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With 12 figures in the text

Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities

R. M. ANDERSON[1] *and* D. M. GORDON[2]

[1] *Department of Pure and Applied Biology, Imperial College, London University, London SW7 2BB*

[2] *Department of Zoology, King's College, London University, London WC2R 2LS*

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SUMMARY

The paper examines the factors which generate various patterns of dispersion in the distribution of parasites within their host populations. Particular emphasis is placed on the role played by chance elements in the growth and decay of parasite populations and on the influence of different types of demographic processes. It is argued that observed distributions are dynamic, rather than static, entities generated by opposing forces, some acting to create over-dispersion and others acting to generate under-dispersion. Monte Carlo simulation experiments, based on probability models of the growth and decay of host and parasite populations, are used to study the dynamics of parasite dispersion. Attention is specifically focused on the role played by parasite-induced host mortality. It is shown that, for certain types of host–parasite associations, convex curves of mean parasite abundance in relation to age (age-intensity curves), concomitant with a decline in the degree of dispersion in the older age classes of hosts, may be evidence of the induction of host mortality by parasite infection. Empirical evidence is examined in light of this prediction. In general, however, simulation studies highlight the technical difficulties inherent in establishing clear evidence of parasite-induced host mortality from ecological studies of hosts and parasites in their natural habitats.

INTRODUCTION

Since the publication of the seminal work of Crofton (1971*a*, *b*), the observed patterns of parasite dispersion within host populations have been the focus of much attention in the parasitological literature (Pennycuick, 1971; Schmid & Robinson, 1972; Rutledge, Ward & Buckwalter, 1973; Anderson, 1974; Boxshall, 1974; Randolph, 1975; Campbell, Ward & Garrie, 1980).

A trend in recent research on parasite ecology has been the recognition of the importance of these dispersion patterns to the population dynamics of host–parasite associations (May, 1977; Anderson, 1978; Anderson & May, 1978, 1979; May & Anderson, 1978, 1979). It has been established, for example, that the aggregated or over-dispersed patterns of parasite numbers/host, widely observed in natural habitats, act to enhance the density-dependent regulation of both host and

parasite abundance (Anderson, 1981). The majority of hosts tend to harbour few parasites while a few hosts harbour the major proportion of the parasite population. It is in these few hosts that density-dependent processes exert their regulatory influence, whether via the suppression of parasite fecundity or survival, or via the influence of the parasite on host survival and fecundity.

A further trend in recent research has been the experimental analysis, in the laboratory, of the processes which generate over-dispersion in the distribution of parasites within their host populations. For example, it has been established that heterogeneity in host behaviour and aggregated spatial patterns of infective-stage distribution can generate high degrees of over-dispersion of parasite numbers/host under carefully controlled experimental infection procedures (Anderson, Whitfield & Dobson, 1978; Keymer & Anderson, 1979).

In natural habitats, the identification of the processes responsible for generating observed patterns of parasite dispersion is fraught with difficulties and has attracted relatively little attention. Such patterns, however, may themselves provide important clues concerning the influence of the parasite on the dynamics of the host population. This topic is the theme of this paper. Specifically, we focus on the following question. Can the frequency distribution of parasite numbers/host be used to establish whether or not a parasite is a significant cause of host mortality? This is a theme that was originally of interest to Crofton (1971*a*) in his treatment of truncated distributions but our approach differs in two substantial ways. First, we reject the idea of a lethal level in which hosts who harbour less than a specified number of parasites survive and those who harbour more die. We simply assume, in accord with the available empirical evidence, that the probability of a host dying in a given time interval is some function of its parasite burden (Anderson, 1978; 1979). Our second major departure from Crofton's conceptual view of this problem is that we regard the observed distribution of parasite numbers/host as a *dynamic* entity which is subject to opposing forces, some acting to increase the degree of aggregation, others acting to reduce it. We are therefore interested in the change through time of the degree of parasite aggregation within the host population. Specifically, we focus on the dispersion of parasites in a cohort of hosts of the same age and examine changes in this pattern as the cohort ages. Data of this form, collected by sampling natural populations, is often presented as age-intensity or age-prevalence curves. The information may be obtained by a cross-sectional survey of the host population at one point in time, or, more appropriately, by following a given cohort of hosts through time.

The paper is organized as follows. *First*, we discuss the various population processes responsible for generating observed patterns of parasite numbers/host. In particular we develop the theme of the dynamic nature of parasite frequency distributions. In the *second* section we construct a simple probability model for the exploration of the influence of various population processes on the dynamics of the frequency distribution and the abundance of parasites within their host population. The *third* section describes the use of this model to examine the impact of parasite-induced host mortalities on age-intensity curves. We discuss our model predictions in the light of observed patterns, specifically focusing on helminth parasites of fish. The *final* section, presents a critical discussion of our results and suggests lines for future theoretical, experimental and field research.

FACTORS INFLUENCING THE DISTRIBUTION OF PARASITE NUMBERS/HOST

By way of an introduction to this section, some comment on terminology is necessary. Much confusion exists in the ecological literature because the word 'distribution' is used in both its colloquial and statistical senses. Colloquially, 'distribution' is synonymous with 'arrangement' or 'pattern'. Statistically, it means the way in which variate values are apportioned, with different frequencies, in a number of possible classes. In this sense there is no implied reference to spatial arrangement. We use the word 'distribution' in this paper in the statistical sense only, where the variate values are the individual hosts and the possible classes are the numbers of parasites contained within, or on, the hosts. We use the word 'dispersion' to refer to the overall form of this statistical distribution of parasites.

Distribution patterns of parasites within their host populations can, broadly speaking, be divided into three distinct categories, namely, under-dispersed (regular or homogeneous), random and over-dispersed (contagious, aggregated or heterogeneous). These patterns are commonly described empirically by three well-known probability distributions; the positive binomial for under-dispersed patterns, the Poisson for random patterns and the negative binomial for over-dispersed patterns. A simple measure of the degree of dispersion is the variance to mean ratio of parasite numbers/host. This statistic will be used throughout this paper and will be denoted by the expression $s^2/\bar{x}$, where s^2 is the sampling variance and $\bar{x}$ is the sample mean. For under-dispersed distributions the variance to mean ratio is less than unity in value, for random patterns it is approximately equal to unity, while for over-dispersed distributions its value is greater than unity.

The generation of variability in the observed abundance of animal populations may be created by two distinct types of mechanisms. First, there is what is termed *demographic stochasticity* (May, 1975). Population growth is a stochastic (chance or random) process since one can say only that there is a certain probability that a particular parasite will die in a given time interval or that a new infection will arrive in the same time interval. The use of the adjective 'stochastic' is intended to draw attention to the random aspect of population changes, due partly to the intrinsically discrete structure of populations (Bartlett, 1960). Second, there is *environmental stochasticity*, which, in the case of parasitic species, implies that the rate processes (i.e. birth, death, immigration and emigration rates) that govern population growth are not in reality constants for a given species but depend on environmental factors, such as climate and host susceptibility or behaviour, which vary both on a temporal and spatial scale. For parasites in which the host provides an environment for population growth, differences in host behaviour and/or susceptibility (due either to genetic factors or past experience of infection) will be of major importance.

As a starting point it is informative to consider demographic factors in isolation. Environmental variability will be superimposed on top of the intrinsic variability generated by population events at a later stage in this paper. Each population process, such as the birth, death, immigration (infection) and emigration rates, generates its own typical pattern of variability. For example, a stochastic population model of a pure death process predicts that the probability distribution

of parasite numbers/host will be positive binomial in form (i.e. under-dispersed). Alternatively, a stochastic model of a pure birth process predicts that at any point in time the probability distribution of population abundance will be negative binomial in form (i.e. over-dispersed). A summary of these results, for each population process considered in isolation, is documented in Table 1. Similarly, each combination of population processes, as a result of demographic stochasticity, will generate its own particular pattern of variability in population abundance. The results for various combinations are also listed in Table 1. In later sections of this paper we will specifically be concerned with helminth infections of vertebrates, such as fish. In this context, parasite population growth within an individual host is invariably controlled by immigration (infection) and death processes (either due to natural mortality or mortality of the host). As such, it can be seen from Table 1 that the underlying pattern of variability in parasite abundance between hosts, generated by stochastic factors of a demographic nature, will be Poisson in form where the variance to mean ratio is aproximately equal to unity. Such patterns, however, are rarely observed due to the action of environmental stochasticity which tends to generate over-dispersion in the distribution of parasites within the host population. Before proceeding to consider such environmental factors one further aspect of demograhic variability must be considered.

