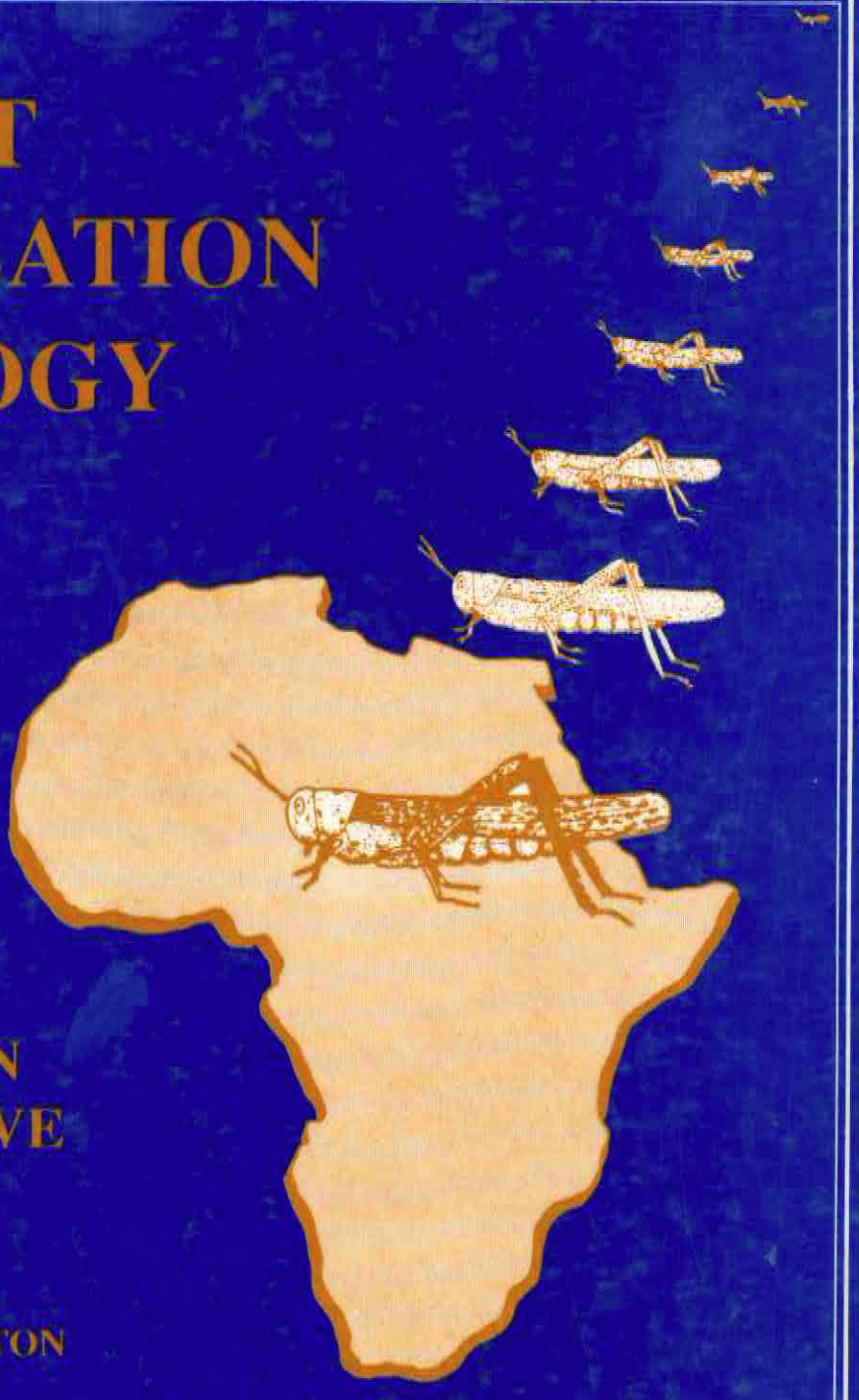
INSECT POPULATION ECOLOGY



AN AFRICAN PERSPECTIVE

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PREFACE

The purpose of this text is to introduce the fundamentals of insect population ecology with a focus on African insects and related arthropods such as mites. The impetus to prepare such a text arose from the graduate course in insect population ecology offered by the African Regional Postgraduate Program in Insect Science (ARPPIS) conducted at the International Center of Insect Physiology and Ecology (ICIPE) in Nairobi, Kenya. The goal of the text is to introduce the students to the basic concepts of insect population ecology. The emphasis is on quantitative methods that students are likely to apply in their field research. The text assumes that students have had a basic training in statistical inference. At the end of each chapter is a set of exercises designed to give the students practice with the material on the chapter. I believe that such practice is critical in helping students master the material and overcome any fears they might have of complex numerical calculations.

I wish to thank the many individuals who have assisted with this project, especially Jeff and Cindy Boettner who made most of the figures and helped with the editing and all phases of the final preparation. I am grateful to Karin Fischer, Gloria Witkus, Rolf Fishburn-Parker Amy Musante, Allan Wright and Don Wakoluk, who helped with gathering references; Sam Englestadt, Zbigniew Dabrowski, Brian Williams, John Edman and Dave Leonard who assisted in logistical support and helped establish the many contacts required to prepare this text. Paula Martin and the ARPPIS students of the 1991 and 1992 classes provided invaluable feedback on the content of the material and exercises in this book. I would also like to thank K. C. Conlan, John Buonaccorsi, Robert Cheke, Roy Van Driesche, Paula Martin, Craig Hollingsworth, Dave Leonard, Sandy Liebhold, Mike Tucker, David Rogers, F. Schulthess and Jeff Waage for reviewing earlier drafts of various chapters. Finally, I am most grateful to the Pew Charitable Trust Foundation and the World Wildlife Fund for providing financial support for the preparation of this text.

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INTRODUCTION TO SAMPLING THEORY

Introduction: General considerations

The science of population ecology is focused on documenting and explaining changes in the abundance of animals. Studies of population dynamics usually begin with the development of sampling techniques which are required to determine the abundance of insects in a habitat. For each important insect species, there has usually been a considerable investment of scientific talent in the development of sampling schemes. Sampling methods vary tremendously with habitat type. Specialized sampling techniques have been developed for insects that live in the soil, in the air, on foliage, and on vertebrate hosts. A large scientific literature exists on the methods developed for each of these habitats (see Southwood (1978) for a general review). In this chapter we focus on some of the general principles that lie behind sampling methodology rather than focus on particular sample methods which are unique to each habitat.

There are several general distinctions one can make about different methods of sampling insects. One of these is to distinguish between **absolute** versus **relative measures of density.** Absolute measures quantify numbers per unit area or volume of habitat. Examples are numbers of aphids per m^2 in quadrat samples or numbers of Collembola per cm^3 of soil. Relative measures express numbers per sample unit. Examples are numbers of leafhoppers captured in a sweep net sample or numbers of male moths captured per pheromone trap. These measures may or may not reflect absolute density. They are frequently much easier to obtain than absolute measures. In many systems, absolute measures may not be feasible to obtain at all, but relative measures may be influenced by many factors (air temperature, for instance) that affect the activity of the insect and its likelihood of being captured. Consequently, much research is needed to be sure that relative measures give an accurate indication of differences in absolute population density. In addition, there are **population indices,** which are indirect measures of density, such as measures of defoliation or frass production.

Another general characteristic of sample methods is that some, such as sticky traps or pheromone traps, obtain data continuously over an interval of time. Others obtain an essentially instantaneous "snapshot" of the population at a particular moment. Examples of the latter include sweep net samples, quadrat samples, and insecticide knockdown samples. Such **instantaneous samples** are susceptible to the effects of time of day or weather conditions at the time of sampling that may influence the number captured.

Continuous samples may also be influenced by weather conditions, but, because they capture over an extended interval, these effects may be averaged over a range of such conditions.

Any investigator planning a sampling program must give careful thought as to how the samples will be selected. Consultations with statisticians with expertise in experimental design prior to collection of data is always wise, as is preliminary sampling or pilot testing to estimate the expected amount of sample variability. Most sampling schemes may be classified as **random, systematic** or **stratified random designs.** In random sampling, the sample units are chosen or placed at randomly selected locations within the sample universe. Random selection is typically done by choosing coordinate points from a table or list of random numbers. In a systematic design, sample units are placed at regular intervals across the sample universe (for example every 20 m or every 10th plant). Systematic samples are frequently much easier to conduct and they assure that the samples are distributed evenly across the sample universe. However, systematic samples violate the assumption that samples be selected independently and at random from the sample universe, as required by most statistical analyses. Whether or not violation of this assumption leads to erroneous conclusions in any particular system is usually debatable. A reasonable compromise between these two approaches is a stratified random or randomized block design. This design involves dividing the sample universe into regularly spaced subunits or blocks. Samples are then selected at random within each block. Differences in density between subunits caused by edge effects or density gradients across the field can be detected with analysis of variance by incorporating block effects into the analysis.

A fundamental principle in scientific research is that studies must be repeated or replicated. This is done to assure that the findings of the study cannot be explained by chance alone. In other words, whatever difference exists between treatment and control groups in the mean value for the variables measured is not caused by the natural variability associated with all measurements.

An important manuscript by Hurlbert (1984) drew attention to the frequent occurrence of **pseudoreplication** in the ecological literature. The following is a hypothetical example of pseudoreplication. A study is conducted on the effects of a cultural control on the density of mealybugs in a cassava crop. Two cassava fields are selected, one of which received the cultural treatment and one of which remained untreated as a control. A large number of quadrat samples are collected from each field and statistical analyses conducted to prove that densities of mealybugs were significantly lower in the field receiving the treatment. The investigator might be tempted to claim that he had proven the effectiveness of the treatment in reducing the density of the mealybugs. In fact, all that was proven was that densities between the two fields were different. The difference might or might not be caused by the treatment. No amount of repeated sampling within the two fields will prove the effect. Only by replicating the treatment between fields, or by replicating the treated and untreated blocks within a field, can the effect of treatment be convincingly demonstrated.

Definition of statistical terms

It is crucial that a field ecologist have adequate training in the principles of statistical analysis. Here we review some basic statistical concepts that are vital to understanding the material in subsequent chapters of this book. The most basic concept of all is the **arithmetic mean:**

$$\bar{x} = \frac{\sum x_i}{n} \tag{1.1}$$

where x_i is the number counted in the i^{th} sample, $1 \leq i \leq n$. The mean is the most common measure of central tendency or typical value for any measurable entity, but it is not necessarily the best. An alternative is the **median,** the value of x_i such that 50% of the values are larger and 50% are smaller. The median is often employed when the distribution of x_i values is highly skewed.

Another fundamental concept is the variance of the population (σ^2) which is estimated from the sample **variance** (s^2) and is a measure of the spread of the data:

$$s^2 = \frac{\sum(x_i - \bar{x})^2}{n-1} = \frac{\sum(x_i^2) - \frac{(\sum x_i)^2}{n}}{n-1} \qquad (1.2)$$

The square root of the variance (s) is known as the **standard deviation** ($s = \sqrt{s^2}$).

The coefficient of variation: ($s/\bar{x}$) is frequently used to compare variability between populations with different means. As we will see in Chapter 2, the variance of density counts typically increases with mean density. Comparison of the coefficient of variation of two or more populations implies that the mean and standard deviation are linearly related. This may not be true; indeed, for estimates of density it is usually not true, (see discussion of Taylor's Power Law in Chapter 2).

It is important to distinguish between the standard deviation and the **standard error of the mean** (usually called standard error for short and denoted SE or $S_{\bar{x}} = \sqrt{(s^2/n)} = s/\sqrt{n}$. The standard error is an estimate of the standard deviation of repeated estimates of the mean. It is a measure of how well the estimated mean predicts the true mean (μ) of the population. It is *not* a measure of variance of population. Increasing the number of samples (n) will reduce the standard error but will not systematically affect the standard deviation, the variance, or the coefficient of variation (Fig. 1.1).

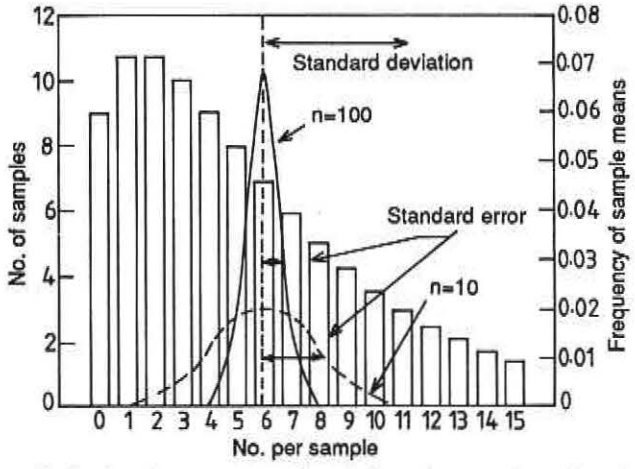


Fig. 1.1 Frequency distribution of counts per sample and of sample means from a hypothetical population.

Relative variation (***RV***) is an expression of the standard error as a percentage of the mean.

$$RV = \frac{SE}{\bar{x}} * 100 \qquad (1.3)$$

This has also been called the coefficient of variability, resulting in some confusion in the literature between this term and the coefficient of variation (Ruesink 1980).

The **Central Limit Theorem** (CLT) is of fundamental importance to most statistical procedures employed for ecological data. The theorem states that if the number of samples (n) is large, then repeated estimates of the mean will be normally distributed regardless of the underlying distribution of the population from which they are drawn, no matter how skewed (Fig. 1.1). As we will see in Chapter 2, the distribution of density counts for most populations is highly skewed (Fig. 1.1). Most parametric statistical procedures, including analysis of variance and linear regression, assume that the sample means will be normally distributed. The CLT indicates that this assumption is valid, provided n is large (typically $n > 30$). The standard error is the standard deviation of the distribution of sample means.

The standard error is related to the concept of a **confidence interval (CI)**: $\bar{x} \pm K\ (SE)$, where K is a constant. If the population variance σ^2 is known, then $K = Z_{\alpha/2}$, Z is the standard normal deviate and α is the probability that the estimated mean lies in the region of the distribution less than Z. For a 95% CI, $Z_{\alpha/2} = 1.96$. In other words, the 95% CI spans the region within 1.96 standard errors of the mean as indicated by the shaded region (dark and light) in Fig. 1.2A. The meaning of a 95% CI is that 95% of all such intervals will contain the true mean (μ).

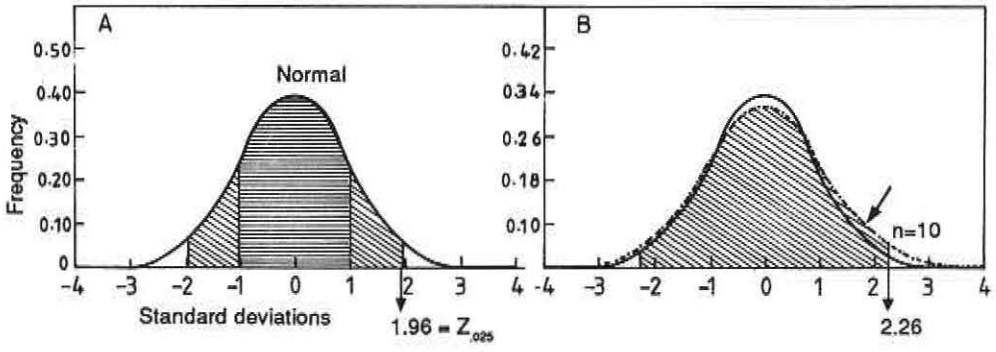


Fig. 1.2 Confidence intervals based on (A) the standard normal curve ($\mu = 0$, $\sigma = 1$) and, (B) the t distribution for a sample size of 10 compared to the limiting case ($n = \infty$) which is the same as the standard normal curve.

Usually the variance of the population is unknown, in which case confidence intervals for large n should be based on the "t" distribution (Fig. 1.2B). The resulting values of K are somewhat larger than Z and depend on the number of samples taken (degrees of freedom, usually $n - 1$). For a 95% CI, for example, if $n = 10$, $k = 2.26$; if $n = 30$, $k = 2.042$, and if $n = \infty$, $k = Z_{\alpha/2} = 1.96$. Throughout the scientific literature it is customary to present data as $\bar{x} \pm SE$. Provided n is large, this approximates a 68% CI by virtue of the CLT, indicated by the darker shading in Fig. 1.2. In other words 68% of all such intervals will contain the true mean. If n is small, the confidence intervals around the mean are asymmetrical.

Determination of sample size

Determination of the number of samples required for a particular study is an important question faced by every field ecologist. Preliminary data can be analyzed to provide guidance on this point. The answer is framed in terms of sample **precision** or **reliability.** Sample precision is defined in terms of the expected difference between the estimated mean ($\bar{x}$) and the true mean (μ). It is measured either by means of a standard error or a confidence interval. In terms of a standard error, we could specify a sufficient number of samples, so that the standard error should be less than a certain fixed percentage of, or within a certain fixed distance from the true mean (μ). Alternatively, we could specify a half-width of a confidence interval that spanned the region within a certain percentage or within a fixed distance of the true mean at a specified level of confidence (95%, for instance). Specifying precision in terms of a standard error is really a special case of specifying it in terms of a confidence interval (i.e. a 68% CI, provided n is large).

Suppose that we define precision in terms of a standard error as a fixed proportion of the mean. In other words we specify a constant D such that:

$$SE = D\bar{x} \tag{1.4}$$

From a normal distribution, or from any distribution provided n is large enough, the number of samples required to achieve a standard error within D of the mean derives directly from the definition of the standard error:

$$SE = \frac{\sqrt{s^2}}{\sqrt{n}}, \qquad (D\,\bar{x})^2 = \frac{s^2}{n}, \qquad n = \frac{s^2}{D^2\,\bar{x}^2} \tag{1.5}$$

Similar mathematical formulae may be derived for samples taken from other distributions discussed in Chapter 2, such as the Poisson and the negative binomial (see Karadinos 1976, Ruesink 1980). If precision is defined in terms of the half-width of a confidence interval as a fixed proportion (d) of the mean, then:

$$n = \frac{\left(Z_{\alpha/2}\right)^2}{d^2} * \frac{s^2}{\bar{x}^2} \tag{1.6}$$

If precision were defined in terms of a confidence interval spanning a fixed distance (h) from the mean, then:

$$n = \frac{\left(Z_{\alpha/2}\right)^2}{h^2} * s^2 \tag{1.7}$$

If the precision levels of two or more sample methods are being compared, their relative costs must be considered. This is accomplished by defining the relative net precision (*RNP*):

$$RNP = \frac{\bar{x}}{SE * C_s} = \frac{100}{RV * C_s} \tag{1.8}$$

where C_s is cost and is computed in wages or time spent per sample for the n samples required to estimate *RV*. Of course, the cost per sample may vary with the number of samples taken and must include travel time between samples, so the computation may be more complex than that implied by the simple equation above.

The following (Table 1.1) illustrates the calculation of *RV* and *RNP* for several methods of sampling the bean flower thrips, *Megalurothrips sjostedti* (Trybom) on cowpeas in Nigeria (Salifu and Singh 1987).

Table 1.1 Cost (C_s) variation (*RV*), and relative net precision (*RNP*) of different methods of sampling *Megalurothrips sjostedti* on cowpeas (Salifu and Singh 1987, reproduced with permission from the International Institute of Entomology, UK)

Sampling method	C_s	*RV*	*RNP*
Absolute			
Cut-and-bag	0.20	8.57	58.34
Relative			
Alcohol	0.11	25.44	35.70
Shaking plants	0.03	9.07	367.51
Sticky traps	0.02	26.2	190.62
Sweep net	0.07	28.44	50.23
Water traps	0.02	10.75	465.12

It is clear that water traps are the best of the relative sampling methods in terms of the precision achieved per unit effort (*RNP*).

Frequently, sampling is conducted at two or more hierarchical levels. The following example comes from a sampling scheme developed for the cassava mealybug, *Phenacoccus manihoti* (Mat.-Ferr.) in Nigeria by Schulthess et al. (1989). Previous work had established that the mealybug density could be estimated by making counts on plant tips. The problem was to determine the optimal number of tips per plant to sample.

The within-plant (S_2^2) and the between-plant variances (S_1^2) were calculated after Cochran (1956):

$$S_1^2 = \frac{\sum_{i=1}^{n}\left(\overline{y_i} - \overline{\overline{y}}\right)^2}{n-1} \tag{1.9}$$

$$S_2^2 = \frac{\sum_{i=1}^{n}\sum_{j=1}^{m}\left(y_{ij} - \overline{y}_i\right)^2}{n(m-1)} \tag{1.10}$$

n = number of plants taken per block,
m = number of tips taken per plant,
$\overline{\overline{y}}$ = mean density per plant,
$\overline{y}_i$ = mean density per tip for plant i,
y_{ij} = density for tip j for plant i

The number of tips (L) to be taken per plant for minimizing costs was calculated after Southwood (1978)

$$L = \sqrt{\frac{S_2^2}{S_1^2} * \frac{C_p}{C_s}} \tag{1.11}$$

C_s = time to sample within the same plant (5 sec),
C_p = time to move from one plant to another (5 sec) plus C_s
S_1^2 = between-plant variance for each field,
S_2^2 = within-plant variance for each field
The S_1^2, S_2^2 and L values are presented in Table 1.2

Table 1.2 Selecting the optimum number of sampling units (L) to be taken per plant when estimating *P. manihoti* densities on cassava in Nigeria (Schulthess et al. 1989)

Field	Variance	Block 1	Block 2	Block 3	Block 4
1	S_1^2	1.29	4.95	4.71	4.44
	S_2^2	0.54	1.62	1.99	1.96
	L	1.2	0.8	0.9	0.9
2	S_1^2	26.1	2123.4	392.7	628.8
	S_2^2	27.3	680.7	177.9	189.8
	L	1.4	0.8	1.0	0.8

These results indicate that counting 1 tip/plant is close to optimum. The number of plants per field or block is then calculated based on whatever level of precision is required (eq. 1.6, 1.7) or can be achieved given the constraints of what can be afforded.

EXERCISES

One hundred whole-plant samples were collected from a field of cowpeas and the number of flower thrips per plant were counted as follows:

0	9	8	15	0	1	7	0	1	0	0	21	0	1	0	2	3	0	11	14
2	0	1	7	0	2	0	0	1	14	12	0	0	8	0	9	0	9	0	0
0	3	7	6	3	2	0	1	3	10	13	0	0	2	1	18	0	1	23	0
0	9	8	15	0	1	7	0	1	0	0	21	0	1	0	2	3	0	2	0
2	0	1	7	0	2	0	0	1	14	12	0	0	8	0	9	0	9	3	27

1. Calculate the following for your sample:

   The mean, variance, standard deviation, standard error, coefficient of variation, the relative variation and a 95% confidence interval about the mean.

2. Calculate the number of samples that would be required to estimate the mean density within 10% of the "true mean" with a 95% probability and/or a standard error that was 10% of the mean.

2

PATTERNS OF DISPERSION

The spatial distribution of animals is fundamental to sampling theory and to many topics in population ecology. In this chapter we explore the various ways in which spatial dispersion can be measured. If a population of animals were placed in a habitat so that the location of each individual was entirely independent of the other individuals, we would say that the population was **randomly distributed** in space. Imagine a population of aphid stem mothers that was deposited by the wind in a cabbage field. Unless there were edge effects or some other anomaly, one might expect the landing to occur at random, meaning that the landing site of each individual was independent of that of all other individuals. The spatial dispersion of these individuals might look like that of (Fig 2.1A). The populations of most species, however, exhibit a **clumped** or **aggregated pattern** of **dispersion** (Fig. 2.1B). This arises for several reasons. For one, the resources upon which species depend are clumped. Most plants have aggregated distributions because the soil and water conditions for a plant are extremely patchy. The patchiness of plant species gives rise to clumped distributions of the herbivores that specialize upon them and thus to the higher trophic levels that feed upon the herbivores. Secondly, patchy distributions arise from reproduction associated with limited dispersal. The aphids that were deposited at random in the example given above will each soon give rise to non-dispersing young that will form a densely packed colony. The resulting distribution of aphids will then be highly clumped.

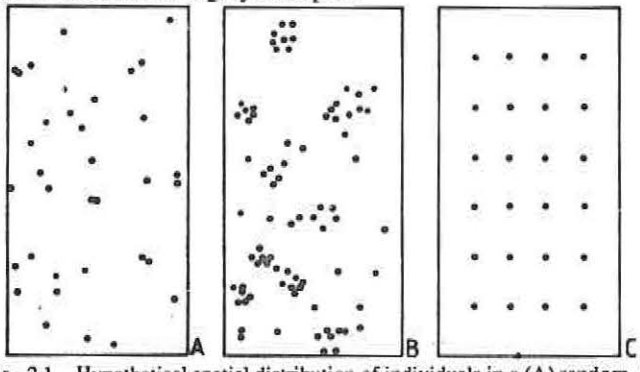


Fig. 2.1 Hypothetical spatial distribution of individuals in a (A) random, (B) aggregated, and (C) uniform pattern.

Uniform patterns of dispersion (Fig. 2.1C) are also quite common in nature, although not as common as aggregated patterns. Uniform patterns typically arise when organisms compete with conspecifics for space. Desert plants are often uniformly distributed, because each plant extracts water from a particular soil depth and prevents the growth of conspecifics within a certain distance. Similarly, ant or termite colonies may be uniformly spaced because each colony controls a territory surrounding the colony from which it extracts food and within which it prevents the formation of other colonies of conspecifics.

More complex patterns of dispersion, such as random clumps or uniform clumps, occur frequently. Indeed, the pattern of dispersion depends upon the spatial scale at which it is measured. The distribution of individual termites is obviously highly clumped, given that termites are social insects that live in colonies, but the colonies are likely to be uniformly distributed. However, on a larger spatial scale that includes areas where soil or vegetation is inappropriate for termites, the distribution of colonies is likely to be clumped.

Dispersion is important in population ecology for several reasons. From a sampling perspective, dispersion influences sample variance and is thus important to the development of sampling schemes and to statistical tests involving density estimates. The greater the degree of aggregation, the higher the variance and the greater the number of samples that must be taken to achieve a required level of precision in estimates of the population mean. Furthermore, the variance of a series of density counts is likely to increase with density. This creates problems in statistical inference involving comparisons between populations with very different means. Standard procedures such as ANOVA or *t*-tests require that variances be equal. Similarly, least squares regression analyses in which population density is the dependent variable plotted against some other variable, assume that the variance does not change with density. Solutions to these problems include transformations of sample data, weighting with 1/variance or use of non-parametric procedures.

Review of frequency distributions

Data on insect counts can be represented as frequency distributions which portray the number of samples containing different numbers of individuals (Fig. 2.2). If both the numbers of insects in each sample and the number of samples taken are large, the resulting frequency distribution may approximate the familiar bell-shaped curve of the normal or Gaussian distribution (Fig. 2.2A).

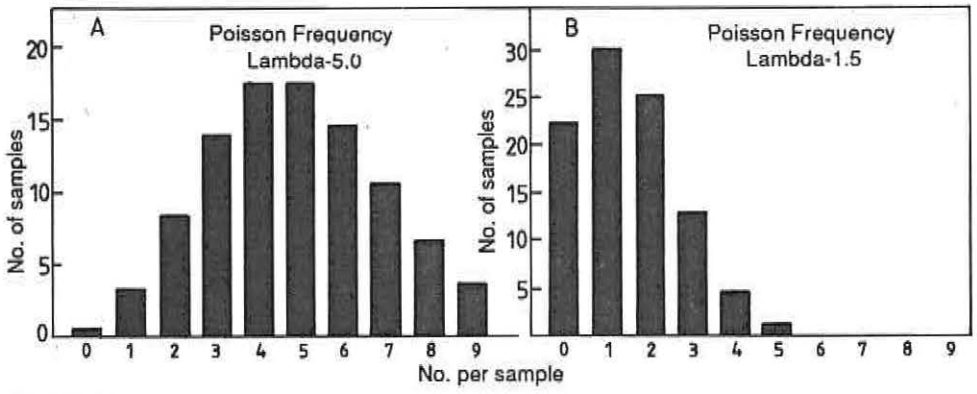


Fig. 2.2 Frequency distribution of sample counts from a population with a random pattern of dispersion, and (A) high, or (B) low density.

More typically, the frequency distribution is strongly skewed as in Fig. 2.2B. If the insects are randomly distributed, the frequency distribution will be well described by a Poisson distribution. In a Poisson distribution, the probability that a sample contains x individuals (denoted $P(X=x)$) is:

$$P(X=x)=\frac{\left(e^{-\lambda}\right)\left(\lambda^{x}\right)}{x!} \tag{2.1}$$

For example, the probability of a sample containing four is:

$$P(X=4)=\frac{\left(e^{-\lambda}\right)\left(\lambda^{4}\right)}{4\cdot 3\cdot 2\cdot 1}$$

The probability that a sample contains zero individuals simplifies to:

$$P(X=0)=e^{-\lambda} \tag{2.2}$$

An important property of the Poisson distribution is that the variance is equal to the mean (λ).

If samples are taken from a population with a clumped distribution, there will be more samples with higher counts and more samples with zero or low counts, than that predicted by the Poisson distribution. A variety of mathematical distributions have been used to represent such data, but the most common is the negative binomial distribution:

$$P(X=x)=\left(1+\frac{\mu}{k}\right)^{-k} * \frac{(k+x-1)!}{x!(k-1)!} * \left(\frac{\mu}{\mu+k}\right)^{x} \tag{2.3}$$

The distribution is defined by the mean μ and the parameter k, which is an indicator of the degree of clumping in the data. The variance is given by:

$$\sigma^2=\mu+\frac{\mu^2}{k} \qquad \text{which rearranges to:} \qquad k=\frac{\mu^2}{\sigma^2-\mu} \tag{2.4}$$

Small values of k indicate a highly clumped distribution; as k approaches ∞, the negative binomial approaches a Poisson distribution. Since k is a positive number, the variance is always greater than the mean. The negative binomial distribution with two different values of k is illustrated in Fig. 2.3.

