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"An Uncertain Life: Demography in Random Environments"

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An Uncertain Life: Demography in Random Environments

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This paper concisely reviews the demography of populations with random vital rates, highlights examples and techniques which yield insight into population dynamics, summarizes the state of significant applications of the theory, and points to open problems. The central picture in this theory is of a time-varying but statistically stationary equilibrium for population, sharply distinct from the notions of classical demography. The deepest biological insights from the theory reveal the temporal structure of life histories to be a rich arena for natural selection. © 1989 Academic Press, Inc.

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1. Introduction

The classical demography of Alfred Lotka has long been a powerful tool in population analysis but it ignores variation in population vital rates (i.e., in birth, death, growth, and similar rates). Many demographers have been interested in extending classical demography to deal with the problems of an uncertain life. One way to describe uncertainty is to treat vital rates as time-dependent random variables. When a population has such random rates, the important constructs of Lotka–Leslie theory (such as the ubiquitous r, the stable age distribution, and reproductive value) have no simple analogs. Instead growth rate, age distribution, and so on become time-dependent random variables. A theory is now available which shows how demography can be done for populations with random rates. This theory and especially its application are the subject of this paper.

The development of demographic theory for random rates represents a coalescence of two directions of study. One goes back as far as 1928 when Norton examined arbitrary temporal change in rates; this study was reborn as demographic ergodicity in the work of Coale (1957), Lopez (1961), and has developed further (Seneta, 1981). The other is a more recent interest in random rate theory, notable by Pollard (1968, 1973), Sykes (1969), Le Bras (1971), and Lee (1974). These directions were brought together for Markovian vital rates into a random ergodic theory of demography by Cohen (1977a). Cohen's work highlighted a stimulating connection with the theory of random matrix products, which has widespread application in science. In particular, populations with random rates follow a lognormal distributional law (Tuljapurkar and Orzack, 1980) derived for matrices (Furstenberg and Kesten, 1960) and transferred to demography. The theory of random demography has since developed on both the formal and applied fronts.

Why does one want a theory for demography under uncertainty? Because substantive questions in human demography, ecology, and evolution involve uncertainty in central ways. Here are some examples. Forecasts of human populations are routinely used for important public and private decision-making, and are routinely incorrect; the inclusion of statistical uncertainty in vital rates is now recognized as crucial in aiding decisions (see, e.g., Lee, 1974, 1977). The same issue arises in the management of populations (setting catch quotas in a fishery), in the design of conservation programs (how big should a refuge be?), and in risk assessment (can a population withstand certain kinds of environmental perturbation?). Ecologists studying population change often observe varying vital rates (Bierzychudek , 1982; Slade and Levenson, 1982; Van Sickle, personal communication, 1986) and wish to incorporate random rates in projection and inference. Evolutionary thinking on the forces which shape life histories has been strongly influenced by the putative effects of random variation (Murphy, 1968; Schaffer, 1974; Giesel, 1976) as a selective force on demographic parameters. These problems are all important and have all benefited (or will) from the use of random rate theory.

The objectives of this paper are to:

(i) concisely review the demographic theory of random rates;

(ii) highlight examples and techniques which yield insight into population dynamics;

(iii) summarize the state of significant applications of the theory;

(iv) point to open problems.

In reading this paper the reader should be aware that several new results are presented here (Sections 3.5, 3.8, 4.1, 4.2.2, 4.3.2, 4.4, 4.5.2, 4.5.4, 5.2, 6.2.3, 7.2), the presentation is biased toward work the author knows best and finds most interesting, and there is no effort to provide historical or complete coverage. The references listed should serve to fill the gaps. The organization of the paper should be clear from the table of contents listed at the start of the paper.

2. The Main Issues

2.1. *Classical Theory: Starting Point*

We begin with a quick reprise of classical demography so that the main aspects of the random theory can be easily set out. In classical discrete-time theory (Keyfitz, 1968; Pollard, 1973) the population at time t is described by a vector of numbers in successive age/stage classes; call this vector $\mathbf{n}_t$.

We will distinguish this population vector from the population structure $\mathbf{y}_t$; the latter is defined as a vector of proportions in successive ages/stages,

$$\begin{aligned} \mathbf{y}_t &= \mathbf{n}_t / P_t, \\ P_t &= \text{total population at } t. \end{aligned} \tag{2.1.1}$$

Although classical theory uses $\mathbf{n}$ and $\mathbf{y}$ almost interchangeably there are important differences in the random theory. The classical dynamics are governed by a fixed matrix $\mathbf{A}$ of vital rates; examples are a Leslie projection matrix, or a stage-structured projection matrix (Lefkovitch, 1965; Werner and Caswell, 1977). Assuming that $\mathbf{A}$ is nonnegative, primitive, and irreducible we are led to the classical results

$$\mathbf{y}_t \to \mathbf{u}, \tag{2.1.2}$$

$$\log(\mathbf{c}, \mathbf{n}_t)/t \to r_0, \tag{2.1.3}$$

$$|\mathbf{y}_t - \mathbf{u}| \sim \exp[-(r_0 - r_1)t] \exp(iw_1 t). \tag{2.1.4}$$

All these results apply in the limit as t increases to infinity. The first of these is *convergence to a stable age distribution*, where the vector $\mathbf{u}$ is the right eigenvector of $\mathbf{A}$ corresponding to the dominant eigenvalue $\exp(r_0)$. The second result says that the *long-run growth rate* of any part of the population is r_0. We have used the scalar product of vectors $\mathbf{a} = (a(i))$, $\mathbf{b} = (b(i))$ defined as

$$(\mathbf{a}, \mathbf{b}) = \sum_i a(i)\, b(i); \tag{2.1.5}$$

in (2.1.3) the vector $\mathbf{c}$ is any vector with bounded nonnegative components. Here and elsewhere in this paper we use natural logarithms. The third result, (2.1.4), says that *the age distribution converges to the stable one in damped oscillations*, with $\exp(r_1)\exp(iw_1)$ being the subdominant eigenvalue of $\mathbf{A}$ closest in magnitude to $\exp(r_0)$. Another result of demographic value says that the left eigenvector $\mathbf{v}$ of $\mathbf{A}$ corresponding to the dominant eigenvalue is a *vector of reproductive values*: the incremental value of one extra individual in a particular class i is proportional to $v(i)$.

About *notation*: boldface indicates a vector or matrix; to the extent possible, lowercase is used for deterministic nonrandom objects, and uppercase for random objects. Exceptions should be clear from the context; an obvious one is $\mathbf{A}$ for the nonrandom average matrix. Some exceptions, such as $\mathbf{A}$, conform to standard usage; in other cases there was a shortage of simple symbols.

2.2. *Random Rates: Models and Questions*

A formal generalization of classical demography is easily made. If we face an uncertain life, vital rates will change over time in potentially unpredictable ways. At time t, let the population vector be $\mathbf{N}_t$, the population structure vector be $\mathbf{Y}_t$. Over the interval t to $t+1$ demographic processes operate on these vectors, and their overall effect is contained in a time-dependent matrix of vital rates $\mathbf{X}_{t+1}$. The dynamics of population are given by the equation

$$\mathbf{N}_{t+1} = \mathbf{X}_{t+1}\,\mathbf{N}_t. \tag{2.2.1}$$

The matrix subscript is $(t+1)$ rather than t to emphasize that these rates apply *to* the vector $\mathbf{N}_t$. Thus in some cases (e.g., the IID model below) $\mathbf{N}_t$ and $\mathbf{X}_{t+1}$ are independent. In order to proceed, we must specify the kind of uncertainty that occurs in the vital rates. The models of most interest here are:

The IID Model. The entries of $\mathbf{X}$ are chosen randomly for each t from the same fixed (in general multivariate) distribution. There may be correlations between vital rates within each period, but there is no serial correlation between rates at different times. Here the environment is completely unpredictable. The number of possible environments can be finite (e.g., a "good" and a "bad" state), or infinite (e.g., if there is a continuously distributed variable like temperature).

The Markov Model. From one time interval to the next, vital rates change according to time-invariant transition probabilities. There are three subcases, according as the set of possible values of vital rates is *finite*, *countable* but infinite (e.g., discrete environmental states but infinitely many of them), or *uncountable* (usually continuously distributed) set. Here the environment is predictable to the extent that there is serial autocorrelation over time.

The ARMA Model. The elements of the vital rates follow a linear times series model of the ARMA type (Box and Jenkins, 1970). This model is most useful in situations where a time series of vital rate values is used to identify and fit a statistical model (cf. Lee, 1974). Ecologists often prefer ARMA models in situations where serial autocorrelation over several time intervals is expected to be important.

The Semi-Markov Model. The possible values (states) of vital rates are as in the Markov case, but the time taken to make a transition from any one state to another in governed by a probability distribution which depends in general on both initial and final states. Here the history of the environment plays a stronger role.

The Catastrophe Model. This is a completely unpredictable environment. One formulation is to suppose that in each time interval there is a very small probability of an event which will cause vital rates to reach extremely low levels; another formulation allows a probability distribution of times between successive catastrophic events, along with a distribution for the intensity of the catastrophic effect on vital rates. The biological view behind this model is of a population buffered against most small changes but vulnerable to large changes in environment.

Irrespective of the particular model used, we shall always assume that the random process generating the vital rates converges toward an ergodic stationary state. *In general, we assume that the random process is in the stationary state*; for the approach to stationarity, see Tuljapurkar and Orzack (1980).

The next question is, what conditions apply to the possible values of the vital rates. The rates here are assumed to be always nonnegative, and in addition we assume *demographic weak ergodicity* (alternatively we assume that the values lie in an ergodic set (Hajnal, 1976)). Ergodicity here means (Lopez, 1961; Keyfitz, 1968; Hajnal, 1976; Cohen, 1977a; Heyde and Cohen, 1985) that if we multiply together enough matrices chosen according to the rules for the $\mathbf{X}$'s in (2.2.1) the resulting product is a matrix with all elements positive. This in turn guarantees that the dynamics of (2.2.1) are *stable* in the following sense. Note that we can rewrite (2.2.1) as an equation for the age structure,

$$\mathbf{Y}_{t+1} = \mathbf{X}_{t+1}\,\mathbf{Y}_t/(\mathbf{e}, \mathbf{X}_{t+1}\,\mathbf{Y}_t) \tag{2.2.2}$$

where we use the scalar product and $\mathbf{e}$ is a vector of 1's. The difference between (2.2.1) and (2.2.2) is that the $\mathbf{Y}$'s, being vectors of proportions, are constrained so that $(\mathbf{e}, \mathbf{Y}) = 1$. Now in (2.2.2) pick two distinct initial structures $\mathbf{b}_0$, $\mathbf{c}_0$ and then apply the same random sequence of vital rates to both; call the resulting sequences of structure vectors $\mathbf{B}_t$, $\mathbf{C}_t$, respectively. Then our ergodicity condition implies that $\mathbf{B}_t$ approaches $\mathbf{C}_t$ as t increases. This is stability, but of a special sort, since the age structures are stable toward a time-varying limit; i.e., there is some sequence of structures $\hat{\mathbf{Y}}_t$, say, and both $\mathbf{B}_t$, $\mathbf{C}_t$ approach $\hat{\mathbf{Y}}_t$.

We now want to know: is there an analog to the stable age distribution? What is the asymptotic growth rate of population? What is the nature of convergence in the random model? Is there something like a reproductive value?

The answers to these questions are summarized in Section 2.3 and are explored and applied in the rest of the paper. First, however, we ask two basic questions: What features of (2.2.1) suggest that it will require a new theory? Why can we not apply insights derived from classical demography

and random but nonstructured models to get a handle on random demography? The answers to these questions lie in the facts that the dynamics of $\mathbf{N}$ are multiplicative and noncommutative, and in addition, the dynamics of $\mathbf{Y}$ are nonlinear. From (2.2.1) note that $\mathbf{N}$ is determined by a product of random (i.e., randomly chosen) matrices, and that these matrices do not in general commute (i.e., if we switch the order in which the matrices appear, the resulting product will change). It may be a good idea to convince oneself of this by, say, multiplying together two 2×2 Leslie matrices whose subdiagonal elements differ. From (2.2.2) note that the difference equation for $\mathbf{Y}$ is nonlinear and thus more messy than (2.2.1) for $\mathbf{N}$. In classical demography this difference is irrelevant, but in the random case the moments of $\mathbf{Y}$ bear a complicated relationship to those of $\mathbf{N}$ and so the linear (2.2.1) does not shed much direct light on the nonlinear (2.2.2).

2.3. *Key Results in Random Theory*

There are many alternative models for random rates, so we cannot expect a complete and universal theory. Instead we present results roughly in decreasing order of generality, alternating between stating mathematical results, and interpreting them demographically.

2.3.1. *General Results*

We begin with

ASSUMPTIONS 2.3.1. (i) *demographic weak ergodicity holds in Eq.* (2.2.1),

(ii) *the random process generating vital rates is stationary and ergodic,*

(iii) *the logarithmic moment of vital rates is bounded,*

$$E \log_+ \|\mathbf{X}_1\| < \infty \tag{2.3.1}$$

where E *indicates an expectation,* $\|\cdot\|$ *is any matrix norm, and* $\log_+(x) = \max\{0, \log x\}$.

Then we have (Furstenberg and Kesten, 1960; Oseledec, 1968; Cohen, 1977a; Raghunathan, 1979; Ruelle, 1979):

(A) the long-run growth rate of the logarithm of total population, or any part of population, is almost surely given by a number a independent of the initial population vector,

$$a = \lim_{t \to \infty} [\log(\mathbf{c}, \mathbf{N}_t)]/t \tag{2.3.2}$$

$$= \operatorname*{Lim}_{t\to\infty} (\log \|\mathbf{X}_t\mathbf{X}_{t-1}\cdots\mathbf{X}_1\|)/t \tag{2.3.3}$$

$$= \operatorname*{Lim}_{t\to\infty} \{E\log(\mathbf{c}, \mathbf{N}_t)\}/t \tag{2.3.4}$$

where $\mathbf{c}$ is any vector of bounded nonnegative numbers.

(B) starting from any initial structure $\mathbf{Y}_0$ the population converges to a (time-dependent) stationary random sequence of structure vectors $\hat{\mathbf{Y}}_t$. This limiting sequence is independent of $\mathbf{Y}_0$.

(C) there is a stationary measure which describes the probability distribution of the joint sequence of vital rates and population structure vectors $\{\mathbf{X}_1, \hat{\mathbf{Y}}_1, \mathbf{X}_2, \hat{\mathbf{Y}}_2, \ldots\}$.

(D) there are constants ρ_i for $i=1$ through $i=$(dimension of $\mathbf{N}$) such that

$$a = \rho_1 \geqslant \rho_2 \geqslant \cdots. \tag{2.3.5}$$

The ρ's are determined by the growth rates of exterior powers of the $\mathbf{X}$'s, and are called Liapunov characteristic exponents. For example, let $\|\mathbf{x}\wedge\mathbf{y}\|$ be the volume of the parallelepiped spanned by vectors, $\mathbf{x}$, $\mathbf{y}$. Choose two nonproportional initial population vectors, say $\mathbf{b}_0$, $\mathbf{c}_0$ and apply (2.2.1) to produce two sequences of random vectors $\mathbf{B}_t$ and $\mathbf{C}_t$. Then the almost sure growth rate of the volume spanned by any two vectors is at most

$$a+\rho_2 = \max_{\{\mathbf{b}_0,\mathbf{c}_0\}} \operatorname*{Lim}_{t\to\infty} \log\|\mathbf{B}_t\wedge\mathbf{C}_t\|/t. \tag{2.3.6}$$

Similar results hold for sums of more exponents. (A notational point: Cohen (1977a, b) writes $\log\lambda$ for the quantity a.)

We get an interesting general result if we add to assumptions 2.3 the

ASSUMPTION 2.3.2. *The random process generating vital rates can be run backwards in time, there being a unique time-reversed process which is stationary and ergodic.*

Then (Ruelle, 1979), we have:

(E) consider the adjoint (time-reversed) process associated with (2.2.2),

$$\mathbf{Z}_t = \mathbf{X}_t^{\mathrm{T}}\mathbf{Z}_{t+1}/(\mathbf{e}, \mathbf{X}_t^{\mathrm{T}}\mathbf{Z}_{t+1}) \tag{2.3.7}$$

where superscript T indicates a transpose. Suppose we fix a vector at time $t=t_1$, say $\mathbf{w}_0=\mathbf{z}_{t_1}$. Then (2.3.7) runs backwards through decreasing values of t, and we have that as $t\to-\infty$ the resulting vectors $\mathbf{Z}_t$ converge to a stationary random sequence of vectors $\hat{\mathbf{Z}}_t$, say, independent of $\mathbf{w}_0$.

2.3.2. *Interpretations*

The central feature is that a is identified as the almost sure growth rate of population. It is also the average growth rate of the population. As definitions (2.3.2)–(2.3.4) show, the value of **a** is a function of the random properties of the rates.

Property (B) is the random rates counterpart of stability of population structure. Although there is a random limit to which the structures converge, there is no information on the properties of the limit. Property (C) expresses the strong overall convergence of rates plus structures to a statistical stationary state. Property (D) identifies an exponential convergence rate for population structures. All of these properties take on substance as we become more explicit about the random process generating vital rates.

Property (E) identifies the stochastic analog of a reproductive value and helps to shed some light on the nature of reproductive value as a concept; more on this will follow.

2.3.3. *Mixing and Limit Theorems*

In addition to Assumptions 2.3.1, let us make

ASSUMPTION 2.3.3. *The random process generating vital rates is rapidly mixing.*

Technical aspects of the mixing condition are discussed by Furstenberg and Kesten (1960), Billingsley (1968), Tuljapurkar and Orzack (1980), and Heyde and Cohen (1985). Given such mixing, we have:

(F) there is some σ such that the asymptotic distribution of total population is lognormal,

$$\log\{(P_t - at)/\sigma\sqrt{t}\} \to N(0, 1). \tag{2.3.8}$$

The interpretation of (2.3.8) is well known in biology (Lewontin and Cohen, 1969; Tuljapurkar and Orzack, 1980). The significance of a as a descriptor of population growth is highlighted by the skewness of the long-run distribution of population. The quantity σ in (2.3.8) determines the asymptotic variance of the logarithm of population size. A limit theorem relevant to estimating σ by Heyde and Cohen (1985) will be discussed in Section 7.1.

2.3.4. *Markovian rates*

The key feature here is that statistical stationarity can be captured in a probability distribution function. Make

ASSUMPTION 2.3.4. *The vital rates follow a countable-state Markov process.*

Assumptions 2.3.1 and 2.3.3 are still in force. Then (Cohen, 1977a, b; Lange, 1979)

(G) there is a joint probability distribution of vital rates and population structures; call it $H(t, A, B) = \Pr\{\mathbf{X}_t \text{ lies in set } A,\ \mathbf{Y}_t \text{ lies in set } B\}$. Then as $t \to \infty$ this distribution converges to an equilibrium distribution, say $H^*(A, B)$, which reproduces itself under the action of (2.2.2).

