three trophic levels (plant–herbivore–predator), emphasizing the importance not only of direct but also of indirect effects that a species may have on others on the same trophic level or on levels below or above it.

9.2 Multiple determinants of the dynamics of populations

fluctuations in abundance are caused by a wide variety of biotic and abiotic factors

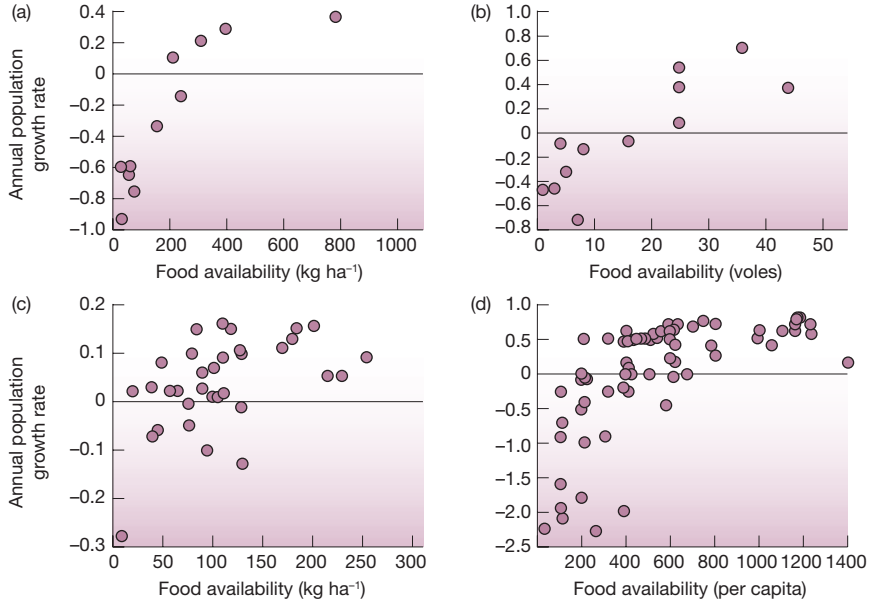
Why are some species rare and others common? Why does a species occur at low population densities in some places and at high densities in others? What factors cause fluctuations in a species' abundance? These are crucial questions when we wish to conserve rare species, or control pests, or manage natural, living resources, or when we wish simply to understand the patterns and dynamics of the natural world. To provide complete answers for even a single species in a single location, we need to know the physicochemical conditions, the level of resources available, the organism's life cycle and the influence of competitors, predators, parasites and so on – and how all these factors influence abundance through effects on birth, death, dispersal and migration. We now bring these factors together and consider how we might discover which actually matter in particular examples.

what total numbers can and cannot tell us

The raw material for the study of abundance is usually some estimate of the numbers of individuals in a population. However, a record of numbers alone can hide vital information. Picture three human populations, shown to contain identical numbers of individuals. One is an old people's residential area, the second is a population of young children and the third is a population of mixed age and sex. In the absence of information beyond mere numbers, it would not be clear that the first population was doomed to extinction (unless maintained by immigration), the second would grow fast but only after a delay and the third would continue to grow steadily. The most satisfactory studies, therefore, estimate not only the numbers of individuals (and their parts, in the case of modular organisms) but also those of different age, sex and size.

what correlations can and cannot tell us

The data that accumulate from estimates of abundance may be used to establish correlations with external factors like food or weather. Correlations may be used to predict the future. For example, high intensities of the disease 'late blight' in potato crops usually occur 15–22 days after a period in which the minimum temperature is above 10°C and relative humidity is more than 75% for two consecutive days. Such a correlation may alert the potato grower to the need for protective spraying. Correlations may also suggest – but not prove – causal relationships. For example, a correlation may be demonstrated between the size of a population and its growth rate. But ultimately 'cause' requires a mechanism. When the population is large, many individuals may starve to death, or may fail to reproduce, or may become aggressive and drive out the weaker members. A correlation cannot tell us which. Nonetheless, correlations can be informative. Figure 9.2, for example, shows four examples in which population growth rate increases with the availability of food. It also suggests that in general, such relationships are likely to level off at the highest food levels where some other factor or factors place an upper limit on abundance.



AFTER SIBLY & HONE, 2002

Figure 9.2

Increases in annual population growth rate with the availability of food, measured as pasture biomass (kg ha^{-1}) in (a) and (c), as vole abundance in (b) and as availability of food per capita in (d). (a) Red kangaroo (Bayliss, 1987). (b) Barn owl (modified from Taylor, 1994). (c) Wildebeest (Krebs et al., 1999). (d) Feral pig (Choquenot, 1998). Positive growth rates indicate increasing abundance; negative growth rates decreasing abundance.

9.2.1 Fluctuation or stability?

many populations are very stable . . .