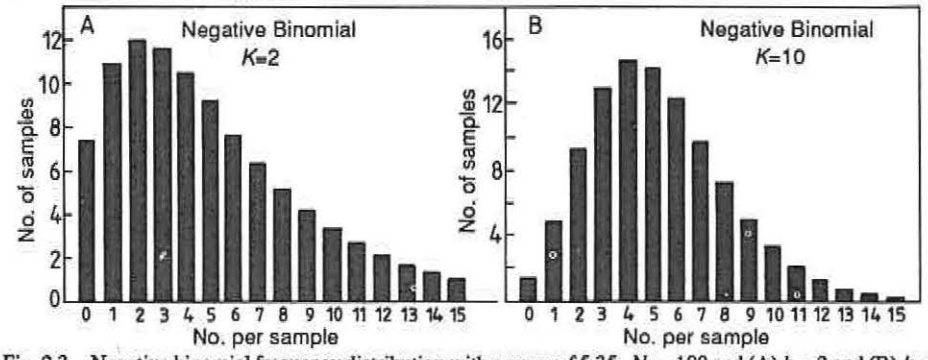


Fig. 2.3 Negative binomial frequency distribution with a mean of 5.35, N = 100 and (A) k = 2 and (B) k = 10.

Equation (2.3) is only defined for integer values of k. However, the individual terms, $P(X=x)$, of the negative binomial can be calculated for all values of k including non-integers as follows:

$$P(X=0)=\left(1+\frac{\bar{x}}{k}\right)^{-k} \tag{2.5}$$

$$P(X=1)=\left(\frac{k}{1}\right)\left(\frac{\bar{x}}{\bar{x}+k}\right) * P(X=0) \tag{2.6}$$

$$P\,(X{=}2) = \left(\frac{k+1}{2}\right)\left(\frac{\bar{x}}{\bar{x}+k}\right) * P\,(X{=}1) \tag{2.7}$$

$$P\,(X{=}n) = \left(\frac{k+n-1}{n}\right)\left(\frac{\bar{x}}{\bar{x}+k}\right) * P\,(X{=}n{-}1) \tag{2.8}$$

When one collects data on population density or counts per sample unit from a field population, it is frequently important to determine whether the pattern of dispersion is clumped, random or uniform. The first step in this process is to compare the mean and variance of the sample data. When the mean and variance are equal, the pattern is random. When the variance is greater than the mean, the pattern is clumped, and when the variance is less than the mean, the pattern is uniform. Of course, the mean is almost never exactly equal to the variance, so the appropriate question is: does the mean differ significantly from the variance? The appropriate statistical test is to determine if the distribution of counts differs significantly from Poisson distribution. Provided that the sample size is sufficiently large, and the mean density is not too small this can be accomplished by means of a chi-square goodness-of-fit test.

An example is given in Table 2.1. Once the sample mean has been determined, the expected number of samples with 0, 1, 2,..., n individuals, assuming a Poisson distribution can be calculated: expected = $N*P\,(X=x)$, where N is the total number of samples and $P\,(X{=}x)$ is the corresponding probability of a sample containing x individuals given above. To avoid biases arising from small sample departures from continuity, one should lump all frequency categories with an expected value of less than one (Elliot 1977). Some authors recommend lumping all categories with an expected value less than 5.0, but unless the mean number of individuals per sample unit is considerably greater than 5.0, the result would constitute a considerable loss of statistical power. In other words, it would be extremely difficult to demonstrate departure from a random distribution. Thus, lumping of frequency categories so that no expected value is less than 1.0 represents a reasonable compromise (Cochran 1954). In our example, we have lumped all samples with four or more individuals per sample into a single category, such that the number in the expected column is 1.55 (> 1.0). In order to calculate the corresponding Poisson probability for this category, we use the fact that the sum of all probabilities ($x = 0, 1, 2,\ldots$) is 1.0. Thus, the probability that $P\,(X \geq 4) = 1.0 - P(0) - P(1) - P(2) - P(3)$.

Table 2.1. Chi-square goodness-of-fit test to a Poisson distribution

No. per sample	No. of samples	Expected $N*P\,(X=x)$[1]	$\frac{(\text{Observed} - \text{Expected})^2}{\text{Expected}}$
0	50	39.06	3.06
1	23	36.72	5.13
2	15	17.26	0.30
3	7	5.41	0.47
4+	5	1.55	7.65
	N= 100	100	$\chi^2 = 16.61$

[1] $P(X=x) = \frac{(e^{-\lambda})\,(\lambda^x)}{x!}$

In this example, the 100 samples collected contained a total of 94 individuals; k is thus 0.94. The sample variance (1.39) is greater than the mean, suggesting that the population may be clumped. The chi-square statistic is calculated in the usual way:

$$\chi^2 = \sum \frac{(\text{Observed} - \text{Expected})^2}{\text{Expected}}$$

With n–2 degrees of freedom, where n is the number of frequency categories. In our example, the number of frequency categories is 5, so one refers to a table for $\chi^2_{n-2} = \chi^2_3 = 7.815$ at $P = 0.05$. Values of the chi-square larger than the cut-off value indicate that the frequency distribution departs significantly from a Poisson distribution. In our example (Table 2.1), the calculated chi-square = 16.61, which is considerably larger than 7.815; hence, we reject the hypothesis that the distribution fits the Poisson.

When the number of samples is large ($N > 30$), the chi-square approximation to the normal distribution can be used to calculate the cut-off value for departure from the Poisson, thereby avoiding the need to refer to a chi-square table:

$$d = \sqrt{2\chi^2} - \sqrt{2v - 1} \tag{2.9}$$

where v = the number of degrees of freedom (3 in our example). If $d > 1.96$, the standard cut-off point at $\alpha = 0.05$ for the standard normal distribution, then sample frequency distribution does not fit the Poisson. In our example, $d = 3.53$, which is much greater than 1.96.

When the number of samples is small ($N \leq 30$) or when the mean density is low (<0.5 per sample), the chi-square goodness-of-fit test lacks statistical power. An alternate calculation based on the following approximation to the chi-square is recommended instead:

$$\left(\frac{s^2}{\bar{x}}\right)(N-1) = \chi^2_{(N-1)} \tag{2.10}$$

where the degrees of freedom (N–1) *are based on the number of samples* taken (N), instead of the number of frequency categories as above. In our example, $(s^2/\bar{x})(N-1) = (1.39/.94)(99) = 146.42$. Since $x^2_{[0.05](99)} = 123.23$, and since $146.42 > 123.23$, we reject the hypothesis that the data fit the Poisson distribution.

Similar calculations can be used to test whether samples from a population fit a negative binomial distribution. To do this, one must first estimate k. This is accomplished by following procedures discussed below (eq. 2.13). One then employs equation (2.8) to calculate the expected frequencies based on a negative binomial distribution. We illustrate this with an example taken from Ramsamy (1981) on the distribution of pupae of the stable fly, *Stomoxys nigra* Macquart, in Mauritius (Table 2.2).

Table 2.2. Comparison of frequency counts of stable flies with the negative binomial distribution (Ramsamy 1981)

No. of pupae per m^2 unit	Observed frequency (f)	Expected frequency (f)	χ^2
0	3032	3033.81	0.00
1	423	386.61	3.43
2	176	175.76	0.00
3	88	99.07	1.24
4	41	61.25	6.69
5	33	39.87	1.18
6	22	26.82	0.87
7	18	18.46	0.01
8	17	12.92	1.29
9	6	9.16	1.09
10	6	6.56	0.05
11	6	4.74	0.96
12	5 (11)	3.45 (8.19)	
13	2	2.52	
14	2	1.85	
15	2 (15)	1.37 (9.53)	3.14
16	9	3.79	
	3888 = N	3888.0	19.95

$\Sigma(fx) = 2271$ $\quad \bar{x} = 0.584$ $\quad S^2 = 3.218$

This population was highly clumped ($s^2 >> \bar{x}$, but the calculated $\chi^2 = 19.95$ is greater than the 5% cut-off value of the chi-square test ($\chi^2_{[0.05](10)} = 18.307 < 19.95$, $P < 0.03$), indicating a poor fit to the negative binomial. The appropriate degrees of freedom are n–3 compared with n–2 of the Poisson test (Table 2.1), because an extra degree of freedom is lost in estimating k.

Indices of dispersion

Having determined that our population is significantly clumped, we may then be interested in quantifying how clumped it is. For example, we might wish to release a predator that aggregates to and consumes high density clumps of prey, and we might want to demonstrate a significant decline in the degree of clumping in the prey population. Several techniques for measuring the degree of clumping, known as indices of dispersion, have been proposed (see Taylor 1984, Kuno 1991 for a general review), but there is no general agreement as to which of these are the best measures. Indeed, it is not intuitively obvious how one determines the validity of a measure of dispersion. However, we could propose the following properties for a desirable index. An ideal dispersion index should be:

1. Defined for all possible patterns of dispersion, uniform to highly aggregated.
2. Unaffected by the number of samples taken.
3. Unaffected by the density of the population.

We might also desire that an index be unaffected by the size of the sample unit, but this is unrealistic, because we know that the pattern of dispersion depends on the spatial scale on which it is measured. While various indices meet points 1 and 2, it is common for changes in density of a population to be accompanied by changes in the value of the dispersion index. When this happens, however, two conclusions are possible: either the dispersion changes with density, or the index itself is affected by density. Unfortunately, without a definition of dispersion that is independent of the index used to measure it, it may not be possible to choose between these two possibilities. Thus, we need to refine the concept or definition of an index that is unaffected by changes in population density. Several investigators have studied effects of density on measures of dispersion in simulated populations (e.g. Myers 1978). One approach would be to create a simulated population with an arbitrary pattern of dispersion, and then to add simulated individuals to increase the density. The problem is knowing how to add individuals without changing the degree of clumping. If we added individuals at randomly selected locations in the simulated space, we would presumably reduce the degree of clumping if the population were aggregated. A less arbitrary approach would be to *remove* individuals, selected at random, from the simulated population. By definition, we could say that an index of dispersion would be independent of density, if by random removal the index remained unchanged. This criterion has been adopted by several investigators, and several of the indices that we will discuss are independent of density by this criterion. However, not everyone accepts this definition. By removing individuals selected at random, more individuals are removed from the patches of high density, and thus one might argue that dispersion is decreased (Pielou 1977, Taylor 1984).

An obvious candidate for an index of dispersion is the **variance/mean ratio** ($s^2/\bar{x}$). For randomly distributed populations $s^2/\bar{x} = 1$; for aggregated populations $s^2/\bar{x} > 1$. The greater the degree of clumping the higher the value of $s^2/\bar{x}$. However, this index is highly subject to changes in density by the criterion given above. Hence, the variance/mean ratio cannot be used to compare the degree of aggregation of two populations that differ in density.

A simple modification of the variance/mean ratio is **Green's index** (Green 1966):

$$\frac{\left(\frac{s^2}{\bar{x}}\right) - 1}{\sum (x) - 1} \qquad (2.11)$$

Green's index ranges from 0 for randomly distributed populations to 1.0 at maximum contagion and is supposedly independent of density. However, the index is influenced by the number of samples taken and thus cannot be used to compare two or more populations from which different numbers of samples have been taken.

A widely used index of dispersion is ***k* of the negative binomial** (Anscombe 1949, 1950). As indicated above, the frequency distribution of counts from many populations with a clumped pattern of dispersion, approximates a negative binomial quite closely. Goodness-of

-fit may be evaluated with a chi-square test, as illustrated above. The negative binomial is defined by two parameters, μ and k. Small values of k imply aggregated distribution; as $k \longrightarrow \infty$, the distribution approaches a Poisson. Values of k near 2.0 are typical for many populations. Frequently $1/k$ is used as the index of dispersion because, unlike k, $1/k$ increases with increasing degree of aggregation. Kuno (1991) and Pielou (1977) have shown that k is not affected by changes in density under the random removal criterion described above. However, k for real populations frequently does change with density, presumably because changes in density are frequently accompanied by changes in patterns of dispersion. For those users who are interested in developing a sequential sampling scheme based on k of the negative binomial (see Chapter 3), techniques for calculating a "common k" have been developed (Bliss and Owen 1958). However, if k changes markedly with density, such calculations are of questionable value and, sequential sampling schemes based on some other distribution are advisable (see Chapter 3).

Estimating k of the negative binomial

A variety of techniques have been devised to estimate k of the negative binomial from sample data (see reviews in Elliott 1977). An approximate method derives directly from the equation (2.4) for the theoretical variance of the negative binomial:

$$k = \frac{\bar{x}^2}{s^2 - \bar{x}} \tag{2.12}$$

As demonstrated by Anscombe (1950), this formula is accurate only for relatively high values of k and/or low values of $\bar{x}$ ($\bar{x} < 4$). However, it is useful for generating a starting value for the more accurate maximum likelihood equation:

$$N \ln\left(1 + \frac{\bar{x}}{k}\right) = \sum_{x=0}^{n}\left(\frac{A_{(x)}}{k + x}\right) \tag{2.13}$$

where

$A_{(x)}$ = number of samples with more than x individuals.
N = total number of samples,
n = maximum number of individuals per sample (i.e., the highest frequency category)

This equation cannot be solved for k. Instead, the correct value for k is determined by entering a starting value for k, derived from the approximate method, into both sides of the equation, and then modifying that value by successive approximation until both sides of the equation are equal. Such a task is easily and quickly accomplished on a computer, for example, with spread-sheet software.

Lloyd's mean-crowding and patchiness indices

Lloyd (1967) proposed indices of crowding and dispersion, that unlike k of the negative binomial, have a biologically meaningful definition. He defined mean crowding (m^*) as the mean number of other individuals per individual per sample unit. For each of the x_i individuals in the i^{th} sample, there are $x_i - 1$ other individuals. Thus:

$$\overset{*}{m} = \frac{\sum_{i=1}^{Q} x_i(x_i - 1)}{\sum x_i} = \frac{\sum(x_i^2)}{\sum x_i} - 1 \tag{2.14}$$

where

Q = total no. of samples
x_i = number of individuals in the i^{th} sample

If the population has been completely censused, so that the population mean (m) and variance (σ^2) can be measured directly, then equation (2.14) can be expressed as:

$$\overset{*}{m} = m + \left(\frac{\sigma^2}{m} - 1 \right). \tag{2.15}$$

When the pattern of dispersion is random, $\sigma^2/m \longrightarrow 1$, $(\sigma^2/m - 1) \longrightarrow 0$. In other words, $m^*=m$ for random patterns, and $m^*>m$ for aggregated populations. The mean-crowding index is a measure of crowding; it is not an index of dispersion. Crowding increases with density (m) and aggregation ($(\sigma^2/m) - 1$). An obvious extension of the mean-crowding index yields the **patchiness index:** m^*/m. Unlike the mean-crowding index, the patchiness index is a measure of dispersion and furthermore, has been shown to be independent of density under the random removal criterion (Pielou 1977).

For populations for which m and σ^2 must be estimated from samples, the most obvious method for computing x^* (an estimate of m^*) is as follows:

$$\overset{*}{x} = \bar{x} + \left(\frac{s^2}{\bar{x}} - 1 \right) \tag{2.16}$$

However, this estimate is not unbiased, although it has been employed by many researchers. A formula that corrects for sample bias is:

$$\overset{*}{x} = \bar{x} + \left(\frac{s^2}{\bar{x}} - 1 \right) \left(1 + \frac{s^2}{N\bar{x}^2} \right) \tag{2.17}$$

If the frequency distribution fits the negative binomial, then:

$$\overset{*}{x} = \bar{x} + \frac{\bar{x}}{k} \tag{2.18}$$

or

$$\frac{\overset{*}{x}}{\bar{x}} = 1 + \frac{1}{k} \tag{2.19}$$

The latter definition provides a biological interpretation for k of the negative binomial. The inverse of k ($1/k$) is the degree to which the patchiness index $x^*/\bar{x}$ exceeds 1.0.

Iwao's patchiness regression

Iwao (1968) extended the concept of Lloyd's patchiness index by calculating a linear regression of x^* vs $\bar{x}$:

$$\overset{*}{x} = \alpha + \beta \bar{x} \tag{2.20}$$

where α and β are the usual regression coefficients (intercept and slope). Iwao considered these coefficients to be separate indices, each expressing aggregation at a different spatial scale and appropriate for measuring aggregation for colonial insects such as aphids (Fig. 2.4).

Iwao referred to the intercept (α) (the value of x^* when $\bar{x} \longrightarrow 0$), as the index of "basic contagion". It is a measure of colony size or of clumping on a scale that is smaller than that of the sample unit. The slope (β) measures clumping on the same scale as the sample unit and is identical to Lloyd's patchiness index.

Substituting $\alpha + \beta m$ for m^* into equation (2.15) and solving for σ^2 yields the following relationships between the mean and variance:

$$s^2 = (\alpha + 1)\bar{x} + (\beta - 1)\bar{x}^2 \tag{2.21}$$

This equation formed the basis of Kuno's (1969) and Iwao's (1975) approach to sequential sampling, as discussed in Chapter 3.

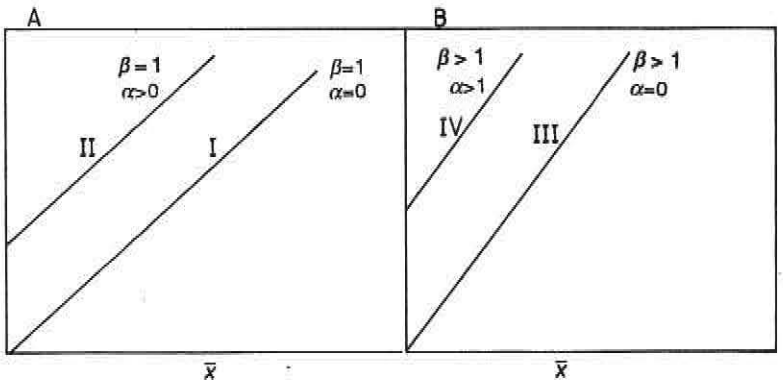


Fig. 2.4 Iwao's patchiness regression of mean crowding (x^*) vs the mean ($\bar{x}$) for (A) randomly distributed solitary (I) or colonies or patches of individuals (II), or (B) aggregated individuals (III) or aggregated colonies (IV).

Taylor's power law

Taylor (1961, 1971, 1984) showed that for many species of insects and other animals, there is a strong linear relationship between the log variance and the log mean for a set of samples that span a range of densities (Fig. 2.5):

$$s^2 = a\bar{x}^{-b} \tag{2.22}$$

or

$$\log\left(s^2\right) = \log(a) + b * \log\left(\bar{x}\right) \tag{2.23}$$

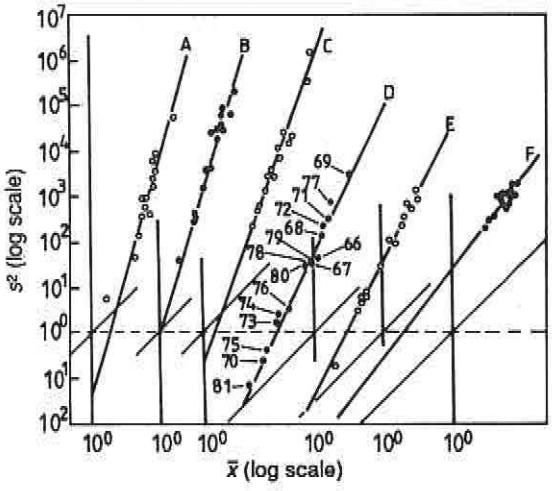


Fig. 2.5 Taylor's power law: Linear relation between log variance and log mean density for different insect species sampled at many sites across Great Britain. Each point represents one year's sample at these sites. The data are for adults of (A) *Aphis sambuci* (b = 3.39), (B) *Lycophotia poorphyrea* (b = 3.11), (C) *Agrotis exclamationis* (b =2.64), (D) *Euproctis chrysorrhoea* (b = 2.04), (E) *Cymatophorima diluta* (b = 1.96), (F) *Epirrhoe altrenata* (b = 1.37). Graphs for each species are displaced along horizontal axis to improve readability of figure. Redrawn from Taylor (1984) with permission from *Annual Reviews of Entomology*.

Taylor proposed that a was a sampling factor but that the slope (b) was an index of aggregation that was independent of the sample procedures and a characteristic of the species. For a random pattern of dispersion, b = 1.0; for aggregated populations $b > 1.0$, and for uniformly distributed populations $b < 1.0$. Unlike the dispersion indices described above, the parameter b can only be estimated by collecting data from different populations that differ in density. Therefore, it cannot be used to assess differences in dispersion between two populations. The primary uses have been in developing sampling schemes and in calculating

the appropriate transformation for purposes of statistical analyses. Sawyer (1989) has demonstrated that b can be affected by the size of the sample unit.

Several authors have attempted to explain the startling universality of Taylor's power law for many different species. Taylor and Taylor (1977) propose a behavioural model to explain it, a balance between density-dependent emigration and aggregation. This concept was embodied in a simulation, the Δ-model (RAJ Taylor 1981 a, b). Details of the Δ-model have been criticized by Thorarinsson (1986). Anderson et al. (1982) offer an alternative explanation based on random population processes of birth, death, emigration, and immigration (see also Hanski 1987).

A debate has ensued as to whether Taylor's power law (eq. 2.22) or Iwao's patchiness regression (eq. 2.21) best describes the relation between mean and variance in samples from most populations and, as a consequence, which approach is best for data transformation or sequential sampling (see Chapter 3). Iwao (1968, 1975) argues that his approach is derived from biological phenomena such as crowding and is therefore preferable. Taylor (1984) argues that for many species the power law remains linear, whereas Iwao's patchiness regression is non-linear in the vicinity of origin. An example of this is shown in the distribution of cassava mealy bug, *P. manihoti* in Nigeria (Schulthess et al. 1989).

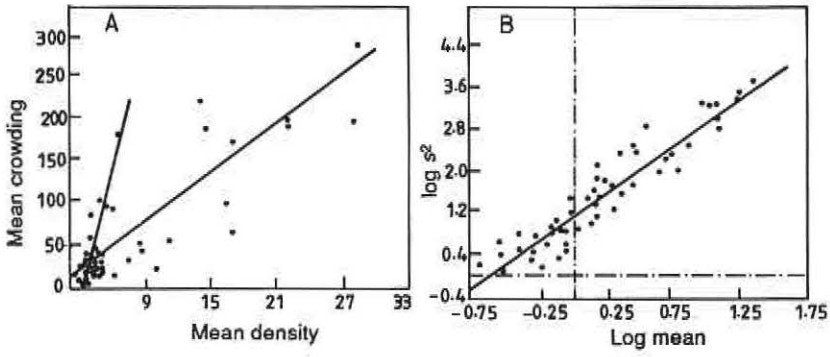


Fig. 2.6 (A) Iwao's patchiness regression of mean crowding vs mean and (B) Taylor's power law regression of log s^2 vs log (mean) for cassava mealybug during the dry season. (Redrawn with permission from Schulthess et al. 1989).

Another index of dispersion is **Morisita's index I_σ** (1959) which is essentially identical to Lloyd's patchiness index, although it is derived in a different manner. As with the patchiness index and k of the negative binomial, it is thought to be independent of density under the random removal criterion.

Effect of size of sample unit on measures of dispersion

We have indicated that the pattern of dispersion depends on the scale on which it is studied. Several authors have studied the systematic variation in the value of dispersion indices as a function of the size of the sample unit: frequently a quadrat, a square area of ground within which counts are made. Greig-Smith (1964) demonstrated that the coefficient of variation ($s^2/\bar{x}$) is maximum when quadrat size and clump size are comparable. Morisita (1959) showed that his index, I_σ, changed rapidly as the quadrat size approached the average clump size. Iwao (1972) proposed an index (ρ) to quantify the relation between clump size and quadrat size:

$$\rho = \frac{\overline{x}^{*}_{i} - \overline{x}^{*}_{(i-1)}}{\overline{x}_{i} - \overline{x}_{(i-1)}} \qquad (2.24)$$

where x_i^* = mean crowding in i^{th} sized quadrat. As with Morisita's index, the value of ρ changed markedly when quadrat size approached clump size.

EXERCISES

Using the same data given in the exercises for Chapter 1, do the following:

1. Construct a frequency histogram.
2. Determine if the population of flower thrips is uniform, random or aggregated. First determine if the variance is greater or less than the mean. Then determine if the distribution is significantly different from random using a chi-square test calculated from the approximate formula and the chi-square goodness-of-fit to the Poisson distribution. Lump together all frequency categories with expected values less than 1.0. Look up χ^2 values in a table and also use the method based on normal approximation to chi-square.
3. If the population is clumped, calculate k of the negative binomial using the approximate and exact (maximum likelihood) formulae. The maximum likelihood calculation can be easily accomplished on a spread-sheet. The value of k is adjusted until the two sides of the equation are equal.
4. Calculate x^* and $x^*/\bar{x}$ using 2 formulae (one with and one without k).

3

SEQUENTIAL SAMPLING

A basic premise of integrated pest management (IPM) is that pest control actions are only implemented when the pest species has attained a density above which the costs of crop losses outweigh the expenses of pest control actions. An **economic threshold** is the pest density at which action is required to prevent the density from reaching the **economic injury level,** or the density at which costs of treatment balance losses Sampling schemes designed to determine whether the pest species has attained the economic threshold, thus lie at the heart of most IPM programs. Sequential sampling is a technique for optimizing sample effort so that only a minimum amount of sampling is done in order to make a rational decision, as to whether densities are high enough to warrant action. Sequential sampling can also be used to determine the number of samples necessary to achieve a specified level of precision in an estimate of mean density (Kuno 1969, Green 1970), as discussed below.

Sequential sampling was developed by Wald (1945) as a technique for assessing the quality of industrial products. It was applied to entomology by Waters (1955).

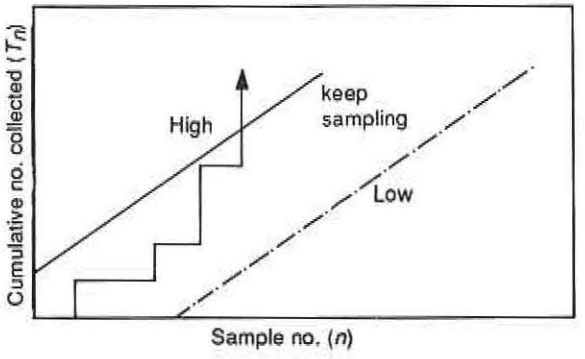


Fig. 3.1 A hypothetical sequential sampling scheme: T_n, the cumulative number of individuals collected is plotted against the number of samples taken (n).

The basic idea of sequential sampling is illustrated in Fig. 3.1. Samples are collected and the cumulative number of individuals collected (T_n) is plotted against the number of samples taken (n). As long as T_n plotted versus n remains between the upper and lower decision lines, sampling continues. If the cumulative number

crosses the upper decision line, we conclude that the population is high and treatment is called for. If the cumulative number crosses the lower decision line, we conclude that the population is low. In a pest management context, we conclude that no treatment is required.

Three types of information are needed in order to develop a sequential sampling scheme. The first of these is a mathematical description or model of the pattern of dispersion, as described in Chapter 2. Secondly, the economic threshold must be determined, or else some other definition is required of what constitutes a high density population. Finally, information is needed on the level of risk we are willing to accept or the probability of making a tolerable wrong decision. There are two types of risk: **Type I error** (α) which is the risk of calling the population high when it is actually low, and **Type II error** (β), which is the risk of calling it low when it is actually high. Typically, growers or pest managers can tolerate much higher Type I errors than Type II. Thus, we might set $\alpha = .25$ and $\beta = .05$.

For sequential sampling schemes based on Wald's (1945) technique (known as the sequential probability ratio test, SPRT), the decision lines (d_1, d_2) are linear and parallel:

$$d_1 = bn + h_1$$
$$d_2 = bn + h_2 \qquad (3.1)$$

where b is the common slope, n is the number of samples taken and h_1, h_2 are the intercepts. The technique requires that we specify two different critical densities (x_1, x_2), such that the population is classified as low (no treatment required) if the density is below x_1, and high (treatment required) if density is above x_2. Formulae for b, h_1 and h_2 are given in Table 3.1 for populations with random (Poisson), aggregated (negative binomial) and binomial distributions.

