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Author(s): T. G. Benton and A. Grant

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HOW TO KEEP FIT IN THE REAL WORLD: ELASTICITY ANALYSES AND SELECTION PRESSURES ON LIFE HISTORIES IN A VARIABLE ENVIRONMENT

T. G. BENTON* AND A. GRANT

School of Environmental Sciences, University of East Anglia, Norwich NR4 7TJ, United Kingdom

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Abstract.—Most life-history theory assumes the environment is invariant. For the first time, analytical and numerical techniques were employed to investigate the impact of environmental variability on selection pressures (elasticities = proportional sensitivities) on a range of life histories. We find that the impact of variability is influenced significantly by the amount of variability an organism experiences (more variability affects selection pressures more), the correlations between variations among the vital rates (negative correlations are more likely to relax selection on fecundities and increase it on survival rates), and the life history in question (shorter life histories are more affected). In addition, the impact of a variable environment on the elasticities of life histories is sensitive to the sampling distribution used to generate the variability, and it is particularly sensitive to extreme values, such as those caused by occasional catastrophic events. The elasticities of life histories in highly variable environments may bear little relationship to those in a constant environment. In detailed optimality or evolutionarily stable strategy (ESS) modeling, variability in vital rates as small as a standard deviation being 10%–15% of the mean may appreciably alter the conclusions. Thus, it may be very important to consider the possible impact of environmental stochasticity and not to assume that it has no effect.

The great majority of life-history theory assumes density and frequency independence and environmental constancy. This allows the use of the classical, deterministic evolutionary theory (developed by, among others, Norton [1928] and Fisher [1930]), in which r from the Euler-Lotka equation is used as a measure of fitness (Charlesworth 1980; Lande 1982; Sibly and Calow 1983). In these studies, r refers to the "per-copy" rate of increase of an allele in a homozygous population (Sibly and Curnow 1993), whereas in ecology r typically refers to the per capita rate of increase. The former refers to the number of allelic copies per allele, the latter to the number of offspring per parent. The justification for the use of r as a measure of fitness is that, with the assumptions made above, the invasion of a rare allele (A) into a population of aa individuals is possible if $r_A > r_{aa}$. Invasion of a rare allele into a population regulated by strong density dependence can also be predicted by using r, in which case, for $r_A > r_{aa}$ to occur, A must increase the genotypic carrying capacity of the environment (see Sibly and Calow 1983 for a proof).

* To whom correspondence should be addressed; present address: 3, Kesters Close, Hardwick, Cambridge CB3 7QY, United Kingdom; E-mail: tbenton@cup.cam.ac.uk.

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Stearns (1992, p. 33) comments, "The most widely used fitness measure is r. Predictions made with r have functioned well in both theory and experiment, but it may not work when environmental stochasticity, frequency dependence or density dependence are important. There is room for more work on how changing fitness definitions changes predictions about life-history evolution." In this article we examine the first situation mentioned by Stearns: life-history evolution in stochastic environments.

Evolution and demography in random environments have been extensively studied by Shripad Tuljapurkar and colleagues since 1980 (reviewed in Tuljapurkar 1990*a*). It is now well known that the fitness of a life history may be considerably different in a random environment compared with a constant environment. For example, variable environments (especially where juvenile mortality is more variable than adult mortality) may cause selection for iteroparity in organisms, when otherwise semelparity would be selected: so-called bet hedging (Murphy 1968; Schaffer 1974; Fox 1993). The effect of a randomly varying environment depends on several factors such as the size of the variation, the covariation between traits, and the life history under investigation. As a result, semelparity may also sometimes be favored by environmental variability (Orzack and Tuljapurkar 1989; Tuljapurkar 1990*a*). Variability may cause selection for delayed reproduction such as occurs in insect diapause, seed dormancy, cohort splitting, or delayed flowering in biennials (Tuljapurkar 1990*a*, 1990*b*). Variability may cause selection for phenotypic plasticity in some situations but not others (Orzack 1985). Variability may induce subtle changes in the proportions of animals adopting different strategies within a population (T. G. Benton and A. Grant, unpublished data). In this article we are interested in investigating the way that variability changes the selection pressures on an organism's life history. Caswell (1989) shows how useful elasticity and sensitivity analyses are for investigating patterns of selection on organisms in deterministic environments. Here, for the first time, we use these analyses on a wide variety of life histories in stochastic environments.

The age-structured life history of an organism can be described by a projection matrix (**M**) of the form

$$\mathbf{M} = \begin{pmatrix} F_1 & F_2 & F_3 & \cdots & F_n \\ S_1 & 0 & 0 & \cdots & 0 \\ 0 & S_2 & 0 & \cdots & 0 \\ \cdots & \cdots & \cdots & \cdots & \cdots \\ 0 & 0 & \cdots & S_{n-1} & 0 \end{pmatrix},$$

where F_x is the number of surviving offspring born to organisms in age class x at time t that remain alive at time $t + 1$, and the S_x are the probabilities of transition (survival rates) from age class x to $x + 1$ during each time step. The zero terms reflect the impossibility of organisms making transitions from age class x to anything other than age class $x + 1$. In size-structured models, these terms may be nonzero. In a constant environment, with no frequency or density dependence,

a population's fecundity and survival rate schedules (and thus the projection matrix) will remain invariant through time. One can also define a vector (**N**) describing the initial (time t equals zero) age structure of a population:

$$\mathbf{N}_0 = \begin{pmatrix} n_1 \\ n_2 \\ n_3 \\ \vdots \\ n_n \end{pmatrix}.$$

Projection of the population can be made using the equation

$$\mathbf{N}_{t+1} = \mathbf{M}\mathbf{N}_t.$$

Upon iteration, the population will converge to a steady, determined rate of population growth given by r, at which time the proportion of organisms in different age classes becomes constant: the stable age distribution. This independence of initial age structure is known as *ergodicity*. However, should the fecundities or survival rates (and thus the matrix **M**) vary with time (perhaps as a result of environmental stochasticity), then there is no constant, determined population growth rate (or age distribution); instead, population growth rate will vary over time.

In a stochastic environment one could estimate population growth rate by calculating r using the averages of the fecundity and survival rate schedules. However, this will almost surely overestimate the population growth rate because population growth is a multiplicative process, and the geometric mean of two numbers is less than or equal to the arithmetic mean. Instead, the appropriate average population growth rate is estimated by a mathematical quantity called the *dominant Lyapunov exponent* (Metz et al. 1992) and termed a by Tuljapurkar (1982).

Estimating population growth rates in stochastic environments is extremely difficult analytically because the population growth rate is determined both by the current environmental state and also by the current age/stage structure, which is itself a product of past events. To calculate analytically the stochastic population growth rate (a), a probability density function for all age (or stage) classes must be computed. So far this has been possible only for a two-age-class life history (Tuljapurkar 1989). However, Tuljapurkar (1982) developed an analytical formula using stochastic linearization that allows the approximation of stochastic growth rate conditions where the magnitude of the variation is "small." This "small noise" approximation is

$$a \approx \ln \lambda - \frac{\tau^2}{2\lambda^2} + \frac{\theta}{\lambda^2},$$

where a is the stochastic analogue of r: the mean population growth rate in an environment varying stochastically in time; λ is the dominant eigenvalue of the average projection matrix, $\ln \lambda = r$; τ is a term that reflects both the amount of

variation in the matrix elements and the pattern of covariation between them; and θ reflects the correlation in environmental states between periods. If there is no correlation between periods (i.e., "good" and "bad" years occur at random), this term can be ignored, which considerably simplifies calculations. (Formulas for τ and θ are given in Tuljapurkar 1982, 1989, 1990*a*.) This "small noise" approximation works best when the standard deviation of the term is less than half the magnitude of the mean, but it may work qualitatively well when the variation is larger (A. Grant and T. G. Benton, unpublished manuscript).

It is also possible to estimate a numerically, using Monte Carlo simulations and the equation

$$a \approx \lim_{t \to \infty} \frac{1}{t} [\ln(N_t) - \ln(N_0)]$$

(Metz et al. 1992).

