of the selective forces acting in this manner is that individuals cannot replace themselves in a bad environment. In consequence, there is strong natural selection to extend life span so that individuals who are born in bad environments will be able to spend at least part of their reproductive period in good environments.

COMPETITION INSPIRED DIVERSITY AND LIFE SPAN

We show how the fitness surface depends on μ and τ in Figure 7. The total variation in R_i depends on both μ and τ, so that the ESS conditions are determined from the total differential

$$dR_i = \frac{\partial R_i}{\partial \mu} d\mu + \frac{\partial R_i}{\partial \tau} d\tau. \tag{32}$$

More explicitly, we have

$$dR_i = -[1 - \exp(\theta \nu_\mu) - \exp(-\theta\tau - \mu\tau) r_i(\theta\tau\nu_\mu - \tau)] d\mu$$
$$-[\exp(-\theta\tau - \mu\tau) r_i(\theta\tau\nu_\tau - \mu - \theta) + \theta\nu_\tau] d\tau, \tag{33}$$

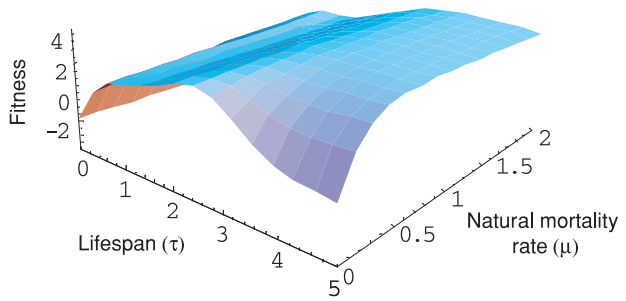

Figure 7. Fitness surface for correlated changes in invader natural mortality rate μ_i and life span τ_i. Fitness is determined by the combined effects of trade-offs in mortality and life span on competitive ability. The surface is relatively flat—multiple strategies have equivalent fitness and coexistence is likely. Optimal fitness depends on negative covariance in life-history traits.

where

$$\nu_\mu = \frac{\dfrac{2(\mu_i - \mu_j)}{\sigma_\mu^2} - \dfrac{2\rho(\tau_i - \tau_j)}{\sigma_\mu \sigma_\tau}}{2(1-\rho^2)}$$

$$\text{and} \quad \nu_\tau = \frac{\dfrac{2(\tau_i - \tau_j)}{\sigma_\tau^2} - \dfrac{2\rho(\mu_i - \mu_j)}{\sigma_\mu \sigma_\tau}}{2(1-\rho^2)} \tag{34}$$

and θ is the trade-off function scaled by the equilibrium abundance of the resident strategy

$$\theta = \gamma_{jj} \exp\left(-\frac{1}{2(1-\rho^2)} \left[\left(\frac{\mu_i - \mu_j}{\sigma_\mu} \right)^2 \right.\right.$$
$$\left.\left. -2\rho \frac{\mu_i - \mu_j}{\sigma_\mu} \frac{\tau_i - \tau_j}{\sigma_\tau} + \left(\frac{\tau_i - \tau_j}{\sigma_\tau} \right)^2 \right] \right) N_j^* m_j^{*3/4}. \tag{35}$$

Covariance in life-history traits leads to a relative broad, flat fitness surface and maximum fitness is predicted when μ is large and τ is long (Fig. 7). Clearly, this is not intuitive and would be difficult to observe because survival to maximum life span is $\exp(-\mu\,\tau)$. Our results show, however, that it is a situation that can arise as a result of selective processes.

We thus predict the co-occurrence of alternative strategies, which is confirmed through numerical simulation (Fig. 8). Correlated changes in trait values between μ and τ lead to different patterns of diversification (Fig. 8). Negative covariance in traits leads different strategies that are tightly clustered in trait-space (Fig. 8A). In contrast, positive (or no) covariance between μ and τ leads to diverse strategies with different survival probabilities (Fig. 8B,C).

ADDITIONAL STRUCTURE IN COMPETITION

The fitness surface around the minimum and maximum points is broad and flat (Fig. 9). Flat fitness surfaces around the minima allow fitness to increase in many different directions in trait space, thus allowing the invasion by many different life histories. Divergent selection around this point could promote phenotypic divergence in trait values. The fitness maximum is found at a broad part of the surface. Selection around this point is likely to be stabilizing and allow several strategies with similar life histories to co-occur. Pairwise invasion dynamics reveal boundaries of coexistence (Fig. 10).