Table 3.1 Equations for estimating decision line parameters: b = slope, h_1 = intercept of the lower line, h_2 = intercept of the upper line for 3 probability distributions: Poisson, Binomial and Negative Binomial (adapted from Waters 1955 and Shepard 1980)

	Poisson	Negative Binomial	Binomial
Slope (b)	$b = \frac{x_2 - x_1}{\ln x_2 - \ln x_1}$	$k \frac{\ln (q_2/q_1)}{\ln [\frac{p_2 q_1}{p_1 q_2}]}$	$\frac{\ln [(1-x_1)/(1-x_2)]}{\ln [(x_2/x_1)]\ [(1-x_1)/(1-x_2)]}$
h_1	$h_1 = \frac{\ln [\beta/(1-\alpha)]}{\ln x_2 - \ln x_1}$	$\frac{\ln [\beta/(1-\alpha)]}{\ln [\frac{p_2 q_1}{p_1 q_2}]}$	$\frac{\ln [\beta/(1-\alpha)]}{\ln [(x_2/x_1)]\ [(1-x_1)/(1-x_2)]}$
h_2	$h_2 = \frac{\ln [(1-\beta)/\alpha]}{\ln x_2 - \ln x_1}$	$\frac{\ln [(1-\beta)/\alpha]}{\ln [\frac{p_2 q_1}{p_1 q_2}]}$	$\frac{\ln [(1-\beta)/\alpha]}{\ln [(x_2/x_1)]\ [(1-x_1)/(1-x_2)]}$

where x_1 and x_2 are the respective lower and upper critical densities that define populations as low or high, α and β are the levels of risk acceptable for incorrectly classifying the densities as respectively high or low, k is k of the negative binomial and $p_1 = x_1/k$, $q_1 = 1 + p_1$, $p_2 = x_2/k$, $q_2 = 1 + p_2$.

Sequential sampling schemes can be evaluated by calculating two curves. The first of these is the operating characteristics (OC) curve (Fig. 3.2A), which expresses the probability of deciding that the population is low over the range of possible values of the true population mean. The second is the average sample number (ASN) curve (Fig. 3.2B), which expresses the expected number of samples that will be taken before a decision is made for any true population mean. For every OC curve there is a complementary curve that expresses the probability of classifying the population as high. Usually, only the former OC curve is given, as in Fig. 3.2A. A sequential sampling plan is only feasible if the slope of the OC curve is steep and the ASN curve indicates that high numbers of counts are required for a narrow range of values for the population mean. The ASN curve reaches a peak and the OC curve equals 0.50 at the critical density. Details of calculating OC and ASN curves for Wald's (1945) are given

in Waters (1955) and in Onsager (1976). More recent sequential sampling techniques require simulation to construct such curves.

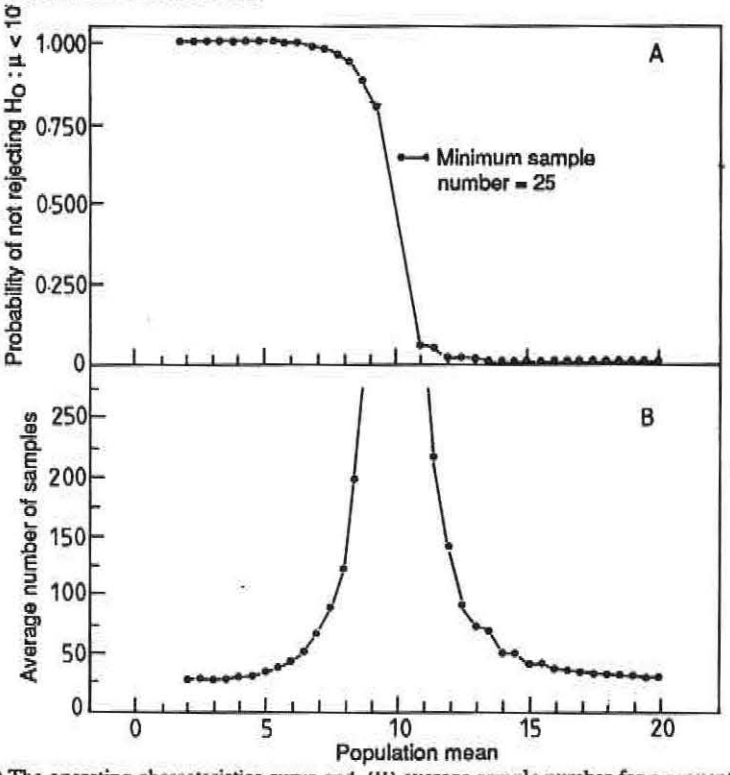


Fig. 3.2 (A) The operating characteristics curve and, (B) average sample number for a sequential sampling scheme for green peach aphids, *Myzus persicae* (Sulzer) on potato. Minimum sample size is set at 25. (Hollingsworth and Gatsonis 1990). Redrawn with permission from the Entomological Society of America.

Iwao (1975) introduced a sequential sampling technique based on his patchiness regression (Chapter 2, Iwao 1968) that offers several advantages over those based on Wald's (1945) SPRT method. First, only one critical density, such as the economic threshold, need be specified. Second, the scheme specifies stopping rules for cases in which T_n remains indefinitely between the upper and lower decision lines. Iwao's technique is very easy to grasp; it is expressed in terms of a confidence interval about the critical density (x_c) or economic threshold. If the estimated population density is equal to x_c, then the estimated confidence interval about x_c is:

$$CI = x_c \pm Z\sqrt{\frac{s^2}{n}} \tag{3.2}$$

where Z is the standard normal deviate, (see Chapter 1) for the appropriate level of allowable risk. Because sequential sampling schemes are based on the accumulated number sampled: $T_n = n^*\bar{x}$, we multiply by n to obtain a confidence band about T_n:

$$\text{upper line} = UL = nx_c + Z_{\frac{\beta}{2}}\sqrt{ns^2}$$

$$\text{lower line} = LL = nx_c - Z_{\frac{\alpha}{2}}\sqrt{ns^2} \tag{3.3}$$

The values of Z may be different for the upper and lower lines reflecting the different levels of allowable risk (α, β), as described above. The variance, s^2, is expressed as a function of the mean density, which, in this case, is equal to the critical density x_c, i.e. ($s^2 = f(x_c)$). Iwao's (1975) method is based on his patchiness regression (eq. 2.21), (with α_1 the intercept and β_1 the slope) so the equation for the decision lines become:

$$UL = n x_c + Z \sqrt{n\left((\alpha_I + 1)x_c + (\beta_I - 1)x_c^2\right)}$$

$$LL = n x_c - Z \sqrt{n\left((\alpha_I + 1)x_c + (\beta_I - 1)x_c^2\right)} \qquad (3.4)$$

The resulting decision lines are non-linear. Other researchers (Green 1970, Maiteki and Lamb 1987, Hollingsworth and Gatsonis 1990), have substituted Taylor's power law (eq. 2.22) instead of eq. 2.21, for s^2, yielding:

$$UL = n x_c + Z \sqrt{n a x_c^b}$$

$$LL = n x_c - Z \sqrt{n a x_c^b} \qquad (3.5)$$

The maximum number of samples to take whenever T_n remains between the upper and lower decision lines is calculated by specifying a level of precision in terms of a half-width of a confidence interval, either as a fixed distance (h, eq. 1.7) or a fixed proportion (d, eq. 1.6), and substituting f(T_n/n) for s^2:

$$n_{max} = \frac{\left(Z_{\frac{\alpha}{2}}\right)^2}{h^2}\left(s^2\right) \qquad (3.6)$$

Sampling for a fixed level of precision

Kuno (1969) proposed similar ideas for using sequential sampling to determine the minimum number of samples required to achieve a predetermined level of precision in the estimated mean density, instead of classifying the population into categories of high and low. As indicated in Chapter 1, standard formulae (eq. 1.5) can be used to estimate the number of samples required to achieve a SE expressed as a proportion (D_o) of the mean. In terms of the cumulative number counted T_n, the mean = T_n/n. Thus :

$$D_0 = \sqrt{\left(\frac{n}{T_n^2}\right)s^2} = \sqrt{\left(\frac{n}{T_n^2}\right) f\left(\frac{T_n}{n}\right)} \qquad (3.7)$$

Substituting equation (2.21) for f(T_n/n) and solving for T_n yields:

$$T_n = \frac{\alpha_I + 1}{\left[D_o^2 - \frac{(\beta_I - 1)}{n}\right]} \qquad (3.8)$$

where D_o is a predetermined level of precision (e.g. 0.10 or 0.25). Plotting T_n against n yields a straight line if the population is randomly dispersed ($\beta_1 = 1$), or a curve otherwise.

Green (1970) proposed using Taylor's power law for f(T_n/n):

$$D_o = \sqrt{\frac{n}{T_n^2} * \frac{a T_n^b}{n^b}} = \sqrt{a n^{1-b} T_n^{b-2}} \qquad (3.9)$$

This rearranges to

$$\log T_n = \frac{\log\left(\frac{D_o^2}{a}\right)}{b-2} + \frac{b-1}{b-2}(\log n) \qquad (3.10)$$

which is a straight line when log T_n is plotted versus log n.

Firempong and Magalit (1990) compared both approaches in samples of the legume pod borer, *Maruca testulalis* (Geyer), a major pest of cowpea in Africa (Table 3.2). Frequently, it is much easier to use a table rather than a graph (as in Fig. 3.1) to determine termination points in the field. Regression estimate of b of Taylor's power law and β of Iwao's patchiness regression indicated that the population was randomly dispersed. Firempong and Magalit concluded that the results support the arguments of Taylor (1984) that fewer samples are required to achieve a given level of precision with his method. Such determinations should be made for each species for which sampling schemes are developed.

Table 3.2 Sequential sampling table for legume pod borer larvae indicating number of samples required to terminate sampling in order to achieve three different levels of precision (from Firempong and Magalit 1990 with permission from ICIPE Science Press)

No. of flowers	Green's (Taylor's) method Precision level (%)			Iwao and Kuno's method Precision level (%)		
	10	20	30	10	20	30
1	253	52	21	–	–	–
2	230	48	19	–	–	–
3	218	45	18	–	–	47
4	209	43	17	–	–	27
5	203	42	17	–	–	22
6	198	41	16	–	168	19
7	194	40	16	–	96	17
8	190	39	16	–	72	16
9	187	38	15	–	61	16
10	185	38	15	–	54	15
12	180	37	15	–	46	15
14	176	36	15	–	42	15
16	173	36	14	–	39	14
18	170	35	14	–	37	14
20	168	35	14	–	36	13
22	166	34	14	1300	35	13
24	164	34	13	672	34	13
26	162	33	13	554	33	13
28	160	33	13	382	32	13
30	158	33	13	326	32	13

– indicates that no estimate is possible.

Presence or absence sampling

Sequential sampling schemes based on the binomial distribution are used when the data collected consist of presence or absence of the insect in a sample rather than counts. Such data are frequently much easier and less expensive to collect, especially for insects such as aphids that exist in colonies so that one sample might contain hundreds of individuals. The variance of the binomial is :

$$s^2 = np(1 - p) \tag{3.11}$$

where np = mean and p is the frequency of samples with one or more individuals. Several approaches have been proposed for estimating p (reviewed in Kuno 1991, Binns and Nyrop 1992); one of these is the zero term of the negative binomial (eq. 2.5):

$$p = 1 - P(X = 0) = 1 - \left(1 + \frac{\bar{x}}{k}\right)^{-k} \tag{3.12}$$

Formulae for binomial sampling based on Wald's SPRT method (1945) are given in Table 3.1.

Double sampling plans (Binns & Nyrop 1992) consist of simplified sequential sampling protocols that involve making a preliminary sample that is used to estimate the population

variance. The sample size required to achieve a specified level of precision in a subsequent sample is then calculated. Such schemes are especially appropriate in cases where counts are made from samples after they have been collected in the field. The workings of a double sampling plan can be illustrated with a figure (Fig. 3.3) from Kuno (1972).

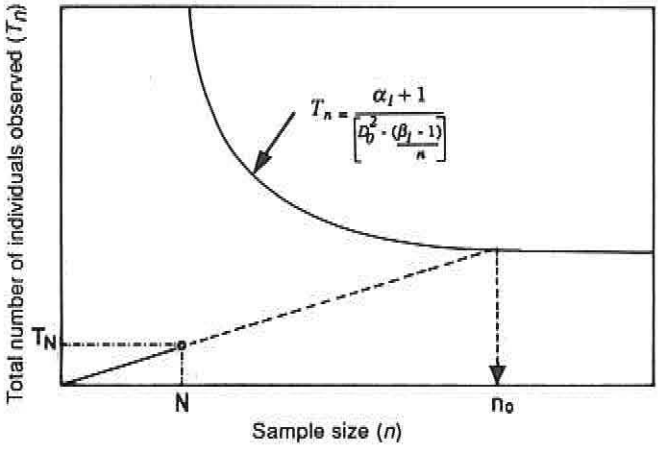


Fig. 3.3 A double sampling plan based on Kuno's equation (eq. 3.8) for a fixed level of precision. Reproduced with permission from the Society of Population Ecology.

The stop line (eq. 3.8) is plotted on a graph of T_n versus n. A preliminary sample of arbitrary size N is collected and T_N is counted. A line drawn from the origin through the point (N,T_N) intersects the stop line at (n_0, T_{n0}). This gives the expected number of samples required (n_0) to achieve the specified level of precision. An additional sample of size n_0–N is then collected from the field in order to achieve the desired level of precision.

EXERCISES

1. Develop a sequential sampling scheme based on the data from the flower thrips/cowpea system that was presented in the exercises for Chapters 1 and 2. Use the maximum likelihood estimate of k of the negative binomial (eq. 2.13) and the appropriate sequential sampling equations for a negative binomial distribution from Table 3.1 for Wald's (1945) SPRT test. Your research shows that no pesticide treatments are necessary whenever densities are less than 5 per plant. You are willing to tolerate a 10% risk of misclassifying your field as less than 5/plant when it is really greater than that, and a 25% risk of calling it greater than 1/plant when it is really less than that. Calculate the equations for the decision lines and draw them on graph paper.
2. Use Iwao's formula (eq. 3.4) to calculate decision lines based on his method. Draw them on the same graph as the SPRT lines above. How do the two methods compare? Calculate a maximum number of samples to take based on a confidence interval of 20% of the mean.

4

MARK-RECAPTURE METHODS

Mark-recapture techniques represent an important methodology for estimating population density, as well as studying dispersal. The technique involves releasing marked individuals into a population and then obtaining samples from the population to determine the proportion of individuals that carry the mark. Seber (1982, 1986) has provided detailed reviews of mark-recapture literature. An excellent nontechnical summary can be found in Begon (1979). Most computations of density are based upon the Lincoln index, also known as the Peterson estimate. The idea embodied in this index is very simple: that the proportion of marked to unmarked individuals in the sample is equal to that in the population:

$$\frac{m}{n} = \frac{M}{N} \tag{4.1}$$

where m = Number of individuals marked in sample
n = Number captured in sample
M = Number marked in the population
N = Number of individuals in the population

Since m and n are determined from the samples, and M is assumed to be equal to the total number of marked individuals released, the density can be calculated:

$$N = M\left(\frac{n}{m}\right) \tag{4.2}$$

This index was first derived by Peterson (1896) and used by Lincoln (1930) to estimate duck populations. Bailey (1951) proposed the following correction for bias for small samples ($m < 10$):

$$N = M\frac{n+1}{m+1} \tag{4.3}$$

Calculations of density based on the Lincoln index, or subsequent derivatives of the Lincoln index, entail making the following assumptions (reviewed in Begon 1979):

1. **All marks are permanent;** marked individuals do not lose their mark.

2. **Marked and unmarked individuals are equally likely to be captured.** This includes the assumptions that marking does not harm the individual or in any way alter its behavior, that individuals do not become trap-shy or trap-happy, and that marked and unmarked individuals are equally likely to emigrate.
3. **Marked individuals and unmarked individuals mix thoroughly** in the population.
4. **The population is homogeneous,** meaning that all individuals have an equal chance of capture regardless of sex, age, or trap location. When such differences exist between subgroups within a population, mark-recapture estimates should be made separately for each group.
5. **Samples are "instantaneous"**; meaning that samples are collected over a short interval relative to the periods between samples and that the population density does not change significantly during the sample interval.

In addition to these assumptions, the different methods of estimating density from mark-recapture data make varying assumptions about the occurrence of gains and losses to the population during the period when samples are collected. Closed population models allow neither gains nor losses. Open models allow both. Gains include additions by birth or immigration. Losses include deaths or emigration. Typically, mark-recapture estimates do not distinguish death from emigration or birth from immigration as sources of loss or gain. Calculations based on the Lincoln index allow for loss but no gain. If we assume loss but no gain, M/N will stay constant, provided that losses affect N and M equally. However, if we assume gain but no loss, M will remain the same, N will increase and, therefore, M/N will decrease. In other words, the marks will be diluted. M/N is thus an estimate of the population size at the moment of sampling, not when population is marked.

Models involving multiple series of mark-recapture events

Closed mark-recapture estimates allow neither birth and immigration, nor death and emigration. However, other assumptions concerning homogeneity of the population or effects of the mark on behavior may be relaxed and statistical procedure for detecting them have been developed. Program CAPTURE (White et al. 1982) is a sophisticated software package for conducting such analyses and selecting the best estimate based on tests of these assumptions, including the assumption of closure. Thus, a closed model may be the appropriate choice, even though no population is truly closed. In addition to estimates of density, the program quantifies home range size. Such analyses are typically used in mark-recapture of small mammals. These animals are relatively long-lived and are territorial; hence the assumption of closure is a good approximation. Insect populations, in contrast, frequently experience high rates of loss and gain.

An additional restriction of most of the closed-model methods used by program CAPTURE is the requirement that each individual be marked uniquely. This can be achieved with some insect marking schemes, such as those that employ patterns of dots on different parts of the body. Many other marking techniques, however, such as those involving fluorescent powders, offer a very limited number of unique marks, so that identifying each marked individual uniquely is impossible. For both of these reasons, open models have been the rule for most mark-recapture studies of insects. Some species, however, such as adult dragonflies or butterflies, are relatively long-lived, occupy restricted home ranges, and can be marked uniquely. For such species, closed models are an appropriate choice.

Open models

Jackson's methods (1937, 1939) are appropriate when individuals cannot be recaptured and released a second time, for example, insects caught on a sticky trap. Jackson's positive method involved a single release followed by multiple recapture occasions. Jackson's negative method involved multiple sets of releases followed by a single recapture occasion.

Each individual was marked and released only once. Both methods yield a single estimate of population size and survival. Jackson developed his methods to estimate the density of tsetse flies, *Glossina morsitans morsitans* (Westwood).

The method of Fisher and Ford (1947) involves multiple release and recapture occasions and entails marking and releasing the same individuals repeatedly. The method assumes a single survival rate over the study period. Density is calculated from the combined recapture data over several intervals. This is the method to use when recaptures are rare, often the case with insects.

The method of Jolly (1965) or Seber (1962, 1965, 1982) has probably been used more often than any other mark-recapture method for insects. As with Fisher-Ford, it involves a series of mark and recapture occasions in which some individuals are repeatedly captured and released. Only the most recent mark is recorded. It calculates a density and new survival rate for each sample occasion. Thus, it does not assume survival is constant over the study interval. It does assume that differences in age of the individual between the first and last sample have no effect on recapture probability.

The following explanation of a Jolly-Seber mark-recapture procedure is adapted from Begon (1979). The procedure is a derivation of the Lincoln index. It differs from the Lincoln index in that M_i, the number of marked individuals available for capture on day (i), is not assumed to be equal to the total number of marked individuals previously released into the population. Instead, a procedure for estimating M_i is presented based on the following tautology:

$$M_i = m_i + (M_i - m_i) \tag{4.4}$$

where

M_i = marks at risk on day i

m_i = number of marks in sample on day (i)

$(M_i - m_i)$ = number of marks at risk not in sample on day (i). Here we use the term "day" to represent the interval between samples, which could be anything.

To estimate M_i, we first estimate $(M_i - m_i)$. For this purpose, several additional parameters are calculated. The parameter z_i is the number of $(M_i - m_i)$ caught on subsequent sample occasions. Furthermore, on day (i), r_i individuals are released, of which (y_i) are caught subsequently. On day (i) we have two groups of marked individuals, $(M_i - m_i)$ and (r_i), and we expect to recapture equal proportions of them on subsequent samples:

$$\frac{z_i}{M_i - m_i} = \frac{y_i}{r_i} \tag{4.5}$$

Since y_i, r_i, m_i, and z_i are known, M_i can be estimated:
$M_i = m_i + (z_i r_i / y_i)$. The population density on day (i), follows directly from the bias-corrected version of the Lincoln index (eq. 4.3, Bailey 1951):

$$N_i = \frac{M_i \ (n_i + 1)}{(m_i + 1)} \tag{4.6}$$

The daily rate of survival ϕ_i (1 – the daily loss rate) is calculated as follows: On day (i), immediately after a sample, there are $(M_i - m_i + r_i)$ marked individuals in the population. On day $(i + 1)$, immediately before the sample, there are $M_i + 1$ individuals. The daily rate of survival is the ratio of these two entities:

$$\phi_i = \frac{M_{i+1}}{M_i - m_i + r_i} \tag{4.7}$$

The number of individuals added to the population (B_i) is:

$$B_i = N_{i+1} - \phi_i N_i \tag{4.8}$$

As indicated above, gains include births and immigration, whereas losses (hence, survival rate) includes deaths and emigration.

Calculation of these parameters involves arranging the mark-recapture data in a so-called Jolly trellis diagram. Marked individuals in each sample are tabulated according to the sample day on which they were last released. If an individual has more than one mark, all but the last mark are ignored. I illustrate this (Table 4.1, 4.2) with the following example from a mark recapture study of a treehole mosquito *Aedes africanus* (Theobald) in Uganda (Sempala 1981). For the sake of brevity, I present only the first eight days of release and recapture. Sempala (1981) continued sampling for a total of 20 days, so that his density estimates differ slightly from what I present here. I employ the notation used in Begon (1979), which I believe is easier to follow than that introduced by Jolly (1965) and used in Southwood (1978) and Sempala (1981).

Table 4.1 A tabulation of recaptures of unfed marked *Aedes africanus* at Zika Forest, Uganda. (Reproduced with permission from Sempala 1981)

			Day Released							
i	n_i	r_i	1	2	3	4	5	6	7	m_i
1		120								
2	166	161	10							10
3	92	90	10	6						16
4	110	107	2	5	4					11
5	97	95	6	2	10	2				20
6	136	120	6	9	2	7	1			25
7	63	58	3	1	1	1	0	1		7
8	108	20	4	8	4	2	1	1	0	20
			$y_i = 41$	31	21	12	2	2	0	

For example, for day 3 we calculate the following parameters:

m_3 = No. of marks captured on day 3 = 10 + 6 = 16 (sum across row for day 3).

y_3 = No. of marks released on day 3 that were subsequently captured = 4 + 10+2 + 1 + 4 = 21 (sum of column for day 3).

z_3 = No. of marks released prior to day 3, not captured on day 3 but captured subsequently (all numbers to the left of the column for day 3 and below row for day 3) = 2+ 6 + 6 + 3 + 4 + 5 + 2 + 9 + 1 + 8 = 46.

Table 4.2 Summary parameters calculated from Table 4.1

Day i	m_i	y_i	z_i	M_i	N_i	ϕ_i
1	41				1.4	
2	10	31	31	171.0	2596	0.7
3	16	21	46	213.1	1166	1.8
4	11	12	56	510.3	4721	3.8
5	20	2	48	2300.0	10733	0.6
6	25	2	25	1525.0	8036	–
7	7	0	20	–	–	–
8	20			–	–	–

For example: $M_3 = 16 + (46)(90)/21 = 213.1$, and since $N_i = M_i\,(n_i + 1)/(m_i + 1)$, $N_3 = (213.1)\,(93)/17 = 1166$.

EXERCISES

This exercise is a version of Guess How Many Beans are in the Jar. We will use Jolly's stochastic method (Jolly 1965) as well as the Peterson estimate to calculate the population size. Place 1–1.5 kg of pinto beans in a bucket or other large-mouth container. Obtain coloured marking pens of seven unique colours. Coloured white-out also works. Working in groups of two or three will facilitate this process.

1. Your sample unit is a 50 ml beaker or other similar small container (ca. 100 beans). Remove a beaker-full from the population, count the beans and mark each one *on both sides* with one colour. Return the beans to the population and shake the jar to ensure mixing.
2. Remove 2 full beakers of beans from the population and discard. This simulates death and emigration. Add 2 full beakers of new unmarked beans to the population. This simulates birth and immigration. In this population birth rate = death rate and the population remains stable.
3. Conduct n samples in this manner, each with a unique colour, where n is the number of colours available. Tabulate the number of recaptures and record the sample occasion when each recaptured bean was last released. In this manner, construct a Jolly trellis. Each recaptured bean receives a new mark before it is returned to the population. In Jolly's method *only the most recent mark is recorded.*
4. Repeat the birth and death processes after each sample.
5. If no beans are available, do steps 6–9 with the following Jolly trellis:

			RECAPTURES					
i	n_i	r_i	1	2	3	4	5	6
1		143						
2	147	145	11					
3	146	144	2	8				
4	159	155	4	7	10			
5	157	157	2	3	4	9		
6	164	160	6	3	8	7	8	
7	148	145	0	0	3	9	12	14

6. Calculate $m_i\, y_i\, z_i\, M_i\, N_i$ and ϕ_i from the trellis diagram.
7. In addition, calculate a Peterson estimate of N_i (with correction factor for small sample size) for each sample occasion. Use the total number of marked beans released up to that sample as your M_i.
8. At the end of the lab, count the beans in the jar.
9. Explain the following:
   a. The difference in the results between the two methods. Why are they different? How close do they come to reality?
   b. The variability in the M_i estimates of Jolly's method — How confident can we be of this estimate?
   c. What are the mark recapture assumptions and to what extent were they violated here?

5

EXPONENTIAL GROWTH AND THE LOGISTIC EQUATION

Exponential growth

A fundamental property of the population dynamics of all species is that the number or density of individuals will increase at an ever-increasing rate when conditions are favourable. The simplest example of such growth is illustrated by the replication of single-celled organisms. A bacterium might divide every half hour, so that a colony that began with one individual would grow to: 2, 4, 8, 16, ... 2^t, where t is the number of replications. With insects, the rate of replication with each generation is potentially much faster. Consider, for example, a house fly, *Musca domestica*, which has a generation time of approximately 1 week and oviposits approximately 200 eggs. If half of those eggs were females and all of them survived to maturity, the population would increase by 100-fold each week. By the end of one year the population will have increased to 10^{104} (much larger than the number of atoms in the galaxy). Even if mortality was much higher, (for instance, suppose that only 1 fly in 10 survives to maturity) the end result would be the same but would take longer to achieve. Mathematically, we refer to this process as exponential growth and typically this is expressed with the following equation (Fig. 5.1A):

$$\frac{dN}{dt} = rN \qquad (5.1)$$

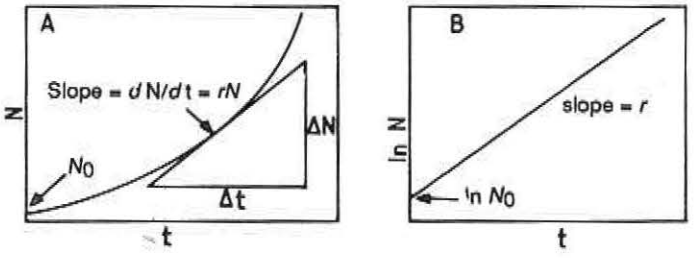


Fig. 5.1 (A) Exponential growth of a population on an arithmetic scale and, (B) a logarithmic scale.

where N is the population size or density, dN/dt is the growth rate (the instantaneous change in density per unit time) and r is a constant. This equation can be rearranged:

$$\frac{dN}{dt}\frac{1}{N} = r \tag{5.2}$$

indicating that r is the per capita rate of increase. The equation can be expressed in its integral form:

$$N_t = N_o e^{rt} \tag{5.3}$$

where N_0 is the initial population density and e is the base of Naperian or natural logarithms (e = 2.178). If N is plotted on a log scale, the resulting graph is linear with slope r and intercept ln (N_0) (Fig. 5.1B).

$$\ln(N_t) = \ln(N_0) + rt \tag{5.4}$$

As indicated in the example given above, r is influenced by the death rate. Specifically:

$$r = b - d \tag{5.5}$$

where b is the instantaneous birth rate and d is the instantaneous death rate. When the birth and death rates are equal, $r = 0$ and the population ceases to grow. When the death rate exceeds the birth rate, r is negative and the population declines.

The relationship between the exponential growth equation and the example of replicating microorganisms is easily derived from the following mathematical identity:

$$a^u = e^{u\ln(a)} \tag{5.6}$$

Where u is any function. Therefore:

$$2^t = e^{t\ln(2)} \quad \text{and} \quad r = \ln(2) = 0.693 \tag{5.7}$$

Strictly speaking, population growth will be exponential if, and only if, the age classes that make up the population exist in a certain ratio, which is known as the stable age structure. In the absence of a stable age structure, the instantaneous per capita growth rate will fluctuate. The value will be greater or less than that of a population with a stable age structure, depending on whether the ages of maximum reproduction are over- or under-represented in the population compared to the stable age structure. Lotka (1925) showed that the *average* instantaneous per capita rate of growth (slope of N_t regressed vs t) will be maximum when the stable age structure is attained. Lotka (1925) further showed that any population growing in an unlimited environment will soon attain such a stable age structure. As long as the birth and death rates of the population remain constant, the age structure of the population will then persist unchanged.

When a population has a stable age structure and is growing exponentially, r is known as the intrinsic rate of natural increase. Some authors or texts use r_m to denote the intrinsic rate of increase (Southwood 1978, Krebs 1978, Andrewartha & Birch 1954). Others including Birch (1948) and May (1976a) use r, a convention we follow here. It is thought that r is a characteristic of the species (Birch 1948), but it is important to note that r is specific to a particular set of environmental conditions. If the conditions change, so will r. Hypothetically, there exists an optimal set of conditions for a species under which r will take on a maximum value.

