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3

From the Individual to the Population in Demographic Models

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ABSTRACT. **Demographic population models are derived from descriptions of how individuals move through their life cycles. In this sense all demographic models are based on information about individuals. Individuals are described in terms of their *i*-states (sensu Metz and Diekmann), which provide the information necessary to specify the response of an individual to its environment. Age, size, and physiological state are typical *i*-state variables. The state of the population, or *p*-state, is derived from the *i*-states. If all individuals experience the same environment (we refer to this condition as mixing), the *p*-state is a distribution function over the set of *i*-states. Most demographic models assume this condition; we call these models *i*-state distribution models. When the mixing assumption fails, for example due to local interactions among individuals, each individual must be followed; we call these models *i*-state configuration models. We use this framework to examine examples of the relations between the individual and the population in demography, including multi-type branching processes and stable population theory, micro-simulations of reproduction and family structure, epidemic models and percolation theory, and hazard analysis.**

Introduction

The use of "individual-based models" (we will propose a different nomenclature in what follows) was urged by Huston et al. (1988) in a paper which led directly to this symposium. They claimed that most ecological models make assumptions that contradict two important properties of organisms. First, grouping individuals into categories violates the principle of the uniqueness of the individual. Second, by not distinguishing among the

locations of individuals, the models violate the principle that interactions are inherently local.

The alternative that they propose is to use models based on explicit representation of individual organisms. Such models must be studied by numerical simulation, but recent increases in available computer power have made them more practical than they have ever been before. Huston et al. (1988) claim that such models escape some of the simplifying assumptions made by other ecological models. They can also be constructed directly from data on individuals, data which are routinely collected in studies of individual physiology and behavior.

In this paper we examine the special case of demographic population models. Some of the claims of Huston et al. (1988) underestimate the capabilities of traditional ecological and demographic models, and the extent to which they are already based on information about individuals. However, we will argue that the explicit inclusion of individual organisms is sometimes necessary, and more often convenient, and that such models can play an important role in theoretical ecology.

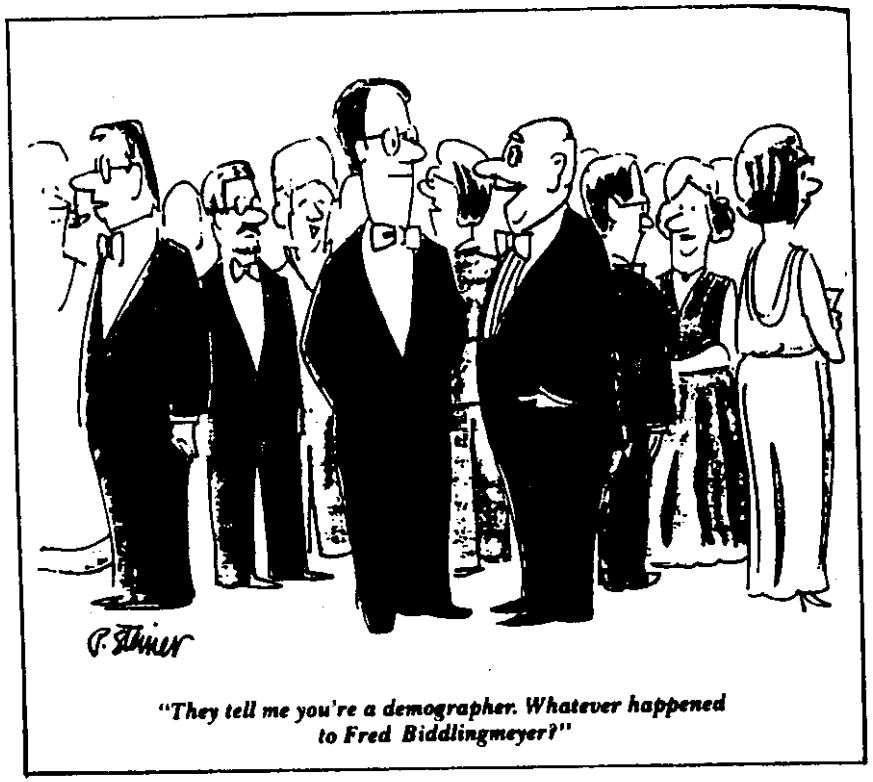
State in Population Models

Demography[1] is an approach to the study of populations which is based on the analysis of individuals. The word "demography" is derived from the Greek, loosely "writing about individuals," and all demographic models are, in a sense, individual-based models.

The simplest way to account for individuals is simply to count them, and the simplest population models are written in terms of the crude numbers of individuals and the crude rates by which those numbers change through time. But such models can have only limited success, and demographic models acknowledge that individuals differ among themselves in many important characteristics as they move through their life cycles. These differences are summarized in the state of the individuals.

It is impossible to predict the response of all but the very simplest natural systems from knowledge of current environmental stimuli alone. The problem is that the past of the system affects its response in the present. The state of a system is that information necessary to account for this history and determine, in combination with the present environment, the

[1]We use the word demography to identify an approach—one based on the characteristics of individuals—and not a subject species. Thus we will refer to "human demography" to denote that species-specific branch of the discipline concerned with a certain large primate. This usage will appear strange to some practitioners of that sub-discipline, but quite natural to demographers sensu lato.



"They tell me you're a demographer. Whatever happened to Fred Biddlingmeyer?"

Figure 3.1. Cartoon by Peter Steiner, reprinted with permission.

system's behavior[2] (Zadeh 1969, Caswell et al. 1972, Metz and Diekmann 1986). Begin with the individual. Following Metz and Diekmann (1986), we define the state of an individual (the *i*-state) as the information needed to specify the response of the individual to its environment. Examples of *i*-state variables include age, size, marital status, instar, employment status, hunger, parity, lipid concentration, and geographical location.