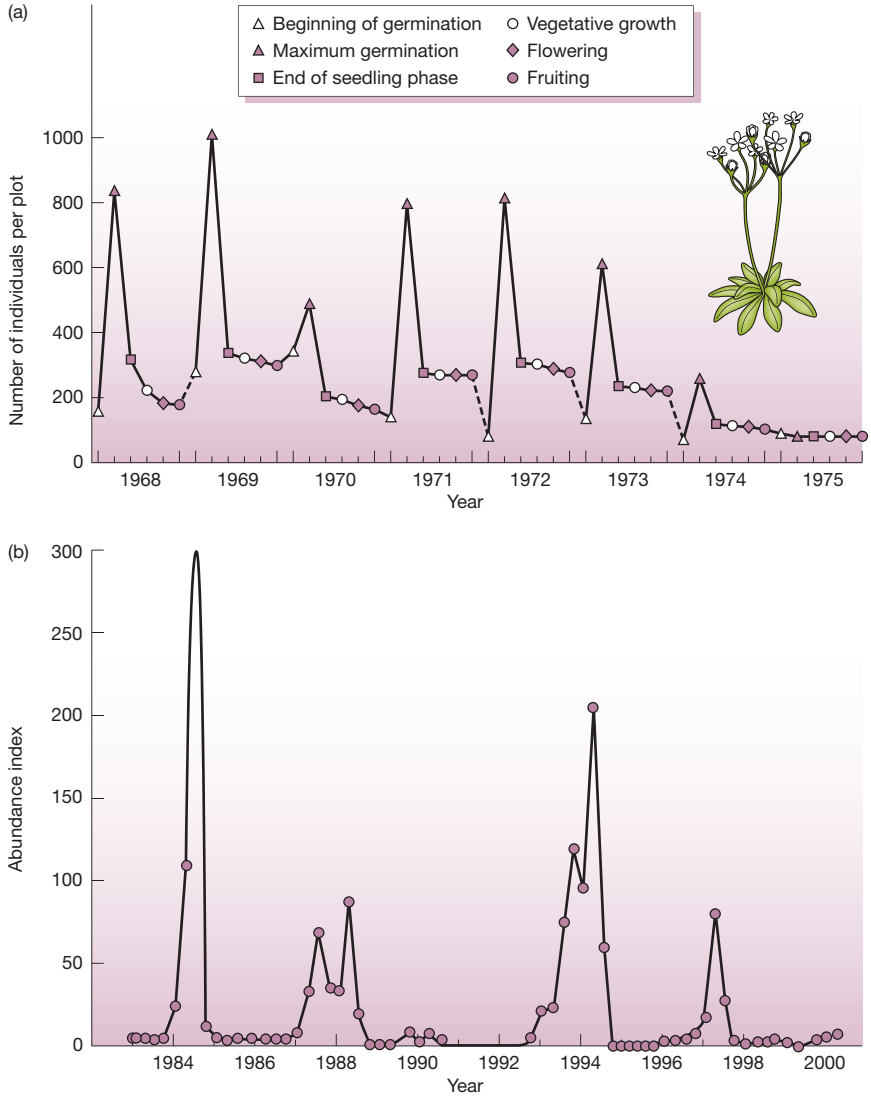
Some populations appear to change very little in size. One study that covered an extended timespan – though it was not necessarily the most scientific – examined swifts (*Micropus apus*) in the village of Selborne in southern England over more than 200 years. In one of the earliest published works on ecology, Gilbert White, who lived in the village, wrote in 1778 (see White, 1789):

> I am now confirmed in the opinion that we have every year the same number of pairs invariably. . . . The number that I constantly find are eight pairs, about half of which reside in the church, and the rest in some of the lowest and meanest thatched cottages.

More than 200 years later, Lawton and May (1984) visited the village and, not surprisingly, found major changes. Swifts are unlikely to have nested in the church for 50 years, and the thatched cottages have either disappeared or had their roofs covered with wire. Yet the number of breeding pairs of swifts regularly to be found in the village is now 12. In view of the many changes that have taken place in the intervening centuries, this number is remarkably close to the eight pairs so consistently found by White.

. . . but stability need not mean 'nothing changes'

But the stability of a population may conceal complex underlying dynamics. Another example of a population showing relatively little change in adult numbers from year to year is seen in an 8-year study in Poland of the small, annual sand-dune plant *Androsace septentrionalis* (Figure 9.3a). Each year, however, there was great flux within the population. Between 150 and 1000 new seedlings per square meter appeared, but subsequent mortality reduced the population by between 30% and 70%. Thus, the population appears to be kept within bounds. At least 50 plants always survived to fruit and produce seeds for the next season. By contrast, the mice in Figure 9.3b have extended periods of relatively low abundance interrupted by sporadic and dramatic irruptions.



