Population ecology

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The study of spatial and temporal patterns in the abundance and distribution of organisms and of the mechanisms that produce those patterns. Species differ dramatically in their average abundance and geographical distributions, and they display a remarkable range of dynamical patterns of abundance over time, including relative constancy, cycles, irregular fluctuations, violent outbreaks, and extinctions. The aims of population ecology are threefold: (1) to elucidate general principles explaining these dynamic patterns; (2) to integrate these principles with mechanistic models and evolutionary interpretations of individual life-history tactics, physiology, and behavior as well as with theories of community and ecosystem dynamics; and (3) to apply these principles to the management and conservation of natural populations.

Definition of a population

A population is the total number of individuals of a given biological species found in one place at one time. In practice, ecologists often deal with density—numbers per unit area for land organisms and numbers per unit volume in aquatic systems—or even weight rather than raw numbers. What may be described as an "individual" depends on the kind of organism and the aim of the scientific enquiry. In most animals, the life cycle starts with a fertilized egg, passes through a largely irreversible process of coupled growth and differentiation, and ends in a tightly integrated, unitary, adult organism. Population size can be measured by merely counting adult units and their juvenile prologues. But in most plants and some colonial animals, growth and differentiation proceed in a modular fashion; growth involves the replication of a basic body unit, so that a fertilized egg generates a spatially distributed "population" of connected modules. Modular organisms show tremendous plasticity in size and form. The connections between modules sometimes break, leading to a dispersed clone of physiologically independent units. For example, one clone of quaking aspen (which spreads by root buds) occupies 200 acres (80 hectares) and is more than 10,000 years old. To a geneticist, this clone constitutes a single huge individual; to an ecologist, each aspen trunk may be considered to be an individual. Modular organisms have an additional dimension of complexity that must be quantified to understand their dynamics. In this article, the population concepts presented apply broadly to both unitary and modular organisms.

There are only four ways a population can change in size: birth, death, immigration, and emigration. If immigration and emigration are negligible, the population is closed, and the difference between birth and death rates drives its dynamics. Terrestrial animals on islands often have closed populations. If immigration and emigration are important, however, the population is open, and its abundance may be substantially influenced by spatially distant events. For example, the number of barnacles that are found on a rocky coastline often reflects