(H) the average growth rate a can be computed as the average one-time-step growth rate. Note from (2.2.1)–(2.2.2) that

$$P_{t+1}/P_t = (\mathbf{e}, \mathbf{X}_{t+1}\mathbf{Y}_t), \tag{2.3.9}$$

so that one has

$$a = E \log(\mathbf{e}, \mathbf{X}_1 \mathbf{Y}_0) \tag{2.3.10}$$

with the average taken with respect to the stationary distribution H^*.

An equation for H^* can be written with some notational effort. Still more is known if we add

ASSUMPTION 2.3.5. *The vital rates follow a finite state Markov process.*

Then (Bharucha, 1961; Kushner, 1966; Pollard, 1968; Cohen 1977b; Tuljapurkar, 1982a) one has:

(I) the moments of the population vector and its tensor powers, $E\mathbf{N}_t$, $E\mathbf{N}_t \otimes \mathbf{N}_t$, $E\mathbf{N}_t \otimes \mathbf{N}_t \otimes \mathbf{N}_t$, ..., can be computed explicitly as functions of time. Asymptotically these moments change geometrically with rates computable as dominant eigenvalues of explicitly known nonnegative matrices.

The final simplification is

ASSUMPTION 2.3.6. *The random vital rates are determined by the IID model* (see Section 2.2).

Then we have:

(J) there is a probability distribution for the population structure vector, say $G(t, B) = \Pr\{\mathbf{Y} \text{ is in set } B\}$; and a corresponding stationary distribution $G^*(B)$ to which $G(t, B)$ converges as t increases.

Examples of G^* and H^* are given by Cohen (1977b), in Section 3, and by Tuljapurkar (1984).

2.3.5. *Simulations*

Useful insight into the consequences of random rates has come from numerical simulations, e.g., Boyce (1977), Cohen (1977b), Tuljapurkar and Orzack (1980), Slade and Levenson (1982). Pollard (1973) describes a way of simulating the more complex case of populations with "demographic" stochasticity added in. There is a large and relevant literature on simulation methodology; the book by Ripley (1987) is a concise introduction. It is fair to say that simulations are most effective when informed by theoretical reasoning, and when their potential limitations are kept carefully in view. No details are given here of any simulations; the reader may refer to the cited papers as well as others mentioned later.

2.3.6. *Assessing the results*

We have presented above the skeleton of a fairly general theory for random rates. However, these results bear a distant relationship to the substantive problems listed in the introduction, in the same way that the Perron–Frobenius theorem is not classical demography. In addition to the obstacles posed by the evident complexity of random rates theory, some theoretical issues remain unresolved. The nature of oscillatory transients in the random theory remains unclear, although Lee (1974) has discussed some of their properties and their significance in population dynamics. The significance of the reproductive value has not been explored. There is limited information about the functional dependence of objects like a and the ρ's on the properties of the underlying vital rates.

This paper now proceeds to the nuts-and-bolts of random rates in demography by studying stylized and practical examples, approximate and exact analytical results, and evolutionary models. In the process we will resolve aspects of the relation between the general theory and applications, develop insight into the consequences of random vital rates, and partially fill some gaps in the theory. We end this section with examples of populations for which this theory is ideally suited.

2.4. *Examples*

The bare bones of Section 2.3 need fleshing out. Here are examples of structured populations usefully modeled by random matrix equations of type (2.2.1). The matrices have a wide variety of structures but are covered by the general assumptions of Section 2.3.

2.4.1. *Age Structure: Humans, Fish, Other Mammals*

Human populations inspired much of demographic theory. The typical age-structured model uses Leslie matrices of the form

$$\begin{pmatrix} f_1 & f_2 & \cdots & \cdot & f_k \\ p_1 & 0 & \cdots & \cdot & 0 \\ \cdot & \cdot & \cdots & \cdot & \cdot \\ 0 & p_2 & \cdots & \cdot & 0 \\ 0 & \cdot & \cdots & p_{k-1} & 0 \end{pmatrix}.$$

Here the f's are fertilities (female offspring per female per time interval) for successive age classes and p_i is the survival rate of individuals aged i in one time interval.

EXAMPLE 1. Human population matrices typically use 5-year age groups with $k = 10$. Usually $f_1 = f_2 = 0$ with peak fertility in age classes 4–6. In modern human populations p_1 reflects the relatively high infant mortality and p_i for $i > 2$ are very close to 1. Mortality rates are stable in the short run with secular increase in p's at high ages. Random fluctuations mainly affect fertilities; Lee (1974) used time series models for the fertilities.

EXAMPLE 2. Long-lived egg-laying fish such as the striped bass and the herring are characterized by year-classes and k of 5 (herring)–20 (bass). Age of first reproduction is at 2 or 3, with fertility increasing with age and then becoming constant late in life. Survival rates are roughly constant, except for the survival rate of eggs to the fingerling stage. Large random fluctuations are common in the egg survival rate.

EXAMPLE 3. Small mammals such as voles (Boyce, 1977, 1979) can be described using year classes with $k = 3$ or 4. Here both fertilities and survival rates can show considerable year-to-year variation in response to environmental conditions. We consider a large mammal, the elk, in Section 8.

2.4.2. *Sizes and Stages: Herbs, Seed Pools, Shrubs*

A variety of populations are best described using size classes or stages instead of age classes.

EXAMPLE 1. The herb jack-in-the-pulpit (*Arisaema triphyllum*) reproduces clonally as well as sexually. Bierzychudek (1982) presents a size and stage-class model in which the time unit is 1 year. Class 1 is seeds, classes 2 through 7 are size classes of plants grouped by leaf area (e.g., class

2 has $\leqslant 50\text{cm}^2$ leaf area, class 3 has $51\text{–}130\text{cm}^2$, and so on). Seeds are only produced sexually, while clonal reproduction leads to direct entry into class 2. The population growth matrix has the form

$$\begin{pmatrix} m_1 & m_2 & \cdots & m_k \\ f_1 & f_2 & \cdots & f_k \\ 0 & m_{32} & \cdots & m_{3k} \\ \cdot & \cdot & \cdots & \cdot \\ 0 & m_{k2} & \cdots & m_{kk} \end{pmatrix}.$$

Here m_i are sexual fertilities (seeds produced per plant); f_i are the sum of clonal fertilities, seed survival (for f_1), and transitions from larger sizes to size class 2; the other m_{jk} are transition rates from class k to class j. Transition rates vary randomly year-to-year.

EXAMPLE 2. Plants with seed pools are of great interest especially in the study of weeds (see e.g., Grime, 1979). Templeton and Levin (1979) studied the evolutionary dynamics of annual plants which bank seeds, suggesting that seed pools are adapted to variable environmental conditions. Charlesworth (1980) formulated a general age-structured model for a plant which lives several years above ground and has a seed pool in which seeds can survive for several years while retaining some probability of germination. The resulting model involves two age-classes vectors, one for seeds and one for germinated plants. The combined vector is acted upon by a growth matrix of the form

$$\left(\begin{array}{ccccc|ccccc} 0 & 0 & \cdots & \cdot & 0 & f_1 & f_2 & \cdots & f_k \\ p_1 & 0 & \cdots & \cdot & 0 & 0 & 0 & \cdots & 0 \\ 0 & p_2 & \cdots & \cdot & 0 & 0 & 0 & \cdots & 0 \\ \cdot & \cdot & \cdots & \cdot & \cdot & \cdot & \cdot & \cdots & \cdot \\ 0 & 0 & \cdots & p_{k-1} & 0 & 0 & 0 & \cdots & 0 \\ \hline g_1 & g_2 & \cdots & \cdot & g_k & & & & \\ & & \mathbf{0} & & & & \mathbf{M} & & \\ & & \leftarrow k \text{ col} \rightarrow & & & & \leftarrow K \text{ col} \rightarrow & & \end{array}\right).$$

Here there are k age classes for seeds, K age classes for plants above ground. The p's are probabilities of survival plus nongermination for seeds, the g's are probabilities of germination, the f's are seed production rates, $\mathbf{0}$ is a block of zeros, and $\mathbf{M}$ contains rates for all transitions between

plants of various age classes, except for reproduction. Vegetative reproduction if it occurs will be described by elements of **M**. Important sources of temporal variability are in seed production rates which can vary widely between seasons, and in survival and germination rates.

EXAMPLE 3. Shrubs which form thickets are an interesting example of modular reproduction (Harper, 1977). Some shrubs are clonal, forming dense thickets in which seed recruitment plays very little part. Huenneke (1987) has done an interesting demographic study of stems and clumps of the speckled alder *Alnus incana* ssp. *rugosa*. She was able to describe the growth of clumps by measuring stem basal diameters within clumps, and then describing clumps by the distribution of stems into 7-stem-diameter classes. Transitions between classes occur due to stem growth or lack of growth, stem death, and clonal growth of new stems. Huenneke observed substantial spatial and temporal variability in growth rates. This is an interesting use of demography to describe modular structure and was expanded on in Huenneke and Marks (1987).

2.4.3. *Dispersal*

Spatial heterogeneity and related population movement have been important themes in population ecology (Gadgil, 1971; Roff, 1975; den Boer, 1981) and genetics (Karlin, 1982). The combined effects of dispersal and temporal variability are thought to be important factors in maintaining population persistence, spatial patchiness, and genetic polymorphisms. In all these situations the population is divided into spatially distinct groups, and there is a sequence of reproduction plus survival within groups followed by dispersal of individuals between groups. The actual order of events and the mating structure of the population matters and can be analyzed as shown by Karlin (1982); here we consider only the simplest case. Thus, let $\mathbf{N}_i$ be the population vector (by age or stage) in spatial group i, and suppose that growth in patch i follows the random rate matrix $\mathbf{X}_{it}$ followed by migration in which a fraction $p_{ij}(a)$ of the population class a at site j moves to site i. Then the dynamics are given by

$$\mathbf{N}_i'(a) = \sum_{j,a} p_{ij}(a)(\mathbf{X}_{jt}\mathbf{N}_j)(a)$$

where $\mathbf{N}_i'$ is the new vector for group i.

It would be of great interest to study this model using random rates theory. Even the simplest case ignoring age structure in the groups is still untreated analytically, and many other biologically interesting cases are worth studying. Cohen (1982) has worked on a closely related problem of

multiregional human population dynamics, although his results are fairly abstract and extend the ones in Section 2.3. Another related problem is of populations structured by birth type (Tuljapurkar and Carey, 1986): an example is a population of Mediterranean fruit fly (*Ceratitits capitata*) which has two available hosts. Individuals laid as eggs in one host have vital rates determined mainly by that host type, but will deposit offspring in both kinds of hosts. This leads to a variant of the dispersal problem, for which detailed results have been obtained with a constant environment; the case of a random environment remains open.

3. Dynamics with Random Rates

It will be instructive and entertaining to begin the study of dynamics with a simple but rich example. This section foreshadows the more general results of the next section.

3.1. *Random Fertility: A Simple Model*

Consider a population with two age classes, labeled young and old, respectively. Let the fertilities of the two ages be random over time so the population model can be written as

$$\mathbf{N}_{t+1} = \mathbf{X}_{t+1}\mathbf{N}_t \tag{3.1}$$

with

$$\mathbf{N}_t = \begin{pmatrix} N_t(1) \\ N_t(2) \end{pmatrix}, \qquad \mathbf{X}_t = \begin{pmatrix} m_1 F_t & m_2 F_t \\ P & 0 \end{pmatrix}. \tag{3.2}$$

Here the m's are positive, $0 < p < 1$, and $\{F_t,\ t = 0, 1, \ldots\}$ is a sequence of independent random variables identically distributed with $(1/F_t)$ having a gamma distribution with probability density function

$$g(w) = (n^n/n-1!)\, w^{n-1} \exp(-nw). \tag{3.3}$$

The parameter n here measures the variance: as $n \to \infty$, F_t approaches the nonrandom value 1. As $n \to 0$ the variance of F_t increases (see (3.6) and (3.10)). To be absolutely clear about the dynamics here, consider how one would simulate (3.1) on a computer. Start with some initial population vector, generate a (pseudo-) random number distributed according to density g, take the reciprocal of this and produce a Leslie matrix as in (3.2), and then use (3.1) to generate a new population vector. Then generate a new random number to get the next fertility and repeat.

According to property (J) of Section 2.3.4 there is a stationary distribution for the population structure. Here the structure is just one number, say the proportion of young; we find it convenient to use instead the random variable

$$R = m_2 N(2)/m_1 N(1). \tag{3.4}$$

Defining $z = (m_1^2/m_2 p)$, the probability density of R may be found as in Tuljapurkar (1984) to be

$$C(x) = (1/K)\, x^{n-1}(1+x)^{-n} \exp(-nzx) \tag{3.5}$$

where K is determined by requiring that the area under the curve of C be unity. The density C is the key to an analysis of the effect of random fertilities on population structure.

3.2. *Average Vital Rates*

Suppose we FIX the vital rates in (3.2) at their AVERAGE values. Noting that

$$\langle F \rangle = E(F) = \int_0^\infty dw\; g(w)\,(1/w) = n/(n-1), \tag{3.6}$$

we can analyze the case of fixed average rates using standard classical theory. In particular, the quantity R in (3.4) converges to a stable value x^* given as the positive root of

$$z\langle F \rangle x^2 + z\langle F \rangle x - 1 = 0, \tag{3.7}$$

which is simply a transformed version of the usual characteristic equation. The long-run growth rate is

$$r_0 = \log(\lambda_0) = \log(m_2 p/m_1 x^*) \tag{3.8}$$

and the convergence rate is determined by

$$r_1 = \log(m_2 \langle F \rangle p) - r_0. \tag{3.9}$$

3.3. *Statistical Steady State Structure*

The first step in exploring the effects of random fertility is to see how the age structure is affected. In the steady state the age structure over time is a sequence of values of the random variable R of Eq. (3.4). According to the general theory, such values have a time-independent probability

distribution, given explicitly here by the function C of Eq. (3.5). Figure 1 shows the density C for illustrative parameter values for $n = 10$, 100, 200. The shape of C shows how the random variation in F is nonlinearly filtered into variability in population structure.

Observe that C is peaked at the most probable value of R, call it $x(n)$, and that $|x(n) - x^*|$ decreases with increasing n. Note from (3.6) that $\langle F \rangle$ approaches 1 as n increases, and that the variance of fertility is

$$\mathrm{Var}(F) = \mathrm{Variance}(F) = \{(n-2)(1-1/n)^2\}^{-1} \sim (1/n). \qquad (3.10)$$

So for large n, the random rates model should approach the model with fixed average rates in Section 3.1. Figure 2 shows the behavior of central moments $E(R)$ and $\mathrm{Var}(R)$; as n increases, these converge to x^* and 0, respectively, as expected. For the curious, the use of $E(R)$ in (3.8) does not accurately predict the stochastic growth rate a.

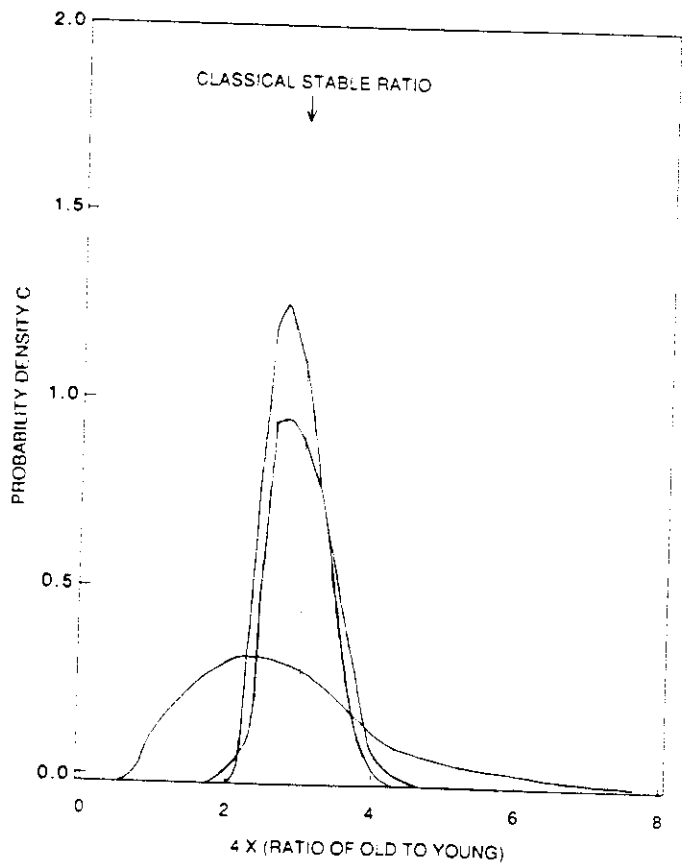

FIG. 1. Stationary probability density of scaled age structure as given by (3.5). Here $m_1 = 0.25$, $m_2 = 1$, $p = 0.75$, and the factor of 4 on the horizontal axis comes from (3.4). We show the classical stable age structure with vital rates fixed at m_1, m_2, p. The most spread-out density is for $n = 10$, the next for $n = 100$, and the most peaked for $n = 200$.

3.4. *Growth Rate a*

The average growth rate a is the most interesting single quantity in the random rates model. From the definition,

$$\begin{aligned} a &= E\log(P_{t+1}/P_t), \qquad \text{calculated in the steady state,} \\ &= E\log(pm_2/m_1R_{t+1}) + E\log(m_2 + m_1R_{t+1}) - E\log(m_2 + m_1R_t) \\ &= E\log(pm_2/m_1) - E\log R_t, \end{aligned} \tag{3.11}$$

where we use stationarity in the last step. This equation is a special case of a new, general result (Section 4.5.4) for age-structured demography.