To do this, one starts with an arbitrary population structure ($\mathbf{N}_0$) and subjects it to a random sequence of projection matrices simulated from the means and variances for each term. Under most conditions, the population growth is ergodic (Caswell 1989). If one repeats the simulation many times, then the average of the population growth rates is the expected growth rate of a population living in that stochastic environment.

Selection pressures on the age-specific vital rates can be predicted by elasticity (proportional sensitivity) analysis (Caswell 1989). If a projection matrix is a representation of a multivariate phenotype, with each age-specific vital rate (matrix element) having some genetic contribution, then we can estimate the sensitivity of fitness to mutant alleles arising that cause small changes in each of the vital rates. If a small change in a matrix element causes very little or no change in fitness, it will not be strongly selected, either for or against. Conversely, if a small change in the matrix element causes a considerable change in fitness, there will be strong selection acting on that allele. Thus, the elasticity of fitness (population growth rate) to changes in the elements of the projection matrix can be used as a measure of the selection pressures on those matrix elements. Although this is a very simplistic view of natural selection, as it assumes selection on each age-specific vital rate is independent and that trade-offs between vital rates do not occur, its simplicity and generality are nonetheless of great value.

In this article, we calculate the selection pressures on a variety of life histories, using both analytical and numerical techniques, and thereby examine the impact of environmental variability on life-history evolution. This allows us to investigate the circumstances under which r can be used as a measure of fitness.

METHODS

Previous investigations (see, e.g., Tuljapurkar 1990*a*) have demonstrated that the impact of stochastic variation on population growth rate is broadly dependent on three factors: the amount of variation in the vital rates, the correlations between variations in vital rates (e.g., whether a good year for fecundities is also a good year for survival rates), and environmental autocorrelation (whether

"good" and "bad" years come in runs or at random). In these investigations we assume that there is no environmental autocorrelation (i.e., the random matrix for each year is independent) but vary the correlations, amount of variation, and life history under investigation. In addition, we investigate the effects of the stochastic variation being sampled from differing sampling distributions.

Using the package MATHEMATICA (Wolfram Research), a procedure was written to calculate Tuljapurkar's "small noise" approximation for a given matrix, amount of variation, and chosen correlation structure. The amount of variation was expressed as the coefficient of variation (CV), whereas the correlations were expressed as three separate correlation coefficients of either one or zero between fecundities, survival rates, and fecundities and survival rates.

Additionally, a program to estimate a using Monte Carlo simulation was written in FORTRAN 77, using routines available from the Numerical Algorithms Group (NAG) library. In its simplest form, this procedure starts with a given matrix, chosen sampling distribution, CV, and chosen rank correlations. The initial population vector is given by the stable age distribution (calculated from its eigenvectors), and each time step's matrix is randomly chosen and depends on the sampling distribution, CV, and correlation structure. Thus, throughout these investigations, each time step's matrix is independent—serial autocorrelation is zero. The procedure then iterates population growth for several to many hundred time steps (depending on the size of the matrix). This realization of "long-run" population growth is repeated several to many hundreds of times to produce an estimate of the mean population growth rate in the "environment" characterized by the CV and correlations. The natural logarithm of this mean long-run population growth rate estimates the dominant Lyapunov exponent, a. The precision of the estimate of a in a single replicate is proportional to 1/(number of generations); the precision of an estimate of a using many replicates is thus a function of 1/[(number of time steps per replicate)·(number of replicates)]. The sample sizes used below (typically 1,000 time steps repeated 1,500 times) are therefore equivalent to simulations of many tens to hundreds of thousands of events.

Numerical differentiation was used to calculate the sensitivity of each matrix element to a small change in that element. Each element of the matrix was varied in turn by a small amount, and a and r were recalculated. The elasticity (e_{ij} = proportional sensitivity; Caswell 1989) is the change in fitness for a given change in the matrix element ($\mathbf{M}_{ij}$) (here we use e^r [$= \lambda$] and e^a as estimates of fitness, as $r \cong 0$):

$$e_{ij} = \frac{\partial a}{\partial \ln \mathbf{M}_{ij}} \text{ or } \frac{\partial r}{\partial \ln \mathbf{M}_{ij}}.$$

The increment added to each $\mathbf{M}_{ij}$ is chosen to be as small as possible such that the function can be best approximated by a straight line. When calculating a or r analytically, this increment only needs to be large enough to ensure the rounding error is much smaller than the change in fitness (we chose a value of 0.01 for the MATHEMATICA procedure, which calculates a and r analytically). When calculating fitness numerically, the increment must be large enough such that it is substantially larger than the sampling variance of the realized value of $\mathbf{M}_{ij}$ (we

chose a value of 0.05 for the FORTRAN procedure, which calculates a and r numerically, and a analytically for comparison). As long as the function ($da/d \ln \mathbf{M}_{ij}$) is close to a straight line, the actual size of the increment should make little difference to the estimate of sensitivity or elasticity. However, to ensure this did not influence our results, when we compare different measures of fitness below ("small noise" a, "simulation" a and r), we use estimates all generated from the same procedure, using the same increment to calculate sensitivity or elasticity.

Using the simulation procedure, different sampling distributions were chosen to assess the effect of distribution on the sensitivities and population growth rate of different life histories. For fecundity terms, we used the lognormal distribution and the normal distribution (truncated at zero to prevent negative fecundities). At very high CVs this truncation produces a distribution that consists of a large mode of zeros with relatively few nonzero terms. For survival rate terms we used the beta distribution (which varies over [0, 1]) and a normal distribution truncated at zero and one. At extreme CVs this produces a more or less bimodal distribution with modes of zero and one. In addition to these continuous distributions, we investigated the effects of two sorts of discrete distributions. In the first we use a mixture of "good" and "bad" years and while maintaining the same mean values increase the difference between "good" and "bad" years to simulate increasing variability. The status of each year was chosen at random, from a uniform distribution. In the second, we maintained very low levels of variation (CVs of 0.001 for both fecundity and survival rate terms) and investigated the impact of increasing frequency of "catastrophic years" when fecundity and/or survival rate terms were reduced to 0.1. Therefore, as the frequency of catastrophes increased, the average vital rates were reduced. For a given frequency of catastrophes, the catastrophic time steps were chosen at random from a uniform distribution.

Table 1 shows the matrices used in this investigation and defines the terminology used below. All numerical results were obtained from running the procedures on the University of East Anglia's MicroVAX 3100/90s.

ANALYTICAL RESULTS

The effect of stochasticity on the elasticity, calculated using Tuljapurkar's small noise approximation, depends on the amount that life-history traits vary over time. At high CVs the elasticity differs most from the deterministic elasticity, independent of the size or shape of the life history (fig 1). Thus, selection pressures on life histories will be different in variable environments and constant environments.

The absolute amount that a given variable environment (as characterized by a given amount of variability and correlation structure) will change the elasticities depends on both the size and the shape of the matrix. Thus, on the average, shorter life histories are most affected by variability (for a CV of one, and zero correlations between variations in the matrix, a 2-yr matrix will have elasticities changed by an average of $0.1390 \pm .067$ per element, a 4-yr by $0.0420 \pm .048$, a 6-yr by $0.0220 \pm .035$, and an 8-yr by $0.0130 \pm .020$; data shown in fig. 1). Addition-

TABLE 1

LIFE HISTORIES USED IN THE PRESENT STUDY

Life History	F_1	F_2	F_3	F_4	F_5	F_6	F_7	F_8	S_x	r	T_0
$2r$	.33333	1.33333							.5	-3.00×10^{-7}	1.6667
$2h$	.55556	.55556							.8	5.54×10^{-6}	1.4444
$2l$	.83333	.83333							.2	3.43×10^{-7}	1.1667
$3f$	0	1.33333	1.33333						.5	-1.07×10^{-6}	2.3333
$4f$	0	1.14286	1.14286	1.14286					.5	-9.72×10^{-7}	2.5714
$4r$	0	.66667	1.33333	2.66667					.5	4.17×10	3.0000
$4h$	0	.51230	.51230	.51230					.8	-5.61×10^{-6}	2.8525
$4l$	0	4.03226	4.03226	4.03226					.2	2.16×10^{-7}	2.2258
$6r$	0	.79188	1.02944	1.33828	1.73976	2.26169			.5	-6.83×10^{-7}	3.2009
$6h$	0	.37185	.37185	.37185	.37185	.37185			.8	2.46×10^{-6}	3.5631
$6l$	0	4.00128	4.00128	4.00128	4.00128	4.00128			.2	-4.55×10^{-8}	2.2484
$8r$	0	0	1.48331	1.95724	2.5826	3.40776	4.49656	5.93325	.5	-5.33×10	4.3998
$8h$	0	0	.42352	.42352	.42352	.42352	.42352	.42352	.8	-1.81×10^{-7}	4.8683
$8l$	0	0	20.0013	20.0013	20.0013	20.0013	20.0013	20.0013	.2	3.06×10^{-7}	3.2496

NOTE.—The name indicates the dimension of the matrix and its characteristics (*f*, "flat"—fecundities unchanging throughout adult life, moderate survival rate; *r*, "rising"—fecundities increasing throughout adult life, moderate survival rate; *h*, high survival rate—fecundity unchanging throughout adult life; *l*, low survival rate—fecundity unchanging throughout adult life). The F_x indicate the value of fecundity at a given age; S_x indicates age-independent survival rate; *r* indicates the deterministic population growth rate; and T_0 is the average generation time for the matrix (see Tuljapurkar 1990*a*).

