27 Population Modeling

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If all significant environmental decisions could be based on predicted or observed effects of chemicals or other agents on organisms, there would be no need for this chapter. However, there are many important decisions for which knowledge of organism-level effects simply is not enough. Some species may, because of their life history or because of their greatly reduced abundance, be more at risk than others, given the same sensitivity of individuals. Mortality to certain individuals is unavoidable, in which case risk managers may be interested in the amount of mortality (or growth reduction) that can be tolerated by the exposed species. It may be necessary to know whether the combined effects of agents affecting several different life stages may reduce the abundance of populations or lead to increased risk of extinction. It may be important to forecast the rate of recovery of populations following an accident or a remedial action.

The toxicity tests and extrapolation models discussed in Chapter 24 and Chapter 26 are insufficient for addressing these problems. From a population viewpoint, the death or impairment of an organism is meaningless, because most organisms die after brief lives (on a human time scale) and few organisms achieve their full reproductive potential or maximum growth. Ecologists have long known that natural populations of many organisms frequently are subjected to extreme environmental variations that cause mass mortalities, and many species are composed of isolated pockets of organisms that occasionally become extinct, and are later reestablished. Modern ecological theory views disturbance and instability as normal, and the constant conditions observed in laboratory experiments to be highly unrealistic.

Many questions of interest in risk assessment relate to effects on the abundance, production, or persistence of populations. Responses of populations cannot be predicted from toxicity tests alone. The response of a fish population, for example, to a contaminant exposure will depend on the spatial pattern of exposure as related to the distribution of individuals in time and space, on the magnitude of other impacts that are imposed (including especially harvesting by fishermen), and the inherent capacity of the population to "compensate" or to evolve in response to exposure. The response of a soil invertebrate population periodically exposed to pesticides depends not only on spatial patterns of exposure and dose–response relationships, but also on the reproductive capacity of the population and on availability of nearby sources of immigrants that can replace organisms killed by the exposure.

Interest among both ecotoxicologists and risk assessors in techniques for predicting responses of populations to chemical exposures has grown rapidly in recent years. Between 1980 and 1990, for example, the journal *Environmental Toxicology and Chemistry* published only three papers dealing with effects of chemicals on populations. This same journal published more than 50 such papers between 1996 and 2005. The increase reflects both renewed awareness on the part of scientists and management agencies that population-level effects are important and can be quantified, and the emergence of new techniques for quantifying population dynamics, many drawn from the emerging field of conservation biology.

For small, short-lived species (e.g., microbes, cladocerans, and some other small arthropods), effects of chemicals on critical population parameters can be measured directly using laboratory experiments. However, these organisms are unrepresentative of the great majority of populations of interest in ecological risk assessment. In a few rare cases, effects of chemicals on the same parameters have been estimated from long-term field studies (e.g., Barnthouse et al. 2003). However, most population-level ecological risk assessments have and will continue to involve using mathematical models of populations to link organism-level experimental data to population-level responses. The objective of this chapter is to show how such models can be developed and applied.

The problem of quantifying population-level responses to death or impairment of organisms is not new. Fish and wildlife managers have struggled for decades with the problem of defining the effects of harvesting, habitat modification, and disease on the abundance and stability of exploited populations. Recently, conservation biologists have developed novel methods for quantifying the effects of environmental variability, habitat fragmentation, and reduced population sizes on the persistence of rare and endangered species. The methods used by resource managers and conservation biologists provide a useful frame of reference for assessments of other sources of stress, including toxic chemicals.

This chapter provides basic definitions, briefly describes some of the models that are especially relevant to assessing ecological effects of toxic chemicals, and discusses some specific applications. Readers interested in a more in-depth treatment of the principles of population biology are advised to consult the excellent textbooks and reviews that are available. Most modern undergraduate-level ecology textbooks (e.g., Begon et al. 1999; Krebs 2002) explain the fundamentals of population analysis. However, many of the most readable and thorough accounts are older. Among these, the most widely cited (and most frequently consulted by the author of this chapter) is the classic text by Andrewartha and Birch (1954). The texts of choice for fish population studies are Hilborn and Walters (1992) and Quinn and Deriso (1999); Bolen and Robinson (2002) provide a good general discussion of wildlife population biology. An overview of the new subdiscipline of "metapopulation biology," which is of substantial relevance to ecological risk assessment, is provided by Hanski and Gilpin (1996). For the mathematically inclined, Caswell (2001) provides a thorough exposition of the theory of matrix population models. Readers interested in developing computer simulation models of populations should consult Swartzman and Kaluzny (1987; unfortunately now out of print), or Jorgensen and Bendoriccio (2001).

Other authors have also written about population-level ecological risk assessment methods. Particularly noteworthy are the books by Newman (2001) and Pastorok et al. (2002). These authors provide useful alternative perspectives concerning issues addressed in this chapter.

One significant topic that is not discussed at all in this chapter is population genetics. Historically, the two principal branches of population biology, demography and population genetics, were intimately related, and many of the pioneers in theoretical population biology made fundamental contributions to both. Some, but not all, of the modeling approaches discussed in this chapter are used in population genetics as well as demography. Impacts of chemicals on the genetic composition and fitness of populations are topics that have been discussed in the ecotoxicological literature (Forbes 1999; Newman 2001); however, genetic impacts on populations have not yet been an issue in any major environmental controversy and no guidance has been developed concerning how risk assessments should or could address these impacts. Until ecological risk assessments begin to more routinely consider population genetics, it seems premature to include the topic in this chapter. Thus, the scope of this chapter is limited to demography.

27.1 BASIC CONCEPTS AND DEFINITIONS

The fundamental objective of population biology is to infer characteristics of groups of organisms (i.e., the populations) from organismal characteristics. The population characteristics of interest include total numbers or biomass, rate of population growth or decline, age, size, sex, or genotypic composition of the population, and the probability that the population will persist into the future. Only a subset of these characteristics is likely to be important in any single assessment. Managers of exploited populations may be interested in the number of organisms available to be harvested; conservationists may be interested in the probability of extinction of a population of a given size. These population characteristics are simply collective expressions of the state and fate of constituent organisms, such as reproduction rates, growth and development rates, and probabilities of death. The individual characteristics are, in turn, governed by (1) innate processes such as development and senescence; (2) the effects of the physical environment; (3) interactions with other organisms; and (4) deliberate or unintentional actions by man.

27.1.1 Population-Level Assessment Endpoints

Population studies address a variety of endpoints of interest in risk assessment. The most basic of these are the endpoints traditionally used in management of exploited populations: total population density or biomass, age or size distribution, and sustainable rate of harvest. In resource management, models relate these endpoints to management actions such as harvest quotas, size limits, or harvest season lengths. Due to concerns about the conservation of endangered species, population biologists have recently formulated new models that contain environmental or demographic stochasticity and spatial heterogeneity, to address endpoints related to persistence of populations in variable environments. These models are used to estimate frequencies or probabilities of extinction within a given time period or expected time to extinction, as functions of population size, size of habitat required, or degree of habitat fragmentation.

27.1.2 Implications of Life History for Population-Level Ecological Risk Assessment

Are some species inherently more vulnerable to environmental stress because of their life history? Are some life stages more important than others to the survival of a population? To date, few ecotoxicologists have attempted to address the influence of life history on the vulnerability of populations to toxic chemicals. Exceptions include Barnthouse et al. (1990), who investigated effects of chemical exposures on two fish species with contrasting life histories and harvesting patterns; Spromberg and Birge (2005), who performed a similar investigation of five general fish life history types; and Calow et al. (1997), who developed a quantitative framework for relating life history traits to population-level effects of chemical exposures. However, the influence of various life history traits on population growth rates was a major topic of theoretical research during the 1960s and 1970s (see review by Stearns 1977). Both theoretical analysis and management experience have shown that long-lived vertebrates such as large mammals, predatory birds, and whales are more sensitive to mortality imposed on adults than are short-lived, highly fecund organisms such as quail and anchovies. Conversely, short-lived species are often vulnerable to short-term catastrophes that affect critical life stages. Populations in which survival or reproduction are strongly related to the density or abundance of the population should, on theoretical grounds, be less vulnerable than populations with a low degree of density-dependence. Qualitatively, it seems clear that the response of a population to a toxic chemical is influenced by the preexisting patterns of natural environmental variability, the age-specific survival and reproduction of the organisms, and the intensity and duration of exposure.

27.1.3 Representation and Propagation of Uncertainty

Chapter 5 discusses a variety of sources of uncertainty that are of interest in ecological risk assessment. Sources potentially important for population analysis include (1) environmental variability in time and space; (2) variations in sensitivity among individuals and life stages; and (3) stochastic birth and death processes. Interindividual and inter-life-stage variability are discussed in Chapter 26. Stochastic birth and death result from the fact that each organism has an indeterminate life span, even if the average life span for the population can be very precisely estimated. In practice, random birth and death processes, usually termed "demographic stochasticity," are important only in small populations (e.g., 50 individuals or fewer). Even for small populations, Goodman (1987) showed that this source of uncertainty is usually quite small compared to environmental variability.

Temporal environmental variability is readily incorporated in population models. Both periodic and stochastic variations have been studied. The principal mathematical tools available include time series analysis and stochastic modeling. These approaches may be used to quantify environmental variability (e.g., to estimate a periodic function of some important driving variable such as temperature or rainfall), or to estimate probability distributions for temporally varying population parameters (e.g., mortality rates). Many of these techniques are mathematically complex and well beyond the scope of this book. Time series analysis is widely used in economics and engineering as well as in ecology; Brockwell and Davis (2003) discuss many of the widely used procedures and software packages. Stochastic population models are discussed by Caswell (2001), and book-length treatments have been written by Nisbet and Gurney (1982) and Tuljapurkar (1990). The problem of fitting stochastic population models to time series data is still an active area of research in population biology, so none of the available textbooks provides a fully up-to-date treatment. For the purpose of typical risk assessments, however, theoretical elegance is often unnecessary. Techniques for using Monte Carlo modeling population-level consequences of environmental variability and parameter uncertainty have been available for more than 20 y (e.g., O'Neill et al. 1982; Barnthouse et al. 1990; Bartell et al. 1992). The popular RAMAS population and ecosystem modeling software (available from Applied Biomathematics, Setaucket, New York, web address http://www.RAMAS.com/) was specifically designed for this purpose.

Spatial variability can now readily be addressed using metapopulation models, i.e., models in which populations are represented as sets of semi-isolated subpopulations, each with potentially different rates of reproduction and mortality that are linked through the processes of immigration and emigration. Although the term "metapopulation" was first used in the 1960s (Levins 1969), most of the methods used to model metapopulations have been developed since 1990. As noted above, Hanski and Gilpin (1996) provide a good introduction to the theory of metapopulations. The most widely used software for metapopulation modeling is the VORTEX model described by Lacy (1993); however, a metapopulation version of RAMAS is also available. A related class of models, termed "spatially explicit" models represents populations either as groups of organisms or as individual organisms interacting on a spatial grid (e.g., Liu 1993; Turner et al. 1994). These techniques provide entirely new ways of quantifying ecological risks of spatially heterogenous chemical exposures; an example is discussed later in this chapter.