For illustrative parameter values, Fig. 3 (solid curve) shows exact values of a computed using (3.11) and (3.5) as n increases. As expected, these values converge toward r_0 in the limit of large n. Notice that $|a - r_0|$ changes linearly in $(1/n)$ for large n; recall that $(1/n)$ measures the variance

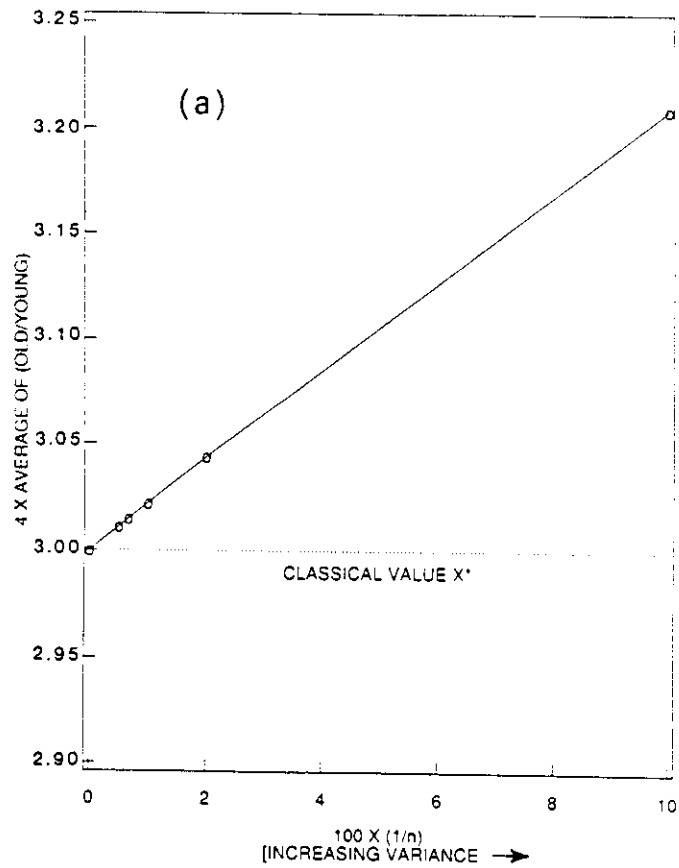

FIG. 2a. Mean of the scaled age structure (3.4). The horizontal axis shows $(1/n)$; on this axis, the zero is the limit of no randomness. Parameter values as for Fig. 1. The classical value is shown.

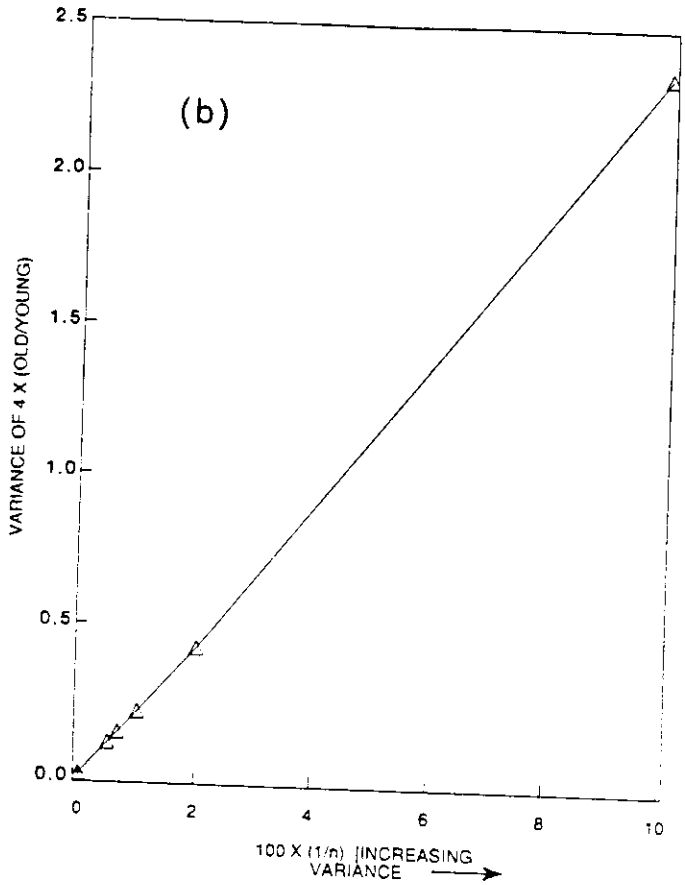

FIG. 2b. Variance of the scaled age structure (3.4) as environmental variance increases.

of the fertilities. This linear dependence is accurately predicted by a general approximation technique discussed later.

3.5. *Convergence Rates: Liapunov Exponent*

The discussion of Liapunov exponents given in Section 2.3.1 was rather terse. So we first describe the geometrical meaning of these exponents, and then calculate them for the random fertility model. The present two-age class model has only two such exponents, the larger being a and the other being ρ_2.

Consider two n-dimensional population vectors $\mathbf{b}$, $\mathbf{c}$ which span the shaded area in Fig. 4. The size of this area is written $\|\mathbf{b} \wedge \mathbf{c}\|$ as in Eq. (2.3.6). The definition of this symbol generalizes to volumes spanned by more than two vectors. To define the vector $\mathbf{b} \wedge \mathbf{c}$ requires the Kronecker (tensor) product of vectors:

$$\mathbf{b} \otimes \mathbf{c} = (b(1)\mathbf{c}, b(2)\mathbf{c}, \ldots). \tag{3.12}$$

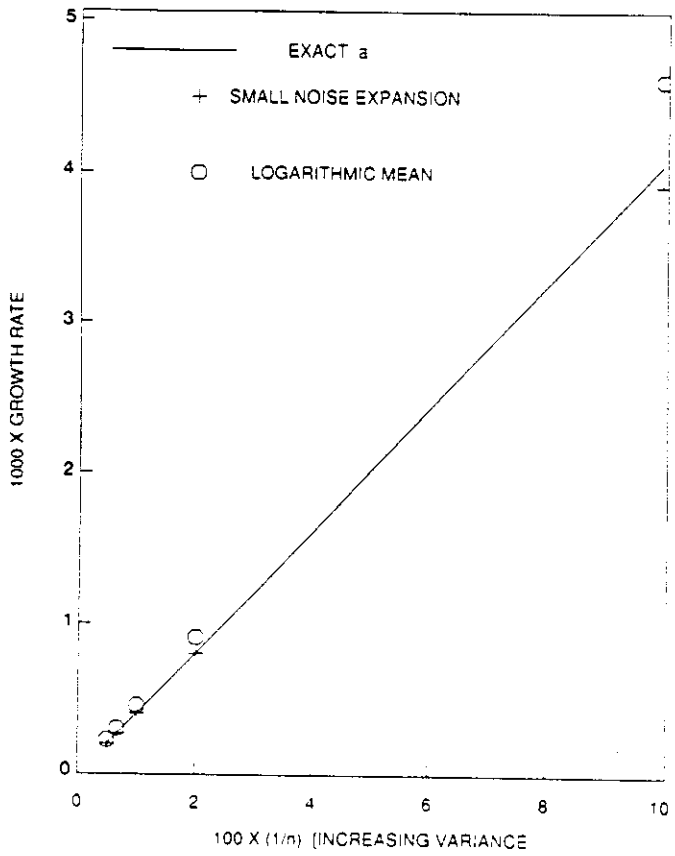

FIG. 3. Exact long-run growth rate a (solid line) plotted against $(1/n)$, computed from (3.11) with $m_1 = 0.2$, $m_2 = 1$, $p = 0.8$. Also plotted are the small noise approximation (3.14) and the logarithmic mean of eigenvalues LM from (3.15).

in terms of which

$$\mathbf{b} \wedge \mathbf{c} = \mathbf{b} \otimes \mathbf{c} - \mathbf{c} \otimes \mathbf{b}. \tag{3.13}$$

Notice that the object in (3.13) has $n(n-1)$ nonzero components, half of which differ only in sign from the other half. The usual definition therefore says that $\mathbf{b} \wedge \mathbf{c}$ has $\{n(n-1)/2\}$ components. A little algebra now shows that

$$\|\mathbf{b} \wedge \mathbf{c}\| = (\mathbf{b} \wedge \mathbf{c}, \mathbf{b} \wedge \mathbf{c})^{1/2}$$

equals the area in Fig. 4 (with a factor of $\sqrt{2}$ if we include all $n(n-1)$ components).

Now consider convergence of population structure. Suppose we start with the vectors $\mathbf{b}$, $\mathbf{c}$ as initial populations in (3.1) and apply the SAME random sequence of vital rates to both. The resulting population vectors

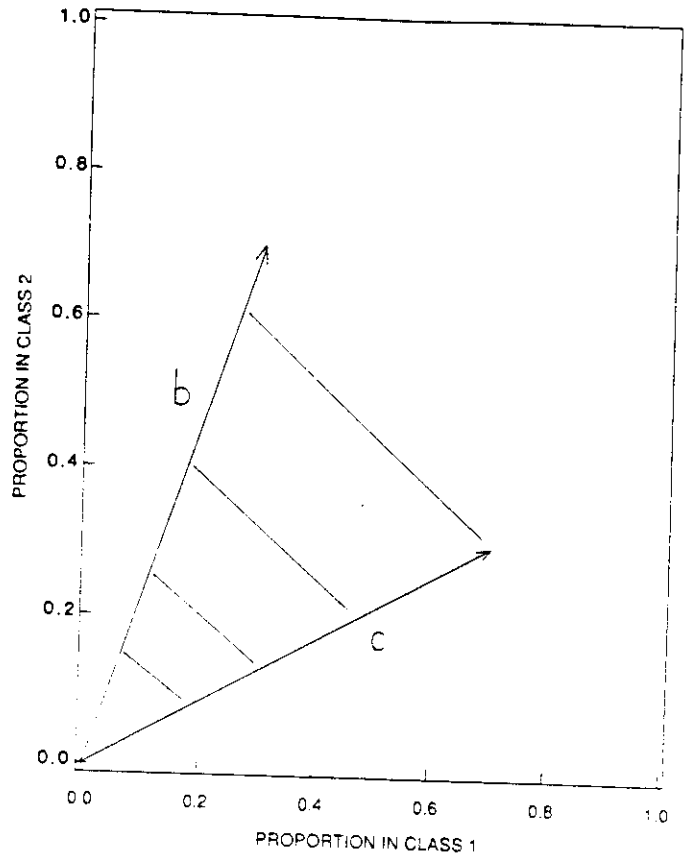

FIG. 4. Two initial vectors **b**, **c** illustrated for a 2-class population. The components of the vectors add to 1 and the shaded region is the area given by the wedge product in (3.13). Under the action of population projection matrices, the angle between vectors decreases asymptotically to zero.

are a sequence, say $\mathbf{B}_t$, $\mathbf{C}_t$. From our assumptions, we have convergence of population structure, i.e., the vectors $\mathbf{B}_t$, $\mathbf{C}_t$ become proportional as t increases. Geometrically, convergence means that if we start with vectors as in Fig. 4, then as t increases, the angle between successive pairs of vectors $\mathbf{B}_t$, $\mathbf{C}_t$ will go to zero since the two vectors ultimately coincide in direction. The asymptotic rate of change of the area between vectors can be heuristically analyzed as follows: suppose the angle between the vectors is θ_t, and consider large t when $\sin(\theta_t) \sim \theta_t$. Then the area spanned by the vectors is $\|\mathbf{B}_t\| \, \|\mathbf{C}_t\| \, \theta_t$. Both vector norms here change at a rate close to $\exp(at)$, so the area overall changes at rate about $\exp(2a+d)t$ where $\exp(dt)$ is the rate of change of θ_t. From the limit in (2.3.6) we now have that d is $(\rho_2 - a) \leqslant 0$. Thus ρ_2 tells us how fast the angle between initially distinct population vectors will shrink.

In the present case the population vector has $n = 2$ components and the

second Liapunov exponent can be found from an identity of Oseledec's (discussed in a demographic context by Tuljapurkar (1986)),

$$\rho_2 = E \log |\text{determinant}(\mathbf{X}_t)| - a,$$
$$= E \log(m_2 F_t p) - a. \tag{3.14}$$

Figure 5 plots values of this second exponent as n changes and shows that $\rho_2 \to r_1$ as n increases. The behavior of ρ_2 can also be accurately described for large n by the analytical approximations discussed later.

In Section 4.3 we show that the Liapunov exponents in the classical theory reduce to the usual convergence rates based on eigenvalues of the vital rate matrix. The meaning of higher order exponents is also described there.

3.6. *Convergence Rates: Distribution of Age Structure*

A different view of convergence in the random model is to ask: if we start with a probability distribution of age structure other than the equilibrium

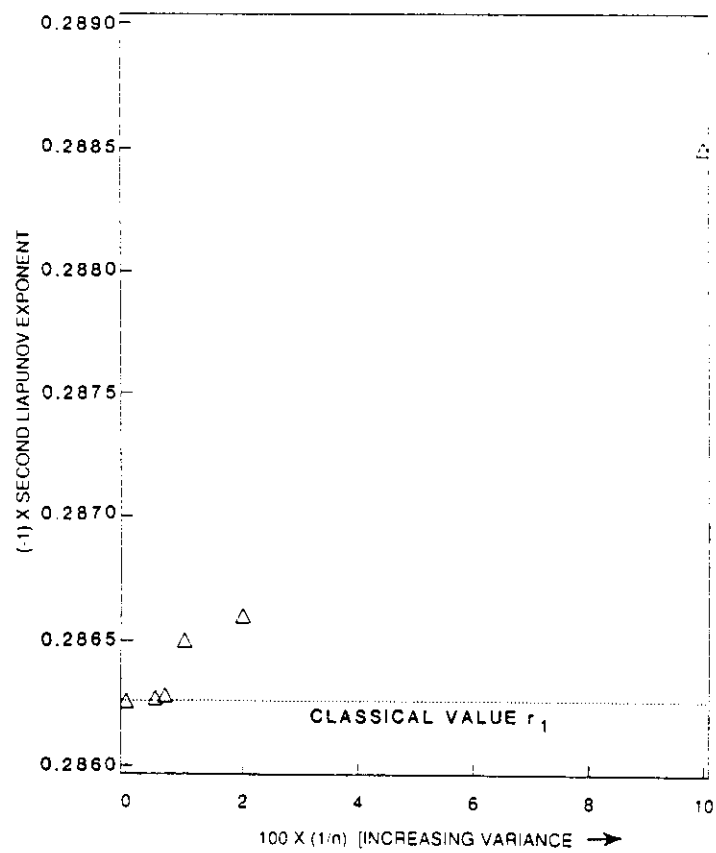

FIG. 5. The second Liapunov exponent giving a convergence rate in the presence of random variation, computed using (3.14) and parameter values as in Fig. 1. Also shown is the classical (deterministic) convergence rate.

distribution C of (3.5), how fast does it converge toward the equilibrium distribution? We answer this question as follows: suppose that $d(t, x)$ $(\neq C)$ is the density of the age-structure variable R. Then Eq. (3.1)–(3.3) lead (Tuljapurkar, 1984) to the transformation

$$d(t+1, x) = \int_0^\infty dy \{ g[zx(1+y)]\, z(1+y) \}\, d(t, y),$$
$$d(t+1, .) = L_g\, d(t, .), \tag{3.15}$$

where the second equation is a linear operator form of the first. The equilibrium density C solves (3.15) identically. Convergence of any $d(t, .)$ to C is governed by the eigenvalues of the linear operator L_g in (3.15). There appears to be little information on these eigenvalues, though they can be numerically approximated. If known these eigenvalues would provide information on oscillatory transient in the approach to statistical stationarity.

3.7. *Approximations for Small Noise*

"Noise" means the amplitude of randomness, measured here by $(1/n)$. There are two different ways of analyzing the dynamics of random rates when the randomness is small.

In the present example, we have seen that the exact density of the age structure is very concentrated near x^* as n increases. This suggests asymptotic expansion in n as a way of computing averages such as a. However, this is tedious and, more importantly, limited since it requires the complete distribution of population structure.

A second approach, preferred here, is to use the approximations developed in Tuljapurkar (1982b), which are based on Ruelle (1979) and on methods exemplified by van Kampen (1981). We defer the general result to later, and write down the approximation to the growth rate in the random fertility model:

$$a \simeq r_0 - (\mathrm{Var}(F)/2\lambda_0^2 T_0^2)[m_1 + (m_2 p/\lambda_0)]^2. \tag{3.16}$$

with $T_0 = 2 - (m_1 \langle F \rangle / \lambda_0)$. Figure 3 compares values of the exact growth rate calculated as in Section 3.4 above with values of the small noise approximation. Clearly (3.16) is a good approximation.

We show later that the approach used to get (3.16) can be extended to compute moments of age structure, transients in the age structure, and other interesting quantities.

3.8. *An "Approximation" Which does not Work*

In Eq. (3.1) if we take X to be a number rather than a matrix, and N to be a number rather than a vector, then the long-run growth rate of N is simply $E \log X$. This fact has led to the suggestion (Schaffer, 1974; Boyce, 1977) that we may approximate a for the matrix case as follows. First, for every random matrix $\mathbf{X}$ find the corresponding dominant eigenvalue $\lambda(\mathbf{X})$. Then use the probability distribution of $\mathbf{X}$ to compute

$$\begin{aligned} \mathrm{LM} &= E \log \lambda(\mathbf{X}), \\ &= \int_0^\infty dw\, g(w) \log r(w), \end{aligned} \tag{3.17}$$

with $r(w) = (m_1/2w)[1 + \{1 + (4w/z)\}^{1/2}]$, with $z = (m_1^2/m_2 p)$ as before. Here we used (3.1)–(3.2) to compute $r(w)$ in (3.17). The notation LM indicates that the object is a logarithmic mean of the dominant eigenvalues of the X's. The geometric mean is $\exp(\mathrm{LM})$.

We now contrast LM in (3.17) with the exact a. The latter must be computed as an average over *both* rates and age structure, see (2.3.10). From (3.1)–(3.2), and (3.4), if $R_t = x$ and $F_t = (1/w)$ the one-period growth rate is

$$\lambda_1(w, x) = m_2[m_1(1 + x) + wp]/[w(m_2 + m_1 x)].$$

Therefore

$$a = \int_0^\infty dw\, g(w) \int_0^\infty dx\, C(x) \log \lambda_1(w, x). \tag{3.18}$$

It is apparent that the single average in (3.17) and the double average in (3.18) are unlikely to agree save in fortuitous circumstances. This is illustrated in Fig. 3 which shows the exact a, the values of LM, and the small noise approximation (3.16). We echo Cohen (1977b, 1979) in saying that LM is NOT a useful approximation to a. See also Sections 4.2.1 and 4.5.2.

3.9. *Population Size and Extinction*

We have not yet considered the population's numbers. The average population vector, $E\mathbf{N}$, changes according to the fixed average vital rates of Section 3.2. The second moments of the population vector can be obtained from the recursion

$$(\mathbf{N}_{t+1} \otimes \mathbf{N}_{t+1}) = (\mathbf{X}_{t+1} \otimes \mathbf{X}_{t+1})(\mathbf{N}_t \otimes \mathbf{N}_t) \tag{3.19}$$

which shows that $E(\mathbf{X}_t \otimes \mathbf{X}_t)$, which is by assumption a primitive nonnegative matrix, determines the dynamics of $E(\mathbf{N}_t \otimes \mathbf{N}_t)$. Further details may be found in Tuljapurkar (1982a).

The other central feature is the lognormality of population size leading to a skew distribution of total population number P at long times. As discussed by Tuljapurkar and Orzack (1980) this skew distribution partly determines the probability of a population reaching an "extinction threshold." That paper discusses the use of a random walk process with drift a and diffusion σ to get estimates of the probability of reaching a particular threshold. We expand on the extinction issue in Section 8.3.

4. General Methods and Results

The example of Section 3 illustrates the effects of random rates in a stylized setting. This section returns to the general problem and shows what can be done. The more abstract results are given very brief treatment.