The *i*-state encapsulates the information necessary to predict the individual's behavior. To move up to the level of the population requires a population state, the *p*-state of Metz and Diekmann. Since a population is a collection of individuals moving through their life cycles, responding to and influencing their environments, and because the additions to and losses from

[2]In deterministic systems it is the behavior that must be determined, in stochastic systems the probability distribution of behavior. The concepts are fundamentally similar in the two cases.

a population come from the reproduction, mortality, immigration and emigration of the individuals that comprise it, we can hope to derive the *p*-state from the information about *i*-state dynamics.[3]

The most direct and comprehensive way to construct a *p*-state is to recognize that a population is a system of interacting individuals and that the state of the population can thus be determined from the *i*-states of the individuals and a constraint function (Caswell et al. 1972) specifying the interactions among individuals. For reasons that will become clear later, we refer to this *p*-state as an *i*-state configuration. Consider, for example, a population of trees, where the *i*-state of a tree consists of its size and vigor. The *i*-state, together with the individual's environment (say, available light and water), provides all the information available on mortality, growth, and changes in vigor. An *i*-state configuration for a population of trees is a list giving the size and vigor of each individual, plus a constraint function specifying the environment of each individual as a function of the *i*-states of the individuals with which it interacts. If interactions are determined by spatial proximity, the *i*-state configuration would consist of the *i*-states and the spatial locations of each individual.

An important simplification occurs when all individuals in the population experience the same environment (we will refer to this as mixing). In this case, all individuals with the same *i*-state will have the same dynamics and can be treated collectively. The response of the population can then be predicted from the number of individuals in each *i*-state; we refer to this *p*-state variable as an *i*-state distribution. Given the assumption of mixing, age as an *i*-state leads to the age distribution as a *p*-state; size leads to the size distribution, and so on.

The *i*-state configuration contains much more information than the *i*-state distribution. Suppose individuals are grouped into discrete age classes 1, 2, 3, ..., k. The *i*-state distribution (the familiar age distribution) is given by the k-vector $\boldsymbol{n}$, where n_i is the abundance of the ith age class. The state space is thus k-dimensional Euclidean space R^k (or perhaps the nonnegative cone of R^k). But suppose that the *p*-state is a configuration of age-classified individuals in two-dimensional space. The *i*-state configuration for N individuals is then a function mapping R^2 (the spatial coordinates of the individuals) to $\{1, 2, \ldots, k\}$ (the set of all *i*-states). The *p*-state space is the set of all possible such mappings, for all values of N (cf. Metz this volume).

[3]We suppose that the behaviors of interest at the population level can be specified in terms of the *i*-states. This may not be the case. Suppose that age is an adequate *i*-state variable for some species, and that population biomass is the response variable of interest. Age is an adequate *p*-state variable only if individual size is determined by age. If it is not, information on size must be included in the population model even though it is not strictly necessary at the individual level (Metz and Diekmann 1986).

The i-state distribution can be obtained from the i-state configuration, but not vice versa.

Construction of a population model requires a function specifying the dynamics of the p-state as determined by the current p-state and the environment. There are three main mathematical frameworks for such models. If the p-states and time are both discrete, the dynamics are modeled with population projection matrices (Caswell 1989). If the p-states are discrete but time is continuous, delay-differential equations are used (Nisbet and Gurney 1982). Finally, if both the p-states and time are continuous, the models are formulated as partial differential equations (Metz and Diekmann 1986). For each of these mathematical formulations, there are well-developed procedures for deriving the p-state population models from the i-state descriptions and powerful mathematical tools (differential equations, matrix algebra, stability theory, etc.) available for their analysis.

These models are what Huston et al. (1988) call "state variable," as opposed to "individual-based" models. The important distinction, however, is not between "individual-based" and "state-variable" models, because any population model must be based on a population state variable. And all demographic models are based, one way or another, on information about individuals. Instead, the important distinction is between two ways of obtaining population states from individual states—one using the complete individual configuration, the other reducing that configuration to a distribution function. The relation between our terminology and that of Huston et al. (1988) is:

This paper	Huston et al. (1988)
i-state configuration models	individual-based models
i-state distribution models	state variable models

The Individual in Demographic Models: Some Examples

We turn now to some examples, in no sense exhaustive, of demographic models and data analyses which display the transition from the individual to the population.

Branching Processes and Stable Population Theory

Stable population theory is used to project the population consequences of patterns of age- or stage-specific vital rates. Matrix population models (Leslie 1945, Caswell 1989) are one familiar version; a square nonnegative matrix $\boldsymbol{A}$ is used to project a stage-distribution vector $\boldsymbol{n}$ according to

$$\boldsymbol{n}(t+1) = \boldsymbol{A}\boldsymbol{n}(t) \tag{1}$$

The matrix entries a_{ij} can be obtained from a directed graph representation

of the movement of individuals through the stages of the life cycle. Once this individual information is incorporated into a matrix, it can be used to calculate the asymptotic rate of population growth, the stable population structure, reproductive value, transient behavior, etc. The entries of $\boldsymbol{A}$ can be made time-varying, stochastic, or density-dependent, albeit at the cost of increased analytical difficulty.

Equation (1) is an archetypal i-state distribution model; the matrix $\boldsymbol{A}$ explicitly maps the stage distribution $\boldsymbol{n}$ from t to $t+1$. A closer look, however, reveals the i-state configuration model, in which each individual is followed, from which it is derived; such a model is called a multi-type branching process.

The simplest branching process model considers only one type of individual. It was one of the earliest stochastic population models, introduced over a century ago to study a problem in conservation biology—the extinction of surnames of English families (Watson and Galton 1874; for reviews of branching processes see Harris 1963, Athreya and Ney 1972).

Consider a semelparous organism with fixed generation length (this reduces the complexity of the i-state space to a minimum). All individuals are identical, and at reproduction each produces offspring, independently of the others, according to the probability distribution

$$P[\text{producing } k \text{ offspring}] = p_k \quad k = 0, 1, 2, \ldots \tag{2}$$

It would be easy to develop and program an individual-based simulation model of this population using the i-state configuration. In fact, what is now known as the Monte Carlo simulation method, which is precisely an i-state configuration simulation approach, was developed by Ulam and Von Neumann at Los Alamos in 1947 to study branching process models for the demography of neutrons—a situation in which the question of population explosion is literal, not figurative (Eckhardt 1989).