The notion of exponential growth was first expressed in the writings of Malthus (1798), who observed that human populations increased geometrically, whereas food production increased arithmetically. As a result, Malthus predicted that mankind will be doomed forever to a life close to the edge of starvation. Malthus did not foresee the large increase in

agricultural production that attended the industrial revolution, but he may yet prove to be correct. Malthus' ideas were fundamental to the development of Darwin's theory of evolution: all organisms must struggle to survive because all species produce more offspring than can survive. Lotka (1925) developed the notion of r and the use of life table analysis as it pertains to human populations. Leslie and Ranson (1940) introduced the concept of r to ecology and Birch (1948) shows how r could be calculated from life table data. We will explore these techniques in the next chapter.

Logistic equation

It is obvious that no population can continue expanding forever. Sooner or later it will reach a density above which individuals can no longer obtain the resources they need to survive. This density is known as the **carrying capacity** of the environment. For different species in different habitats the carrying capacity will be determined by competition for particular resources. For desert plants, water is typically the limiting resource. For many animals, food supply determines the carrying capacity. As populations expand towards the carrying capacity, the rate of growth slows down. This process is typically represented by the logistic equation (Fig. 5.2A):

$$\frac{dN}{dt} = rN\left(\frac{K - N}{K}\right) \tag{5.8}$$

where K is the carrying capacity and r and N are defined as above.

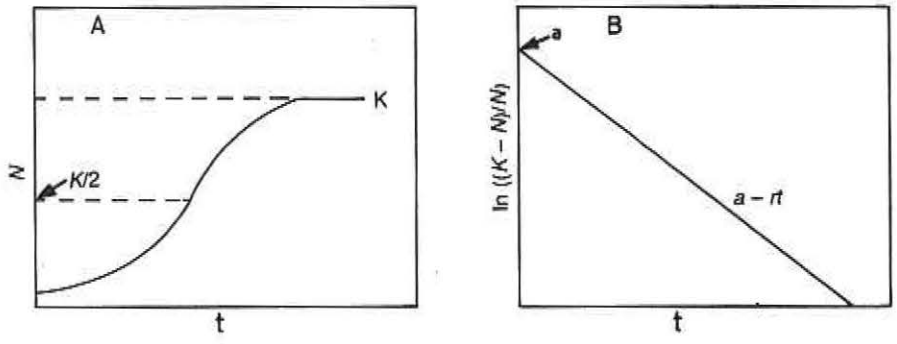


Fig. 5.2 (A) Logistic growth curve and, (B) linear form.

This equation is best understood if it is expanded:

$$\frac{dN}{dt} = rN - \frac{rN^2}{K} \tag{5.9}$$

The first term (rN) represents exponential growth. The effect of the second term, which has been called environmental resistance, increases as N becomes large. As $N \dashrightarrow K$, the rate of growth (dN/dt) approaches zero. The integral form of this equation:

$$N_t = \frac{K}{\left(1 + e^{a - rt}\right)} \tag{5.10}$$

can be rewritten in a linear form (Fig. 5.2B):

$$\ln\left(\frac{K - N}{N}\right) = a - rt \tag{5.11}$$

This can be fitted by regression of ln $((K-N)/N)$ against t, provided one knows the value of K.

The logistic equation was independently applied to population growth by Verhulst (1838) and Pearl and Reed (1920). Variations of the logistic have been explored by many individuals; indeed, it is the foundation of a large body of work in theoretical population ecology. Lotka (1925) and Volterra (1926) extended the logistic to describe competition between species and predator-prey interactions.

There are a number of assumptions inherent in the use of the logistic equation to represent population growth. The first of these is that the population will remain stable about the carrying capacity (K), unless otherwise disturbed. In actuality, most populations fluctuate in density, even populations kept in the laboratory under constant environmental conditions.

Another assumption involves an inherent linearity in per capita growth rate as a function of density, which can be seen if we rewrite the logistic:

$$\frac{dN}{dt}\frac{1}{N} = r - \frac{rN}{K} \tag{5.12}$$

As a result of this linearity, the point of inflection of the logistic (Fig. 5.2) occurs where $N = K/2$ and the shape of the curve is symmetrical above and below this point. Biologically, this means that the effects of the factor that limits the growth of the population will be felt even at the very lowest density. In actuality, there may be no effect of environmental resistance until densities approach the carrying capacity. For instance, many animals compete for space; caddisflies, for example, are limited by availability of space on rocks in a streambed. At low density there is no lack of space and competition for space is nonexistent. Only at densities near the carrying capacity will failure to find appropriate space constitute a significant drag on population growth. In other words, there is no biological reason why population growth should be symmetrical about $K/2$.

The discussion of the assumptions given above illustrates that few real populations can be expected to grow in a manner that is exactly described by the logistic equation. Indeed, there is no biological reason for choosing the logistic over any other function that gives a sigmoid shape. The importance of the logistic is its contribution to theoretical ecology. It captures the most basic processes of population dynamics: exponential growth and the effects of factors that limit growth. The great advantage of the logistic is its mathematical simplicity, which enables mathematicians to explore its properties in ways that would be impossible for more complex but biologically realistic equations. For these reasons the logistic equation occupies a central place in population ecology.

Modifications to the logistic

Any number of modifications have been made to the basic logistic equation (eq. 5.8) for the purpose of making it more realistic and for addressing some of the limitations listed above. For example, Gilpin and Ayala (1973) suggested the following generalization:

$$\frac{dN}{dt} = rN\left[1 - \left(\frac{N}{K}\right)^{\theta}\right] \tag{5.13}$$

where θ is a parameter that controls the point of inflection. If $\theta < 1.0$, the inflection point occurs at $N < K/2$; if $\theta > 1.0$, the inflection point occurs at $N > K/2$; and if $\theta = 1.0$, the equation reverts to the original logistic and the inflection point occurs at $N = K/2$. Other modifications concern the introduction of time delays into logistic growth on the grounds that many forms of environmental resistance have more effect on the density of the offspring of the population than on the immediate density. (Cunningham 1954, Beddington 1974, Beddington and May 1975, May 1981). For example, Cunningham (1954) suggested the following modification:

$$\frac{dN}{dt} = rN_t\left(\frac{1 - N_{t-\tau}}{K}\right) \qquad (5.14)$$

When t = time since the start of the population growth
τ = time lag (typically the generation time)

At low values of τ the population density approaches K smoothly and asymptotically. At high values of τ the population density oscillates about K. (Fig. 5.3).

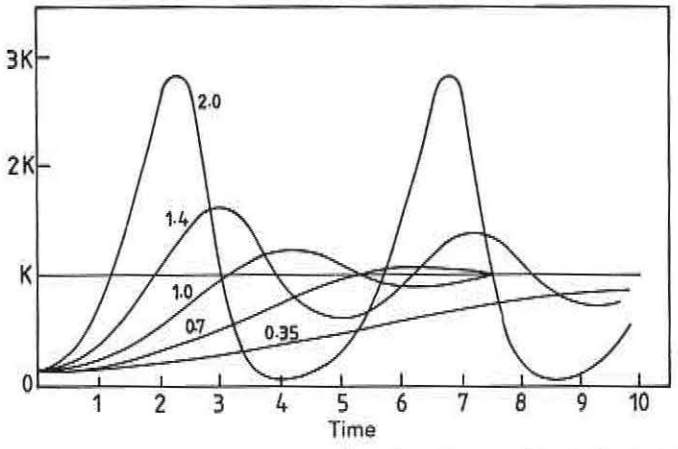


Fig. 5.3 The effect of different values of a time delay (τ) on the trajectory of density in a logistic equation (eq. 5.14) from Cunningham (1954).

Chaos

In a series of important papers, Robert May (1974, 1976b, 1986) explored the behavior of a version of the logistic appropriate for species with discrete generations:

$$N_{t+1} = N_t \exp\left[r\left(1 - \frac{N_t}{K}\right)\right] \qquad (5.15)$$

where N_t and N_{t+1} are population densities in successive generations

At low rates of reproduction (low values of r), the system stabilizes at a single equilibrium (Fig. 5.4A). At $r=2$, a 2 point cycle, that is a cycle that repeats every 2 generations (Fig. 5.4B) appears. As r increases, 4 point (Fig. 5.4C), 8 point, 16 point ... 2^n point cycles appear. This process is known as bifurcation. At values of r above 2.69, truly astonishing behavior appears (Fig. 5.4D) in which the dynamics of the population appear completely irregular or chaotic. These findings had profound implications. Prior to this, population biologists had always assumed that simple deterministic population models would have simple dynamics; they would yield stable density equilibria or regular cycles. It had always been assumed that the irregular temporal pattern of population change, or the erratic occurrence of population outbreaks, characteristic of many insect species, arose from stochastic processes such as weather conditions, which vary from year to year in an entirely irregular and unpredictable way. May's findings raised the possibility that such irregular dynamics were a mathematical consequence of underlying population processes.

Subsequent to the publication of May's work, a debate ensued as to whether populations of most species were driven by "deterministic chaos" or by stochastic events. For most population models, such as the discrete logistic given above, chaos only appeared at high values of the critical parameter, in this case when reproductive rate was high. Blackith and Albrect (1979) calculated the reproductive rate of the desert locust, *Schistocerca gregaria* (Forsk.) and the red locust, *Nomadacris septemfasciata*, (Serv.) and concluded that both species had reproductive rates sufficiently high to create chaotic dynamics. A logistic population model based on the parameter values is shown for the desert locust (Fig. 5.5A)

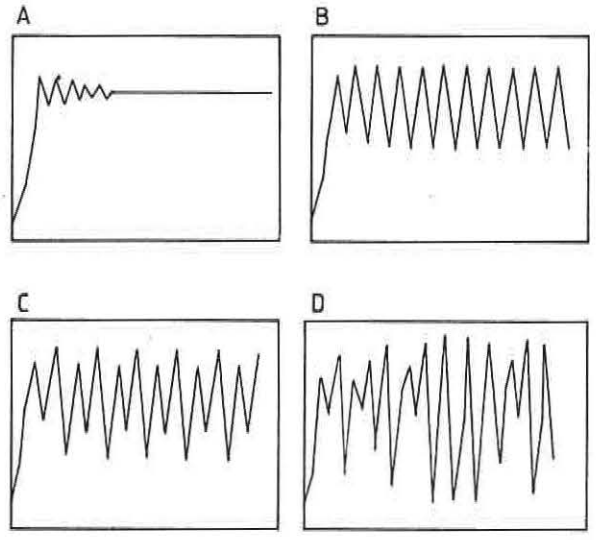
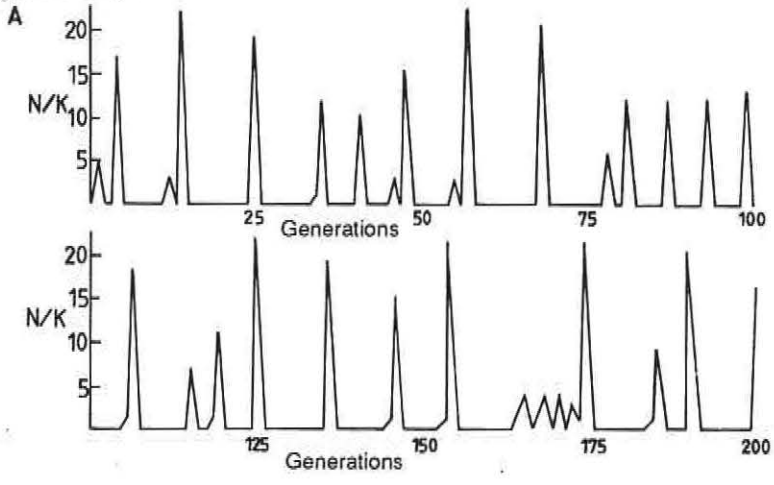


Fig. 5.4 Density of a hypothetical population plotted versus time for the discrete logistic (May 1976b) under different values of r (increasing from A–D) illustrating (A) a steady state, (B) a two point cycle, (C) a four point cycle and — (D) chaos.

compared to the frequency and duration of outbreaks (Fig. 5.5B) of several locust species (Farrow and Longstaff 1986).



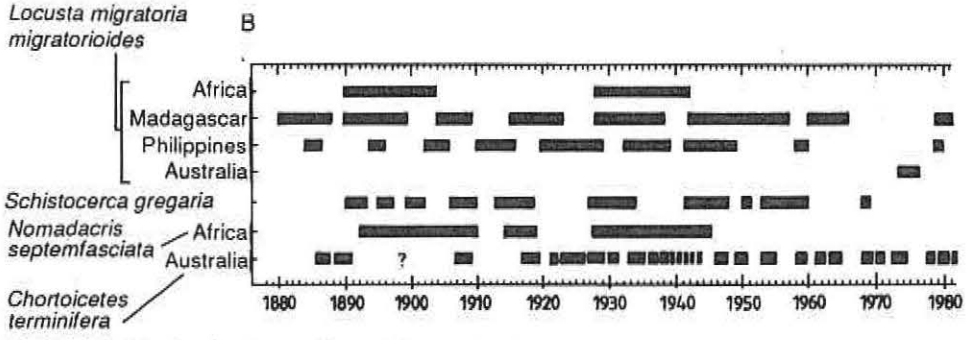


Fig. 5.5 (A) Simulated trajectory of population density of the desert locust over 200 generations built on the discrete logistic equation (eq. 5.15) (from Blackith and Albrect 1979 with permission). (B) The temporal pattern of outbreaks of several major locust species (from Farrow and Longstaff 1986 with permission from Oikos).

The latter authors disagreed with the conclusions of Blackith and Albrect. They argued that the irregular dynamics of most locust species is caused by stochastic meteorological events, as most scientists had always assumed. The stochastic nature of weather, may itself arise from chaotic interactions amongst the physical variables (temperature, pressure etc.) responsible for driving the weather, as first demonstrated by Lorenz (1963), who is known as the founder of the modern science of chaos. Recent analyses by Cheke and Holt (1993) of long-term data on abundance of the desert locust, were inconclusive regarding the existence of chaotic dynamics in the population fluctuations of this species. The degree to which deterministic chaos characterizes the population dynamics of most species remains debatable. However, the science of chaotic dynamics has permeated many fields of study, including planetary orbital mechanics, meteorology, economics, and physiology.

EXERCISES

You are raising a colony of cassava green mites in a greenhouse and you make daily counts of the number of individuals present. No counts were made when the colony was first started (day 0). It is clear that the population size is starting to level off after several days and you want to estimate the maximum size the population will attain in the future. You decide to estimate it using the logistic equation. Here are the counts:

Day No. t	Number of individuals N
1	67
2	188
3	466
4	920
5	1360
6	1618

1. Plot these data on a piece of graph paper and draw in an S-shaped curve by eye.
2. Estimate what you think K is.
3. Enter the data in the computer and run a regression of:

$$\ln\left(\frac{K-N}{N}\right) = a - rt \tag{5.11}$$

using whatever spread-sheet or statistical package is available

4. Try out several values of K until you find the one that gives you the best fit (the one which gives an R^2 close to 1.0). Since there is no sampling error you should be able to find an $R^2 = .9999$.
5. Determine the intrinsic rate of natural increase (r) and the initial population size (N_0, the value of N at $t = 0$). Check your results by calculating

$$N_t = \frac{K}{\left(1 + e^{a-rt}\right)} \qquad \text{for } t = 1, 2 \ldots 6$$

6. Given the values you have calculated for r and N_0, plot what the growth would have been had the population density increased exponentially without environmental resistance. Plot this on the same graph that you drew the S-shaped logistic curve. Also, plot the natural log of density versus days for this curve and draw a regression line. Calculate the time (in days) that it takes this population to double in size.

7. Explore the chaotic behavior of the discrete version of the logistic ($N_{t+1} = N_t \exp [r(1 - N/K)]$) as in May (1974, 1976b). Use the same values of N_0 and K used in the exercises given above. Enter the above equation into a spread-sheet and calculate N_{t+1} for 100 or more successive generations. Vary r to determine the values at which you see the onset of chaos and of cycles every 2, 4, and 8 generations.

6

LIFE TABLES FOR SPECIES WITH OVERLAPPING GENERATIONS

Life tables are fundamental requisites to the analysis of population processes and to understanding population dynamics. A life table is a listing of the number or densities of individuals in a population surviving to specific ages or stages in the life cycle. Life tables were first developed for human populations. They are the actuarial tables used by insurance companies to calculate life insurance rates based on the probability that an individual will live to a particular age. Life tables were introduced to ecology by Deevey (1947) in a study of Dall sheep *Ovis dalli* in Alaska. Life table data are used to determine the relative importance of specific mortality factors in tests for density dependence or key factor analysis. We will address these topics in chapter 8.

A life table is a tabular form of a survivorship curve in which the number surviving to each age category in a population is plotted on a log scale against age (Pearl 1928) (Fig. 6.1).

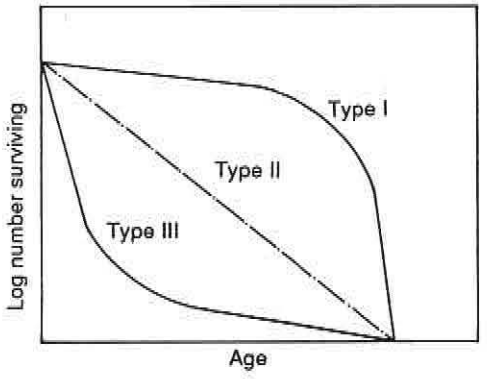


Fig. 6.1 Number surviving (on a log scale) to successive ages for species in which mortality rates increase (Type I), decrease (Type III), or remain constant (Type II) with age.

Different species exhibit different characteristically shaped survivorship curves. In a Type I curve, the rate of mortality increases at old age. Humans have a survivorship curve that is close to Type I. A Type II curve is linear (provided number surviving is plotted on a log scale), representing an exponential decline in numbers. In Type II, the rate of mortality is constant

through life. Some bird species exhibit Type II survivorship. In Type III survivorship, the greatest mortality rates occur in the youngest age category. Most insects and other invertebrates exhibit a Type III survivorship. However, many species exhibit a combination of idealized patterns indicated above. Humans, for instance experience relatively high rates of mortality in the first year of life, a trend which is superimposed on the basic Type I pattern (Fig. 6.2):

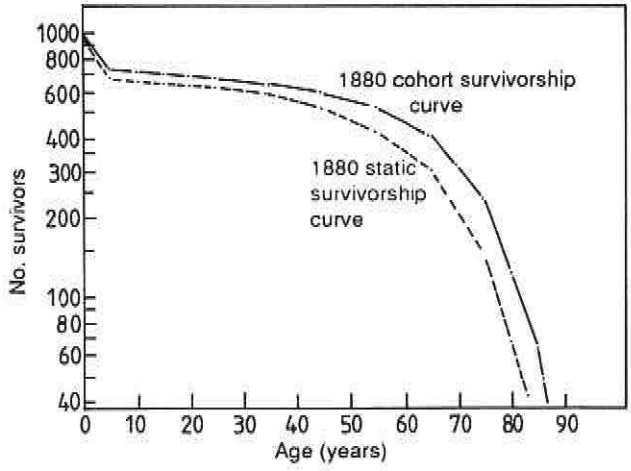


Fig. 6.2 Survivorship curve with a complex shape characteristic of humans, indicating the difference between cohort and time specific survivorship (redrawn with permission from Krebs 1978).

There are two types of life tables. A **horizontal or cohort life table** tabulates the survival of a cohort of individuals (all born at the same time) as they age. Horizontal life tables are principally used for insect species with discrete generations. **Vertical life tables** (also called **static**, or **time specific life tables**) provide an instantaneous picture of the age structure of a population, appropriate for populations with overlapping generations. Such an age structure reflects the mortality that occurs at each age and, provided the intrinsic rate of natural increase (r) is close to zero (Caughley 1977), the age structure approximates the probability of survival of cohorts, as illustrated in Fig. 6.2. Such life tables also typically list the age-specific fecundity in addition to survival.

We shall illustrate the concept of a life table with a hypothetical example (Table 6.1). First, we define some symbols: Let x be an age class, and l_x be the proportion of those individuals that enter the first age class ($x = 0$) that survive to successive age classes. In other words, l_x is calculated by dividing the number or density of individuals surviving at the beginning of each age class "x", by the number entering the first age class. Let m_x be the average number of offspring produced by individuals during age interval x. Usually only females are tabulated in life tables.

Table 6.1 Hypothetical life table

Age	Proportion surviving	No. born per individual	Expected offspring	
x	l_x	m_x	$l_x m_x$	$x l_x m_x$
0	1.000	0	0	
1	.700	0	0	
2	.500	0	0	
3	.300	10	3	9
4	.100	20	2	8
5	.050	40	2	10
6	.020	10	0.2	1.2
7	.010	0	0	
		R_0 = Reproductive rate=	7.2	28.2

The proportion surviving (l_x) is computed by dividing the number present at the beginning of each age category (x) by the number in the first age category. The net reproductive rate, R_0, is given by $\sum l_x m_x$ and is the net rate of increase per generation. For a population that remains unchanged, $R_0 = 1.0$. For a growing population $R_0 > 1.0$ and for a declining population, $R_0 < 1.0$. In the life table given above, $R_0 = 7.2$. The generation time (T_c), the mean time between parents and offspring, is calculated:

$$T_c = \frac{\sum(xl_x m_x)}{\sum(l_x m_x)} = \frac{\sum(xl_x m_x)}{R_o} \tag{6.1}$$

which for our example is: (28.2/7.2) = 3.92.

We can use this information to calculate r, the intrinsic rate of natural increase for a population. An approximate calculation is given by:

$$N_{T_c} = N_o \cdot e^{rt} \tag{6.2}$$

and since

$$\frac{N_{T_c}}{N_o} = R_o \tag{6.3}$$

then:

$$R_o = e^{rT_c} \tag{6.4}$$

which rearranges to:

$$r = \frac{\ln(R_0)}{T_c} \tag{6.5}$$

In our example, $r = 0.504$.

This estimate is only approximate. According to May (1976a), these calculations represent a good approximation to r when R_0 is close to 1.0 (a stationary population), or if the variance of the $l_x m_x$ distribution is small, a condition that occurs when reproduction is concentrated in a narrow age range.

A more precise estimate of r may be obtained from (Lotka 1907, Birch 1948):

$$\sum\left(e^{-rx} l_x m_x\right) = 1.0 \tag{6.6}$$

This equation cannot be solved for r. Instead, the correct value may be obtained by successive approximation until a value for r is found, such that the left hand side of the equation is sufficiently close to 1.0. Birch (1948) suggested a graphical method and linear interpolation; modern computers make successive approximation a trivial task. Accordingly, we arrive at a value for $r = .5304$ such that , $\sum(e^{-rx} l_x m_x) = 1.0$ as illustrated below (Table 6.2).

Table 6.2 An expanded hypothetical life table (as in Table 6.1) showing calculation of r

Age = x	l_x	m_x	$l_x m_x$	$xl_x m_x$	e^{-rx}	$l_x m_x e^{-rx}$
0	1.00	0	0	0	1	0
1	.70	0	0	0	.588	0
2	.50	0	0	0	.346	0
3	.30	10	3	9	.204	.611
4	.10	20	2	8	.120	.240
5	.05	40	2	10	.070	.141
6	.02	10	0.2	1.2	.041	.008
7	.01	0	0	0	.024	0
Total			$R_0 = 7.2$	28.2		1.00

Frequently, we want to know how fast such a population is increasing in a given time period. This can be calculated from the finite rate of increase which expresses the relative rate of growth (N_{t+1}/N_t) per unit time.

$$\frac{N_{t+1}}{N_t} = e^r \tag{6.7}$$

Similarly, we can calculate the population doubling time T_d:

$$\frac{N_{T_d}}{N_0} = e^{rT_d} = 2 \tag{6.8}$$

Solving for T_d yields:

$$T_d = \frac{\ln(2)}{r} = \frac{0.693}{r} \tag{6.9}$$

As indicated in the previous chapter, Lotka (1925) showed that a population growing under constant conditions will soon attain a stable age structure. If r is known, we can predict the proportion of individuals in each age class:

$$C_x = \frac{e^{-rx}l_x}{\sum\left(e^{-ri}l_i\right)} \tag{6.10}$$

where C_x is the proportion in age class x, and i goes from age class 0 to the maximum age class. Alternatively, we may express it as a fraction of those in the youngest age class: C_x/C_0:

$$\frac{C_x}{C_0} = e^{-rx}l_x \tag{6.11}$$

The following example illustrates the calculation of these life table statistics for the cassava green mite, *Mononychellus tanajoa* (Bondar), reared at different temperatures in a growth chamber by Yaninek et al. (1989a). This mite was introduced to Uganda in 1971. It has since spread across the cassava growing region of Africa and has become a major pest (Yaninek et al. 1989b).

Table 6.3 Net reproduction rate (R_0), intrinsic rate of increase (r), doubling time (T_d), and generation time (T_c) of *M. tanajoa* on leaf disks in calendar days at five temperatures (from Yaninek et al., 1989a with permission from the Entomological Society of America)

Temp. °C	R_0	r	T_d	T_c
20	13.9	0.0960	7.2	28.1
24	44.4	0.1800	3.9	22.2
27	43.2	0.2450	2.8	16.3
31	30.5	0.2810	2.5	13.0
34	3.2	0.1190	5.8	9.9

For another example, we turn to tsetse fly, *Glossina* spp., for which several static life tables have been published, based on capturing wild adult females and determining their age by ovarian dissection. An example is the life table published by Ryan (1981, Table 6.4).

The calculation of r from such a life table required some assumptions about mortality among immature stages. The value of r (0.000522) thus obtained is very close to 0, suggesting that tsetse has an extremely low rate of growth compared to other insects. However, Caughley (1977) and Van Sickle (1988) point out that calculation of r based on the age structure of the population at a moment in time (a static or vertical life table), is a flawed procedure. The age structure of the population will be equivalent to the survival of individual cohorts followed

Table 6.4 Life table for *G. morsitans centralis* Machado, collected within the Kabulwebulwe Resettlement Area, Zambia (15° S, 27° E), in June and July 1980, using the recorded mean monthly temperature of 18°C (from Ryan 1981 with permission from the International Institute of Entomology)

Ovarian category	Mean age (days)	No. caught N	l_x	m_x	$l_x m_x$	$x l_x m_x$
P	32.2	[83]	1	0	0	0
0	69	65	0.700	0	0	0
1	80.1	41	0.659	0.500	0.33	26.40
2	98	29	0.597	0.457	0.27	26.74
3	120.5	23	0.493	0.400	0.20	23.76
4	143	31	0.388	0.344	0.13	19.09
5	165	26	0.283	0.270	0.08	12.65
6	188	7	0.179	0.200	0.04	6.73
7	210.5	10	0.074	0.148	0.01	2.31
		$\Sigma = 232$			$R_0 = 1.06$	$\Sigma = 117.78$

through time only if $r = 0$. If this assumption is not correct, the true value of r can deviate widely from the calculated value. The only way to properly calculate r is to document the survival of cohorts through time. Unfortunately, this is usually a very difficult task in field populations.

Despite this objection, there is no doubt that tsetse flies have very low reproductive rates compared to most insects. As such, tsetse is a classic example of a ***K*-selected** organism (MacArthur and Wilson 1967), characterized by low reproductive rates and large amounts of parental investment in the rearing of young. This contrasts with ***r*-selected** species such as the cassava green mite (Table 6.3), which have high reproductive rates and experience high levels of juvenile mortality. Such traits are typical of animals that colonize new habitats.

EXERCISES

1. Given the following life table, calculate R_0, T_c, and approximate value for r. Then calculate the exact r using the Lotka equation ($\Sigma(e^{-rx} l_x m_x)=1.0$). Calculate the doubling time and the fraction of individuals expected in each age class.

Age (x) in days	Proportion surviving (l_x)	Age specific fecundity (m_x)
0	1.00	0
1	.90	0
2	.80	2.0
3	.65	3.2
4	.50	4.7
5	.42	3.9
6	.31	2.7
7	.20	2.0
8	.10	1.5
9	.05	1.0
10	.01	0

   Calculation of exact r can be accomplished by plotting your various estimates of r versus $\Sigma(e^{-rx} l_x m_x)$ and connecting the points (Birch 1948). The correct value for r is where the line intersects $\Sigma(e^{-rx} l_x m_x) = 1.0$. This procedure can be more easily accomplished by entering the formula into a spread-sheet program and adjusting the value of r until $\Sigma(e^{-rx} l_x m_x)=1.0$.

2. Using this value of r and $N_0=5$, plot the exponential growth of this population for 10 days. With the same r and N_0, plot a logistic curve (eq. 5.10) for this population when $K = 1500$.

7

CONSTRUCTION OF LIFE TABLES FOR INSECTS WITH DISCRETE GENERATIONS

In the previous chapter, we examined the methods available for constructing life tables for animals with overlapping generations. In this chapter, we examine methods available for animals with discrete generations. By definition, these are horizontal life tables, since they involve documenting the survival of discrete cohorts. These methods were pioneered by Morris and Miller (1954), who applied them to population studies of spruce budworm. In a subsequent chapter, we will consider the methods that have been developed for analyzing life table data. It is important to realize that construction of a life table does not imply a particular type of analysis. Furthermore, a single life table will not reveal anything about the factors responsible for population change, because a life table merely expresses the schedule of births and deaths in a single generation. Only by analysis of how life tables change between successive generations of the same population, or differ between populations under different experimental protocols, will the processes of population change be dissected. We will address such analyses in Chapter 9.

In many population studies, data are collected on the proportion of animals dying from a particular agent of interest. In biological control programs, for instance, we typically want to assess the effectiveness of a particular predator or parasitoid. Or, we might want to study the impact of some cultural treatment on the mortality caused by natural enemies. Such studies are frequently conducted without collecting data sufficient to build complete life tables. Nevertheless, guidelines are needed as to how to quantify mortality. The methods are the same as those required to construct life tables and are elaborated in this chapter.