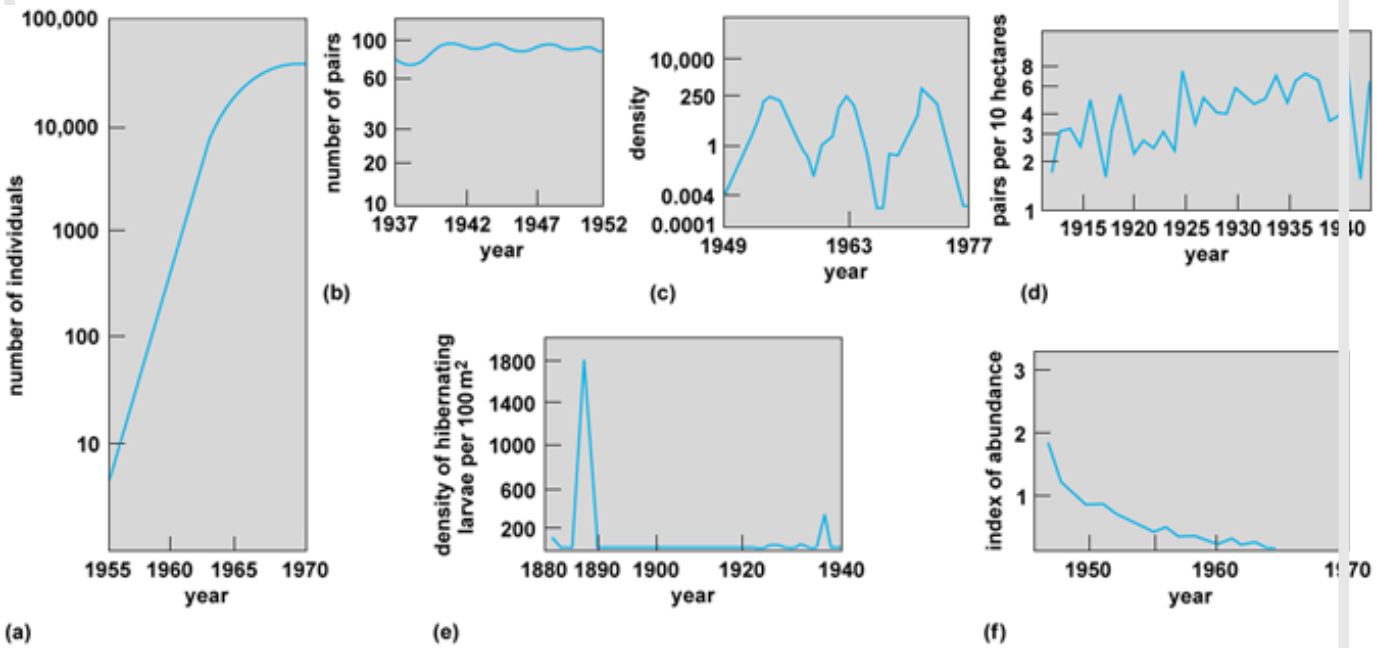

Fig. 1 **Graphs of dynamical behaviors showing the diversity of population dynamics. (*a*) Collared dove; a phase of exponential growth asymptoting toward carrying capacity. (*b*) Yellow-eyed penguin; steady state. (*c*) Budmoth; regular cycles. (*d*) Songbird; irregular fluctuations. (*e*) *Dendrolimus* moth; outbreaks. (*f*) Blue whale; decline toward extinction.**

the density of setting larvae, which in turn is governed by events in offshore waters. If a population that is under study is found to be highly open, the spatial scale of the study may be too narrowly circumscribed to capture the important mechanisms of its population dynamics.

Basic population models

Populations exhibit a great variety of dynamical patterns, ranging from explosive outbreaks, to local extinctions, to regular cycles or relatively constant abundances (**Fig. 1**). To help describe and explain these patterns, ecologists rely on population models. Simple life cycles and closed populations provide a useful starting point in developing population models. Many temperate-zone insects have one annual generation, and so at any given time all individuals are at the same stage of life. For a population with discrete, nonoverlapping generations, if $N(t)$ is the number of adults censused in generation t, and $R(t)$ is the number of adult offspring in generation $t + 1$ produced per adult in generation t, the number of individuals in the next generation is given by Eq. (1).

$$N(t+1) = N(t)R(t) \tag{1}$$

The quantity $R(t)$ is the growth rate of the population for generation t. Iterating this discrete time growth model for subsequent generations allows one to project population numbers through time. In the limit of very small census intervals, population growth is described by the differential equation (2),

$$\frac{dN}{dt} = r(t)N(t) \tag{2}$$

where $r(t)$, the instantaneous per-capita growth rate, is the difference between per-capita birth and death rates. Equation (2) is literally true only if populations grow continuously with overlapping generations (as is approximately true for some microbes). Constant values for R or r imply exponential growth. If $R > 1$ ($r > 0$), a population grows without bounds; if $R < 1$ ($r < 0$), it declines to extinction. The theoretical framework of population ecology largely consists of elaborations of these basic growth models, including extensions to more complicated life cycles and multiple species.

Exponential growth has a snowballing effect: if the growth rate is constant, then the more individuals there are, the faster the population grows. Even low growth rates eventually lead to populations of enormous sizes. Some natural populations show transient phases of exponential growth, particularly in colonizing episodes: for instance, the collared turtledove invaded Great Britain in 1955 and increased exponentially for nearly a decade (Fig. 1*a*). The per-capita rate of growth during exponential growth is called the intrinsic rate of increase, r_0. The value of r_0 quantitatively expresses the interplay of individual traits, such as life history strategies, with the environment.