Standard treatments of branching processes begin by showing that, given the stated assumptions, the sequence of population sizes $N(t)$, $t = 0, 1, 2, \ldots$ is a Markov chain. That is,

$$P[N_{t+1} = k | N_t, N_{t-1}, \ldots, N_0] = P[N_{t+1} = k | N_t] \tag{3}$$

In other words, knowing N_t provides all the information there is to be known about N_{t+1}. This shows that the i-state distribution N_t is a p-state variable for this process.

N_t is a random variable. Its stochastic dynamics depend on the distribution of offspring number. Let $f(s)$ be the probability generating function of the distribution p_k:

$$f(s) = \sum_0^\infty p_k s^k, \quad |s| \le 1 \tag{4}$$

where s is a complex variable. Let $g_t(s)$ denote the probability generating function of N_t. This generating function satisfies a recursive relationship

$$g_t(s) = g_{t-1}(f(s)) \tag{5}$$

Given an initial condition N_0, (5) can be iterated to find the probability distribution of N_t for all t. The derivation of (5) involves writing the offspring produced at time t as a sum $\xi_1 + \xi_2 + \ldots + \xi_{N_t}$, where ξ_i is the number of offspring produced by the ith individual present at t. The crucial step is the assumption that the ξ_i are independently and identically distributed random variables from the distribution p_k. This is, in this case, the operational version of the mixing condition—that each individual experiences the same conditions as every other.

This approach can be generalized to time-varying environments, in which the p_i change with time, and to nonlinear models, in which the p_i are functions of N_t; in these cases the recursion relation (5) becomes a time-varying or nonlinear relation.

Explicit calculation of the generating function $g_t(s)$ by solving (5) is generally impossible. However, the expected population size can be found easily. Suppose that $f(s)$ does not change with time. Then

$$E[N_t] = g_t'(1) \tag{6}$$
$$= (f'(1))^t \tag{7}$$
$$= (R_0)^t \tag{8}$$

where R_0 is the mean number of offspring produced per individual and the primes denote differentiation with respect to s. Thus a constant environment produces exponential growth of the mean population at a rate given by the net reproductive rate. The extinction probability can also be calculated (Harris 1963, Athreya and Ney 1972), which is what Galton wanted in the first place.

Now consider the multi-type case, where the individuals are not identical. Instead, any finite number of types may be distinguished. The demography of an individual of type j is specified by a multivariate probability distribution $p^{(j)}(r_1, r_2, \ldots, r_k)$ which gives the probability that an individual of type j produces r_1 offspring of type 1, r_2 offspring of type 2, ..., and r_k offspring of type k. (The production of "offspring" may be the result of reproduction, of growth, of survival in the same state, etc.) All individuals are assumed to behave independently. The simplest assumption is that the probabilities $p^{(j)}$ are time-homogeneous, although this can be relaxed.

Again, it is not difficult to imagine an individual-based simulation of this population. Such a simulation is not necessary, however, since the properties of the population can be analyzed by straightforward generalizations

of the analysis for the single-type process (although the notation does become more cumbersome). In particular, the sequence $\boldsymbol{n}(t)$, $t = 0, 1, 2, \ldots$, where $\boldsymbol{n}$ is a vector giving the number of individuals of each type, can be shown to be a Markov chain. Thus the distribution of individuals into the different types is a p-state corresponding to the i-state model specified by the $p^{(j)}$. In terms of the probability generating functions

$$f^{(j)}(\boldsymbol{s}) = \sum_{r_1} \sum_{r_2} \cdots \sum_{r_k} p^{(j)}(r_1, r_2, \ldots, r_k) s_1^{r_1} s_2^{r_2} \cdots s_k^{r_k} \tag{9}$$

the mean process is given by

$$\mathrm{E}[\boldsymbol{n}(t+1) | \boldsymbol{n}(t)] = \boldsymbol{A}\boldsymbol{n}(t) \tag{10}$$

where the entries of the matrix $\boldsymbol{A}$ are the means

$$a_{ij} = \left. \frac{\partial f^{(j)}}{\partial s_i} \right|_{s=(1,\ldots,1)} \tag{11}$$

Equation (10) is the familiar population projection matrix model (1); we have identified it as the expected value operator for a multi-type branching process.[4] The life cycle graph approach to constructing projection matrices (Hubbell and Werner 1979, Caswell 1982, 1989) is a simple way to specify directly the mean transition rates in (10).

Thus, lurking behind the well-known population projection models of demography is a family of stochastic descriptions of individuals. Given the mixing assumptions, those individual descriptions translate directly into the familiar i-state distributions without loss of information.

Micro-Simulation in Human Demography

In this section, we consider a tradition in human demography which uses explicit i-state configuration models, rather than relying on the reduction of these models to i-state distribution form. The emphasis in human demography since World War II has been on i-state transitions, following individuals through the life cycle (birth, sexual maturity, marriage, widowhood, migration, death), or through portions of the life cycle such as the female reproductive period (fecund, pregnant, post-partum infecundable). These studies are generally referred to as "micro-demography." They often involve

[4] A more elaborate and sophisticated treatment of these issues has been produced by Mode (1985) using age-dependent multi-type branching processes.

drawing population inferences from detailed data on individuals. Some micro-demographic models are merely particularly detailed *i*-state distribution models (e.g., Keyfitz 1977, Sheps and Menken 1973, Bongaarts 1977, John 1990), but human demographers have also made extensive use of *i*-state configuration models, which they refer to as micro-simulation models.

Since their introduction in the 1960's (Sheps 1967, Perrin 1967), micro-simulation models have been used extensively in the study of human reproduction and marriage. In these studies, reproductive histories of individual women are generated according to distribution functions defining transition probabilities between *i*-states. Once the individual reproductive histories have been generated, aggregation of the "data" yields the descriptive reproductive measures for cohorts (e.g., age specific or parity-specific fertility rates) and for the population (e.g., the total fertility rate or net reproduction rate).

