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[Article]

Demographic and Evolutionary Consequences of Selective Mortality: Predictions from an Eco-Genetic Model for Smallmouth Bass

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Abstract.—We use an individual-based eco-genetic model to examine the demographic and evolutionary consequences of selective mortality on a species with parental care, the smallmouth bass *Micropterus dolomieu*. Our analyses are grounded in a long-term (1936–2003) empirical study of the dynamics of two populations that differ widely in both density and life history. The model we construct extends previous approaches by including phenotypic plasticity in age and size at maturation, by permitting density-dependent somatic growth, and by analyzing how costs associated with parental care alter model predictions. We show that, first, additional mortality on age-0 individuals applied for 100 years causes reduced population abundance and biomass, faster somatic growth rates, and phenotypic plasticity toward slightly larger sizes at maturation. Second, mortality on individuals above a minimum size limit, also applied for 100 years, has a small influence on population abundance and somatic growth, causes a reduction of biomass, and substantial evolution of the probabilistic maturation reaction norm, leading to younger ages and smaller sizes at maturation. Third, the incorporation of body-size-dependent survival costs associated with parental care (i.e., by reducing the number of small breeding adults at high population densities, increasing the mortality of parents that breed at small body sizes, or increasing the mortality of offspring originating from small-sized parents) reduces the amount of evolution predicted to occur within 100 years. Together, these results underscore that selective harvest can cause both phenotypically plastic responses and rapid evolution; however, the rate and magnitude of the evolved changes are sensitive to a species' life history characteristics.

To understand the effects of selective mortality on a population, it is critical to distinguish between plastic and evolutionary responses in key life history traits such as age and size at maturation (Reznick 1993; Rijnsdorp 1993). Selective mortality can induce rapid evolutionary changes in both age and size at maturation (e.g., Reznick et al. 1990; Haugen and Vøllestad 2001) by favoring those individuals that reach maturity while minimizing their exposure to mortality (Law 2000). Selective mortality can also cause a phenotypically plastic response in age and size at maturation by altering environmental conditions for somatic growth through effects on population density and food availability (Law 2000; Hutchings 2004; Reznick and Ghalambor 2005; Dunlop et al. 2005b). Disentangling genetic and plastic responses is of particular significance to resource managers because genetic responses might take much longer to reverse than mere plastic changes (Law 2000). In the wild, however, it can be difficult to rule out the influence of confounding variables and to collect the data necessary for distinguishing between plastic and genetic responses. Models are therefore indispensable for predicting and understanding the consequences of selective mortality because they enable full control of any extraneous variables and salient assumptions.

In this study, we develop and analyze a model of mortality-induced evolution and parameterize it for a species with parental care, the smallmouth bass *Micropterus dolomieu*. We take a step beyond previous

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models of mortality-induced evolution (e.g., Law 1979; Abrams and Rowe 1996; Heino 1998; Martinez-Garmendia 1998; Ratner and Lande 2001) by permitting phenotypic plasticity in both the age and size at maturation by allowing somatic growth to be density-dependent and by including costs associated with parental care. This approach permits a more realistic framework in which to predict both the rate of evolutionary change and its endpoint. We include phenotypic plasticity in our study by modeling maturation reaction norms (MRNs). An MRN predicts the phenotypically plastic response of age and size at maturation to environmental variation in somatic growth rate (e.g., Stearns and Koella 1986; Roff 1992; Ernande et al. 2004). Plastic changes in response to environmental variation in somatic growth will cause the realized combination of age and size at maturation to be shifted along the MRN, whereas selection is expected to force an evolutionary shift of the entire MRN away from its original position (Heino et al. 2002). Using this approach, studies of maturation in several marine fish stocks have demonstrated consistent correlations between high levels of harvest mortality and shifts in MRN position (Grift et al. 2003; Barot et al. 2004b; Olsen et al. 2004, 2005).

Despite the growing number of studies examining fishing-induced evolution (e.g., Baskett et al. 2005; Olsen et al. 2005; Reznick and Ghalambor 2005), it is not fully known how a species' life history characteristics influence the rate of the evolutionary response to selective mortality. Parental care is one important aspect of life history that is present in numerous fish species (Mank et al. 2005), and yet its influence on the response to selective mortality has received little, if any, attention. Although parental care can improve offspring survival, such care can be costly by demanding a large expenditure of energy and by increasing a parent's exposure to predators (Clutton-Brock 1991). If sufficiently severe, these costs imply selective forces that might alter a population's predicted evolutionary response to mortality.

The focal species of our study, the smallmouth bass, shows paternal care in the form of an extended nest-guarding period that can last up to 6 weeks in the spring, during which the male parent fans the eggs to prevent fungal infection and defends the brood from potential predators (Ridgway 1988, 1989). The male feeds at a much diminished rate during the nest-guarding period (Ridgway and Shuter 1994), relying on stored energy reserves for sustenance (Mackereth et al. 1999). Evidence suggests that the presence of parental care in smallmouth bass is altering the selective forces acting on parental body size through three mechanisms. First, small males are disadvantaged because they emerge from winter with proportionately lower weight-specific energy reserves than larger males (Mackereth et al. 1999) and experience higher weight-specific maintenance costs (Shuter and Post 1990). Consequently, when population density is high and food availability low, small males possess insufficient energy reserves for maintaining adult home ranges and for nest-guarding (Ridgway et al. 2002). Therefore, small males might not initiate spawning, even when they are mature, which could explain why the fraction of mature males that form a nest decreases with increasing population density (Ridgway et al. 2002). Second, smaller males might suffer higher overwinter mortality because their reserves are further depleted after the energetically demanding nest-guarding period (Shuter et al. 1980; Mackereth et al. 1999). Third, smaller males might be less effective at protecting their brood from predators, and accordingly, their offspring might suffer higher mortality (Ridgway and Friesen 1992; Wiegmann and Baylis 1995; Knotek and Orth 1998). The selective pressures resulting from the above three mechanisms favor the evolution of larger body sizes at maturation and might thus directly oppose those resulting from size-selective mortality on larger individuals, which if acting alone, would favor maturation at smaller body sizes.

Two well-studied populations of smallmouth bass provide the empirical basis for assessing the usefulness of our modeling approach. The two study populations, one from Provoking Lake and the other from Opeongo Lake (both in Algonquin Provincial Park, Ontario, Canada, 45°42′N, 78°22′W), were most likely introduced from a common source in the early 1900s. The Opeongo Lake population has been studied continuously since 1936 and the Provoking Lake population has been studied periodically from the late 1940s. Since their original introduction, these two populations have diverged, Provoking Lake now supporting a population with a higher density, slower somatic growth rates, and smaller sizes at maturation relative to the Opeongo Lake population (Dunlop et al. 2005a). Empirical evidence suggests that the lower rates of predation on age-0 smallmouth bass in Provoking Lake, resulting from the depauperate fish community, might have contributed to the higher population density and slower somatic growth rates in that population (Dunlop et al. 2005a). Empirical evidence also indicates that, despite the existence of higher levels of mortality on the large size-classes in Provoking Lake (Dunlop et al. 2005a), the two populations exhibit similar maturation reaction norms (Dunlop et al. 2005b). We assessed how the costs of parental care might be affecting the rates of MRN evolution in these populations and whether it is reasonable to expect detectable changes in the MRN after only 100 years of life in the different selective environments of Provoking and Opeongo lakes.