(a) AFTER SYMONIDES, 1979; (b) AFTER SINGLETON ET AL., 2001

Figure 9.3

(a) The population dynamics of *Androsace septentrionalis* during an 8-year study. (b) Irregular irruptions in the abundance of house mice (*Mus domesticus*) in an agricultural habitat in Victoria, Australia, where the mice, when they irrupt, are serious pests. The 'abundance index' is the number caught per 100 trap-nights. In the fall of 1984 the index exceeded 300.

9.2.2 Determination and regulation of abundance

Is the move from eight to 12 pairs of swifts over 200 years an indication of consistency or of change? Is the similarity between eight and 12 of most interest – or the difference between them? Some investigators have emphasized the apparent constancy of populations; others have emphasized the fluctuations.

Those who have emphasized constancy argue that we need to look for stabilizing forces within populations to explain why the populations do not exhibit unfettered increase or a decline to extinction (generally, density-dependent forces: for instance, competition between crowded individuals for limited resources). Those who have emphasized fluctuations often look to external factors, weather or disturbance, to explain the changes. Can the two sides be brought together to form a consensus?

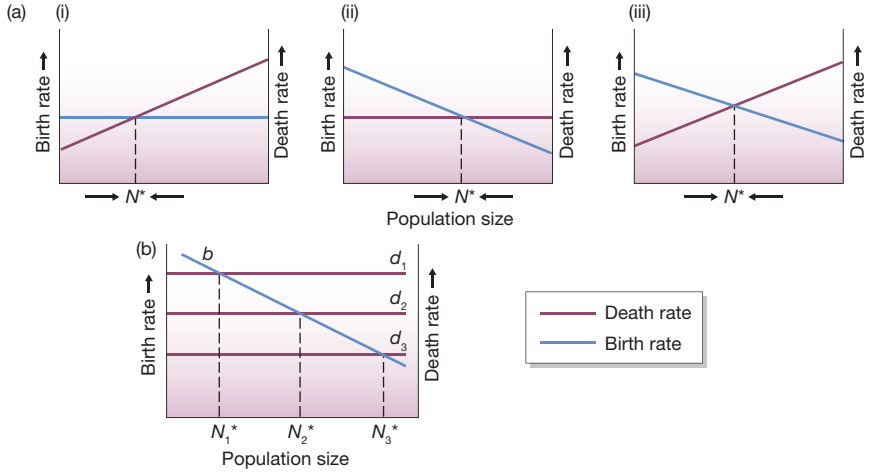


Figure 9.4

(a) Population regulation with: (i) density-independent birth and density-dependent death; (ii) density-dependent birth and density-independent death; and (iii) density-dependent birth and death. Population size increases when birth rate exceeds death rate and decreases when death rate exceeds birth rate. N^* is therefore a stable equilibrium population size. The actual value of the equilibrium population size is seen to depend on both the magnitude of the density-independent rate and the magnitude and slope of any density-dependent processes. (b) Population regulation with density-dependent birth, b, and density-independent death, d. Death rates are determined by physical conditions which differ in three sites (death rates d_1, d_2 and d_3). Equilibrium population size varies as a result (N_1^*, N_2^*, N_3^*).

the distinction between determination and regulation

To do so, it is important to understand clearly the difference between questions about the ways in which abundance is *determined* and questions about the way in which abundance is *regulated*. Regulation is the tendency of a population to decrease in size when it is above a particular level, but to increase in size when below that level. In other words, regulation of a population can, by definition, occur only as a result of one or more density-dependent processes (see Chapters 3 and 5) that act on rates of birth and/or death and/or movement (Figure 9.4a). Various potentially density-dependent processes have been discussed in earlier chapters on competition, predation and parasitism. We must look at regulation, therefore, to understand how it is that a population tends to remain within defined upper and lower limits.

On the other hand, the precise abundance of individuals will be determined by the combined effects of all the factors and all the processes that affect a population, whether they are dependent or independent of density (Figure 9.4b). We must look at the determination of abundance, therefore, to understand how it is that a particular population exhibits a particular abundance at a particular time, and not some other abundance.

In the past, certainly, some have believed that density-dependent, biotic interactions play the main role not only in regulating but also in determining population size, holding populations in a state of balance in their environments. Others have felt that most natural populations could be viewed as passing through a repeated sequence of setbacks and recovery. This view tends to reject any subdivision of the environment into density-dependent and density independent 'factors', preferring instead to see populations as sitting at the

center of an ecological web, where various factors and processes interact in their effects on the population.

both are perfectly valid interests

There is really no conflict between the two views. The first is preoccupied with what regulates population size and the second with what determines population size – and both are perfectly valid interests. No population can be absolutely free of regulation – long-term unrestrained population growth is unknown, and unrestrained declines to extinction are rare. Furthermore, any suggestion that density-dependent processes are rare or generally of only minor importance would be wrong. A very large number of studies have been made of various kinds of animals, especially of insects. Density dependence has by no means always been detected but it is commonly seen when studies are continued for many generations. For instance, density dependence was detected in 80% or more of studies of insects that lasted for more than 10 years (Hassell et al., 1989; Woiwod & Hanski, 1992).