For parasite sub-populations within individual hosts, the distribution of parasite numbers within the host population is also influenced by the rate of mortality acting on the host population. For adult helminth parasites (and most other parasitic organisms) the death of the host normally results in the death of the parasites contained within. In two or three host life-cycles, host death by predation may result in parasite transmission to the definitive host or the next intermediate host in the developmental sequence. With respect to the larval parasite population such losses are equivalent to mortality but they obviously form an immigration or gain term for the next population in the developmental cycle. If the death rate of the host is independent of the burden of parasites harboured (irrespective of whether death is due to predation or other causes), then the distribution of parasite abundance throughout the host population will simply be controlled by the population processes influencing the growth of the sub-populations within individual hosts. For example, if the growth of a parasite sub-population is controlled by immigration and death processes, then the resultant distribution of parasite abundance between hosts will be Poisson in form irrespective of the magnitude of the death rate of the hosts, provided this rate is independent of parasite burden. This result is established in Appendix A. However, if the host death is dependent on parasite burden (whether due to indirect or direct causes), as commonly appears to be the case for helminth infections of vertebrates (see Anderson & May, 1978; Anderson, 1978, 1979), then the resultant distribution of parasite numbers/host will differ from that predicted by stochastic models of the population processes which influence the growth of any one sub-population of parasites within a host (i.e. from those listed in Table 1). On intuitive grounds, it would appear that a host death rate which is positively correlated with parasite burden will tend to generate under-dispersion in the distribution of parasites within the total host population. This is indeed the case, but this result is more difficult to establish by

Table 1. *Probability distributions generated by stochastic models of various population processes considered in isolation and in combination (see Bailey, 1964)*

Population process(es)	Rate symbol(s)	Mean population size at time t, $E\{N_t\}$	Variance of N_t, $V\{N_t\}$	Probability distribution of population size, N_t	Variance to mean ratio
Death	μ	$N_0 \exp(-\mu t)$*	$N_0 \exp(-\mu t)[1-\exp(-\mu t)]$	Positive binomial	< 1
Birth	λ	$N_0 \exp(\lambda t)$	$N_0 \exp\{\lambda t\}[\exp(\lambda t)-1]$	Negative binomial	> 1
Immigration	Λ	$N_0+\Lambda t$	$N_0+\Lambda t$	Poisson	1
Emigration	γ	$N_0 \exp(-\gamma t)$	$N_0 \exp(-\gamma t)[1-\exp(-\gamma t)]$	Positive binomial	< 1
Immigration and death	Λ, μ	$N_0 \exp(-\mu t)+(\Lambda/\mu)[1-\exp(-\mu t)]$	$N_0 \exp(-\mu t)+(\Lambda/\mu)[1-\exp(-\mu t)]$	Poisson	1
Birth and death	r, where $r=\lambda-\mu$	$N_0 \exp(rt)$	$[N_0(\lambda+\mu)/r]\exp(rt)[\exp(rt)-1]$	Over-dispersed in form, similar to the negative binomial	> 1
Immigration, birth, death and emigration (For $N_0=0$ at time $t=0$)	$\Lambda, \hat{r}$ where $\hat{r}=\lambda-\mu-\gamma$	$(\Lambda/\hat{r})[\exp(\hat{r}t)-1]$	$(\Lambda/\hat{r})[\exp(\hat{r}t)-1][1+(\lambda/\hat{r})[\exp(\hat{r}t)-1]]$	Negative binomial	> 1

* N_0 = initial population size at time $t=0$.

analytical procedures. However, we provide numerical support for this suggestion in the following section, by means of Monte Carlo simulation methods (Pielou, 1969). The precise degree of under-dispersion generated by parasite-induced host mortalities will of course depend on their severity. If the immigration rate of parasites into the hosts is high and the parasite-induced death rate low, then the resulting distribution will be almost Poisson in form. Alternatively, if the immigration rate is low and the death rate high, the resultant distribution will have a variance to mean ratio much less than unity. Other factors, in addition to parasite-induced host mortality, can induce under-dispersion. Specifically, density-dependent processes, whether acting on the infection (immigration) rate, Λ, or the natural parasite mortality rate μ, can, independent of the degree or nature of host mortality, induce variance to mean ratios less than unity. Here again, these results are difficult to establish by analytical means but are easily demonstrated by simulation methods (see the following section).

As mentioned earlier, the prime cause of over-dispersion in the distribution of parasite numbers within a host population will be stochastic factors of an environmental, as opposed to a demographic, nature. Such factors include changes in physical parameters of the environment, either through time or space and, most importantly, differences in host susceptibility to infection whether induced by behavioural differences, genetic factors or varying past experiences of infection. Variability in past exposure to infection and the genetic constitution of the host population can also influence the death rates of parasites and hosts such as to create heterogeneity in the dispersion of parasites within the host population. These environmental factors (taking the environment to include both the host and the host's habitat) act to make the population rate parameters, which control the size of any one parasite sub-population, random variables rather than constants. In other words the rate parameters, such as the immigration and death rates, will vary in time, in space and between different sub-populations of parasites within the total host population. Here, the term sub-population refers to the number of parasites within an individual host as opposed to the total parasite population within the entire host population. An immediate consequence of heterogeneity in these rate processes, is the generation of over-dispersion in the distribution of parasite numbers/host. To briefly illustrate this point consider a sub-population of parasites controlled by immigration and death processes. We already know that if the rates are constant in time and between hosts, the resultant distribution of parasite numbers/host will be Poisson in form (see Table 1). However, if the immigration rate Λ, varies between hosts such that the distribution of the value of Λ within the host population is itself Poisson in form, then the resultant distribution of parasite numbers/host, within the total host population, will be Neyman Type A in form (see Pielou, 1969). This distribution is over-dispersed in character, where the variance is greater than the mean. This result is established in Appendix B.

It is clear from the preceeding discussion that the observed distribution of parasite numbers within a host population at any one point in time will be the resultant of two sets of opposing forces, one acting to decrease the degree of dispersion and the other acting to increase the degree of dispersion. Fig. 1 presents a diagrammatic summary of this concept, listing the various factors which

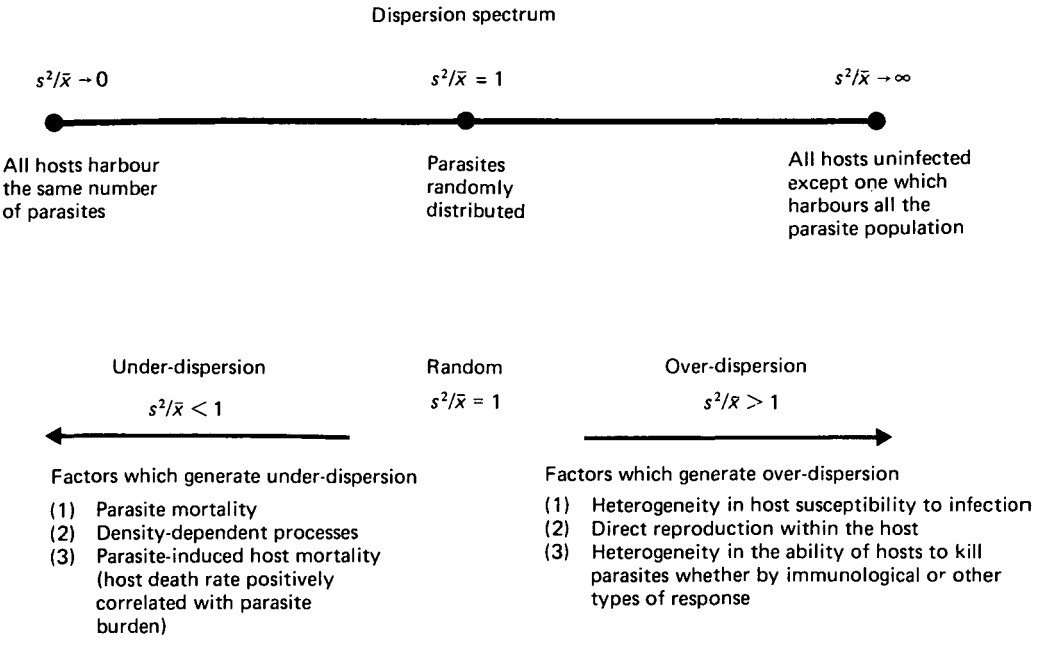

Fig. 1. Diagrammatic representation of a dispersion spectrum denoting the factors which create under-dispersion and the factors which generate over-dispersion.

generate under-dispersion and over-dispersion. These are presented in the framework of a dispersion spectrum. The two conceptual limits of this spectrum are (1) infinite over-dispersion where all hosts are uninfected bar one which harbours all the parasites and (2) zero dispersion where all hosts harbour the same number of parasites. Somewhere between these two limits lies the point at which the parasites are randomly distributed within the host population.

We wish to stress that an observed distribution is not a static entity, but is truly dynamic as a consequence of changes in the relative magnitudes of the factors which determine the degree of dispersion, both through time and in space. The measurement of changes in parasite dispersion may therefore provide additional information concerning the factors which control the population dynamics of host–parasite interactions, over and above the more commonly measured statistics such as the mean parasite abundance (average intensity of infection) and the prevalence of infection. The interpretation of changes in this latter statistic, for example, is critically dependent on a knowledge of changes in the pattern of parasite dispersion. Some empirical examples, to illustrate the dynamic nature of parasite dispersion patterns through time, are presented in Fig. 2.

In order to interpret what such changes imply in terms of the dynamics of host–parasite interactions it is clearly necessary to pursue in more detail the influence of various combinations of population processes and environmental factors on parasite distributions. This problem is pursued in the following section.

