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REGULATION AND STABILITY OF HOST-PARASITE POPULATION INTERACTIONS

I. REGULATORY PROCESSES

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SUMMARY

(1) Several models describing the dynamics of host-parasite associations are discussed.

(2) The models contain the central assumption that the parasite increases the rate of host mortalities. The parasite induced changes in this rate are formulated as functions of the parasite numbers per host and hence of the statistical distribution of the parasites within the host population.

(3) The parameters influencing the ability of the parasite to regulate the growth of its host's population, and the stability of parasite induced equilibria, are examined for each model.

(4) Three specific categories of population processes are shown to be of particular significance in stabilizing the dynamical behaviour of host-parasite interactions and enhancing the regulatory role of the parasite.

These categories are overdispersion of parasite numbers per host, nonlinear functional relationships between parasite burden per host and host death rate, and density dependent constraints on parasite population growth within individual hosts.

INTRODUCTION

Eucaryotic parasites of one kind or another play a part in the natural history of many, if not most, animals. Man is not exempt: considering helminth parasites alone, roughly 200 million people are affected by the trematode species which cause schistosomiasis and 300 million by the filarial nematode parasites. A multitude of other internal and external parasites of man produce effects ranging from minor irritations to major diseases.

The relation between populations of such parasites and their hosts can be regarded as a particular manifestation of the general predator-prey interaction. Predator-prey theory has received much attention since the work of Lotka and Volterra in the 1920s, and all contemporary ecology texts give a lot of coverage to the subject. This work, however, tends to draw its inspiration, and its empirical basis, from the world of moose and wolves (with differential equations for continuously overlapping generations) or of arthropod predators and their prey (with difference equations for discrete, non-overlapping generations); see, e.g. the reviews by May (1976, ch. 4.) and Hassell (1976). That is, the extensive predator-pray literature pertains mainly to situations where (at least in effect) the predators kill and eat their prey.

Analogous studies of the special kinds of predator-prey relations that are of parasito-

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logical interest are comparatively few. Thus relatively little is known about the effects that parasites may have upon the population dynamics of their hosts.

In this paper we explore the dynamical properties of some simple models which aim to capture the essential biological features of host-parasite associations. In analogy with earlier work on arthropod systems (Hassell & May 1973; Hassell, Lawton & Beddington 1976; Beddington, Hassell & Lawton 1976), we have tried to build the models on biological assumptions that are rooted in empirical evidence.

The paper is organized as follows. After giving a more precise definition of what we mean by the term 'parasite', we outline the biological assumptions upon which the 'Basic model' rests, and expound the dynamical properties of this model. The Basic model assumes parasites to be distributed independently randomly among hosts; various patterns of non-random parasite distribution are then discussed, and introduced into 'model A', which is then investigated. The effects of varying the way the host's mortality depends on the density of parasites ('model B'), and of introducing density dependence into the parasites' intrinsic death rate ('model C') are explored. The following paper (May & Anderson 1978) goes on to discuss various destabilizing influences and encompasses the interaction between host and parasite reproduction rates, transmission factors, and time delays.

Throughout, the text aims to be descriptive with results displayed graphically and the emphasis on the biology of host-parasite associations. The mathematical analysis of the stability properties of the various models is relegated to appendices.

THE TERM 'PARASITISM'

Parasitism may be regarded as an ecological association between species in which one the parasite, lives on or in the body of the other, the host. The parasite may spend the majority of its life in association with one or more host species, or alternatively it may spend only short periods, adopting a free-living mode for the major part of its developmental cycle. During the parasitic phase of its life cycle, the organism depends upon its host for the synthesis of one or more nutrients essential for its own metabolism. The relationship is usually regarded as obligatory for the parasite and harmful or damaging for the host. To classify an animal species as parasitic we therefore require that three conditions be satisfied: utilization of the host as a habitat; nutritional dependence; and causing 'harm' to its host.

When one considers such interactions at the population level, the terminology now used for labelling animal associations appears rather confusing and imprecise (see Starr 1975; Askew 1971; Dogiel 1964). This is particularly apparent when one tries to formalize the nature of the harmful effect of a parasitic species on the population growth of its host. These difficulties do not always arise. For example, insect parasitoids as a developmental necessity invariably kill their host, the parasite surviving the death it induced by the adoption of a free-living mode of life in the adult phase. Askew (1971) has termed such species 'protean parasites' since they are parasitic as larval forms and free-living when adult.