On the other hand, for many populations weather is typically the major determinant of abundance and other factors are of relatively minor importance. For instance, in one famous, classic study of a pest, apple thrips, weather accounted for 78% of the variation in the number of thrips (Davidson & Andrewartha, 1948); for predicting thrips' abundance, information on the weather is of paramount importance. So, what regulates the size of a population need not determine its size for most of the time. It would be wrong to give regulation or density dependence some kind of pre-eminence. It may be occurring only infrequently or intermittently, and it is likely that no natural population is ever truly at equilibrium: even when regulation is occurring, it may be drawing abundance toward a level that is itself changing in response to changing levels of resources. Thus, there are a range of possibilities: some populations in nature are almost always recovering from the last disaster (Figure 9.5a), others are usually limited by an abundant resource (Figure 9.5b) or by a scarce resource (Figure 9.5c), and others are usually in decline after sudden episodes of colonization (Figure 9.5d).

9.2.3 Key factor analysis

We can distinguish clearly between what regulates and what determines the abundance of a population, and see how regulation and determination relate to one another, by examining an approach known as *key factor analysis*. It has been applied to many insects and some other animals and plants and is based on calculating what are known as *k*-values for each phase of the life cycle. In fact, key factor analysis is poorly named, since it identifies key *phases* (rather than key factors) in the life of a study organism (those most important in determining abundance). Details are described in Box 9.1, but the approach can be understood simply by appreciating that the *k*-values measure the amount of mortality: the higher the *k*-value, the greater the mortality (*k* stands for 'killing power').

Colorado potato beetles

For a key factor analysis to be carried out, data are compiled in the form of a life table (see Chapter 5), such as that done for a Canadian population of the Colorado potato beetle (*Leptinotarsa decemlineata*) in Box 9.1. The sampling program in that case provided estimates of the population at seven stages: eggs, early larvae, late larvae, pupae, summer adults, hibernating adults and spring adults. One further category was included, females 2, to take account of any unequal sex ratios among the summer adults.