To do this, we developed an individual-based model aimed at incorporating the salient ecological processes underlying mortality-induced evolution and introduce the term "eco-genetic" to characterize this model. That is, the model predicts the rates at which quantitative genetic traits evolve while at the same time integrates key aspects of the ecological setting (e.g., age and size structure, density-dependent growth, and phenotypic plasticity) into the selective environment that determines those rates. On this basis, we model the introduction of a smallmouth bass population into environments with differing levels of age-0 mortality or size-selective mortality and observe the implications of these differences for population dynamics, somatic growth, and MRN evolution. We then incorporate the three possible body-size-related effects of parental care discussed above and determine how these additions alter predicted changes in demography and predicted rates of MRN evolution. The detailed empirical data available for the Provoking Lake and Opeongo Lake populations provide a realistic context in which to evaluate these predictions and assess the possible role of parental care in determining how these populations have diverged since their introduction.

Methods

The individual-based eco-genetic model we constructed examines the introduction of a smallmouth bass population into an environment with selective mortality. In accordance with the historical timeframe of the smallmouth bass introductions into Opeongo and Provoking lakes, we ran this eco-genetic model for 100 years in discrete, 1-year increments.

Data sources.—Data from the Provoking Lake and Opeongo Lake smallmouth bass populations were used to parameterize the model. Empirical data have been collected on the Opeongo Lake population since its introduction in the early 1900s via an access point creel survey (Shuter et al. 1987). These creel data include temporal estimates of growth, population density, and mortality, corrected for changes in survey effectiveness (Shuter et al. 1987; Shuter and Ridgway 2002). Data also came from a multiyear spawning study conducted on Opeongo Lake from the 1980s to the present, as well as from detailed studies of growth and maturation conducted in 1981–1982 and 2000–2003 on Provoking and Opeongo lakes (Ridgway et al. 1991; Dunlop et al. 2005a, b). The empirical relationships used to parameterize the model are depicted in Figure 1 and detailed parameter values are provided in Appendix 1.

Probabilistic maturation reaction norms.—To account for the inherent stochasticity in the maturation process (Bernardo 1993), Heino et al. (2002) introduced the probabilistic maturation reaction norm (PMRN), defined as the probability that an individual will mature during the next season based on its current size and age. The midpoint of such a PMRN is defined separately for each age as the size at which the probability of maturing is 50% (Figure 1). The probabilistic envelope around the midpoint is given by the contour lines of equal maturation probability, ranging from just over 0 to just under 100% probability of maturation. The envelope's width is defined separately for each age as the size interval within which the probability of maturation rises some amount (e.g., 1–99%). This PMRN approach has been used in recent studies to isolate the influence of growth on maturation (Dunlop et al. 2005b) and to reveal shifts in PMRNs suggesting evolutionary responses to selective harvest (Grift et al. 2003; Barot et al. 2004b; Olsen et al. 2004, 2005). In our model, we considered a linear PMRN with an evolving midpoint slope and intercept and a constant width among ages. We modeled linear reaction norms (de Jong 1990) to reduce model complexity, and we considered the slope and intercept of the reaction norm as separate evolving traits because they experience different selective pressures (e.g., Brommer et al. 2005).

Genetic structure.—We used quantitative genetics principles (Falconer and Mackay 1996) to model the underlying genetic component of our populations and assumed that phenotypic plasticity in maturation is heritable by modeling genetically based reaction norms that are passed from parents to offspring (e.g., Brommer et al. 2005; Nussey et al. 2005). In the model the evolving traits that describe the reaction norms (PMRN intercept and slope) are initially assumed to be normally distributed with a given mean and variance. Both traits are passed on to offspring via incomplete inheritance.

Each individual in the population has a PMRN that is characterized by a midpoint slope (X), midpoint intercept (Y), and envelope width. The distribution of individual PMRNs in the population represents the genetic variance (van Noordwijk 1989; Windig 1994). The population-level PMRN is the mean of the individual PMRNs, and its envelope width represents the phenotypic variation in length at maturation for each age. At each age a, the population's phenotypic variance in length at maturation, $\sigma^2_{P,a}$, is composed of its genetic variance in length at maturation, $\sigma^2_{G,a}$, and its variance in length at maturation resulting from environmental factors, $\sigma^2_{E,a}$ (van Noordwijk 1989; Falconer and Mackay 1996). Heritability in length at

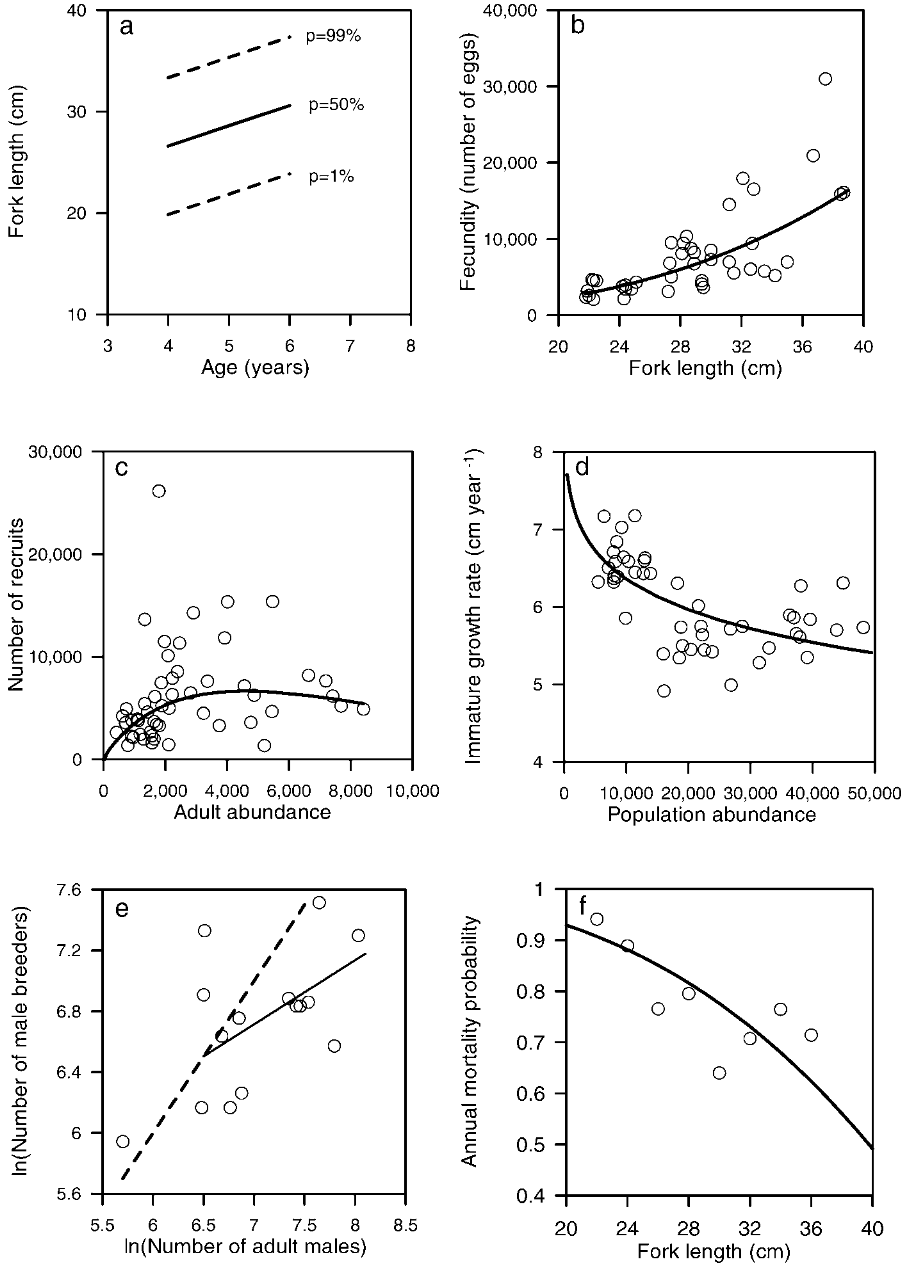

FIGURE 1.—Empirically derived functions for smallmouth bass populations in two Ontario lakes. Panels are as follows: **(a)** probabilistic maturation reaction norm estimated for the Opeongo Lake population in the 1930s and 1940s showing the 50% (midpoint), 1%, and 99% probability curves; **(b)** relationship between fecundity and body size for Provoking and Opeongo lakes (reproduced from Dunlop et al. 2005a); **(c)** stock–recruitment relationship for Opeongo Lake (reproduced from Shuter and Ridgway 2002); **(d)** relationship between immature somatic growth rate and population abundance in Opeongo Lake (reproduced from Shuter et al. 1987); **(e)** relationship between the number of breeding males and the number of adult males used in parental care mechanism 1 (solid line [reproduced from Ridgway et al. 2002]; the dashed line shows the 1:1 relationship); and **(f)** relationship between the annual probability of mortality and the fork length of male nest-guarders used in parental care mechanism 2 (reproduced from Dunlop et al. 2005b; see Appendix 3).