4.1. *Limits on Population Structure*

An interesting question about random rates is: how much variation can they produce in population structure? By structure we mean the vector of proportions $\mathbf{Y}$. One way of formalizing the question is: suppose the vital rates are bounded, so that for each i, j on has either

$$(\mathbf{X}_t)_{ij} = 0 \qquad \text{for all} \quad t, \tag{4.1.1}$$

or

$$0 < (\mathbf{L})_{ij} \leqslant (\mathbf{X}_t)_{ij} \leqslant (\mathbf{U})_{ij} < \infty \qquad \text{for all} \quad t. \tag{4.1.2}$$

Then are there bounds such that in the statistical steady state for each t, i

$$b(i) \leqslant Y_t(i) \leqslant c(i)? \tag{4.1.3}$$

General results on this question are given by Seneta (1984) who provides references to earlier work. His main result is that under the ergodicity conditions used here, there is a nonempty limiting set of population structures which is approached geometrically fast. However, this result does not describe the limiting set.

Explicit bounds on steady state structure can be obtained for age-structured populations whose Leslie matrices obey (4.1.1) except in the first

row and subdiagonal. Specifically we can find an explicit equation for the vectors $\mathbf{c}$ and $\mathbf{b}$ in (4.1.3). Details are in the Appendix.

4.2. *Small Randomness Approximations*

Nonlinear random problems are notorious for their difficulty in ecology, genetics, physics, and chemistry. Simulation is a useful and increasingly affordable approach to such problems, but there is great need for analytically based results. Random demography has available a combination of some exactly solvable cases and approximate results which reveal a great deal of the story of how randomness affects populations. The approximation methods we now present take the magnitude of external random effects to be small. This strategy lies behind the use of diffusion equations in genetics and ecology, and stochastic expansion methods in physics and chemistry (see, e.g., Van Kampen, 1981). The basic method was developed in Tuljapurkar (1982b) and has been applied to the evolution of homeostasis by Orzack (1985), to stochastic forecasting by Tuljapurkar (1987) and Tuljapurkar and Lee (1987), to life history evolution by Orzack and Tuljapurkar (1989), to projection by Wallace (1986), and to the problem of population conservation by Lande (1987). We first analyze growth rates and then age structure. Throughout this subsection we consider general nonnegative matrices, except when specifically stated otherwise.

The small randomness strategy requires splitting the random matrix $\mathbf{X}_t$ into an average matrix and a deviation: with

$$\mathbf{A} = E(\mathbf{X}),$$

and

$$\mathbf{B} = \mathbf{X}_t - \mathbf{A}$$

one writes

$$\mathbf{N}_{t+1} = (\mathbf{A} + \mathbf{B}_{t+1})\mathbf{N}_t. \tag{4.2.1}$$

The average vital rate matrix $\mathbf{A}$ is taken to have dominant eigenvalue $\lambda_0 = \exp(r_0)$, with corresponding right, left eigenvectors $\mathbf{u}$, $\mathbf{v}$. Assumptions 2.3.1 are taken to hold; additionally we assume that the stationary moments of the random $\mathbf{B}$'s are known:

$$E(\mathbf{B}_t) = 0,$$
$$E(\mathbf{B}_{t+m} \otimes \mathbf{B}_t) = \mathbf{S}_m, \qquad \text{for integer } m. \tag{4.2.2}$$

A central assumption is that the $\mathbf{B}$'s are "small" deviations, and a formal

version of this assumption is that all moments of $\mathbf{B}$ are bounded, and that the average absolute deviation in any element of $\mathbf{X}$ from its mean value is small compared to that mean. A convenient way of keeping track of the magnitude is to introduce a positive parameter ε which multiplies the $\mathbf{B}$ in (4.2.1) and then track powers of ε so the results are useful when ε is near zero. We do not do this here.

4.2.1. *Growth Rate a*

The goal is to compute

$$a = \mathrm{Lim}(1/t) E \log P_t \qquad \text{as} \quad t \to \infty.$$

In view of demographic weak ergodicity we will get the same answer irrespective of the initial population vector so choose $\mathbf{N}_0 = \mathbf{u}$. Since we will take the limit of large t, we may compute the average growth rate as

$$a = \mathrm{Lim}(1/t) E \log(\mathbf{v}, \mathbf{U}_t) \tag{4.2.3}$$

where $\mathbf{U}_t$ is the sequence obtained from (4.2.1) when $\mathbf{N}_t = \mathbf{u}$. From (4.2.1)

$$\mathbf{U}_t = (\mathbf{A} + \mathbf{B}_t)(\mathbf{A} + \mathbf{B}_{t-1}) \cdots (\mathbf{A} + \mathbf{B}_1)\mathbf{u}$$

which with (4.2.3) leads to

$$a = r_0 - \mathrm{Lim}(1/t) E \log(1 + \mathbf{T}_{1t} + \mathbf{T}_{2t} + \cdots) \tag{4.2.4}$$

where for each i the $\mathbf{T}_{it}$ contains all terms with exactly i of the $\mathbf{B}$'s. Now expand the logarithm in (4.2.4) and evaluate successive terms in the limit (as in Tuljapurkar (1982b)). We omit the details and go to the main result. This requires a spectral decomposition of $\mathbf{A}$ in the form

$$\mathbf{A}/\lambda_0 = \mathbf{P} + \mathbf{Q} \tag{4.2.5}$$

where $\mathbf{P} = \mathbf{u}\mathbf{v}^{\mathrm{T}}/(\mathbf{v}, \mathbf{u})$ is the matrix projecting onto $\mathbf{u}$. Then one gets

$$a \simeq r_0 - \frac{1}{2\lambda_0^2(\mathbf{v}, \mathbf{u})^2} (\mathbf{v} \otimes \mathbf{v})^{\mathrm{T}} S_0 (\mathbf{u} \otimes \mathbf{u}) + \frac{1}{\lambda_0^2(\mathbf{v}, \mathbf{u})} \mathbf{v}^{\mathrm{T}} \left\{ E \sum_{t=1}^{\infty} \mathbf{B}_{t+1} \mathbf{Q}^{t-1} \mathbf{B}_1 \right\} \mathbf{u}. \tag{4.2.6}$$

The first term on the right-hand side of (4.2.6) is simply the growth rate if vital rates were fixed at their average values. The second term describes the way in which variances and covariances of vital rates within a single time interval affect long-run growth rate. The third term describes the effect of between-period (serial) autocorrelation.

The "bottom line" for an approximation is how well it performs. In a large number of tests by several investigators using different methods the approximation (4.2.6) is found quantitatively accurate when vital rates have a coefficient of variation (i.e., ratio of standard deviation to average) of 0.3 or less. For higher levels of randomness the approximations becomes quantitatively inaccurate but continues to describe qualitative dependence accurately.

There is a way of rewriting (4.2.6) which gives insight into the formalism and is useful in applications. For simplicity focus on the first two terms on the right of (4.2.6); these are the only relevant terms when there is no serial dependence between vital rates. We use a standard fact from perturbation theory (a general reference is Kato (1966)), which states that for the average vital rate matrix **A**,

$$\partial r_0/\partial A_{ij} = v(i)\, u(j)/\lambda_0(\mathbf{v}, \mathbf{u}).$$

The derivative $\partial r_0/\partial A_{ij}$ is the SENSITIVITY of r to the elements of **A** (cf. Caswell, 1978). Now the second term of approximation (4.2.6) can be rewritten in terms of these sensitivities. Let $\mathrm{Var}(ij)$ be the stationary one-period variance of $(\mathbf{X}_t)_{ij}$ and $\mathrm{Cov}(ij, kl)$ be the stationary one-period covariance between $(\mathbf{X}_t)_{ij}$ and $(\mathbf{X}_t)_{kl}$. Then a matrix multiplication shows that for IID vital rates

$$a \simeq r_0 - \frac{1}{2}\sum_{i,j} (\partial r_0/\partial A_{ij})^2 \,\mathrm{Var}(ij) - \sum_{\substack{i,j,k,l \\ (i,j)\neq(k,l)}} (\partial r_0/\partial A_{ij})(\partial r_0/\partial A_{kl})\,\mathrm{Cov}(ij, kl). \qquad (4.2.7)$$

This form is very useful for qualitative interpretation. The more general expression in (4.2.6) can be similarly recast using higher eigenvalues of the matrix **A**. Since these higher eigenvalues are not as well characterized as r_0, the value of such a rewriting is moot.

The following features of (4.2.6) are important.

(i) The terms in (4.2.6) are only the first three terms of a systematic expansion which incorporates successively higher order moments of the **B**'s.

(ii) The expansion is what one obtains from Ruelle's (1979) result that a is an analytic function of the rule which selects random matrices. See Tuljapurkar (1982b) for this approach.

(iii) A particularly important point is that expansion (4.2.6) is very different from the Taylor expansions of "utility" functions in foraging theory (Lacey *et al.*, 1983). The qualitative reason for this is the need to

keep track of the ordering of vital rates in this demographic calculation. For the quantitative reason consider Markovian vital rates for which, according to (2.3.10),

$$a = E\log(\mathbf{e}, \mathbf{X}_1\mathbf{Y}_0), \qquad \text{in the steady state,}$$
$$= E\log(\mathbf{e}, \{\mathbf{A}+\mathbf{B}_1\}\{E\mathbf{Y}_0 + \hat{\mathbf{Y}}_0\}$$

where

$$\hat{\mathbf{Y}}_0 = Y_0 - E\mathbf{Y}_0.$$

Now it is tempting to assume that $E\mathbf{Y}_0$ equals $\mathbf{u}$, and expand the logarithm to second order. However, a careful analysis shows that $E\mathbf{Y}_0$ differs from $\mathbf{u}$ by an amount of the same order as the second derivative terms which come from an expansion of the logarithm. In addition, it is necessary here to take into account the serial autocorrelation between $\mathbf{B}_1$ and $\hat{\mathbf{Y}}_0$, an issue which does not arise in present formulations of foraging theory. Hence the mean-variance "tradeoffs" contained in (4.2.6) are different both in quantity and kind from those in foraging theory.

(iv) A point related to (iii) concerns the incorrect notion that (4.2.6) or (4.2.7) are related to the logarithmic mean (LM) of dominant eigenvalues. We defined LM in (3.17) and indeed a Taylor expansion in the spirit of foraging theory shows that for small noise

$$\mathrm{LM} = E\log\lambda(\mathbf{X})$$
$$\approx \log\lambda_0 - \frac{1}{2\lambda_0^2}\sum_{i,j,k,l}\left(\frac{\partial^2\lambda_0}{\partial A_{ij}\,\partial A_{kl}}\right)\mathrm{Cov}(ij, kl).$$

This is very different from (4.2.7). In addition, LM does not take account of serial correlations, which (4.2.6) does. See also Section 4.5.2.

4.2.2. *Population Structure: Moments, Power Spectrum*

The expansion method used for a also yields information about the steady state behavior of the population structure vector $\mathbf{Y}_t$. Recall that $\mathbf{Y}_t$ obeys Eq. (2.2.2) which is a nonlinear stochastic difference equation, so the methods typically used for such equations (Bartlett, 1978; Ellner, 1986) can and will be used here. However, the method of Section 4.2.1 makes a nice and decisive use of demographic weak ergodicity, and we begin with this method. This presentation is brief; details are in Tuljapurkar and Lee (1987).

The objective is to obtain information on $\mathbf{Y}_t$ for large t; ideally we want the stationary probability distribution but will settle for approximations to the moments. Demographic weak ergodicity means that as $t \to \infty$ the

population's structure becomes independent of the initial structure. We can therefore choose an initial structure which makes the calculations easy. As in Section 4.2.1 take $\mathbf{Y}_0 = \mathbf{u}$, and observe that

$$\mathbf{Y}_t = \mathbf{M}_t\mathbf{u}/(\mathbf{e}, \mathbf{M}_t\mathbf{u}) \tag{4.2.8}$$

where

$$\begin{aligned}\mathbf{M}_t &= \mathbf{X}_t\mathbf{X}_{t-1}\cdots\mathbf{X}_1 \\ &= (\mathbf{A}+\mathbf{B}_t)(\mathbf{A}+\mathbf{B}_{t-1})\cdots(\mathbf{A}+\mathbf{B}_1).\end{aligned}$$

Now expand the numerator and denominator in (4.2.8), invert the denominator and collect terms to get to second order in the $\mathbf{B}$'s.

$$\mathbf{Y}_t \cong \mathbf{u} + (1-\mathbf{u}\mathbf{e}^{\mathrm{T}})\mathbf{R}_{1t} + (1-\mathbf{u}\mathbf{e}^{\mathrm{T}})[\mathbf{R}_{2t} - (\mathbf{e}, \mathbf{R}_{1t})\mathbf{R}_{1t}] \tag{4.2.9}$$

with

$$\mathbf{R}_{1t} = \sum_{i=1}^{t} (\mathbf{A}/\lambda)^{t-i}(\mathbf{B}_i/\lambda)\mathbf{u},$$

$$\mathbf{R}_{2t} = \sum_{i=0}^{t-2}\sum_{j=0}^{t-i-2} (\mathbf{A}/\lambda)^{t-i-j-2}(\mathbf{B}_{t-j+2}/\lambda)(\mathbf{A}/\lambda)^{j}(\mathbf{B}_{t-1}/\lambda)(\mathbf{A}/\lambda)^{i}\mathbf{u}.$$

These expressions depend on the choice of initial population structure; to get steady state information we will examine various moments and in each case take the limit of large t.

Consider the important features of (4.2.9). First note that the factor $(1-\mathbf{u}\mathbf{e}^{\mathrm{T}})$ which multiplies every term with $\mathbf{B}$'s in it has two effects: it ensures that $(\mathbf{e}, \mathbf{Y}_t) = 1$; and it is a projection of sorts because

$$\begin{aligned}(1-\mathbf{u}\mathbf{e}^{\mathrm{T}})(\mathbf{A}/\lambda)^{j} &= (1-\mathbf{u}\mathbf{e}^{\mathrm{T}})(\mathbf{P}+\mathbf{Q}^{j}), \qquad \text{from (4.2.5)}, \\ &= (1-\mathbf{u}\mathbf{e}^{\mathrm{T}})\mathbf{Q}^{j}.\end{aligned} \tag{4.2.10}$$

Now from (4.2.5) it should be clear that

$$\mathbf{Q}^{j} \to 0 \qquad \text{as} \quad j \to \infty. \tag{4.2.11}$$

With these facts (4.2.9) leads to the following observations about the population structure.

(i) The second term in (4.2.9) is the leading term in the fluctuations of the structure vector $\mathbf{Y}$ about its average value. This term is essentially a weighted average of the "shocks" to the age structure due to successive random shifts in the vital rates, with the most recent vital rates being most heavily weighted.

(ii) The average population structure does not depend closely on the recent history of vital rates, because $E\mathbf{R}_{1t} = 0$. The current structure $\mathbf{Y}_t$ tracks the environment closely, but the average structure does not. Equations for $E\mathbf{Y}_t$ are given in the Appendix. The difference between $E\mathbf{Y}_t$ and $\mathbf{u}$ depends on the second-order moments of the $\mathbf{B}$'s, as pointed out near the end of Section 4.2.1.

(iii) The variance of the population structure vector can also be obtained from (4.2.9). It is important to recognize the difference between the variance of $\mathbf{Y}$, which is bounded in any case since the components of $\mathbf{Y}$ must sum to 1, and the variance of the population vector $\mathbf{N}$. They are of course related but in a complicated way. It is relatively easy to compute the steady state variance of population structure up to second order in moments of the random rates, in the form

$$E(\Delta\mathbf{Y})_t \otimes (\Delta\mathbf{Y})_t \sim (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}}) \otimes (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\mathbf{R}_1^*, \tag{4.2.12}$$

where the result holds for large times t, and

$$\Delta\mathbf{Y}_t = \mathbf{Y}_t - E\mathbf{Y}_t, \tag{4.2.13}$$

The expression for $\mathbf{R}_1^*$ is written out in the Appendix, Eq. (A9).

(iv) Finally, the autocorrelation function and power spectrum of fluctuations in population structure can be obtained from (4.2.9). We present here only the simplest leading order terms derived by writing a linear difference equation for the fluctuations in the fashion of Bartlett (1978). Define the autocorrelation function

$$\mathbf{C}(m) = E(\mathbf{Y}_{t+m} - \mathbf{u}) \otimes (\mathbf{Y}_t - \mathbf{u}). \tag{4.2.14}$$

A general expression is given in the Appendix.

For serially uncorrelated rates, the autocorrelation is dominated by transients expressed in $\mathbf{Q}$ (recall 4.2.5)

$$\mathbf{C}(m) \cong (1 - \mathbf{L} \otimes \mathbf{L})^{-1} E(1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})(\mathbf{Q}^m \mathbf{B}_1) \otimes \mathbf{B}_1(\mathbf{u} \otimes \mathbf{u}), \tag{4.2.15}$$

with $\mathbf{L} = (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\mathbf{Q}$ and $m \geq 0$.

The multivariate power spectrum of population fluctuations can in principle be obtained by Fourier transforming $\mathbf{C}(m)$. However, it is easier to return to the linear equation from which one gets $\mathbf{C}$ and Fourier transform it. Defining

$$\begin{aligned} \hat{\mathbf{f}}(\omega) &= \sum_{-\infty}^{\infty} e^{-i\omega t}(1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\,\mathbf{B}_t\mathbf{u}, \\ \hat{\mathbf{Z}}(\omega) &= \sum_{-\infty}^{\infty} e^{-i\omega t}(\mathbf{Y}_t - \mathbf{u}), \end{aligned} \tag{4.2.17}$$

one obtains to leading order in the **B**'s that

$$\hat{\mathbf{Z}}(\omega) \simeq (e^{i\omega} - \mathbf{L})^{-1}\,\hat{\mathbf{f}}(\omega), \tag{4.2.18}$$

and the various cross-spectral functions are the elements of $E\hat{\mathbf{Z}}(\omega) \otimes \hat{\mathbf{Z}}^*(\omega)$, where the asterisk indicates a complex conjugate. Note that when $\{\mathbf{B}_t\}$ is serially uncorrelated $\hat{\mathbf{f}}$ is a constant and the spectrum will have peaks at frequencies determined by population transients. When $\{\mathbf{B}_t\}$ is serially correlated, the structure of (4.2.18) shows that the population power spectrum is essentially a product of the spectrum of transients (in **Q**) and the power spectrum of the random rates. Lee (1974) made this observation indirectly by showing that the leading population transient, which is Coale's (1972) generational cycle, shows up as a peak in the population's power spectrum when rates vary randomly. Lee (personal communication) has also earlier derived the product result stated above.

The really challenging task of determining the interaction between the transients in population and in random rates is open. Such interactions will presumably be important when random variation in the rates is large.