In Table 7.1, a life table is presented for a hypothetical insect with four life stages: egg, larva, pupa and adult. Survival to each successive life stage (l_x) has the same meaning as that presented in the previous chapter in life tables for species with overlapping generations, except that, by convention, l_x is frequently standardized to 1000 instead of 1.0, so that it expresses numbers surviving out of an initial cohort of 1000. In other words, $l_x = 1000 * a_x/a_0$, where a_x is the number present at the beginning of each stage. The standardized numbers dying (d_x) in each stage, is $l_{x+1} - l_x$. In other studies, l_x is used synonymously with a_x (see Table 7.2), and d_x is the number dying in terms of actual population counts or densities.

When measuring changes in density between generations in the same population, it is important to keep track of the actual, rather than the standardized, densities. Ultimately, it doesn't matter which of these conventions is followed, as long as readers are clear about the meaning of the symbol in any particular treatment.

The proportion or percentage dying in each stage can be calculated in several ways. The **stage specific** or **apparent mortality** (q_x), is the proportion of those who entered the stage that died during the same stage (d_x/l_x). Frequently, it is more convenient to perform calculations with the stage specific survival ($1-q_x$). **Real mortality** is the proportion or percentage of those that began the generation that died during the stage (d_x/l_0). The total mortality in the stage is the sum of the real mortalities in each stage.

Table 7.1 Hypothetical life table

Stage x	No. surviv. l_x	No. dying d_x	Appar. mort. $q_x = d_x/l_x$	Appar. survival $1-q_x$	Real mort. d_x/l_0	k
Eggs	1000	600	.60	.40	.60	.40
Larva	400	200	.50	.50	.20	.30
Pupa	200	180	.90	.10	.18	1.00
Adult	20					
Total		980			.98	1.7

In Table 7.1, the total mortality is 0.98, which equals the sum of the real mortalities in each stage (0.6 + 0.2 + 0.18). The total proportion surviving the generation (0.02), is the product of the stage-specific survival in each stage (0.4 * 0.5 * 0.1). **Indispensable mortality** is another measure of mortality (see Southwood 1978), that has been proposed, but is seldom calculated and is of limited utility (Bellows et al. 1992). Adding a given amount of stage-specific mortality, say 30%, to a population will cause a corresponding 30 % decline in the total generational survival or nut reproductive rate R_0, regardless of which stage it attacks. This is true, provided the level of mortality caused by other sources of mortality remains unaffected.

Apparent or stage-specific mortality can be expressed in logarithmic form as a ***k*-value** or killing power (Haldane 1949, Varley and Gradwell 1960) which is defined as:

$$k = \log_{10}(N_x/N_{x+1}) = \log_{10}(N_x) - \log_{10}(N_{x+1}) \qquad (7.1)$$

where N_x is the number entering stage x on which the mortality acts, and N_{x+1} is the number entering the following stage. In our example, the k-value for the larval stage is:

$$\log_{10}(400) - \log_{10}(200) = .30$$

We should note here that we are referring to the total mortality occurring in the stage. Later, we will demonstrate how to compute k-values for individual causes of mortality occurring within the stage. Since the stage-specific survival ($1-q_x$) is equal to N_{x+1}/N_x; then

$$k = \log_{10}(1/(1-q_x)) = -1.0 * \log_{10}(1-q_x) \qquad (7.2)$$

A stage specific mortality of 90% results in a k-value of 1.0; 99% mortality corresponds to $k = 2.0$, 99.9% corresponds to $k = 3.0$, and so forth. There are several advantages to using k-values.

In studies where mortality is related to some other factor such as population density (see Chapter 10), by means of regression analysis, k-values are more likely to result in a linear

relationship than percent mortalities, which, by definition, are constrained between 0 and 100%. Furthermore, the total generational mortality, expressed as (K) is easily computed; it is the sum of the individual k-values for each life stage:

$$K = k_1 + k_2 + k_3 + \ldots + k_n \tag{7.3}$$

This is true because the total generational survival is the product of the stage-specific survivals.

Up to this point, we have considered k-values in terms of mortality. However, any complete analysis of population change must take account of changes in fecundity as well. Price (1990) argued that the life table methods pioneered by Morris and others have placed too much emphasis on mortality and have caused population ecologists to ignore important factors influencing the oviposition behaviour of adults, and hence, fecundity. Many life table studies, including those of Morris, treated fecundity as a constant. Varley and Gradwell (1968), on the other hand, incorporated change in fecundity by calculating it as a k-value in their life table studies. They did this by expressing the observed fecundity (f_{obs}) as a proportion of the maximum possible fecundity (f_{max}) under the specific conditions of the population being studied. The k-value for fecundity was thus:

$$k_f = -\log_{10}(f_{obs}/f_{max}) \tag{7.4}$$

and total K was not mortality, but the total change in numbers between generations:

$$K = \log_{10}(N_t/N_{t+1}) \tag{7.5}$$

where N_t and N_{t+1} are the numbers entering the same stage (usually the egg stage) in successive generations. Since the net reproductive rate R_0 is N_{t+1}/N_t,

$$K = -\log_{10}(R_0) \tag{7.6}$$

Sex ratio is another factor that must be incorporated into life table studies. This is accomplished in several ways. The best is to express the densities in the life table in terms of females only. This is straightforward when the sex ratio is 50/50 throughout the life cycle of the animal. For many insects, the sex ratio may change during the course of the life cycles due to differences between sexes in mortality, and, furthermore, the sex of immature stages may be impossible to determine. In these cases, it is still better to express densities in terms of females, but an alternative is to calculate a k-value (k_{sr}) for sex ratio:

$$k_{sr} = -\log_{10}(N_{fx}/N_x) \tag{7.7}$$

where N_{fx} is the number of females in stage x.

Estimating numbers entering a stage

A common misconception in population ecology is that estimating densities for life table studies, or quantifying mortality from various causes, is simply a matter of obtaining an unbiased estimate of density at periodic intervals and determining the proportion of animals that die from particular causes over those intervals. Unfortunately, for a life table or for an estimate of stage-specific mortality, we need to estimate the number *recruited* to the host stage over the generation, which may be quite different from the number present at any moment. The problem arises because for most species, populations consist of a mixture of different life stages (Fig. 7.1), caused by the fact that oviposition and egg hatch are distributed over an extended period of time. Furthermore, individuals that hatch at the same time may develop at different rates due to inherent genetic or phenotypic variation or to the differences in microclimate that they experience.

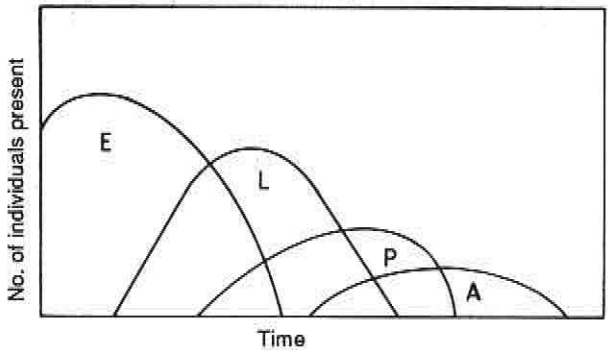


Fig. 7.1 Temporal distribution of life stages typical of many insects, E = eggs, L = larvae, P = pupae, A = adults.

When stages overlap, as in Fig. 7.1, no measure of density taken at any moment will constitute a valid estimate of the numbers that *enter* any particular stage. Several techniques have been developed that allow one to estimate the numbers recruited to a particular life stage, based on a series of samples that estimate the numbers present in particular life stages at periodic sample intervals. Such data are known as stage frequency data. We shall review some of these techniques here.

The graphical method of Southwood and Jepson (1962) has been used quite widely. A series of sample estimates of density of the particular life stage of interest is plotted against physiological time (Fig. 7.2). Physiological time is usually computed in terms of day-degrees (see Chapter 10), and attempts to correct for effects on developmental time of differences in temperature experienced by different populations.

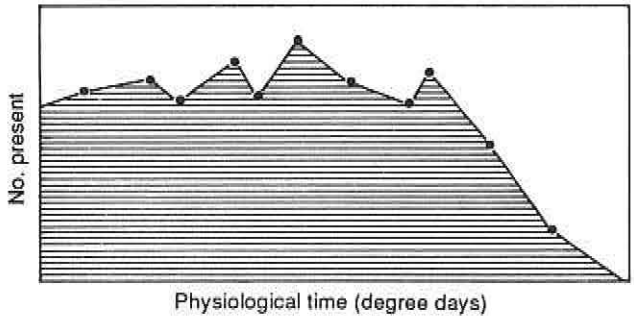


Fig. 7.2 Southwood and Jepson (1962) graphical technique for estimating recruitment to a stage.

The area under the curve (Fig. 7.2) is computed by summing the areas below each line segment drawn between each successive density estimate. The result is an estimate of the number of degree days consumed by the entire population. This number is divided by the number of degree days required by one individual to complete development (assuming that such data have been obtained in laboratory studies), to yield an estimate of the number of individuals that passed through the stage. A key assumption of the method is that all mortality occurs near the end of the stage; departures from this will cause an underestimate in numbers recruited. Sawyer and Haynes (1984) offer techniques for correcting some of these errors. Bellows et al. (1989) explored the sources of bias in the Southwood and Jepson technique and concluded that it should be avoided, or used with extreme caution, in most cases.

Richards and Waloff (1954) proposed a method based on the assumption of a constant rate of mortality during a life stage. For each sample occasion, the numbers present in the stage of interest, and all subsequent stages, are plotted on a log scale against time (Fig. 7.3). As animals are recruited into the stage, the numbers increase with each successive sample occasion. Once recruitment to the stage has been completed, mortality causes the numbers

present to decline with time. Advancement to succeeding stages does not influence the decline in numbers present, because all subsequent stages are included in the density counts. Thus, the decline in numbers represents the effect of mortality only. The slope of a line drawn through the declining portion of the curve (Fig. 7.3) is log (ø) where ø is the daily rate of survival:

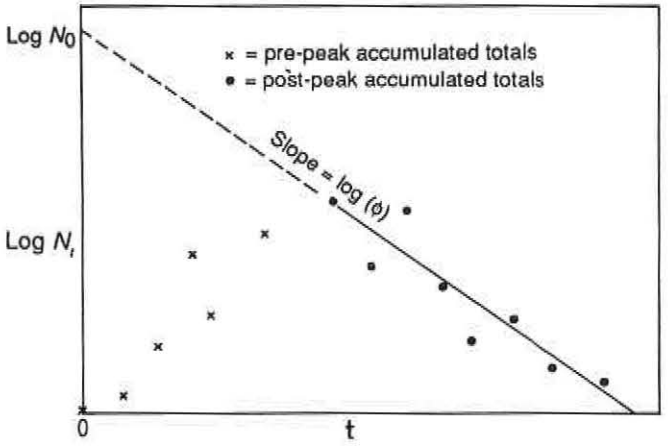


Fig. 7.3 Richards and Waloff's (1954) technique of estimating number entering a stage (from Southwood 1978 with permission from the Natural Resources Institute).

$$N_t = N_0 \, ø^t \tag{7.8}$$

or

$$\log(N_t) = \log(N_0) + \log(ø) * t \tag{7.9}$$

N_0 is the numbers present at start of recruitment to the stage, and $\log(N_0)$ is the intercept, which can be obtained by linear regression. There are several obvious constraints that limit the application of this technique. The first of these is that the daily rate of survival (ø) may not be constant but may vary over the interval. This may be especially true when most of the population has progressed to successive stages. Knowledge about when recruitment has been completed may be imperfect, so it may not be clear which points to include near the peak of the density curve. Differences in decisions on this point will make a very big difference in the estimate of N_0 because it is plotted on a log scale. Applications of this technique in population ecology have not been frequent.

Van Driesche and Bellows (1988) offered a straightforward solution to the problem of estimating recruitment of specific life stages. They proposed that recruitment be measured directly. For example, recruitment of herbivorous insects to the egg stage could be made by measuring the daily rate of egg deposition on "sentinel plants", which are sampled repeatedly for the arrival (recruitment) of new individuals. After each sampling, eggs would then be removed, so that counts of eggs made on subsequent visits would reflect only eggs deposited during the interval since the previous sample occasion. The number or density per plant of individuals entering the egg stage is simply the total number counted on each recruitment interval over the oviposition period. This method has a lot to recommend it since it is a direct measure of what is needed for a life table, and avoids the sources of bias discussed above for methods based on stage frequency analysis. On the other hand, it is not always possible to use this method. Mobile life stages, for example, may move onto sentinel plants from surrounding vegetation so that recruitment via dispersal cannot be separated from graduation from earlier life stages.

Quantifying mortality

Many studies of insect populations are focused on measuring the impact of particular agents of mortality, including predators, parasitoids, or disease agents. Only a small fraction

of such studies gather sufficient information to construct a complete or even a partial life table for the study insect. For example, we might be interested in measuring the impact of some crop management practice on the levels of parasitism occurring in a particular pest species. The usual approach would be to collect the host insect on a series of sample dates from the different treatment populations and to determine, either by rearing or dissection, what fraction of hosts was parasitized. However, the fraction of hosts parasitized is affected by the interaction of host and parasitoid phenology (Van Driesche 1983). It is another aspect of the same problem that arises in estimating the numbers entering a stage discussed in the previous section. The problem is best illustrated with a simple hypothetical example from Van Driesche (1983), in which 100 hosts were attacked by 50 parasitoids (i.e., apparent or stage-specific mortality was 0.5) (Fig. 7.4).

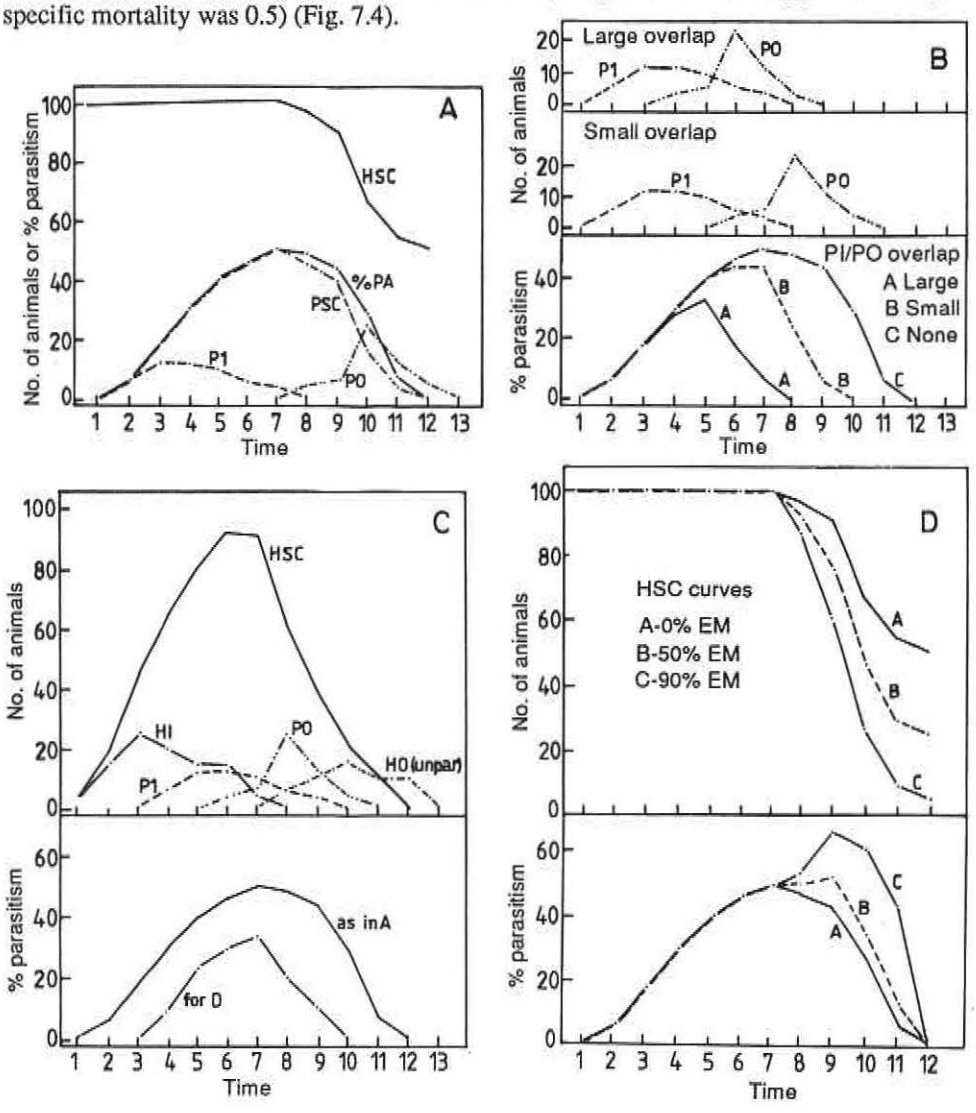


Fig. 7.4 Effect of host and parasitoid phenology on observed levels of parasitism in samples extracted from a hypothetical population (reproduced from Van Driesche 1983 with permission from the Entomological Society of America). See text for further explanation.

In the first and simplest case (Fig. 7.4A), host recruitment is complete before parasitoid recruitment commences, as indicated by the curve labeled *HSC* (host standing crop), which is the number of hosts present. As oviposition (*PI* = parasitoid input) proceeds, the fraction of hosts parasitized, *% PA*, as measured from a sample collected at a given instant, increases up to the 50% level, equivalent to the stage-specific mortality for that stage. The percentage

of hosts in a sample that is parasitized, *%PA*, is given by: (*PSC/HSC*) * 100, where *PSC*, the parasitoid standing crop, is the number of hosts present bearing immature parasitoids. However, if the first parasitoids to attack complete development inside the host and emerge (*PO* = parasitoid output) prior to the completion of parasitoid oviposition (Fig. 7.4B), then at no time will all the parasitoids be present as immatures inside the host population, and levels of percent parasitism in any sample will never attain the value of 50%, the actual stage-specific mortality. Further complications arise when the recruitment of hosts to the susceptible stage (*HI*) or graduation of hosts to a subsequent stage (*HO*) overlaps with oviposition (*PI*) or emergence (*PO*) of parasitoids (Fig. 7.4C). In all these cases, the proportion of sample hosts parasitized vastly underestimates the stage-specific mortality. In some systems, parasitism may delay host development, with the result that healthy, unparasitized hosts graduate to the next stage, which may not be sampled, whereas the parasitized hosts remain behind in the susceptible stage (Fig. 7.4D). In this situation, the fraction of sample hosts parasitized rises above the level (50%) represented by the stage-specific mortality. In the extreme case, it might be that 100% of the hosts remaining in the population at the end of the host stage might be parasitized, even though impact of the parasitoid on the host population (50% mortality) was the same for all of these examples. Thus, two or more populations may have exactly the same levels of parasitism, yet differ dramatically in sample percent parasitism, caused solely by differences in host and parasitoid phenology.

Relatively few studies have considered this problem (Van Driesche 1983), and there is great variation in how percent parasitism data is presented or calculated. Many studies record parasitism based on a single sample taken from the host population. Such measures are meaningful only if they occur during the window of time between parasitoid oviposition (*PI*) and emergence (*PO*) (Fig. 7.4A), which does not overlap with host recruitment. Other studies may present the average percent parasitism among samples collected at various times or may pool all collections and present the total percent parasitism. Such values are likely to have little meaning and to depart widely from the true level of parasitism represented by the stage-specific or apparent mortality.

There are several possible solutions to this problem (discussed in detail in Van Driesche et al. 1991). The solutions involve applying the same methods listed above for estimating the numbers entering a stage. In this case, they are applied separately to both the host and immature parasitoid populations. The stage-specific or apparent mortality in any host stage is given by: $\Sigma P_i/\Sigma H_i$, where P_i and H_i are the respective number of immature parasitoids and hosts recruited during the i^{th} interval. Any combination of the three methods (Southwood and Jepson 1962, Richards and Waloff 1954, or Van Driesche and Bellows 1988) could be applied. Of these, the recruitment method of Van Driesche and Bellows (1988) is the most direct and bias-free, although, as indicated above, it may not always be possible.

Van Driesche and Bellows (1988) suggest two possible techniques for directly measuring recruitment of parasitoids. One of these involves use of trap hosts, which are uninfected hosts that are placed in the field for specific intervals of time. Trap hosts may behave differently from naturally occurring hosts, resulting in altered attack rates by parasitoids. Examples of this may be found in Gould et al. (1992). Another method involves use of a "short marker stage". Here, a developmental stage of sufficiently short duration (such as the parasitoid egg stage) is periodically counted. This count gives an approximate estimate of the number recruited during the previous short interval.

Contemporaneous mortality

The life table methods presented above focus on quantifying total mortality within a stage. As I have indicated, however, we are usually interested in partitioning that mortality into its components and expressing mortality from different sources acting contemporaneously within a stage. In most life tables, this is presented in the d_x column; total d_x is partitioned into components, the number that die from parasitoid A, parasitoid B, etc. An example of this is given (Table 7.2) from a study of the legume pod borer, *Maruca testulalis* Geyer on cowpeas (Okeyo-Owuor and Oloo 1991).

Table 7.2 Life table for the generation of *M. testulalis* on the cowpea crop at site 1 during 1984 (late crop) long rain season (May–August) in Kenya (with permission from Okeyo-Owuor and Oloo 1991 and ICIPE Science Press)

x	l_x	d_xF	d_x	$100r_x$	$100q_x$	Marginal rate	k
Egg	(232019)	Total (Disappear.)*	(59569)	(25.7)	(25.7)	25.7	.129
Larva I–II	172450	Disease	15311	6.6	8.9	8.9	.040
		Disappear.	23108	10.0	13.4	14.7	.069
		Total	38419	16.6	22.3	22.3	.109
III	134031	Disease	24549	10.6	18.3	18.3	.088
		Disappear.	11527	5.0	8.6	10.5	.048
		Total	36076	15.6	26.9	26.9	.136
IV	97955	Disease	8718	3.8	8.9	8.9	.041
		Disappear.	9763	4.2	10.0	10.9	.050
		Total	18481	8.0	18.9	18.9	.091
V	79474	Disease	42942	18.5	54.0	54.0	.338
		Parasitism	0	0.0	0.0	0	0
		Disappear.	30070	13.0	82.3	37.8	.752
		Total	73012	31.5	91.8	91.8	1.090
Pupa	6462	Disease	908	0.4	14.1	14.1	.066
		Parasitism	298	0.1	4.6	5.3	.024
		Disappear.	1519	0.7	23.5	28.9	.148
		Total	2725	1.2	42.2	42.2	.238
Adult	3737						
		Grand total	228282	98.6	98.4		1.795

x = age interval.
l_x = Number alive during x.
d_xF = Factors responsible for mortality (d_x).
d_x = Number dying during x.
q_x = Stage-specific (apparent) mortality.
r_x = Real mortality

Figures in parentheses are estimated.
*Disappearance includes losses due to predation, emigration and all other unknown causes.

As in virtually all life tables constructed for field populations, there is a residual mortality, which is unknown mortality in the form of changes in density not accounted for by the measured mortality from known agents, usually parasitism or disease. In Table 7.2 this is listed as disappearance which includes losses due to predators.

The apparent mortality (q_x) is typically calculated for each agent in the same way ($q_x = d_x/l_x$) as for total losses to the stage. However, if this is done, the q_x values sum to the total q_x for the stage, but the product of $(1 - q_x)$ for each factor is not equal to $1 - q_x$ for the stage. For example, in Table 7.2 the apparent mortality of first stage larvae: 0.089 + 0.134 = 22.3, but $(1 - 0.089)(1 - 0.134)$ does not equal $(1 - 0.223)$. As a consequence, k-values for each factor cannot be calculated as $k = -\log_{10}(1-q_x)$.

The problem is best illustrated by the following hypothetical example. Suppose that there are two parasitoids, A and B, that together comprise the total mortality that occurs during a particular stage or age interval. Suppose further that the two agents attack hosts indiscriminately and that in a given stage, 80% are attacked by A and 50% by B. As indicated by Royama (1981), this process can be represented by a Venn diagram (Fig. 7.5), and the proportions attacked (0.8 and 0.5, Fig. 7.5A) constitute the marginal probabilities of dying or marginal attack rates (m_A and m_B respectively).

Varley et al. (1973) offer a straightforward solution to this problem, used in Table 7.2, that works in most cases and yields the marginal rates (although they did not use this term). Their technique was based on dissection of hosts to determine the presence of parasitoids or disease

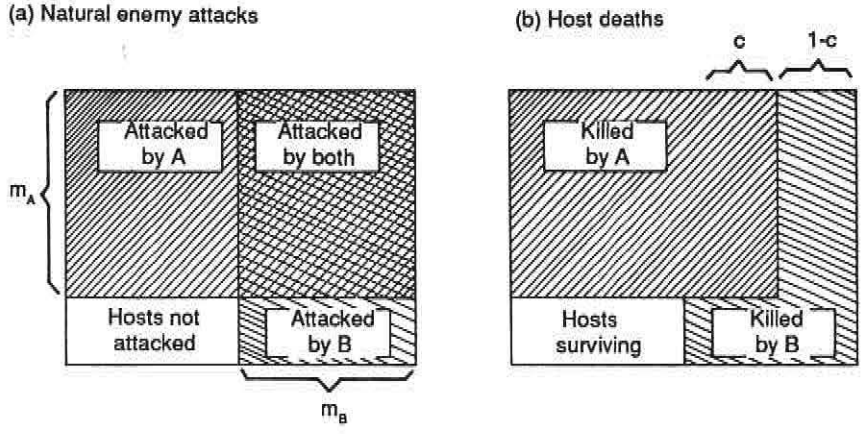


Fig. 7.5 A Venn diagram after Royama (1981) indicating (A) the proportion of hosts attacked, and (B) the proportion from which adult parasitoids emerge for two nondiscriminating hypothetical parasitoids (reproduced from Elkinton et al. 1992 with permission from the Society of Population Ecology).

agents. Each agent was assumed to act sequentially. In our hypothetical example given above, 100 hosts would be dissected and on average, 80% would contain A, of which 40 would have A alone and 40 would have both A and B; 50% would contain B, of which 10 would contain B alone, and 40 both A and B. If A goes first it is attributed with 80%, B is assigned to 10 of the remaining 20, or 50%. The same answer is obtained when B goes first. (See rules of constructing life tables; Varley et al. 1973, p. 100). In Table 7.2, the disease was assumed to attack first and kill 8.9% of the I and II stage larvae. The *k*-value for disease in this stage is $-\log_{10}(1-0.089)= 0.040$. The disappearance was assumed to occur next and killed 23108 of the remaining larvae (172450 – 15311), or 14.7%, of the individuals that survived the disease attack. This value is the marginal rate, and $-\log_{10}(1-0.147) = 0.069$ is the *k*-value for disappearance in this stage.

In many studies of insect population dynamics, however, mortality from parasitism or disease is assessed by rearing field collected hosts rather than dissecting hosts to score numbers attacked; such an approach often is the only possible way to score mortality. In the example given above, there are 40 hosts attacked by both A and B. If the system were such that only A or B, but not both, could successfully emerge from the host, then some fraction c of these multiparasitized hosts might yield A, and $1-c$ would yield B. If half the multiparasitized hosts yielded A and the other half B, then the proportion of hosts killed by A (d_A) would be 0.60 and proportion killed by B (d_B) would be 0.30. Although $0.60 + 0.30 = 0.90$, $(1-0.6)(1-0.9) \neq 0.10$. Instead, it is the marginal rates of survival (1– marginal rate of mortality) that multiply to the total survival: $1 - 0.8)(1 - 0.5) = 1 - 0.9 = 0.1$. If the data consists of proportion dying (d_A, d_B) obtained by rearing of field collected hosts, the marginal rates can be recovered from the emergence rates for the different agents, using the following equation from Buonaccorsi and Elkinton 1990, Elkinton et al. 1992:

$$m_A = \frac{b - \sqrt{b^2 - 4cd_A}}{2c} \tag{7.10}$$

$$m_B = \frac{d_B}{(1 - cm_A)} \tag{7.11}$$

where $b = c(d_A + d_B) + 1 - d_B$, and c is the proportion of hosts attacked by both A and B from which A emerges.

The value for c used in equation (7.10) and (7.11) could be determined experimentally by rearing hosts parasitized by both A and B, although in field populations the value may vary

depending on the temporal distribution of parasitoid attacks. For many hosts and parasitoids, the values for c will be unknown and some assumption concerning its value will be necessary. It might be reasonable to assume, for instance, that $c = 0.5$, so that half of the individuals attacked by both A and B yield A and the other half yield B. Another reasonable assumption might be that the first agent to attack is the winner. This would always be the case with two or more predators. In this situation the marginal attack rates are:

$$m_i = 1-(1-d)^{d_i/d} \tag{7.12}$$

where m_i and d_i are the respective marginal rates and death rates from the i^{th} cause and d is the death rate from all causes combined (Elkinton et al. 1992). A great advantage of this equation over (7.10, 7.11) is that it is easily extended to more than two contemporaneous agents. In the case of a contemporaneous predator and parasitoid the predator always "wins" in any host attacked by both. In this situation the marginal attack rate for predation is the same as the death rate and is observed directly, ($m_A = d_A$). The marginal attack rate for the parasitoid (B) is:

$$m_B = \frac{d_B}{1-m_A} \tag{7.13}$$

This formula applies if predation is scored by classifying the remains of the host after it has been eaten by the predator. If predation is equated with disappearance, as in Table 7.2, the k-value for predation is obtained by subtracting the k-value for disease and parasitism, combined from total k for the stage (based on observed change in density = $\log_{10}(l_x) - \log_{10}(l_{x+1})$. The only ambiguity in Table 7.2 occurs in pupal stages where the above marginal rate calculations (e.g. 7.10, 7.11, 7.12) must be applied to obtain marginal rates and k values separately for disease and parasitism, assuming that these values were obtained by rearing and not by dissection.