> The actual simulation procedure for this model, written for the IBM-7094 computer, consists, of course, of putting an individual woman through the process for a given number of months, randomly selecting at the proper point and according to the specified distributions both the state to which the individual will pass next and the length of stay in that state. This process is repeated for a specified number of women . . . The output for the simulated cohort of females is designed to provide the usual information of demographic interest, together with some optional features. (Perrin 1967, p. 139)

REPMOD (Bongaarts 1977, Bongaarts and Potter 1983) is an example of a human reproduction simulation model, in which the possible *i*-states for a woman in a reproductive union are: fecundable, pregnant, post-partum infecundable, and sterile, possibly in combination with age and lactational history (Figure 3.2). The model is a semi-Markov process, in which the dynamics of the *i*-state distribution are determined by the waiting time distributions for each state transition. These distributions may be based on model output (e.g., the age at marriage is determined by the Coale (1971) marriage model), or hypothesized distribution functions (e.g., the waiting time to conceive has a geometric distribution, the duration of post-partum infecundability has a Pascal distribution, and the distribution of post-abortion infecundability has a geometric distribution). The addition of heterogeneity is straightforward: for example, the distribution of the waiting time in the fecund state may be heterogeneous with respect to woman's age and lactational history.

The output of REPMOD includes age-specific birth rates, birth interval distributions, and parity distributions. Since these rates and distributions are typical of the sort of data which can be collected from real populations, comparison of model aggregate output with data serves as an indication of how well the underlying (and often unmeasurable) individual processes are

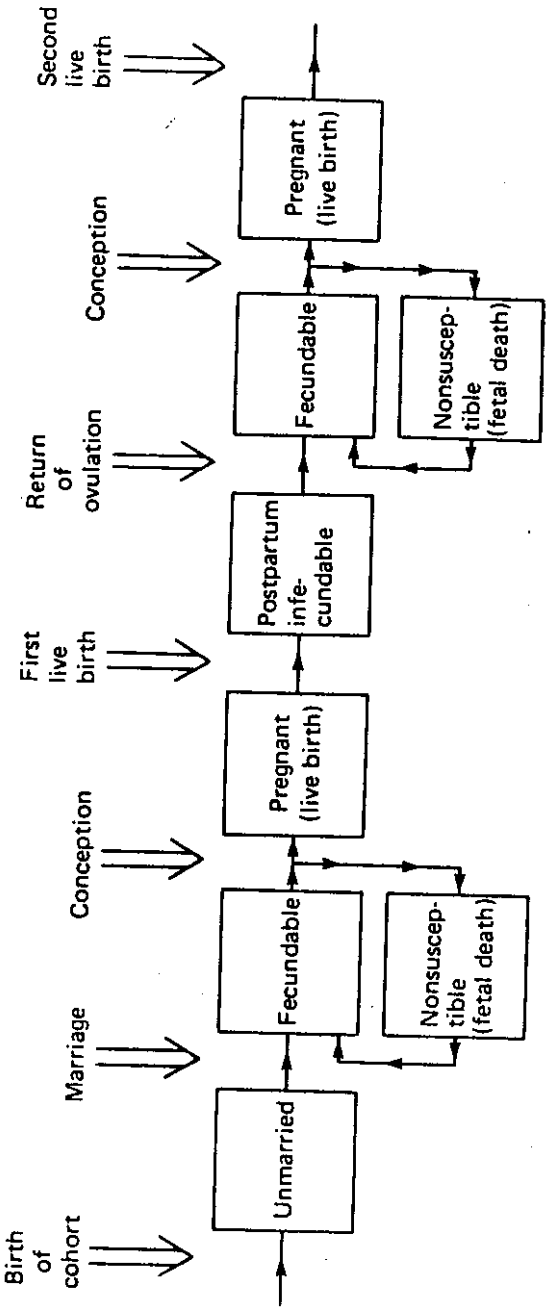


Figure 3.2. An *i*-state transition diagram for human reproduction from the micro-simulation model REPMOD (Bongaarts and Potter 1983).

captured by the model. A well-behaved micro-simulation model also permits the testing of the impact of individual behavioral variation on population aggregates; models of this sort have proven useful in attempts to predict the effect of changing age patterns of marriage or family building patterns (number and timing of births) on human reproduction patterns at the population level. For example, such models have demonstrated that China can achieve the same long-run target levels of population growth either by maintaining a one-child per couple policy, or by adopting a two-child policy with the mother's age at first birth relatively late and the children widely spaced, so that the mean generation length is increased.

Individuals in REPMOD proceed through their *i*-states without interaction. Thus the mixing condition is satisfied and the model can be analyzed as an *i*-state distribution model. In fact, the REPMOD simulation programs are written to permit either mode of analysis.

When interactions are important, individuals in the same *i*-state may experience different conditions, depending on their interactions. In such cases, micro-simulation models must use the complete *i*-state configuration. For example, Hammel et al. (1979) analyzed the demographic consequences of incest tabus in small human populations. They hypothesized that restricting marriage because of relatedness might significantly limit fertility, and that the effect at the population level should vary with population size. To test this hypothesis, they used a micro-simulation package (SOCSIM) in which individuals were characterized by age and sex and in which genealogical information was recorded to permit evaluation of relatedness. Marriages were generated in the model only between individuals eligible to marry under the incest rules being evaluated. Several hundred replicate simulations were performed for each of several population sizes and four patterns of incest tabu. The results showed that incest tabus could significantly restrict marital fertility, and that the effect decreased rapidly as population size increased.

MacCluer and Dyke (1976) used a similar approach to study the minimum viable size of human populations. Their model followed individual men and women characterized by age and relatedness. Marriages between siblings and between parents and offspring were prohibited, and restrictions were placed on the age differences between mates. Three mating patterns (monogamy, polygamy, monogamy with restrictions on first cousin marriage) and two levels of fertility and mortality were used. Populations were simulated for 200 years from each of several initial population sizes. Over this time scale, the minimum viable population size appeared to be fewer than three hundred, and perhaps fewer than one hundred individuals.

Epidemic Models

In models of aerogenically transmitted infectious disease, individuals move through a series of epidemiologic states (*i*-states) of variable duration. The distribution of individuals among these states at any point in time gives the *i*-state distribution. The dynamics of this distribution are derived from hypotheses about the rates of movement of individuals among the *i*-states.

In the simplest deterministic model (Kermack and McKendrick 1927, Hethcote 1989) the host population is divided into three classes: susceptible (S), infectious (I), and recovered, hence immune (R). The host population is assumed constant, N, so at all times $S(t) + I(t) + R(t) = N$. If b denotes the crude birth rate and μ denotes the crude death rate, then bN gives total births and μN gives total deaths. Since the population is stationary, the expectation of life at birth is $1/\mu$. These relationships constitute the demographic elements of the standard epidemiologic model.