Numerical solutions show coexistence of species with different physiologies can occur through mutation and replacement dynamics (Fig. 11). Strategies with different physiologies may persist for long periods of time, and the number that co-occur might exceed the diversity observed in the terminal assemblage. In addition, different co-occurring strategies give rise to different mortality trajectories: differences in physiology give rise to heterogeneous mortality trajectories (Mangel and Bonsall 2004) within and between strategies (Fig. 11).

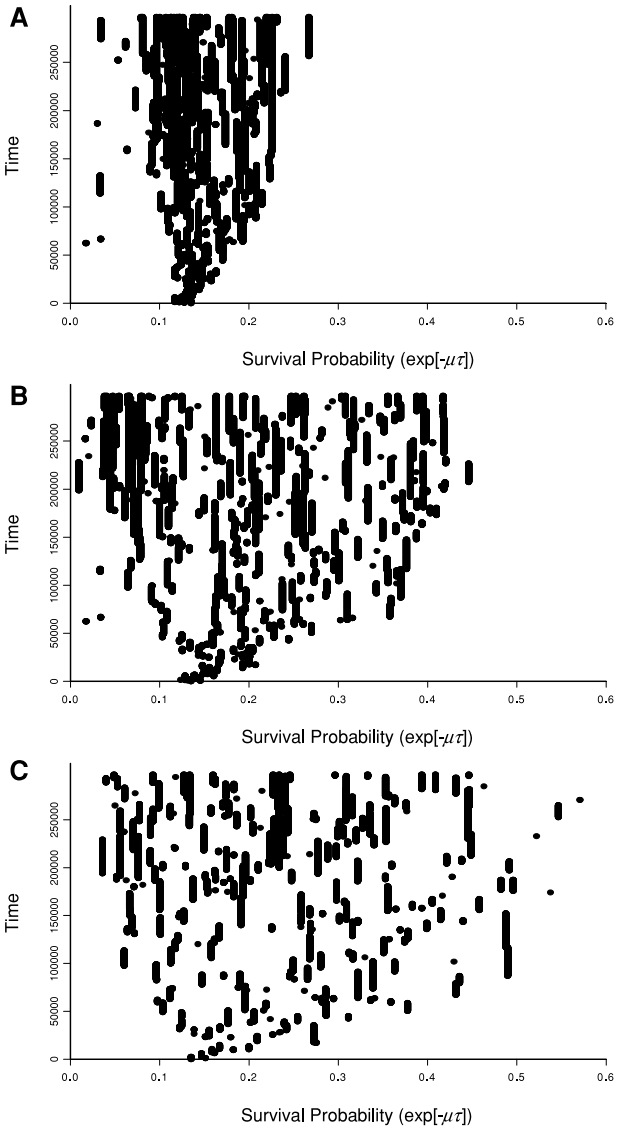

Figure 8. Evolutionary dynamics and coexistence of strategies under different trade-offs represented in terms of survival probabilities exp ($-\mu\tau$) for correlated changes in traits: $\rho = -0.75$ (panel A) $= 0$ (panel B) or $= 0.75$ (panel C).

THE IMPORTANCE OF THE STRENGTH OF COMPETITION

Fixed strategy differences respond differently (nonparallel slopes) to changes in the breadth of competition (σ_μ) (Fig. 12). Numerical simulations confirm that the width of the competition window is crucially important to diversity and diversification (Fig. 13). Narrow windows of competition imply strong, myopic competition between alternative strategies with very similar life histories, whereas weak competition exists between widely divergent strategies. In contrast, broad competition captures all invading strategies and is global. Although the former promotes diversity, the latter does not (Fig. 13). Multiple strategies with negative correlations

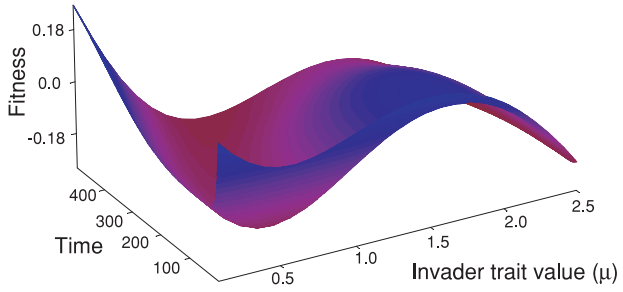

Figure 9. Fitness surface for changes in invader mortality rate μ_i through time. Optimal fitness depends on how mortality is distributed (complexity of physiology) in both invader and resident strategies. Time-dependency in fitness reflects the dynamic interaction between physiological, invasion, and evolutionary dynamics. (Also see Bonsall 2006.)

in these life-history traits suggest that coexistence of multiple life histories may be possible (Fig. 14). The selection differential confirms that the magnitude of selection for correlations in the range $[-1, -0.75]$ is close to zero when fitness in this boundary is positive (Fig. 14).