maturation at age a, h_a^2, is given by the ratio between $\sigma_{G,a}^2$ and $\sigma_{P,a}^2$ (Falconer and Mackay 1996). At each age a, the population's genetic variance in length at maturation is

$$\sigma_{G,a}^2 = a\sigma_X^2 + \sigma_Y^2, \quad (1)$$

where σ_X^2 and σ_Y^2 are the population's additive genetic variance in midpoint slope and intercept, respectively, at age a. Equation (1) follows directly from the assumed linearity of PMRNs.

In our model, we assume levels of genetic variance for the two traits X and Y and, together with an empirical estimate of phenotypic variance in length at maturation, use it to estimate heritability in the initial population (see below). The genetic variances are then free to change through time.

Initial population structure.—The parameters of the initial population were estimated from Opeongo Lake creel data collected on the earliest studied cohorts (1930s and 1940s). In our model, the introduced population consists of 200 yearlings (individuals of age 1) with a normal distribution of initial body sizes based on the mean and standard deviation of the Opeongo 1932 year-class. Back-calculations (Francis 1990) of body lengths at age 1, measured from scale samples collected from individuals of the 1932 cohort, were used to estimate the mean and standard deviation for yearling body size (via a validated back-calculation technique described in Dunlop and Shuter 2006).

The initial population-level PMRN midpoint slope, midpoint intercept, and envelope width were estimated empirically from Opeongo Lake creel data using the procedure introduced by Barot et al. (2004a). This procedure involves deriving the probability of maturing at age a and size s,

$$p_m(a,s) = \frac{o(a,s) - o(a-1, s-\Delta s[a])}{1 - o(a-1, s-\Delta s[a])}, \quad (2)$$

from the maturity ogive $o(a,\ s)$ describing the probability of being mature at a given age and size (calculated using logistic regression) and from the growth increment Δs from age $a-1$ to age a (Barot et al. 2004a). We pooled the 1930s–1940s cohorts to obtain a representative sample. Sufficient data allowed the estimation of the population-level PMRN for ages 4, 5, and 6; a linear regression of the midpoints of these three ages was then used to estimate the midpoint slope and intercept of the initial population's PMRN (Figure 1a). The 1% and 99% maturation probability percentiles of 4-year-olds (the age-class with the largest sample size) were used to determine the width of the initial population's PMRN (Figure 1a).

Given that the actual distribution of genetic variation between midpoint slope and intercept is unknown for the considered smallmouth bass populations, we parsimoniously assumed the same 10% level for the coefficients of genetic variation (CV = 100·standard deviation/mean) in both traits in the initial population. We chose 10% because it produces a realistic value of heritability (see below). All individuals in the initial population are then assigned a PMRN midpoint slope and intercept from a normal distribution with a mean equal to the population-level PMRN's midpoint (Figure 1a) and a standard deviation given by the assumed coefficient of genetic variation (10%) in midpoint slope and intercept. To examine robustness, we also determined the sensitivity of our model's results to 99 other combinations of the two coefficients of genetic variation (Appendix 2).

We calculated the heritability ($\sigma_{G,a}^2/\sigma_{P,a}^2$) for 4-year-olds in our initial population. We chose this age-group because this is the most common age at maturation, and sample sizes are accordingly large. The value of genetic variance ($\sigma_{G,a}^2$) was calculated from equation (1) and our assumed coefficients of genetic variation. The initial population-level PMRN width for age 4 represents the range of body sizes over which maturation occurs for this age; this PMRN width was therefore used as our estimate of phenotypic variance ($\sigma_{P,a}^2$) in size at maturation for age 4. Using this approach, we estimated the initial population's heritability in size at maturation for age 4 to be 0.26, which is in accordance with published estimates of heritabilities in life history traits (Mousseau and Roff 1987; Law 2000).

Maturation and reproduction.—In any given year, immature individuals in our model become mature according to the maturation probability given by their PMRN in conjunction with their age and size. In our model, mating is size assortative and occurs between pairs of mature individuals (assuming a 1:1 sex ratio) that are similar in body size. We parsimoniously assumed strict size-assortative mating because there is evidence that smallmouth bass are strongly size assortative in mate preference (Ridgway et al. 1991; Mackereth et al. 1999), and we did not want to increase model complexity by introducing an additional parameter describing strength of the preference (note, relaxing this assumption does not change the direction of predictions but does cause a slight increase in the magnitude of evolutionary change in response to size-selective mortality).

The number of offspring produced by a reproductive pair is estimated from the body size, L, of the largest parent in the pair following an empirically derived allometric relationship (Figure 1b), namely,

$$F_1 = (H_1 L)^{H_2}, \quad (3)$$

where H_1 and H_2 are constants. The number of new individuals recruiting to the population at age 1 is determined from a modified Ricker-type stock–recruitment function (Figure 1c), namely,

$$r = AS_a^d e^{-bS_a + cT}, \quad (4)$$

where S_a is the number of adults; T is the mean air temperature for June through September (kept constant at 15°C); and A, b, c, and d are empirically determined constants based on a detailed analysis of annual variation in recruitment, adult population size, and summer air temperatures for the Opeongo Lake population over the period 1937–1992 (Shuter and Ridgway 2002). The survival probability from eggs to age 1 is defined as the ratio between r and the total number of eggs produced by the mature population. Because stock–recruitment relationships can be highly variable, we tested our model's sensitivity to adding stochastic noise to the recruitment process (Appendix 2).

To model the inheritance of quantitative traits, we assumed that offspring trait values are equally determined by maternal and paternal trait values. In particular, the PMRN midpoint slope and midpoint intercept of each newborn are drawn from a normal distribution centered on the corresponding midparental values (mean of the two parent's trait values) and possessing a variance (σ_o^2) equal to half the population variance ($\sigma_{M,F}^2$) in the parental generation (Cavalli-Sforza and Feldman 1976; Baskett et al. 2005),

$$\sigma_o^2 = \sigma_{M,F}^2/2. \quad (5)$$

This ensures that the inheritance process maintains the population variance and follows from the parsimonious assumption that the maternal and paternal trait variances (σ_M^2 and σ_F^2) are equal.