It is usually assumed that the disease is propagated through a "catalytic" or "mass action" transmission process in which infections are acquired at a rate proportional to the number of encounters between susceptible and infectious individuals. The rate at which individuals pass from the infected class to the recovered class is constant and denoted γ. The model is written as a system of differential equations:

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu N - \mu S(t) - \beta S(t)I(t) \\
\frac{dI(t)}{dt} &= \beta S(t)I(t) - (\mu + \gamma)I(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t).
\end{aligned} \tag{12}$$

Summing the three equations yields $dN/dt = 0$, satisfying the assumption that population size is constant.

This simple model is typical of models of infectious disease transmission dynamics for developed countries where the assumptions of constant population size and negligible mortality until old age are credible, particularly if one is considering epidemics which occur on a shorter time scale than that required for significant changes in a slowly growing population.

The *i*-states in this model include no demographic detail. Expanding the *i*-state to include age, sex, or other demographic variables is an important problem. One approach (John 1990a,b, Tuljapurkar and John 1991) incorporates a demographic process and an epidemiologic process connected by a pair of link functions. The demographic system governs the dynamics of the age distribution $N(a, t)$, while the epidemiologic system governs transitions of individuals among the epidemiologic classes at each age: $S(a, t)$, $I(a, t)$ and $R(a, t)$. Thus the p-state of the model is a joint distribution of individuals among age and epidemiologic classes. In the absence of disease in the population, the simulation model reduces to a population projection model based on a Leslie matrix.

Analytical solution of these demographic-epidemiologic systems for the equilibrium population structure and the equilibrium force of infection is difficult. In addition, perturbation results for the effects of changes in the demography (i.e., the fertility or mortality schedules) or the epidemiology (e.g., changes in immunization schedules) cannot readily be found. Given these analytical difficulties, *i*-state configuration simulations might be an effective approach to the study of these complex models. Such simulations have been used by Ackerman et al. (1984) to study structurally simpler models of polio and influenza.

A second extension of the basic epidemiologic models is to situations in which infection spreads only to neighboring individuals. In such case *i*-state configuration models are a necessity, not a convenience, because the assumption that all individuals in a given *i*-state experience the same environment is obviously violated. Introduced by Bailey (1967) and Mollison (1977), and termed variously "contact epidemic processes" or "general epidemic models," these models have received more attention from mathematicians and physicists (e.g., Grassberger 1983, MacKay and Jan 1984, von Niessen and Blumen 1986, Durrett 1988, 1989) than from ecologists or epidemiologists.

Imagine an infinite d-dimensional integer lattice Z^d, at each point of which is an individual which may be healthy and susceptible (S), infected (I), or recovered (R). Let x denote a point on the lattice, let $\xi_t(x)$ denote the state of the individual at point x at time t, let k denote the number of immediate neighbors of x that are infected and let p denote the probability of transmitting the infection to a healthy neighbor. The dynamics are simple:

$$\begin{aligned} &\xi_t(x) = R \Rightarrow \xi_{t+1}(x) = R \\ &\xi_t(x) = I \Rightarrow \xi_{t+1}(x) = R \\ &\xi_t(x) = S \Rightarrow \begin{cases} P[\xi_{t+1}(x) = S|\xi_t(x)] = (1-p)^k \\ P[\xi_{t+1}(x) = I|\xi_t(x)] = 1-(1-p)^k \end{cases} \end{aligned} \tag{13}$$

This formulation assumes that infection lasts for only a single time step and that there is no latent period; both assumptions may be relaxed, as may be the assumption of discrete time (Durrett 1989). In any case, Eqs. (13) is a stochastic, spatially localized version of the deterministic model (12).

The states of individuals in this model are clearly the same as those for the individuals in Eqs. (12). The p-state, however, is no longer the vector $[S(t), I(t), R(t)]$. Instead it is a function $\xi_t : Z^d \rightarrow \{S, I, R\}$ (Durrett 1988); that is, a list of the *i*-states of the individuals at each location in space. Thus this is an archetypal *i*-state configuration model.

Remarkably little is known about these systems, and little of that is known rigorously. Most of the available results rely on the equivalence of the contact epidemic model and percolation models[5] (Durrett 1989, Grimmett 1989). Thus there is a threshold value p_c for the probability of infection such that when $p < p_c$ the infection dies out with probability 1, and when $p > p_c$ a single infected individual gives rise, with probability 1, to an infection which spreads infinitely far. The value of p_c is known for only a few special cases.

There is a phase transition at $p = p_c$, and physicists have made much of analogies to statistical mechanics, noting that such quantities as the percolation probability, the mean size of the infection arising from a single individual, and the number of infected particles per individual scale allometrically with p in the neighborhood of p_c. They conjecture that the exponents describing these scaling relationships are universal; i.e., their value is independent of the details of the model, depending only on the dimensionality of the lattice. Mathematicians seem fond of pointing out that none of this is proven (Grimmett 1989).

Considerable attention has been devoted to theorems describing the asymptotic shape and growth rate of an infection beginning from a single infected individual (Durrett 1988, 1989). For $p > p_c$, the shape is convex and grows linearly in time, as $t \rightarrow \infty$. Computer simulations on a 2-dimensional lattice (Durrett 1988) show this clearly. As $p \downarrow p_c$, however, the smooth convex shape is very slow to appear. There seems to be a correlation distance, below which the spatial pattern of infection is fractal, with a dimension less than 2. This distance seems to scale roughly as $(p - p_c)^{-2}$. Thus, close to the threshold probability, spatial pattern s will remain irregular until the infection has spread over a large area.

The analysis of these models has challenged some outstanding mathematicians, the results are published in journals of probability theory, physics, and mathematics, and the model itself (identical individuals living on the points of a regular, infinite, and homogeneous lattice passing infection only to their immediate neighbors) is absurdly simple. Durrett (1988) notes that most of the available results are qualitative rather than quantitative, and that they do not lend themselves to computation. It is a sobering thought that such difficulties may be generic to *i*-state configuration models.