Other eucaryotic parasitic organisms such as lice, fleas, ticks, mites, protozoa and helminths exhibit the nutritional and habitat requirements of a parasite but appear to do very little harm to their hosts, unless present in very large numbers. Such species do not kill their hosts as a prerequisite for successful development; indeed, in contrast to parasitoid insects, these organisms are often themselves killed if they cause the death of

their host. Although parasite induced host deaths usually result when heavy infections occur, the precise meaning of the word heavy will very much depend on the size of the parasite in relation to its host, and the niche and mode of life adopted by the parasite within or on the host.

In this paper, we use the term parasite to refer to species which *do not kill their host as a prerequisite for successful development*. This distinguishes parasites from parasitoids.

Parasites exhibit a wide degree of variability between species in the degree of harm or damage they cause to their hosts. At one extreme of the spectrum, parasites merge into the parasitoid type of association with their close relationship (in population terms) with predator-prey interactions. At this extreme, death will invariably result from parasite infection, but in contrast to parasitoids such host deaths will also kill the parasites contained within. At the other end of the spectrum lie the symbiotic forms of association in which the symbiont lives on or in the host with a degree of nutritional dependence akin to a permissive gastronomic hospitality. Species at this end of the spectrum cause negligible, if any, harm to the host even when present in very large numbers.

In terms of their population dynamics, there will be differences between parasites at the two ends of this spectrum; between the parasitoid like parasites and the symbionts. Crofton (1971a, b) has stressed the importance of quantifying these notions, and has suggested that a useful first step lies in the definition of a 'lethal level', which measures the typical number of parasites required to kill a host. As Crofton notes, quantitative information about such lethal levels is hard to come by.

We shall return to these quantitative questions below: for the present we propose that an organism only be classified as a parasite if it has a detrimental effect on the intrinsic growth rate of its host population.

BASIC MODEL: BIOLOGICAL ASSUMPTIONS

We define $H(t)$ and $P(t)$ to be the magnitudes of the host and parasite populations, respectively at time t; the average number of parasites per host is then $P(t)/H(t)$. We assume that the vast majority of protozoan and helminth parasites exhibit continuous population growth, where generations overlap completely. The equations describing the way the host and parasite populations change in time are thus formulated as differential equations.

The basic model is for parasite species which do not reproduce directly within their definitive or final host, but which produce transmission stages such as eggs, spores or cysts which, as a developmental necessity, pass out of the host. This type of parasite life cycle is shown by many protozoan, helminth and arthropod species. In the following paper (May & Anderson (1978) in model E) this basic framework is modified to encompass species, such as some parasitic protozoa, which have a reproductive phase which directly contributes to the size of the parasite population within the host.

We assume that all parasitic species are capable of multiply infecting a proportion of the host population and that the birth and death rates of infected hosts are altered by the number of parasites they harbour. The precise functional relationship between the number of parasites harboured and the host's chances of surviving or reproducing varies greatly among different host-parasite associations. The rate of parasite induced host mortalities may increase linearly with parasite burden or as an exponential or power law function. Some examples of these functional relationships for protozoan, helminth and arthropod parasites of both vertebrate and invertebrate host species are shown in Fig. 1. Sometimes the relationship may be of a more complex form than suggested by these

examples. The parasite, for instance, may act in an all-or-nothing manner where low burdens do not influence the hosts survival chances but at a given threshold burden the death rate rises very rapidly resulting in certain death for the hosts. In general, however, where quantitative rate estimates are available the 'harmful' effect of the parasite is usually of the more gradual forms indicated in Fig. 1.

In the majority of host-parasite associations it appears to be the death rate rather than the reproductive rate of the host which is influenced by parasitic infection. Exceptions to this general pattern are particularly noticeable in the associations between larval digenean parasites and their molluscan intermediate hosts. Many parasitic arthropods also decrease the reproductive power of their hosts, and in certain cases complete parasitic castration occurs.

Accordingly, the majority of our models assume that the parasite increases the host death rate. Attention is given to the population consequences of parasite induced reduction of host reproductive potential (in Model D) in the following paper (May & Anderson 1978).

TABLE 1. Description of the principal population parameters used in the models

Parameter	Description
a	Instantaneous host birth rate (/host/unit of time).
b	Instantaneous host death rate, where mortalities are due to 'natural causes' (/host/unit of time).
α	Instantaneous host death rate, where mortalities are due to the influence of the parasite (/host/unit of time).
λ	Instantaneous birth rate of parasite transmission stages where birth results in the production of stages, which pass out of the host, and are responsible for transmission of the parasite within the host population (i.e. eggs, cysts, spores or larvae) (/parasite/unit of time).
μ	Instantaneous death rate of parasites within the host, due to either natural or host induced (immunological) causes (/parasite/unit of time).
H_0	Transmission efficiency constant, varying inversely with the proportion of parasite transmission stages which infect members of the host population.
r	Instantaneous birth rate of parasite, whose birth results in the production of parasitic stages which remain within the host in which they were produced (/parasite/unit of time).