Exponential growth during colonization often involves expansion across space as well as an increase through time. Equation (2) can be expanded to include immigration and emigration, as given in Eq. (3).

$$\frac{dN}{dt} = rN + (\text{net change due to dispersal}) \tag{3}$$

When individuals move down spatial density gradients, which is analogous to chemical diffusion along concentration gradients, their rate of movement is characterized by a diffusion coefficient, D. In a homogeneous environment, this model predicts that the expanding population wave advances at a velocity $2(rD)^{1/2}$, implying a linear expansion in range area with time. Data from a number of colonizing populations show exactly this relationship.

Population heterogeneity

Models such as those given in Eqs. (1) and (2), when interpreted literally, assume that all members of a population are identical. This is rarely true. Birth and death rates typically vary as a function of age, body weight, and genotype. A great deal of work in population ecology is devoted to elucidating age-specific schedules of mortality and fecundity, using these patterns to predict population growth, and interpreting these patterns in the light of evolutionary theory. To study age-structured population dynamics, the number of individuals in each age class must be monitored. The two ingredients needed to project changes in population size and age structure are the mortality schedule or survivorship curve, which describes the fraction of newborns surviving to each age, and the fecundity schedule, which describes the rate of female births per female at each age. It is a formidable task to measure complete fecundity and mortality schedules in natural populations, but if these schedules are given, the geometric growth model of Eq. (1) can be generalized to a matrix model, as in Eq. (4),

$$N(t+1) = A(t)N(t) \tag{4}$$

where $N(t)$ is a vector in which each element is the number of individuals in an age class, and $A(t)$ is a matrix incorporating the fecundity and mortality schedules. When individuals can be categorized into discrete stages such as body size (for example, number of connected modules in a clonal organism) in addition to age, more general matrices can describe population growth; the matrix elements are rates of transition between each pair of stages. [The continuous time model of Eq. (2) can be similarly generalized by using partial differential equations.] A fundamental principle of demographic theory is that if these schedules are constant, a population will (with rare exceptions) eventually settle into a stable age distribution in which each age class comprises a constant fraction of the total population. A population in its stable age distribution grows geometrically, as in Eq. (1), at a rate of increase uniquely determined from the mortality and fecundity schedules. A population displaced from its stable age distribution may exhibit transient phases of growth or decline, divergent from its long-term growth pattern.

All populations are genetically variable. If different genotypes have different fecundity or mortality schedules, genetic variation can influence population dynamics. Models that simultaneously incorporate changes in genetic composition and population growth can be quite complex; however, they may be important in describing some populations and are necessary for linking population ecology with evolutionary theory.

Population limitation and regulation

Populations cannot expand exponentially forever. For instance, the growth rate of the collared dove substantially declined in the second decade of its invasion, probably because mortality rates rose or birth rates declined as a result of competition for limited resources such as food or nest sites. Such mechanisms are called negatively density-dependent factors. The notion of density dependence is a specific example of the more general concept of feedback. An alternative hypothesis is that the environment worsened for reasons unrelated to dove density,

such as a shift in the weather. Such causes for variation in birth or death rates are called density-independent factors. Ecologists have long disputed the relative importance of density-dependent and density-independent factors in determining population size. The current consensus is that both are important but to differing degrees in different species and environments. For a population to be regulated, it must tend to increase when below a certain size and decrease when above that size. If growth rates vary with time but in a fashion unrelated to density, closed populations will eventually fluctuate to extinction or expand without limit. If a closed population persists over long periods of time, it must be regulated to some degree.

As illustrated below, population regulation by no means implies population stability. In general, a population is stable if it returns to equilibrium following a perturbation. Moreover, many local populations may not be persistent over long periods of time and so may not be regulated in the usual sense. Open populations, by definition coupled by dispersal with other populations, can become reestablished by immigration following a local extinction. The total population of a species may persist, even though no single local population survives, because there is a spreading of risk among an ensemble of local populations (which experience somewhat different environmental conditions) that are loosely coupled by dispersal. *See also:* POPULATION DISPERSAL.