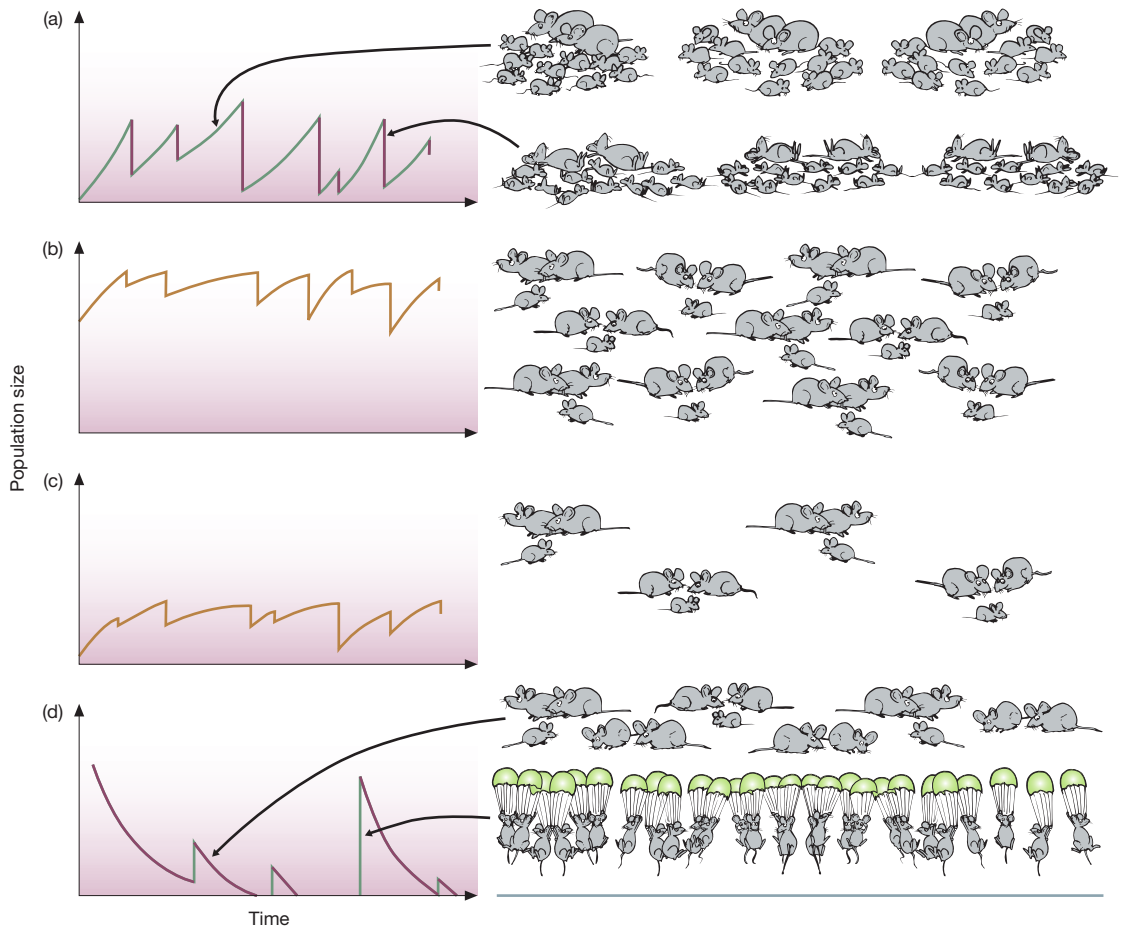


Figure 9.5

Idealized diagrams of population dynamics: (a) dynamics dominated by phases of population growth after disasters; (b) dynamics dominated by limitations on environmental carrying capacity, where the carrying capacity is high; (c) same as (b) but where the carrying capacity is low; (d) dynamics within a habitable site dominated by population decay after more or less sudden episodes of colonization or recruitment.

9.1 Quantitative aspects

9.1 QUANTITATIVE ASPECTS

Determining *k*-values for key factor analysis

Table 9.1 sets out a typical set of life table data, collected by Harcourt (1971) for the Colorado potato beetle, *Leptinotarsa decemlineata*, in Canada. The first column lists the various phases of the life cycle. *Spring adults* emerge from hibernation around the middle of June, when potato plants are breaking through the ground. Within 3 or 4 days egg laying begins, and it continues for about 1 month. The eggs

Table 9.1

Life table data for the Canadian Colorado potato beetle.

AGE INTERVAL	NUMBERS PER 96 POTATO HILLS	NUMBERS DYING	MORTALITY FACTOR	FACTOR $LOG_{10}N$	*k*-VALUE	
Eggs	11,799	2,531	Not deposited	4.072	0.105	(k_{1a})
	9,268	445	Infertile	3.967	0.021	(k_{1b})
	8,823	408	Rainfall	3.946	0.021	(k_{1c})
	8,415	1,147	Cannibalism	3.925	0.064	(k_{1d})
	7,268	376	Predators	3.861	0.023	(k_{1e})
Early larvae	6,892	0	Rainfall	3.838	0	(k_2)
Late larvae	6,892	3,722	Starvation	3.838	0.337	(k_3)
Pupal cells	3,170	16	Parasitism	3.501	0.002	(k_4)
Summer adults	3,154	–126	Sex (52%♀)	3.499	–0.017	(k_5)
Females 2	3,280	3,264	Emigration	3.516	2.312	(k_6)
Hibernating adults	16	2	Frost	1.204	0.058	(k_7)
Spring adults	14			1.146		
					2.926	(k_{total})

are laid in clusters (approximately 34 eggs) on the lower leaf surface, and the larvae crawl to the top of the plant, where they feed throughout their development, passing through four stages. When mature, they drop to the ground and form pupal cells in the soil. *Summer adults* emerge in early August, feed, and then re-enter the soil at the beginning of September to hibernate and become the next season's spring adults.

An adult Colorado potato beetle (*Leptinotarsa decemlineata*) taking off from its host plant. Emigration by summer adults represents the key phase in the population dynamics of potato beetles.

The next column lists the estimated numbers (per 96 potato hills) at the start of each phase, and the third column then lists the numbers dying in each phase, before the start of the next. This is followed, in the fourth column, by what were believed to be the main causes of deaths in each stage of the life cycle. The fifth and sixth columns then show how *k*-values are calculated. In the fifth column, the logarithms of the numbers at the start of each phase are listed. The *k*-values in the sixth column are then simply the differences between successive values in column 5. Thus, each value refers to deaths in one of the phases, and, similarly to column 3, the total of the column refers to the total death throughout the life cycle. Moreover, each *k*-value measures the rate or intensity of mortality in its own phase, whereas this is not true for the values in column 3 – there, values tend to be higher earlier in the life cycle simply because there are more individuals 'available' to die. These useful characteristics of *k*-values are put to use in *key factor analysis*.

Table 9.2

Summary of the life table analysis for Canadian Colorado beetle populations (see Box 9.1).