Interactions among individuals are not limited to spatial neighbors. More detailed *i*-state configuration models, along the lines of the micro-simulation models used to study human fertility, or of the epidemic models of Ackerman et al. (1984) would be appropriate for modeling diseases whose trans-

[5]Percolation models (Frisch and Hammersley 1963) originated as idealizations of fluid flow through porous media. Imagine a lattice of sites, with lines or "bonds" connecting the sites. Each bond is open with probability p and blocked with probability $1 - p$. A liquid introduced at a site(s) can flow only along open bonds. Percolation is said to occur if a liquid introduced at a single site spreads through the entire medium. This model is referred to as bond percolation; there is a corresponding site percolation model in which all bonds are open and sites may be blocked or not.

mission is influenced by interactions among household members or sexual partners (e.g., Barrett 1988).

Population Inference From Individual Data: Hazard Analysis and Related Topics

The construction of demographic models necessitates inferring p-state parameters from data on i-state transitions. Huston et al. (1988) emphasize the ease of collecting individual data as a benefit of the i-state configuration approach. However, the transition rates which form the basis of an i-state distribution model can also be obtained from data on individuals. One of the most powerful analytical tools is hazard analysis (e.g., Cox and Oakes 1984; for applications see Trussell and Hammerslough 1983, John et al. 1987, John 1988).

Individual data generally take one of two forms: cross-sectional (also called current status, or period) data, in which information about the individual's state at a single time is recorded, or prospective data, in which individuals are followed for an extended period and their movements among states recorded. For humans, there is a third type of data—retrospective history data—in which the life history of the individual for a specified period prior to the interview is reported at the survey interview. An example is the retrospective maternity history collected for individual women in the World Fertility Surveys (Cleland and Scott 1987). These surveys were conducted in 44 developing countries in the late 1970s and early 1980s; in each country a standardized survey interview was used to record reproductive histories for approximately 5,000 women.

The p-state parameters that one wishes to estimate from these data are rates or probabilities (of survival, of reproduction, of conception, etc.). The data consist of individual transitions. In essence, hazard models are regression models in which the dependent variable is the rate at which the transition occurs, known as the hazard. For example, for a reproduction model, the dependent variable may be the monthly rate of conception.

Formally, let $f(t)$ denote the probability of the event happening at time t and let $F(t)$ denote the cumulative probability of the event happening before time t; i.e., $F(t) = \int_0^t f(s)ds$. Then the hazard function is $f(t)/(1 - F(t))$; it gives the risk of the event occurring at t, given that it has not yet occurred. This formulation is familiar to ecologists in the context of life table analysis, where $f(t)$ is the distribution of age at death, $1 - F(t)$ is the survivorship curve, and the hazard is the mortality rate $\mu(t)$. However, we emphasize that the use of hazard analysis is not restricted to mortality; to illustrate we will use as an example the transition from fecund to pregnant.

There are several ways of formulating hazard models; their common feature is the expression of the hazard as a function of a linear combination of explanatory variables. Let λ denote the hazard, z the vector of variables, and β a vector of coefficients. Then the hazard is written as $\lambda(\beta'z)$; the coefficients in β describe the relationship between the covariates and the hazard (e.g., the relation between lactation and the risk of conception).

Let $\lambda_i(t)$ denote the hazard for individual i at time t (e.g., the probability of individual i conceiving at time t conditional on not yet having conceived by time t). The two most common forms relating λ_i to the covariates z_i are the loglinear form

$$\lambda_i(t) = e^{\beta' z_i} \tag{14}$$

and the logistic form, which uses the log-odds of the hazard as the dependent variable in a linear model

$$\ln \frac{\lambda_i(t)}{1 - \lambda_i(t)} = \beta' z_i \tag{15}$$

or equivalently,

$$\lambda_i(t) = \frac{e^{\beta' z_i}}{1 + e^{\beta' z_i}} \tag{16}$$

These forms always result in nonnegative hazards. If the hazard were modeled simply as a linear function of z_i, some combinations of β and z_i would yield an estimated hazard less than zero, which is inconsistent with the definition of a hazard. While (14) and (16) appear quite different, the estimated coefficients are similar when the hazard $\lambda_i(t)$ being estimated is small (Allison 1982, Foster et al. 1986).

The hazard model is a particularly powerful tool because it can incorporate censored data; i.e., data in which the fate of some individuals is not observed. For example, a group of fecund women may be observed either until they conceive or until the survey ends; at the end of the survey the only information available about some women is that their wait was at least as long as the period of observation. Excluding these women from the analysis biases the sample to those with the shortest conception waits. However, in the hazard model, these women can be included as censored observations, thus rendering the sample unbiased. The models can also incorporate time-varying covariates; e.g., a fecund woman may move from the lactating to non-lactating state while she waits to conceive, and this transition may have a significant impact on her hazard.

Hazard models yield estimates of population level transition rates, but they are estimated from event history data on individuals. The data consist of information on whether the event has happened, and on covariates de-

scribing the individual (e.g., age, sex) and her environment (e.g., population density). These individual results and the population-level hazard model are connected by the likelihood function. The likelihood of the model given the data is proportional to the probability of the data given the model. The parameters in the model are estimated by maximizing the likelihood function. This usually requires iterative calculation, but the necessary routines can be found in the survival analysis routines in such software as SAS, STATA, and BMDP.

Using hazard analysis, complex information on i-state transition rates can be obtained from individual data. John (1988), for example, used a logistic hazard model to examine the factors determining conception rate (following a birth) in a sample of Bangladeshi women. The data, obtained in a prospective study, consisted of monthly interviews with 403 individual women over a period of five years. Covariates included time since resumption of menses, age, religion, weight, season, proportion to the month the couple are separated, husband's occupation, and the duration and extent of breastfeeding. The results demonstrated that time since resumption of menses, age, and the proportion of the month separated all had significant effects, but that season, socio-economic variables, and body size had little effect. Lactation effects were of particular interest, and the analysis showed that they had significant effects, reducing the probability of conception. Not only did the results indicate the significance of the covariates, but also provided a set of equations from which the conception rates could be calculated as functions of the covariates.