4.3. *Exponential Convergence and Liapunov Exponents*

Sections 2.3.1 and especially 3.5 have shown that Liapunov exponents for the product of random vital rate matrices describe the exponential rate at which population structure (and also reproductive value) converges to its stationary stable value. Here we describe what these exponents mean in the classical case, followed by a small noise expansion.

4.3.1. *Liapunov Exponents in Classical Demography*

In the classical case as in Section 2.1 there is a fixed matrix **A** of vital rates, with dominant eigenvalue λ_0 and corresponding right, left eigenvectors **u**, **v**. This matrix also has additional eigenvalues λ_i, $i \geqslant 1$, which we assume to be distinct with magnitudes ordered so that

$$\lambda_0 > |\lambda_1| \geqslant \cdots. \tag{4.3.1}$$

Consider now nonnegative nonproportional vectors **b**, **c** and generate the population vector sequences $\mathbf{B}_t$, $\mathbf{C}_t$ where

$$\mathbf{B}_t = \mathbf{A}^t\mathbf{b}, \tag{4.3.2}$$

and $\mathbf{C}_t$ is defined likewise. To study convergence is to study such differences as $\{\mathbf{B}_t \otimes \mathbf{C}_t - \mathbf{C}_t \otimes \mathbf{B}_t\}$, where the tensor product is as defined in (3.12).

For nonparallel vectors $\mathbf{b}$, $\mathbf{c}$, we can use the spectral decomposition of $\mathbf{A}$ (Keyfitz, 1968) to show that

$$\lim_{t\to\infty}\frac{1}{t}\log\|\mathbf{B}_t\wedge\mathbf{C}_t\|\leqslant\log\lambda_0+\log|\lambda_1|. \tag{4.3.3}$$

The right-hand side of (4.3.3) is the largest value possible for the left taken over all possible starting vectors $\mathbf{b}$, $\mathbf{c}$. From definition (2.3.6) we see that the second Liapunov exponent for a fixed vital rate matrix is the absolute value of the leading subdominant eigenvalue.

The higher (in fact, smaller in magnitude) eigenvalues of the vital rate matrix $\mathbf{A}$ are related to the other Liapunov exponents in an analogous way.

4.3.2. *Small Noise Expansion of* ρ_2

Just as expansion (4.2.6) for a provides insight into the determinants of stochastic growth rate, one expects a similar expansion of the next higher Liapunov exponent to be informative about convergence. The main steps in the latter expansion are as follows.

The plan is to take nonparallel vectors $\mathbf{b}$, $\mathbf{c}$ and compute leading terms in the asymptotic behavior of $(\mathbf{M}_t\mathbf{b}\wedge\mathbf{M}_t\mathbf{c})$ where

$$\mathbf{M}_t=(\mathbf{A}+\mathbf{B}_t)(\mathbf{A}+\mathbf{B}_{t-1})\cdots(\mathbf{A}+\mathbf{B}_1), \tag{4.3.4}$$

as in Section 4.2. For the matrix $\mathbf{A}$ we assume all the properties given in Section 4.3.1. Next we choose

$$\mathbf{b}=\mathbf{u},\qquad \mathbf{c}=\mathbf{u}+k\mathbf{u}_1+k^*\mathbf{u}_1^*, \tag{4.3.5}$$

where $\mathbf{u}_1$ is the right eigenvector of $\mathbf{A}$ corresponding to the eigenvalue λ_1 closest in magnitude to λ_0. This choice is the simplest possible. The asterisks in (4.3.5) indicate complex conjugates.

Now use (4.3.4) to write $(\mathbf{M}_t\mathbf{b}\wedge\mathbf{M}_t\mathbf{c})$ as a series of terms which are successively linear, quadratic, ..., in the $\mathbf{B}$'s. It is now possible to use methods analogous to those mentioned in Section 4.2.1 to obtain the rate of change of this quantity when the $\mathbf{B}$'s are small. Details are given in Tuljapurkar (1988). In the IID case one gets

$$\begin{aligned}\lim_{t\to\infty}\frac{1}{t}E\log\|\mathbf{M}_t\mathbf{b}\wedge\mathbf{M}_t\mathbf{c}\|&\\ =a+\rho_2&\\ \simeq\log\lambda_0+\log|\lambda_1|-\frac{1}{2\lambda_0^2}\frac{(\mathbf{v}\otimes\mathbf{v},\,\mathbf{S}_0\mathbf{u}\otimes\mathbf{u})}{(\mathbf{v},\,\mathbf{u})^2}-\psi_1,&\end{aligned} \tag{4.3.6}$$

where

$$\psi_1 = \frac{1}{2}\,\mathrm{Re}\left\{\frac{(\mathbf{v}_1 \otimes \mathbf{v}_1, \mathbf{S}_0\mathbf{u}_1 \otimes \mathbf{u}_1)}{(1-\xi_1^2)(\mathbf{v}_1, \mathbf{u}_1)^2}\right\} + \mathrm{Re}\left\{\frac{(\mathbf{v}_1 \otimes \mathbf{v}, \mathbf{S}_0\mathbf{u} \otimes \mathbf{u}_1)}{(1-\xi_1)(\mathbf{v}_1, \mathbf{u}_1)(\mathbf{v}, \mathbf{u})}\right\}. \tag{4.3.7}$$

Here $\mathbf{S}_0$ is the second moment matrix of the $\mathbf{B}_t$'s defined in (4.2.2). Recall now the earlier expansion (4.2.6) for a, in which only the first two terms are needed for the IID case. Using that result in (4.3.6), one gets finally

$$\rho_2 \approx \log|\lambda_1| - \psi_1. \tag{4.3.8}$$

The qualitative features of (4.3.8) need to be explored. This approximation works accurately for the example of Section 3.

4.4. *Reproductive Value*

The notion of reproductive value is useful in classical demography and population genetics. It has been particularly significant in life history theory where reproductive value maximization has been used (see, e.g., Stearns, 1976) to predict the direction of life history evolution. This subsection develops the concept as it applies to time-varying and randomly varying rates.

4.4.1. *Time-Varying Vital Rates*

It is simplest to begin with a population whose vital rates are changing in some arbitrary time-dependent manner, but with demographic weak ergodicity holding (cf. Section 2.2). The population follows Eq. (2.2.1) in which the vital rates have the interpretation

$$(\mathbf{X}_t)_{ij} = \text{Number of class } i \text{ individuals at time } t \text{ per class } j \text{ individual at time } t-1. \tag{4.4.1}$$

Consider now a quantity $W_t(i)$, to be called the undiscounted reproductive value of an individual in class i at time t, and defined as the total number of descendants (i.e., children, grandchildren, and so on) produced by an individual who is in class i at time t. From this definition and the interpretation of the vital rates above it follows that

$$W_t(i) = \sum_j (\mathbf{X}_{t+1})_{ji}\, W_{t+1}(j). \tag{4.4.2}$$

The vector form of this recursion is

$$\mathbf{W}_t = \mathbf{X}_{t+1}^{\mathrm{T}}\mathbf{W}_{t+1} = \mathbf{X}_{t+1}^{\mathrm{T}}\mathbf{X}_{t+2}^{\mathrm{T}}\cdots\mathbf{X}_{t+m}^{\mathrm{T}}\mathbf{W}_{t+m}. \tag{4.4.3}$$

To deal with the obvious possibility that the **W**'s are likely to be unbounded, define instead the normalized (or discounted) reproductive value vector

$$\mathbf{V}_t = \mathbf{W}_t/(\mathbf{e}, \mathbf{W}_t) \tag{4.4.4}$$

which follows the recursion

$$\mathbf{V}_t = \mathbf{X}_{t+1}^{\mathrm{T}}\mathbf{V}_{t+1}/(\mathbf{e}, \mathbf{X}_{t+1}^{\mathrm{T}}\mathbf{V}_{t+1}). \tag{4.4.5}$$

Observe that (4.4.5) appeared earlier as (2.3.7), and is clearly very similar to (2.2.2). The dynamics of both equations can be understood by considering matrix products.

Take two times, k and $l=(k+m)>k$, and consider

$$\mathbf{Z}(k, l) = \mathbf{X}_l\mathbf{X}_{l-1}\cdots\mathbf{X}_{k+1} \tag{4.4.6}$$

which is a product of m matrices. Demographic weak ergodicity means (Hajnal, 1976) that this product will have its rows all proportional as m increases. Thus there is a number $R(k, l)$ and vectors $\mathbf{v}(k, m)$ and $\mathbf{u}(l, m)$ such that

$$\mathbf{Z}(k, l) \sim R(k, l)\,\mathbf{u}(l, m)\,\mathbf{v}^{\mathrm{T}}(k, m) \qquad \text{as} \quad m\uparrow.$$

Further we know that there is stability of age structure, meaning that $\mathbf{u}(l, m)$ approaches some $\mathbf{u}(l)$ asymptotically independent of m; similarly $\mathbf{v}(k, m)$ approaches some $\mathbf{v}(k)$. Thus asymptotically for large m

$$\mathbf{Z}(k, l) \sim R(k, l)\,\mathbf{u}(l)\,\mathbf{v}^{\mathrm{T}}(k). \tag{4.4.7}$$

Numerical insights into (4.4.7) are to be found in Kim and Skyes (1976). Suppose now that we start with a population vector $\mathbf{n}^*$ at time $t=k$. Then at time l we have asymptotically a population vector

$$\mathbf{N}_l \sim R(k, l)(\mathbf{v}(k), \mathbf{n}^*)\,\mathbf{u}(l),$$

with population structure $\mathbf{Y}_l \sim \mathbf{u}(l)$. The growth rate here is contained in $R(k, l)$, so that $\log R(k, l)/(l-k) \to a$ for stochastic rates as $(l-k)\to\infty$. The normalized reproductive value at time k is $\mathbf{V}_k \sim \mathbf{v}(k)$. To find $\mathbf{v}(k)$ simply start with an arbitrary nonnegative vector $\hat{\mathbf{v}} \neq \mathbf{0}$ at time $t=l \gg k$ in (4.4.5) and iterate backwards.

Notice that the vital rate matrices **X** in (2.2.2) act to propagate popula-

tion vectors forward in time, and that the stable age structure at each time is an accumulation of the past. In contrast, the transposed vital rate matrices $\mathbf{X}^T$ in (4.4.5) act to propagate reproductive value backward in time, and the reproductive value at each time is a summation of the future.

4.4.2. *Random Rates*

The foregoing clearly applies when vital rates follow a random process as in Section 2.2. However, the vital rates in (4.4.5) appear in time-reversed order and this brings us back to Assumption 2.3.2 in Section 2.3: to make probabilistic statements about reproductive value requires that the random process generating vital rates can be run backwards in time giving rise to a unique, stationary, and ergodic random process. Such backwards processes are easily defined in the Markov rates model. Given this assumption, everything that applies to the dynamics of the population structure vector will apply to the reproductive value vector.

In particular, there will be a joint stationary distribution of rates and reproductive values. In the Markov case, this distribution is computable using the appropriate version of Cohen's (1977b) equation as applicable to (4.4.5). The small noise approximation results of the preceding subsection will apply to provide approximations to the average reproductive value vector. It is important to note, however, that the expectations in equations such as (4.2.12) must be replaced by expectations with respect to the time-reversed process, and correspondingly the ordering of time indices must be reversed.

4.5. *Growth Rates*

4.5.1. *Inequalities*

Consider a model of random vital rates formed as in (4.2.1) by adding a random term to a deterministic matrix of vital rates, so the time-dependent matrix of rates is

$$\mathbf{X}_t = \mathbf{A} + \mathbf{B}_t.$$

Take a fixed population vector $\mathbf{n}_0$ and produce the *deterministic* population vector

$$\mathbf{n}_t = \mathbf{A}^t \mathbf{n}_0$$

and the *stochastic* population vector,

$$\mathbf{N}_t = \mathbf{X}_t \mathbf{X}_{t-1} \cdots \mathbf{X}_1 \mathbf{n}_0.$$

Let the corresponding total population sizes be p_t, P_t.

We have focused on the long-run stochastic growth rate

$$a = \operatorname*{Lim}_{t \to \infty} \frac{1}{t} E \log(P_t/p_0), \tag{4.5.1}$$

which is relevant in most demographic, ecological, and evolutionary problems. It is sometimes useful to consider the long-run growth rate of average population size,

$$\log \mu = \operatorname*{Lim}_{t \to \infty} \frac{1}{t} \log[(EP_t)/p_0]. \tag{4.5.2}$$

Now it is a consequence of Jensen's inequality (Karlin and Taylor, 1975) that

$$a \leqslant \log \mu, \tag{4.5.3}$$

with inequality expected in general. The further consequences of this inequality are discussed by Lewontin and Cohen (1969), Tuljapurkar and Orzack (1980).

How do these stochastic growth rates compare with the classical deterministic growth rate

$$r_0 = \operatorname*{Lim}_{t \to \infty} \frac{1}{t} \log(p_t/p_0)? \tag{4.5.4}$$

When vital rates are IID,

$$r_0 = \log \mu \geqslant a. \tag{4.5.5}$$

In *general* (Cohen, 1979), however, serial autocorrelation in the rates can complicate matters. Depending on the particular random rates, it is possible to have

$$\log \mu > r_0 > a$$

or

$$\log \mu > a > r_0.$$

Some conditions for these situations can be deduced, for example, by using the expansions for $\log \mu$ in Tuljapurkar (1982a) and for a in Section 4.2.1.

4.5.2. *Synergistic Effects of Environment*

This paper has made several references to the distinction between the multidimensional dynamics of structured populations and simpler scalar

growth models. A striking illustration of this difference is provided by situations in which the randomness of the environment makes the difference between population growth and decline.

Consider a population with two age classes and suppose that the population's vital rates are given by one of two Leslie matrices,

$$\mathbf{A} = \begin{pmatrix} \frac{1}{4} & 3-x \\ \frac{1}{4} & 0 \end{pmatrix},$$

$$\mathbf{B} = \begin{pmatrix} \frac{15}{16} - 0.1 & \frac{1}{16} + 0.09 \\ 1 & 0 \end{pmatrix}.$$

Take the environment to change randomly so that in each time interval the population's Leslie matrix is $\mathbf{A}$ with probability p or $\mathbf{B}$ with probability $(1-p)$. If we set $x = 0.142857$ and $p = 0.5$ a numerical simulation (of 5000 iterations of the stochastic growth process) yields an estimate $a = +0.1954$ with a sample standard error of $\hat{s} = 0.0047$. Thus, the population should increase with probability one over the long term. Yet computation of dominant eigenvalues of the matrices shows

$$\log \lambda_0(\mathbf{A}) = -0.0209,$$

$$\log \lambda_0(\mathbf{B}) = -0.0087.$$

Therefore in the absence of a random environment with matrix $\mathbf{A}$ or $\mathbf{B}$ fixed forever, the population would decline. We have here a synergistic effect of random variation. Key (1986) suggested this term in the context of a multitype branching process using special kinds of matrices. The example given above is easily generalized to more parameters or dimensions.

An equally important aspect of this example is that it shows decisively that the logarithmic mean dominant eigenvalue *cannot* accurately describe a, since

$$\mathrm{LM} = 0.5[\log \lambda_0(\mathbf{A}) + \log \lambda_0(\mathbf{B})] < 0,$$

whereas $a > 0$. It should also be obvious that this example derives fundamentally from the multidimensional character of the problem, and would not be possible without age structure. See the discussion in Sections 3.8 and 4.2.1. We have not used small noise approximations here since the noise is large.

4.5.3. *Convexity Properties*

The sensitivity of growth rate to parameters affecting vital rates is important in classical demography. The results of Section 4.2.1 provide some information on the sensitivity of a to stochastic properties of vital rates.

There are many problems in which the vital rates depend on parameters and one wants to know how changes in these parameters will affect a. Cohen (1980) has established the following general result which provides some information.

Let $(\theta_1, \theta_2, ..., \theta_m) = \boldsymbol{\theta}$ be a set of real parameters, and suppose that the random vital rates in (2.2.1) depend on these:

$$(\mathbf{X}_t)_{ij} = F_{tij}(\boldsymbol{\theta}). \tag{4.5.6}$$

Here the F_{tij} are random functions, some of which are identically zero. *Assume* that for every t, i, j

either $F_{tij}(\boldsymbol{\theta}) = 0,$

or $\log F_{tij}(\boldsymbol{\theta})$ is a convex function of $\boldsymbol{\theta}$.

Then a defined via (2.2.1) for each $\boldsymbol{\theta}$ is also a function $a(\boldsymbol{\theta})$, and Cohen proves that $a(\boldsymbol{\theta})$ is a convex function of $\boldsymbol{\theta}$. A similar result holds for the $\log \mu$ defined in (4.5.2).

4.5.4. *A Simplification for Age-Structure: Estimating a*

Given Markovian vital rates, the growth rate a is in general computed according to (2.3.10) which requires an average over the joint distribution of vital rates and population structure. However, for *age-structured* populations, a remarkable simplification in (2.3.10) can be obtained as follows. In the Appendix it is shown that

$$a = E \log S_t(1) - E \log Z_t(2), \tag{4.5.7}$$

where $S_t(1)$ is the (random) survival rate of the youngest age class, and $Z_t(2) = [N_t(2)/N_t(1)]$ is the ratio of number in age-class 2 to the number in age-class 1. This equation is remarkably close to the classical analog which can be written

$$r = \log p(1) - \log[u(2)/u(1)]$$

where $u(1)$, $u(2)$ are stable proportions in age classes 1, 2. In practical cases where one has partial census information together with some statistics on $S_t(1)$, (4.5.7) yields a quick estimate of a. Equation (3.11) is, of course, a special case of (4.5.7) for two age classes.

4.6. *Questions*

The results above show how much we do not know. Here is a partial list of interesting questions and directions.

(i) The distribution of age structure is difficult to get at either numerically (see the 2-age class calculation in Cohen (1977b) which looks an unlikely candidate with more age classes) or analytically (see Tuljapurkar (1984)). Are there efficient numerical approaches to learning about the distribution or the approach to stability? Can one establish conditions under which the distribution is absolutely continuous as opposed to singular? Will asymptotic methods work for small noise, i.e., can one expand the distribution around say a Gaussian whose variance depends on the magnitude of noise?

(ii) Are there other ways of learning about the analytics of a and ρ's? Can one get analytical information on the behavior of these for large noise? Is there a scaling theory for these exponents (cf. the literature on characteristic exponents in nonlinear equations, e.g., Napiorkowski and Zaus (1986))?

(iii) What is the behavior of the power spectrum in higher orders (result (4.2.21) is very rough and reveals nothing about the interaction between transients induced by random rates)? Biologically, the power spectrum tells us about cohort synchrony and population cycling, both subjects of great practical interest.

(iv) Exact results for a seem very difficult to get in biologically interesting cases. Even apparently simple cases (e.g., only one survival rate varies and there are simplifying structural assumptions on the matrix elements) have so far been intractable, although some cases have been solved (see e.g., Section 3).