A useful method for considering the interplay of density-dependent and density-independent factors in determining population size is to plot birth and death rates as functions of density (**Fig. 2**). The carrying capacity *K* of a population in a given environment is defined to be the largest number of individuals for which the birth rate just matches the death rate. The population decreases above *K* and increases below *K*. A given change in density-independent death rates can produce very different changes in population size, depending on the form of the underlying density dependence. If density dependence is weak (Fig. 2*b*), fluctuations in mortality generate large oscillations in population size; if density dependence is strong (Fig. 2*c*), the population readily buffers such fluctuations. Density-dependent factors are necessary to regulate populations, but density-independent factors must also be considered to understand fully what limits populations to a given value of *K*.

Evidence for density dependence

Statistical analyses of time series of population data can suggest density dependence, but it is difficult to demonstrate density dependence conclusively by using such data. Sometimes density dependence can be shown for particular stages in the life cycle, and in general, density dependence may be observed in births, deaths, or dispersal. However, without examining density dependence at each stage in the life cycle, it is difficult to infer the regulatory importance of any single stage. Stronger evidence comes from manipulative experiments in which control populations are compared with artificially enhanced or depressed populations.

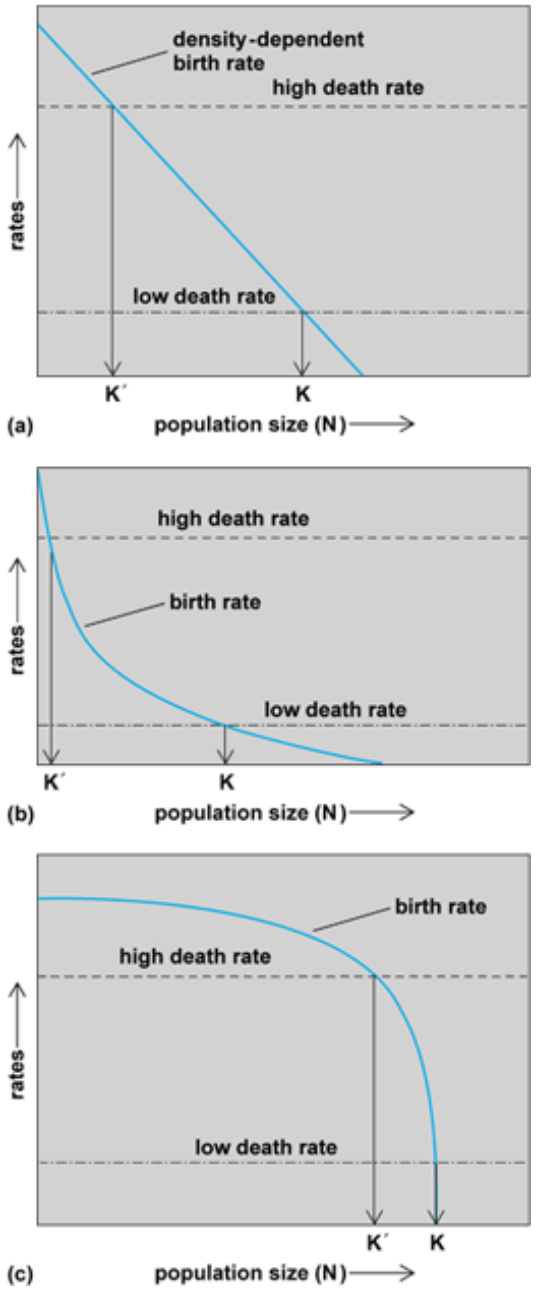

Fig. 2 **Carrying capacity as a reflection of both density dependence and the intensity of density-independent mortality. Births are considered to be purely density-dependent, deaths purely density-independent. The three curves show the effects on population size of (*a*) an increase in density-independent mortality (a high death rate), (*b*) weak density dependence (near *K*), and (*c*) strong density dependence (near *K*).**