EXERCISES

To construct a life table for insects with discrete generations but overlapping stages, a method of estimating the numbers entering a stage is needed. In this exercise we will determine the number of armyworms entering the larval and pupal stages and measure the impact of a parasitoid wasp on the larval stage. The field has been visited every day and counts have been made of the number of armyworm larvae found per plant. Certain "sentinel" plants were visited every day and the number of newly emerged larvae were counted and removed. Trap hosts (unparasitized larvae reared in the laboratory) were placed out in the field each day. The trap hosts and naturally occurring hosts were brought back to the laboratory on subsequent sample dates and the proportion parasitized determined. Ecdysed armyworm pupae were those that successfully molted to the adult stage leaving behind a pupal skin. The following counts (all expressed in terms of individuals per plant) were obtained:

DAY	MEAN TEMP.	NO. LARVAE RECRUITED	NO. LARVAE PRESENT	NO. PUPAE PRESENT	ECDYSED PUPAE PRESENT
1	24	0.1	.1	0	0
2	23	1.9	1.7	0	0
3	28	7.0	8.5	0	0
4	26	8.2	14.4	0	0
5	23	6.0	17.6	0	0
6	22	4.6	12.3	0.7	0
7	20	3.9	10.5	1.3	0
8	23	1.2	7.3	2.2	0
9	28	0.1	3.4	4.1	0
10	29	0	0.2	3.1	0
11	27	0	0	2.0	0.6
12	23	0	0	1.1	0.8
13	18	0	0	0.6	1.2
14	17	0	0	0.1	1.5
15	22	0	0	0	1.5

DAY	PROPORTION TRAP HOSTS PARASITIZED	PROPORTION OF NATURAL LARVAE PARASITIZED
1	0.02	0.02
2	0.03	0.05
3	0.09	0.11
4	0.11	0.13
5	0.07	0.12
6	0.09	0.09
7	0.09	0.09
8	0.06	0.08
9	0.07	0.07
10	0.02	0.05

1. The Graphical Method (Based on Southwood & Jepson 1962):

Plot the counts of larvae and pupae versus day-degrees. Assume a threshold temperature of 10°. The formula is: Day-degrees = (Temperature – threshold temperature) * No. of days between samples (1.0). Compute this for each day and add up the degrees accumulated. Estimate the area for the larvae by counting the number of squares under the curve of the plot on graph paper. Make sure you convert the area to the proper units (individuals times day-degrees). Alternatively, write a spread-sheet formula that calculates and sums the areas of successive trapezoids on the graph. Calculate the number of larvae and pupae that entered the stage using 51.3 as the average day degrees required to complete larval development, and 44.0 as the average day-degrees for pupae.

2. Richard and Waloff's (1954) first method:

   Plot $\log_{10}$ density of larvae and pupae plus subsequent stages versus time. Draw the regression line using whatever statistical package or spread-sheet program you have available. Estimate the number that entered the stage by extrapolating back to the vertical axis.

3. Recruitment technique (Van Driesche & Bellows 1988):

   Calculate the total number recruited to the host stage and the total number recruited to the immature parasitoid stage. The latter is the product of the proportion of trap hosts parasitized and the number of larvae present in the population at a given moment in time. Calculate the total parasitism for the larval stage and compare to the peak and average proportion parasitized in the naturally occurring hosts. Why might these estimates differ?

4. Determine the total mortality that occurred in the larval stage and the fraction of it caused by the parasitoid. To do this, you will have to compare the relative numbers that entered the larval and pupal stages. Compare the three estimates of numbers entering the larval host stage. Explain why the estimates may differ.

8

REGULATION OF POPULATION DENSITY: THE ROLE OF DENSITY DEPENDENT VERSUS DENSITY INDEPENDENT FACTORS

A fundamental controversy has raged in the population dynamics literature for more than 50 years, as to whether or not the population densities of most species are held at equilibrium by forces that return the population to equilibrium density following a perturbation. The concept of **density dependence** is fundamental to this debate. A mortality factor is said to be density dependent if the proportion or percentage of the population killed by the factor varies systematically with density (Solomon 1949). A mortality factor is **density independent** when it kills the same proportion of the population regardless of density (Fig. 8.1). An increase in the proportion dying with increasing density is called **positive density dependence;** a decrease in the proportion dying with increasing

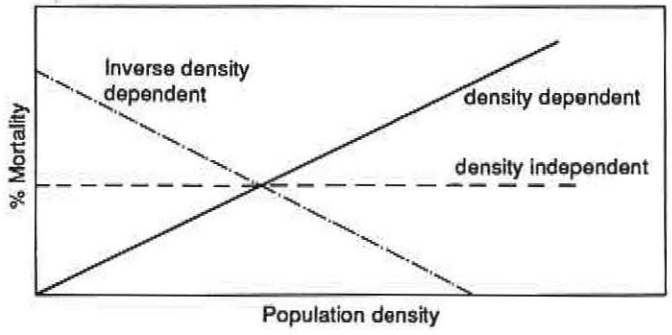


Fig. 8.1 Density dependent, density independent and inverse density dependent mortality.

density is termed **negative** or **inverse density dependence.**

Frequently, the term density dependence is used synonymously with positive density dependence. If population densities are at equilibrium, then by definition, the factors responsible for maintaining that population at equilibrium are density dependent. For example, suppose there is a species in which each female produces exactly 100 female eggs. For the population to remain stable, each female must replace herself once, which means that 99% of her offspring must die before reaching reproductive maturity. If only 98% die, the population will double in size. For

an equilibrium to exist, increases or decreases in density must trigger increases or decreases in the proportion dying, so that the population returns to the equilibrium level at which fecundity is exactly balanced by mortality.

It is important to emphasize that density dependence is expressed in terms of the proportion dying and not the number dying. As population density of a prey species increases, it is typical for predators to consume more of them. Despite the increase in numbers consumed, the proportion of the population consumed may actually decrease. Suppose, for example, the density of the prey may increase from 10 to 1000, and the number consumed increases from 5 to 50. Despite a tenfold increase in the number consumed the proportion consumed declines from 0.5 to 0.05. When the number dying is plotted against density (Fig. 8.2), density independence results whenever the number dying increases with density along a straight line drawn back to the origin. (i.e. $N_a = a + bN_t$ where $a = 0$).

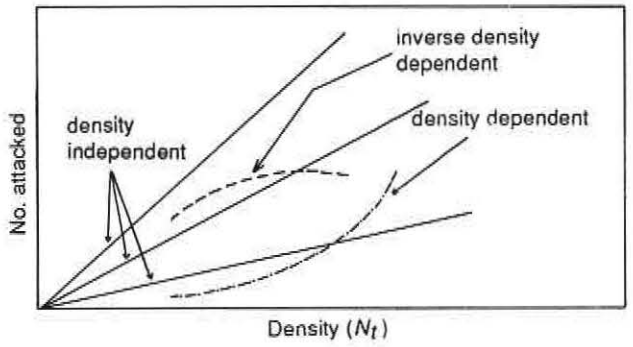


Fig. 8.2 Density dependence in terms of number killed.

The concept of density dependence is typically applied to mortality agents. However, the term can be equally applied to fecundity, or indeed, to any factor that increases or decreases its impact on the population as a function of density. For example, the fecundity of an insect species may decline at high density.

Factors that act in a positive, density-dependent manner may only do so over a short range of density. It is not uncommon for a predator to cause positive, density dependent mortality in populations of low prey density and inverse density dependent mortality at higher prey density. Figure 8.3, for example, depicts the impact of small mammal predators on gypsy moth as hypothesized by Campbell (1975).

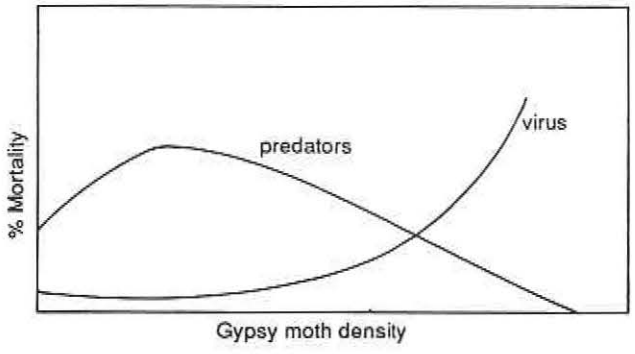


Fig. 8.3 Gypsy moth density and percent mortality caused by various agents. Redrawn from Campbell (1975).

At low gypsy moth density, the predators supposedly consume a larger fraction of the population as gypsy moth density increases. At high gypsy moth density, the proportion consumed declines. In this phase the population increases very rapidly and an outbreak ensues. Outbreak populations are subject to a different suite of density dependent factors, such as a virus disease and starvation, that only become major sources of mortality when

densities are high. These factors can maintain populations at a high density equilibrium, but more frequently, they cause the collapse of populations back down to a low density, endemic phase.

Southwood and Comins (1976) proposed a "synoptic model", very similar to the Campbell (1975) gypsy moth hypothesis, as a general feature of insects that occasionally go into an outbreak phase. The model is depicted by plotting R_0, the net reproductive rate, against density. The resulting figure (Fig. 8.4), is the mirror image of the Campbell model: higher mortality produces a "natural enemy ravine" at low density at which the population is maintained at equilibrium ($R_0=1$) by natural enemies. The natural enemy ravine separates two "ridges", one at high and one at low density, where mortality is lower and population densities increase. At very high density, other mortality factors such as starvation and disease cause the populations to collapse. At the extremes of low density, the "Allé effect" comes into play caused by the failure of individuals to find mates. Populations in this range decline inexorably to extinction. Such densities are probably infrequent in most natural populations.

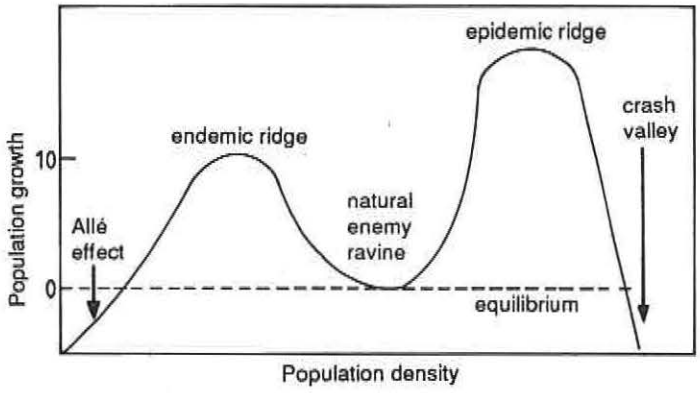


Fig. 8.4 The synoptic model of complex density dependence. Redrawn from Southwood and Comins (1976) with permission from Blackwell Scientific Publications.

The inverse density dependence evident at the mid-range of densities is a common feature of predators and parasitoids whose densities are constrained or determined by factors other than the density of their prey. This phenomenon is explained by the following concepts. Density dependent predation or parasitism may arise from two different sources: the **numerical response** and the **functional response** (Solomon 1949). The numerical response is an increase in the density or number of predators or parasitoids in response to increasing prey density. The functional response is an increase in the number of prey taken per predator or parasitoid at increasing prey density.

Important contributions to the understanding of the functional response were made by Holling (1959 a,b, 1965). Holling simulated predation with the "disc experiment" in which blindfolded human subjects searched for sandpaper discs on a table. As the density of discs increased, the subjects found more of them, but a declining percentage. The explanation is that each disc required a certain fixed handling time, so that the total time left for searching declined as more discs were found at higher density. Holling's results have subsequently been verified in field and laboratory experiments with many types of animals foraging for a food resource. The effects of handling time include latent periods following a meal during which the animals engage in activities other than searching for prey. All animals have an upper limit in their capacity for consumption. Above this limit, further increases in prey density will not cause higher consumption. The implications are that the functional response is inherently inverse density dependent. Without a numerical response, predators and parasitoids are unlikely to stabilize a host population. The numerical response of many predators and parasitoids may be limited by a variety of factors. Parasitoids may be constrained by the need for obligate alternate hosts or may be regulated themselves by hyperparasitoids. Many

vertebrate predators are constrained by fixed territory sizes and depend on a wide variety of food sources. Rarely would their densities be affected by increases in the density of particular prey species.

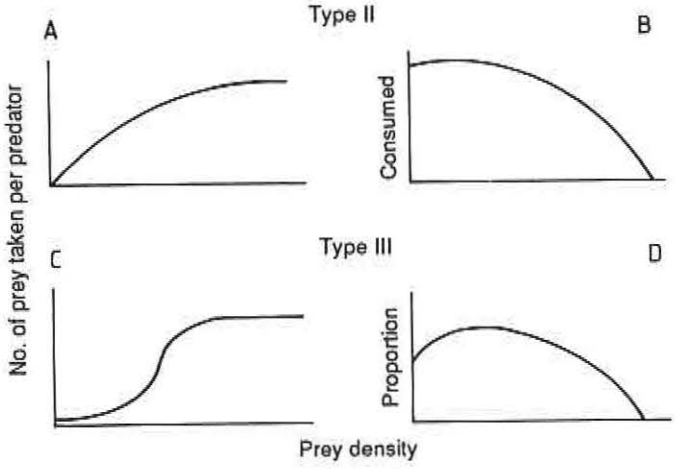


Fig. 8.5 Number of prey consumed per predator and the corresponding proportion consumed for a type II (A, B) and type III (C, D) functional response.

Further work by Holling showed that under some important conditions, the functional response can lead to positive, density-dependent predation. Whenever increases in prey density results in some change in the foraging behaviour of the predator or parasitoid, such that they forage more efficiently or concentrate their efforts on the particular prey species, the number taken will accelerate with increasing host density (Fig. 8.5C), and the proportion taken will increase (Fig. 8.5D) over the lowest range of prey densities. Holling termed this a type III functional response in contrast to type II, which is the continuous decline in proportion taken evident in the disc experiment. It occurs whenever foraging behaviour changes with prey density (Fig. 8.5 C,D). Holling (1965) demonstrated a type III response for shrews foraging for sawfly pupae. He envisioned type III responses to be characteristic of vertebrate predators, which have a relatively high capacity for learning and behavioral change. However, the type III functional response has subsequently been demonstrated in many invertebrates. Murdoch and Oaten (1975) showed that predatory snails would switch their foraging from a preferred prey species to a less preferred species, as the latter became more common, resulting in a type III response to the less preferred species. Hassell et al. (1977) showed that several parasitoid species have a type III response. This occurs because they intensify their searching activity after locating and attacking a host, but engage in other activities (resting, preening), if they fail to find a host after a period of searching. A large body of scientific research in the realm of optimal foraging theory (Krebs and Davies 1984) has demonstrated that such changes in foraging behaviour are typical of most animals that search for resources.

Changes in behaviour that involve aggregation of predators of parasites into pockets of high host density may be classified as either a functional or numerical response, depending on the scale of the response in relation to the population under study. If aggregation causes an increase in density of predators and parasitoids from outside the study area, it is typically classified as a numerical response. If aggregation increases predation rates in pockets of high host density within the population under study, it would be classified as a type III functional response. Studies of simple host-parasitoid models suggest that aggregation is a very important ingredient of the stability of many host-parasitoid systems (Hassell and May 1973, 1974, May 1978).

Delayed density dependence

It is typical for a numerical response, based on reproduction of a predator or parasitoid to a particular prey density, to be expressed in the following generation. The density of the predator or parasitoid thus lags behind that of its host, resulting in a classic predator-prey oscillation. The highest rates of attack on the host occur at peak predator density which occur after the host population density has declined. Plotting percent mortality from the predator or parasitoid against host density would not reveal a consistent pattern of positive density dependence, even though the predator or parasitoid is clearly maintaining the host within a narrow range of density. This type of response is termed delayed density dependence (Hassell and Huffaker 1969).

Controversy over the role of density dependent factors: A historical sketch

Howard and Fiske (1911) were the first population ecologists to focus on the importance of density dependent factors in population dynamics. They believed that all natural populations are in balance (equilibrium). For equilibria to exist, at least one of the many factors acting on a population, must be "facultative", which was their term for positive density dependence. Other factors could act in a "catastrophic" manner (density independent). Other factors would act in a manner opposite to facultative factors (i.e. inverse density dependence), although they had no term for it. The actual terms that we use today (density dependence and density independence) were coined by Smith (1935).

The Australian entomologist A. J. Nicholson (1933, 1957, 1958) was the next major proponent of the importance of density dependent factors in population dynamics. He reiterated the ideas of Howard and Fiske (1911), that most populations are held at equilibrium density by density dependent factors. Density dependent factors included the action of natural enemies such as predators and parasites that hold the populations of many organisms below the carrying capacity of the environment. They also include intraspecific competition of animals for resources that maintains densities at the carrying capacity. The level of the carrying capacity may be determined by the abiotic environment, but it is density dependent competition for food, space, or other resources, that regulates the population.

The factor that regulates the population is not necessarily the one that causes the most mortality. For example, weather factors such as winter kill may account for 90% of the mortality, but if each female deposits an average of 100 female eggs and there is no other mortality, the population will still increase 10-fold each generation. If there are less than 1% survivors, then populations would decline. Thus, an additional, density dependent mortality, causing between 0 and 10% mortality, would regulate the population in this hypothetical example.

According to Nicholson, nearly all natural populations vary within the limits of a characteristic abundance, which is maintained by density dependent factors. It is patently obvious that populations do not increase without limit, nor do they decline to extinction (except rarely). Density independent factors are not fine-tuned enough to maintain populations at the replacement rate for any length of time. Without density dependent factors, all populations would randomly walk to outbreak or extinction.

The Nicholsonian view of equilibrium processes was strongly challenged by Andrewartha and Birch (1954). They argued that most populations are not held at equilibrium density. Rather, densities merely fluctuate. The "characteristic abundance" of animals viewed by Nicholson as an equilibrium density is no more than the mean value of these fluctuating densities. In response to Nicholson's argument that populations governed by density independent factors cannot be fine tuned adequately to prevent eventual extinction, Andrewartha and Birch claimed that extinction of local subpopulations occurs quite frequently. However, such events occur asynchronously among subpopulations which are linked by dispersal, so that extinct sub-populations are continually recolonized. Thus, the species persists indefinitely over the entire region.

Andrewartha and Birch cited examples from a number of population systems to support their views. For example, the density of grasshopper species in the desert fringes of Australia fluctuated dramatically with variation in the density of grasses on which they fed, which, in turn was determined by rainfall in the spring. In particular, they focused on an analysis of the abundance of the flower thrips, *Thrips imajinis*, in rose blossoms in Adelaide, Australia (Davidson and Andrewartha 1948). Multiple regression analyses indicated that changes in weather explained 78% of variance in population size. They found no evidence for any significant density dependent effects. Smith (1961) reanalyzed this data and purportedly discovered evidence for density dependence. His statistical procedures, however, have been challenged by others (Eberhardt 1970).

The conflict between the views of Nicholson and Andrewartha and Birch culminated in a symposium held at Cold Spring Harbor, New York in 1957. Several population ecologists of this era attempted to find a middle ground. One of these was Milne (1957), who distinguished between perfect density dependence, (competition for resources), and imperfect density dependence, (attacks by natural enemies). The latter is imperfect because it is affected by many factors other than prey density, and is thus, only weakly density dependent.

Hairston et al. (1960) argued from a trophic level perspective for the universal prevalence of equilibrium densities maintained density dependent factors. Decomposers must ultimately be food limited, because organic matter does not accumulate. In contrast, herbivores are mostly limited by predators, because few plants are completely consumed by herbivores. Carnivores are mostly food limited. They limit their own supply. Plants are limited by competition for resources such as light and water.

Subsequent authors critiqued the views of Hairston et al. Murdoch (1966), observed that just because plants are not totally defoliated does not mean that herbivores are food limited; not all plant parts are edible. Even if herbivores are not food limited, this does not mean that they are limited by predators. Other types of limitation exist, such as competition for space or self-regulation. Ehrlich and Birch (1967) observed that the failure of organic matter to accumulate, does not mean that decomposers are generally food limited. For example, suppose we had a community with 100 decomposers, of which 99 were limited by predation and the remaining one was food limited and consumed the remaining organic matter.

The arguments about the importance of the density dependent factor and the existence of population equilibria, have persisted in recent years. Den Boer (1968, 1981, 1985, 1986), and his associate Reddingius (1971), supported the views of Andrewartha and Birch. They promoted the idea of **"spreading of risk"**, that most populations are composed of subunits, each with its own independent dynamics, but coupled by dispersal so that while individual subunits might go extinct quite frequently, the population as a whole (the meta-population) would persist indefinitely. Den Boer (1986) reanalyzed one of the classic examples of population regulation, the winter moth data of Varley and Gradwell (1960). He disputed the notion that the populations were stabilized by density dependent predation on pupae. In response, Latto and Hassell (1987) argue that Den Boer chose values for mortality at random *from the field data* and therefore, incorporated the effects of density dependent predation inadvertently.

Strong (1984) argued that most mortality factors are **"density vague"**. There is so much variability in the action of density dependent factors that the notion of regulation around an equilibrium is untenable. However, density dependence, although extremely variable and unpredictable, was sufficient to prevent extinction.

Several authors have reviewed the published literature on a variety of insect population systems for evidence of regulation by density dependent factors. Dempster (1983) analyzed twenty-four published life tables from the Lepidoptera. Only three of these contained evidence of density dependent factors and, even then, it was not clear that the host was regulated by the density dependent agent. A similar study, covering a wider taxonomic range and with a similar conclusion, was completed by Stiling (1987). Murdoch et al. (1984) found no evidence for density dependence in the olive scale/parasitoid interaction in California—

one of the best examples of successful biocontrol. Olive scale, *Parlatoria oleae* (Corvee) is completely suppressed by *Aphytis*, but there was no evidence that it did so in a density dependent manner. This interpretation was challenged by Huffaker et al. (1986). Murdoch et al. (1985) analyzed a series of the most successful bio-control efforts on record. In most cases, there was no evidence for density dependence or the existence of low density equilibrium.

In response to these analyses, Hassell (1985) pointed out that most life table data is inadequate and that statistical problems with detecting density dependence may obscure underlying density dependence. We will examine these techniques in the next chapter.

Spatial vs temporal density dependence

The classical analyses, by which Varley and Gradwell (1960) detected density dependence in winter moth populations, were performed on a single population followed over many generations. This is known as **temporal density** dependence. Many other studies record mortality acting on different populations with different density. This is known as **spatial density dependence**. While most would agree that temporal density dependence results in population equilibrium, there is no consensus as to whether spatial density dependent results necessarily in temporal density dependence. (see Hassell 1985, 1986, Hassell et al. 1987, Dempster and Pollard 1986, Stewart-Oaten and Murdoch 1990).

A related question concerns the spatial scale on which density dependence operates. Studies with the holly leafminer (Heeds and Lawton 1983) and the viburnum whitefly (Hassell et al. 1987), show that density dependent processes may only be detectable at certain spatial scales.

Tsetse fly: A case study

Discussing these issues in the context of a particular insect, the tsetse fly, *Glossina* spp. may clarify the relevance of this rather arcane debate. Rogers and Randolph (1984) review the evidence for density dependence in the population dynamics of tsetse flies. Tseste flies are continuously breeding insects with overlapping generations, as we saw in Chapter 5. The female has the unusual biology of maturing larvae inside her body cavity and depositing them on the ground just prior to pupation. The larvae then burrow into the soil to pupate. A variety of studies have linked tsetse population densities to climatic variables. For example, the adult population densities have been related to both time of year and rainfall (Nash 1937, Nash and Page 1953, Rogers and Randolph 1984) (Fig. 8.6).

An early review (Glasgow 1963) of tsetse ecology concluded that the dynamics of this insect were driven by density independent abiotic factors.

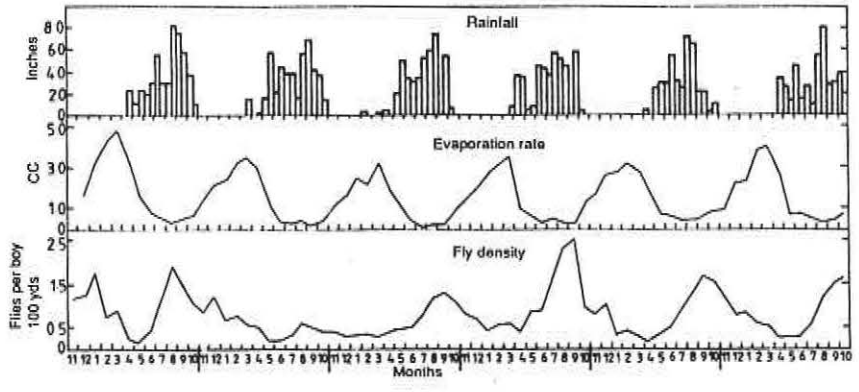


Fig. 8.6. Seasonal fluctuations in (A) rainfall, (B) atmospheric humidity and (C) density of *G. palpalis* in Nigeria (redrawn from Nash and Page 1953 with permission from the Royal Entomological Society).

A variety of evidence, summarized by Rogers and Randolph (1984, 1985), points to the existence of density dependent population processes, superimposed on the abiotic, density independent influences evident in Fig. 8.6. For example, capture rates of adult flies in traps in an area that had been sprayed, increased exponentially with time (Fig. 8.7) (linear on a log scale), but then tapered off at an upper limit (Turner and Brightwell 1986).

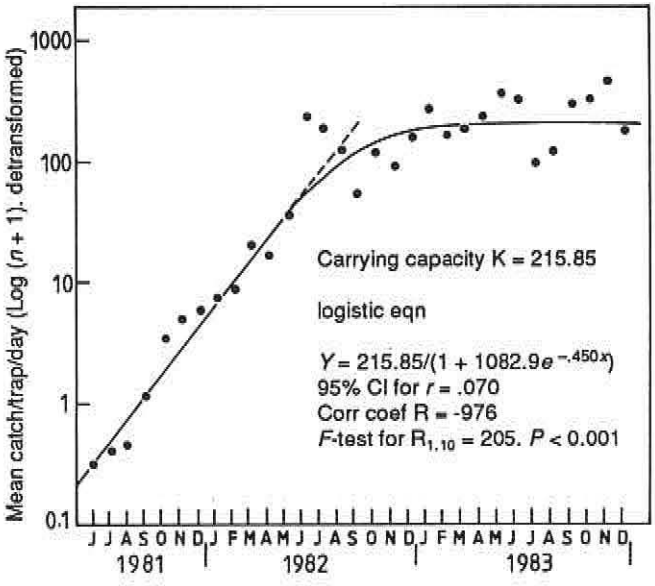


Fig. 8.7. Mean catch per trap per day of *G. pallidipes* following aerial insecticide spray in Kenya (redrawn from Turner and Brightwell 1986 with permission from the International Institute of Entomology).

Analyses of published tsetse life table data (e.g. Table 6.4) showed that the rates of increase (r) calculated using the methods outlined in Chapter 6, were negatively related to density (Rogers and Randolph 1984, Fig. 8.8).

The mechanisms regulating tsetse fly populations remain poorly understood. Experiments with released tsetse pupae at different densities revealed density dependent predation (Rogers 1974). Vale (1977) showed that the proportion of *G. morsitans* (Westwood) obtaining a blood

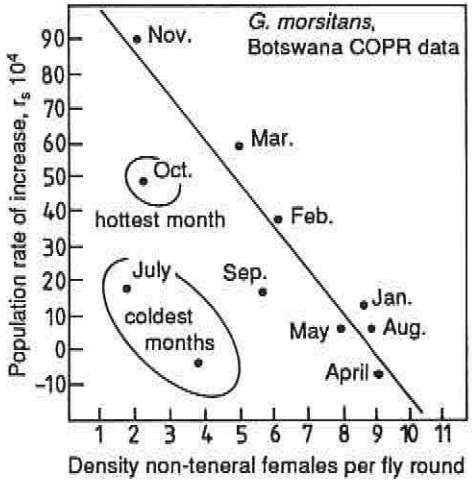


Fig. 8.8. Relationship between the rate of increase of *G. morsitans* in Botswana (calculated from monthly ovarian age dissections, data from R. Allsopp of NRI) and the catch of non-teneral females on fly rounds. (Reproduced with permission from Rogers and Randolph 1984).

meal declined with increasing fly density. Whatever the actual mechanism of density dependence, Rogers and Randolph (1984) observe that the densities of tsetse flies are far more stable than could be explained by density independent factors alone. In other words, what is noteworthy about the change in density evident in Fig. 8.6C, is not how much, but how little, it changes from year to year.

Other important African insect species exhibit far greater variation in density. The desert locust, *S. gregaria*, is an example. For such species, the Southwood and Comins (1976) synoptic model is likely to describe the overall dynamics. Density dependent factors maintain population in an endemic phase, but these factors are sometimes overwhelmed and the population shifts into outbreak phase.