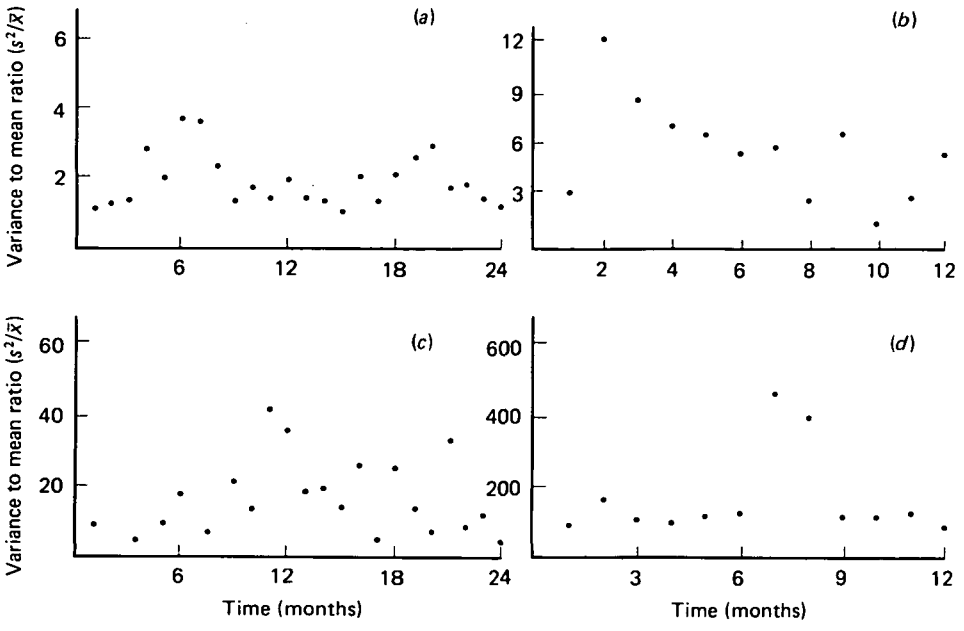

Fig. 2. Some examples of temporal changes in the dispersion pattern of parasite numbers/host. Graph (*a*), *Lepeophtheirus pectoralis* on *Platichthys flesus* (data from Boxshall (1974)). Graph (*b*), *Caryophyllaeus laticeps* in *Abramis brama* (Anderson, 1974). Graph (*c*) *Diplostomum gasterostei* in *Perca fluviatilis* (Kennedy & Burrough, 1977). Graph (*d*) *Diplozoon paradoxum* on *Abramis brama* (Anderson, unpublished data).

MONTE CARLO SIMULATION MODELS

To simplify the following analysis we restrict our attention to the accumulation and loss of parasites within a newly born cohort of hosts of identical age. We then follow the dispersion pattern of parasites as this cohort of hosts ages through time. To further simplify matters we focus on just one segment of a parasite's life-cycle, namely the parasite sub-population within the host population, without reference to the other transmission stages in the cycle. The sub-populations, however, may be adult parasites in the definitive host or larval parasites in an intermediate host. We are specifically concerned with helminth parasites, partly as a result of the comparative wealth of empirical data concerning the ecology of these species, and hence we envisage the growth of sub-populations to be the consequence of immigration and death processes as opposed to birth and death processes. In other words we assume that the parasites do not multiply within the host to directly increase their population size.

Because of the difficulty of obtaining analytical results from non-linear population models which encorporate stochastic elements created by both demographic and environmental factors we employ Monte Carlo simulation techniques in order to analyse model behaviour (Pielou, 1969).

Basic model

As a starting point, we consider a parasite sub-population, of size N, subject to a constant immigration rate Λ and a constant *per capita* death rate μ. At all times

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there are exactly two possibilities for the next event: it may be the arrival of an immigrant, in which case sub-population size will increase from N to $N+1$; or it may be the death of an established parasite, in which case sub-population size will decrease from N to $N-1$. The net rate at which events occur may be expressed as follows:

$$N \to N+1 = \Lambda, \tag{1}$$

$$N \to N-1 = \mu N. \tag{2}$$

These events are mutually exclusive and exhaustive and thus the event probabilities, Pr, are of the form

$$\Pr(N \to N+1) = \Lambda/(\Lambda+\mu N), \tag{3}$$

$$\Pr(N \to N-1) = \mu N/(\Lambda+\mu N). \tag{4}$$

The time between events is assumed to have an exponential distribution with parameter, $\Lambda+\mu N$ (see Cox & Miller, 1965; Pielou, 1969). A sequence of events, and the times between each event, may be simulated by the use of a pseudo-random number generator, to mimic changes in the size of a sub-population of parasites within a given host. The simulation may then be repeated a large number of times, with different sequences of random numbers, to mimic the changes in parasite sub-populations with a population of hosts whose size is determined by the number of replicate simulations. We assume that the events that occur in any given host are independent of events occurring in other hosts within the population. So far this procedure is straightforward and an analytical solution is available for this form of process; the resultant probability distribution of parasite sub-population size is Poisson in form (see Table 1). The complication arises, however, as a consequence of the fact that each sub-population may be extinguished at any point in time by the death of the host. In the light of the available empirical evidence we assume that the probability that a host dies is a function of the parasite burden (see Anderson, 1978, 1979; Anderson & May, 1978). For simplicity we assume that this function is linear such that the rate at which an individual host dies, $b(N)$, is given by

$$b(N) = a+\alpha N, \tag{5}$$

where N is the size of the parasite burden or sub-population. The parameter a denotes the *per capita* rate of natural host mortality, where $1/a$ represents host life-expectancy in the absence of infection, and the parameter α measures the severity of the parasite's influence on host mortality.

By following through time a newly born cohort of hosts, of size H_0 at birth, and $H(t)$ at time t, we are only interested in the time between events since only one type of event occurs; namely, a death. For a given host the waiting time to death is taken to have an exponential distribution with parameter, $b(N)N$ (Pielou, 1969). Note that in any given simulation this parameter changes in value through time as events occur in the parasite sub-population. A computer programme was written to carry out Monte Carlo simulations of changes in the size of parasite sub-populations through time and changes in the size of the cohort of hosts under the influence of natural mortality and parasite-induced host mortality. At a sequence of points through time summary statistics were calculated such as the size of the

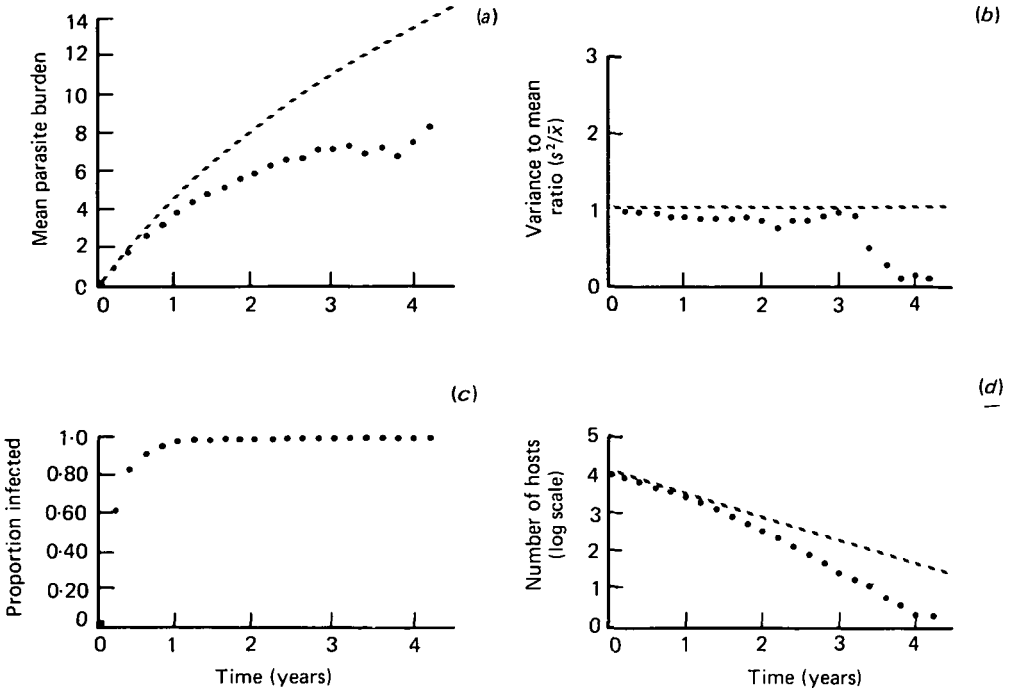

Fig. 3. Simulation experiment of the influence of parasite-induced host mortality (acting at a rate $\alpha = 0{\cdot}3$/parasite/host/year) on various statistics of the distribution of parasite numbers/host. In this experiment the cohort of hosts was set at 10000 individuals at time $t = 0$, the natural mortality rate of the parasite, μ, was set at 0·2/parasite/year and the natural host death rate, a, was set at 0·6/host/year. The immigration rate, Λ, was assumed to be constant for each host in the population, and through time and was set at 5/host/year. Graph (a), temporal behaviour in the mean parasite burden/host. The dots are simulation results, while the dashed line denotes the predicted behaviour of the system in the absence of parasite-induced host mortality ($\alpha = 0$). Graph (b), temporal behaviour of the variance to mean ratio, $s^2/\bar{x}$, where the dots and dashed line are as explained for graph (a). Graph (c), temporal behaviour of the prevalence (proportion of infected hosts) of infection. Graph (d), decay in the number of hosts through time. Dots and dashed line as explained in graph (a).

host population, the mean parasite burden, the proportion of hosts infected and the variance to mean ratio of the number of parasites/host. Various modifications were made to the basic model, to incorporate the various population parameters as random variables themselves and to encompass density-dependent parasite mortality within any given host.