Discussion

Evolutionary maintenance of diversity is essentially a phenomenon determined by the underlying shape and topology of the fitness surface. In particular, flat (Banoli and Bezzi 1997) or indifferent (Orzack 1997) fitness surfaces are predicted to favor diversity as different strategies solve the life-history allocation problem in different ways to achieve equivalent fitness. The correlation between mortality rate (a proxy for the inverse of mean life span) and maximum life span plays a crucial role in shaping both diversity and life span.

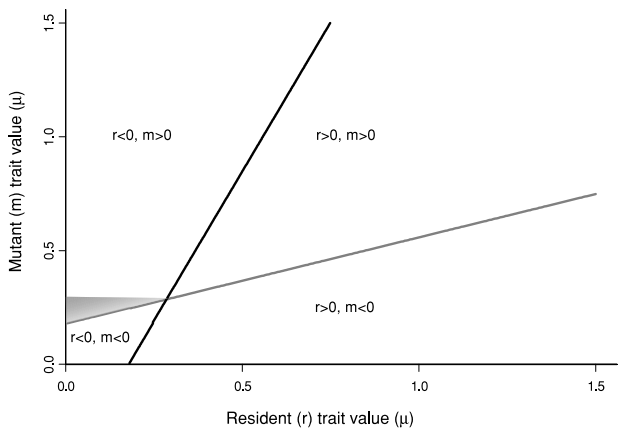

Figure 10. Pairwise invasion boundaries for strategies with distributed mortalities. Invasion is successful if the invading (*m*) strategy outcompetes the resident (*r*) (above the gray line or to the right of the black line). Both long- and short-lived strategies with distributed mortality are predicted to co-occur (Bonsall 2006).

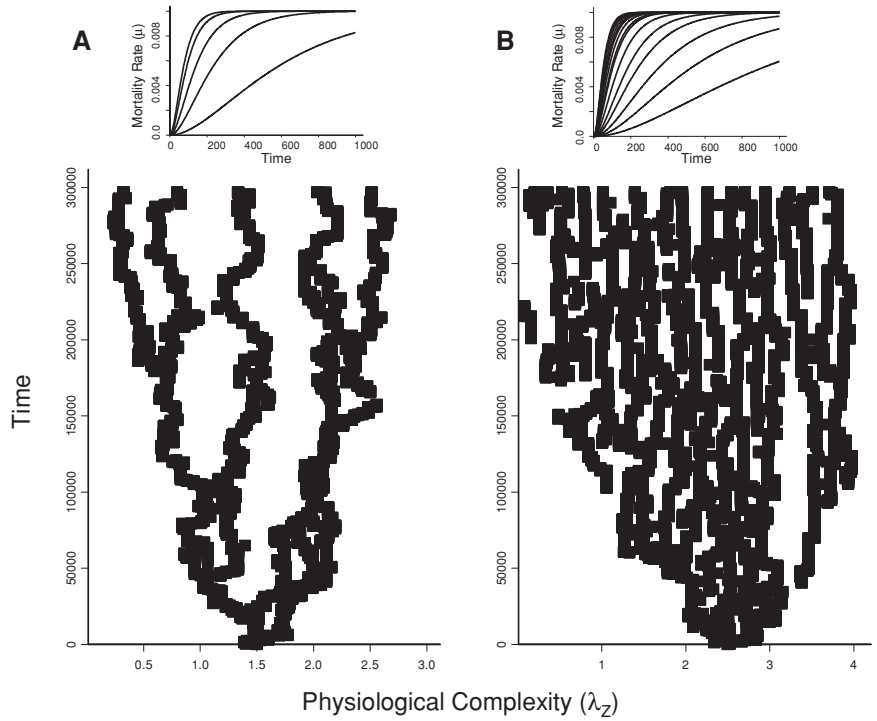

Figure 11. Evolutionary dynamics and coexistence of strategies with different physiologies λ_z. Evolution of physiology governs how mortality cascades through the life cycle of the strategy and λ_z measures the ability to deal with damage (high λ_z is indicative of low ability to deal with damage and conversely low λ_z is indicative of high ability to deal with damage). The insert shows that different strategies have different mortality rates (Bonsall 2006). The rate of mutation is 0.05 in A and 0.1 in B.