Somatic growth.—We use the biphasic growth model proposed by Lester et al. (2004) to describe somatic growth. Accordingly, the growth of immature individuals is linear, and a mean growth rate $\bar{h}_i$ is calculated from the population abundance, D, using a simple variant of the competition equation described by Begon et al. (1996),

$$\bar{h}_i = \frac{h_{\max}}{1 + jD^q}, \quad (6)$$

where j and q are constants and $h_{\max}$ is the maximum growth rate (at $D = 0$). Equation (6) provides a good description of the temporal association between growth and abundance exhibited by the Opeongo Lake population (Figure 1d). In the model of Lester et al. (2004), mature individuals grow according to von Bertalanffy's growth function, reaching length

$$L_a = L_\infty[1 - e^{-k(a-a_0)}] \quad (7)$$

at age a; the parameters are calculated as follows:

$$L_\infty = \frac{3h_i}{g}, \quad (8)$$

$$k = \log_e\left(1 + \frac{g}{3}\Delta a\right), \quad (9)$$

and

$$a_0 = a_m + \left[\log_e\left(1 - \frac{g}{3}a_m\right)\right]/k, \quad (10)$$

where g is the reproductive investment rate, Δa is the increment between age-classes (1 year), and a_m is the age at which the "decision" to mature is made (this equals the age at maturation in our model). The reproductive investment rate g was estimated from the growth curve of mature Opeongo Lake smallmouth bass (following Lester et al. 2004) captured in 2000–2001 (Dunlop et al. 2005a); for simplicity, g is assumed to be identical and constant over time for all individuals. To allow variation in growth among individuals, growth rates h_i are randomly drawn, separately in each year, from a normal distribution with mean $\bar{h}_i$ and standard deviation h_s calculated from Opeongo Lake creel data (Shuter et al. 1987).

Mortality.—Age-specific annual mortality probabilities were measured from Opeongo Lake creel data (Shuter et al. 1987) and are applied annually to individuals of ages 1–3 ($m_{1-3} = 0.27$) and of ages 4 and older ($m_{4+} = 0.54$). On top of this background mortality, we apply different levels of selective harvest mortality from the time of introduction onwards. We apply selective annual mortality probabilities of 0.1–0.5 (in increments of 0.1) at the individual level on either age-0 individuals or individuals above a minimum size limit. We chose 18 cm as the minimum size limit because this is the hypothesized size at which mortality differences begin to emerge between the Provoking Lake and Opeongo Lake populations (Dunlop et al. 2005a). The size-selective mortality is applied to individuals regardless of maturation status; typically, the 18-cm size-class contains both juveniles and adults (Dunlop et al. 2005a). We also tested the sensitivity of our model results to decreasing m_{4+} to 0.27 and to increasing the minimum size limit to between 20 and 28 cm (Appendix 2).

Parental care.—We explore the consequences of parental care by considering three different ecological mechanisms. To delineate the evolutionary consequences of parental care, we investigate how the evolutionary response to a 0.3 probability of size-selective mortality, as applied to individuals above an

TABLE 1.—Detailed model results for smallmouth bass (mean ± SD of 100 independent model runs) after 100 years of exposure to a particular annual selective mortality probability (m) and one of the following parental care effects: no parental care mechanism (0), small parents do not breed if the number of adults is large (1), small parents experience survival cost (2), offspring of small parents experience survival cost (3; $\tilde{M}_{p,0} = 0.5$ [see text]). Population-level results are shown separately for selective mortality of age-0 fish and selective mortality of fish longer than 18 cm.

Parental care effects	m	Age at maturation (years)	Size at maturation (cm)	Immature growth rate (cm/year)	Asymptotic length (cm)	Total population abundance
			Age-0 fish			
0	0.1	3.3 ± 0.2	21.1 ± 1.2	6.2 ± 0.0	50.3 ± 0.2	9,714 ± 340
0	0.2	3.3 ± 0.2	21.4 ± 1.4	6.3 ± 0.0	51.0 ± 0.3	8,059 ± 481
0	0.3	3.3 ± 0.2	21.6 ± 1.4	6.4 ± 0.0	51.9 ± 0.3	6,350 ± 470
0	0.4	3.4 ± 0.2	22.4 ± 1.5	6.6 ± 0.0	53.2 ± 0.4	4,460 ± 450
0	0.5	3.3 ± 0.2	23.0 ± 1.4	6.8 ± 0.1	55.0 ± 0.6	2,722 ± 382
			Fish >18 cm			
0	0.1	2.9 ± 0.2	18.3 ± 1.7	6.2 ± 0.0	50.0 ± 0.2	10,480 ± 477
0	0.2	2.5 ± 0.3	16.0 ± 2.1	6.2 ± 0.1	50.2 ± 0.4	9,807 ± 785
0	0.3	2.2 ± 0.3	13.9 ± 2.4	6.2 ± 0.1	50.5 ± 0.7	9,271 ± 1,169
0	0.4	1.9 ± 0.3	11.8 ± 2.3	6.2 ± 0.1	50.6 ± 1.0	8,954 ± 1,451
0	0.5	1.7 ± 0.3	10.7 ± 2.7	6.3 ± 0.2	50.9 ± 1.6	8,475 ± 1,704
1	0.3	3.1 ± 0.0	19.6 ± 0.3	6.3 ± 0.0	51.2 ± 0.4	7,706 ± 628
2	0.3	2.9 ± 0.3	20.1 ± 2.3	6.8 ± 0.2	54.9 ± 1.3	3,363 ± 1,098
3	0.3	2.4 ± 0.3	15.4 ± 2.6	6.3 ± 0.1	50.9 ± 1.0	8,472 ± 1,681
1 + 2 + 3	0.3	3.3 ± 0.2	23.7 ± 1.7	7.1 ± 0.1	57.6 ± 0.8	1,615 ± 374

18 cm size limit, varies in the presence and absence of each of the three mechanisms. We also assess the evolutionary effect of all three mechanisms combined.

For the first mechanism, we assume that small males possess insufficient energy reserves for nest-guarding when population density is high. Accordingly, we introduce a dependence of the number of breeding mature individuals, R, on the abundance of mature individuals in the population, D_m:

$$R = C_1 D_m^{C_2}, \qquad (11)$$

where C_1 and C_2 are empirically based constants (Figure 1e). The largest R mature individuals in the population are then chosen for breeding, implying that for $R < D_m$ the smallest mature individuals in the population do not breed.

For the second mechanism, we assume elevated mortality levels for parents that breed at small body sizes. We used mark–recapture data on nesting males in Opeongo Lake to derive the relationship between the size of a parent and its mortality (Figure 1f; Appendix 3). We incorporate this second mechanism by applying, following reproduction, the mortality probability ($M_{p,L}$) to the largest parent in each reproducing pair.

For the third mechanism, we assume reduced survival of offspring of small-sized parents. Due to a lack of empirical data for calibrating this effect, we assumed a linearly decreasing relationship between the mortality probability of the offspring produced by a given pair of parents in a given year and the size L of the pair's larger parent. Specifically, we assume this mortality probability to decrease from a maximum of $\tilde{M}_{p,0} = 0.5$ at $L = 0$ cm to a minimum of 0 at $L \geq L_0$, such that

$$\tilde{M}_{p,s} = \tilde{M}_{p,0}(1 - L/L_0)_+. \qquad (12)$$

We chose $L_0 = 40$ cm because parents above this size appear to be particularly aggressive and effective nest-guarders (E. Dunlop, unpublished data). We also varied $\tilde{M}_{p,0}$ to observe the effect on model results (Appendix 2).

Results

Age-0 smallmouth bass mortality causes slower population expansion, lower population biomass and abundance, faster somatic growth rates, and maturation at slightly larger sizes (Table 1; Figure 2); however, there is little to no effect on the PMRN's slope and intercept (Figure 2e). Selective mortality of individuals above 18 cm in the absence of parental care has a small influence on population abundance and somatic growth rate, but causes a substantial decrease in population biomass and in age and size at maturation (Table 1; Figure 2); these changes are accompanied by large decreases in the PMRN's slope and intercept (Figure 2f).