Factors which generate under-dispersion

A sample output of one simulation experiment, for a cohort of hosts of size 10000 at birth, subject to infection at a constant rate, Λ, and parasite-induced host mortality of the form defined in equation (5), is displayed in Fig. 3. The results presented in this figure confirm the earlier conjecture that parasite-induced host mortality tends to generate under-dispersion ($s^2/\bar{x} < 1$) in parasite numbers/host.

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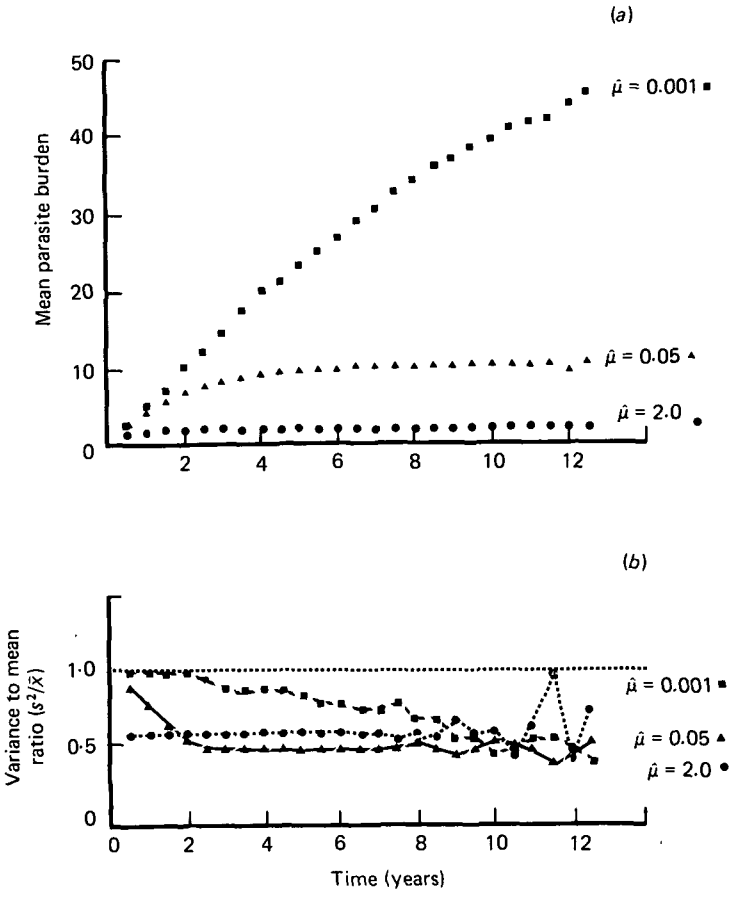

Fig. 4. Simulation experiments of the impact of density-dependent parasite mortality (of the form $\mu(N) = \hat{\mu}N^2$) on changes in the mean parasite abundance and degree of dispersion through time. A series of experiments are shown for different values of the parameter, $\hat{\mu}$, which measures the severity of density-dependent natural parasite mortality ($\hat{\mu} = 0{\cdot}001$, $\hat{\mu} = 0{\cdot}05$, $\hat{\mu} = 2{\cdot}0$). The other parameters were set at $\alpha = 0$, $a = 0{\cdot}6$ and $\Lambda = 5{\cdot}0$ (units of α are/parasite/host/year while the units for a and Λ are/host/year). The value of Λ was assumed to be constant within the host population (host cohort size at time $t = 0$, was set at 10000).

In Fig. 3*a* and *b*, for example, the dashed line denotes the behaviour of the model in the absence of parasite-induced host mortality, while the dots represent the simulated results in which the parasite influences the survival rate of the host. A comparison of these two sets of results reveals the degree of depression of both parasite abundance (Fig. 3*a*), and the degree of dispersion (Fig. 3*b*), generated by parasite-induced host mortality.

Similar patterns can be created when the parasite is non-pathogenic to its host (the α in equation (5) equal to zero), provided the immigration rate and/or the natural death rate of the parasite is density dependent. This point is illustrated by the simulation results portrayed in Fig. 4, in which the death rate $\mu(N)$ was assumed to increase with parasite burden as a power law relationship ($\mu(N) = \hat{\mu}N^2$).

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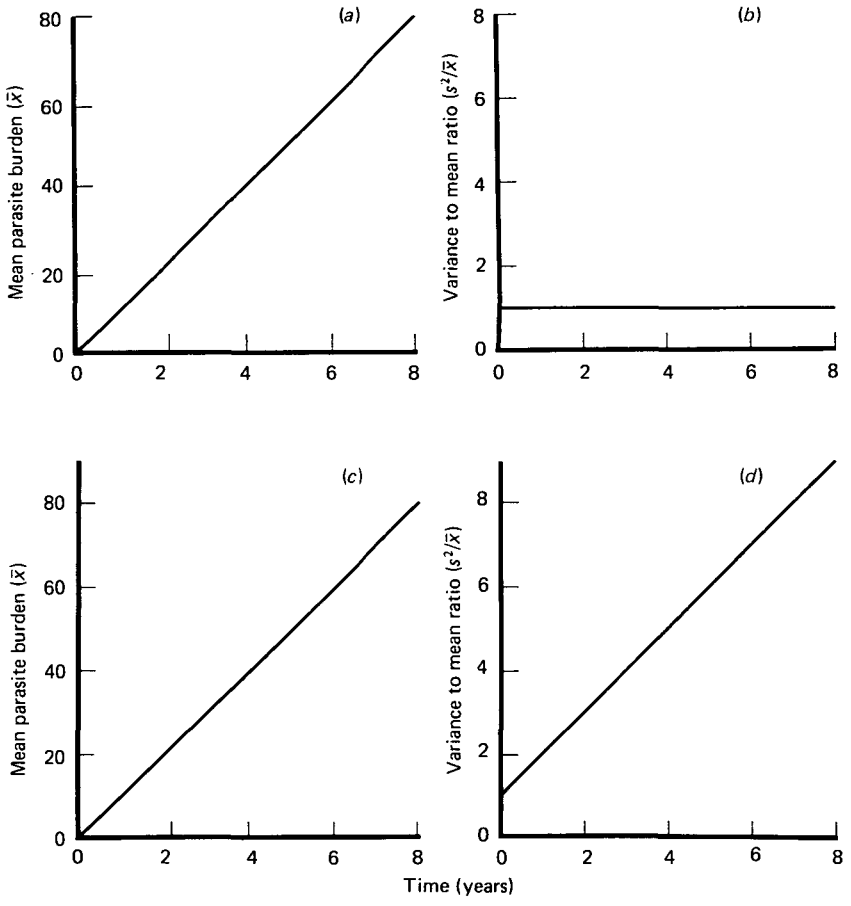

Fig. 5. The influence of heterogeneity in susceptibility to infection on the degree of parasite dispersion ($s^2/\bar{x}$). In graphs (*a*) and (*b*) the parasite population is simply subject to an immigration Λ which is constant within the host population ($\Lambda = 10{\cdot}0$). The mean parasite burden/host is thus $E\{N_t\} = \Lambda t$ (graph (*a*)) with variance to mean ratio equal to unity (graph (*b*)) (see Appendix A). In graphs (*c*) and (*d*) the parasite population is again subject to a constant immigration rate, but this rate is assumed to be a Poisson variate within the host population with mean $\bar{\Lambda}$ ($\bar{\Lambda} = 10{\cdot}0$). The mean parasite burden is thus $E\{N_t\} = \bar{\Lambda}t$ (graph (*c*)) with variance to mean ratio $E\{N_t\}/\mathrm{Var}\{N_t\} = 1 + t$ (graph (*d*)) (see Appendix B). (Other parameter values $\alpha = \mu = a = 0{\cdot}0$).

Factors which generate over-dispersion

In natural populations of hosts the principal factor responsible for the generation of over-dispersion is heterogeneity between hosts in their exposure, susceptibility to infection, or defensive capabilities (specific or non-specific responses). As mentioned earlier the causes of such heterogeneity may be many and varied.