27.1.4 Density Dependence

Population regulation has been a fundamental problem in population biology since its inception. What prevents populations with high reproductive rates from increasing without bounds? How do fish and wildlife populations persist in the face of intensive exploitation by

humans? The simplest answer to these questions is that in many, and perhaps in most or all, populations, either mortality, reproduction, or both change with the size of the population. When numbers are high, mortality increases and reproduction decreases; when numbers are low, mortality decreases and reproduction increases. Many empirical studies have documented effects of density on growth or reproduction of organisms; the widespread existence of density-dependence at the level of organisms is beyond doubt. However, the importance of these mechanisms for stabilizing populations and ensuring their persistence in variable environments is still widely debated. Rose et al. (2001) discussed the difficulty of quantifying effects of density-dependence in fish, the taxonomic group that has been the most intensively studied. In spite of the fact that harvested populations of fish could not sustain themselves if survival or reproduction was not strongly density-dependent, it continues to be very difficult to detect and quantify density-dependence in specific fish populations. In addition, populations of many types of organisms appear to be stabilized through dispersal of organisms between habitat patches, so that the population as a whole may persist indefinitely even though the subpopulations inhabiting individual patches frequently become extinct (den Boer 1968; Wu and Loucks 1995). Regardless of the mechanisms involved, it seems clear that some form of density-dependence, acting either within populations or between interacting subpopulations, is necessary to ensure the persistence of species (Murdoch 1994; Lande et al. 2002).

It is not always necessary to build density-dependence into population models used for management or risk assessment. Fish and wildlife managers have been reasonably successful with density-independent models, provided that the prediction horizon is short and that the population changes modeled are relatively small. For long-term predictions, however, explicit incorporation of density-dependence is usually necessary to provide realistic simulations. Projections from purely density-independent models inevitably grow either to infinite size or decline to zero, even without the imposition of anthropogenic agents such as harvesting or toxic chemicals.

27.2 APPROACHES TO POPULATION ANALYSIS

A variety of approaches to population analysis have been developed over the past several decades. This section provides an overview of the principal methods, with an emphasis on their conceptual relationships and past applications. Representative case studies involving toxic chemicals are presented in Section 27.3.

27.2.1 Potential Population Growth Rate

The simplest approach to population analysis is quantification of the population growth rate. The theory in its present form was developed by Lotka (1924), Fisher (1930), and Cole (1954). Before the 1960s, analysis of the relationships between life history traits and population growth was the only approach to population dynamics used outside fisheries management. The approach requires only a compilation of (1) the fraction of organisms surviving from one age to the next, and (2) the average number of offspring of an organism of a given age. Define l_x as the fraction of organisms surviving from birth to age x, and m_x as the average number of offspring produced by an organism of age x. Suppose the maximum age or organisms in the population is n years. If l_x and m_x are constant, these parameters uniquely determine the relationship between reproduction, mortality, longevity, and population growth. This relationship is mathematically described by Equation 27.1:

$$\sum_{x=1}^{n} e^{-rx} l_x m_x = 1 \tag{27.1}$$

The parameter r, the coefficient required to make the left side of Equation 27.1 sum to 1, has been termed the "Malthusian parameter," the "intrinsic rate of natural increase," or the "geometric rate of increase." In this chapter it will be termed the instantaneous rate of population change. If r is greater than 0, the population will increase indefinitely. If r is less than 0, the population will decline toward extinction, and if r is exactly 0 it will remain unchanged. It can be shown that if undisturbed, the age composition of this population will converge to a "stable age distribution" in which the fraction of organisms in each age class is the same from each generation to the next. Once this state is achieved, both the population as a whole and the number of organisms in each age class will grow (or shrink) exponentially with time:

$$N_t = N_0 e^{rt} \tag{27.2}$$

where N_t = population time at time t, and N_0 = population size at time 0.

Equation 27.1 and Equation 27.2 are often expressed in alternative forms:

$$\sum_{x=1}^{n} \lambda^{-x} l_x \, m_x = 1 \tag{27.3}$$

$$N_t = N_0 \, \lambda^t \tag{27.4}$$

where $\lambda = e^r$ = the finite rate of population change.

The value of r and changes in r that might relate to the decline or extinction of a population are important for population management. Many authors have investigated the sensitivity of r to changes in fecundity or mortality. Mertz (1971) showed that because of its very low reproductive rate, the California condor population was extremely vulnerable to increased mortality of adults due to hunting. Mertz also concluded that management actions designed to increase reproductive success in this population were unlikely to improve the prospects for recovery. Unfortunately, Mertz's prediction proved correct and the California condor became extinct in the wild about 10 y later. During the 1980s, numerous authors used similar methods to assess the viability of northern spotted owl (Dawson et al. 1987; Lande 1988).

In ecotoxicology, the potential population growth rate approach is now frequently used to interpret results of chronic toxicity tests performed using cladocerans and other small, short-lived species (e.g., Daniels and Allan 1981; Gentile et al. 1983; Meyer et al. 1986; Walthall and Stark 1997; Kuhn et al. 2001; Salice and Miller 2003). Measurements of daily survival and reproduction obtained from these tests are sufficient to obtain estimates of r. Changes in r resulting from exposure to toxicants can be used as a relative index of chronic effects on populations. Although the calculated values of r cannot be directly extrapolated to the field, this approach to test data interpretation has the advantage of combining information on survival and reproduction into a single index. Forbes and Calow (1999) compiled a list of 41 such studies, including a total of 28 species and 44 chemicals. In addition to being used to assess risks to populations of the tested species, test-derived estimates of r for multiple species have been proposed as a potential method for deriving water quality criteria that protect aquatic communities (Forbes et al. 2001; discussed later in this chapter).

27.2.2 Projection Matrices

Age-structured or stage-structured projection matrices are an important extension of the potential population growth rate approach. The simplest matrix model is the linear "Leslie matrix" (Leslie 1945; Caswell 2001). The Leslie matrix contains exactly the same information as the potential population growth rate model, but the information is expressed in matrix form. The change in abundance of each population in time can be represented by the matrix equation:

$$\mathbf{N}(t) = \mathbf{L}\mathbf{N}(t-1) \tag{27.5}$$

where $\mathbf{N}(t)$ and $\mathbf{N}(t-1)$ are vectors containing the numbers of organisms in each age class $(N_0,\ldots,N_k)$, and $\mathbf{L}$ is the matrix defined by

$$\mathbf{L} = \begin{pmatrix} s_0 f_1 & s_1 f_2 & s_2 f_3 & \cdots & s_{k-1} f_k & 0 \\ s_0 & 0 & 0 & \cdots & 0 & 0 \\ 0 & s_1 & 0 & \cdots & 0 & 0 \\ 0 & 0 & s_2 & \cdots & 0 & 0 \\ \cdots & \cdots & \cdots & \cdots & \cdots & \cdots \\ 0 & 0 & 0 & \cdots & s_k & 0 \end{pmatrix} \tag{27.6}$$

where s_k = age-specific probability of surviving from one time interval to the next, and f_k = average fecundity of an organisms at age k.

The Leslie matrix can also be expressed in a graphical form (Figure 27.1a) in which the different age classes are depicted as nodes and the survival and reproduction parameters are depicted as arrows connecting the nodes.

The matrix analog to Equation 27.4 is

$$\mathbf{N}(t) = \mathbf{L}^t\,\mathbf{N}(0) \tag{27.7}$$

where $\mathbf{N}(0)$ = age distribution vector at time 0, and $\mathbf{L}^t$ = the matrix $\mathbf{L}$ raised to the power t.

Leslie (1945) showed that any population growing according to Equation 27.7 will converge to a stable age distribution, after which it will grow according to

$$\mathbf{N}(t) = \lambda^t\,\mathbf{N}(0) \tag{27.8}$$

The parameter λ, which in matrix algebra is termed the "dominant eigenvalue" of the matrix $\mathbf{L}$, is the same finite rate of population change that appears in Equation 27.3 and Equation 27.4.

Alternatively, a matrix of life stages and stage transitions (as opposed to ages and age transitions) can be constructed. Caswell (2001) presents a detailed discussion of the mathematics of stage-classified models. In addition to representing survival and reproduction rates, the coefficients of a stage-classified model can represent probabilities of transition from one size class or life stage to the next.

For example, in a stage-based alternative to the Leslie matrix, an organism alive during any time step might either remain in the same stage or size class during the next time step or transition to the next class. The transition matrix for a model of such as population could be

$$\mathbf{A} = \begin{pmatrix} P_0 & F_1 & F_2 & \cdots & F_k \\ G_0 & P_1 & 0 & \cdots & 0 \\ 0 & G_1 & P_2 & \cdots & 0 \\ 0 & 0 & G_2 & \cdots & 0 \\ \cdots & \cdots & \cdots & \cdots & \cdots \\ 0 & 0 & 0 & G_{k-1} & P_k \end{pmatrix} \tag{27.9}$$

In this matrix, the elements along the diagonal (P_i) are probabilities that an organism will survive and remain in the same class; elements along the subdiagonal (G_i) are probabilities that an organism will survive and transition to the next class. A graphical version of a stage-based model is provided in Figure 27.1b. The projection equation for this matrix is

$$\mathbf{N}(t) = \mathbf{A}^t\,\mathbf{N}(0) \tag{27.10}$$

Ecological examples of stage-based models include Sinko and Streifer (1967, 1969), Taylor (1979), Law (1983), Law and Edley (1990), De Roos et al. (1992), and many other papers cited by Caswell (2001). These models appear especially useful for plants and invertebrates, which have complex life cycles in which population dynamics are more strongly influenced by size and developmental stage than by age. Emlen and Pikitch (1989) used stage-classified models of generalized vertebrate populations to analyze the sensitivity of different types of vertebrate life cycle to mortality imposed on different stages. Munns et al. (1997) developed a

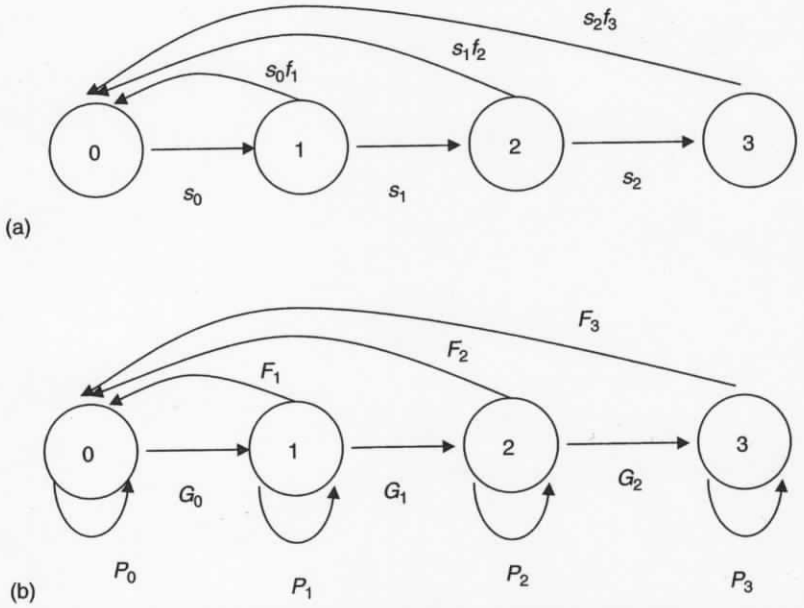

FIGURE 27.1 Life cycle graphs corresponding to age-based (Leslie) and stage-based projection models. (a) In the age-based model, organisms surviving at each time step transition to the next age group. No organisms survive beyond age group 3. (b) In the stage-based model, organisms surviving at each time step may either transition to the next stage or stay in the same stage. In both models, reproduction occurs immediately following the age–stage transition.

stage-based model of a mummichog population exposed to dioxin and polychlorinated biphenyls (PCBs).