Including effects of parental care generally reduces the rate of evolution. The 100-year response in the PMRN to a 0.3 probability of size-selective mortality is not appreciably different when we make the number of small-sized parents that actually breed dependent on the abundance of adults (mechanism 1); however, it is significantly reduced when we include a survival cost for small parents (mechanism 2), include a survival

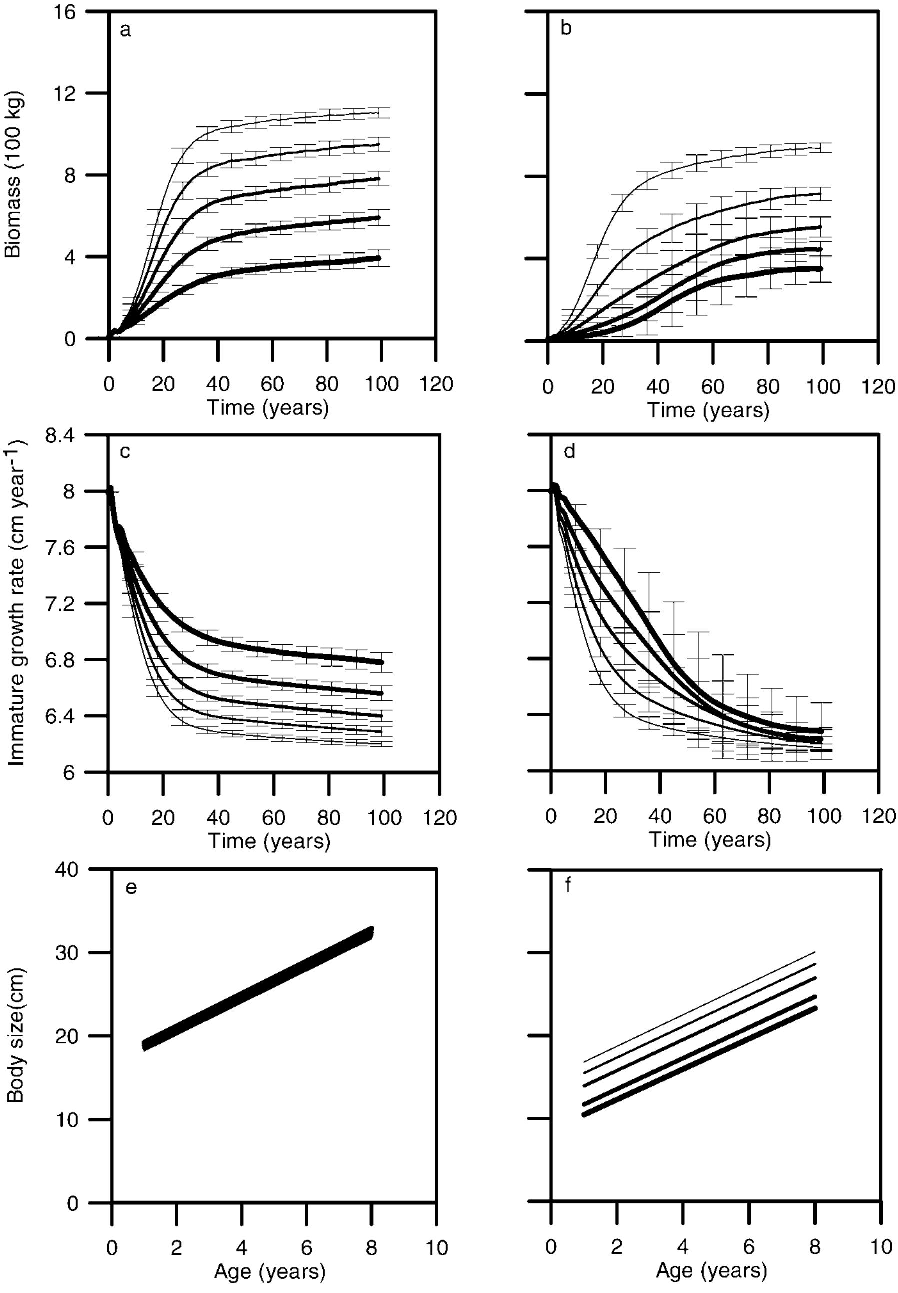

FIGURE 2.—Model results in the absence of parental care for different probabilities of selective mortality (0.1–0.5; line thickness increases with increasing mortality probability) on individual age-0 smallmouth bass or individuals longer than 18 cm. All results are the averages for 100 independent model runs. Panels are as follows: **(a)** population biomass ± SD (every 10th year) for mortality on age-0 individuals; **(b)** population biomass ± SD for mortality on individuals longer than 18 cm; **(c)** immature somatic growth rate ± SD for mortality on age-0 individuals; **(d)** immature somatic growth rate ± SD for mortality on individuals longer than 18 cm; **(e)** final position (year 100) of population-level probabilistic maturation reaction norm (PMRN) midpoint for mortality on age-0 individuals; and **(f)** final population-level PMRN midpoint for mortality on individuals longer than 18 cm. Biomass was estimated by converting body length to body mass via an empirically derived length–weight relationship (data from Dunlop et al. 2005a).

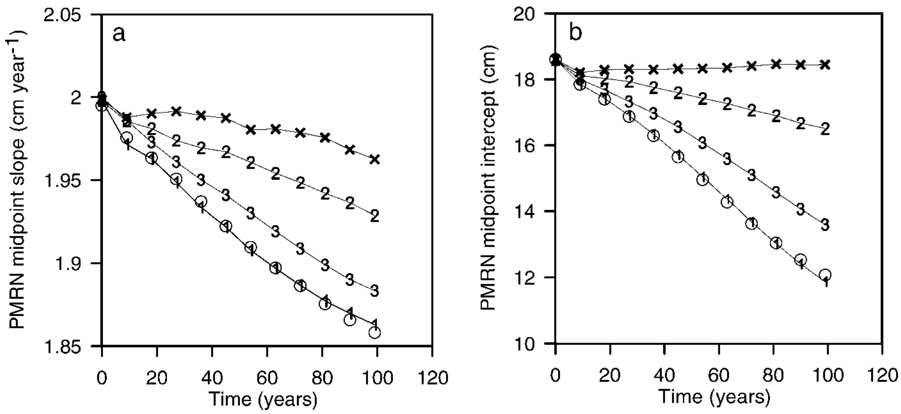

FIGURE 3.—Influence of parental care on model results with an annual selective mortality probability of 0.3 on individual smallmouth bass longer than 18 cm. All results are the averages for 100 independent model runs. Panels **(a)** and (b) show the changes in the slope and intercept of the probabilistic maturation reaction norm (PMRN) in the absence of parental care (circles), when small parents do not breed because the number of adults is large (1), when small parents experience a survival cost (2), when the offspring of small parents experience a survival cost (3, with $\tilde{M}_{p,0} = 0.5$), and when all three effects of parental care are combined (*x*s).

cost for the offspring of small-sized parents (mechanism 3), or combine all three mechanisms (Table 1; Figure 3). In addition, final population sizes are significantly reduced when these parental care effects are included (Table 1).

Discussion

Our model predicts that, at least in the absence of parental care, introduction of a population into a system with high size-selective mortality will cause a dramatic shift of the PMRN, corresponding to evolution toward smaller sizes and younger ages at maturation. This is because individuals able to reach maturation before succumbing to mortality are more likely to reproduce and pass on their traits to the next generation. These predictions are supported by observations of PMRN shifts by Barot et al. (2004b) and Olsen et al. (2004, 2005) in several stocks of Atlantic cod *Gadus morhua* and by Grift et al. (2003) in plaice *Pleuronectes platessa* that were subjected to the type of selective mortality we considered in our model. In Atlantic cod, the midpoint of the PMRN dropped by 10 cm in only 7 years (Olsen et al. 2004), providing empirical evidence that the magnitude of responses predicted from our model are also possible in nature. However, these predictions contrast sharply with the observed stability of the PMRN exhibited by both the Provoking Lake and Opeongo Lake smallmouth bass populations after 100 years of living under different mortality rates (Dunlop et al. 2005a, 2005b).