We capture this feature of host–parasite associations in our model by making the assumption that the immigration rate, Λ, is a random variable such that the distribution of its values is Poisson in form with mean $\bar{\Lambda}$. In our model, when calculating the behaviour of each sub-population of parasites through time, a value of Λ is chosen for each host from a Poisson distribution with mean $\bar{\Lambda}$ by the use

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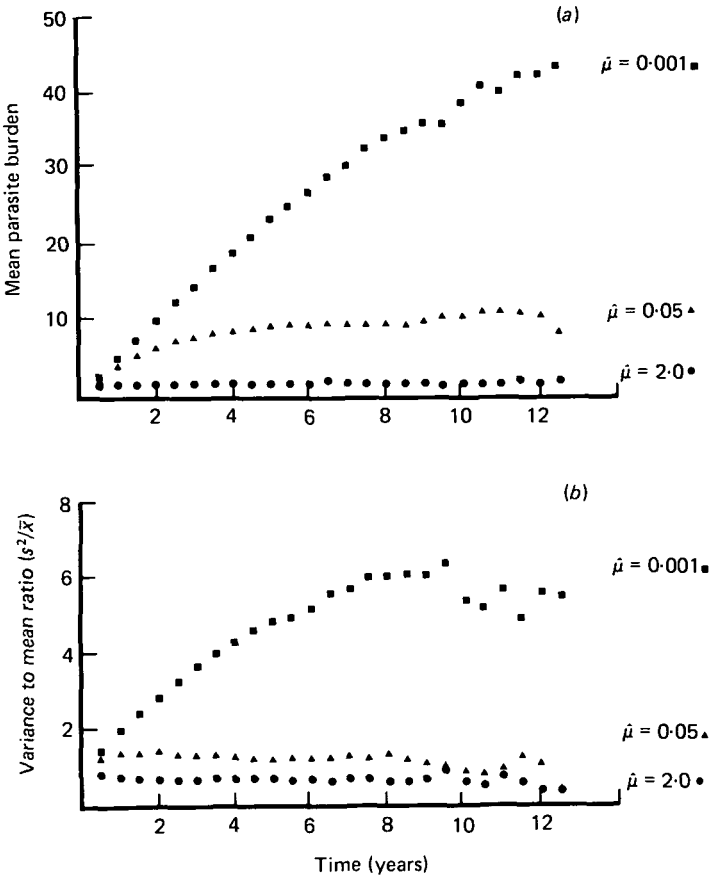

Fig. 6. This figure presents the results of three simulation experiments designed to illustrate the dynamic balance between factors which generate over-dispersion and factors which create under-dispersion. The model employed and the parameter values used are identical to those described in the legend to Fig. 4 except that the immigration rate is assumed to be a Poisson variate with mean $\bar{\Lambda} = 5{\cdot}0$. Heterogeneity in this immigration rate within the host population generates over-dispersion while density-dependent natural parasite mortality (of severity $\hat{\mu}$) creates under-dispersion. The resultant degree of dispersion, measured by the variance to mean ratio, $s^2/\bar{x}$, is set by the relative magnitudes of the two opposing forces (graph (*b*)) as is the mean abundance, $\bar{x}$/host (graph (*a*)). The host cohort size at time $t = 0$ was set at 10000.

of a pseudo-random number generator. A simple illustration of the influence of such heterogeneity on the variance to mean ratio ($s^2/\bar{x}$) of parasite numbers/host is displayed in Fig. 5. The situation presented in this figure is one in which hosts accumulate parasites at a mean rate, $\bar{\Lambda}$, and die at a rate, a, which is independent of parasite burden. If the immigration rate Λ is constant for each host (in the absence of host mortality) then the distribution of parasites/host is Poisson with a variance to mean ratio of unity (Table 1 and Fig. 5*a* and *b*). When Λ is a Poisson variate the distribution is Neyman Type A in form with a variance to mean ratio of $s^2/\bar{x} = 1 + t$, where t = time (Appendix B, Fig. 5*c* and *d*). Since $s^2/\bar{x} > 1$, the distribution is over-dispersed.

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The observed degree of dispersion of parasite numbers/host (measured by the variance to mean ratio, $s^2/\bar{x}$) within any given cohort of hosts, will depend on the relative magnitudes of the factors which generate over-dispersion and under-dispersion. This principle is illustrated in Fig. 6, where simulation results are presented of the accumulation of parasites by a cohort of hosts. The factor responsible for over-dispersion is heterogeneity between hosts in the rate of acquisition of parasites, Λ, while the factor generating under-dispersion is density-dependent natural parasite mortality. The death rate of the host is assumed to be independent of parasite burden. Fig. 6 displays three simulation experiments in which the mean infection rate, Λ, and the natural host death rate, a, are both held constant, while the severity of density-dependent parasite mortality is allowed to vary. The main point to note in this figure is that either under-dispersed or over-dispersed patterns (Fig. 6*b*) can be generated, depending on the severity of density-dependent constraints on parasite survival.

The relationship between variability and mean parasite abundance/host

In an important paper published in 1961, Taylor pointed out that for most, if not all, animal and plant species, variability in abundance between sampling units is empirically related to mean abundance by a simple power law (Taylor, 1961). Specifically, the variance of the numbers/sampling unit, s^2, is related to the mean number/sampling unit, $\bar{x}$, by the empirical relationship

$$s^2 = c\bar{x}^d, \tag{6}$$

where c and d are constants. Taylor (1961), argued that the parameters c and d were constant for a given species and hence represented, in some manner, certain biological characteristics of the species in question. He provided empirical evidence in support of his hypothesis drawn from a very wide variety of ecological studies, of species ranging from virus through to man. In particular, he emphasized the linear relationship observed between log s^2 and log $\bar{x}$ and suggested that the magnitude of the slope of this linear relationship, d, measured a behavioural attribute; namely, the tendency of the species to aggregate with a habitat (Taylor & Taylor, 1977).

The 'empirical law' between variability and abundance, defined by Taylor's equation (equation (6)), certainly provides a good general description of observed patterns (Taylor, Woiwood & Perry, 1978; Taylor & Woiwood, 1980). In many respects, however, it is rather unsatisfactory since its usefulness as a descriptive tool has tended to distract attention from more fundamental questions. For example, why is variability related to abundance? Are the biological or physical determinants of this relationship species-specific? These questions are of very general ecological interest and their discussion is beyond the scope of this present paper. However, we wish to emphasize three points of particular relevance to the study of host–parasite associations. First, from an empirical standpoint, parasites are no exception to the general trend for variability to be related to mean abundance. Specifically, plots on a log scale of observed variances and means of the number of parasites/host, tend to produce highly significant linear relationships (Fig. 7). The second point we wish to make, is that it is our belief that the reason

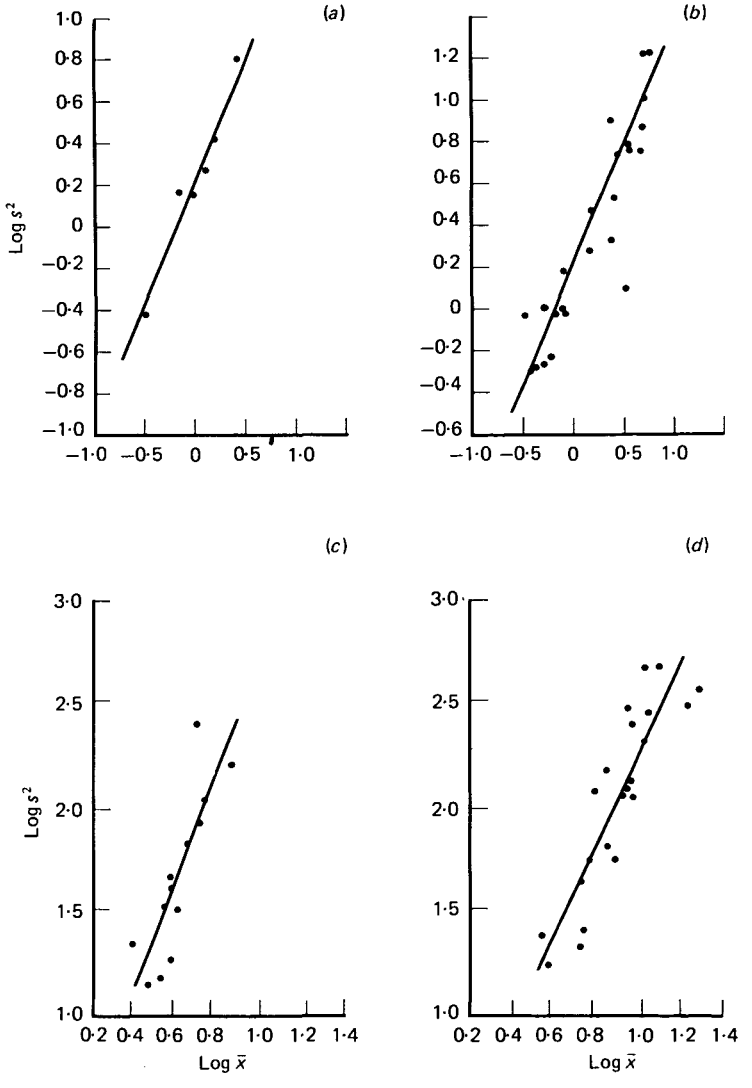

Fig. 7. Examples of the relationship between the mean abundance of parasites/host and the variance plotted on a log scale. The graphs illustrate the linear nature of such relationships. The solid line denotes the best fit linear model of the form $\log s^2 = C + d \log \bar{x}$. The dots are observed values. Graph (*a*), *Polymorphus minutus* in *Gammarus pulex* (data from Crofton, 1971). The slope and the correlation coefficent of the best fit linear model are respectively $d = 1{\cdot}16$, $r = 0{\cdot}96$. Graph (*b*), *Lepeophtheirus pectoralis* on *Platichthys flesus* (Boxshall, 1974) $d = 1{\cdot}18$, $r = 0{\cdot}96$. Graph (*c*), *Bothriocephalus rarus* in *Notophthalmus viridescens* (Jarroll, 1979) $d = 2{\cdot}70$, $r = 0{\cdot}85$. Graph (*d*), *Diplostomum gasterostei* in *Perca fluviatilis* (Kennedy & Burrough, 1977) $d = 2{\cdot}12$, $r = 0{\cdot}88$.