The evolution of life span necessitates a shift in life history and physiological trade-offs. Our phylogenetic analysis and theoretical predictions show that size at maturity is an explanatory variable for maximum life span and not the converse. Late maturation leads to longer-lived individuals who are buffered from high rates of extrinsic mortality by larger size. In guppies, Reznick et al. (2004) show that although populations that experience higher extrinsic mortality mature earlier, the onset of senescence is not correlated with either extrinsic mortality or reproduction. They argue that more derived theories or approaches to understanding the evolution of senescence are necessary. In particular, the time has come to focus on internal mechanisms of aging, to explain, for example, why such guppies die in the laboratory in the absence of predators or disease. To say that they die because they senesce is insufficient. Our work using damage shows how one may approach this question.

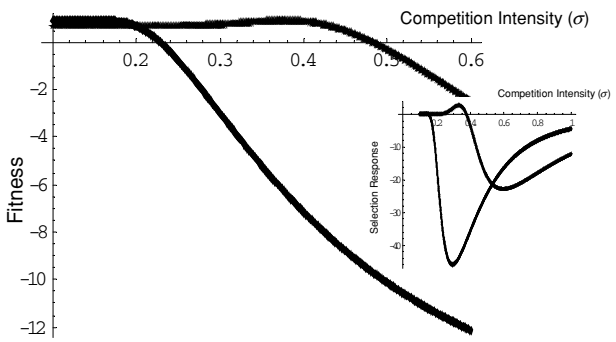

Figure 12. Effects of the intensity of competition σ_μ on invader fitness. The fitness surface is relatively flat over much of the range of competition intensities for fixed differences in invader and resident trait values $\mu_i - \mu_j$ (in the upper lines, the difference $\mu_i - \mu_j$ is large [0.5] and the lower lines it is small [0.05]). The insert shows the fitness response $\frac{dR_i}{d\sigma_\mu}$ to selection is specific to the underlying difference in traits (nonparallel slopes) and this response confirms that multiple strategies have equivalent fitness because $\frac{dR_i}{d\sigma_\mu} \sim 0$.

Selection can act both within and between strategies to lead to the evolution of longer life spans. Within species selection experiments on *Drosophila melanogaster* show that restricting reproduction to later in life consistently extends life span (Rose and Charlesworth 1980; Partridge et al. 1999). However, this necessitates that the maintenance of somatic function to be traded off against other physiological (e.g., reproductive capacity—Kirkwood 1977) or ecological (e.g., competitive ability—Levins and Culver 1971) processes. Investment in competitive ability is one mechanism through which evolution to greater life span is feasible. Life-history trade-offs and resource allocation decisions cascade to affect an organism's ability to defend itself, forage for food, and resist infection; these can be manifest as

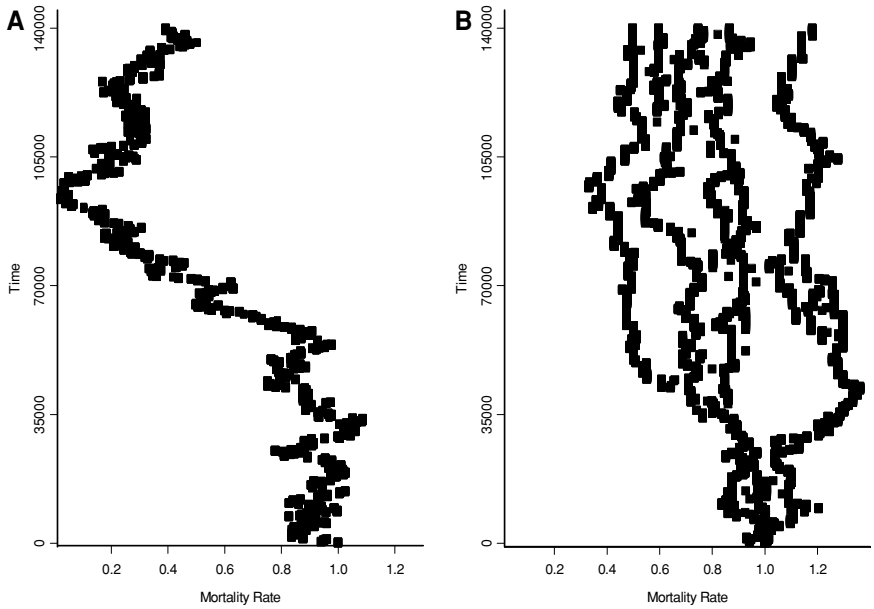

Figure 13. Evolutionary dynamics under (A) high ($\sigma_\mu = 0.75$) and (B) low ($\sigma_\mu = 0.5$) competition windows. Intensity of competition among different strategies governs the likelihood of coexistence and can affect the overall diversity of a terminal assemblage.

ecological trade-offs in competitive ability (Levins and Culver 1971). Here, we have explored how a competition-life span trade-off could facilitate coexistence. Covariance in natural mortality and maximum life span in a structured population setting leads to a broad, relatively flat fitness surface: there a multiple ways of achieving the same fitness by trading mortality against competitive ability.