The relative influence of different life history characteristics on the population growth can be calculated using a property termed "elasticity" (deKroon et al. 1986; Caswell 2001). Elasticity is a measure of the proportional sensitivity of λ to each element of the population transition matrix (a_{ij}):

$$e_{ij} = (a_{ij}/\lambda)(\partial\lambda/\partial a_{ij}) \tag{27.11}$$

The elasticities of all the elements (a_{ij}) of a population projection matrix sum to 1.0. Spromberg and Birge (2005) and Forbes et al. (2001) used the elasticity index to compare the relative influence of different life history characteristics on the population growth rates of species with different life history strategies.

For a population that is *not* at a stable age distribution, Equation 27.7 or Equation 27.10 can be used to predict the abundance and age distribution of the population over the next few time intervals, given any initial age distribution. Otherwise, as long as the coefficients of the matrices **L** or **A** are viewed as constant parameters, the matrix projection approach is essentially equivalent to the population growth rate approach. The real power of the matrix representation of population dynamics is its flexibility. The coefficients of **L** or **A** can be viewed not as constants, but as random variables or functions of environmental parameters.

These modifications permit entirely new types of analyses, many of substantial value for risk assessment. In the strictly linear and deterministic models discussed so far, the only endpoint that can be addressed is the future trend of abundance: will the population increase or decline in the future, and how sensitive is the rate of increase or decline to changes in life history parameters? Modification of the matrix approach in the ways described below permits assessment of any endpoint for which an operational definition can be formulated.

The remainder of this section deals with matrix models in which the elements are variables rather than constants. One straightforward modification is to make the coefficients random variables. Stochastic matrix models are generalizations of age- or stage-based models, in which one or more of the matrix coefficients are assumed to be a random variable. In very small populations, substantial fluctuations in abundance, and even extinction, can occur simply because of the random nature of birth and death processes. The most important application of these models for ecological risk assessment is in quantifying the probability of extinction, either due to strictly demographic and environmental stochasticity or due to stress (e.g., excessive harvesting, toxic chemical exposure, or periodic catastrophic mortality) imposed on stochastically varying populations.

The theoretical literature on stochastic matrix models emphasizes the use of analytical mathematics to obtain general results. In applications to specific populations, however, numerical simulations using Monte Carlo methods are usually adequate and are much easier to perform. Barnthouse et al. (1990) used Monte Carlo simulation to evaluate the combined effects of environmental variability and uncertainty of chemical toxicity on the abundance and risk of extinction of two fish populations. Snell and Serra (2000) describe a similar approach to quantify impacts of chemical exposures on rotifer populations influenced by short-term environmental variability and episodic catastrophes. Software designed for easy development of stochastic matrix models is now readily available; however, in many cases special software is not needed. Equation 27.3 and Equation 27.5 can be readily implemented on a spreadsheet, and Monte Carlo analysis of spreadsheets can be easily performed using add-on programs such as Crystal Ball®.

Another common modification of the basic matrix model is to make the coefficients density-dependent, i.e., to make one or more of the vital rates a function of the number of

individuals either in the entire population or in some of the age classes. The purpose of incorporating density-dependence is to account for the fact that, in spite of fluctuations in abundance, populations in nature are always bounded within limits (Section 27.1.4).

As a practical matter, explicit incorporation of density-dependence in population models is often necessary to obtain realistic population projections. Inspection of Equation 27.2, Equation 27.4, Equation 27.8, and Equation 27.10 shows why. For deterministic models, unless r is *exactly* 0 (λ *exactly* 1), the model population must either grow without bounds or decline to zero. Inclusion of stochasticity does not alter this behavior. Given enough time, any density-independent model population will either grow to infinite size or become extinct. For this reason, most models intended to simulate population behavior over more than one generation incorporate density-dependence. Two common functions for density-dependent survival (s) are the Beverton–Holt function:

$$s = c_1/(1 + c_2 n) \tag{27.12}$$

where c_1, c_2 = constant parameters, and n = population size; and the Ricker function:

$$s = \alpha e^{-\beta n} \tag{27.13}$$

where α, β = constant parameters.

These functions are introduced here simply as convenient examples, because both have been extensively studied and are easy to apply to age- and stage-structured models. Both functions were originally developed for application to fish populations; explanations of their derivations and uses can be found in Hilborn and Walters (1992) or in Quinn and Deriso (1999). The key parameters of these two functions are the constants c_1 in the Beverton–Holt model and α in the Ricker model. They represent the maximum possible values of the survival rate s. When the population size (n) is very low, s is approximately equal to c_1 (Beverton–Holt model) or α (Ricker model). As n increases, s decreases in both models (although at a different rate in each model). In the fisheries literature, the parameters c_1 and α are sometimes termed estimates of "compensatory reserve," because they are measures of the capacity of populations to grow at low population sizes. Populations with high compensatory reserves (i.e., populations that grow rapidly at low population sizes) are more resilient to environmental disturbances, and can often sustain higher rates of harvesting, than can populations with low compensatory reserves (Christensen and Goodyear 1988; Rose et al. 2001).

The functional forms of Equation 27.12 and Equation 27.13 do not reflect specific biological processes, and there is no way to directly measure the critical parameters (c_1, c_2, α, and β). Moreover, there is no way to determine a priori which functional form is appropriate for a given population. In principle, it should be possible to determine which model is appropriate by fitting both models to a time series of data on population abundance. In practice, the available data are rarely sufficient to unambiguously distinguish between the two models. For the purpose of risk assessments, uncertainty due to differences between alternative models of the same process can usually best be dealt with by performing alternative simulations using all of the alternatives. If the results are not affected by the choice of models, the most convenient model may be selected; if the results are highly dependent on the choice of models, it may be better to perform parallel simulations using both.

Predictions about future population behavior or response to stress can be extremely sensitive to variations in functional forms and parameter values. For this reason, management applications of density-dependence have had mixed success and are often highly controversial (Rose et al. 2001). As in most other applications of mathematical models in risk assessment, these difficulties are minimized if the models are used for comparative

purposes rather than for the purpose of predicting future states of nature. For example, Barnthouse et al. (1990) used stochastic, density-dependent population models to examine the relative influence of uncertainties concerning life history, harvesting mortality, environmental variability, and chemical toxicity on the predicted responses of fish populations to chemical exposures. They found that uncertainties related to the characteristics of the exposed populations were negligibly small compared with uncertainties related to laboratory-to-field extrapolation of toxicity test data.

27.2.3 Aggregated Models

Many readers of this book will be familiar with other kinds of population models, such as the logistic model and stock-recruitment model. These approaches to population modeling differ from the models discussed above in that all organisms are aggregated into one or two components such as "population size" or "parents and offspring." The logistic model has a long history in population biology, and is discussed in all college-level ecology textbooks. It is perhaps the simplest model that simulates the growth and stabilization of populations. A form of the logistic model, known as the Shaeffer surplus production model, is used in fisheries management. Good discussions of surplus production models and of the various forms of stock-recruitment models can be found in Hilborn and Walters (1992). Because of its extreme simplicity, many applied population biologists view the logistic model and its variants as being of little practical value. However, as long as precise numerical predictions are not required, the logistic model may be used as an approximation to more complex models. The model is usually expressed in differential equation form:

$$\frac{dN}{dt} = rN\frac{(K-N)}{K} \tag{27.14}$$

where K = population carrying capacity.

In integral form, the model can be expressed as

$$\mathbf{N}_t = \frac{K}{1+e^{rt}} \tag{27.15}$$

When the size of the population is very small relative to its carrying capacity, the rate of growth of the population is close to the maximum possible rate r, which is the same intrinsic rate of population growth defined in Equation 27.1. The rate of population growth declines as the population grows, and approaches zero as the population approaches the carrying capacity, K. The logistic model can also be expressed as a recursive discrete-time equation, which may be useful for simulating recovery of a population from repeated disturbance events:

$$\mathbf{N}_{t+1} = \mathbf{N}_t + r\mathbf{N}_t\left(\frac{K-\mathbf{N}_t}{K}\right) \tag{27.16}$$

A disturbance is simulated by eliminating a fraction of the population present on the day of the disturbance. The population immediately begins growing again toward carrying capacity, until the next disturbance occurs. Barnthouse (2004) used both the continuous and the discrete forms of the logistic model to estimate approximate population recovery times for various aquatic organisms following simulated mortality due to agricultural chemical applications. Nakamaru et al. (2002) used a stochastic version of the continuous logistic model to

quantify the influence of DDT exposure on the probability of extinction of herring gull populations. Snell and Serra (2000) used a variant of the discrete logistic model to quantify the influence of generalized chemical exposures on the probability of extinction of rotifer populations. These studies are discussed later in this chapter.

27.2.4 Metapopulation Models

Many species, and in particular most terrestrial species, do not exist as continuous interbreeding populations. Instead, they consist of subpopulations inhabiting patches of suitable habitat interspersed among patches or regions of unsuitable habitat. All of these patches are subject to environmental variability. Small populations frequently become extinct, but new populations can be established in empty habitat patches by colonists arriving from other patches. This view of species as "metapopulations" was first formalized by Andrewartha and Birch (1954), although they did not use the term. Levins (1969) is credited with developing the first formal metapopulation model. He formulated a simple relationship between the fraction of habitat patches occupied by a species at any given time ($p(t)$), the rate of extinction of occupied patches (e), and the rate of production of propagules from each occupied patch (m). At any time t, the number of propagules produced is equal to the rate of production per occupied patch multiplied by the fraction of patches occupied. If each propagule has an equal probability of dispersing to occupied and unoccupied patches, a fraction equal to $(1 - p)$ of the propagules will colonize unoccupied patches. At the same time, a total number of patches equal to ep would become extinct. The rate of change in p at any time would be determined by the equation:

$$\frac{dp}{dt} = mp(1 - p) - ep \tag{27.17}$$

The equilibrium frequency of occupied patches (p^*) is determined by the ration of the extinction and colonization rates:

$$p^* = 1 - e/m \tag{27.18}$$

If extinction is more likely than dispersal (i.e., e is larger than m), extinction of the metapopulation is inevitable. This result is intuitively obvious, even without the model. What is not obvious, however, is that if e and m are nearly equal, the fraction of occupied patches can be expected to be very small, even if the rate of dispersal of propagules from occupied patches is very high. Under this circumstance, random variations in extinction and colonization rates can cause metapopulation extinction, even if under constant conditions the metapopulation would persist.