Not surprisingly, age-0 smallmouth bass mortality causes no evolutionary response in the PMRN. In our model, age-0 smallmouth bass mortality is applied to individuals during their first year of life and is not applied throughout the juvenile period. We took this approach because many predators of smallmouth bass are relatively small in body size and tend to feed on the smallmouth bass eggs and larvae (Knotek and Orth 1998; Dorn and Mittelbach 2004; Steinhart et al. 2004). Our approach contrasts with models in which the life history strategy affects survival throughout the juvenile period (e.g., Abrams and Rowe 1996) or in which the considered juvenile mortality extends right up to maturation (e.g., Ernande et al. 2004). In such models, juvenile mortality is predicted to cause evolution in the age or size at maturation. However, because mortality during age 0 occurs far in advance of maturation and individuals undergo such mortality regardless of their maturation traits, it is clear that the age-0 smallmouth bass mortality in our model exerts no selective pressure on the PMRN.

The detailed studies that have been conducted on the Provoking Lake and Opeongo Lake smallmouth bass populations permit comparisons between model predictions and empirical observations. Our model predicts that introduction to a system with low mortality on age-0 individuals produces higher population density, slower somatic growth rates, and smaller sizes at maturation. These model predictions match the empirical observations of the Provoking Lake population, where evidence suggests that there is less predation of age-0 individuals than in the Opeongo Lake population (Dunlop et al. 2005a). Empirical evidence also suggests that mortality on typical adult size-classes is higher in Provoking Lake than in Opeongo Lake (Orendorff 1983; Dunlop et al. 2005a). For example, mortality probabilities following nest-guarding, as estimated from return rates of tagged individuals, are related to body size (Figure 1f) and average 89% in Provoking Lake and 63% in Opeongo Lake (Dunlop et al. 2005a). These differences in mortality are not due to recreational harvest because that harvest is low in Opeongo Lake (Shuter et al. 1987) and very low in Provoking Lake (<3% per year; Orendorff 1983); instead, these differences are probably related to the low availability of large prey in Provoking Lake (Dunlop et al. 2005a). Interestingly however, these differences in size-selective mortality between the Provoking and Opeongo populations have not led to detectable evolutionary divergence in their PMRNs (Dunlop et al. 2005b).

The predictions of our model—namely, that parental care reduces the rate of evolutionary response to mortality—are supported by the above observations that the Opeongo and Provoking populations show no evolutionary divergence in their PMRNs, despite between-population differences in size-selective mortality. Inclusion of parental care mechanisms in our model alters the selective environment and reduces the evolutionary response in the PMRN. In the wild, larger parents not only possess higher energy reserves to better survive the energetic costs of nest-guarding (Mackereth et al. 1999) but are also better able to defend their brood from predators (Wiegmann and Baylis 1995; Knotek and Orth 1998). Large body size also has its benefits in species without parental care, whereby more experienced spawners produce eggs and larvae with higher survival probabilities (e.g., Trippel 1998). Our model illustrates that the selective pressures favoring large body size in parents might counteract the selective pressures towards maturation at smaller body size imposed by mortality on larger individuals, and this could thereby alter the total selective forces acting on the PMRN. However, this is not to say that very intensive harvest will not cause substantial evolution of the PMRN in a species with parental care; rather, because of the opposing selective forces, the evolutionary response to intense harvest would probably be lessened in a species with parental care compared with a species without parental care. This increase in evolutionary inertia, imposed on the PMRN by parental care effects, has demographic consequences: Final population sizes in the presence of parental care are reduced. This reduction occurs because parental care effects constrain the compensating increases in population growth rate that arise from evolutionary adaptation in the PMRN. This finding suggests that the presence of parental care in the face of the added mortality imposed by intense harvest can impose additional limitations on the ability of a population to persist by adaptation.

The observed patterns in the age and size at maturation of smallmouth bass support our hypothesis that the survival costs associated with parental care are significant factors in this system. The lower range of ages and sizes at maturation that our model predicts for a species without parental care experiencing the high-mortality regimes are not commonly observed in the smallmouth bass of Provoking or Opeongo lakes (Dunlop et al. 2005a) or in wild populations of smallmouth bass in general (Dunlop 2005). However, once we include parental care in the model, maturation occurs across a range of more typical ages and sizes, suggesting that accounting for this life history characteristic improves the realism of our model.

Our evolutionary model includes several simplifications. First, the model describes evolution in quantitative traits rather than genes. Second, we parsimoniously assume coefficients of genetic variation of 10% in our initial population. As expected (Falconer and Mackay 1996), the speed of evolution is positively related to the assumed values (Appendix 2). Encouragingly, however, our assumed coefficient results in an estimated heritability of length at maturation for 4-year-olds (about 0.26) that is the same as the mean value of 0.26 reported for life history traits by Mousseau and Roff (1987). Third, we assume no genetic correlation between the two evolving quantitative traits, and we do not consider potential joint evolution in many other interesting life history traits, such as reproductive investment or somatic growth rate. We did not allow these other traits to evolve in the model because the inclusion of PMRNs already is a substantial advancement over previous models, and we wanted to keep model analyses and predictions reasonably simple. However, there is evidence that reproductive investment increases with higher mortality (Reznick et al. 1990; Lester et al. 2004) and that rapid evolution of somatic growth rate can occur in response to selective mortality on large individuals (Conover and Munch 2002); it is thus conceivable that size-selective mortality could induce evolutionary shifts in these other life history traits in the smallmouth bass.

A promising extension of our model would be to include sex differences. In our model, males and females are assumed to have a 1:1 ratio. We made this simplification because there is no sexual dimorphism in the somatic growth rates of smallmouth bass (Dunlop et al. 2005a). This is likely because females invest a large amount of energy into gonads, whereas males invest energy into parental care, leading to a similar reduction in somatic growth in both sexes. If we had modeled males and females separately and allowed only males to exhibit parental care, the impact on the parental care results would have probably been minimal because of size-assortative mating (Ridgway et al. 1991; Mackereth et al. 1999). In the wild, small females might not be able to breed if there are no small males for them to breed with (Dunlop et al. 2005a); if the number of small males that breed is dependent on population density (our first parental care mechanism), the same relationship will hold true for small females. Similarly, because of size-assortative mating the offspring of large females will also have a higher probability of survival (our third parental care mechanism). When considering a mortality cost for small parents (our second parental care mechanism), we only apply this cost to one of the parents to mimic a situation where only one parent is providing care. Therefore, modeling separate sexes was not necessary for the purposes of this study but, in future, could provide further insights depending on the focal species and research question at hand.

The eco-genetic modeling framework presented here provides a powerful tool for studying the mortality-induced evolution of PMRNs. The majority of related models to date have not adequately considered phenotypic plasticity in the maturation process because they directly modeled age and size at maturation as quantitative traits, without accounting for their dependence on the environment (e.g., Law 1979; Abrams and Rowe 1996; Heino 1998; Martinez-Garmendia 1998; Ratner and Lande 2001). The model by Ernande et al. (2004) did account for phenotypic plasticity by following the evolution of MRNs but employed deterministic maturation dynamics and focused on the prediction of evolutionary endpoints rather than of the time course of evolutionary change. In Ernande et al. (2004), size-selective harvest of individuals larger than a minimum size limit caused evolutionary shifts of the MRN towards younger ages and smaller sizes. While these findings are qualitatively similar to ours, using an eco-genetic modeling approach readily allowed for several novel developments. First, we could model maturation as being probabilistic, which is probably more realistic given the considerable stochasticity involved in the maturation process (Heino et al. 2002). This added realism actually alters the model's predictions by slowing down the evolutionary response more than models in which MRNs are unrealistically treated as deterministic (Appendix 2). Second, our choice of model allowed examination of the speed of evolution, something that is not possible based on traditional optimization models (e.g., Law 1979) or adaptive dynamics models (e.g., Heino 1998; Ernande et al. 2004), both of which focus on evolutionary endpoints instead. Third, our model permitted somatic growth rates to vary with population density, a realistic feature missing from most of the related previous evolutionary models (e.g., Martinez-Garmendia 1998; Ratner and Lande 2001; Ernande et al. 2004; Tenhumberg et al. 2004; Baskett et al. 2005). Modeling somatic growth as density-dependent is important in the context of mortality-induced evolution. Although mortality can influence growth through its impact on population abundance, growth controls an individual's body size, which in turn influences its fitness, especially in an environment where mortality is size-selective. Accounting for density-dependent growth is also important when examining the dynamics of recently introduced populations because the density and growth of such populations undergo considerable temporal shifts in the wake of the introduction. Our analysis of the effect of growth on the evolutionary process clearly illustrates the danger in assuming a constant growth rate that does not change with population abundance: the rates of evolution can be severely over- or underestimated when density-dependence is not included (Appendix 2). Also, we would not have been able to predict the plastic effect of age-0 mortality (which alters abundance) on growth and maturation patterns without the presence of density-dependent growth.