5. Evolution

Population biologists have long been concerned with the evolution of demographic vital rates, and so with the analysis of combined genetic–demographic models. This section reviews available results about such models in random environments, and an application by Orzack (1985) to the evolution of homeostasis. Other applications are considered in Section 7.

5.1. *One Locus, Age Structure, Invasion*

The simplest situation has two alleles 1, 2 at a locus in a diploid random-mating population. Various assumptions (e.g., the population is monoecious, or there is a fixed relationship between vital rates in the sexes) allow one to ignore sex differences, and the resulting deterministic theory was studied by Norton (1928) and extended by Charlesworth (1980, and

references therein). Tuljapurkar (1982b) studied the case of randomly varying vital rates with the following results.

It is only necessary to track the frequency of allele 1 in the youngest age class. The resulting equations are complex but a boundary analysis (as in Haldane and Jayakar, 1963; Karlin and Lieberman, 1974) can be done with the following results. Consider invasion: the population initially is almost all genotype 11 and we introduce a few individuals carrying allele 2. To each genotype ij one can assign a long-run stochastic growth rate a_{ij}, which is the growth rate of a homogeneous population whose vital rates are those corresponding to genotype ij. The boundary analysis shows that allele 2 increases in frequency at the expense of allele 1 if

$$a_{12} > a_{11}. \tag{5.1.1}$$

Protected polymorphism occurs if $a_{11} < a_{12} > a_{22}$.

Condition (5.1.1) shows that *a plays the same role for random rates* as r_0 in classical deterministic evolutionary theory. Charlesworth (1980, Chapter 5) discusses the latter situation and his remarks are pertinent here. In particular a is a suitable fitness measure for studies of life history evolution in random environments. Little is known in the stochastic case about interior gene frequency dynamics away from the boundaries (although see Section 5.3).

5.2. *One Locus, Stage Structure, Cloning*

Consider a problem which looks quite different from the one just discussed: the dynamics at one locus in a size/stage structured population in which individuals reproduce both sexually and by cloning. A nice example is jack-in-the-pulpit for which a stage structured description was given in Section 2.4.2 following Bierzychudek (1982). Sexually produced offspring here are seeds which make up stage class 1. The smallest (in leaf area) group of plants make up stage class 2, which contains both plants produced by germinating seeds as well as all clonally produced offspring. In the present genetic context we have three genotypes each of which has a growth matrix of type (2.4.2.1).

We focus on invasion and so need only track the frequency Q_t of allele 1, say, in stage class 1. Let $B_{ijt} = B_{jit}$ = (number of sexually produced seeds of genotype ij at time t). Then

$$Q_{t+1} = \sum_j (B_{ijt+1} + B_{jit+1})/2B_{t+1},$$

with $B_{t+1} = \sum_{i,j} B_{ijt+1}$. These numbers of seeds depend of course on the entire population structure at time t, and thus on the clonal reproductive

rate. Now suppose that the stage class vectors of genotypes 12, 21, and 22 are ε_{ijt}, with very small elements compared to the numbers of genotype 11. Linearization of the frequency recursions leads to the equations

$$\begin{aligned} \boldsymbol{\varepsilon}_{12t+1} &= \mathbf{L}_{12t+1}\boldsymbol{\varepsilon}_{12t} + \begin{pmatrix} \mathbf{m}_{22t+1}^{\mathrm{T}} \\ \mathbf{0} \end{pmatrix} \boldsymbol{\varepsilon}_{22t}, \\ \boldsymbol{\varepsilon}_{22t+1} &= \begin{pmatrix} \mathbf{0}^{\mathrm{T}} \\ \mathbf{S}_{22t+1} \end{pmatrix} \boldsymbol{\varepsilon}_{22t}. \end{aligned} \tag{5.2.1}$$

Here (cf. Section 2.3.2) $\mathbf{L}_{12}$ is the growth matrix for heterozgotes, $\mathbf{m}_{22}$ is the vector of stage-specific sexual fecundity (female seeds/female), and $\mathbf{S}_{22}$ contains clonal reproduction rates. Therefore $\boldsymbol{\varepsilon}_{22t}$ need not decrease over time and the invasion dynamics of (5.2.1) are not independent of the 22 homozygotes. Indeed one can have invasion of allele 2 even when $a_{12} < a_{11}$, if genotype 22 has a sufficiently high cloning rate.

5.3. *Homeostasis or the Lack of It*

An interesting application of (5.1.1) is to inquire into conditions under which evolution might favor decreased homeostasis in vital rates. Orzack (1985) observed that homeostasis measures the buffering (lack of response) of vital rates (among other things) to environmental change, and thus decreased homeostasis could correspond to increased variance in vital rates over time. In a random environment, the fitness a depends on the variance in vital rates, so one can ask when an increase in this variance leads to an advantage in a.

Expansion (4.2.6) shows immediately that in the absence of serial autocorrelation, a must decrease as variance in vital rates increases, all other things being equal. This goes along with the classical argument that homeostasis is selectively advantageous. However, in the presence of serial autocorrelation, there is an additional term in (4.2.6) and Orzack shows that with sufficiently high serial autocorrelation, decreased homeostasis can lead to increased a. It is easy to see qualitatively how this happens. High serial autocorrelation means that there are long periods when vital rates change relatively little over time (the environment has "runs"), separated by sudden large changes in the rates. Thus an organism with decreased homeostasis might be able to exploit these runs to achieve a higher long-run growth rate. Indeed, Orzack shows that such a less homeostatic genotype can fix in a population in a serially correlated environment.

To illustrate the phenomenon, consider genotypes whose fertility fluctuates over time according to a stationary random process with serial autocorrelation s. (A specific model is a two-state Markov chain for

fertility.) The result of calculations based on (4.2.6), confirmed by stochastic simulations, is illustrated in Fig. 6. The curves shown are schematic and based on Orzack (1985). The solid curve shows how the least homeostatic (most variable fertility) genotype has an a which increases with s. The dashed curve shows how a more homeostatic genotype has higher fitness when $s=0$ but that as s increases, this fitness advantage is eventually reversed.

Orzack argues that such an effect may underlie some of the phenotypic plasticity observed in nature. He shows that decreased homeostasis can be selected for in both positively and negatively correlated environments. In addition, covariation of vital rates can allow an increase in variance for a particular vital rate. His results show that the temporal structure of a life history is an arena for natural selection. Thus, Gillespie's (1977) principle

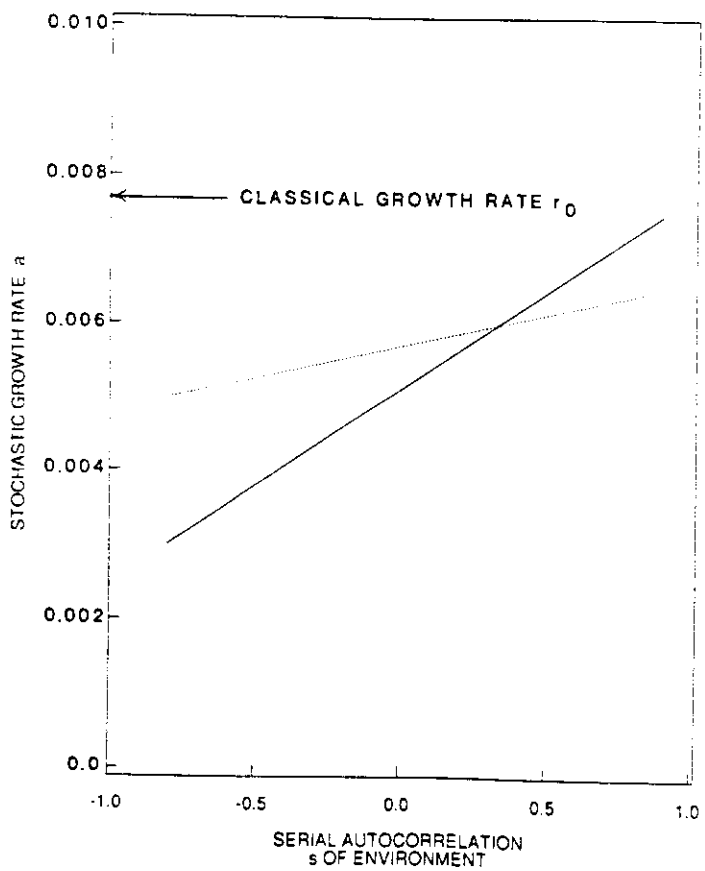

FIG. 6. Schematic illustration of the effect of serial environmental autocorrelation s on stochastic growth rate. Here r_0 is the growth rate in the absence of random variation. For $s=0$ the life history represented by the long-dashed line has higher fitness in the presence of random variation than the life history represented by the short-dashed line. As s increases this fitness difference decreases and is actually reversed for high autocorrelation.

that there should always be selection for increased homeostasis in offspring number needs to be weakened for complex life histories in correlated environments.

5.4. *Open Questions*

Among the most interesting ones:

(a) what happens in the interior of gene frequency space; how does a protected polymorphism here differ from the scalar situation; will diffusion approximations work?

(b) what happens in two-locus cases?

(c) how does one develop a quantitative genetic theory; does deterministic weak-selection theory (Lande, 1982) provide a starting point?

(d) how does one extend the invasion (ESS) analysis of Section 5.1 to complex life cycles, especially those involving dormancy, dispersal, or a mixed sexual-cloning pattern of reproduction?

6. Life History Evolution

6.1. *The Tangled Background*

Fisher (1930) posed one of the central questions about life histories, asking how the apportionment of reproduction over life might have evolved. Cole (1954) framed the style of many recent studies in comparing the evolutionary advantage of semelparity (reproducing once) and iteroparity (reproducing more than once). Since then there has been considerable work on general features of life history evolution (e.g., Williams, 1966; the review by Stearns, 1976; Begon *et al.*, 1986), and on features specific to certain species or genera (e.g., Denno and Dingle, 1981; Jackson *et al.*, 1985). It is clear from Lewontin (1965) that classical demographic arguments can only account for some of the life historical patterns in nature, and later workers have tried to incorporate factors outside the classical framework. One of these is an environment which produces random variations in vital rates and thereby generates selection pressures on life histories.

Although there has been considerable interest in the evolutionary consequences of random vital rates, the theoretical picture has been rather confusing. A review by Stearns (1976) lists features of two contradictory views of how randomness affects life history evolution. One, the r–K view, relied on deterministic theory to conclude that so-called "r-selected" life histories

were most advantageous in the presence or variability. The other, the bet-hedging view, used some version of stochastic theory to argue that "more" iterations life histories were more advantageous. This tentative state of the theory is undoubtedly due to the fact that until recently no analysis has dealt with (1) age structure, (2) random environmental variability, (3) life histories of arbitrary length, (4) variation and covariation of components of the life history, (5) a genetic basis for life history differences, to yield predictions about the rate and direction of natural selection on particular types of life histories.

The situation has begin to change with the work of Orzack and Tuljapurkar (1987) on the evolution of iteroparity and Roerdink (1987) on the difference between biennials and perennials.

6.2. *Iteroparity*

The problem: does environmentally driven fluctuation in vital rates result in an evolutionary advantage for iteroparous life histories? This question was addressed by Murphy (1968), Schaffer (1974), Giesel (1976), Hastings and Caswell (1979), and Goodman (1984), none of whom used the theory of stochastic demography. Orzack and Tuljapurkar (1989) have used the theory discussed in Sections 3 and 4 of this paper, taking iteroparity to be a continuous character described by the temporal clumping and positioning of reproduction during life. They combined analytical and simulation results to study the relative fitness (a) of iteroparous life histories chosen from constrained phenotype sets; they assumed zero serial autocorrelation between environments over time. The main conclusions were as follows.

6.2.1. *Crossover*

Think of a life history in a random environment as a phenotype whose vital rates have some average values plus stochastic fluctuation around this average. In the absence of fluctuations, the average rates determine the intrinsic rate of increase r_0, and the usual theory (Charlesworth, 1980) compares the relative fitness of genotypes by comparing r_0 values. In a random environment the key difference (cf. Section 5) is that one must compare a values between phenotypes. The crucial new feature that emerges is that random variation in rates can completely reverse the relative fitness ranking of life history phenotypes; call this the CROSSOVER effect.

An illustration is provided by a set of life histories in which random variation affects the survival of the youngest age class. Consider three life histories (in the spirit of Murphy (1968)) which have geometrically declining survivorship curves, equal fertilities in all reproductive years

(Fig. 7a) and the same average net reproduction rate R_0. Suppose that the survival rate of juveniles in these life histories is variable; this translates into random variation of adult fertility with perfect correlation between all pairs of fertilities. If the coefficient of variation of the fertilities is C, (4.2.6) reduces to

$$a \simeq \log \lambda_0 - \frac{C^2}{2T_0^2} \tag{6.2.1}$$

where T_0 is the mean generation length ($T_0 = \sum i m_i l_i \lambda_0^{-i}$) and we consider reproductive spans beginning at age 2 or greater. Equation (6.2.1) implies that as C (the level of uncertainty in the environment) increases the value of a will decrease. In addition, the decrease will be relatively greater for populations with lower mean generation lengths. The three life histories in Fig. 7a are labelled so that $r_0(1) > r_0(2) > r_0(3)$. However, the figure suggests, and calculation confirms, that the mean lengths of generation are ordered as $T_0(1) < T_0(2) < T_0(3)$. Therefore (6.2.1) predicts that for

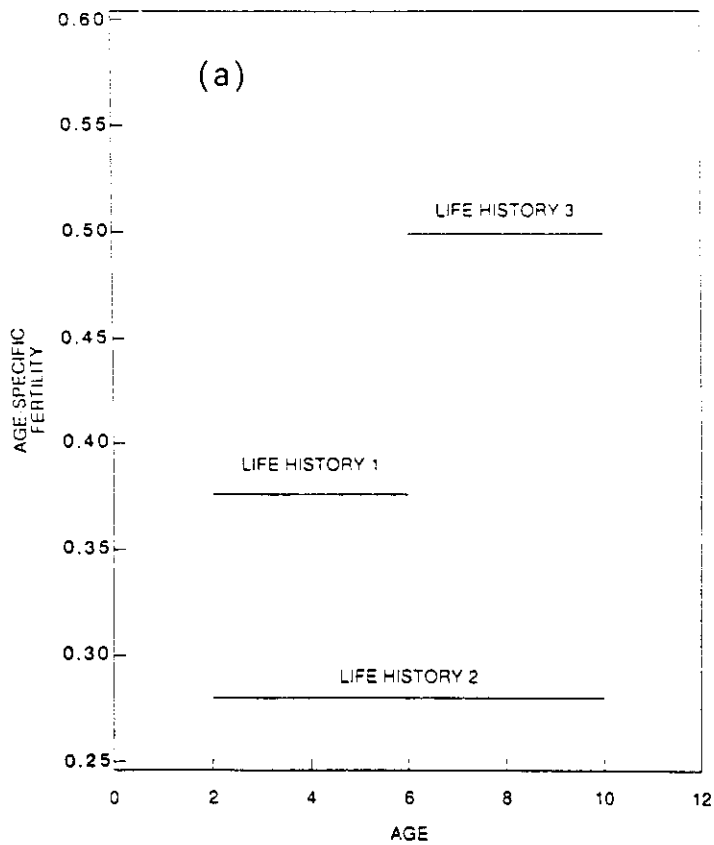

FIG. 7a. Three stylized life histories. Each has the same average net reproductive rate, exponentially declining survivorship, and the deterministic r_0 values are ranked as $1 > 2 > 3$.

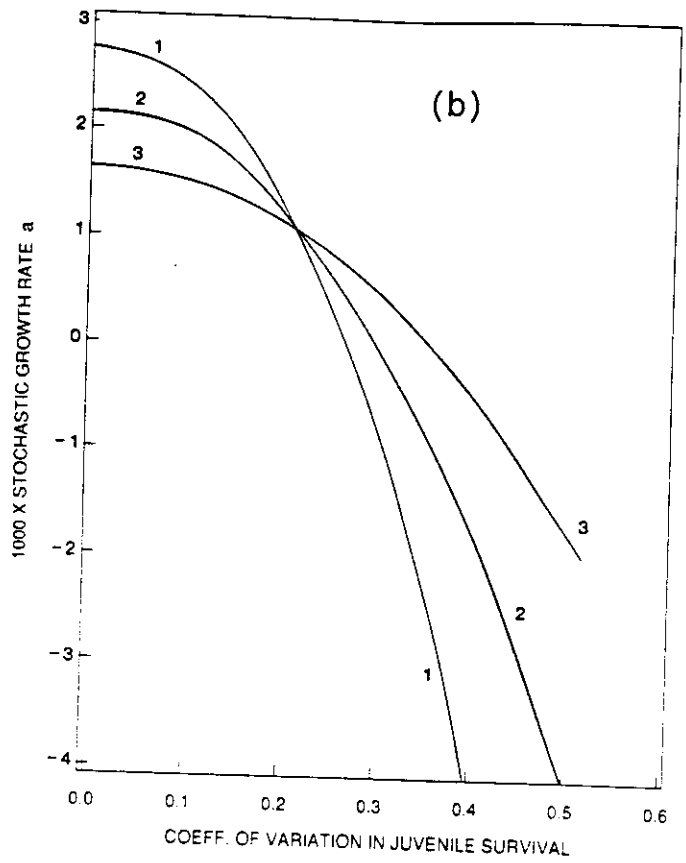

FIG. 7b. Stochastic growth rate a for increasing coefficient of variation C for the three life histories in Fig. 7a. For high C there is a crossover effect so that the fitness ranking of the life histories is reversd from the deterministic one.

sufficiently high C one will have a CROSSOVER in growth rates, so that $a(1) < a(2) < a(3)$. This analytical prediction is nicely borne out by simulations with the result shown in Fig. 7b: sufficiently high variability in the environment reverses the relative advantages of these three life histories.

This example shows that uncertainty can have dramatic impact but it is premature to conclude for example that iteroparity is generally advantageous in a randomly varying environment. The magnitude of random variability and the structure of the life history are important in the relative ranking of life histories, and distinct life histories can have very similar a for the same level of random variation.

Two relatively general conclusions:

(1) The growth rate a for life histories can be distinguished by differences in r_0 and the degree of discounting of environmental fluctuations by the mean generation length.

(2) There can be selection between genotypes purely on the basis of

the sign of the covariances between vital rates. For example, assume that environmental variability only affects fertilities. Then, a mutant heterozygote with negative correlations among fertilities can enter a population composed of a genotype with *identical* average fertilities which are independent or positively correlated. Hence some cases of negative correlations between life history components may be the product of natural selection instead of indicating an unavoidable constraint or "cost" of reproduction.

6.2.2. *Levels of Variability*

The crossover discussed above is a robust feature of random vital rates, occurring over a wide range of life histories with fluctuations in a variety of age-specific rates. Examination of many cases suggests three regimes of randomness:

(a) LOW VARIABILITY: r_0 SELECTION. Here random variation has some effect but not enough to alter a ranking of life histories in terms of their r_0 values. We expect classical arguments, employed for example by Lewontin (1965), to describe much of the selective pressure on life histories.