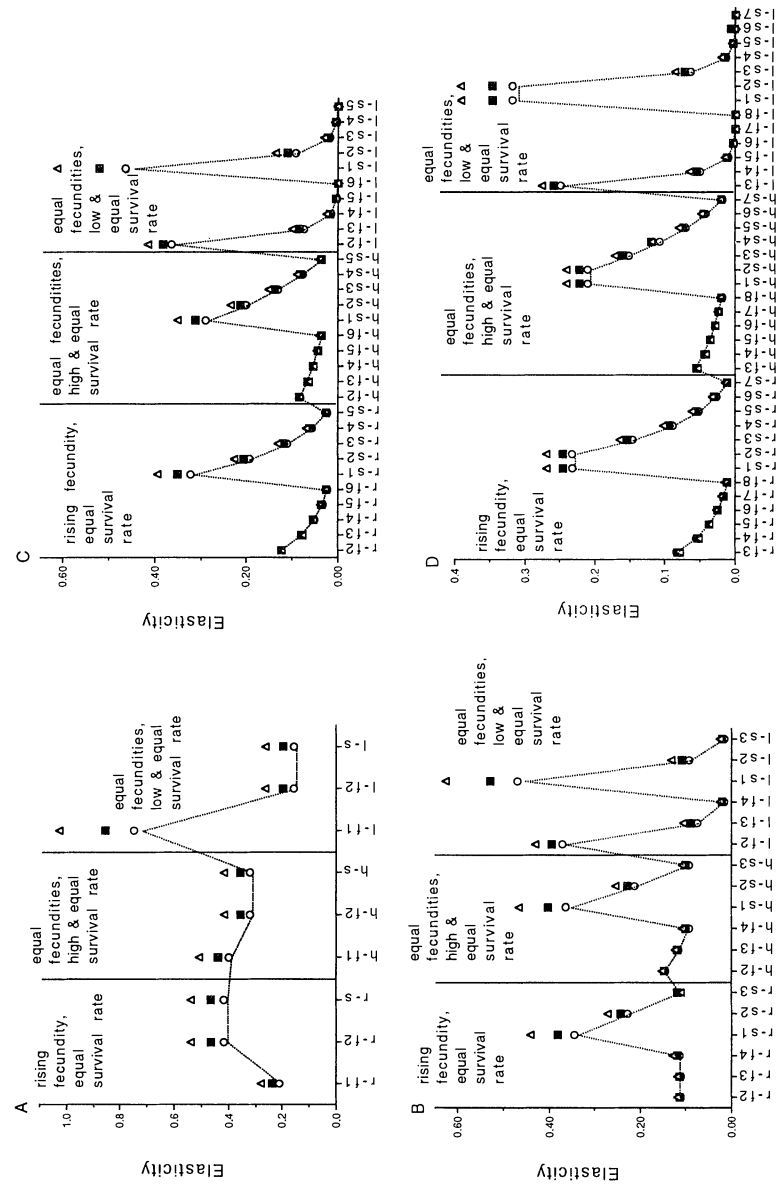

FIG. 1.—Elasticity of λ ($= e^r$) and e^a to changes in the vital rates of a variety of life histories, in a variety of environments characterized by different levels of variability (as measured by the CV: ratio of standard deviation to mean). Elasticity is the proportional change in λ (equals e^a) for the same change in the vital rates (e.g., an elasticity of one means a 1% change in the vital rate would cause a 1% change in λ). Elasticities were calculated using Tuljapurkar's approximation. Variability is zero (*dotted line*), CV of 0.33 (*open circle*), 0.67 (*solid square*), and 1.0 (*open triangle*). Variations in each vital rate are independent (all correlations equal zero). *A*, 2-yr life histories (2*r*, 2*h*, 2*l*; see table 1 for descriptions); *B*, 4-yr life histories (4*r*, 4*h*, 4*l*); *C*, 6-yr life histories (6*r*, 6*h*, 6*l*); and *D*, 8-yr life histories (8*r*, 8*h*, 8*l*). Throughout the figures, the vital rates are indicated using the convention $X - Yn$, where X is r, h, or l and describes the matrix (see table 1), Y is f or s (for fecundity or survival rate), and n indicates age class. Also, the dotted line used (here for CV equals zero) is to aid clarity; its use should not be taken to indicate continuous variation between categories.

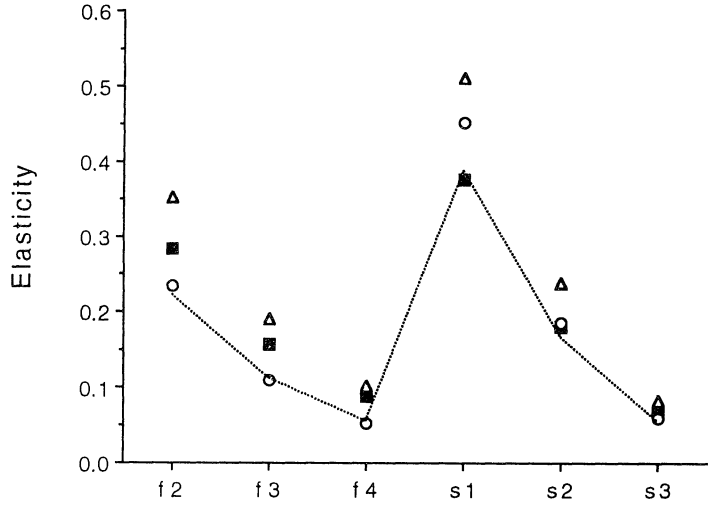

FIG. 2.—The effect of varying fecundities, survival rates, or both on elasticity. Elasticities are calculated using Tuljapurkar's approximation: deterministic elasticity (*dotted line*); fecundity CV = 1, survival rate invariant (*solid squares*); fecundity invariant, survival rate CV = 0.5 (*open circles*); fecundity CV = 1.0, survival rate CV = 0.5 (*open triangles*). The life history is 4*f* (see table 1). Correlations are +1.

ally, on the average, life histories with a low adult survival rate and high fecundity (i.e., 2*l*, 4*l*, 6*l*, and 8*l*) are more affected than those with a high adult survival rate and low fecundity (i.e., 2*h*, 4*h*, 6*h*, and 8*h*), which are affected approximately equally to life histories with a medium adult survival rate and increasing fecundity through life (i.e., 2*r*, 4*r*, 6*r*, and 8*r*). For a CV of one and correlations of zero, life histories with a low survival rate will have elasticities changed by an average of 0.0470 ± .069, those with a high survival rate by 0.0270 ± .036, and those with rising fecundity by 0.0270 ± .040 (data shown in fig. 1).

The fecundity and survival rate terms are also affected differently. In general, variability has a greater effect on survival rate elasticities than fecundity elasticities (for a CV of one and zero correlations, fecundity elasticities are changed by 0.0260 ± .055, median 0.005; survival rate elasticities by 0.0410 ± .046, median 0.022; data shown in fig. 1). For the analyses shown in figure 1, the effect of increasing variation generally increases the elasticities for both survival rates and fecundities. There is a very slight reduction of elasticities with increasing variation only in the first two fecundities of the 6*r*, 6*h*, 8*r*, and 8*h* life histories, which is almost imperceptible in figure 1.