When the i-State Distribution is not Enough

Dynamic i-state distribution models are the fundamental tools of demographic theory, which relies on them to link the properties of individuals and the dynamics of populations. They produce equations (ordinary differential, partial differential, or difference) which can be attacked by a variety of powerful mathematical tools. The attack may be resisted, but if successful its results are more powerful and more general than the simulation of i-state configuration models. For example, simulation of individuals following a multi-type branching process might suggest that population size eventually increases exponentially. Repeating the simulation many times might suggest that this always happens, and that the rate depends on the stage-specific vital rates. But branching process theory provides powerful general results that go beyond those obtained from simulations (e.g., if the dominant eigenvalue of the expected value matrix $\boldsymbol{A}$ is greater than one, the population converges with probability 1 to a random vector proportional to the dominant right eigenvector $\boldsymbol{w}_1$ of $\boldsymbol{A}$). Simple matrix algebra applied to $\boldsymbol{A}$ reveals the conditions (primitivity of the expected value matrix) under which this asymptotic growth pattern appears, what happens when those conditions are not fulfilled, and how to calculate the eventual rate of increase. These conclusions could not be reached by simulation alone, no matter how elaborate or sophisticated. This power is not something to be lightly discarded, even though it may require approximations in the development of the models.

Be that as it may, there are at least three situations in which i-state distribution models become intractable or impossible, and in which a direct attack on the i-state configuration may be the only possible tactic. These situations are: complicated i-states, small populations, and local interactions.

As the complexity of the i-state increases, the number of dimensions in the i-state distribution may become inconveniently large. Simulation of an i-state configuration model may become easier than numerical solution of a complicated i-state distribution model. There is a trend in population ecology toward incorporating more detailed individual information in models (e.g., the work of the inter-continental *Daphnia* modeling conspiracy, the usual suspects in which include Kooijman (1986), Hallam et al. (1990), McCauley et al. (1990), and Gurney et al. (1990)). Combined with increased computer power, this trend will probably lead to increased use of i-state configuration simulations. The difficulties encountered by John (1990a,b) and Tuljapurkar and John (1991) in the analysis of age-classified epidemic models suggests that an i-state configuration analysis might be useful here as well.

It is widely appreciated that the dynamics of small populations may be strongly affected by demographic stochasticity. Because population projection models are expected value operators for underlying multi-type branching process models, their accuracy declines when population size is small. The underlying branching process models, which include demographic stochasticity, are i-state distribution models, just as the expected value models are. However, their analysis may be difficult enough that i-state configuration simulations become an attractive approach to their study. Interest in demographic analysis of threatened or endangered species, and questions of the viability of small populations, will likely lead to increased use of i-state configuration simulations in this context.

These uses of i-state configuration models are practical conveniences rather than theoretical necessities. There are, after all, no theoretical limits on, say, the size of a population projection matrix. The situation is different when the assumption of mixing fails. In this case, i-state configuration models play a fundamental role. If the failure of mixing results from simple spatial heterogeneity, where individuals in different locations experience different environmental conditions, it can be remedied by enlarging the i-state to include location. But if the heterogeneity results from local interaction of individuals, the entire configuration of the population is fundamentally required to predict its dynamics. The interactions may be in geographical space, as in the generalized epidemic models and their percolation theory counter-

parts, or in some more abstract space, as in the kinship-based micro-simulation model of Hammel et al. (1979). In either case, Huston et al. (1988) are correct to emphasize the importance of local interactions as a problem leading to *i*-state configuration models.

Farmer (1990) draws a potentially useful distinction between fixed and dynamic interaction structures in a recent paper on 'connectionist' models, which

> "consist of elementary units which can be 'connected' together to form a network. The form of the resulting connection diagram is often called the architecture of the network . . . Since the modeler has control over how the connections are made, the architecture is plastic. This contrasts with the usual approach in dynamics and bifurcation theory, where the dynamical system is a fixed object whose variability is concentrated into a few parameters . . . Dynamics occurs on as many as three levels, that of the states of the network, the values of connection strengths, and the architecture of the connections themselves." (Farmer 1990, p. 154)

Farmer cites neural networks, classifier systems, immune networks, and autocatalytic chemical reaction networks as examples. These are differentiated from models like cellular automata, in which the units interact, but the interactions are "fixed, completely regular, and have no dynamics." *i*-state configuration models in which the interactions themselves evolve (e.g., due to relatedness, household structure, or pair formation) may share some properties with other connectionist models.

Parting Thoughts

We conclude with some thoughts about future uses and future problems with *i*-state configuration models.

First, *i*-state configuration models may be useful for estimating the parameters in *i*-state distribution models. Suppose that the set of *i*-states is large and the rules for transitions among them complicated. An *i*-state configuration model could provide "data" from which the waiting time distributions and transition probabilities could be estimated using hazard models. Or, the probabilities calculated from the individual-based simulation could be used to derive maximum likelihood estimates of parameter values (A. Solow, pers. comm.).

Second, most analyses of most models are interested in asymptotic behavior. Since *i*-state configuration models are usually stochastic, analyzing and interpreting asymptotic behavior will require extra care because results on convergence of stochastic processes are generally more subtle than the results for the convergence of deterministic series.

Stochastic matrix population models provide an excellent example. The

asymptotic behavior of time-invariant models is unambiguously described by the dominant eigenvalue of the projection matrix. The demographic strong ergodic theorem guarantees that every initial population will eventually grow at this rate. If the vital rates vary stochastically, however, population size is a random variable, and one must choose how to describe its asymptotic properties. One can calculate the asymptotic growth rate of the mean population size

$$\ln \mu = \lim_{t\to\infty} \frac{1}{t} \ln E[N(t)] \tag{17}$$

or the mean population growth rate

$$\ln \lambda_s = \lim_{t\to\infty} \frac{1}{t} E[\ln N(t)] \tag{18}$$

where $N(t) = \Sigma_i n_i(t)$ denotes total population size. The interesting part is that these two rates are in general not equal; $\ln \lambda_s \leq \ln \mu$, with strict inequality in general. Which rate to choose? The answer is provided by the ergodic theory of random matrix products (Furstenburg and Kesten 1960, Cohen 1976a, Tuljapurkar and Orzack 1980; see reviews by Tuljapurkar 1989, 1990), which proves that, under a wide array of conditions,

$$\lim_{t\to\infty} \frac{1}{t} \ln N(t) = \ln \lambda_s \tag{19}$$

with probability 1. That is, except for a set of measure zero, every realization of this process eventually grows at the rate $\ln \lambda_s$, even though the mean population size is growing at the faster rate $\ln \mu$. Thus it is perfectly possible to have the mean population size increasing exponentially ($\ln \mu > 0$) while the probability of extinction is approaching 1 ($\ln \lambda_s < 0$). Without the guidance of the ergodic theory, it is doubtful that simulation analysis (e.g., Boyce 1977) would uncover the true nature of the asymptotic behavior of the model.