Local competition promotes ecological coexistence among relatively sessile organisms (e.g., Murrell and Law 2003) but how the structure of the competition kernel might affect the evolutionary consequences of life span remains relatively unexplored. Bagnoli and Bezzi (1997) suggest that increasing the intensity of short-range competition over a trait space is more likely to favor speciation events. Our results confirm that for particular covariances in trait space, short-range competition is more likely to facilitate diversification: negative covariances are more likely to favor the invasion of novel, alternative strategies. Moreover, selection on maximum life span is a nonlinear function of population size, suggesting that changes in population abundance or dynamics might have as an important effect as changes in life histories. Appreciating the extent of these nonlinear interactions at hierarchical scales is essential to understanding how life span evolves (Mangel 2002). In the northeast Pacific Ocean, many shorter-lived species (such as sardines and anchovies) exhibit large geographical variation in response to fluctuations in the climate (MacCall 1990). Individuals are highly mobile, early maturing, and short-lived. We may envision that the strength of competition between resident and invading strategies is intense across a wide range of trait space. On the other hand, for the rockfishes, individuals are more sedentary and the net effect may be intense competition across a narrower range of trait space, allowing for the persistence of more diversity in life histories.

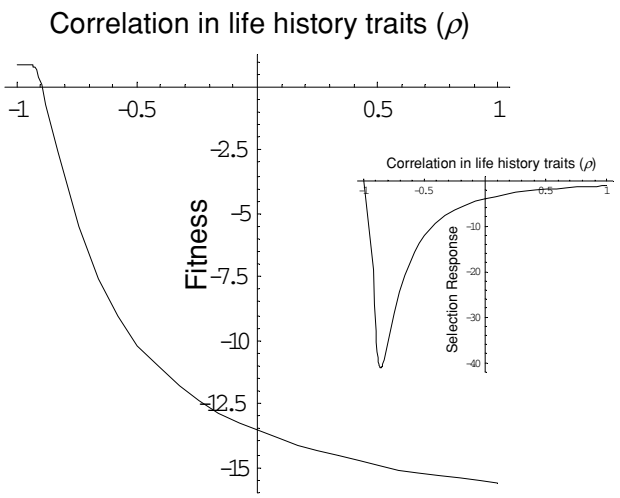

Figure 14. Effects of covariance ρ in life-history traits on invader fitness. Positive fitness is observed when there is strong negative covariance between μ_i and τ_i. As correlation in traits increases, fitness declines.

Investment strategies are physiologically costly (Zera and Harshman 2001), and different allocation patterns have evolutionary consequences on the variability in life histories observed within a species (e.g., Metcalfe and Monaghan 2001; Mangel and Munch 2005) and between species (Bonsall 2006). Although physiological complexity can lead to diversification mediated by

flat fitness surfaces, selection acting at the physiological level (through complexity in growth and mortality) is critically important to the evolution of diversity through life-history traits such as life span. For instance, the likelihood of canalization (Waddington 1957; Schlichting and Pigliucci 1998) as a mechanism for flat fitness surfaces at the developmental and/or physiological level is entirely possible. How the effects of genetic canalization (epigenetic effects), the formation of flat fitness surfaces and environmental fluctuations all interact to affect diversification through the evolution of life span remains relatively unexplored.

In general, species that show indeterminate growth are predicted to invest in processes that allow them to live longer. This necessitates being able to deal with variability in physical environments, natural enemies, and competitors. Highly variable environments are likely to favor phenotypic plasticity and promote a range of optimal responses to different environmental circumstances (Kirkwood and Austad 2000). Although this effect is known to affect a wide range of organisms (Klass and Hirsh 1976; Metcalfe and Monaghan 2001), the between-species implications and consequences for diversification have remained relatively unexplored. We have shown that fluctuating environments are likely to favor flat fitness surfaces suggesting that species that live in close proximity can have highly disparate lifestyles.

We have shown that the maintenance of diversity and the evolution to longer life spans in the rockfishes is likely to be driven by life-history evolution giving rise to flat fitness surfaces. A variety of mechanisms may be at work and that these may be density independent or density dependent. In the former case, fluctuating environments or physiological complexity are the keys to the evolution of diversity and life span. In the latter case, the strength of competition between two trait types is the key to the evolution of diversity and life span. The necessity for more integrated theory to understand the evolution and evolutionary consequences of life span is clearly warranted.

ACKNOWLEDGMENTS

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