		MEAN	COEFFICIENT OF REGRESSION ON k_{TOTAL}
Eggs not deposited	k_{1a}	0.095	−0.020
Eggs infertile	k_{1b}	0.026	−0.005
Rainfall on eggs	k_{1c}	0.006	0.000
Eggs cannibalized	k_{1d}	0.090	−0.002
Egg predation	k_{1e}	0.036	−0.011
Larvae 1 (rainfall)	k_2	0.091	0.010
Larvae 2 (starvation)	k_3	0.185	0.136
Pupae (parasitism)	k_4	0.033	−0.029
Unequal sex ratio	k_5	−0.012	0.004
Emigration	k_6	1.543	0.906
Frost	k_7	0.170	0.010
	k_{total}	2.263	

AFTER HARCOURT, 1971

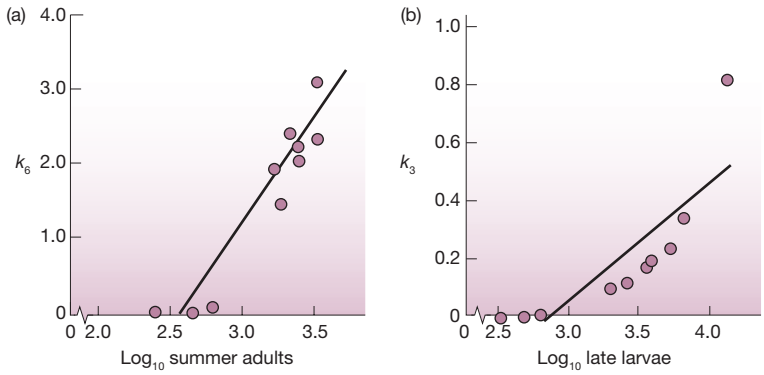
when does most mortality occur?

The first question we can ask is: 'How much of the total "mortality" tends to occur in each of the phases?' (Mortality is in inverted commas because it refers to all losses from the population.) The question can be answered by calculating the mean *k*-values for each phase, in this case determined over 10 seasons (that is, from 10 tables like the one in Box 9.1). These are presented in the third column of Table 9.2. Thus, here, most loss occurred amongst summer adults – in fact, mostly through emigration rather than mortality as such. There was also substantial loss of older larvae (starvation), of hibernating adults (frost-induced mortality), of young larvae (rainfall) and of eggs (cannibalization and 'not being laid').

the phases that determine abundance . . .

It is usually more valuable, however, to ask a second question: 'What is the relative importance of these phases as determinants of year-to-year *fluctuations* in mortality, and hence of year-to-year fluctuations in abundance?' This is rather different. For instance, a phase might repeatedly witness a significant toll being taken from a population (a high mean *k*-value), but if that toll is always roughly the same, it will play little part in determining the particular rate of mortality (and thus the particular population size) in any particular year. In other words, this second question is much more concerned with discovering what *determines* particular abundances at particular times, and it can be addressed in the following way.

Mortality during a phase that is important in determining population change – referred to as a *key phase* – will vary in line with total mortality in terms of both size and direction. It is a key phase in the sense that when mortality during it is high, total mortality tends to be high and the population declines – whereas when phase mortality is low, total mortality tends to be low and the population tends to remain large, and so on. By contrast, a phase with a *k*-value that varies quite randomly with respect to total *k* will, by definition, have little influence on changes in mortality and hence little influence on population size. We need therefore to measure the relationship between phase mortality and total mortality, and this is achieved by the *regression coefficient* of the former on the latter. The largest regression coefficient will be associated with the key phase causing population change, whereas phase mortality that varies at random with total mortality will generate a regression coefficient close to zero.



AFTER HARCOURT, 1971

Figure 9.6

(a) Density-dependent emigration by Colorado beetle summer adults (slope = 2.65).
(b) Density-dependent starvation of larvae (slope = 0.37).

In the present example (Table 9.2), the summer adults, with a regression coefficient of 0.906, are the key phase. Other phases (with the possible exception of older larvae) have a negligible effect on the changes in generation mortality.

. . . and the factors that regulate abundance

What, though, about the possible role of these phases in the *regulation* of the Colorado beetle population? In other words, which, if any, act in a density-dependent way? This can be answered most easily by plotting *k*-values for each phase against the numbers present at the start of the phase. For density dependence, the *k*-value should be highest (that is, mortality greatest) when density is highest. For the beetle population, two phases are notable in this respect: for both summer adults (the key phase) and older larvae there is evidence that losses are density-dependent (Figure 9.6) and thus a possible role of those losses in regulating the size of the beetle population. In this case, therefore, the phases with the largest role in determining abundance are also those that seem likely to play the largest part in regulating abundance. But as we see next, this is by no means a general rule.

two further examples of key factor analysis

Key factor analysis has been applied to a great many insect populations, but to far fewer vertebrate or plant populations. Examples of these, though, are shown in Table 9.3 and Figure 9.7.