why variability is related to mean abundance is a simple consequence of demographic stochasticity (Anderson, Gordon, Crawley & Hassell, 1982). In particular, the precise relationship will depend on the stochastic contributions of each of the various population rate processes, such as birth, death and immigration rates, which determine the abundance of a given population (see Table 1). For example,

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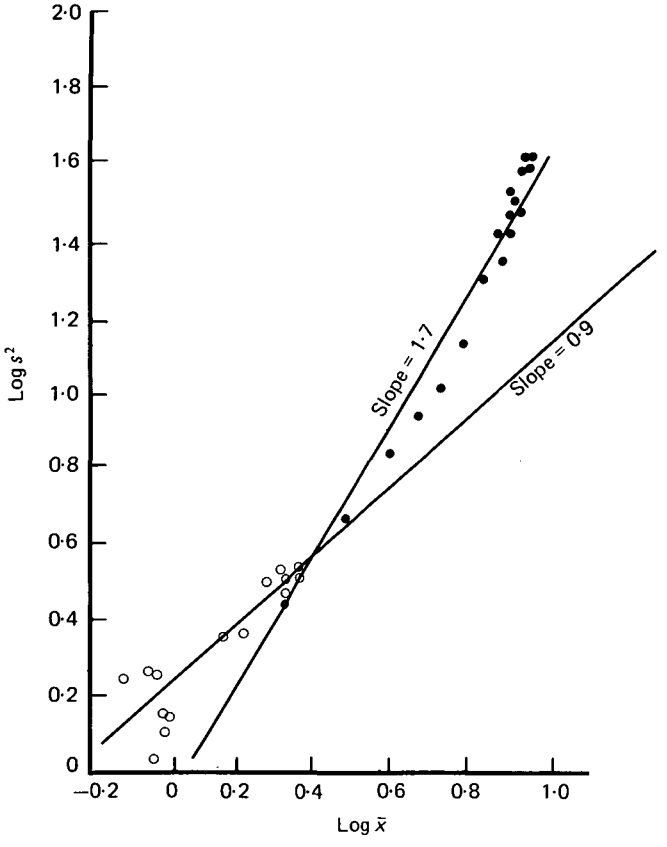

Fig. 8. The influence of the rate of parasite-induced host mortality on the slope of the best fit linear model to the variance (s^2) to mean relationship plotted on a log scale. The results presented are from two simulation experiments in which the immigration rate was assumed to be a Poisson variate ($\bar{\Lambda} = 5{\cdot}0$) and the parameters a and μ were set at 0·6 and 0·0 respectively. In one experiment the value of α was set at 0·9/parasite/host/year which gave r and d values of 0·89 and 0·9 respectively. In the second experiment α was set at 0·1/parasite/host/year which gave r and d values of 0·98 and 1·7 respectively (d is the slope of the best fit linear model and r is the correlation coefficient).

in the case of a population controlled by birth, immigration and death processes, the resultant probability distribution of population abundance at any point in time will be negative binomial in form (Table 1). In such cases, a plot of log s^2 against log $\bar{x}$, will be approximately linear with a slope of roughly 2·0. The final point we wish to make is that the numerical value of the slope of the relationship between log s^2 and log $\bar{x}$ may tend to be species-specific, but not necessarily so. The slope is, to a large extent, set by the dynamic balance between population processes which create under-dispersion and over-dispersion. Such processes, for a given host–parasite association, often vary both in space and through time (i.e. different seasons of the year, see Fig. 2). This point may be simply illustrated by Monte Carlo simulation experiments. For example, Fig. 8 shows the relationship between log s^2 and log $\bar{x}$ for two simulation experiments of infection within a cohort of hosts, in

which the immigration rate, Λ, is a Poisson variate and the host population is subject to natural and parasite-induced host mortality. When the rate of parasite-induced host mortality is high the slope of the best fit linear model to the logs of the variances and means is low ($\alpha = 0{\cdot}9$, slope = 0·9), while conversely, if the rate of mortality is low the slope is high ($\alpha = 0{\cdot}1$, slope = 1·7). For a given parasite species, parameters such as α tend to vary on a seasonal basis and often depend on host age.

The influence of parasite-induced host mortality on dispersion patterns

In this section we return to the question posed in the introduction of this paper. Can observed dispersion patterns of parasite numbers within their host populations provide clues concerning the impact of the parasite on the dynamics of the host population? Specifically we focus on the impact of parasite-induced host mortalities and use our simulation model to examine changes in the dispersion of parasite numbers through time as a cohort of hosts ages. The first general point to emerge from simulation experiments is that given a constant mean infection rate, $\bar{\Lambda}$ through time, parasite-induced host mortality tends to induce curved, or peaked, age-intensity curves (Fig. 9*a*). The maximum mean parasite burden occurs in hosts of intermediary age, with intensity falling off in the older age classes as a consequence of the more rapid death of heavily infected hosts. Simulation studies suggest that such patterns cannot be induced by density-dependent natural parasite mortality acting in isolation of the influence of the parasite on host survival. The second general point, is that concomitant with the decline in mean parasite burden in the older age classes of hosts, the degree of dispersion (as measured by the variance to mean ratio, $s^2/\bar{x}$) also tends to decrease in value (Fig. 9*b*). Note that the severity of decline of both the intensity of infection and the degree of dispersion in the older hosts, is critically dependent on the magnitude of the influence of the parasite on host survival (the value of α). For low values of α the mean intensity will still tend to fall off in older hosts but the degree of dispersion may remain high (Fig. 9).

The practical conclusion to be drawn from these experimental simulations is that peaked age-intensity curves, concomitant with a decline in the degree of dispersion of parasites in the older age classes of hosts, can provide evidence of parasite-induced host mortality where the rate of mortality is positively correlated with parasite burden. This conclusion, however, is subject to a series of important qualifications which are as follows. (1) Peaked, or curved age-intensity curves can of course be generated by age-related changes in the average rate of infection, $\bar{\Lambda}$. In many situations, for example, older hosts may cease to acquire parasites as a consequence of either changes in feeding, changes in habitat utilization or the acquisition of acquired immunity. It is important to note, however, that these factors will not normally create a concomitant decline in the degree of dispersion in older hosts except under the circumstances listed in the following qualification. (2) A decline in the degree of parasite dispersion in the older age classes of hosts may be generated by two factors in addition to parasite-induced host mortalities. These are a reduction in the degree of heterogeneity in the infection rate within older age classes of hosts and the action of acquired immunity on parasite mortality,

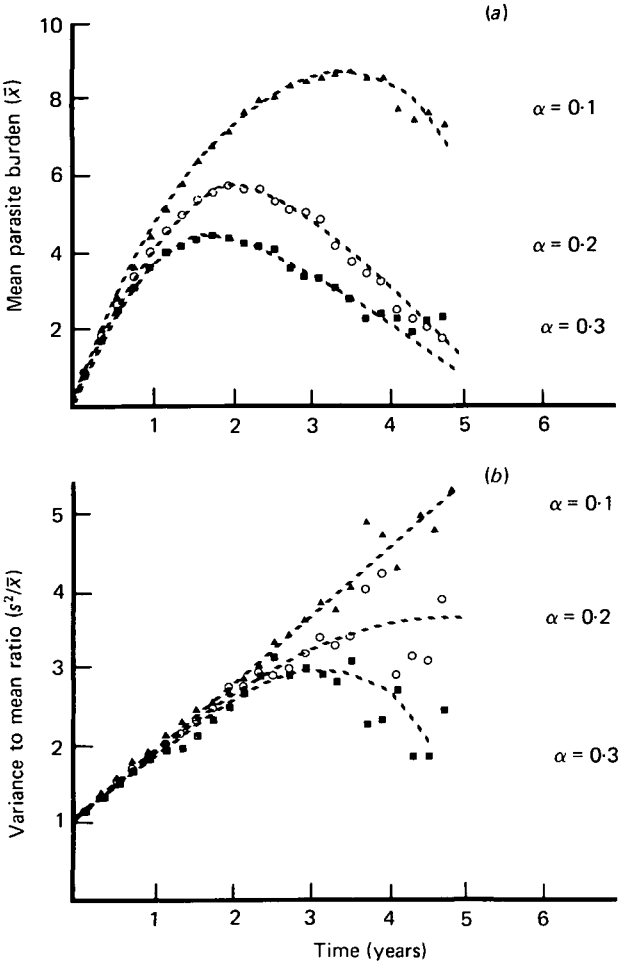

Fig. 9. Simulation experiments of the impact of the rate of parasite-induced host mortality (α) on the temporal behaviour of mean parasite abundance (graph (*a*)) and the degree of parasite dispersion, $s^2/\bar{x}$ (graph (*b*)). Three simulation experiments are presented in which the value of α ranged from 0·1 to 0·3/parasite/host/year. The immigration rate was assumed to be a Poisson variate with mean $\bar{\Lambda} = 5{\cdot}0$. The natural host and parasite death rates (a and μ) were set at 0·6 and 0·0 respectively. The points are the experimental results and the dashed lines are curves fitted by eye to illustrate the general trends. (Cohort size at time $t = 0$ was set at 10000.)