(b) INTERMEDIATE VARIABILITY: NEUTRAL OUTCOMES. Here crossover has just begun to occur and life histories with very different r_0 values have very similar a values. This regime allows distinct temporal distributions of reproduction and survival to be selectively neutral with respect to one another. Thus one expects sets of equally plausible evolutionary endpoints, the precise course of evolution being determined by historical or chance events.

(c) HIGH VARIABILITY: a SELECTION. Crossover is complete here and the ranking of life histories by r_0 values is reversed. Selective advantage will lie with those life histories for which reproduction is more spread out over life, i.e., "more" iteroparous life histories.

The identification of regimes depends on the extent of variation, and it is important to ask: what does a coefficient of variation mean biologically? The answer is that the variance in a vital rate is a result of (1) the variance in the environment to which the organisms are responding, and (2) the environmental sensitivity of the organism which translates environmental change into change in vital rates. Thus an organism which is well buffered might see a particular environment as type (a) above, while to a poorly buffered organism the same environment might be type (c). In addition, the impact of variability on a depends on the covariance of rates, so the fact that just one vital rate has high variance can be misleading without information on other vital rates.

A key difference between these results and much life history theory is in the importance of polymorphism. The neutral (with respect to a) life histories we identify can coexist in the same sense as neutral enzyme polymorphisms do. On the other hand, we have following (5.1.3) the conditions for a protected polymorphism. When heterozygous individuals are intermediate in average life history between constituent homozygotes in the crossover regime of intermediate variability, it is clearly possible to have selectively maintained polymorphisms.

These results also bear on the relationship between plasticity and genetic polymorphism (Bradshaw, 1965; Jain, 1979; Scheiner and Goodnight, 1984). Phenotypic plasticity is measured by the individual coefficients of variation for vital rates and the pairwise correlation between vital rates. Since the growth rate a depends sensitively on these measures of plasticity,

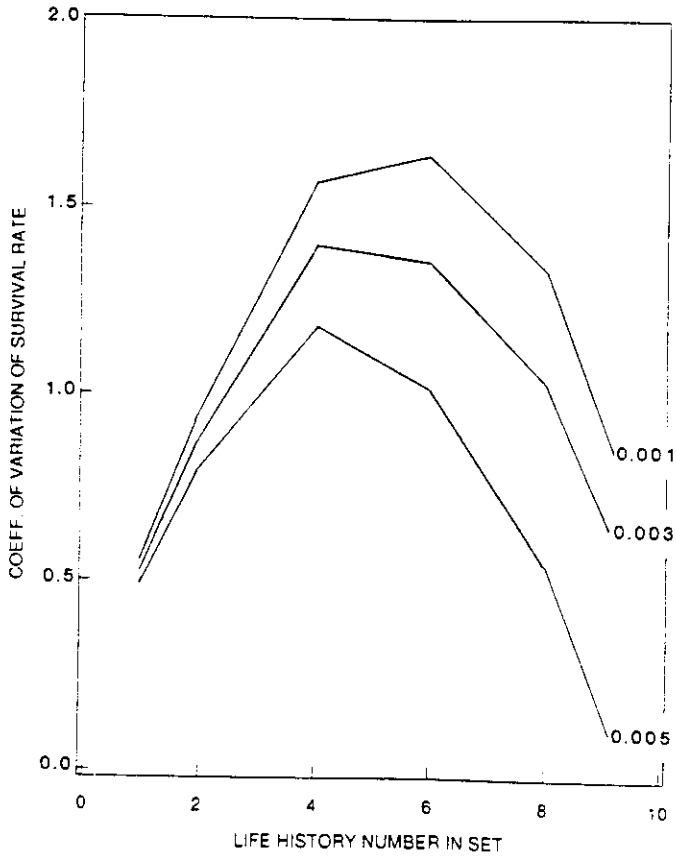

FIG. 8. Life histories numbered 1 through 9 belong to a set described below. Each curve in the figure links C (level of variation) values at which all live histories have equal a (i.e., are selectively neutral). Each curve is labeled by the corresponding value of a. The life histories have equal average net reproductive rate R_0, and net maternity schedule (i.e., $l_x m_x$) declining with age (x). Life histories 1 to 5 have first reproduction at age $\alpha = 1$ and last reproduction at age $\omega = 2$ through 10 in steps of 2. Numbers 3 to 9 have $\omega = 10$ and α increasing from 3 to 9 in steps of 2.

and in turn a determines conditions for polymorphism, there is a direct relationship implied by the theory. As pointed out by Scheiner and Goodnight, the relationship is not necessarily antagonistic. Selection can be coupled: for example, if plasticity were to increase in a variable environment, there would be an increase in C and an increasing advantage to iteroparous life histories. In such a case, genetic variation might decrease as plasticity increases. However, an increase in plasticity which pushed a population from low C to intermediate C regimes would open the population to invasion by a wide range of life histories, and here increasing plasticity could drive an increase in genetic polymorphism.

6.2.3. *Murphy's Result Revisited*

The crossover phenomenon discussed above can be looked at in a different way. Consider a set of life histories which obey a common constraint, say that their average vital rates all lead to the same net reproductive rate. Suppose now that each life history in the set is subject to random variation

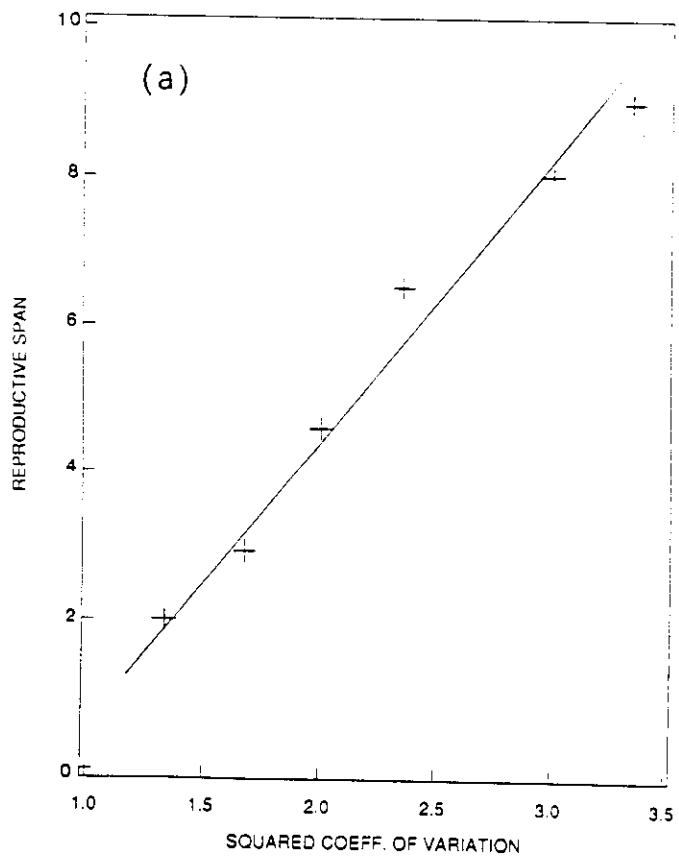

FIG. 9a. Plot of reproductive span against squared coefficient of variation for a fixed a. This theoretical prediction for selectively neutral life histories is based on the $a = 0.001$ curve in Fig. 8 using life histories 1–5. The straight line is an eyeball fit.

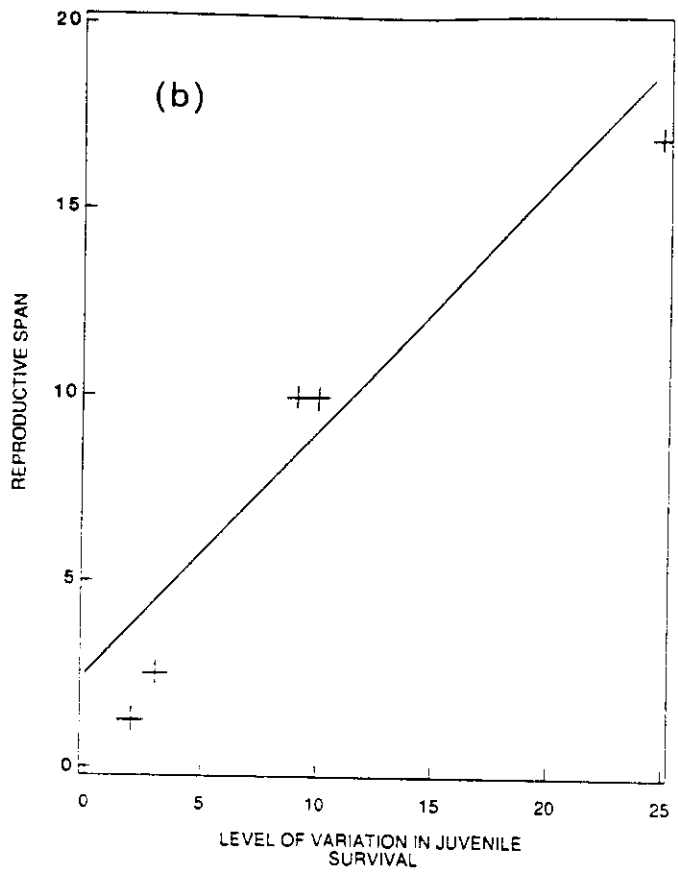

FIG. 9b. Based on Murphy's (1968) Figure 2 for various fish. The vertical axis is a measure of reproductive span in years, and the horizontal axis approximates the level of random variation in survival rate of eggs. There is a strong concordance with Fig. 9a, although the measures of variation are only similar not equivalent. The straight line is an eyeball fit.

in the same vital rates with a common coefficient of variation C. By determining the value of a as a function of C for each life history, one can plot an "indifference curve" which shows values of C at which different life histories in the set have the same value of a. Figure 8 provides an illustration.

These indifference curves have direct application. Consider a set of life histories such as those in Murphy (1968) which are constrained as above and differ mainly in their length of reproductive span. The life histories for which Fig. 8 is computed make up such a set, and in Fig. 9a the indifference curves are used to plot the relationship between reproductive span and C for a family of equally fit life histories. Note that the strong similarity between this plot and the one in Fig. 9b which is based on Murphy's (1968) Figure 2. Clearly, Murphy's conclusion that the spreading-out of reproduction increases with the level of random variation is strongly supported by the results described here.

6.3. *Annuals versus Biennials versus Perennials*

Cole (1954) found little advantage in repeated reproduction as compared with reproducing once; later workers have argued reasons which undo this conclusion. Holgate (1967) examined a stochastic model comparing annuals with biennials and incorporating "demographic" stochasticity. He found that the extinction probability for biennials was usually smaller than that for annuals, assuming an equal r_0 for both. Klinkhammer and de Jong (1983) considered the effects of a random environment which affected the seed set of flowering plants. They compared strict biennials (flowering in the second year of life, then death) with biennials which live for several years with some fraction flowering every year and used simulations to show that delayed flowering (i.e., a flowering fraction less than one) can be advantageous. Roerdink (1987) applied stochastic demographic methods to this problem and his interesting results are sketched below.

His model considers two age classes: plants up to a year old, and plants over a year old. Letting $\mathbf{N}_t$ be the population vector at time t, f (between 0 and 1) be the fraction of plants older than 1 year which flower each year, S_t be the per-capita production of seeds per flowering plant per year, and p the fraction of older plants surviving each year, the random dymanics are described by

$$\mathbf{N}_{t+1} = \begin{pmatrix} 0 & fS_t \\ p & (1-f)S_t \end{pmatrix} \mathbf{N}_t. \tag{6.3.1}$$

Environmental randomness is contained in the series of S_t's; Roerdink assumes this to be an IID random sequence with a gamma distribution. The resulting model is analyzed exactly as in Section 3, and (3.11) is used to compare a exactly. Roerdink's key result is that a INCREASES with f as f increases from 0, and in general has a maximum at a value of f less than 1. Therefore delayed flowering, with some individuals waiting beyond the second year of life to flower, is a life history which can invade a population of annuals or strict biennials.

Note two other features of Roerdink's paper. First, he studies the variance of one-period growth rates (his Eq. (3.9b)) and assumes that it equals the long-run variance σ^2 in population size (2.4.8). This is an error since the latter variance is obtained from many-period growth rates (S. Orzack, personal communication, October 1987); we shall say more about variances in Section 7. The other feature is that he compares the results of approximation (4.2.6) with his exact calculations, and finds that for moderate values of f the approximation is excellent, but at high f it fails to display the maximum that the exact result displays. This is disappointing since the maximum is the biologically interesting thing.

6.4. *Other Questions*

The results outlined above make clear that stochastic demography can be used to attack important biological problems. There are many open questions, several of which can be studied with existing theoretical results: (a) what happens to the arguments of Sections 6.2 and 6.3 when there is serial autocorrelation in the environment? (b) what is the importance of random variation as force selecting for more complex life cycles? Particular examples are the seed pools discussed briefly in Section 2.3.2, and the "escape in time" strategies of insect diapause and dispersal (cf. Section 2.3.3).

Istock (1967) studied complex life cycles in a general setting, considering life histories in which different stages have different ecological requirements. Random environmental effects are expected to be major forces in shaping complex life cycles, examples of which are found in plants, insects, and marine organisms. The theoretical analysis of such life histories remains relatively undeveloped; see Istock (1981) for some interesting directions.

7. Projection and Estimation

Projection and estimation are complementary. To make projections, we need to set confidence intervals of some kind around a point projection; we also need estimates of the model parameters. The central issue is to determine how variance in vital rates works its way into population variance. Two approaches will be considered here: one is to focus on the long run, in which case the key task is to estimate a and σ^2 in order to make total population forecasts; the other is to ask for more detailed forecasts in the shorter run and to use estimates of randomness in vital rates as the inputs.

7.1. *The Long Run: Heyde–Cohen*

Heyde and Cohen (1985) provide estimators for a and σ starting from a time series of counts of total population. Their theory was developed for a closed population but will probably work for certain kinds of immigration patterns as well (Heyde, 1985). The estimator for growth rate is pleasantly simple:

$$\hat{a} = (\log P_T - \log P_1)/(T-1),$$

where the P's are total population counts over a total of T time units.

The variance estimator is more complicated and it may be useful to explain why. From (2.2.1) it follows (see (2.3.9)) that one can write

$$P_T = r_T r_{T-1} \cdots r_2 P_1$$

where r_t is the growth rate at period t and depends on both the state of the environment and the population structure. Now from the general results (2.3.8) about the long-run lognormal distribution of population size it follows that

$$\begin{aligned}\sigma^2 &= \operatorname{Lim}(1/T)\operatorname{Var}\log(P_T/P_0)\\ &= \operatorname{Lim}(1/T)\left\{\sum_x \operatorname{Var}(r_x) + \sum_{x\neq y}\operatorname{Cov}(r_x r_y)\right\}\\ &= \operatorname{Var}(r_t) + \mathrm{COV},\end{aligned}$$

where Var means variance, Cov means covariance, and COV is the limit of the second term in the middle equation. The stationary Var in the last line is the one-period variance computed by Roerdink (1987) (see Section 6.3) as an estimate of σ^2. In a simulation study one can estimate σ by the procedure of generating many strings of r_t's in successive independent simulations, so that an estimate of COV can be obtained. However, when one has only a relatively short series of P's, something different is needed.

Heyde and Cohen provide such an estimator:

$$\begin{aligned}\hat{\sigma} = \frac{1}{2}(\pi/2)^{1/2}\Bigg\{&[\log(T-1)]^{-1}\sum_{j=1}^{T-1} j^{-3/2}\\ &\times|\log P_{j+1} - \log P_1 - j\hat{a}| + [\log(T-2)]^{-1}\\ &\times\sum_{j=1}^{T-2} j^{-3/2}|\log P_{j+2} - \log P_1 - j\hat{a}|\Bigg\}.\end{aligned}$$

This estimator is quite unusual compared with the more common estimates of variance. See Cohen (1986) for an application and comparisons with other methods of generating confidence intervals.

These results are of interest in making long-run forecasts. In practical situations, however, one would like to use recently observed population structure and Lee (1987, personal communication) has pointed out that the asymptotic estimators are probably not optimal in the short to medium term. The Heyde–Cohen estimates provide information only on long-run growth.

7.2. *The Short Run: Variance Decomposition*

Lee (1974) used the procedure of fitting a time series (ARMA, Box and Jenkins, 1970) model to fertility rates and then generating population forecasts based on these fertility models. Time series methods have since been used by MacDonald (1979), Saboia (1977), Lee (1977), and Alho and

Spencer (1985). Tuljapurkar (1987) pointed out that all these methods use an approximation in which the nonlinear demographic model (2.2.2) is linearized. The generation of point forecasts and confidence intervals in the linearized model is relatively straightforward. However, the linear model does not reflect the geometric character of a full projection, in which (2.2.2) is used. Tuljapurkar (1987) discusses differences between the two approaches, but important questions remain unanswered. Here we consider only one result which lies at the heart of the difference between linear and nonlinear models.

Lee's pioneering 1974 paper asserts that a linearization of (2.2.2) might be expected to work because, with random vital rates, the variance in growth rates is dominated by variance in rates as opposed to variance in population structure. Tuljapurkar and Lee (1987, unpublished) used the methods in Section 4.2 to analyze the relative contributions to the variance, with the following results. Recall notation from Section 4.2, in which the time-dependent matrix $\mathbf{X}_t$ of random vital rates is decomposed into an average matrix $\mathbf{A}$ plus a matrix of random deviations $\mathbf{B}_t$. The average matrix determines a vector $\mathbf{u}$ of population structure which would be stable in the absence of random effects. The one-period population growth rate is from (2.3.9),

$$\begin{aligned}\lambda_t &= (\mathbf{e}, \mathbf{X}_{t+1}\mathbf{Y}_t)\\ &= (\mathbf{e}, \mathbf{Au} + \mathbf{A}(\mathbf{Y}_t - \mathbf{u}) + \mathbf{B}_{t+1}\mathbf{u} + \mathbf{B}_{t+1}(\mathbf{Y}_t - \mathbf{u}))\\ &= \lambda_0 + \Delta_A + \Delta_R + \Delta_{AR},\end{aligned} \tag{7.2.1}$$

where the terms in the last line correspond exactly to the terms in the line preceding it. These terms can be interpreted as follows: Δ_A is the average vital rates acting on the random variation in population structure, Δ_R is the random part of vital rates acting on the average population structure, and Δ_{AR} is the interaction between variation in structure and rates. These identifications are not exact (cf. the discussion in Section 4.2.2) but are close enough for the purpose of asking how the two sources of variation compare. Now define the variance in λ_t due to population structure fluctuations as

$$V_A = E\,\Delta_A^2 \tag{7.2.2}$$

and the variance in λ_t due to random rate fluctuations as

$$V_R = E\,\Delta_R^2. \tag{7.2.3}$$

The results of Section 4.2.2 are easily adapted to compute these variances and we can now ask: what is the ratio of V_A to V_R? Tuljapurkar and Lee

(1987) examined the case of a human population typical of the United States in the 1960s, and found that V_A is about $0.7V_R$. This number is computed using the small noise expansions and was verified by simulations. This means that in the situation which Lee (1974) considered, roughly a third of the one-period variance in growth rate was due to changing age structure. The implication is that one ought to do better at short-term forecasts using full information on age structure than without.