The above model is too simplistic to be of much value in the management of real populations. However, the fundamental processes and variables considered in the model, i.e., dispersal, extinction, and percent occupancy of available habitat, are central issues in conservation biology. In the 1980s, conservation biologists turned to metapopulation theory as a means of designing preservation strategies for vertebrate species that, although once widespread, were becoming restricted to isolated subpopulations because of increasing habitat fragmentation. Levins' original model has been extended to include influences of local population size, local population structure, spatial dispersal patterns, interspecies interactions, and population genetics. Hanski (1999) has provided an excellent overview of metapopulation ecology, including both theoretical and empirical aspects.

Metapopulation biology provides ecological risk assessors with both a conceptual framework and modeling techniques for addressing the effects of spatially variable chemical

exposures on populations inhabiting spatially heterogeneous environments. Maurer and Holt (1996) used a metapopulation model to demonstrate that pesticide applications can endanger the regional persistence of species by reducing the pool of sites available for colonization. Spromberg et al. (1998) developed a generalized metapopulation model based on an extension of Equation 27.17 and used it to examine the influence of the spatial arrangement and connectivity of patches on the response of the metapopulation to a toxic chemical that affects one of the patches. Chaumot et al. (2002, 2003) used a multipopulation extension of the Leslie matrix approach to model the impacts of cadmium discharges on a brown trout metapopulation inhabiting a hypothetical river network. This study is discussed in more detail later in the chapter.

27.2.5 Individual-Based Models

Ultimately, the health of a population is no more than a collective expression of the health of the individual organisms. The models discussed are at best abstractions that capture the general (we hope the essential) features of the biology of the organisms. Some, like the potential population growth rate model or the density-dependent Leslie matrix, are basically bookkeeping devices with which the deaths and births occurring during a given time are tabulated, while the biological mechanisms responsible for reproduction and mortality are ignored. All organisms within a given class (however defined) are assumed to be indistinguishable. Clearly, all organisms are *not* indistinguishable, and variations between individuals can have substantial influences on the responses of populations to anthropogenic stresses or management actions. Recognition of these problems has led to interest in "individual-based" models, i.e., models in which population dynamics are represented in terms of the physiological, behavioral, or other properties of the individual organisms. The general procedure is to develop a model of the individual organism to whatever level of detail is required, and then to infer the properties of the population as a whole either by analytical solution of equations or by numerical simulation of the activities of hundreds or thousands of individual organisms (Figure 27.2).

Individual-based models have made important contributions to understanding successional patterns in forests (Huston and Smith 1987), comparing the structure and development of different forest types (Shugart 1984), and predicting the effects of environmental stress on forest composition (Dale and Gardner 1987). A substantial number of applications to fish populations have also been published (Sperber et al. 1977; Adams and DeAngelis, 1987; DeAngelis et al. 1990; Madenjian and Carpenter 1991; Rose and Cowan 1993; Rose et al. 2003).

There are two broad approaches to developing individual-based models, of which one emphasizes Monte Carlo simulation and the other, analytical solutions to equations. The subtleties of the approaches and criteria for choosing one over the other have been discussed by Caswell and John (1992) and DeAngelis and Rose (1992). Elegant examples of the analytical approach have been published by McCauley et al. (1990) and Hallam et al. (1990). The principal advantage of the analytical approach is that results obtained are general, easy to verify, and easy to understand. The level of detail, however, must be compromised to achieve analytical tractability. In practice, the published biological applications of analytical individual-based models all deal with relatively simple organisms such as *Daphnia*.

The most widely known analytical individual-based models have emphasized physiological characteristics such as metabolism, growth, and chemical toxicodynamics. McCauley et al. (1990) developed a model of *Daphnia* growth and reproduction based on energetics and used the model to predict time-dependent changes in the age and size structure of *Daphnia*

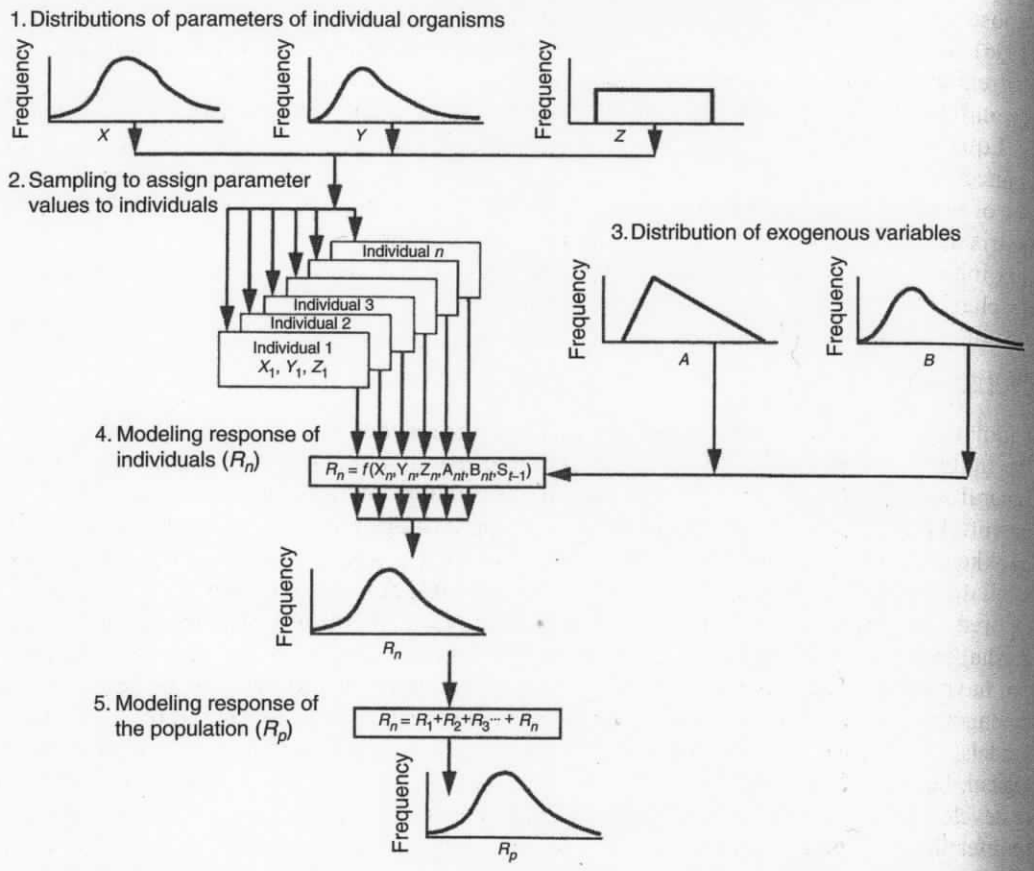

FIGURE 27.2 A schematic representation of individual-based population modeling. X_n, Y_n, and Z_n are characteristics of an individual organism n such as size or leaf area. A_{nt} and B_{nt} are characteristics of the environment experienced by individual n at time t such as temperature, pollutant concentrations, and prey availability. S_{t-1} is the state of the organism at the previous time step. R_n is the response of individuals such as death or maturation. R_p is the response of the population such as abundance or harvestable biomass.

populations in response to changes in food availability. Work on metabolism and toxicodynamics was pioneered by Kooijman and Metz (1984), who examined the influence of toxic chemicals on metabolism and population growth using *Daphnia* as a model organism. Hallam and Lassiter extended this approach to include (1) a thermodynamically based model of the uptake of contaminants from aqueous media, and (2) a definition of death in terms of the internal dissolved contaminant concentration within an organism (Hallam et al. 1990; Lassiter and Hallam 1990). Kooijman (2000) used the principles developed in these early studies as the basis of a formal framework for physiologically structured population modeling termed "Dynamic Energy Budget" (DEB) modeling. The DEB approach links physiological characteristics of the organisms to the growth rates and age distributions of populations, and also the exposure concentrations and modes of action of toxic chemicals to the physiological characteristics of the individuals. A software package (DEBtox; Kooijman and Bedaux 1996) has been developed for the purpose of estimating the chemical effects parameters used in DEB models from toxicity test data collected using standard Organization for Economic Cooperation and Development (OECD) protocols.

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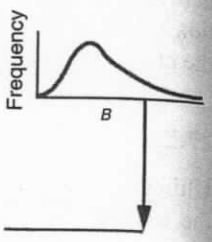

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lism and toxicody- influence of toxic organism. Hallam y based model of ath in terms of the :t al. 1990; Lassiter se early studies as ı modeling termed ysiological charac- ulations, and also the physiological and Bedaux 1996) eters used in DEB conomic Cooper-

The DEB approach is intended for application to aquatic organisms with relatively simple life cycles, inhabiting homogeneous environments. The models are very general in form, and analysis of the models emphasizes analytical investigation of the equations. An entirely different approach has been used to develop individual-based models of organisms with more complex life cycles, inhabiting more complex environments. DeAngelis et al. (1991) and Rose and Cowan (1993) developed models of fish populations that include metabolism, growth, foraging behavior, and prey selection as functions of the life stage and age of the fish. The approach followed in developing both of these models was to use the existing extensive literature on bioenergetics, reproduction, and foraging of individual fish, coupled with exhaustive evaluation of the life history of specific fish species, to develop detailed models of each life stage from egg through to reproductive adult. Population-level consequences of changes in the physiology, behavior, or reproduction of individual fish were inferred by brute-force simulation of the birth, growth, and death of hundreds or thousands of individual fish. The models were calibrated to extensive data sets collected for specific fish populations. This approach was later used by Jaworska et al. (1997a) to model the effects of simulated PCB exposures on young-of-the-year largemouth bass in southeastern US reservoirs. Jaworska et al. (1997b) used individual-based models of walleye and yellow perch populations to test whether causes of adverse changes in populations could be inferred from observed patterns of abundance, growth, and age structure. Rose et al. (2003) used an individual-based model of an Atlantic croaker population to link experimental data on the effects of PCB exposures on fecundity, egg survival, and larval predator avoidance ability in this species to population-level effects. This study is discussed in greater detail later in the chapter.