Finally, and perhaps most importantly, our results are novel because we show that inherent selective forces, such as those produced by parental care, can greatly alter the evolutionary response typically observed under size-selective harvest. This underscores the importance of adequately reflecting the life history characteristics of the considered species when assessing its vulnerability to the selective pressures imposed by exploitation.

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Appendix follows

Appendix 1: Model Parameters and Their Values

TABLE A.1.1.—Parameters and values used in an eco-genetic model for smallmouth bass examining the evolutionary consequences of selective mortality. Data sources are (1) Opeongo Lake creel data reproduced from Shuter et al. (1987), (2) Opeongo Lake creel and spawning data reproduced from Shutter and Ridgway (2002), and (3) Opeongo and Provoking Lake data reproduced from Dunlop et al. (2005a, b).

Variable	Description	Equation	Data source	Value
$\overline{L}$	Initial mean body size (cm)		1	7.71
L_s	Initial standard deviation of body size (cm)		1	1.43
$\overline{X}$	Initial mean reaction norm slope (cm/year)		1	2.0
$\overline{Y}$	Initial mean reaction norm intercept (cm)		1	18.6
W	Width of reaction norm (cm)		1	13.5
$m_{1–3}$	Mortality probability for ages 1 to 3		1	0.27
m_{4+}	Mortality probability for ages 4 and older		1	0.54
H_1	Constant in fecundity function (per cm)	3	3	0.64
H_2	Constant in fecundity function	3	3	3.02
d	Constant in stock–recruitment function	4	2	0.89
A	Constant in stock–recruitment function	4	2	1.4×10^{-4}
b	Constant in stock–recruitment function	4	2	1.9×10^{-4}
c	Constant in stock–recruitment function (per °C)	4	2	0.72
h_{max}	Maximum immature growth rate (cm/year)	6	1	9.12
q	Constant in growth function	6	1	0.29
j	Constant in growth function	6	1	0.031
h_s	Standard deviation of growth rate (cm/year)		1	0.91
g	Reproductive investment rate (per year)	8	3	0.37
C_1	Constant in first parental care function	9	2	42.9
C_2	Constant in first parental care function	9	2	0.42
I_s	Constant in second parental care function (per year cm)	A3.1	3	–2.85
I_i	Constant in second parental care function (per year)	A3.1	3	11.19
$\tilde{M}_{p,0}$	Constant in third parental care function	12		0.5
L_0	Constant in third parental care function (cm)	12		40

Appendix 2: Sensitivity Analyses

We performed eight sensitivity analyses on various model parameters and assumptions. We carried these out with size-selective mortality applied to individuals longer than 18 cm and examined the effect of the changes in parameters or assumptions on the final position (i.e., in year 100) of the probabilistic maturation reaction norm (PMRN) midpoint. The measure of sensitivity used is therefore the amount of evolution observed in the PMRN over a 100-year period.

(1) *Maximum mortality probability in the third parental care mechanism*.—We varied the $\tilde{M}_{p,0}$ in equation (12) between 0.1 and 0.5 to explore the consequences of the third parental care mechanism. We found that as we increased the mortality rate ($\tilde{M}_{p,0}$) applied to the offspring of small parents, the evolutionary response to size-selective mortality decreased (Table A.2.1).

(2) *Minimum size limit in the size-selective mortality regime*.—We tested the effect of raising the minimum size limit in our size-selective harvesting regime to 20, 22, 24, 26, and 28 cm. The results show that raising the minimum size limit to 20 or 22 cm causes a slight increase in the amount of PMRN evolution observed, whereas raising it to 24 cm or more lessens the amount of evolution (Table A.2.1).

(3) *Combination of age-0 mortality and size-selective mortality*.—We determined the combined effect of an age-0 mortality of 0.3 and a size-selective mortality on individuals greater than 18 cm of 0.3. This causes only a minor decrease in the amount of PMRN evolution relative to a scenario with only 0.3 mortality on individuals greater than 18 cm (Table A.2.1).

(4) *Stochasticity in the stock–recruitment relationship*.—We added normally distributed noise into the stock–recruitment relationship by drawing recruitment numbers randomly from a normal distribution centered on the deterministic number of recruits (from equation 4) with a standard deviation equal to either 10% or 20% of the

TABLE A.2.1.—Sensitivity analyses of the effects of different model parameters and assumptions on the final position of the probabilistic maturation reaction norm (PMRN) midpoint slope and intercept after 100 years of annual selective mortality probability applied to smallmouth bass.

Modification made in sensitivity analysis	Size targets of selective mortality (cm)	Selective mortality probability	PMRN	
			Slope (cm/year)	Intercept (cm)
Baseline scenario[a]	>18	0.3	1.86	12.08
$\tilde{M}_{p,0} = 0.1$	>18	0.3	1.87	12.30
$\tilde{M}_{p,0} = 0.2$	>18	0.3	1.87	12.32
$\tilde{M}_{p,0} = 0.3$	>18	0.3	1.87	12.86
$\tilde{M}_{p,0} = 0.4$	>18	0.3	1.88	13.30
$\tilde{M}_{p,0} = 0.5$	>18	0.3	1.88	13.60
Minimum size limit	>20	0.3	1.85	12.13
Minimum size limit	>22	0.3	1.84	11.86
Minimum size limit	>24	0.3	1.82	12.13
Minimum size limit	>26	0.3	1.84	12.72
Minimum size limit	>28	0.3	1.84	13.37
Size-selective (and age-0)	>18 (and age 0)	0.3 (and 0.3)	1.88	13.05
10% stochasticity in SR[b]	>18	0.3	1.84	12.00
20% stochasticity in SR[b]	>18	0.3	1.86	11.86
Deterministic MRN[c]	>18	0.3	1.85	10.05
Constant mean growth[d] = 6 cm/year	>18	0.3	1.84	11.58
Constant mean growth[d] = 7 cm/year	>18	0.3	1.87	14.17
Constant mean growth[d] = 8 cm/year	>18	0.3	1.94	16.73
Constant mean growth[d] = 9.12[e] cm/year	>18	0.3	1.98	18.10
Constant mean growth[d] = 10 cm/year	>18	0.3	1.99	18.69

[a] The baseline scenario is included for comparison; it consists of the standard results (i.e., without any of the modifications considered in the sensitivity analyses) for 100 years of selective mortality probability of 0.3 on individuals exceeding 18 cm in length.
[b] SR = stock–recruitment relationship (equation 4).
[c] The slope and intercept represent a deterministic maturation reaction norm (MRN) with vanishing envelope width.
[d] Density-dependent growth is disregarded in these analyses.
[e] Note that $\bar{h}_i = h_{max} = 9.12$ cm follows from equation (6) for population abundance $(D) = 0$.

number of recruits. This had little effect on the evolutionary response, there being only a slight tendency for the amount of PMRN evolution to increase (Table A.2.1).