The effect of varying fecundities, survival rates, or both is shown in figure 2 for a single matrix. If only fecundities are variable, fecundity elasticities are affected most (increased when correlations are +1), though survival rate elasticities are very slightly affected (decreased when correlations are +1); the reverse is true if only survival rates are affected. The effect of a given level of variation is greater for any element if all vital rates are variable rather than if only survival rates or fecundities are variable (fig. 2).

Different correlations between the elements in the life-history matrix cause different changes in the elasticities with given amounts of variation (fig. 3). If all variations in the vital rates are independent (i.e., correlations are zero), a given amount of variability has the least effect. Strong positive correlations between the variations in the vital rates magnify the effects of a given level of variation (with all elasticities increasing). However, if there are strong positive correlations between fecundities and between survival rates, yet strong negative correlations between fecundities and survival rates, some elasticities are increased and some may be markedly decreased (fig. 3). In longer life histories, selection pressures on fecundities tend to be decreased, whereas those on survival rates increase (fig. 3*B–D*). In very short life histories, the reverse may be true (fig. 3*A*), with selection pressures on fecundities increasing, whereas those on survival rates decrease.

The data shown in figure 3 are with a CV of 1.0 for fecundities and survival rates. For some organisms, for example, some living in the marine environment (see, e.g., Loosanoff 1964), much higher CVs are possible. Very large variations in fecundity may have a huge effect on the elasticities (fig. 4). The data in figure 4 are for a CV of 3.0 for fecundities and 0.5 for survival rates. In this situation, if any elasticities are reduced (they are in 4-yr and larger matrices for independent variations and negative correlations between survival rate and fecundity variations), then they are survival rate elasticities. In an environment in which fecundities are highly variable, therefore, selection on survival rate terms may be reduced, while selection on fecundities is increased.

Thus, the amount that selection is affected by variability depends on the amount of variation (both fecundity and survival rate variations), the correlations between variations, and also the life history in question. If there are positive correlations between all variations, then selection is increased on all vital rates. However, in environments in which there is negative correlation between variations in fecundity and survival rate, variability may increase the selection pressure on survival rates and decrease it on fecundities (longer life histories, moderate variation) or vice versa (shorter life histories, moderate variation; longer life histories, very large variation).

SIMULATION RESULTS

As expected, both the amount of variation and the correlations between vital rates affect the magnitude of fitness measured by a, the dominant Lyapunov exponent estimated numerically (fig. 5). In particular, higher levels of stochastic variation reduce fitness most. Different correlation structures have markedly different effects. The largest reduction in fitness occurs when there are positive correlations between variations in fecundities and survival rates. At intermediate to high levels of variation (CV greater than 0.5), the least reduction in fitness occurs with strong negative correlations between variations in fecundities and survival rates. The sampling distribution from which the variations in vital rates were chosen also affects the magnitude of fitness: especially at high CVs, sampling from different distributions may produce markedly different estimates of

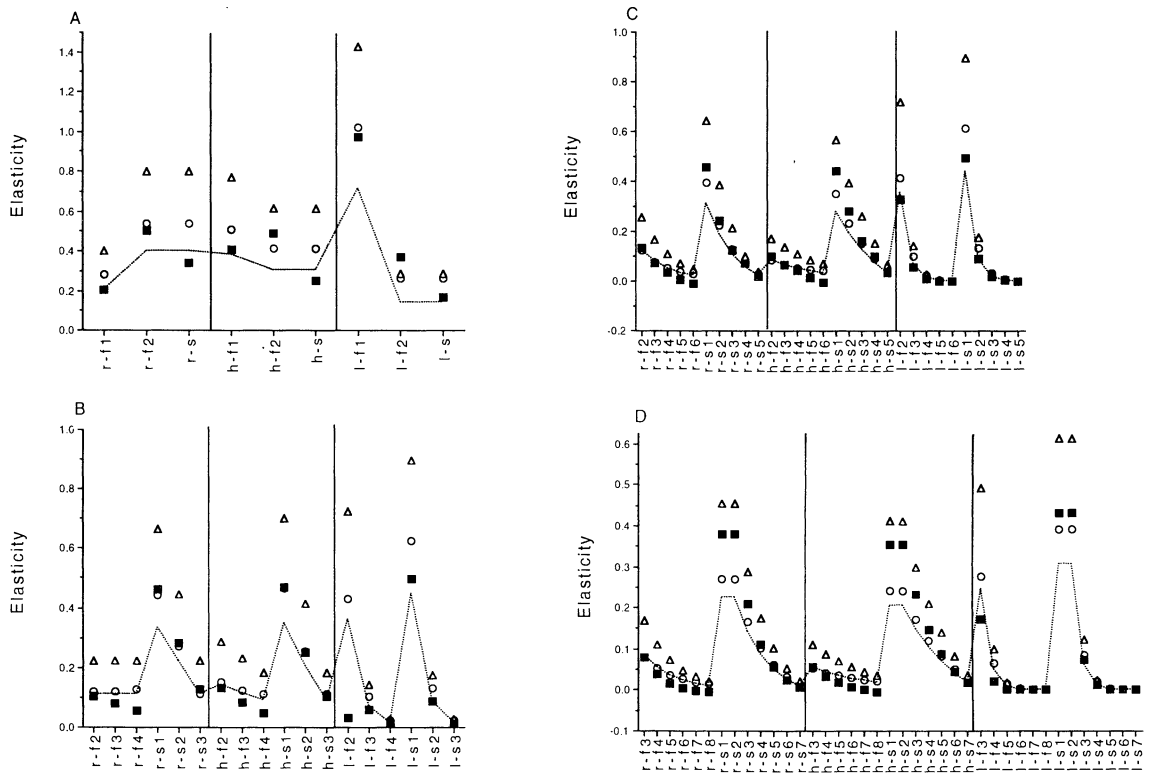

FIG. 3.—The effect of different correlations between variations in the vital rates on the elasticities of a variety of life histories. Elasticities are calculated using Tuljapurkar's approximation: deterministic elasticity (i.e., no variability) (*dotted line*); all variations in vital rates independent (*open circles*); all variations in vital rates positively correlated (correlations = +1) (*open triangles*); variations in fecundities positively correlated (+1), variations in survival rates positively correlated (+1), variations in fecundities and survival rates negatively correlated (−1) (*solid squares*); CV is 1.0 for both fecundities and survival rates. *A*, 2-yr life histories (2*r*, 2*h*, 2*l*; see table 1 for descriptions); *B*, 4-yr life histories (4*r*, 4*h*, 4*l*); *C*, 6-yr life histories (6*r*, 6*h*, 6*l*); *D*, 8-yr life histories (8*r*, 8*h*, 8*l*); fx = fecundity at age x; sx = survival rate from age x to $x + 1$.