The metapopulation model VORTEX, originally described by Lacy (1993) and subsequently applied to a wide variety of endangered vertebrate populations, is fundamentally an individual-based model. The core of VORTEX is a stochastic model of the birth, growth, movement, reproduction, and death of each animal present in a population. The growth, decline, or extinction of a population is calculated by simulating the fate of each animal and its offspring for multiple generations. Estimates of the probability of persistence and expected time to extinction of the simulated populations are obtained by performing multiple runs in which random values of key parameters are drawn from prespecified statistical distributions.

Within the last decade, as the availability of Geographic Information System (GIS) technology has expanded, ecologists have used this technology as a basis for a new class of individual-based models termed spatially explicit models. In spatially explicit models, organisms are distributed over a realistic landscape composed of habitat patches of different types and suitability for utilization by the species of interest. The spatially explicit approach permits ecologists to integrate theory and observations on foraging behavior and reproduction in individual animals, relate these to specific measurable habitat characteristics, and infer influences of habitat change on populations.

Thorough and well-tested models of this type have specially been developed for populations of ungulates foraging in Yellowstone National Park (Turner 1993; Turner et al. 1994) and for the population of Bachmann's Sparrow nesting on the US Department of Energy's Savannah River site. Recently, an individual-based model of skylarks utilizing an agricultural landscape in Denmark has been used to compare the relative influences of pesticide applications and land use change on the abundance and persistence of this species (Topping and Odderskær 2004). This study is discussed in greater detail later in the chapter.

27.3 APPLICATIONS TO TOXIC CHEMICALS

Most of the modeling approaches (DEB modeling is an important exception) discussed were developed to address theoretical problems, to manage exploited populations, or to aid in the conservation of endangered species. However, interest in applying these approaches to

ecological risk assessment of toxic chemicals has grown over the past few years. In addition to numerous papers published in the scientific literature, three recent international workshops have addressed potential ecotoxicological applications of population models (Kammenga and Laskowski 2000; Baird and Burton 2001; Barnthouse et al. 2006). The following case studies provide examples of these applications.

27.3.1 Quantifying Uncertainties in Individual-to-Population Extrapolations

Barnthouse et al. (1987, 1988, 1990) developed a series of models that directly link toxicity test data to fish population models, and then used the combined models to evaluate the ecological implications of toxicity test data. Although this work was published nearly 20 y ago, it is still relevant today, for at least two reasons. First, it provides examples of applications of some of the extrapolation approaches discussed in Chapter 26 of this book. Second, it explores the relative uncertainties inherent in extrapolation of laboratory test data to effects on populations in the field in a way that has not yet been duplicated or superseded by more advanced methods.

Two different approaches to population modeling were used in these studies. In the first two papers of this series, estimates of the survival and reproduction parameters used in the Leslie matrix were used to calculate an "index of reproductive potential." The index was defined (Barnthouse et al. 1987) as the expected contribution of a female recruit (a 1-y-old female fish, in fisheries science terminology) to future generations of recruits, taking into account (1) her annual probability of survival (s_i), probability of being sexually mature (m_i), and age-specific fecundity (f_i); and (2) the probability that a spawned egg will hatch and survive to age 1 (s_0). The reproductive potential of a 1-y-old female recruit is given by

$$P = s_0 \sum_{i=1}^{n} s_i f_i m_i \tag{27.19}$$

Although Equation 27.19 contains the same parameters found in the Leslie matrix (Equation 27.6), the reproductive potential index is not used to calculate the future abundance or age composition of a population. Instead, it is used as a relative measure of the effect of changes in mortality or fecundity on a population, expressed as a fractional reduction in reproductive potential (R_s):

$$R_s = (P - P_s)/P \tag{27.20}$$

where P_s is the reproductive potential index in the presence of a stress that reduces survival, fecundity, or both. The value of P_s is calculated from

$$P_s = s_0(1 - C_m) \sum_{i=1}^{n} s_i (1 - C_r)^{i-1} f_i C_i m_i \tag{27.21}$$

where C_m = probability of stress-induced mortality during the first year of life; C_r = probability of stress-induced mortality for 1-y-old and older fish (assumed to be equal for all age classes); and C_f = proportional reduction in fecundity due to stress (assumed equal for all reproducing age classes).

The reproductive potential index was originally used to assess impacts of power plant cooling systems on fish populations (Barnthouse et al. 1986). Since the mid-1980s, a variant of the index termed the "spawning stock biomass per recruit" index has been widely used in marine fisheries management (Goodyear 1993).

The reproductive potential approach, like the density-independent Leslie matrix, cannot account for natural environmental variability or density-dependence. To explore the influence of these processes on responses of fish populations to toxic chemicals, Barnthouse et al. (1990) developed density-dependent, stochastic matrix projection models for two especially well-studied populations: the Gulf of Mexico menhaden population and the Chesapeake Bay striped bass population. The models employ conventional projection matrices, but with the survival coefficient for young-of-the-year fish (s_0) containing both density-dependent and randomly varying components. Estimates of the coefficients were obtained from published abundance, age structure, and mortality statistics for these two populations.

Survival of young-of-the-year fish was calculated using

$$s_0 = e^{-\alpha + R_i\sigma - 0.5\sigma^2 - \beta N_0} \tag{27.22}$$

where α = expected annual instantaneous rate of density-independent mortality; σ = standard deviation of α; R_i = a unit random normal deviate; and β = coefficient of density-dependence. Effects of chemicals on young-of-the-year are incorporated by replacing α in Equation 27.19 with

$$\alpha' = \alpha - \ln(1 - C_m) \tag{27.23}$$

where C_m = fraction of young-of-the-year expected to die from effects of chemical exposure. Effects of chemical exposure on fecundity were incorporated by multiplying each age-specific fecundity rate (f_i) in the population matrix by a fecundity reduction factor.

The contaminant effects factors were estimated from standard life-stage-specific toxicity data using concentration–response models and extrapolation models (inter-life-stage and interspecies) described earlier in this book. These procedures were used to develop exposure–response relationships that explicitly incorporate three types of uncertainty in lab-to-field extrapolations: test variability, species-to-species uncertainty, and acute-to-chronic uncertainty. The concentration–response function used in these analyses was the logistic model:

$$P = e^{a+BX}/(1 + e^{a+BX}) \tag{27.24}$$

where P = fractional response of the exposed population; X = exposure concentration; and a and B = fitted parameters with no direct biological interpretation. When fitted to concentration–response data, the logistic function has a sigmoid shape similar to the probit model. Concentration–response data sets were fitted to Equation 27.24 using nonlinear least-squares regression. Uncertainty concerning the shape and position of the concentration–response function, as reflected in the variances and covariances of a and B, were represented graphically as confidence bands surrounding the fitted functions.

Concentration–response functions specific to each life stage were combined to produce integrated functions that express the effects of chemical exposures, including uncertainty, on population-level response variables. An example is provided in Figure 27.3, which shows a concentration–response function for brook trout exposed to methylmercuric chloride (data from McKim et al. 1976), with female reproductive potential as a response variable. The maximum acceptable toxicant concentration (MATC) for the data set (calculated as the geometric mean of the no observed effect concentration (NOEC) and the lowest observed effect concentration (LOEC) and also known as the chronic value) corresponds to a 55% to 70% reduction in brook trout reproductive potential. This result, and other similar ones presented in Barnthouse et al. (1987), demonstrated that MATCs calculated from life cycle

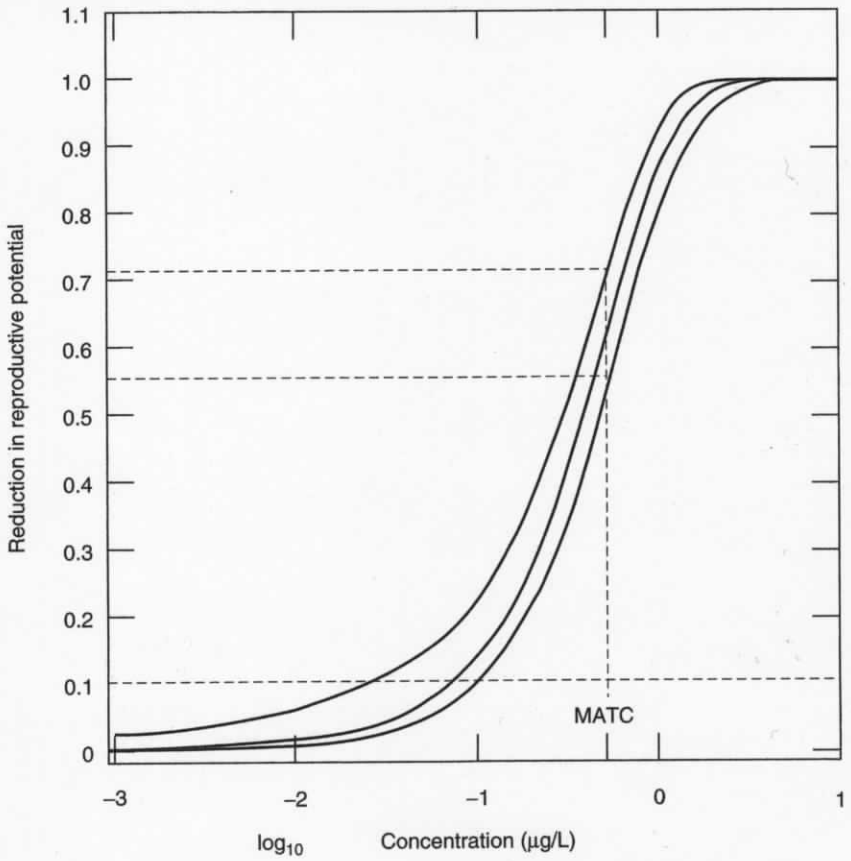
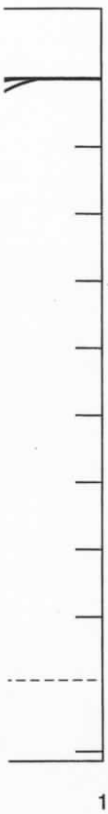


FIGURE 27.3 Concentration–response function and uncertainty band for the reduction in female reproductive potential of brook trout exposed to methylmercuric chloride. The lower dashed line denotes the 10% effect level (EC_{10}). The two upper dashed lines denote the 90% confidence band for the effects level associated with the maximum acceptable toxicant concentration (MATC). (From Barnthouse, L.W., Suter, G.W., II, Rosen, A.E., and Beauchamp, J.J., *Environ. Toxicol. Chem.*, 6, 811, 1987.)

toxicity tests often correspond to surprisingly high population-level effects and cannot in general be construed as ecological effects thresholds.