9

INTERPRETATION OF LIFE TABLES: KEY FACTOR ANALYSIS AND DETECTION OF DENSITY DEPENDENCE

Key factor analysis

A **key factor**, as defined by Morris (1959), is the mortality most highly correlated with overall change in density from one generation to the next. As with density dependence, a key factor could be mortality caused by a particular agent, or it may be the total mortality from all causes occurring during a particular life stage. Expressed in another way, a key factor is the agent most responsible for the observed changes in density in a population. It is important, however, not to overemphasize the importance of this term. A key factor is not necessarily the agent responsible for maintaining the typical density of the population, nor is it necessarily responsible for regulating or stabilizing the host population. It is simply the factor most responsible for population change under current conditions. A simple example might clarify this point. Suppose we have a population for which the average fecundity is 100 eggs and there are two sources of mortality. The first mortality caused, say by predators, consumes a predictable and constant 95% of the population every generation. Another mortality factor, say a parasitoid, might kill the remaining 1–5%. Such mortality might or might not be density dependent, but either way, the parasitoid is the key factor because it is the only source of generation to generation variation in mortality and population change. The fact that the parasitoids are a key factor says nothing about what would happen if the system were altered and predation rates were changed. Obviously, such a hypothetical example is an unlikely scenario. A factor that causes a large amount of mortality (apparent mortality) is also likely to be a key factor, if it is also highly variable, as most sources of mortality are.

The methods that Morris introduced to calculate a key factor, involved regressing densities of successive life stages against the densities of eggs in the following generation. The methods have proved unreliable (Luck 1971, Kuno 1973). However, Varley and Gradwell (1960) introduced similar techniques involving data that are collected from the same population over many successive generations (at least 15). The method involved plotting the total generational mortality (expressed as K) and each of its components

(k_1, k_2...) representing the mortalities occurring in successive life stages against generation number (Fig. 9.1). The key factor is the one whose change is most highly correlated with the change in total K.

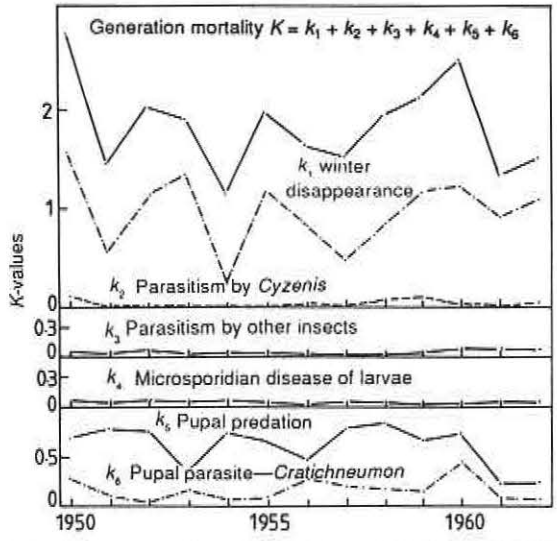


Fig. 9.1 Graphical key factor analysis of winter moth by Varley and Gradwell (1960). Redrawn from Varley et al. 1973 with permission from Blackwell Scientific Publications.

In Fig. 9.1, it is the k_1, the total change in density during the overwintering stage that is most highly correlated with K. It is important to realize that the Varley and Gradwell method treated K as a measure of total generation change including fecundity, although it was framed as a measure of mortality. Fecundity was incorporated as a k-value by calculating the actual fecundity as a fraction of the potential fecundity, expressed by the female under ideal conditions. In practice, this means dividing the observed mean fecundity (E_{obs}) by the maximum fecundity (E_{max}), and converting to a k-value: $k_f = -\log_{10}(E_{obs}/E_{max})$. Total K then measures the total change in numbers from one generation to the next.

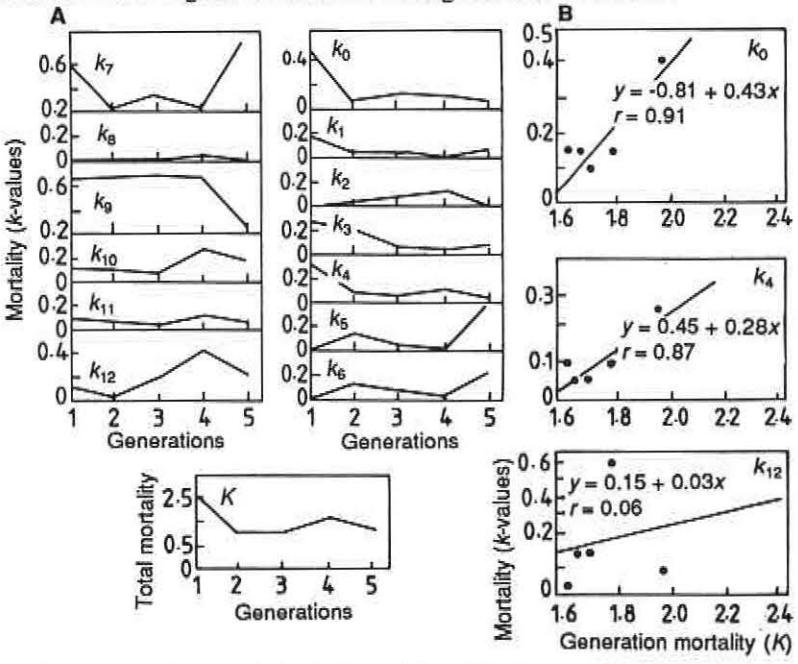


Fig. 9.2 A key factor analysis by Okeyo-Owuor and Oloo (1991) of a population of the legume pod borer with (A) the graphical technique of Varley and Gradwell (1960) and (B) the regression technique of Podoler and Rogers (1975). Redrawn with permission from Okeyo-Owuor and the ICIPE Science Press.

The graphical method of Varley and Gradwell may give an ambiguous result. There may be no factor that is clearly more correlated than others with the change in total K. Podoler and Rogers (1975) proposed a more rigorous analytic method based on a regression of k_1, k_2, k_3...against total K. The factor with the highest slope is the key factor. A slope of 1.0 indicates that the factor accounts for all of the total variation in K.

An example of both of these approaches is found in the life table study, discussed in a previous chapter (Table 7.2, Okeyo-Owuor and Oloo 1991) of the legume pod borer *M. testulalis* in Kenya. With the large number of factors (12) and only 5 generations, the graphical approach gives no clear answer. With the Podoler and Rogers (1975) technique, only 3 of the 12 mortality factors were positively correlated with total K (Fig. 9.2B). Of these K_0 egg disappearance, had the steepest slope and was thus the key factor. Such a conclusion is a reasonable one, based on the graphical comparison (Fig. 9.2A). Obviously, these conclusions are tentative, given the limited number of generations presented.

Detection of density dependence

An important ingredient in the analysis of the impact of various mortality factors on natural populations is to determine which, if any, of them act in a density dependent manner and thus might contribute to the stability of the population. The most obvious way to test for density dependence is to plot the stage-specific percent mortality against the density of the population during the stage on which the mortality acts. Since percentages are constrained to values between 0 and 100, it is obvious that such a plot is likely to be non-linear, provided it covers a sufficiently wide enough range of values of mortality. This is one reason why k-values are a better expression of mortality than percentage or proportion dying, when used in a regression context. Expressing mortality as a k-value, one would plot k_t versus log 10 (N_t), where N_t is the stage on which the population acts (Fig. 9.3).

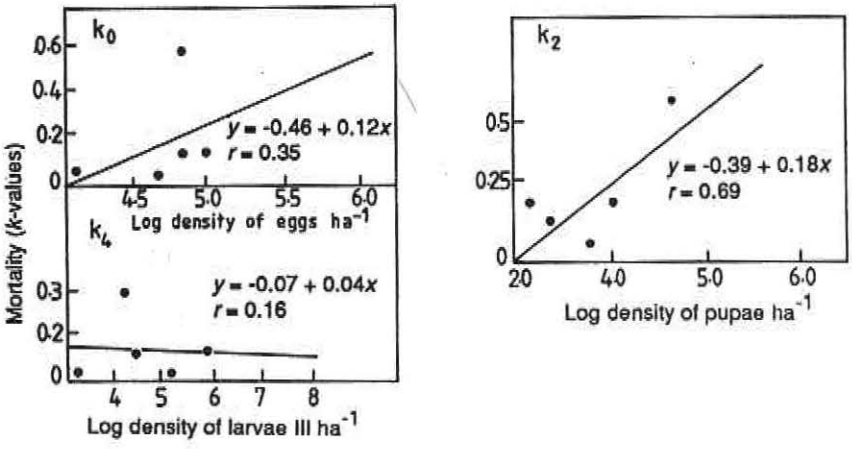


Fig. 9.3 Testing for density dependence: regression of k versus log density from a study of legume pod borer, Okeyo-Owuor and Oloo (1991). Redrawn with permission of ICIPE Science Press.

There is no reason to expect that such a regression should be linear. However, if a linear regression approximates the data well, then the slope (b) of the regression is an expression of the strength of the density dependence. If b is not statistically different from zero, then there is no evidence for density dependence. If $b < 0$ there is inverse density dependence; if $b > 0$, we have positive density dependence and the value of the slope has implication for the trajectory of the population density as a function of time. If $b = 1.0$, the mortality factor (if it alone acts on the population) would return the population to the equilibrium level in a single

generation following a disturbance that caused the population to depart from the equilibrium value (Fig. 9.4). This is known as perfect density dependence (Varley et al. 1973). If $0 < b < 1.0$, the density dependence is known as **under-compensating.**

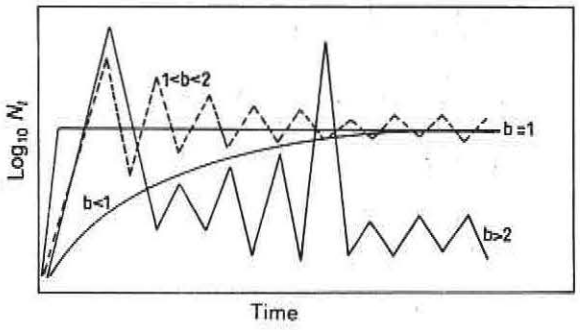


Fig. 9.4 Effect of under-compensating and over compensating density dependence on population trajectory. Redrawn from Varley et al. 1973 with permission from Blackwell Scientific Publications.

Populations with such mortality factors will move in a monotonic fashion towards equilibrium over a period of several generations. If $b > 1.0$, oscillations occur and this condition is known as **over-compensating density dependence**. For values of b between 1.0 and 2.0, damped oscillations occur. At $b = 2.0$, stable oscillations occur, and at $b > 2.0$, the population fluctuates erratically (chaos) as discussed in chapter 5.

There are several statistical problems that arise when k is regressed against log density (St. Amant 1970, Benson 1973, Eberhardt 1970, Reddingius 1971). First of all, the independent variable ($\log_{10}(N_t)$) figures also in the calculation of the dependent variable (k), so that the two measures are not independent. Errors in the estimation of N_t will consequently result in spurious correlations. Secondly, an important assumption of linear regression is that the independent variable is measured without error, whereas, in fact, there is likely to be substantial error in the estimation of N_t. Also, the values for N_t and k are not selected at random from a universe of all possible values for these variables. Rather, the values for N_t in one generation are likely to be correlated with the values for N_t in the following or preceding generation. This is known as serial correlation.

Varley and Gradwell (1968) proposed a second method for relating mortality to density that dealt with the first two of these problems. They proposed a regression of the log number of survivors ($\log_{10}N_s$) in a life stage against the log number that entered the stage ($\log_{10}N_t$): $\log_{10}N_s = \alpha_1+\beta_1(\log_{10}N_t)$. As a check on the effect of error residing only with the dependent variable, another regression is performed: $\log_{10}N_t = \alpha_2+\beta_2(\log_{10}N_s)$ reversing the dependent and independent variables. Both of these regressions are plotted on the same axis (Fig. 9.5).

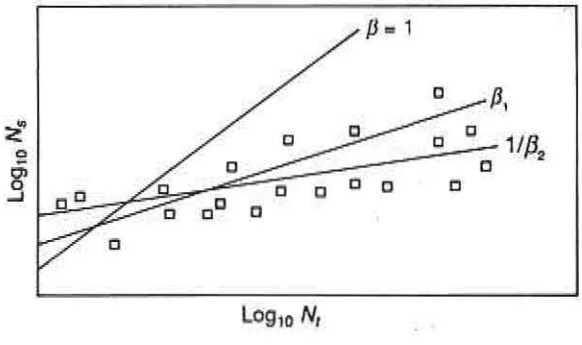


Fig. 9.5 Varley and Gradwell's second method (1968) for detecting density dependence.

The two regressions differ in that the first assumes that the measurement error resides entirely in N_t; the latter assumes the error resides in N_s. The relationship of this approach to the regression of k versus $\log(N_t)$ is as follows: According to the first method:

$$k = \log_{10} N_t - \log_{10} N_s = a + b \log_{10} N_t$$

$$\log_{10} N_s = -a - b \log_{10}(N_t) + \log_{10}(N_t)$$

$$= -a + (1 - b)\log_{10} N_t$$

According to the second method:

$$\log_{10} N_S = -\alpha + \beta \log_{10} (N_t)$$

$\alpha = -a$ of the first method,

$\beta = (1 - b)$ of the first method.

Thus, with the second method we conclude density independence when $\beta = 1.0$, which occurs when the number of survivors is always a constant fraction of the numbers that entered the stage and is equivalent to $b = 0$ of the first method. We conclude density dependence if $\beta < 1.0$ and inverse density dependence when $\beta > 1.0$. Perfect density dependence ($\beta = 0$) occurs when N_s is constant, regardless of any value for N_t.

Detection of delayed density dependence

Frequently, density dependent factors act on a population in subsequent generations. Such behaviour is evident in simple models of host-parasitoid interaction. The impact of host density on the numerical response of the parasitoid is felt in the next generation, resulting in the pattern of host-parasitoid oscillations, in which the peak densities of the parasitoid lag one or two generations behind that of the host. Plotting % parasitism or k-values against host density of the same generation would reveal no density dependence, although it is clear in this model system, that the parasitoid is regulating the host density in the vicinity of an equilibrium value. Hassell and Huffaker (1969) proposed that delayed density dependence could be detected in such plots by connecting points in successive generations. If the system had delayed density dependence, a counter-clockwise spiral would result (Fig. 9.6).

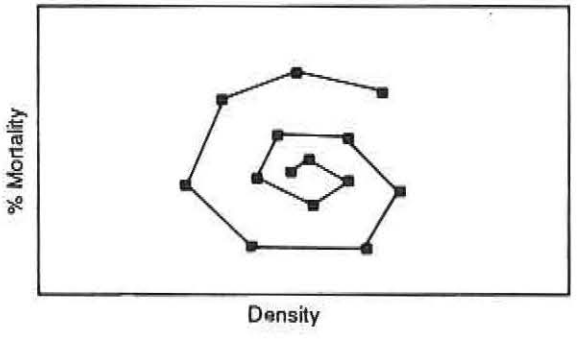


Fig. 9.6 Graphical technique for detecting delayed density dependence.

Autocorrelation methods of testing for density dependence

The second method of Varley and Gradwell does not successfully cope with the problem of serial correlation and several more recent methods have been proposed that are specifically designed for analysis of time series data (Bulmer 1975). These methods are appropriate when data from only a single life stage are available. The basic idea is to relate the number occurring in any generation to the numbers present in the same stage in previous generations.

$$\log(N_t) = a + b \log(N_{t-1}) + c \log(N_{t-2}) \qquad (9.3)$$

This model is analogous to Varley and Gradwell's second method replacing N_s with N_t. Direct density dependence is detected when $b<1$. Most of the modern treatments of detecting density dependence are based on this approach and attempt to solve the statistical problems inherent in Varley and Gradwell's method. (See Bulmer 1975, Royama 1977, Gaston and Lawton 1987, Pollard et al. 1987, Reddingius and Den Boer 1989, Turchin 1990). Turchin 1990, used autocorrelation analysis to detect delayed density dependence in 8 out of 14 species of forest insects.

EXERCISES

A long-term study of a population of hypothetical insects has yielded the following life table for 20 consecutive generations. Calculate and construct a detailed table for the first generation, containing the l_x, d_x, real mortality, apparent mortality, and k-values for each life stage. Using k-values calculated for all subsequent generations, run a key factor analysis using the graphical method of Varley and Gradwell (1960, 1968), as well as the regression method of Podoler and Rogers (1975). Which factor is the key factor? Do the two methods agree?

The next step is to determine which factors might be regulating the population densities. Do this using both the first and second methods of Varley and Gradwell. Write a paragraph summarizing what you have learned about the population dynamics of this insect.

The following table contains the densities of each life stage for 20 generations.

	GENERATION				
	1	**2**	**3**	**4**	**5**
Eggs	11,530	13,040	12,340	14,710	10,640
Larvae	6,555	5,393	9,972	3,108	4,808
Pupae	168	156	172	133	147
Adults	65	62	74	53	60
	6	**7**	**8**	**9**	**10**
Eggs	11,960	12,160	14,270	10,640	11,460
Larvae	5,105	8,986	3,106	4,106	9,016
Pupae	155	160	128	141	191
Adults	61	71	53	57	71
	11	**12**	**13**	**14**	**15**
Eggs	14,290	10,120	13,680	11,530	11,830
Larvae	2,563	7,766	4,203	4,260	10,730
Pupae	120	167	148	155	204
Adults	51	68	58	59	75
	16	**17**	**18**	**19**	**20**
Eggs	15,010	13,998	12,500	10,600	11,800
Larvae	13,759	11,379	4,368	2,370	3,776
Pupae	230	205	175	170	165
Adults	70	68	53	59	72

10

EFFECTS OF RAINFALL AND TEMPERATURE ON POPULATION GROWTH

Rainfall and humidity

In many parts of Africa, seasonal contrasts in rainfall far exceed seasonal variation in temperature. Furthermore, many of the major insect pests in Africa, such as the desert locust, *Schistocerca gregaria* (Forsk), or the African armyworm *Spodoptera exempta* (Walker), occupy arid or semi arid regions and feed on ephemeral vegetation that grows in response to rain. It is thus not surprising that the populations of many important species is linked to the occurrence of rain, and each of them responds to moisture in many direct and indirect ways. The eggs of nearly all grasshoppers require moisture in order to complete development (Dempster 1963). For the desert locust, approximately 20 mm of rain are required for successful egg development (Bennet 1976). Adults seek moist soil in which to deposit eggs. Presumably these conditions assure sufficient vegetation for larval development. Rainfall and the consequent high breeding success elicit development of the gregarious phase of *S. gregaria*, during which adults aggregate into swarms that engage in long range flight. Such swarms are transported downwind. In summer months, these winds flow into the intertropical convergence zone across the southern Sahara, where winds from northern and southern hemispheres meet (Rainey 1951, 1979). Such convergence zones are characterized by general upwelling of the surface winds and the rising air typically produces rain. As a consequence, the swarms of desert locust concentrate in regions where rainfall typically assures an adequate food supply.

Occasional massive outbreaks of desert locust or plagues have been recorded since biblical times. The onset of such plagues have been linked to the occurrence of above average rainfall for several consecutive seasons in the winter-spring breeding areas (Waloff 1976). Bennet (1976) showed that the regular occurrence of such rainfall was important, as well as the total amount, and that the collapse of plague populations was frequently associated with inadequate rain.

Similar associations have been described for *S. exempta*. Outbreaks of armyworm move with prevailing winds that concentrate the moths in regions where rainfall is adequate to sustain larval growth (Haggis 1986). Tucker and Pedgley (1983)

showed that concentrated oviposition leading to armyworm outbreaks in east Africa are associated with specific rainstorms in January to March. Analyses by Tucker (1984, 1993) showed that armyworm outbreaks were usually associated with rainstorms that were followed by dry periods. Prolonged heavy rainfall in October through December usually resulted in low densities for *S. exempta*, probably because it caused high larval mortality that offset the positive effect of rain on food supply.

Links to rainfall or atmospheric moisture have been noted for many other important African insects. Atmospheric humidity has been related to population density of tsetse fly, *G. palpalis* in Nigeria (Nash and Page 1953, Rogers and Randolph 1984), as we have previously noted in Chapter 8. During the dry season the range of the fly becomes restricted to riparian edges. The relationship between humidity and population growth for this species has been summarized by Rogers and Randolph (Fig. 10.1, 1984).

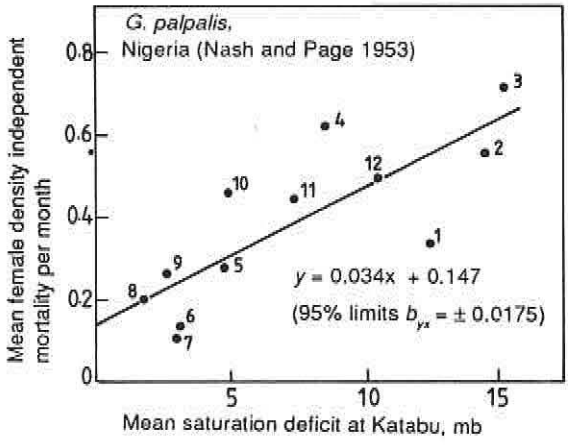


Fig. 10.1 Average (over 6 years) monthly density independent mortality of *G. palpalis* increases with average saturation deficit at Katabu, Nigeria (data from Nash and Page, 1953: 1 = January etc.). Redrawn with permission from Rogers and Randolph (1984) and ICIPE Science Press.

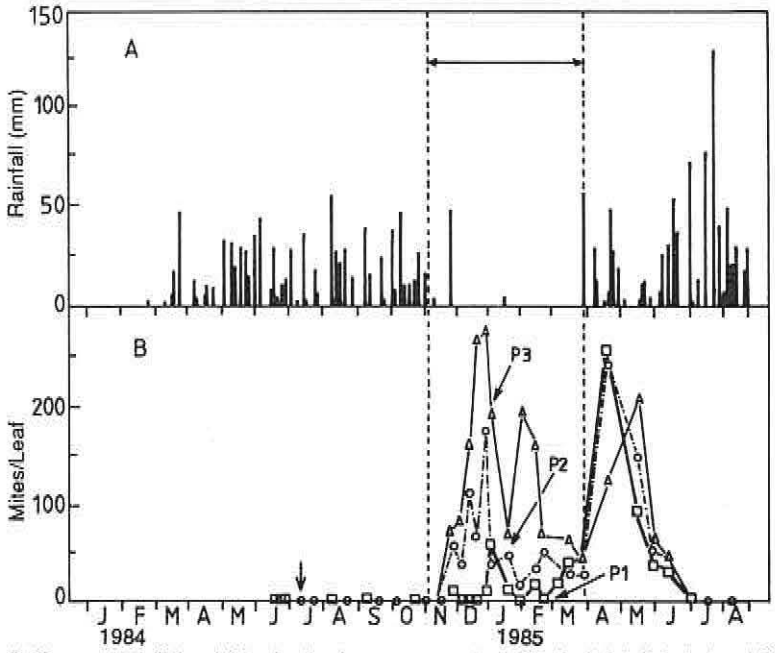


Fig. 10.2 (A) Seasonal rainfall and (B) mite density on cassava planted in April (p_1), July (p_2) and October (p_3) 1984 in Nigeria. Reproduced from Yaninek et al. (1989c) with permission from the Entomological Society of America.

The saturation deficit is one of several measures of atmospheric humidity. It is defined as the difference between the actual vapour pressure of water and the maximum possible vapour pressure at a given temperature. The saturation deficit is more closely related to drying power of the air than the more commonly expressed relative humidity (*RH*), which is the ratio of the actual to the maximum vapour pressure. Consequently, saturation deficit is more useful than *RH* for scientific work.

In contrast to the examples given above, other species are favoured by dry conditions. Increases in cassava green mite, *M. tanajoa*, populations have been associated with the mid-winter dry season in west Africa (Fig. 10.2).

The principal reason for the decline in mite density during the rainy season is that rain washes the mites off the leaves; in other words rain is a direct source of mortality (Yaninek et al. 1987, 1989c). Other indirect factors are also involved, including effects of rain on plant development and foliage quality. Mite population growth rates were actually highest immediately after the start of the rainy season in April, because reproduction is enhanced on the flush of new growth that comes as a response to the rain (Yaninek et al. 1989c). This effect is subsequently superseded by the washing-off effect.

This example illustrates that effects of rainfall may be multifaceted and difficult to unravel from other factors that vary systematically with the season, such as temperature and plant growth.

Effects of temperature

Insects are poikilothermic animals which means that their body temperatures, and consequently their rate of development, are determined by the temperature of the environment in which they live. Predicting the rate of growth from the ambient temperature has an important place in insect population ecology. The rate of growth (R) could be measured in several ways, including weight gain per unit time, but typically it is expressed as the inverse of the developmental time. For instance, if a house fly, *Musca domestica*, completes the larval stage in 4.0 days at 27°C then the rate of development is 0.25 per day. In other words, the houseflies complete 25% of larval development each day. The relationship between temperature and rate of growth is typically determined by rearing insects at constant temperatures in growth chambers. A typical pattern is seen in Fig. 10.3.

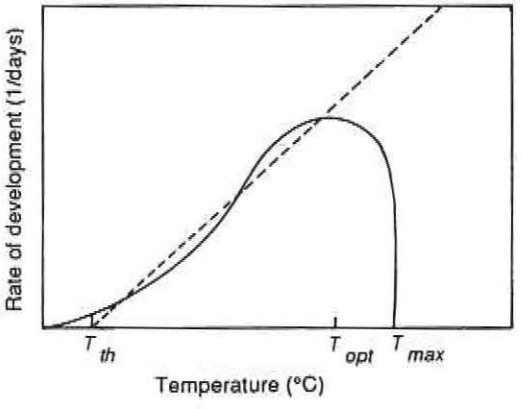


Fig. 10.3 Rate of growth as a function of temperature for a typical insect. The curved line represents actual rate of growth, the straight line is a day-degree approximation.

As temperature rises, so does rate of growth, until some optimum temperature (T_{opt}) is reached. At higher temperatures, the rate of growth declines dramatically, and above this range (T_{max}) the insect will die. However, most species are well adapted to the environment

in which they live and rarely experience temperatures above T_{opt}. Thus, the rate of growth increases with temperature and can be approximated by a straight line (Fig. 10.3). This is the basis of the concept of the day-degree, a measure of physiological time.

The concept of a day-degree, first proposed by de Reamur (1735), entails a thermal constant (K): K = (Days) $(T - T_{th})$ where T is air temperature in °C and T_{th} is a threshold temperature, normally around 10°C, below which no growth occurs. Insects or crop plants require a certain number of day-degrees to complete development. For example, if a maize crop requires 2400 day-degrees to mature from germination to harvest, this could be accomplished at a variety of hypothetical temperatures: 240 days at 10°, 120 days at 20° or 60 days at 40°, where temperature is expressed as degrees above threshold $(T - T_{th})$. If data on the rate of growth at different temperatures are obtained as in Fig. 10.1, computation of the number of day-degrees required to complete development is accomplished by fitting a linear regression to the data points, excluding all points that depart strongly from the line at high temperature: $R = a + bT$, where a and b are the intercept and slope, respectively. The threshold temperature (T_{th}) is the horizontal intercept (Fig. 10.1): $T_{th} = -a/b$. The number of day-degrees required to complete development is $1/b$.

Day-degree estimations are widely used in IPM applications to predict the phenological development of crops or the emergence of various insect pests. The concept is embodied in many simulations of crop-pest systems. The use of the concept implies certain assumptions. The most obvious of these is that growth is a linear function of temperature. Clearly, this is only an approximation which departs most strongly from the true rate of growth at the low and high temperature extremes. This problem can be partially solved by setting growth to the maximum value at temperatures above optimum, or to zero at temperatures above T_{max}.

A more serious source of error in day-degree calculations is caused by substantial differences between air temperature, as recorded by a weather station and the actual body temperature of insects. Microclimate variation of temperature at different heights within vegetation can be substantial. Insects, like any physical object exposed to direct sunlight, can absorb heat that elevates the temperature to several degrees C above ambient. An example occurs in studies of Lance et al. (1987), who showed that the body temperatures of larval gypsy moths were elevated 2–3°C above ambient. This occurred especially in high density populations that caused defoliation of forest trees, allowing the sunlight to penetrate the canopy. Furthermore, many insects engage in behaviour that permits them to avoid high temperature extremes or to elevate body temperature when air temperatures are low. Basking in sunlight or generating metabolic heat are common in many flying insects.

Fitting a day-degree model to the entire developmental period of an insect species entails the assumption that the rate of growth does not vary as the insect ages. This assumption may not hold, particularly for long-lived univoltine insects that may be adapted to grow under very different temperature regimes in early versus late instars. This limitation is easily solved by fitting a separate model for each stage or instar.

Another assumption embodied in the day-degree concept is that insects reared at constant temperature grow at the same rate as insects that accumulate the same number of day-degrees, but at fluctuating temperature. Eubank et al. (1973) showed that this assumption was inaccurate for *Heliothis* eggs.

Calculation of day-degrees from daily temperature records

The number of day-degrees accumulated by a population is frequently calculated from daily records of air temperature collected at weather stations. Exact calculations are possible where continuous records exist, such as those obtained with a pen-chart recorder, or with a digital system that records many temperature readings each day (hourly, for instance), as illustrated in Fig. 10.4.