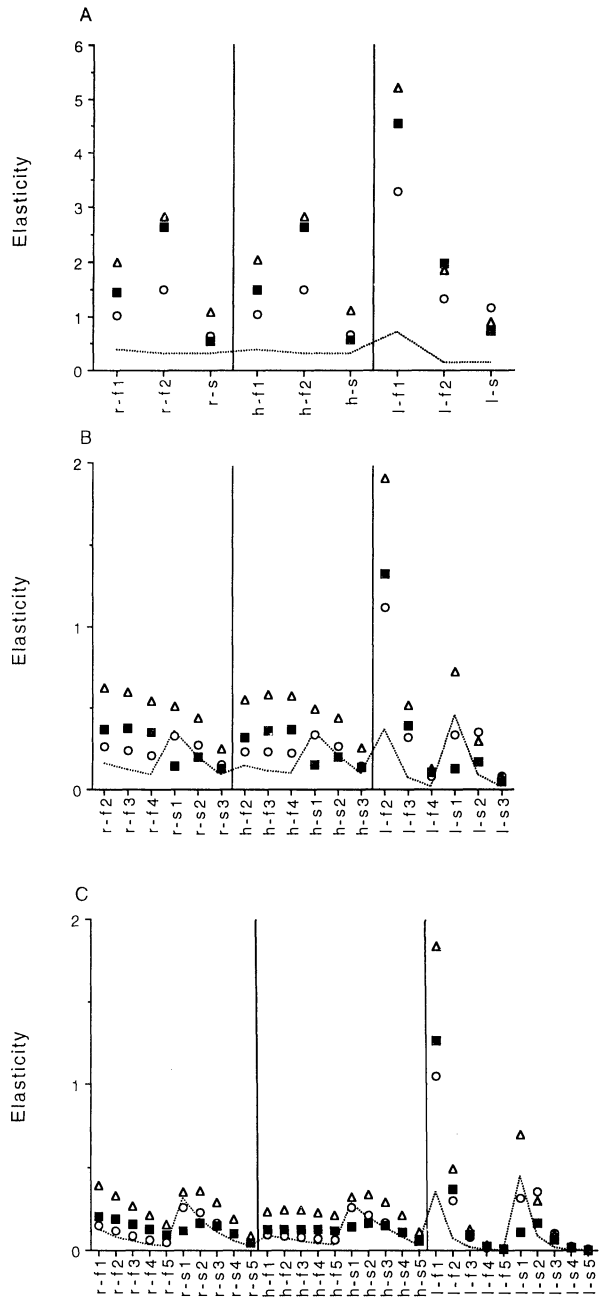

FIG. 4.—As for fig. 3, but using a CV for fecundities of 3.0 and CV for survival rates of 0.5

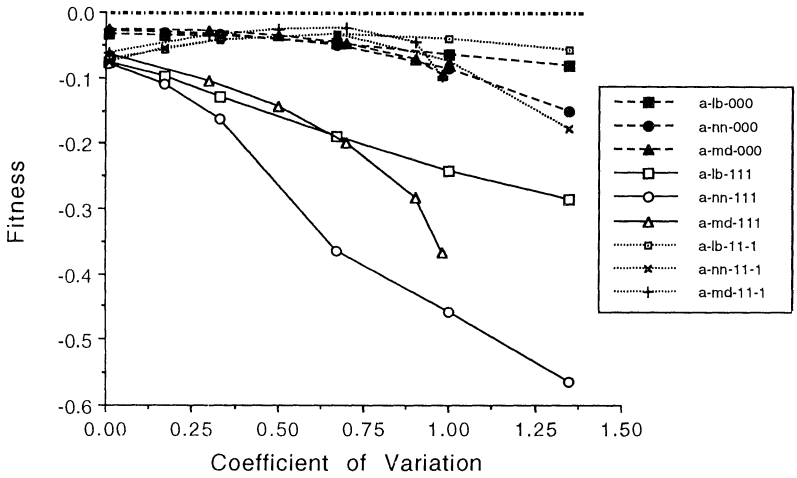

FIG. 5.—The relationship between fitness (as measured by a, calculated numerically) and the amount of variation in fecundities, the correlations between variations among the vital rates, and the sampling distribution for the random variation. The matrix used was 4r (see table 1). The lines are labeled according to the convention *a-xx-yyy*, where *xx* refers to sampling distributions (*lb* is lognormal for fecundities, beta for survival rates; *nn* is truncated normal distributions for both fecundities and survival rates; *md* is mixed distribution of either "good" or "bad" years), and *yyy* refers to the magnitude of the correlations between fecundities, survival rates, and fecundities and survival rates. The variation in survival rates was fixed at a CV of 0.5; that of fecundities was varied. For each set of parameter values, simulations of *nn* consisted of 600 time steps repeated 390 times; of *lb*, 250 time steps repeated 250 times; and of *md*, 750 time steps repeated 500 times. The 95% CI error bars are smaller than the symbols. The line at which fitness equals zero refers to the fitness of the life history in a constant environment ($a = r = 0$) or the usual estimate of fitness (calculated as r) if stochasticity is ignored.

fitness (fig. 5). At a CV of one for fecundities and 0.5 for survival rates, sampling from normal distributions reduced fitness most, whereas sampling from lognormal (fecundities) and beta distributions (survival rates) had the least effect. These results are independent of the life history under investigation.

As with the analytical results, the simulations show that increasing variation has the greatest effect on the elasticities (fig. 6*A*). Additionally, different correlations produce different magnitudes of change in the elasticities (fig. 6*B*). However, the simulation results in figure 6 show some differences from the analytical results. For the results shown, there is an accentuation of the impact of variation on survival rate elasticities, whereas the elasticities of fecundities mostly do not differ from the deterministic elasticity.

Different shapes and sizes of life history influence the amount that elasticities are affected by variation, as expected (fig. 7). However, the simulation results in figure 7 are different from the analytical results shown in figure 3. The results in figure 3 lead us to expect that for high CVs and strong positive correlations, the elasticities should all be greater in the stochastic environment than the deterministic environment. The results in figure 7 show that in sampling from mixed distributions (good years and bad years, and no "in-between" years), variation does

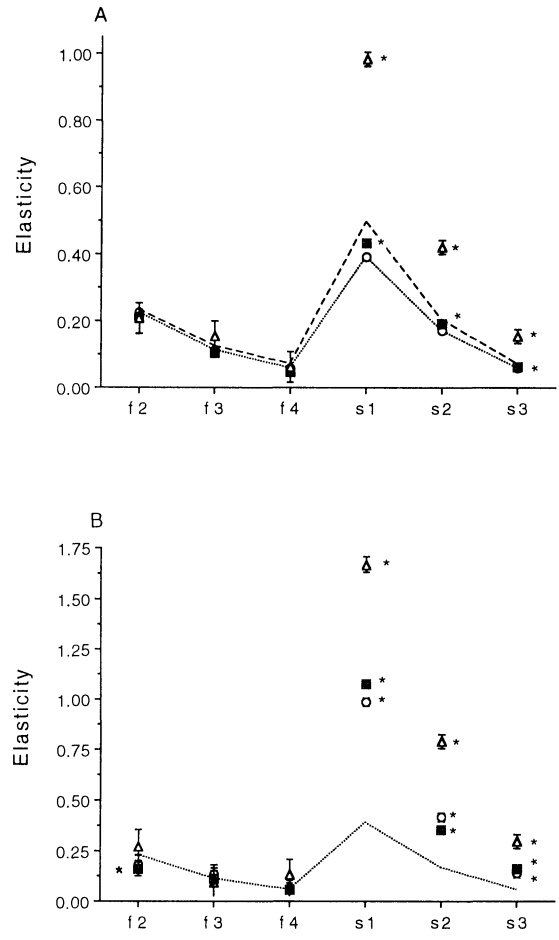

FIG. 6.—*A*, The effect of variability (in both fecundities and survival rates) on elasticities calculated numerically. Deterministic elasticity (*dotted line*) is indicated, CV of 0.01 (*open circles*), CV of 0.5 (*solid squares*), CV of 0.9 (*open triangles*). The dashed line indicates elasticities calculated using Tuljapurkar's formula, with a CV of 0.9. *B*, The effect of different correlations on elasticities calculated numerically. Deterministic elasticity (*dotted line*) is shown; variations in all vital rates independent (*open circles*), variations in all vital rates correlated at +1 (*open triangles*), correlations between survival rates and between fecundities is +1, between survival rates and fecundities is −1 (*solid squares*). Error bars are 95% confidence estimates. Asterisks indicate significant differences ($P < .05$) between the stochastic and deterministic elasticity. The life history used was $4f$ (see table 1). The sampling distribution was mixed (i.e., good years and bad years). The simulations consisted of 1,000 time steps repeated 1,500 times.