where the severity of such responses is more marked in older hosts. (3) Chance effects can generate very bizarre age-related patterns in mean abundance and the degree of dispersion, when the size of a given cohort of hosts falls to a low level. In practical terms, similar effects can be generated by small sample sizes which themselves, of course, may be a consequence of the comparative scarcity of older hosts in the population. In illustration of this point, Fig. 10 records the results of one simulation experiment in which the rate of parasite-induced mortality was high. As the size of cohort drops to a low level, the chance survival of a few heavily infected hosts results in a rise in the degree of dispersion and average abundance in the old age classes. Such effects will be most marked when the parasite is highly

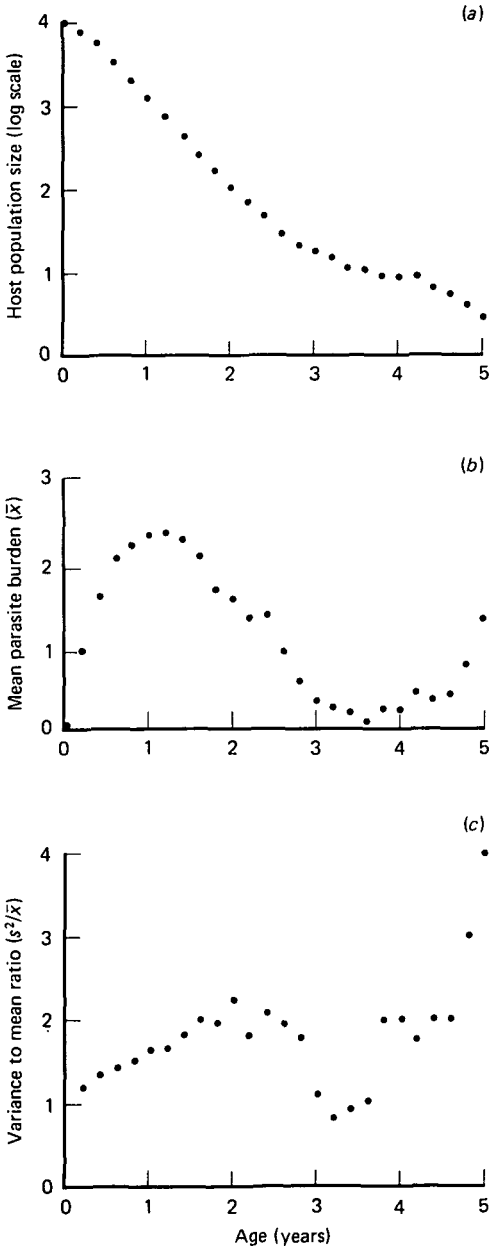

Fig. 10. An example of a simulation experiment which illustrates the impact of chance effects when host density is low, on the trends in mean abundance and the degree of dispersion. The model employed and parameter values used were identical to those described in the legend to Fig. 9 except that the value of α was set at 0·9/parasite/host/unit of time. Graph (*a*) records the decline in host numbers (on a log scale); graph (*b*) shows the temporal behaviour of the mean parasite abundance and graph (*c*) shows the temporal behaviour of the degree of dispersion, $s^2/\bar{x}$. Note that when host density falls to a low level (less than 10–20 individuals) both mean abundance and the degree of dispersion start to increase in value. This is a consequence of the chance survival of a few heavily infected hosts.

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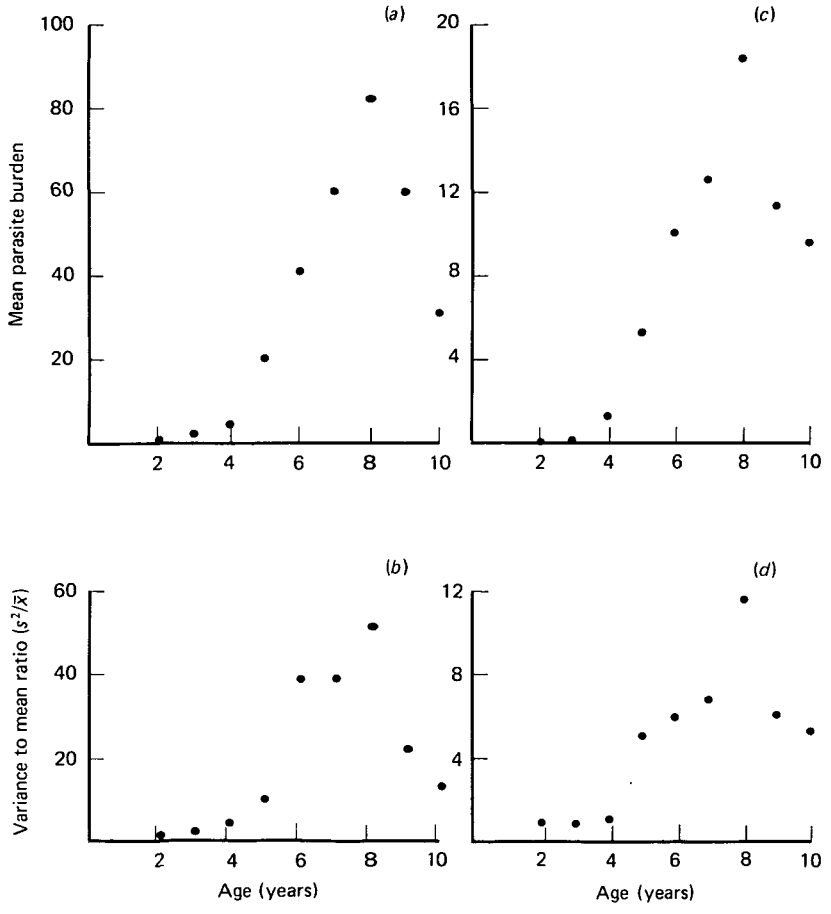

Fig. 11. Changes in the mean abundance and the variance to mean ratios of the plerocercoids of *Diphyllobothrium* in the char *Salvelinus alpinus* with host age. Graphs (*a*) and (*b*), *D. dendriticum* (Henricson, 1977). Graphs (*c*) and (*d*), *D. ditremum* (Henricson, 1977).

pathogenic (α large), since the number of hosts surviving to reach old age will tend to be low. The recognition of the significance of chance effects when host sample size is small, is of major importance to the interpretation of trends in dispersion, intensity and prevalence in field studies of natural populations of hosts and parasites.

Generally speaking, in view of these qualifications, it is clear that it will be difficult to detect the presence of parasite-induced host mortality from field data concerning parasite abundance and dispersion. There are, however, certain types of host–parasite associations in which the qualifications outlined above are of limited significance. Specifically, we have in mind certain larval helminth parasites of vertebrate and invertebrate species, such as metacercarial digenean stages or larval tapeworms in fish hosts and larval acathocephalans or tapeworms in arthropod hosts. As a broad generalization, the life-spans of such parasites tend to be long in relation to that of their host such that parasite losses within their

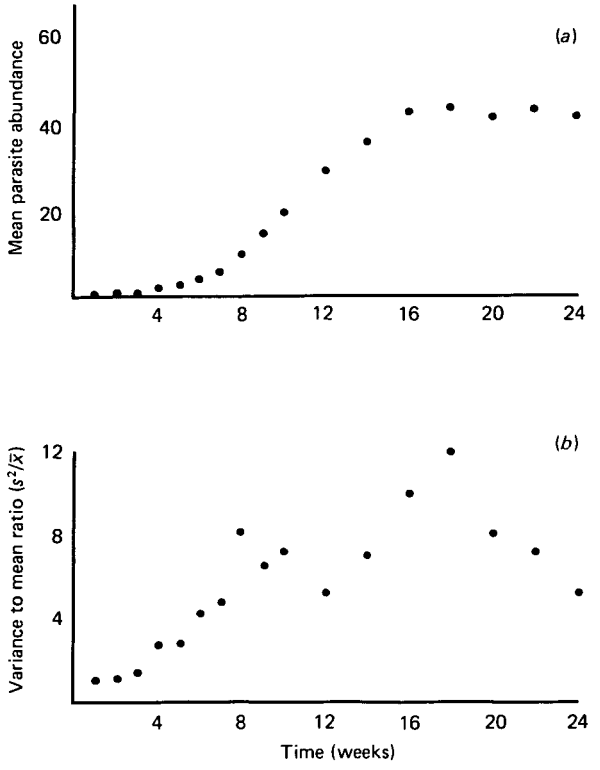

Fig. 12. Changes in the distribution of the tetracotyle *Apatemon gracilis* in the stickleback *Culaea inconstans* with time. Graph (*a*), mean abundance; graph (*b*), variance to mean ratios (Gordon & Rau, 1982).