Barnthouse et al. (1987, 1988, 1990) used the above models for a variety of purposes. Extrapolated population responses were compared to MATCs derived from the same data sets, demonstrating that MATCs are not equivalent to population-level no-effects thresholds (Barnthouse et al. 1987, 1988). Comparisons of uncertainties associated with different test endpoints showed that fecundity responses are substantially more variable and introduce more uncertainty into risk assessments than do mortality responses (Barnthouse et al. 1988). Comparisons of uncertainty introduced in different extrapolation steps and toxicity test types were used to quantify the relative value for risk assessment of different toxicity testing strategies (Barnthouse et al. 1990). Comparisons between responses of menhaden and striped bass populations showed that, for typical screening-level assessments, uncertainties related to life history, environmental variability, and harvesting intensity would be negligible compared to toxicological uncertainties resulting from the use of quantitative structure–activity relationships and short-term toxicity tests to predict long-term population responses.



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27.3.2 Life History–Based Ecological Risk Assessment

Calow et al. (1997) and Forbes et al. (2001) proposed an approach for assessing population and community-level risks based on a combination of toxicity test data and simplified life history models. The model used in this approach was first described by Calow and Sibly (1990). It assumes a highly simplified life history involving only two stages: juveniles and adults. It has only five parameters: the fractions of juveniles surviving to first breeding (S_j); the fraction of adults surviving between breeding events (S_i); the time from birth to first breeding (t_j); the time between breeding attempts (t_a); and the number of offspring produced per individual at each breeding (n). Each organism is assumed to have a potentially infinite life span. Only female organisms are included in the model. Under these assumptions, Calow and Sibly (1990) derived a simple formula for calculating the finite population growth rate (8) from the discrete form of the fundamental population growth rate equation (Equation 27.3). The procedure involves reformulating the fundamental equation as an infinite sum, recalling that the survivorship term (l_x) in the equation can be expressed as the product of survival fractions from birth to age x:

$$\begin{aligned} 1 &= \sum_{x=1}^{\infty} \lambda^{-x} l_x m_x = \lambda^{-t_j} S_j n + \lambda^{-(t_j+t_a)} S_j S_a n + \lambda^{-(t_j+2t_a)} S_j S_a^2 n + \cdots \\ &= n S_j \lambda^{-t_j} \left(1 + \lambda^{-t_a} S_a + \lambda^{-2t_a} S_a^2 + \cdots\right) \end{aligned} \tag{27.25}$$

Substituting $y = \lambda^{-t_a} S_a$:

$$1 = n S_j \lambda^{-t_j} \left(1 + y + y^2 + y^3 + \cdots\right) \tag{27.26}$$

Because both λ^{-t_a} and S_a are limited to values between 0 and 1, y must also be a number between 0 and 1. A theorem from the mathematics of infinite series states that for any number y between 0 and 1:

$$\sum_{x=1}^{\infty} (1 + y^x) = \frac{1}{1-y} \tag{27.27}$$

Therefore, Equation (27.22) can be restated as

$$1 = \frac{n S_j \lambda^{-t_j}}{1-y} = \frac{n S_j \lambda^{-t_j}}{1 - \lambda^{-t_a} S_a} \tag{27.28}$$

which can be rearranged as

$$1 = n S_j \lambda^{-t_j} + S_a \lambda^{-t_a} \tag{27.29}$$

Calow et al. (1997) used this model to illustrate the influence of chemical toxicity on species with different general life history types: semelparous species (species that reproduce only once before dying, so that $S_a = 0$); moderately iteroparous species ($S_a = 0.5$); and strongly iteroparous species ($S_a = 0.9$). They examined the effects of chemicals that affected juvenile survival, reproduction, or adult survival on species with these life history types. Not surprisingly, reductions in juvenile survival or reproduction per breeding event had the greatest effect on λ for semelparous species and the least effect on strongly iteroparous species. Reductions in adult survival, on the other hand, had the greatest effect on strongly

iteroparous species and the least effect on semelparous species. The relative durations of the time to first reproduction (t_j) and the interval between reproductive events (t_a) also affected the responses of the model populations to reductions in survival or reproduction. The shorter the value of t_j relative to t_a, the greater is the sensitivity of λ to reductions in survival or reproduction.

The model described above is obviously quite simplistic, but it is also highly adaptable. Basic information on time-to-maturity, reproductive rate, and longevity are available for most types of organisms. Provided that dose–response models describing the relationships between chemical concentrations and effects on survival and reproduction can be developed, models relating chemical concentrations to changes in λ for different general life history type can be defined. Given information on the frequency distribution of different life history types present in a given ecosystem, "life history sensitivity distributions," analogous to the species sensitivity distributions (SSDs) described in Section 26.2.3, can be constructed.

Forbes et al. (2001) expanded on this idea in a subsequent paper, in which they compared hypothetical aquatic life protection criteria derived from population growth rate analysis to criteria derived using conventional toxicology-based approaches. In this paper, the authors defined four life cycle types representative of major organisms found in aquatic ecosystems: benthic invertebrates, fish, zooplankton (daphnids), and algae. They applied the life history model described above to each life history type, assuming that the long-term population growth rates (λ) for each organism type were approximately 1.0. They then investigated the influence of small changes in juvenile survival (S_j) on the population growth rate, using the elasticity index defined earlier in this chapter. Elasticities measure the proportional contribution of each life cycle parameter to the overall population growth rate. The authors found that the population growth rate of benthic invertebrates was the most elastic to small changes in juvenile survival, and that the population growth rate of daphnids was the least elastic. This means that, for any given decrease in survival caused by exposure to a contaminant, the corresponding reduction in λ would be greatest for the benthic invertebrate life cycle and least for the daphnid life cycle.

The authors then simulated a community composed of a mix of species with the four life history types, and compared protection strategies based on several conventional toxicity-based approaches to a protection strategy based on protection of population growth rates. The analysis assumed that the contaminant in question affected only juvenile survival (S_j). First, they developed a hypothetical dose–response function so that changes in juvenile survival could be translated into corresponding contaminant concentrations. For each life cycle type, they calculated the contaminant concentration corresponding to 10% reduction in λ. This value was assumed to be a safe concentration for each life cycle type, i.e., a population-level NOEC.

They then developed a series of scenarios in which both the relative toxicities of a hypothetical chemical to organisms with different life cycle types, and the relative contributions of different life cycle types to the total community, were varied. Variability in sensitivity among species within the same life cycle type was simulated by assuming a log-normal distribution of NOECs within each group. Monte Carlo simulation was used to calculate (1) the average NOEC for the community, i.e., the concentration at which the average value of 8 for all species was reduced by 10%; and (2) a 95% protection level for the community, i.e., the concentration at which only 5% of the species-specific NOECs is exceeded. These values were compared with protective concentrations developed using conventional toxicity-based standard-setting toxicological methods: an application factor method and an SSD-based method. Forbes et al. (2001) found that the protective concentration based on a 10% reduction in λ was, in general, substantially higher than the concentrations calculated using the application factor or the SSD. Hence, conventional risk assessment approaches appeared

to produce protective estimates of environmentally safe contaminant concentrations, and in some cases these approaches appeared to produce substantially overprotective concentrations. There were, however, some circumstances (e.g., a preponderance of species with a sensitive life cycle type and also high chemical sensitivity) in which the conventional approaches might produce underprotective concentrations. Forbes et al. (2001) concluded that more research on the relative contributions of different life history types to aquatic communities is needed to refine environmental protection protocols so that they provide truly protective, but not overprotective, estimates of safe exposure limits.

27.3.3 Quantifying Impacts of Chemical Exposures on Risk of Extinction

Snell and Serra (2000) used an empirically derived, stochastic model of temperate rotifer populations to simulate the impacts of a hypothetical chemical exposure on the long-term risk of population extinction. Their model is based on a density-dependent variant of the exponential growth model:

$$\mathbf{N}_t = \mathbf{N}_{t-1} e^{r_t} \tag{27.30}$$

Rather than being a constant as in Equation 27.2, the growth rate (r_t) is a function of previous population density and environmental variability. The authors obtained values for the parameters of the growth rate function by analyzing a time series of population density data for a natural rotifer population. Under most circumstances, rotifers are parthenogenetic (i.e., all individuals are female, and produce eggs that hatch into other females that produce only female eggs). At high densities, however, a fraction of the offspring produced comprises "mictic" females capable of sexual reproduction. Unfertilized eggs produced by mictic females hatch as male rotifers capable of fertilizing mictic females. The eggs produced from sexual reproduction are "resting" eggs, which fall to the sediment. Resting eggs are subject to mortality during both summer and winter, and they are the only life stage that survives through the winter. In the model simulation, the growing season for rotifers is assumed to last for 240 d, after which all individuals in the water column die. The following spring, the population in the water column is reestablished from resting eggs that hatch from the sediment. Only 10% of resting eggs hatch during any given spring, and those that do not hatch remain in the sediment as an "egg bank" that can potentially repopulate the water column for several years, and provide insurance against catastrophic events that may kill all rotifers in the water column.

Persistence and extinction in the model of Snell and Serra (2000) is determined by the density of resting eggs. If, during any year, the density of resting eggs falls below a critical value, the population is assumed to become extinct. The authors calculated extinction risks by simulating the fate of 1000 populations, all starting from the same initial population size, over a period of 100 y. They found that, even without chemical exposures, there is about a 5% extinction risk for a rotifer population over a 100 y period. The authors simulated three types of perturbations that could increase extinction risks: a continuous reduction in r caused by a chronic chemical exposure, an intermittent reduction in r caused by an intermittent chemical exposure, and a series of catastrophes occurring with different frequencies.

Effects of chemical exposures were simulated by multiplying each value of r_t by a constant fraction. The authors found that even a continuous reduction in r as small as 10% raised the extinction risk for a rotifer population to approximately 20%, and that a reduction in r of 30% or greater raised the risk to nearly 100%. The influence of intermittent reductions in r was simulated by assuming that r_t is reduced by 25% on a percentage of days ranging from 20% to 100%. Extinction risks were lower in the case of intermittent reduction than in the case of

continuous reduction; however, reducing r_t on only 40% of days still increased the 100 y extinction risk to 30%. Simulated chemical exposures also reduced the abilities of the model rotifer population to survive periodic catastrophes. In the absence of chemical exposures, a catastrophe frequency of once every other year increased the risk of extinction from 5% to about 25%. If, in addition, a 10% continuous reduction in r occurs due to chemical exposure, the extinction risk is raised to 60%.

According to the authors, the underlying reason for the sensitivity of their model to chemical exposures is the dynamics of the resting egg bank. A minimum number of resting eggs must hatch in the spring so that the density of parthenogenetic rotifers can increase rapidly to the threshold density required for sexual reproduction. Reducing r reduces the rate of growth toward the threshold, and also the number of resting eggs that can be produced before the end of the growing season. If insufficient resting eggs are produced, the size of the egg bank begins to fall, reducing the number available for hatching the following spring. This further extends the time required for growth to the sexual reproduction threshold, again reducing the number of resting eggs produced. Eventually, the egg bank declines below the extinction threshold.

Snell and Serra (2000) concluded, based on their analysis, that even small reductions in r, including levels that are often presumed to be safe in toxicity tests, can pose significant extinction threats to natural rotifer populations. They argued for increased use of population-based methods in risk assessments.