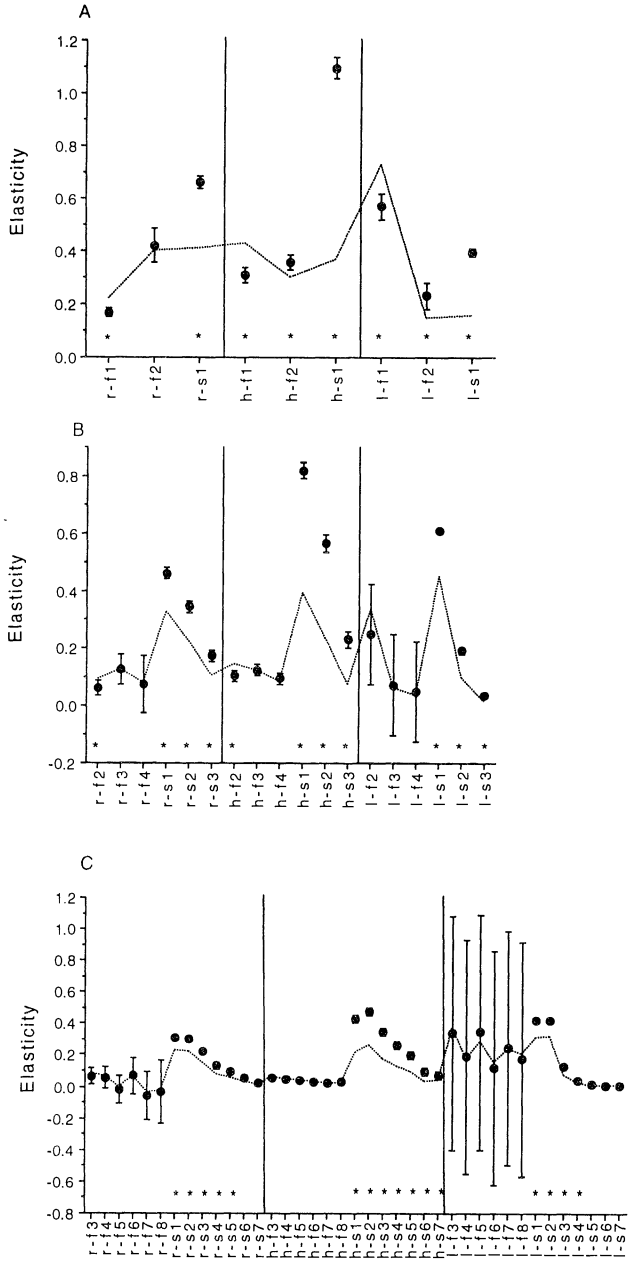

FIG. 7.—Elasticities for a variety of life histories calculated numerically for an environment characterized by variability in fecundities of CV equals 0.9; that of survival rates CV equals 0.5, and correlations among all variations equal +1. The sampling distribution was mixed (i.e., good years and bad years). The simulations consisted of 1,000 time steps repeated 1,500 times. Deterministic elasticity (*dotted line*) and stochastic elasticity (*solid circles*) are shown. Error bars are 95% confidence estimates. Asterisks indicate significant differences ($P < .05$) between the stochastic and deterministic elasticities. *A*, 2-yr life histories (2*r*, 2*h*, 2*l*); *B*, 4-yr life histories (4*r*, 4*h*, 4*l*); and *C*, 8-yr life histories (8*r*, 8*h*, 8*l*).

increase survival rate elasticities, but it may reduce, increase, or have little effect on fecundity elasticities. In shorter life histories, variation can reduce the selection pressures on fecundities when we would expect them to be increased.

The results shown in figure 7 are calculated with sampling from only one, of many possible, sampling distributions. Especially with shorter life histories, the sampling distribution used for the variations in vital rates may make a big difference to the amount that a given, variable environment affects the elasticities (fig. 8). Sampling from unimodal distributions (lognormal and beta used here) causes the greatest correspondence between analytical and numerical results. Sampling from truncated normal distributions causes survival rate elasticities to be greatly increased over the expected levels. At high CVs, truncated normal distributions become "odd" shapes: for fecundities, in which truncation occurs at zero, the distribution has a very large mode at zero with a very long, flat tail; for survival rates, in which truncation occurs at zero and one, the distribution becomes essentially bimodal, with modes at zero and one. Sampling from mixed distributions may give results close to the expected analytical results for larger but not smaller matrices. For the 2-yr life history (fig. 8*A*), the numerical results are all significantly different from each other (using three sampling distributions) and from the expected (other than for the survival rate elasticity, with sampling from lognormal and beta distributions, when there was no difference between the numerical and analytical sensitivities). With the 4- and 8-yr life histories, the numerical elasticities calculated from the truncated-normal distributions are generally significantly different from the elasticities calculated with sampling from the other distributions, which are much more similar to each other.

That the specific sampling distribution used in the simulations also affects the results is unsurprising but has important implications for the interpretation of observations on real organisms. As an extreme, consider the case of an organism living in a constant environment with occasional catastrophic occurrences that cause average fecundities and survival rates to become very low for a short time (realistic examples are such things as occasional storms, droughts, famines, floods, freezes, etc.). Even if these catastrophes are very rare, there may be appreciable differences in the elasticities of the organism's vital rates (fig. 9). Events as rare as once every 100 time steps cause slight, but significant, differences in the selection pressures on all the vital rates in an organism's life history. These differences become more marked as the frequency of catastrophes increases. When the catastrophe affects both survival rates and fecundities, then elasticities are reduced, which signifies a relaxation in selection pressures. This result indicates that marginal changes in life-history terms have little effect on the magnitude of fitness compared to the frequency of catastrophes. When catastrophes affect only fecundities, selection pressure is reduced on early fecundities and survival rates but increased on late fecundity and survival rate, which perhaps indicates that if catastrophes strike when organisms are young, selection is increased on their survival and later fecundity in order to compensate. When survival rates only are hit, selection pressures tend to be increased on fecundities and reduced on survival rates perhaps in order to compensate for possible early death brought about by the event.

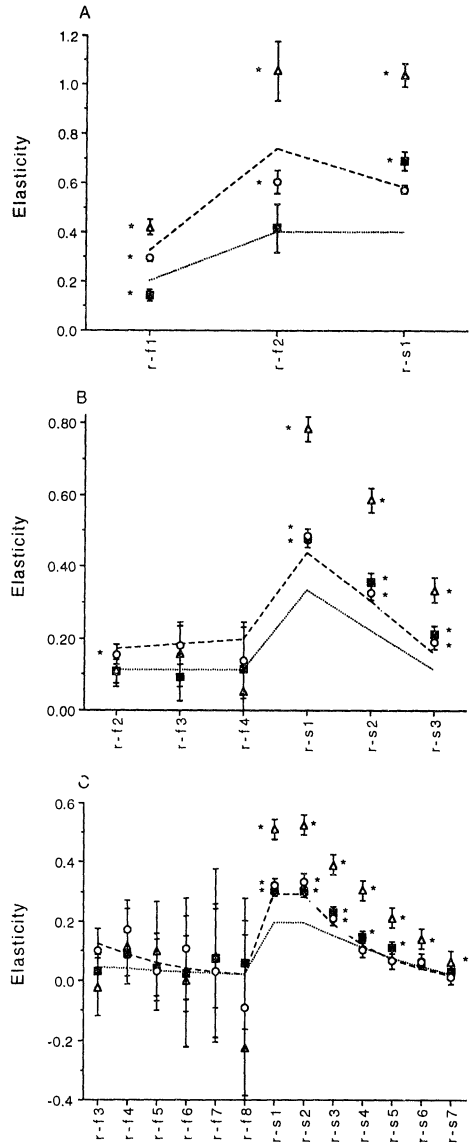

FIG. 8.—The effect of different sampling distributions on elasticities calculated numerically. Deterministic elasticity (*dotted line*), lognormal-beta sampling distributions, *lb* (*open circles*), normal-normal distributions, *nn* (*open triangles*), mixed distributions, *md* (*solid squares*), elasticities calculated using Tuljapurkar's formula (*dashed line*). Error bars are 95% confidence estimates. Asterisks indicate significant differences ($P < .05$) between the stochastic and deterministic elasticity. Variations between all vital rates are correlated at +1; coefficient of variation for fecundity equals 1.0, and that of survival rates equal 0.5. *A*, 2-yr life history (2*r*); simulations consisted of 1,000 time steps repeated 1,500 times. Tuljapurkar's elasticities are significantly different to all other estimates except for the survival rate estimated using sampling from *lb*. *B*, 4-yr life history (4*r*); simulations consisted of 1,000 time steps repeated 1,500 times. Tuljapurkar's elasticities are significantly different to *f*2 (*nn* and *md*), *s*1–*s*3 (*nn*, *lb*, and *md*). *C*, 8-yr life history (8*r*); simulations consisted of 1,000 time steps repeated 1,500 times (*md*), 1,000 time steps repeated 1,250 times (*nn*), and 500 time steps repeated 700 times (*lb*). Tuljapurkar's elasticities are significantly different to *f*3 (*nn* and *md*), *s*1–*s*7 (*nn*), *s*1–*s*2 (*lb*) and *s*3–*s*5 (*md*).