populations are principally the result of host death (which may or may not lead to transmission to the next host in the life-cycle). Parasite mortality as a result of host responses may occur but, in general, dead and encapsulated parasites are still observable on dissection of the host and, as such, are recorded in the measurement of parasite abundance. In addition, in many of these associations parasites are acquired throughout the life-span of the host, such that the sub-population size within a host of any given age is primarily controlled by immigration, while the total parasite population is controlled by average infection rates and death rates induced by host mortality. One example of such an association is that between the plerocercoids of *Diphyllobothrium* and the fish intermediate host *Salvelinus alpinus*. Data obtained by Henricson (1977) in a field study of the ecology of this association revealed patterns in the mean abundance and dispersion of the parasite within the sampled host population reminiscent of simulation sequences mimicking the action of parasite-induced host mortality. Specifically, the mean abundance and degree of dispersion of the parasite declined in the older age classes of the fish population (Fig. 11). A further example is provided by a field study of the digenean *Apatemon gracilis* in its fish intermediate host *Culaea inconstans* by Gordon & Rau (1982). In this case, the study is a longitudinal one of a given cohort of hosts, and infection is known to occur

throughout the sampling period. Furthermore, the natural mortality rate of the parasite within the fish host is thought to be negligible. As such, the plateau in mean abundance observed as the fish aged, plus the rise and subsequent fall in the degree of parasite dispersion within the fish samples, strongly suggests that the parasite is an important determinant of the rate of host mortality (perhaps due to selective predation by birds on the more heavily parasitized hosts) (Fig. 12).

DISCUSSION

The simulation studies reported in this paper suggest that changes in the frequency distribution of parasite numbers/host will not necessarily provide clear evidence of mortality within a host population induced by parasitic infection. A decline in the degree of dispersion within older age classes of hosts, concomitant with a decline of mean abundance, will be indicative of the presence of such mortality but does not necessarily provide conclusive evidence. Other factors, such as the age-related acquisition of immunity, may complicate the interpretation of such patterns.

The observed balance, at one point in time, between factors which generate over-dispersion and factors which create under-dispersion is controlled by a number of distinct population processes. Identifying the principal mechanisms which generate such patterns of parasite dispersion is thus not easily achieved simply on the basis of monitoring time-related changes in parasite abundance and distribution within the host population. Ideally, it is necessary to obtain independent evidence concerning the magnitude of one or more of the individual population processes that control the size of parasite sub-populations. Evidence of this type may be provided by the age distribution of parasites within the host population, or by the relationship between population size and parasite mortality. Such evidence is invariably difficult to obtain by field study and hence we are drawn to the conclusion that experimental studies in the laboratory, under controlled and defined conditions, provide the initial means for exploring the factors responsible for generating different patterns of parasite dispersion. Simulation studies can provide invaluable help in the design of such experiments.

One of the most important points raised by the study of probability events in the population dynamics of animal species, concerns the influence of individual population processes on the resultant probability distribution of abundance (Table 1). Aside from the issue of heterogeneity in susceptibility to infection, it is clear from Table 1 that parasite populations, which are controlled by birth, death and immigration processes, will tend to exhibit greater degrees of variability in parasite numbers/host than those simply controlled by immigration and death processes. We might therefore expect that parasites which reproduce directly on or within their hosts, such as monogeneans belonging to the genus *Gyrodactylus*, will exhibit higher degrees of over-dispersion than species such as many cestodes, nematodes and acanthocephalans which do not exhibit direct reproduction. In a similar vein, host–parasite associations which characteristically exhibit low degrees of over-dispersion (the vast majority of observed relationships show over-dispersion rather than random or under-dispersed patterns) will tend to be subject to either tight density-dependent constraints on parasite population growth or a marked degree

of homogeneity in susceptibility to infection within the host population. The former explanation appears much more likely than the latter. Density dependence may be due either to constraints on the rate of infection or parasite survival, or to the impact of parasite-induced host mortality.

The final point we wish to emphasize concerns the consequence of chance events within small populations. If chance plays a major role in the growth or decay of parasite and host populations, as it must do in most habitats, then the detection of general patterns in parasite abundance and distribution is critically dependent on the acquisition of large samples of hosts. In small populations, or small samples of hosts, the same set of population rates may generate very different levels of parasite abundance in two different populations or samples. There is little one can do to overcome this problem since population size will often be small in the older age classes of hosts as a simple consequence of host mortality through time. In such cases, it is clearly important to recognize the vagaries of chance.

APPENDIX A

This appendix outlines the derivation of the probability distribution of parasite numbers/host in the presence and absence of host mortality. We first assume that host mortality does not occur and consider a parasite sub-population within a host, subject to constant rates of immigration and death. We define the size of this sub-population at time t by the discrete random variable $N(t)$. The probability distribution at time t of observing n parasites in a host is defined as

$$Q\{N(t) = n\} = g_n(t) \tag{A 1}$$

and the probability generating function (p.g.f) of this distribution is defined as

$$\pi(z,t) = \sum_n q_n(t)\, z^n. \tag{A 2}$$

If the chance of an immigrant arriving in the time interval Δt is $\Lambda \Delta t$ and the chance of a death occurring in a population of n parasites in the same time interval is $\mu n \Delta t$, then

$$q_n(t+\Delta t) = \Lambda q_{n-1}\Delta t + \mu(n+1)\, q_{n+1}\Delta t + q_n \Delta t(1-\Lambda-\mu n). \tag{A 3}$$

The differential-difference equation for the rate of change of $q_n(t)$ with respect to time is thus:

$$\frac{dg_n(t)}{dt} = \Lambda q_{n-1} + \mu(n+1)\, q_{n+1} - \Lambda q_n - \mu n q_n. \tag{A 4}$$

Following standard lines, the partial differential equation for the p.g.f of $q_n(t)$ can be derived directly from equation (A 3) to give

$$\frac{d\pi(z,t)}{dt} = \Lambda \pi(z,t) - \frac{z\mu\, d\pi(z,t)}{dz}. \tag{A 5}$$

Given the initial conditions $\pi(z,0) = 1$, the solution of this equation is

$$\pi(z,t) = \exp\left[(z-1)\frac{\Lambda}{\mu}[1-e^{-\mu t}]\right]. \tag{A 6}$$

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This is the p.g.f. of a Poisson distribution with mean and variance

$$E\{N\} = V\{N\} = \frac{\Lambda}{\mu}[1-e^{-\mu t}]. \tag{A 7}$$

If the host population is now subject to mortality at a *per capita* rate b, which is independent of the random variable $N(t)$, then the differential-difference equation for the probability $g_n(t)$ is identical to that defined in equation (A 4). The resultant distribution of parasite numbers/host is therefore Poisson in form, with the same mean as defined in equation (A 7).

APPENDIX B

This appendix outlines the derivation of the probability distribution of parasite numbers/host when the infection rate is itself a discrete random variable of Poisson form. We define this random variable as I with mean $\bar{\Lambda}$ which is assumed to be constant through time. The moment generating function (m.g.f.) of this random variable is defined as

$$M(\theta) = e^{-\bar{\Lambda}} e^{\bar{\Lambda} e^{\theta}}. \tag{B 1}$$

We assume that the chance of an immigrant arriving in the time interval Δt is $\bar{\Lambda}\Delta t$ and the chance of death occurring in a sub-population of n parasites in the same time interval is $\mu n \Delta t$. Given that when the infection rate I is a constant, the probability distribution of parasite numbers/host is Poisson (as derived in Appendix A) then when I is a random variable of Poisson form itself, the new p.g.f. of the probability of observed n parasites at time t, $S(z,t)$, is given by

$$S(z,t) = E\left\{ \exp\left[\left(\frac{I}{\mu}(1-e^{-\mu t})\right)(z-1)\right]\right\}. \tag{B 2}$$

With the m.g.f. of I as defined in equation (B 1) then

$$S(z,t) = e^{-\bar{\Lambda}} \exp\left[\bar{\Lambda} \exp\left[\frac{1}{\mu}(1-e^{-\mu t})(z-1)\right]\right]. \tag{B 3}$$

This is the p.g.f. of a Neyman Type A distribution (see Pielou (1969)) with mean;

$$E\{N\} = \frac{\bar{\Lambda}}{\mu}\left[1-e^{-\mu t}\right] \tag{B 4}$$

and variance

$$V\{N\} = \frac{\bar{\Lambda}}{\mu}[1-e^{-\mu t}]\left[1+\frac{1}{\mu}[1-e^{-\mu t}]\right]. \tag{B 5}$$

Clearly, the variance is greater than the mean and hence the distribution of parasite numbers/host is over-dispersed.

If the death rate, μ, is zero in the above model, equations (B 4) and (B 5) become

$$E\{N\} = \bar{\Lambda}t, \tag{B 6}$$

$$V\{N\} = \bar{\Lambda}t(1+t). \tag{B 7}$$

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