Density-dependent population models

The exponential growth model [Eq. (2)] can be modified to include density dependence by expressing r as a function of N. The simplest model that generalizes Eq. (2) is the logistic equation (5),

$$\frac{dN}{dt} = r_0 N \left(1 - \frac{N}{K}\right) \tag{5}$$

in which per-capita growth rate declines linearly with increasing density. Populations displaced from K converge smoothly back to it, without overshoots. Population growth is maximal when $N = K/2$. This model provides a good fit to some laboratory colonies and captures much of the qualitative behavior of more complex models; it is, however, a somewhat crude first approximation to accurate models of population growth.

The logistic model can be improved upon in numerous ways. The simplest method is to use a nonlinear function for the per-capita growth rate. Further refinements in the model can be achieved by incorporating threshold effects and explicitly incorporating submodels that encapsulate the mechanisms of density dependence. At densities far below carrying capacity, there may be an Allee effect—a positive relationship between density and per-capita growth rates. One intriguing possibility that arises in more complex models is that the population may exhibit alternative stable states; the one it actually occupies will depend upon accidents of history. For instance, insects may be regulated at low densities by bird predation, but at a higher threshold density the birds may be satiated and thus no longer regulate insect density; the insect population will then grow until checked by some other factor.

The discrete-time model [Eq. (1)] can be similarly modified to incorporate density dependence by expressing R as a function of N. Analysis of such models has led ecologists to reevaluate their traditional assumptions about the causes of population fluctuations in nature and about the relationship between regulation and stability. For instance, an analog of Eq. (5) is Eq. (6),

$$N(t+1) = N(t) \exp\left[r\left(1 - \frac{N}{K}\right)\right] \tag{6}$$

which, along with similar equations, reveals a rich array of dynamical patterns. If $r < 2$, the population equilibrates stably at K; if $2 < r < 2.7$, the population fluctuates cyclically; if $r > 2.7$, the population exhibits chaotic behavior, with cycles of arbitrary periodicity or even aperiodic fluctuations. Such fluctuations are intriguingly similar to the fluctuations in real-world data, which in the past were assumed to be produced by random environmental noise. The qualitative properties of the model that trigger pronounced population fluctuations are the time lag implicit in the discrete-time formulation, high growth rates at low densities, and

strong density dependence at high densities. This suggests that a potential for complex dynamical behavior exists whenever there are time lags in the feedback between population size and population growth rates. Age structure is a ubiquitous source of time lags in populations, simply because time is required to reach reproductive maturity. Similarly, interactions between two, three, or more species can introduce long time lags, together with strong density dependence. It is an open question whether observed variability in natural populations reflects to any significant extent the complex dynamics latent in deterministic growth models instead of the force of fluctuations in the physical environment.

Mechanisms of density dependence

Given that density dependence exists, the mechanisms generating it can be used both to predict the consequences of environmental change for population dynamics and to provide insight into systems where experimental manipulations are difficult. Density dependence often arises from competition, which is said to exist when organisms utilize common limiting resources and thereby negatively affect each other. (A resource is limiting if an increase in its supply increases per-capita growth rates.)

There are two principal sorts of competition, interference and exploitative. Interference competition occurs when one individual directly harms another. Interference may be dramatic, as in lethal aggression, or subtle, as when social interactions reduce the time available for gathering resources or increase the risk of predation. A surprising number of animal species are cannibalistic. Large scorpions, for instance, eat with relish their smaller-bodied conspecifics. Because encounter rate increases with increasing population size, cannibalism is likely to be a potent density-dependent factor in scorpion populations. Exploitative competition occurs when one individual consumes a resource such as food that otherwise would have been consumed by another individual. Because exploitative competition is mediated indirectly through a shared resource base, it can be more difficult to demonstrate than interference. In territorial animals, such as many songbirds, less space is available for additional territory holders as population size increases. As a result, competition for space can sharply cap population numbers.