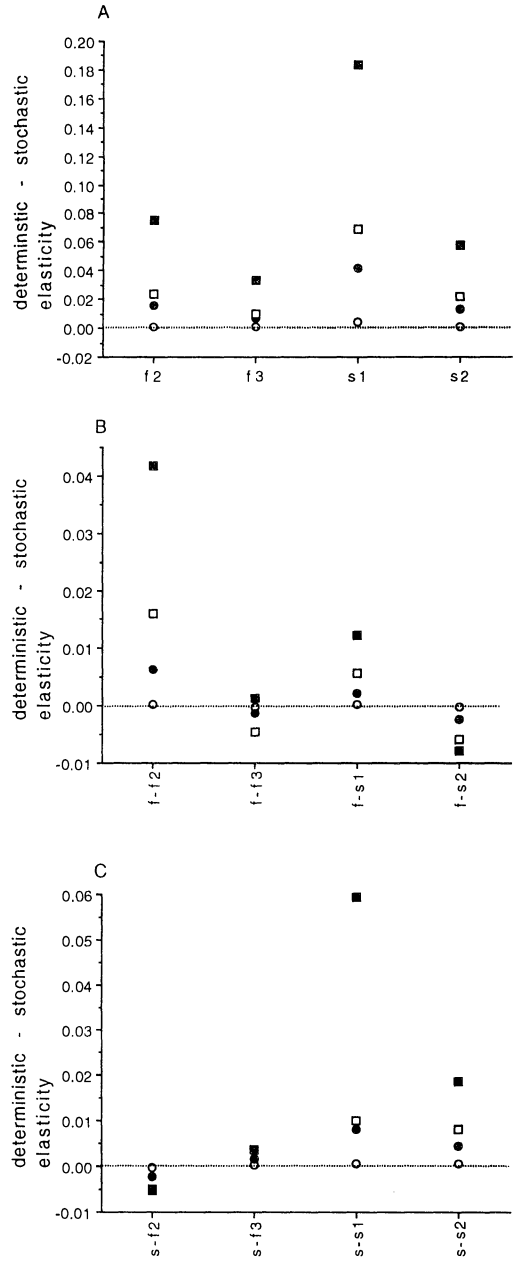

FIG. 9.—The effect of occasional catastrophic years on elasticities. The *Y*-axis is deterministic elasticity minus stochastic elasticity. *A,* Both fecundities and survival rates are set to 0.1 in a catastrophic year; *B,* fecundities only are reduced to 0.1 in a catastrophic year; *C,* survival rates only are reduced in a catastrophic year. The dotted line indicates no difference between stochastic and deterministic elasticities; catastrophe frequency is 1 per 107.1 yr (*open circles*), 1 per 10.6 yr (*solid circles*), 1 per 6.5 yr (*open squares*), and 1 per 2.3 yr (*solid squares*). The 95% CI is smaller than symbols: *all* differences between deterministic and stochastic elasticities are significantly different ($P < .05$). The matrix used was $3f$. Background variation was a CV of 0.001. Simulations were 750 time steps repeated 750 times.

DISCUSSION

Stochastic variation in an environment, if it affects the life histories of organisms within that environment, causes changes in the elasticities of, and thus selection pressures on, their life histories. Previous studies have compared a variety of life histories in both constant and variable environments (Tuljapurkar 1989, 1990*a*). These have shown that the life history favored in the constant environment (the one with greatest r) is not necessarily the one favored in a variable environment (the one with the greatest a). Tuljapurkar terms this the "cross-over effect," and it results from the differential reduction in fitness on different life histories caused by stochasticity. For example, shorter life histories tend to suffer a bigger reduction in fitness for a given variability than longer life histories. Thus, a short life history may be favored in a constant environment (Sibly and Calow 1986), whereas in a variable environment a longer life history may instead be favored. This study now illustrates that selection on a life history in variable environments can differ appreciably from selection in constant environments. Therefore, our conclusion is similar to earlier studies: different traits are favored by selection depending on the amount (and correlation structure, etc.) of variation, and we give some indications of the circumstances in which variability is likely to matter appreciably. Thus, detailed optimality studies of life history should at least consider the effects of stochastic variation, especially in shorter-lived organisms or organisms known to live in variable environments, otherwise their conclusions may be inaccurate. Perhaps a good indication of the necessity of considering variation would be if the results of deterministic analyses suggested that the organism in question was behaving maladaptively (e.g., the expected "optimal" clutch size of great tits in Whytham Wood is greater than the observed; Boyce and Perrins 1987). Maladaptivity is a possibility, but also so is the possibility that the assumptions of the simple deterministic analysis are so grossly contravened that the analysis does not indicate adaptation.

The real world does cause variations in the fecundities and survival rates of organisms. In several reports, the year-to-year variability in organisms' life histories has been noted: red deer, *Cervus elephas* (Benton et al., in press); barnacle geese, *Branta leucopsis* (Middleton et al. 1993); barnacles, *Semibalanus balanoides* (Wethey 1985); gorgonian coral, *Leptogorgia virgulata* (Gotelli 1991); a winter annual, *Collinsia verna* (Kalisz 1991); perennial bunchgrass, *Danthonia sericea* (Moloney 1988); and marine green alga, *Ascophyllum nodosum* (Aberg 1992). Collating these reports, we find that the median CV for fecundities was 0.675 (interquartile range = 0.475–1.156, $n = 9$; i.e., five species at nine sites) and that of survival rates is 0.339 (interquartile range = 0.225–0.451, $n = 11$; i.e., six species over 11 sites). In addition, there are usually quite strong covariances between the vital rates (T. G. Benton and A. Grant, unpublished manuscript). For a variety of reasons it is possible that the levels of variability experienced by many organisms may exceed those reported in the literature. There may be a bias toward studying organisms whose life history is known not to be particularly variable; short-term studies are unlikely to experience the whole range of environmental variation, and so on. In some environments, organisms

may be more affected by environmental variation than others. For example, in the marine environment large variations in recruitment occur in fish (Cushing 1975; Laevastu and Favorite 1988) and in benthic invertebrates (Coe 1953). For year-to-year variation in numbers of newly settled juvenile starfish (*Asterias forbesi*) at a single site, CVs range up to 2.5 (our calculations on data in Loosanoff 1964). Rare events such as severe winters can have a significant impact on the population ecology of intertidal species for more than a decade (Sebens and Lewis 1985). Large amounts of stochastic variation in several life-history components (such as survival, seed set, and age to first reproduction) have been found in a long-term study of sand dune plants (Klinkhamer and de Jong 1983; de Jong and Klinkhamer 1988), such that delayed flowering in these biennial plants is favored.

The results reported here indicate that increasing environmental stochasticity causes increasing changes in the selection pressures (elasticities) of life histories. For high levels of variability, the elasticities may be very different from the deterministic elasticities. For a given amount of variation, the correlations between variations in the vital rates determine the amount the elasticities are affected. The analytical results indicate that correlations between variations in the vital rates that are zero or positive tend to increase the selection pressures on vital rates, whereas negative correlations between fecundity and survival rate variations may increase some selection pressures (notably survival rates in longer life histories) and decrease others (notably fecundities in longer life histories). This is especially evident in the 4-yr life history with low adult survival rate (fig. 3*B*). With strong negative correlations, the selection pressures on fecundities are reduced to very low levels, whereas selection pressures on survival rates are increased. This is similar to bet hedging (Seger and Brockmann 1987) but does not involve a "cost of reproduction" trade-off. It is interesting that the analytical results indicate it is most likely with negative correlations between fecundity and survival rate variations but not with zero or positive correlations.