(5) *Deterministic maturation reaction norm*.—To test the implications of removing stochasticity from the maturation process, we modeled the response of a deterministic maturation reaction norm to size-selective mortality. Not surprisingly, making the maturation reaction norm deterministic resulted in a larger evolutionary response (Table A.2.1).

(6) *Density-dependent growth*.—In our test of the sensitivity of model results to density dependence, we disregarded the relationship between the growth rate and population abundance. Immature growth then differed between individuals and years only stochastically around the mean immature growth rate of the initial population ($\bar{h}_i$). The results show that in the absence of density dependence the amount of PMRN evolution decreases when the mean immature growth rate is increased (Table A.2.1). When the mean immature growth rate is held constant at a slow rate (i.e., at 6 cm/year), the amount of PMRN evolution observed is larger than in the case of density dependence; the opposite pattern is observed when the mean immature growth rate is held constant at higher rates (≥7 cm/year; Table A.2.1).

(7) *Background mortality*.—We performed a sensitivity analysis on the background level of age-specific mortality applied to older individuals (ages 4 and older) to ascertain whether the amount of evolution observed in response to size-selective mortality would be larger than the effect of the elevated age-specific mortality of older individuals. To do this, we altered the background age-specific annual mortality of older individuals from 0.54 (the original level) to 0.27 to make it equal to that of younger individuals while at the same time applying a size-selective annual mortality of 0.5 to individuals exceeding 18 cm. The results indicate that decreasing the background mortality of older individuals reduces the amount of evolution in response to size-selective harvest (i.e., the PMRN midpoint for a 5-year-old decreases by 10.82 cm with the original background mortality of 0.54 and by 8.36 cm when the background mortality is reduced to 0.27). Therefore, the evolutionary response with the reduced

background mortality is still substantial and is significantly larger than the difference in response between the two background mortalities.

(8) *Coefficients of genetic variation in the initial population.*—We performed a sensitivity analysis on the initial population's coefficients of genetic variation in the PMRN midpoint slope and intercept. We tested 100 combinations, allowing both coefficients of variation to range between 1% and 10% in steps of 1% and observed the effect on model results. We performed this sensitivity analysis using an annual size-selective mortality of 0.5 applied to individuals exceeding 18 cm. As expected, we found that the amount of evolution in the PMRN slope increases with the initial variance of this slope and that the amount of evolution in the PMRN intercept increases with the initial variance of this intercept (Figure A.2.1).

Appendix 3: Estimation of Size-Dependent Parental Mortality

From 1997 to 2003, all of the nesting males in Jones Bay (perimeter = 5 km) of Opeongo Lake were captured with fishing rods, had their body sizes measured, had 3–6 scales removed for aging purposes, were tagged with internal passive integrated transponder tags (Biomark, Boise, Idaho) and external T-bar tags (Halprint Ltd., Victor Harbor, Australia), and were released back into their nests within 5 min (Ridgway et al. 1991). Smallmouth bass show extremely high nest site fidelity (i.e., over multiple years males tend to nest within close proximity of their original nest site). In the Opeongo Lake population, 94% of previously nesting males returned to within 200 m of their original nest site and 35% returned to within 20 m (the modal distance category; Ridgway et al. 2002). Therefore, by sampling all nesting males in Jones Bay in multiple years, it was possible to identify which male parents did not return to nest in a subsequent year. Under the reasonable assumptions that the number of males straying outside of the sampling area was negligible and that those that did not return had suffered mortality, the size-dependent annual mortality probability was estimated by dividing, separately for each size-class, the number of males that did not return to nest in a subsequent year by the total number of males that were tagged (following Dunlop et al. 2005b). These annual mortality probabilities of reproducing individuals ($M_{r,L}$) were converted to instantaneous mortality rates ($I_{r,L}$) and regressed on body size L ($I_{r,L} = I_s L + I_i$, where I_s and I_i are regression coefficients; Figure 1f shows this relationship in terms of annual mortality probabilities). We then assumed that the background annual mortality probability of 0.54 experienced by older fish was the annual mortality probability (M_b) of nonreproducing individuals. The increase in annual mortality probability induced by reproduction for a parent of body size L ($M_{p,L}$) is thus given by the equation

$$M_{p,L} = M_b - M_{r,L}.$$

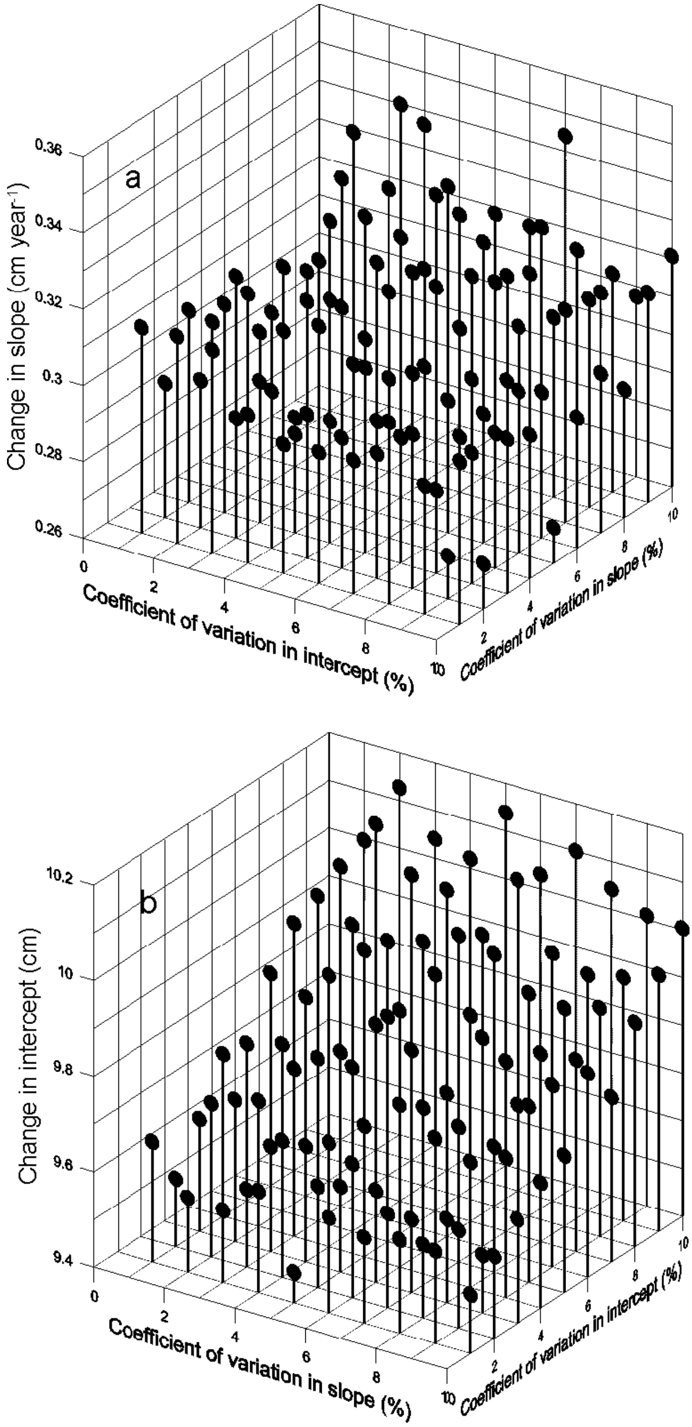

FIGURE A.2.1.—Influence of different initial combinations of coefficients of genetic variation on the evolutionary response of smallmouth bass to 100 years of size-selective mortality: **(a)** evolution of the probabilistic maturation reaction norm (PMRN) midpoint slope, and **(b)** the PMRN midpoint intercept, where the midpoints are measured in terms of the absolute difference between the initial (year 1) and final (year 100) slope or intercept. The results were averaged over 50 independent model runs. The annual size-selective mortality probability was 0.5 for individuals longer than 18 cm.