For an individual-based example, consider a model of the gregarious settlement of larval benthic invertebrates. Suppose there is a finite number of patches in which larva may settle, and that the attractiveness of a patch is directly proportional to the number of settled individuals in that patch. Begin with all patches equal (say, with a single individual in each) and follow the settlement of a population of larvae. Figure 3.3a shows the results of a simulation of 1000 individuals for the simple case of two patches. The occupancy of the two patches converges to proportions of 0.6334 and 0.3666.

Since this is a stochastic simulation, we had better replicate it. Figure

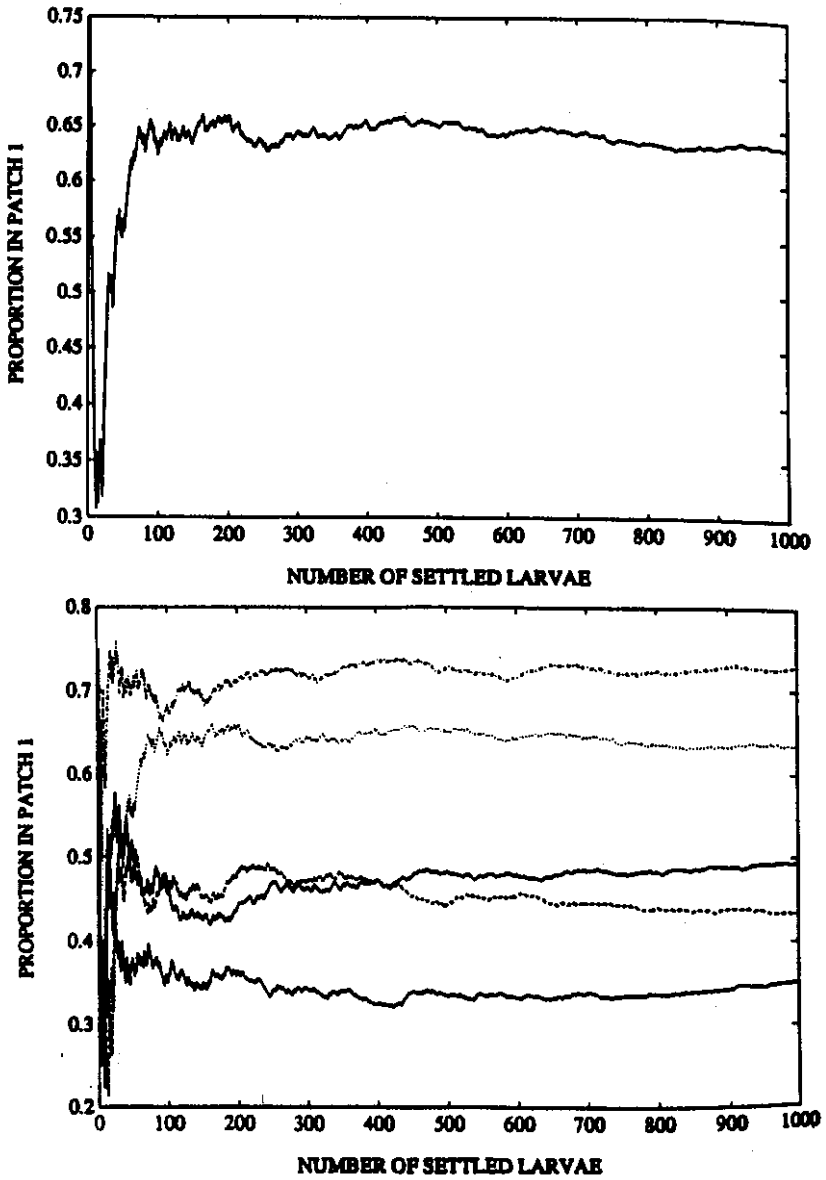


Figure 3.3. (a) The results of a simulation of gregarious settlement into two patches by individual planktonic larvae of a benthic invertebrate. There are two patches; the proportion of larvae settling in Patch 1 is plotted against time. (b) The results of five replicates of the experiment in (a).

3.3b shows the results of five replicates. Each converges to a fixed proportion of settled larvae in each patch, but the proportion is different each time. This model, introduced to population biologists by Cohen (1976b), is an example of a Polya urn scheme (Eggenberger and Polya 1923). It has the surprising property that, with probability 1, every realization converges to fixed proportions in each patch, but that the proportions are "nondegenerate limit" random variables, uniformly distributed between 0 and 1 (or, for k patches, uniformly distributed on the unit simplex). See Arthur et al. (1987) for an application to the problem of industrial firms choosing locations; the analogy to larval settlement is not coincidental.

These examples underscore the likelihood that study of the asymptotic properties of stochastic i-state configuration models will require concepts and methods that go beyond the stability analyses so useful in studying similar deterministic models.

Conclusions

The individual plays an essential role in demographic models. Choosing a p-state variable is the crucial step in constructing a population model from individual information. Demographic theory has been developed using the i-state distribution as a p-state variable. There are powerful methods, including hazard models, for estimating the parameters in these models from data on individual organisms, and equally powerful analytical techniques for analyzing the models. However, when the i-state space is very complicated, simulation of i-state configuration models may be an alternative to the solution of the dynamic equations for an i-state distribution model. More fundamentally, when local interactions (in either geographical or some other, more abstract space) are important, the mixing assumption required for i-state distribution models fails. In these cases, i-state configuration models are a necessity, not a convenience. As models of individual organisms become more detailed, and computers become more powerful, i-state configuration models will become increasingly useful in demography.

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