Nakamaru et al. (2002) used a different modeling approach to quantify extinction risks in a herring gull population exposed to DDT. These authors modeled population growth and extinction using a stochastic version of the logistic model (Equation 27.14):

$$\frac{dN}{dt} = rN\left(\frac{K-N}{K}\right) + \sigma_e \xi_e(t) \circ N + \sigma_d \xi_d(t) \cdot \sqrt{N} \tag{27.31}$$

In this equation the terms N, r, and K are defined in the same way as before, as population size, intrinsic rate of population growth, and carrying capacity. The additional terms in the equation represent environmental ($\sigma_e \xi_e$) and demographic ($\sigma_d \xi_d$) stochasticity. Environmental stochasticity is simply random variation in environmental factors affecting population growth. Demographic stochasticity is random variation in population due to the fact that, in stochastic models, births and deaths of individual organisms are random events. The symbols ($\circ$ and $\cdot$) used in the equation denote mathematical operations performed, using the "Stratonovich calculus" and the "Ito calculus," respectively. Readers interested in what these operations are and how they are performed should consult a good textbook on stochastic differential equations and be prepared for some heavy reading. The benefit of using a model like this rather than performing hundreds or thousands of Monte Carlo simulations is that mathematicians (in this case Hakoyama and Iwasa 2000) can derive an integrated equation that describes the influence of key model parameters on the risk of population extinction. The relationship between extinction time, carrying capacity, and environmental stochasticity is defined by

$$T = \frac{2}{\sigma_e^2} \int_0^K \int_0^\infty e^{-R(y-x)} \left(\frac{y+D}{x+D}\right)^{R(K+D)+1} \frac{1}{(y+D)y} dy dx \tag{27.32}$$

The term R represents the more complex term $2r/(\sigma_e^2 K)$ and the term D represents $1/\sigma_e^2$. The mean time to extinction (T) is defined in units of generation time. Hence, Equation 27.31 can be used to calculate the mean number of generations a population will persist before becoming extinct, given estimates of r, K, and σ_e^2.

How does DDT affect the risk of extinction of a herring gull population? Nakamura et al. (2002) addressed this question by modifying the parameters r and K in Equation 27.32 to include a reduction caused by DDT exposure:

$$r' = r - \alpha \tag{27.33}$$

$$K' = (r - \alpha)\, K/r \tag{27.34}$$

Although the mathematics may be abstruse, the conceptual basis of the model can be very simply summarized: mean time to extinction is a function of population growth rate, carrying capacity, environmental variability, and DDT exposure. DDT decreases both population growth rate and carrying capacity, thereby shortening the expected time to extinction.

The authors used data from herring gull populations in Long Island, New York, to illustrate how the model can be used. They used observed doubling times of newly established herring gull populations and measures of interannual variability in long-established populations to derive estimates of r and σ_e^2. They estimated a range of carrying capacities (K), corresponding to large and small herring gull populations. They estimated effects of DDT on r and K using a multistep procedure that considered both age-specific fertility in female herring gulls and influence of DDT exposures on female fertility. Using this information, together with published estimates of DDT biomagnification factors and historical concentrations of DDT in Long Island Sound, Nakamura et al. (2002) estimated that DDT exposures during the 1960s reduced r in herring gulls by approximately 20%.

Because herring gull populations are fairly large, and interannual variability in herring gull abundance is small compared to many other types of animals, Nakamura et al. (2002) found that the expected time to extinction for both exposed and unexposed herring populations was quite long. For example, for unexposed populations ranging in size from 100 to 100,000 adult females, mean extinction times ranged from 10^5 to 10^{30} generations. Since the generation time of herring gulls is approximately 8 y according to the authors, this means that a population consisting of only 100 birds would be likely to persist for approximately 1 million years.

Perhaps more interesting than the extinction risks themselves are the calculations performed by Nakamura et al. (2002) to compare risks due to DDT exposures with risks caused by habitat disturbance. The authors argued that, because chemical exposures and habitat disturbance both reduce the carrying capacity of a population, their model could be used to compare both types of disturbance in common units of reduced time to extinction. The method for making the comparison is quite simple in concept (although not so simple mathematically). For a specific value of α, corresponding to a specific concentration of DDT in the environment, Equation 27.32 is used to calculate the change in time to extinction caused by DDT exposure. Assuming that the carrying capacity of a population is directly proportional to the size of its habitat, one then uses a version of the model in which only K is reduced (rather than both r and K as in the case of DDT exposure) to calculate the reduction in K required to produce the same change in time to extinction.

The authors found that the equivalent habitat loss for any given DDT concentration is strongly dependent on the size of the exposed population. For example, according to Table 2 in Nakamura et al. (2002), a water-column DDT concentration of 0.1 ng/L would cause the same change in time to extinction for a population of 100 herring gulls as would a 50.5% reduction in habitat area. For a population of 100,000 birds, a reduction in habitat area of 96.5% would be required to produce the same change in time to extinction.

Nakamura et al. (2002) qualified their results on the grounds that many of their parameter values were only approximations and that herring gulls are a very abundant species with a very low risk of extinction at present. They argued, however, that their method could be used

to calculate mitigation requirements (e.g., habitat protection or enhancement) needed to protect endangered species exposed to DDT or other hazardous substances.

27.3.4 Quantifying Impacts of Chemicals on Metapopulations

Maurer and Holt (1996) investigated the impacts of pesticide exposures on spatially distributed populations in which some subpopulations reside in habitats that are exposed to pesticides and other subpopulations reside in unexposed or "safe" habitats. If migration of organisms occurs between the safe habitats and the exposed habitats, it is possible in theory for the exposed habitats to become "sinks" that reduce the size of the population in the safe habitat, perhaps ultimately resulting in the extinction of the entire population. Typical agricultural landscapes consist of mosaics of vegetation types, some of which are treated with pesticides and others of which are not. If applying these chemicals to only a fraction of the vegetation patches in a region could still cause extinction of nontarget populations throughout the entire region, protocols for field-testing of pesticides would need to consider this possibility. The authors used two alternative metapopulation models to determine conditions under which this phenomenon could occur.

The first model used by Maurer and Holt (1996) is based on two coupled discrete-time equations that describe births, deaths, and migrations within and between safe and exposed habitat patches:

$$N_s(t) = N_s(t-1) + r_s N_s(t-1) - mN_s(t-1) + mN_e(t-1) \tag{27.35}$$

$$N_e(t) = N_e(t-1) + r_e N_e(t-1) + mN_s(t-1) - mN_e(t-1) \tag{27.36}$$

The terms r_s and r_e are net rates of population growth within safe and exposed habitats, respectively, accounting for both births and deaths. Since organisms in the exposed habitat should be declining in number, the growth rate (r_e) in that habitat is assumed to be negative. The term m refers to the rate of migration of organisms between habitats, assumed to be the same for both habitat types. The change in number of organisms within each habitat from time $(t-1)$ to time (t) is thus equal to the number present at time $(t-1)$ plus the number born in the habitat or migrating in, minus the number dying or migrating out.

The growth rate of the entire population in both habitats can be calculated by writing Equation 27.35 and Equation 27.36 as a matrix equation and then using the methods of matrix algebra (Caswell 2001) to find the dominant eigenvalue. The resulting population growth rate is given by

$$\lambda = \frac{2 + r_s + r_e - 2m + \sqrt{(r_s - r_e)^2 + 4m^2}}{2} \tag{27.37}$$

As long as the rate of population decline in the exposed habitat is smaller than, or equal to, the rate of population growth in the safe habitat (i.e., $|r_e| \leq r_s$), λ will be greater than 1 and the population will increase. However, if the rate of decline in the exposed habitat is greater than the rate of growth in the safe habitat, the population will decline to extinction if the migration rate (m) is too high. If $|r_e| > r_s$, the threshold value for m, above which the population will decline to zero, is given by

$$m = \frac{r_s r_e}{r_s + r_e} \tag{27.38}$$

To account for the possibility that density-dependent survival or reproduction might stabilize the population and permit persistence under conditions in which the above model predicts extinction, Maurer and Holt (1996) investigated an alternative model, based on the logistic equation:

$$\frac{dN_s}{dt} = r_s N_s\left(1 - \frac{N_s}{K_s}\right) - mN_s + mN_e$$
$$\frac{dN_e}{dt} = r_e N_e + mN_s - mN_e \tag{27.39}$$

Note that density-dependence is assumed to occur only in safe habitat. The population in the exposed habitat would, as in the density-independent model, decline toward zero in the absence of immigration from the safe habitat.

In this model, because of density-dependence, the total population will stabilize at an equilibrium value determined by the values of the growth rate, migration rate, and carrying capacity parameters. The population persists if and only if this equilibrium population size is greater than zero. Maurer and Holt (1996) found that the condition for an equilibrium population size greater than zero is exactly the same as the condition for a positive value of λ in the density-independent model.

These results, according to the authors, have important and counterintuitive implications for the design of pesticide application programs. The authors noted that in both models the likelihood that the total population will be able to persist is a decreasing function of the migration rate between habitats. Hence, the ability of organisms to replenish exposed habitats through migration actually increases the risk that a regional population inhabiting a mosaic of treated and untreated habitats will become extinct. Persistence is more likely if little or no migration occurs between treated and untreated habitats. Moreover, the likelihood of persistence declines as the population growth rate in the safe habitat declines. The implication of this result is that species with very high maximal growth rates, including many pest species, can persist in the face of localized pesticide applications under conditions in which species with lower growth rates, including nontarget vertebrate species, might become extinct. On the basis of their analyses, Maurer and Holt (1996) concluded that typical approaches to pesticide risk assessment that emphasize measurement of effects in laboratory studies and test applications to individual fields are inadequate because they fail to consider the spatial structure of exposed populations.

Chaumot et al. (2002, 2003) used a multiregion matrix population model to investigate the responses of a hypothetical spatially distributed population of brown trout to cadmium discharges affecting a river network. In both studies, the trout population was distributed among 15 hierarchically organized compartments representing a network of first- through fourth-order stream segments. Three life stages were represented: alevins (age 0 trout), juveniles, and adults. Trout were assumed to migrate seasonally between compartments, with spawning occurring in the first-order segments, and the different age groups of trout distributed during the nonspawning season according to age distributions observed in field data. In the first paper (Chaumot et al. 2002), all trout in any compartment had the same probability of migrating during the spring season. In the second paper (Chaumot et al. 2003), the complete mixing assumption was relaxed to account for the observation that some trout did not migrate, and both a spring and a fall migration season were modeled. In the first paper, survival and reproduction were represented in an extended version of the Leslie matrix (Equation 27.6):

$$\mathbf{L} = \begin{bmatrix} 0 & 0 & \mathbf{FP}_H \\ \mathbf{S}_1 & 0 & 0 \\ 0 & \mathbf{S}_2 & \mathbf{S}_3 \end{bmatrix} \tag{27.40}$$

In Equation 27.40, the elements $\mathbf{S}_1$, $\mathbf{S}_2$, and $\mathbf{S}_3$ are diagonal matrices containing compartment-specific survival rates for each life stage. The element $\mathbf{FP}_H$ is a matrix containing compartment-specific fecundities and migration probabilities. The corresponding matrix in the second paper is similar, but includes additional terms resulting from the more complex representation of the trout life cycle.