We start with populations of the wood frog (*Rana sylvatica*) in three regions of the United States (Table 9.3). The larval period was the key phase determining abundance in all regions, largely as a result of year-to-year variations in rainfall. In low-rainfall years, the ponds often dry out, reducing larval survival to catastrophic levels. Such mortality, however, was inconsistently related to the size of the larval population (only one of two ponds in Maryland, and only approaching significance in Virginia) and hence it played an inconsistent part in regulating the sizes of the populations. Rather, in two regions it was during the adult phase that mortality was clearly density-dependent (apparently as a result of competition for food) and, indeed, in two regions mortality was also most intense in the adult phase (first data column).

The key phase determining abundance in a Polish population of the sand-dune annual plant *Androsace septentrionalis* (Figure 9.7) were the seeds in the soil. Once again, however, mortality there did not operate in a density-dependent manner, whereas mortality of seedlings (not the key phase) was density-dependent.

Overall, therefore, key factor analysis (its rather misleading name apart) is useful in identifying important phases in the life cycles of study organisms, and useful too in distinguishing the variety of ways in which phases may be important:

Table 9.3

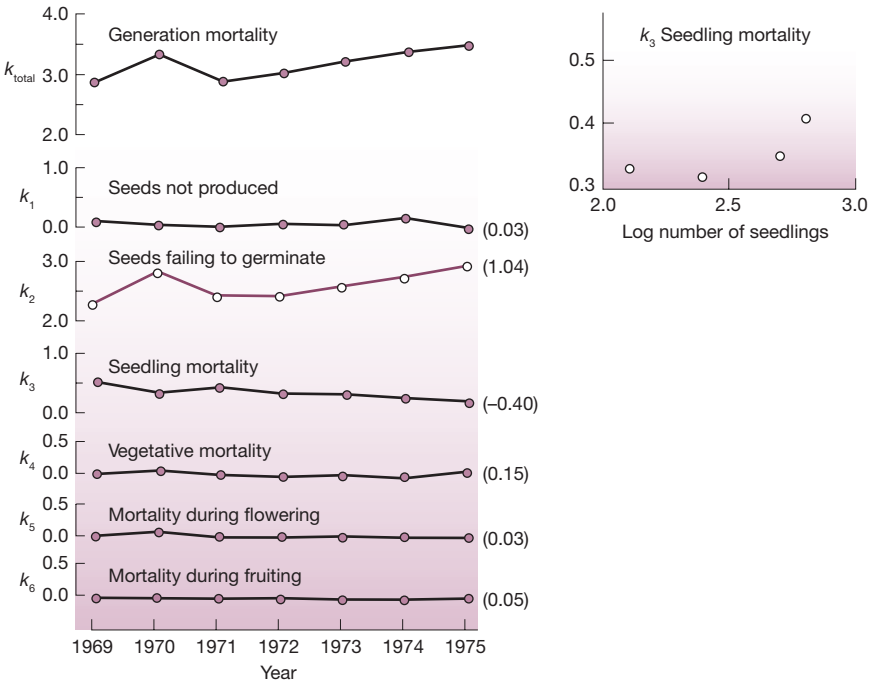
Key factor (or key phase) analysis for wood frog populations in the United States: Maryland (two ponds, 1977–1982), Virginia (seven ponds, 1976–1982) and Michigan (one pond, 1980–1993). In each area, the phase with the highest mean *k*-value, the key phase and any phase showing density dependence are highlighted in bold.

AGE INTERVAL	MEAN *k*-VALUE	COEFFICIENT OF REGRESSION ON k_{TOTAL}	COEFFICIENT OF REGRESSION ON LOG (POPULATION SIZE)
Maryland			
Larval period	1.94	**0.85**	**Pond 1 : 1.03 ($P = 0.04$)** Pond 2 : 0.39 ($P = 0.50$)
Juvenile: up to 1 year	0.49	0.05	0.12 ($P = 0.50$)
Adult: 1–3 years	**2.35**	0.10	0.11 ($P = 0.46$)
Total	4.78		
Virginia			
Larval period	**2.35**	**0.73**	0.58 ($P = 0.09$)
Juvenile: up to 1 year	1.10	0.05	–0.20 ($P = 0.46$)
Adult: 1–3 years	1.14	0.22	**0.26 ($P = 0.05$)**
Total	4.59		
Michigan			
Larval period	1.12	**1.40**	1.18 ($P = 0.33$)
Juvenile: up to 1 year	0.64	1.02	0.01 ($P = 0.96$)
Adult: 1–3 years	**3.45**	–1.42	**0.18 ($P = 0.005$)**
Total	5.21		

AFTER BERVEN, 1995

Figure 9.7

Key factor analysis of the sand-dune annual plant *Androsace septentrionalis*. A graph of total generation mortality (k_{total}) and of various *k*-factors is presented. The values of the regression coefficients of each individual *k*-value on k_{total} are given in brackets. The largest regression coefficient signifies the key phase and is shown as a maroon line. Alongside is shown the one *k*-value that varies in a density-dependent manner.