The number of day-degrees accumulated each day is given by the area between the line that plots the changing air temperature as the day progresses, and the low temperature threshold (T_{th}), excluding all intervals for which temperature falls below threshold. If

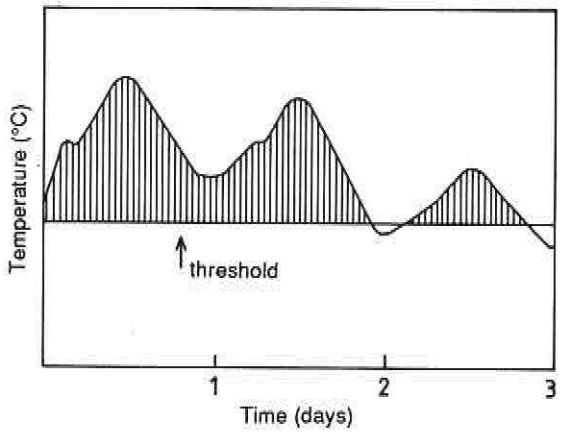


Fig. 10.4 Hypothetical continuous record of temperature over the course of 2–3 days.

temperature is recorded at regular intervals, then the number of day-degrees accumulated can be estimated as follows:

$$\frac{\sum_{i=1}^{n} T_i - T_{th}}{n} \quad \text{for all } T_i \geq T_{th} \tag{10.1}$$

where T_i is the air temperature at interval i for $i = 1,2,...,n$ intervals in the day. The number of day-degrees thus computed is equivalent to the value that would have occurred, had the temperature remained constant at the mean temperature for the day (again excluding any values below threshold). This scheme can be modified to include an upper temperature threshold (T_{th2}, Fig. 10.3), thus reducing a major part of the departure from linearity that occurs when temperatures rise into the region above optimum.

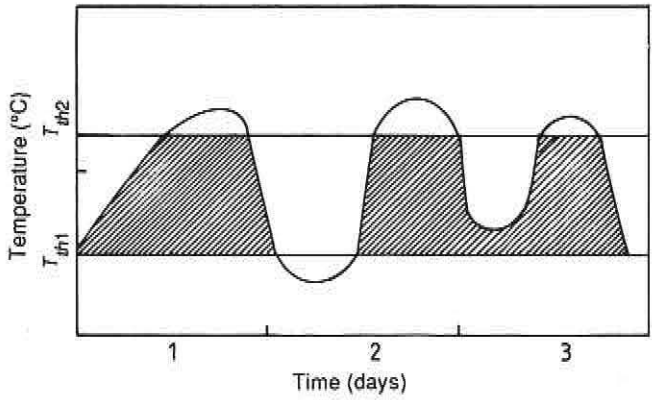


Fig. 10.5 Day-degree model with upper temperature threshold.

Day-degrees are thus estimated:

$$\frac{\sum_{i=1}^{n}(T_i - T_{th1})}{n} \quad - \quad \frac{\sum_{i=1}^{n}(T_i - T_{th2})}{n} \tag{10.2}$$

$$\text{for all } T_i - T_{th1} \geq 0 \qquad \text{for all } T_i - T_{th2} \geq 0$$

Frequently, the only temperature records available are those of daily minimum and maximum. Day-degrees accumulated on each day can be approximated by $(T_{mean}-T_{th})$, where T_{mean} is the mean temperature estimated from:

$$T_{mean} = \frac{Max + Min}{2} \tag{10.3}$$

As long as the minimum temperature remains above T_{th}, this calculation is usually quite accurate. An extreme example of this problem is illustrated in Fig. 10.4. The mean temperature is below the threshold, so the day-degrees estimated is equal to zero. In contrast the actual day-degrees accumulated during the warm hours of the day are considerable, as indicated by the shaded region in Fig. 10.6.

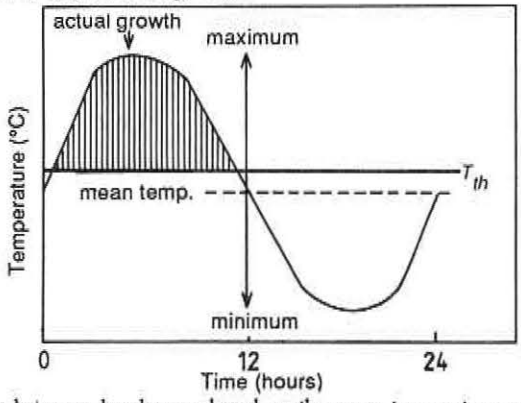


Fig. 10.6 Discrepancy between day-degrees based on the mean temperature and the actual day-degrees accumulated.

This source of error can be largely eliminated by fitting a sine wave to the daily Min, Max data (Arnold 1960), as one can see by inspecting Fig. 10.4. Computer programs that do this are widely available, including that of Allen (1976) which includes both upper and lower temperature thresholds, or Baskerville and Emin (1969), which allows the user to choose between no growth or maximum growth at temperatures above the upper threshold. Allen's program allows users to calculate cooling day-degrees, which are the accumulation of temperature units below some threshold. These might be useful for insects that require an accumulation of cool temperatures to complete diapause.

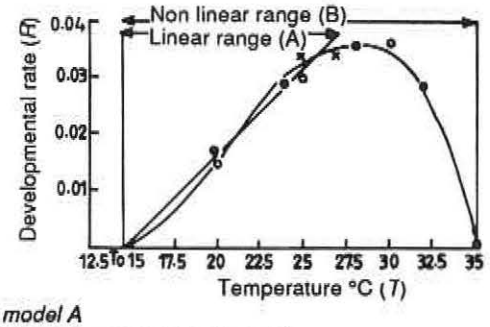
Non-linear rate-of-growth models

The obvious non-linearity of growth as a function of temperature (Fig. 10.1) has spawned many attempts to fit nonlinear models of growth. One of the earliest efforts was by Davidson (1944), who fit a logistic equation to growth, using the linearized transform of the logistic, discussed in chapter 5. A more recent application of the logistic to insect growth can be found in Casagrande et al. (1987). Use of the linear regression approach requires that the upper threshold (K) be estimated independently. Modern, non-linear, maximum likelihood methods can be used to estimate all three parameters (K,a,b) simultaneously. An obvious limitation of the logistic is that it approaches optimum growth rate asymptotically and does not capture the precipitous decline in growth that occurs between T_{opt} and T_{max}.

This limitation is avoided with polynomial regression:

$$R = a + b_1 T + b_2 T^2 + b_3 T^3 \tag{10.4}$$

The second and third order terms capture the non-linear shape of the curve. An example of this approach (Fig. 10.7) is a laboratory study by Schulthess et al. (1987) of the cassava mealybug, *Phenacoccus manihoti* (Mat.-Ferr.).



Linear model A

$R = 0.0032 \times T - 0.047$ with $r^2 = 0.968$

$T_0 = 14.66$ (temperature threshold)

$K = 312.5$ (thermal constant)

Non-linear model B

$R = 0$ if $(14.6 \geq T \geq 35.0)$

$R = 0.236 - 0.038 \times T + 0.002\ T^2 - 0.0000304 \times T^3$ for range $(14.6 < T < 35.0)$

Fig. 10.7 A third order polynomial model of rate of growth of cassava mealy bug as a function of temperature. Redrawn with permission from Schulthess et al. (1987). Different symbols represent data from different sources.

The decline in growth rates above T_{opt} is sometimes extremely abrupt and cannot be adequately mimicked with polynomial regression. A variety of nonlinear models have been proposed that address this problem. Some of these are derived from physiological principles (e.g. Logan et al. 1976, Hilbert and Logan 1983, Sharpe and de Michele 1977, Schoolfield et al. 1981).

Variability in developmental rate

Thus far we have focused only on the mean developmental rate as a function of temperature. The variability in developmental rate as a function of temperature is also an important issue, particularly for those engaged in the construction of detailed simulations that include the changing distribution of individuals between instars, as a population matures (Fig. 7.1). Largely because individuals in a population are exposed to different microclimates (or feed on different hosts), they mature at different rates. As a result, the population is frequently distributed narrowly among the first one or two instars after hatch, but become more spread out between different instars as the population progresses toward the adult stage (Fig. 7.1).

Several methods have been proposed to quantify developmental variance among populations reared at constant temperature under laboratory conditions (Regniere 1984, Wagner et al. 1984). The method of Wagner et al. (1984), for example, involves fitting a Weibull function to the cumulative developmental times of individuals reared at each temperature. These cumulative times are normalized so that a single function can be derived for all temperatures, since the variability in developmental rate decreases, and hence, the slope of the cumulative developmental curve increases at increasing temperatures up to T_{opt}.

The model of Wagner et al. (1984) estimates variability due to inherent differences (genetic and non-genetic) in growth rate between individuals reared at constant temperature in the laboratory. However, most of the variability that exists in field populations, arises from microclimate and other environmental effects. A technique that captures all sources of variability by fitting a developmental model to the instar distribution data (as in Fig. 7.1) has been proposed by Dennis et al. (1986) and Kemp et al. (1986). The technique involves fitting a multinomial distribution to the instar distribution data. A disadvantage of this method is that it must be performed separately for each field population.

EXERCISES

The following data has been collected on the duration of the larval stage of the mango weevil at different temperatures. The experiment was done by rearing the weevils in growth chambers at constant temperature.

Temp.	Mean no. days to complete development
10°C	90
15°C	40
20°C	30
23°C	20
26°C	13
28°C	7
31°C	6
32°C	7
33°C	12

1. Plot the rate of development (1/Days) vs temperature on graph paper. Excluding the values above 32°C, plot a linear regression of 1/Days vs temperatures. What is the low temperature threshold and how many day-degrees are required to complete development?

2. Using the equation given for the logistic given in Chapter 5, fit a regression line to the data using the linear form of the logistic, (eq. 5.11). Estimate K by eye, a maximum rate of development and transform the rate of development into the appropriate linearized form. Does this equation fit the data better (have a higher R^2) than the linear day-degree model?

3. The following is a temperature record made every two hours at a weather station for a ten day period. Using the day-degree calculations in part 1, determine the number of day-degrees accumulated over the ten day period. Calculate "true day-degrees" based on the accumulated bi-hourly temperature (minus threshold temperature). Also use the daily minimum and maximum reading to compute day degrees based on estimated daily mean (T_{mean} = (Min + Max)/2). Explain why these two procedures might give you a different answer.

	Day									
	1	2	3	4	5	6	7	8	9	10
Hour	Temperature °C									
2:00	6	10	5	5	10	12	15	12	17	5
4:00	3	7	5	3	9	10	12	10	15	3
6:00	3	5	3	3	7	10	10	12	15	4
8:00	4	8	3	5	9	10	12	13	17	5
10:00	10	12	10	10	13	15	17	16	20	9
12:00	15	17	12	11	15	18	20	18	21	10
14:00	20	22	15	13	19	23	24	22	27	15
16:00	20	23	17	12	18	23	25	23	26	13
18:00	18	19	15	11	16	20	22	19	24	12
20:00	14	13	11	9	14	19	19	18	19	10
22:00	10	9	7	5	13	15	17	18	13	9
24:00	9	8	6	5	10	14	13	19	9	8

11

DISPERSAL AND MIGRATION

Movement and dispersal of insects are dominant features in the population dynamics of many species. These processes frequently overwhelm population growth and mortality occurring at the local level. For many important African species, such as the desert locust, *S. gregaria*, and the African armyworm, *S. exempta*, regional outbreaks arise from source populations in relatively

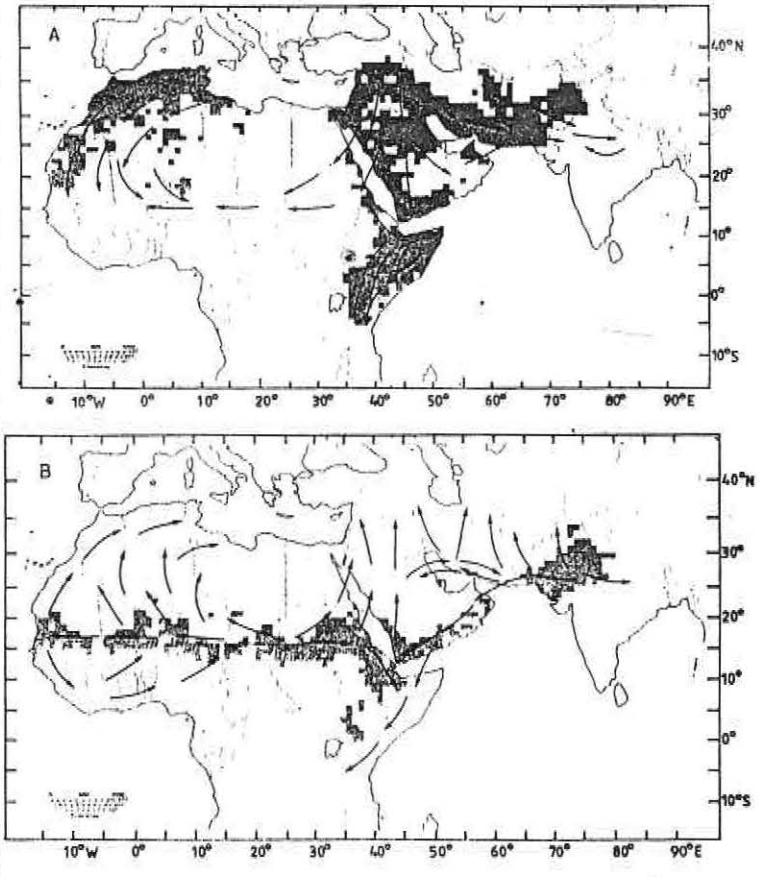


Fig. 11.1 Breeding and migration of desert locust into (A) the spring breeding areas and (B) the intertropical convergence zone in summer 1954 (redrawn from Steedman 1990, based on Rainey 1979 with permission from the Natural Resources Institute).

restricted areas and then spread to vast regions on a continental scale. Population studies have focused on understanding and predicting the movement of these organisms. **Dispersal** is usually defined as the spreading out of individuals away from others, especially from parents or siblings. **Migration** refers to the mass movement of individuals in a particular direction. Migration may be seasonal, as in the movement of locusts or birds, or diurnal, as in the daily movement of plankton between surface and deeper layers of water.

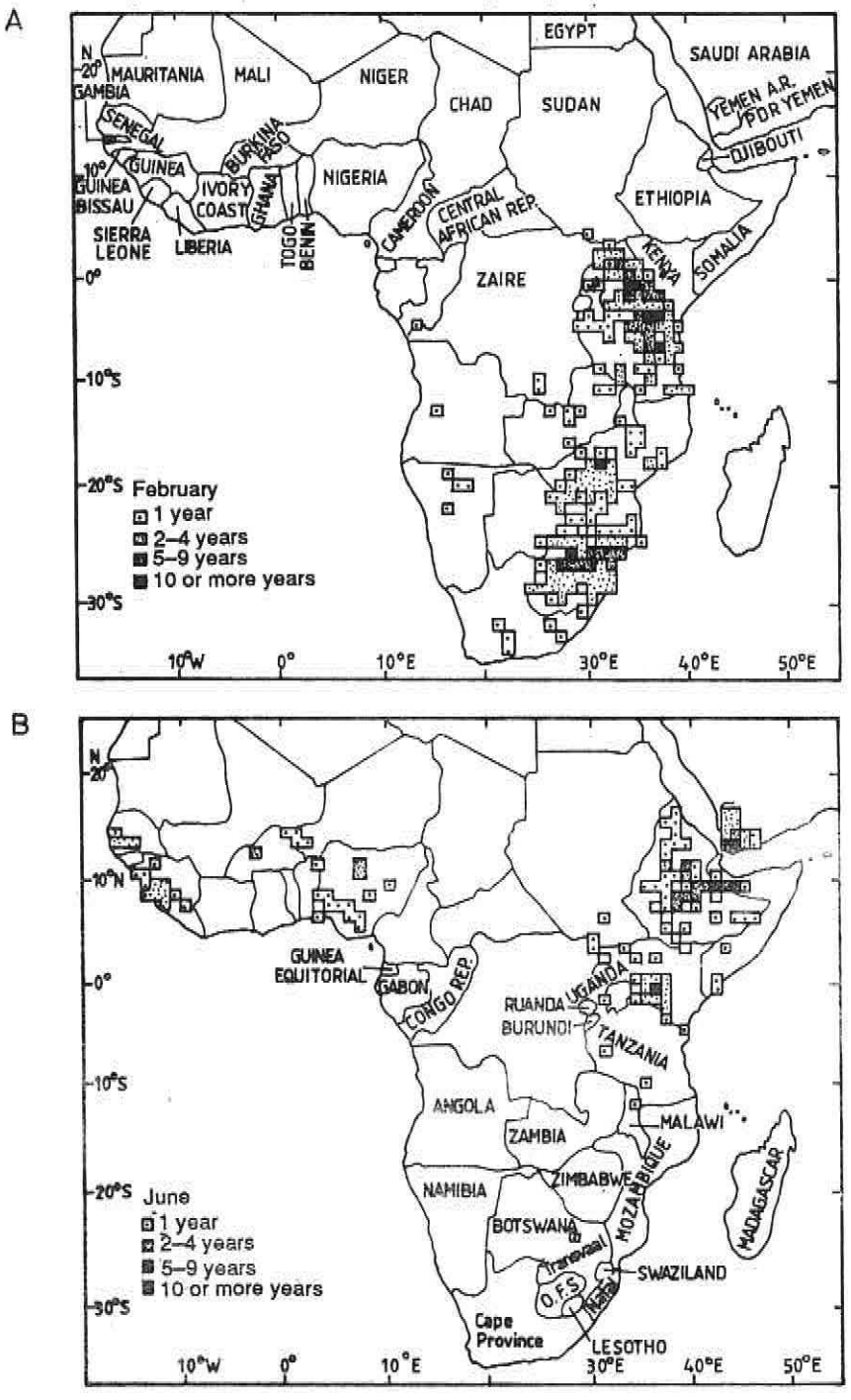


Fig. 11.2 Frequencies of outbreak areas of African armyworm 1940–1982 in (A) February and (B) June caused by wind-driven migration during the period 1940–1982 (reproduced from Haggis 1986 with permission from the International Institute of Entomology).

Outbreak populations of the desert locust arise when favorable conditions, notably adequate rainfall, elicit development of the gregarious phase of *S. gregaria*, during which adults aggregate into swarms that engage in long range flight. Such swarms are transported downwind. In summer months these winds flow into the intertropical convergence zone across the southern Sahara (Fig. 11.1), where winds from northern and southern hemispheres come together (Rainey 1951 1979).

Similar associations have been described for *S. exempta*. Outbreaks of armyworm move with prevailing winds northwards in east Africa from April to June (Fig. 11.2, Haggis 1986, Tucker 1993). As we observed in chapter 10, these wind patterns concentrate the moths in regions where rainfall is adequate to sustain larval growth.

Movement of a different type occurs when a species invades a region where it previously did not exist. Many of the major pest species around the world have been spread from one continent to another. An example of this in Africa is the cassava green mite, *Mononychellus tanajoa* (Bondar) (Yaninek et al. 1989a). This mite was introduced to Uganda in 1971 from South America. It has since spread across the cassava growing region of Africa and has become a major pest (Yaninek et al. 1989b). The spread of green mite is illustrated in Fig. 11.3.

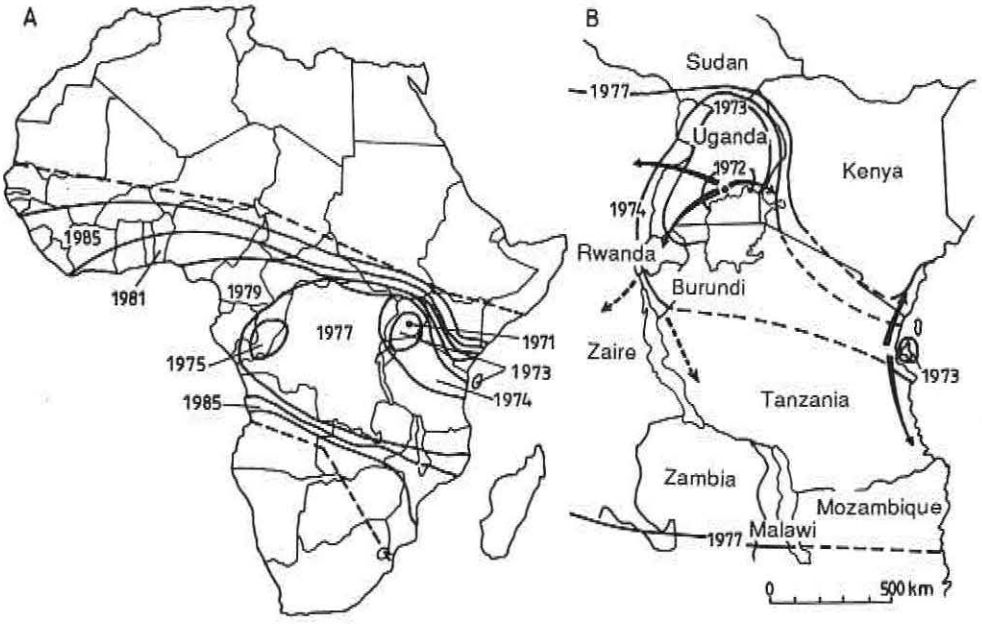


Fig. 11.3 Spread of the cassava green mite from its point of introduction near Kampala in 1971 (reproduced with permission from Yaninek 1989b).

Attempts to model the movement of animals and to predict the rate of spread from a point source have mostly been based on analogies to molecular diffusion (Dobzhansky and Wright 1943, Skellam 1951, Karieva 1983). Such analogies assume that movement consists of a random walk: a series of small steps, with direction at each step chosen at random, and a constant mean rate of movement. Each animal is assumed to behave independently of all other individuals. If such assumptions hold, then the density of animals released from a point source will be normally distributed along any axis away from the release point, with the mode of the distribution at the source (Fig. 11.4A). The following diffusion equation relates the changes in density (N) at any point (x,y) of animals at time (t) after release from a point source at ($x = 0, y = 0$):

$$\frac{\partial N}{\partial t}(x, y, t) = D\left[\frac{\partial^2 N}{\partial x^2} + \frac{\partial^2 N}{\partial y^2}\right] \qquad (11.1)$$

where D is the coefficient of diffusion, a parameter that expresses the rate of diffusion for the species in question. The variance (σ^2) of the distribution of animals at time (t) in Fig. (11.4A) in any direction is $2Dt$ (Kareiva 1983). Thus, if the animals can be censused or sampled in such a way that the distance or displacement between each animal and the point of release can be measured, D can thus be estimated.

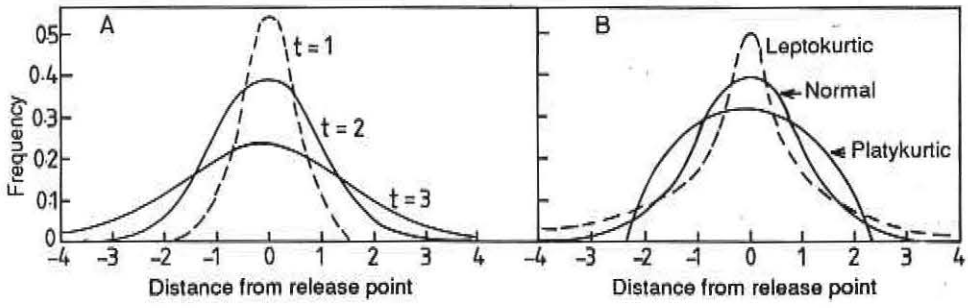


Fig. 11.4 (A) Normal distribution of animal density according to a random walk model along any direction extending away from a point source release at increasing times t_1, t_2 and t_3 after release. (B) comparison of normal, platykurtic and leptokurtic distributions.

Such estimations are only possible where a complete census or estimate of density can be made at all points (x,y) away from the source, and where losses of animals due to mortality or other causes are negligible or can otherwise be estimated. Frequently, it is only possible to obtain estimates of density (N_t) at specific locations at distance x from the source. If so, D can be estimated from the following (Scotter 1971):

$$N_t = \frac{M}{4\pi Dt} \exp\left[\frac{-x^2}{4Dt}\right] \qquad (11.2)$$

where M is the number of "marks at risk", i.e. the number of insects released at the center point that still survive at time t. This equation cannot be solved for D, but values of D that make both sides of the equation equal can be found by iteration, provided independent estimates of M are possible. Two such values of D will be found; these correspond to the increase and subsequent decrease in density that occurs at any point. Scotter (1971) presents extensions of eq. 11.2 for conditions in which density is estimated across a distance interval (a ring surrounding the source), instead of a point, or when density estimates are obtained from sampling devices that capture over an interval of time.

If the distribution of animals can be measured, departure from normality (i.e. departure from a random walk model) can be determined by estimating the kurtosis of the distribution (Fig. 11.4B, Dobzhansky and Wright 1943, Kareiva 1983). If dispersal is density dependent and the animals are more dispersive at the central release point, or if the population consists of individuals with unequal dispersal abilities (Kareiva 1983), the distribution will be leptokurtic (Fig. 11.4B). If dispersal rates increase with distance from the source, the population will be platykurtic. Leptokurtosis is quite common in nature and may arise from a variety of causes (see Kareiva 1983).

Departure from a random walk model can also be detected by plotting density (N) versus distance (x) from the source. Under the assumption of a random walk model (normal distribution):

$$N = \exp(a + bx^2), \quad \text{or equivalently:}$$

$$\text{Log } N = a + bx^2 \qquad (11.3)$$

Taylor (1978) showed that few populations conformed to this pattern, as determined by non-linear least squares analyses. He proposed that eq. 11.3 be considered a special case of the more general model:

$$N = \exp(a + bx^c) \quad \text{or equivalently}$$
$$\text{Log } N = a + bx^c \tag{11.4}$$

where the parameter c determined whether the population was more leptokurtic ($c < 2$) or more platykurtic ($c > 2$) than the normal curve ($c = 2$, Fig. 11.4B).

Jackson 1941 presented data from mark-recapture study of tsetse flies released from a single point and recaptured in a spiral grid of traps (fly rounds), extending several km from the release points. Tsetse dispersal is complicated by the occurrence of feeding cycles in the adult flies, resulting in cyclical variation in propensity to be trapped. As a result, analysis of recapture results has not been straightforward. Jackson concluded that tsetse movement was restricted to specific ambits or fixed flight paths. However, Bursell (1970) and Rogers (1977) reanalyzed Jackson's data and other data and concluded that tsetse dispersal was consistent with a random walk model. Further analyses by Hargrove (1981) concluded that no simple random walk model could account for Jackson's data, because the rate of movement and mortality varied with fly age.

Skellam (1951) coupled a diffusion model (11.1) with exponential population growth to predict the rate of spread of an invading organism, such as the cassava green mite illustrated in Fig. 11.3:

$$\frac{\partial N}{\partial t}(x, y, t) = rN + D\left[\frac{\partial^2 N}{\partial x^2} + \frac{\partial^2 N}{\partial y^2}\right] \tag{11.5}$$

where r is the intrinsic rate of natural increase. He applied his model to predict the rate of spread of muskrats in Europe. One of the predictions of the model is that the front of infestation moves at the rate $2\sqrt{rD}$. Andow et al. (1990) fit the model to a series of data sets on invading organisms.

The models discussed above all assume that there is no net displacement of the population in any particular direction (downwind, for instance). This assumption will not hold for most airborne insects, as illustrated in Figs. 11.1–11.2. Net movement can be incorporated by adding a term for advection to eq. 11.1 or 11.5. Atmospheric physicists who study the downwind movement of air pollutants use the analogous Gaussian plume model (Pasquill 1974) for a continuously-releasing point source. These models assume that the average concentration of airborne particles downwind of an elevated point source is normally distributed in any axis perpendicular to the downwind direction (Fig. 11.5). Such models have frequently been applied to the dispersion of pheromones from a point source (reviewed in Elkinton and Cardé 1984).

Mason and McManus 1981, added a term for settling velocity to the Gaussian plume model and computed deposition of gypsy moths downwind of a point source. A key distinction is whether the source is viewed as releasing insects continuously, or whether a single instantaneous release occurs, as in the tsetse experiments cited above. If the latter is the case an analogous equation (Pasquill 1974) applies.

Attempts to fit these models to any particular data set or species is likely to encounter complications. The cassava green mite, for instance, clearly spread more rapidly east-west than north-south (Fig. 11.3). North-south spread is limited by the range of cassava cultivation. Also, a major jump in the distribution from east to west Africa occurred in 1975. On a local scale, systematic changes or differences between individuals in behaviour, as illustrated with the tsetse fly examples above, and nonrandom fluctuation in wind direction on a variety of temporal scales have often frustrated the few attempts to apply these models to particular cases. As with many of the theoretical concepts presented in these chapters, the importance of these models is largely conceptual. They form a starting point from which we can begin to understand the natural process of dispersal.

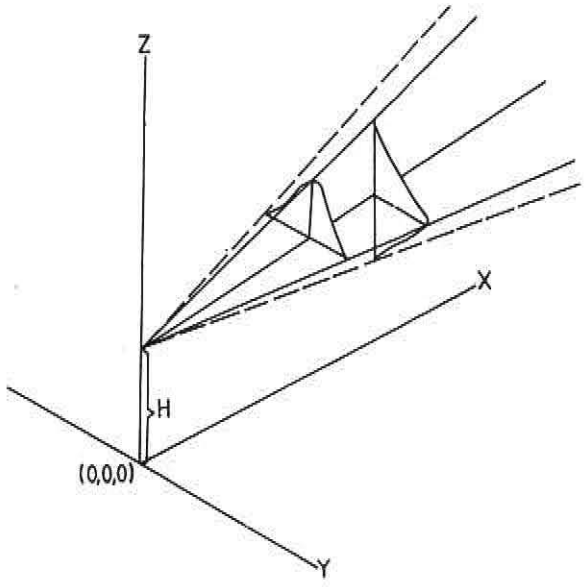


Fig. 11.5 A Gaussian plume model of the concentration of airborne particles downwind (along x axis) from an elevated point source. Reproduced from Elkinton and Cardé (1984) with permission from Chapman and Hall.

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