The variance (7.2.3) involves the transient behavior determined by the average vital rates. It is known (Coale, 1972) that transients are governed by the width of the next maternity function, and so it is interesting to ask how the ratio (V_A/V_R) changes as the reproductive span is concentrated relative to the age of last reproduction. Consider a series of stylized flat net maternity functions in which the age of last reproduction is fixed at 10, and the age of first reproduction is increased from 2 to 8, holding the net reproductive rate at a fixed value, and allow serially uncorrelated fluctations in fertility at all ages. The ratio (V_A/V_R) is found to increase from 0.65 for age of first reproduction 1 to a value of 0.89 for age of first reproduction 8. Thus, there is a sizeable increase but not a dramatic one.

7.3. *Simulations: From Fur Seals to Social Security*

Theoretical progress on problems arising in projection has been slow. In the absence of usable theory one can use simulation to study particular populations, and an example is the work of Gerrodette *et al.* (1985) on the northern fur seal *Callorhinus ursinus*. Average vital rates for the population in year classes are given by a 23 age-class Leslie matrix, with first reproduction at 3 years. Fecundity has a peak at about 10 years and then falls slowly. Survival rates increase with age till age 9–10 and then fall. All vital rates are allowed to vary stochastically, with estimated coefficients of variation about 0.05. In addition, there is a within-period pairwise correlation between fecundities of 0.9, between survival rates of 0.9, and between fecundities and survival rates of 0.5. There is no serial autocorrelation between rates over time. These authors performed simulations which confirmed the lognormal distribution (2.3.8). They also note that a geometric mean transformation (raising P_t to the power $1/t$) was even more effective as a normalizing transformation for small t. These normalizing transformations were used to generate 95 and 99% confidence intervals for the total female fur-seal population. This kind of effort is necessary for practical short-term projection.

A different projection important for human populations is of particular age-class proportions, and of ratios of numbers in different age classes. Keyfitz (1986) considers old age pensions and social security in the United States, which are very sensitive to the *dependency ratio*: the ratio of the

retired population, say ages 65+, to the working population, say ages 15–64. The usual method of defining a high, low, and median variant of forecast can greatly mislead. As Keyfitz shows by simulation, if we allow rates to vary randomly within the possible range over time, the dependency ratio acquires substantially more variance over time. He estimates approximate confidence regions based on simulations.

For practical application, the problems of this section are very important and open questions abound:

(a) how rapidly do linear forecasting methods fail;

(b) what are good methods for short-term forecasts?

(c) how does one make good predictions of age structure?

8. Management

Harvesting, resource management, conservation, and risk assessment are all "management" issues. In many ways these are prediction problems, and the remarks of Section 7 are relevant. However, most management questions are studied in much greater ignorance; it is often unclear what questions need asking and what predictions will be meaningful. Here we illustrate these problems by describing some interesting cases. As with prediction, many significant questions remain unanswered.

8.1. *The Striped Bass*

The striped bass (*Morone saxatilis*) populations in the Potomac and Hudson Rivers are celebrated in the business of studying environmental impacts. These populations are under considerable fishing pressure and also subject to potentially poor environments caused by power plants which alter the water quality in various ways. There have been several unpublished studies of these populations, at least one of which (Tuljapurkar *et al.*, 1983) used the random rates theory. Two notable published studies by Cohen *et al.* (1983) and Goodyear *et al.* (1985) illustrate some of the issues in applying stochastic demography to "real world" situations.

The first of these develops a stochastic age-structured model for the Potomac fishery. The key notion is that random variation mainly affects egg survival rate and adult survival rate is constant in time. Statistical tests showed that the egg survival rate series is an uncorrelated random sequence but did not yield an unambiguous form for the underlying probability distribution. Observed values were used to generate a discrete distribution, and simulations were run with survival rates drawn from this distribution. Estimates of a were obtained and used to discuss the possible

impacts of environmental factors on the fishery. The results obtained depend in a clearly stated way on estimates of adult survival rates.

The second cited study focuses on the Cheaspeake Bay population and uses the same method to estimate egg survival rates over time. Assuming other vital rates fixed, a replacement survival rate was computed at which the population would just replace itself. There appears to be a clear downward secular trend in egg survival rates to well below the replacement value. An alternative interpretation of the results is that egg survival stayed constant but adult survival fell. In either case the results indicate population decline and suggest the magnitudes of intervention effort required to maintain stocks.

8.2. *The Rocky Mountain Elk*

There is a population of Rocky Mountain Elk, *Cervis elaphus nelsoni*, living essentially unhunted in the Upper Cedar River Watershed near Seattle, Washington. These are descendants of a transplant in 1913 of 50 elk from Yellowstone National Park, and have been studied for some 20 years. Wallace (1986) constructed and applied a random rates model for this population.

The model divided the population into three classes: calves, yearlings, and all adults 2 years older. This lumping of adults was necessary because it is not possible to tell ages apart any finer in the field. Population estimates were used to generate rough estimates of vital rates and it was concluded that calf survival is most variable. A regression study of annual calf survival against precipitation and temperature at different times of year yielded two main environmental indices. No evidence of serial autocorrelation was found in these variables and so a discrete environmental state variable was constructed which had one of 90 possible values with specified probabilities of occurrence. Corresponding estimates of calf survival rates were available from the regression. Just as in Section 8.1, this is an IID random rate model. All other vital rates were estimated and assumed to be constant over time. The resulting model described the population trajectory reasonably well, but indicated that the population might be reaching some kind of plateau. Population projections, confidence intervals, and potential harvest numbers were computed.

8.3. *The Spotted Owl and Extinction Theory*

Simberloff (1987) highlights the recent cause celebre of the Spotted Owl, *Strix occidentalis*, in the Western United States. Simply stated, the owl depends on old forests and there is considerable economic pressure to log

these forests. The conflict between conservation and economics has involved academic ecologists and the full machinery, such as it is, of modern ecological science. The central scientific question is one of extinction probability. It is interesting to quote Simberloff (1987, p. 769): "there is little treatment in the theoretical literature of environmental variation." He also treats demographic stochasticity as completely separate from environmental effects. Clearly there is an awareness gap here. There are also many open problems since a formal general theory of extinction exists in Athreya and Karlin (1971) but application requires considerable further effort.

Interesting studies on extinction have recently been done by Mode and Jacobsen (1987a, b). Their age-structured model has both demographic and environmental stochasticity. They make a technical statistical independence assumption, but it is not clear whether this is a real limitation. They model the random environment by a one-lag or two-lag autoregressive process. Monte Carlo simulation is used to obtain extinction probabilities and also critical population sizes (defined as the smallest population from which extinction occurs with no more than some specified tolerable probability). They find extinction probabilities very sensitive to the amount of environmental randomness and the serial correlation structure of the environment.

An important analytical approach by Lande and Orzack (1988) starts with the lognormal distribution (Sections 2.3, 3, and 3.9) and asks how one can approximate the dynamics of population size P_t by a diffusion process. The difficulty lies in finding a way to incorporate the effect of initial age structure. Lande and Orzack make the crucial observation that differences in initial structure $\mathbf{y}_0$ should be weighted by reproductive value. For small fluctuations they model $\log P_t$ as a diffusion with mean a, variance σ^2, and initial point $\log[(\mathbf{v}_0, \mathbf{y}_0)P_0]$, where $\mathbf{v}_0$ is the (normalized) reproductive value vector for the average Leslie matrix $\mathbf{A}$. This model is in excellent agreement with simulations. Their striking results do not incorporate serially correlated environments or demographic stochasticity. It seems clear that extinction probabilities need further study and that conservation biology will benefit from it.

9. Epilog

This paper has presented many questions and a few, mostly incomplete, answers. These questions range from very abstract to rather concrete, but all promise to yield better insights into the biological world.

The deepest insights from this theory are into the temporal structure of life histories as an arena for natural selection. We have seen (Sections 5 and

6) the many ways in which organisms can achieve a "fit" to a varying environment. There is a hierarchy of adaptative possibilities, from the average distribution of reproduction and survival over the life course to the ways in which the environment is filtered into variation, covariation, and serial autocorrelation of vital rates. The results strongly suggest that such adaptation is the key evolutionary force underlying many complex life cycles, and such phenomena as diapause, dispersal, and seed pools are ripe for further exploration.

At a more immediately applicable level, this theory yields novel insights into the dynamics of real populations. The notion of a population as being a time-varying, dynamic, yet statistically equilibrial state is very different from the notions of classical demography. It is also the consequence of the unpredictable and variable vital rates which we might expect in the real world. Thus we should expect as a matter of course that: population structure tracks environment with the recent past writ large; the long-term average of population structure is not as sensitive to runs in environmental conditions; populations will display oscillatory behavior, including the temporal dominance of cohorts. Many other conclusions from the analysis of Section 4 are central to the analysis of observed population and the prediction of future population.

Finally, there is interesting related work on disordered systems as diverse as alloys, glasses and neural nets. See Ziman (1979), Demetrius (1987) for physical examples, and Cohen *et al.* (1986) for a wider discussion.

Appendix

Appendix to Section 4.1

Label the age classes 1 through k, where $k = \{$last age class which is reproductive$\}$, and describe population structure by the variables $Z(i)$ defined as

$$\begin{aligned} Z_t(i) &= N_t(i)/N_t(1), \\ \mathbf{Z}_t &= (Z_t(2), \ldots, Z_t(k)). \end{aligned} \tag{A1}$$

The basic equation (2.2.1) now becomes

$$\begin{aligned} Z_{t+1}(i+1) &= S_{t+1}(i)\, Z_t(i)/(\mathbf{M}_{t+1}, \mathbf{W}_t), \qquad i > 1, \\ &= S_{t+1}(i)\, Z_t(i)\, h(\mathbf{M}_{t+1}, \mathbf{Z}_t), \end{aligned} \tag{A2}$$

where $S_t(i) = (\mathbf{X}_t)_{i+1,i}$, $M_t(i) = (\mathbf{X}_t)_{1i}$, the vectors are $\mathbf{S} = (S(i))$, $\mathbf{M} =$

$(M(i))$, $\mathbf{W}=(1, \mathbf{Z})$, and the function $h(\mathbf{m}, \mathbf{z})=1/(\mathbf{m}, \mathbf{z})$. Suppose that the S's and M's are bounded for all t,

$$\mathbf{0}<\mathbf{s}_1 \leqslant \mathbf{S}_t \leqslant \mathbf{s}_2<\mathbf{1}, \qquad \mathbf{0} \leqslant \mathbf{m}_1 \leqslant \mathbf{M}_t \leqslant \mathbf{m}_2<\infty. \tag{A3}$$

Here and elsewhere inequalities between vectors are shorthand for inequalities which hold componentwise; similarly for matrices.

To find bounds on age structure resulting from the bounds on vital rates, observe that h in (A2) is a decreasing function of $\mathbf{Z}$, and also that

$$h_1(\mathbf{Z})=h(\mathbf{m}_1, \mathbf{Z}) \geqslant h(\mathbf{M}_t, \mathbf{Z}) \geqslant h(\mathbf{m}_2, \mathbf{Z})=h_2(\mathbf{Z}). \tag{A4}$$

Starting with any initial nonzero $\mathbf{Z}_0$ in (A2) such that $\mathbf{0} \leqslant \mathbf{Z}_0<\infty$, generate upper bounds as

$$Z_1(2) \leqslant s_2(1)\, h_1(0)=c_1(2),$$
$$Z_2(3) \leqslant s_2(2)\, c_1(2)\, h_1(0)=c_1(3),$$

and so on to get a complete vector of upper bounds, $\mathbf{c}_1 \geqslant \mathbf{Z}_k$. These upper bounds can be used to generate a set of lower bounds,

$$Z_1(2) \geqslant s_2(1)\, h_2(c_1)=b_1(2),$$
$$Z_2(3) \geqslant s_2(2)\, h_2(c_1)=b_1(3),$$

and so on to get a vector $\mathbf{b}_1 \leqslant \mathbf{Z}_k$. We can now iterate this procedure to generate a sequence of upper bounds $\mathbf{c}_m$ and lower bounds $\mathbf{b}_m$, $m=1, 2, \ldots$, using the mappings

$$\begin{aligned} c_{m+1}(i+1) &= s_2(i)\, c_{m+1}(i)\, h_1(\mathbf{b}_m), \\ b_{m+1}(i+1) &= s_1(i)\, b_{m+1}(i)\, h_2(\mathbf{c}_m), \end{aligned} \tag{A5}$$

where we define $b_m(1)=c_m(1)$ for all m, and $i=1, \ldots, k-1$. It is easy to see that $\mathbf{c}_{m+1} \leqslant \mathbf{c}_m$, and $\mathbf{b}_{m+1} \geqslant \mathbf{b}$. Thus the bounds converge to a limit which can be computed by solving for an equilibrium in (A5). Specifically we look for $\mathbf{c}$ and $\mathbf{b}$ which reproduce themselves under the mappings given above. A little algebra shows that if we set

$$t=h_1(\mathbf{b}),$$

$$l_j(i)=\begin{cases} 1 & \text{if } i=1, \\ s_j(1)\, s_j(2) \cdots s_j(i-1) & \text{if } i>1, \end{cases} \qquad \text{for } j=1, 2,$$

$$Q(t)=\sum_{x=1}^{k} l_2(x)\, m_2(x)\, t^x,$$

then we can find t as the root of the polynomial equation

$$\sum_{x=1}^{k} l_1(x)\, m_1(x)\, t^x Q^{-x-1} = 1, \tag{A6}$$

In simple cases this equation yields a nice interpretation in terms of iterated products of matrices (cf. Tuljapurkar, 1984).

Appendix to Section 4.2.2

The average population structure (see Eq. (4.2.11)) is

$$E\mathbf{Y}_t \cong \mathbf{u} + (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})(E\mathbf{R}_{2t} - E\{\mathbf{R}_{1t}(\mathbf{e}, \mathbf{R}_{1t})\}). \tag{A7}$$

Taking the limit as $t \to \infty$ yields the steady state average

$$E\mathbf{Y}_t \simeq \mathbf{u} + (1 - \mathbf{Q})^{-1} E\left\{\sum_{l=1}^{\infty} (\mathbf{B}_{l+1}/\lambda)(1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\, \mathbf{Q}^{l-1}(\mathbf{B}_1/\lambda)\right\}\mathbf{u}$$

$$- \sum_{l=1}^{\infty} \mathbf{Q}^{l-1} E\{(\mathbf{B}_1/\lambda)(\mathbf{A}/\lambda)^{l-1}(\mathbf{B}_1/\lambda)\}\,\mathbf{u}. \tag{A8}$$

The variance of $\mathbf{Y}_t$ in (4.2.12) depends on

$$\mathbf{R}_1^* = \lim_{t \to \infty} E[(1 - \mathbf{P})\,\mathbf{R}_{1t} \otimes (1 - \mathbf{P})\,\mathbf{R}_{1t}]$$

$$= \Big\{(1 - \mathbf{P}) \otimes (1 - \mathbf{P})\,\mathbf{S}_0$$

$$+ \mathbf{Q} \otimes \mathbf{Q}\{1 - \mathbf{Q} \otimes \mathbf{Q}\}^{-1}\mathbf{S}_0$$

$$+ \sum_{j=1}^{\infty} [(1 - \mathbf{P}) \otimes \mathbf{Q}^j \mathbf{S}_j + \mathbf{Q}^j \otimes (1 - \mathbf{P})\,\mathbf{S}_{-j}]$$

$$+ \sum_{j=2}^{\infty} \mathbf{Q}^j \otimes \sum_{i=1}^{j-1} [\mathbf{S}_i + \mathbf{S}_{-i}]\Big\}(\mathbf{u} \otimes \mathbf{u}). \tag{A9}$$

The above expressions simplify greatly in the IID case when $\mathbf{S}_m = 0$ for $m \neq 0$. The general small noise expression for the autocorrelation function of population structure is

$$\mathbf{C}(m) \cong (1 - \mathbf{L} \otimes \mathbf{L})^{-1}\Big\{(1 - \mathbf{u}\mathbf{e}^{\mathrm{T}}) \otimes (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\,\mathbf{S}_m$$

$$+ E \sum_{j=0}^{m-1} [(1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\,\mathbf{Q}^{m-j}\mathbf{B}_{j+1}] \otimes \mathbf{B}_1\Big\}(\mathbf{u} \otimes \mathbf{u}), \tag{A10}$$

with $\mathbf{L} = (1 - \mathbf{u}\mathbf{e}^{\mathrm{T}})\,\mathbf{Q}$ and $m \geqslant 0$.

Appendix to Section 4.5.4

Use the scaled variables

$$Z_t(i) = N_t(i)/N_t(1)$$

of Section 4.1, and recall Eqs. (A2)–(A3) and the corresponding notations. The one-period growth rate is obtained by noting that

$$(\mathbf{e}, \mathbf{N}_{t+1}) = (\mathbf{M}_{t+1}, \mathbf{N}_t) + \sum_{i \geqslant 1} S_{t+1}(i)\, N_t(i) \tag{A11}$$

so that

$$\begin{aligned}(\mathbf{e}, \mathbf{N}_{t+1})/(\mathbf{e}, \mathbf{N}_t) &= \left\{(\mathbf{M}_{t+1}, \mathbf{W}_t) + \sum_{i \geqslant 1} S_{t+1}(i)\, Z_t(i)\right\} \Big/ (\mathbf{e}, \mathbf{W}_t) \\ &= (\mathbf{M}_{t+1}, \mathbf{W}_t)\{(\mathbf{e}, \mathbf{W}_{t+1})/(\mathbf{e}, \mathbf{W}_t)\}. \end{aligned} \tag{A12}$$

Now from (4.1.5) for $i = 2$ one has $(\mathbf{M}_{t+1}, \mathbf{W}_t) = S_{t+1}(1)/Z_{t+1}(2)$ and so the logarithmic growth rate becomes

$$\begin{aligned} a &= E \log\{(\mathbf{e}, \mathbf{N}_{t+1})/(\mathbf{e}, \mathbf{N}_t)\} \\ &= E \log S_{t+1}(1) - E \log Z_{t+1}(2) \\ &\quad + E \log(\mathbf{e}, \mathbf{W}_{t+1}) - E \log(\mathbf{e}, \mathbf{W}_t). \end{aligned} \tag{A13}$$

Stationarity implies that the last two terms will cancel and so finally we get (4.5.7).

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