Population regulation and interspecific interactions

Negative density dependence may arise from interspecific interactions. A schematic classification of interactions between two species comes from considering the positive (+) or negative (−) effect that individuals of one species have on the growth rate of the other. In interspecific competition the interaction is (−,−); in mutualism it is (+,+). Natural enemies, defined broadly to include predators, herbivores, and parasites, are often engaged in (+,−) relations with their prey or hosts. Most species are potential prey to one or more natural enemies; even top-level carnivores may be beset by parasites.

Obviously, competitors and mutualists can dramatically affect the size of a given population and so must be considered when studying population limitation. However, natural enemies are far more likely to be regulatory

agents than are either competitors or mutualists. If two species are competing and one increases in density, the other will decrease. This will relax the interspecific competition on the first, which can then increase even more. Hence, competitive loops (and for similar reasons, mutualist loops) tend to produce positive feedback and so will not regulate population growth. By contrast, predator–prey interactions may produce negative density dependence acting across several time scales on both the predator and prey. As prey in one habitat patch become more numerous, predators may almost immediately become more active or switch over from other prey types or patches. Predators may also show an intergenerational numerical response to increased prey availability. Because an increase in predator numbers usually decreases prey numbers, this induces delayed density dependence in both the predator and its prey. In host–pathogen systems, these numerical responses may be pronounced, even within a host generation. For example, in tropical trees, seedling mortality from fungal pathogens increases with increasing seedling density, because the rate of spore dispersal increases as the distance between hosts declines. There is mounting evidence that parasites are significant regulatory factors in a wide range of natural populations, including economically important forest insects and game animals.

Several cautionary remarks about natural enemies and density dependence are in order. Predation and intraspecific competition can interact in complex ways. Compensatory responses by prey populations sometimes diminish the impact of enemies. For instance, plants typically produce many more seeds than can possibly become reproductive adults. Predation on some seeds may simply enhance the survivorship of the nonconsumed seeds, leading to no net effect on adult numbers. Conversely, predation can intensify intraspecific competition in mobile animals by restricting them to limited refuges. Generalist natural enemies can greatly depress prey numbers without being important regulatory agents, because by depending on many prey types they are less likely to show a strong numerical response to any one. Indeed, generalist predators may often be destabilizing, driving local prey populations to extinction. Finally, density-dependent responses by natural enemies often involve time lags, setting up the possibility of oscillatory behavior. Host–pathogen systems seem particularly likely candidates for generating strongly cyclic or chaotic dynamics.

Population growth models can incorporate interspecific interactions by taking models such as those in Eqs. (5) and (6) and adding expressions that describe how competitors, mutualists, or natural enemies affect the growth rate of a given species. The dynamics of two or more coupled species is studied in theoretical community ecology, which among other things seeks to understand how the species richness and stability of communities is related to the pattern and strength of interspecific interactions. Analyses of models of interacting species suggest that strong intraspecific density dependence is required for community stability. For two competing species to persist at a stable equilibrium, the negative effect that each species exerts on its own growth must on average exceed the cross-species negative effects. In like manner, predator–prey interactions are most stable when one or both populations experience intraspecific density dependence.

Applied population ecology

In addition to its intrinsic conceptual appeal, population ecology has great practical utility. Control programs for agricultural pests or human diseases ideally attempt to reduce the intrinsic rate of increase of those organisms to very low values. Analyses of the population dynamics of infectious diseases have successfully guided the development of vaccination programs. In the exploitation of renewable resources, such as in forestry or fisheries biology, population models are required in order to devise sensible harvesting strategies that maximize the sustainable yield extracted from exploited populations. Conservation biology is increasingly concerned with the consequences of habitat fragmentation for species preservation. Population models can help characterize minimum viable population sizes below which a species is vulnerable to rapid extinction, and can help guide the development of interventionist policies to save endangered species. Finally, population ecology must be an integral part of any attempt to bring the world's burgeoning human population into harmonious balance with the environment. *See also:* ECOLOGY; MATHEMATICAL ECOLOGY; THEORETICAL ECOLOGY.

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