In both papers, effects of cadmium on brown trout were modeled using dose–response data from laboratory toxicity tests. Life-stage-specific concentration–response curves were derived using a logistic regression approach similar to the method used by Barnthouse et al. (1987, 1988, 1990). The matrix elements were then modified by multiplying the stage-specific fecundity and mortality rates by reduction coefficients derived from the concentration–response functions. The first paper considered only chronic exposures; the second considered both chronic and acute exposures.

In both studies, the authors used the elasticity analysis to determine the effects of cadmium exposure on the population growth rate, as a function of the location and intensity of a hypothetical discharge. In addition, they calculated the effects of hypothetical discharges on the age structure and spatial distribution of trout. In the second paper, the authors showed how discharges of cadmium into different levels of the stream hierarchy could have greatly different impacts on the spatial distribution of trout while having exactly the same effect on the population growth rate.

The authors made no claims concerning the management implications of their results, other than arguing that their approach could be adapted to a wide variety of river network types, be made highly site-specific, and be used to model other stresses in addition to toxic chemicals.

27.3.5 Individual-Based Models

Topping and Odderskær (2004) described an individual-based model of a skylark population inhabiting an agricultural landscape in Denmark. The purpose of the study was to evaluate the influence of pesticides in relation to weather and agricultural practices as influences on skylark populations.

The landscape component of the model is a spatially explicit GIS-based system that includes three types of farms, each of which has its own characteristic crop rotation pattern, and detailed rules for simulating the sequence of activities (e.g., pesticide application, watering, plowing, sowing) that occur on each farm. Roadside vegetation, hedgerows, and other noncultivated areas are also included in the model landscape.

The skylark component of the model simulates the behavior of individual birds, including territory establishment, foraging, nest-building, incubation, and rearing. Each bird is modeled as an "agent" that engages in various behaviors contributing to survival and reproductive success according to sets of decision rules that take into account the bird's size, age, location, nest status, and other characteristics. The influence of weather on skylark reproduction is simulated by treating bad weather conditions (cold, wind, and heavy rain) as categorical variables (i.e., either present or absent during a given half-day time step), with the probability distributions of bad weather events determined from local meteorological records. Skylark foraging success is influenced by vegetation structure, which is determined by vegetation type and growth rate (explicitly simulated in the landscape model), and by the available insect biomass within a given vegetation type.

Topping and Odderskær (2004) developed parameter sets for the model based on landscape data for a specific region in central Jutland, Denmark, and evaluated the impacts of pesticide application, field size, crop heterogeneity, and weather on the skylark population present in the modeled region. Calculated population-level endpoints included total population size, number of breeding pairs, and total number of birds successfully fledged.

The pesticides and herbicides typically used in the modeled region have relatively low direct toxicity to birds. These chemicals were assumed to affect skylarks indirectly, through reductions in the availability of arthropod prey. Field size and crop heterogeneity affect skylarks by influencing the diversity of vegetation types and growth stages present in the territory of a typical bird, which in turn influence arthropod abundance and foraging success. Weather acts directly on the birds, influencing both incubation time, fledgling mortality, and foraging success.

Skylark populations were simulated over five 11 y weather cycles under four combinations of pesticide application patterns (used and not used) and field sizes (large and small). Impacts of crop heterogeneity were evaluated by assuming that only a single crop (spring barley) is cultivated throughout the region.

Results of the simulation showed that, although pesticide applications affected skylark abundance, the effects of pesticides were small compared with effects of landscape structure and weather. Doubling the average field size reduced mean skylark abundance over the 55 y simulation period by 37%. Applying pesticides to the fields using label-specified application rates and frequencies reduced skylark abundance by only about 4%. Variations in weather conditions between years resulted in annual variations in fledgling production between +19% and −13% compared with long-term average fledgling production. Assuming that only barley is grown in the region resulted in dramatically lower skylark abundance, including extinction of some or all model skylark populations.

The authors concluded that, although pesticide use has potentially adverse impacts on skylark populations in central Denmark, agricultural practices have a much greater influence. Intensification of farming practices, including increased farm size and decreased diversity of crops appear to be a more significant threat.

Rose et al. (2003) used an individual-based model to link laboratory observations of effects of PCBs on Atlantic croaker to field data on coastwide abundance trends in this species. The experiments providing the laboratory observations measured effects of PCBs on female fecundity, egg survival, larval swimming speed, and larval predator avoidance ability. The dynamics of the coastwide population were simulated using a matrix projection model similar to those discussed elsewhere in this chapter. Adults were assumed to spawn in the mid-Atlantic bight; larvae were assumed to migrate to nursery areas in North Carolina and Virginia, and to return to the ocean as juveniles.

The effects of PCBs on the behavior of Atlantic croaker larvae, which occur on time scales of seconds to hours, were linked to effects on the coastwide population, which occur on time scales of months to years, using a model of the feeding, growth, and mortality of individual Atlantic croaker larvae. The laboratory experiments measured effects of PCBs on larval avoidance behavior and swimming speed. Larvae exposed to PCBs through maternal transfer from PCB-exposed adult females were found to swim more slowly and respond less actively to simulated predator attacks than were unexposed larvae. Rose et al. (2003) used a statistical method referred to as a "regression tree" to translate the responses observed in the experiment to a probability that a larva encountering a predator would escape predation. Reduced swimming speed, in addition to reducing the probability that a larva would escape predation, would be expected to reduce the rate at which the larva would encounter zooplankton prey organisms. Hence, PCB exposure would be expected to result in reduced prey consumption, slower growth, and potential mortality due to starvation. The individual-based model used by

Rose et al. (2003) simulates the daily activities of a larval Atlantic croaker from hatching to transformation to the juvenile stage. It includes a bioenergetics submodel, a foraging submodel, and a predation submodel. Parameters for these submodels are derived from a variety of laboratory and field studies of larval fish. This same approach has been used in a number of other individual-based fish population models (e.g., DeAngelis et al. 1991; Rose and Cowan 1993; Rose et al. 1999). PCB effects were incorporated in the model through effects of predator avoidance and swim speed on the daily rates of mortality and growth. The daily mortality and growth rates, in turn, were used to modify the mortality and stage duration parameters used in the matrix projection model.

The matrix projection model was calibrated by adjusting the juvenile-stage mortality rates so that, in the absence of PCB exposures, the population would be stable, with interannual variability in juvenile abundance being similar to the variability observed in long-term monitoring data collected in Virginia and North Carolina. PCB exposures were simulated assuming that juveniles reared in North Carolina nursery areas are exposed during juvenile development, with effects expressed when the females spawn 1 to 2 y later. Fecundity and egg survival in the model were reduced by values observed in the laboratory experiments. The growth, development, and survival of PCB-exposed larvae were followed during the estuarine nursery phase of the life cycle, with the predator avoidance and swim speed parameters reduced according to the results of the regression tree analysis.

Two exposure scenarios were evaluated. In the first, females were assumed to be impaired only during their first spawning, and to completely depurate their PCBs during that spawning event. In the second, females were assumed to be impaired throughout their entire lifetime.

Rose et al. (2003) found that, under the first-time-only scenario, the predicted effects of PCBs on the long-term abundance of Atlantic croaker were negligible. Under the lifetime impairment scenario, long-term average abundance was about 10% lower than the baseline. In addition to demonstrating that their model could be used to link behavioral toxicity data to long-term effects on a population, the authors suggested that in the future, a better-verified version of the model could be used to quantify cumulative impacts of multiple stresses, such as PCB exposures combined with increased harvesting.

27.4 FUTURE OF POPULATION MODELING IN ECOLOGICAL RISK ASSESSMENT

The examples discussed in this chapter cover a wide range of applications of population models in ecological risk assessment. The first edition of this textbook suggested that projection matrices, stochastic extinction models, and individual-based models would in the future be used in ecological risk assessments performed to support Superfund assessments, pesticide risk assessments, natural resource damage assessments, and other types of regulatory activities. Research applications are clearly widespread, but applications of population models in chemical risk assessment and management are still relatively uncommon.

The US Environmental Protection Agency's (US EPA) recent guidance on endpoints for ecological risk assessments (EPA 2003) identifies several population-level endpoints (extirpation, abundance, and production) as being relevant to the agency's assessments; however, the agency's primary focus is still on organism-level attributes such as morphological anomalies, survival, reproduction, and growth (Chapter 16). The agency justifies this emphasis on the grounds of legal requirements, regulatory precedents, and practicality. Laws and regulations are beyond the scope of assessment science; however, practicality is an issue that can be directly addressed through research, demonstration, and guidance development.

A recent workshop on population-level ecological risk assessment (Barnthouse et al. 2006) identified a number of steps that could be taken to increase the use of population-level methods in ecological risk assessments. The most obvious of these steps is development of guidance documents explaining the availability, use, and interpretation of common modeling tools. Such guidance would cover the selection of models suitable for different types of assessment problems, methods for parameter estimation, and rules for model use and interpretation. Guidance on field data collection relevant to population-level assessment would likely also be needed. Beyond technical guidance, broader guidance intended to inform risk managers, and stakeholders concerning how, and under what circumstances, population-level assessment tools can lead to better environmental decisions. The workshop report includes a framework for population-level ecological risk assessment analogous to the well-known framework in the EPA's guidelines (EPA 1998a), and also contains recommendations for incorporating population-level considerations in risk management decisions.

Training and education are also important. Risk assessment practitioners would benefit from enhanced opportunities for training in population ecology theory, empirical field and laboratory methods, and GIS technology. Beyond training programs, perhaps the best single educational activity would be actual application of population models in one or several high-profile assessments. There can be no doubt that the widespread development and use of metapopulation models in conservation biology were enhanced by the use of these models to assess impacts of habitat fragmentation on the northern spotted owl. Similar case-specific applications involving environmental chemicals would educate both risk managers and the assessment practitioners themselves concerning the benefits, limitations, and proper use of population models in ecological risk assessments.

Even though population models are not yet being routinely used in ecological risk assessments, the integration of ecotoxicology and population biology that was envisioned by the authors of this book 20 y ago has occurred. Approaches originally developed for use in resource management and conservation biology and even for purely theoretical purposes are now being applied in ecotoxicology. Equally important, young scientists and assessment practitioners are entering the field with the training and expertise needed to understand, use, and advance these approaches.

Future editions of this book, or maybe successors to this book, will very likely discuss concrete regulatory applications of population models, and will provide specific recommendations concerning the uses of these models based on successes and failures in on-the-ground applications.