The simulation results presented here are not so clear-cut. The general results hold: elasticities change according to the amount of variation and the correlations experienced, shorter life histories are prone to larger changes in elasticities in a given environment, and survival rate terms are generally more affected than fecundity terms. However, the exact pattern of changes depends on the sampling distribution used for the variations in vital rates. For example, a reduction in selection pressures on fecundities, and increase on survival rates, may occur with positive correlations (rather than just negative correlations) if variability is bimodal: good and bad years, with no in-betweens (fig. 7). Sampling from different distributions may produce significantly different results, both between distributions and from the analytical expectations (fig. 8). The closest match between analytical results and simulation results occurs if the variability in fecundity terms is sampled from a lognormal distribution and variation in survival rate terms is sampled from a beta distribution. We explore the accuracy of Tuljapurkar's small noise approximation in detail elsewhere (A. Grant and T. G. Benton, unpublished manuscript). The dependence of results on sampling distribution is exemplified by the effect that occasional catastrophic events have on an organism living in an otherwise constant environment (fig. 9).

Catastrophic events (such as severe weather, famine, etc.) can cause small, but significant, changes in elasticities even if they occur as infrequently as once every 100 yr. It therefore appears unlikely that it is possible to characterize fully the stochastic population growth rate for any organism, simply because the distributions of fecundities and survival rates (and correlations between them) must be fully known, which would perhaps require several hundred years' data.

Deterministic theory predicts that low adult survival and high juvenile survival (roughly equivalent to the "low" life histories used herein—2*l*, etc.) lead to semelparity, whereas high adult survival and low juvenile survival (the "high" life histories—2*h*, etc.) lead to iteroparity (see, e.g., Stearns 1992). The deterministic elasticities tend to support this: in the "low" life histories, early survival rate and fecundity have the greatest elasticities, whereas later vital rates have lower elasticities. Conversely, the "high" life histories have more uniform elasticities for both survival rates and fecundities. Schaffer (1974) introduced variability into the deterministic analysis and showed that variable juvenile survival tends to lead to semelparity, whereas variable adult survival tends to lead to iteroparity. When only fecundity or survival rate varies (fig. 2), the largest effect is on the elasticity of the variable vital rates, though there are slight changes in the elasticities of the nonvariable vital rates. Therefore, variable fecundities (which includes juvenile survival rate to the following census) strengthen selection on fecundities throughout life and slightly increase selection on survival rates during the reproductive phase of life. Conversely, variable adult survival strengthens selection on early survival and fecundity (slightly) and slightly decreases selection on fecundity late in life. Thus, the results in figure 2 are consistent with the findings of Schaffer (1974).

In an environment in which a life history has evolved to its "optimum," we might expect that there would be no selection pressures on the vital rates (Caswell 1989); that is, the elasticities (incorporating trade-offs between vital rates) would tend to zero. If we assume that to incorporate suitable trade-offs into the elasticity analysis would reduce deterministic elasticities to zero, we assume that stochastic elasticities are reduced by the same amount (thus mentally flattening the figures such that deterministic elasticity is zero for all elements). The difference between deterministic and stochastic elasticities will therefore be similar to the selection pressures on life histories (incorporating trade-offs) of an optimum life history in a constant environment transferred to a variable environment. Thus, we can perhaps approximate patterns of selection pressures on life histories when trade-offs are incorporated. For example, consider the elasticities shown for the "low" life history in figure 3*B*. When variations in vital rates are independent, the differences between a elasticity and r elasticity are generally small but positive. Thus, if the r elasticities are assumed to be zero (i.e., the life history is optimal), there will be weak positive selection on the vital rates in a variable environment. If variations in vital rates are positively correlated, there will be very strong selection on prereproductive survival and early fecundity, which implies that the life history will tend to evolve toward a semelparous life history. Conversely, if the correlation between fecundities and survival rates is negative, there will be selec-

tion against increasing early fecundity, which implies that evolution will occur toward a more iteroparous life history. The comparison of these results to the simple predictions of deterministic models is not always straightforward; the effect of variability on selection on life histories is complex and depends on many factors, not just the relative amounts of adult and juvenile survival (Tuljapurkar 1990*a*). If the form of the trade-off between fecundity and survival is known, the impact of random variation on the selection pressures can be explicitly calculated (Benton et al., in press).

Changes in the logarithm of population size can be approximated as a diffusion (or Weiner) process, which allows extinction probabilities and the mean, variance, and modal time to extinction to be calculated (Levinton and Ginzburg 1984; Lande and Orzack 1988; Orzack and Tuljapurkar 1989; Orzack 1993). If a is greater than zero, then the probability of ultimate extinction is given by

$$\exp\left(\frac{-2ax_0}{\sigma^2}\right),$$

where x_0 is the logarithm of initial population size, and σ^2, the variance of population growth rate, is approximated by $2(r - a)$ (Tuljapurkar 1982; Orzack 1993). Thus, a small change in the life history that increases a will decrease the extinction probability *unless* the elasticity of r to the change is so much greater than the elasticity of a that σ^2 increases at a faster rate than a. Using this approach, we could examine in detail the impact of different demographic parameters on the probability of population and time taken to extinction. This is, however, beyond the scope of this article.

This study, like many theoretical studies in evolutionary biology, and especially life-history evolution (see reviews in Caswell 1989; Stearns 1992), assumes that populations can grow at a rate unconstrained by density and that there is no frequency-dependent selection. At first sight these assumptions may appear biologically unrealistic, especially that of density independence. However, these assumptions are justified in many ways. First, for many species, especially invertebrates with an annual life cycle, such as insects (Taylor 1979), population growth indeed may be exponential throughout their short lifetimes. In other cases, populations may experience periods when density dependence is relaxed, which allows their population to grow exponentially (MacArthur and Wilson 1967; Murray 1994), especially if the population is predation limited, in which case density dependence may be very weak. Second, although density-dependent effects are widespread in many organisms (Dolman and Sutherland 1995), there is still heuristic value in projecting population growth into the future. A projection is an attempt to describe what *would* happen given certain hypotheses, not to predict what *will* happen. As such, projection is a powerful way to study the present conditions experienced by a population through examining the consequences of conditions remaining the way they currently are. A car's speedometer fulfills a similar role. For example, a 60-mph speed does not predict that in an hour 60 miles will have been covered but rather that the vehicle is traveling at a certain current speed (Caswell 1989). Third, assuming density dependence in theoretical

models necessitates choosing some mathematical function to describe the effects of increasing density on population dynamics. As is well known, different functions and different parameter values can produce widely different dynamic effects (see, e.g., May 1974), so assuming density independence may have a generality not achieved by choice of a specific density function.

Incorporating the effects of stochasticity into models may result in differing predictions concerning life-history evolution (e.g., delayed maturity: Tuljapurkar 1990*b;* iteroparity: Orzack and Tuljapurkar 1989; phenotypic plasticity: Orzack 1985; migration: Wiener and Tuljapurkar 1994). This is especially true of optimality or ESS investigations, when the relative benefits to different strategies do not differ much. In a population of scorpions studied in England, males may mature at either the sixth or seventh instar (Benton 1992). Seventh instar males are larger and have a higher reproductive success than sixth instar males, but they mature later than sixth instar males. Approximately half the males mature at each stage and, once mature, do not continue growing. Using data on the observed costs and benefits of maturing at different sizes, it is possible to predict when animals should mature, given their size at the fifth instar. From this is it possible to predict a ratio of sixth to seventh instar males. This ratio is very sensitive to environmental variability. If the environment is assumed to be constant (and r is used as a measure of fitness), the predicted ratio of sixth to seventh instar males is 1:3.2, which differs greatly from the observed 1:1. A CV of only about 0.125 (and fitness calculated using a) predicts a ratio of 1:1 (T. G. Benton and A. Grant, unpublished data).

However, for many organisms, especially long-lived ones living in relatively stable environments, there may be little appreciable effect of stochasticity when the sort of analyses detailed above are conducted, even if there are appreciable density-independent demographic changes caused by, for example, the weather (Benton et al., in press). Stearns (1992) may be correct in stating that the predictions of classical life-history theory are fairly robust, and he is undoubtedly correct in saying (p. 33) that "fitness definitions should be judged by how well they do a particular job and replaced if necessary." However, if deterministic theory highlights traits that appear maladaptive, it is highly likely that a factor such as environmental stochasticity is being ignored.

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