AFTER SYMONIDES, 1979; ANALYSIS IN SILVERTOWN, 1982

in contributing significantly to the overall sum of mortality; in contributing significantly to variations in mortality, and hence in *determining* abundance; and in contributing significantly to the *regulation* of abundance by virtue of the density dependence of the mortality. Box 9.2 presents an account of a topical problem, an understanding of which could benefit from key factor analysis.

9.2 Topical ECOncerns

9.2 TOPICAL ECONCERNS

Acorns, mice, ticks, deer and human disease: complex population interactions

Ecologists have been trying to uncover the complex interactions among acorn production, populations of mice and deer, parasitic ticks and, ultimately, a bacterial pathogen carried by the ticks that can affect people. It is clear that a thorough understanding of the abiotic factors that determine the size of the acorn crop and of the various population interactions can enable scientists to predict years when the risk of human disease is high. This is the topic of the following newspaper article in the *Contra Costa Times* on Friday, February 13, 1998, by Paul Recer.

More acorns may mean a rise in Lyme disease

A big acorn crop last fall could mean a major outbreak of Lyme disease next year, according to a study that linked acorns, mice and deer to the number of ticks that carry the Lyme disease parasite.

Female deer tick (*Ixodes dammini*), which carries Lyme disease (7).

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Based on the study, researchers at the Institute of Ecosystem Studies in Millbrook, New York, say that 1999 may see a dramatic upswing in the number of Lyme disease cases among people who visit the oak forests of the Northeast.

'We had a bumper crop of acorns this year, so in 1999, two years after the event, we should also have a bumper year for Lyme disease', said Clive G. Jones, a researcher at the Institute of Ecosystem Studies; '1999 should be a year of high risk for Lyme disease'.

Lyme disease is caused by a bacterium carried by ticks. The ticks normally live on mice and deer, but they can bite humans. Lyme disease first causes a mild rash, but left untreated can damage the heart and nervous system and cause a type of arthritis.

Jones, along with researchers at the University of Connecticut, Storrs, and Oregon State University, Corvallis, found that the number of mice, the number of ticks, the deer population and even the number of gypsy moths are linked directly to the production of acorns in the oak forest.

Jones said that in years following a big acorn crop, the number of tick larvae is eight times greater than in years following a poor acorn crop.

Additionally, he said, there are about 40 percent more ticks on each mouse.

The researchers tested the effect of acorns by manipulating the population of mice and the availability of acorns in forest plots along the Hudson River. Jones said the work, extended over several seasons, proved the theory that mice and tick populations rise and fall based on the availability of acorns.

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How could a key factor analysis be used to pinpoint the phases of importance in determining risk of human disease?

9.3 Dispersal, patches and metapopulation dynamics

dispersal is ignored at the ecologist's peril

In many studies of abundance, the assumption has been made that the major events all occur within the study area, and that immigrants and emigrants can safely be ignored. But migration can be a vital factor in determining and/or regulating abundance. We have already seen, for example, that emigration was the predominant reason for the loss of summer adults of the Colorado potato beetle, which was both the key phase in determining population fluctuations and one in which loss was strongly density-dependent.

habitable sites and dispersal distance

Dispersal has a particularly important role to play when populations are fragmented and patchy – as many are. The abundance of patchily distributed organisms can be thought of as being determined by the properties of two features: the 'habitable site' and the 'dispersal distance' (Gadgil, 1971). Thus, a population may be small if its habitable sites are themselves small or short-lived or only few in number; but it may also be small if the dispersal distance between habitable sites is great relative to the dispersibility of the species, such that habitable sites that go extinct locally are unlikely to be recolonized.

To discover the limitations that the accessibility of habitable sites places on abundance, though, it is necessary to identify habitable sites that are not inhabited. This is possible, for example, for a number of butterfly species, because their larvae feed only on one or a few species of patchily distributed plants. Thus, by identifying habitable sites with these plants, whether or not they were inhabited, Thomas et al. (1992) found that the silver-studded blue butterfly *Plebejus argus* was able to colonize virtually all habitable sites less than 1 km from existing populations, but those further away (beyond the dispersal powers of the butterfly) remained uninhabited. The overall size of the population was determined as much by the accessibility of this patchy resource as by the total amount of the resource. Indeed, the habitability of some of these isolated sites was established when the butterfly was successfully introduced there (Thomas & Harrison, 1992). This, after all, is the crucial test of whether an uninhabited 'habitable' site is really habitable or not.

metapopulations

A radical change in the way ecologists think about populations has involved combining patchiness and dispersal in the concept of a *metapopulation*, the origins of which are described in Box 9.3. A population can be described as a