The two basic equations, for dH/dt and dP/dt, are constructed from several components, each of which represents specific biological assumptions. A summary of our notation is given in Table 1. These components will now be discussed, one by one.

The growth of the host population

We assume that the rate of growth of the host population is simply determined by the natural intrinsic rate of increase in the absence of parasitic infection minus the rate of parasite induced host mortalities. Both the host reproductive rate a, and the rate of 'natural' mortalities b, are represented as constants unaffected by density dependent constraints on population growth. We use the term 'natural' mortalities to encompass all deaths due to causes other than parasitic infection, e.g. predation and senescence.

Our omission of density dependent constraints on host population growth is deliberate. We recognize that in the real world host population growth will be limited by, among other factors, intraspecific competition for finite resources. Since our aim, however, is to provide qualitative insights into the mechanisms by which parasites regulate host population growth, we have excluded the concept of a carrying capacity of the hosts' environment to simplify algebraic manipulations. Such simplification clarifies predictions of biological interest.

We assume that if the parasite fails to control host population growth, exponential increase of the host population occurs until resource limitation results in the gradual approach to a carrying capacity.

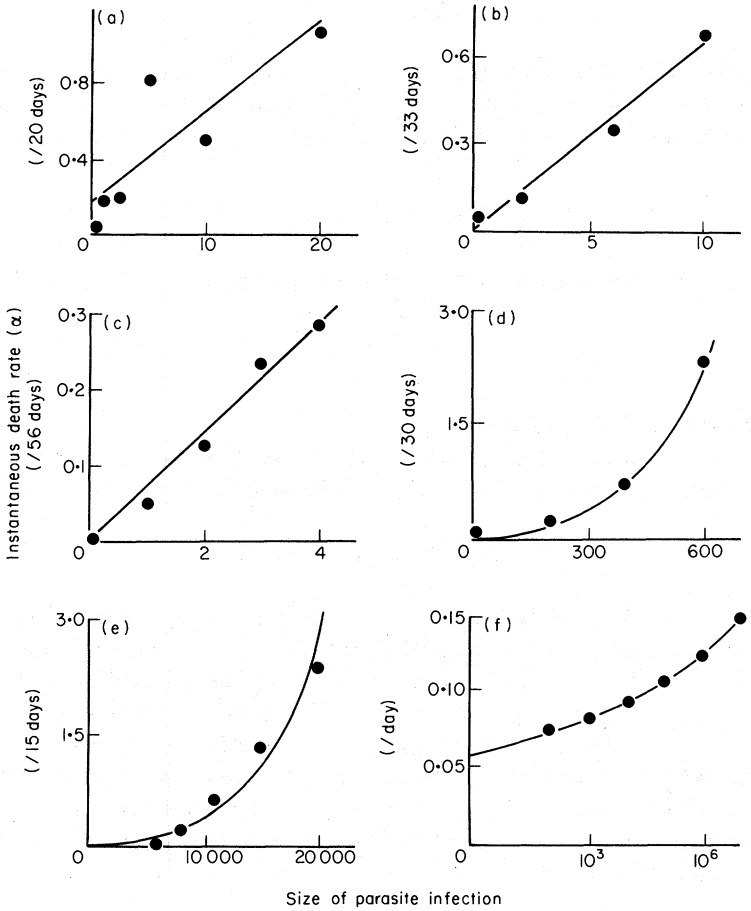

FIG. 1. Some examples of the functional relationship between the rate of parasite induced host mortalities (α) and the parasite burden (i) per host. (The straight lines in graphs (a), (b) and (c) are the least squares best fit linear models and the curves in the graphs (d), (e) and (f) are the least squares best fit exponential models). (a) Snail host *Lymnea gedrosiana* (Annandale and Prashed) parasitized by the larval stages of the digenean *Ornithobilharzia turkestanicum* (Skrjabin) (data from Massoud 1974); (b) aquatic Hemipteran *Hydrometra myrae* (Bueno) parasitized by the mite *Hydryphantes tenuabilis* (Marshall) (data from Lanciani 1975); (c) laboratory mouse parasitized by the digenean *Fasciola hepatica* (L.) (data from Hayes, Bailer & Mitovic 1973); (d) laboratory mouse parasitized by the nematode *Heligmosomoides polygyrus* (Dujardin) (data from Forrester 1971); (e) laboratory rat parasitized by the nematode *Nippostrongylus brasiliensis* (Yokogawa) (data from Hunter & Leigh 1961); (f) laboratory mouse parasitized by the blood protozoan *Plasmodium vinckei* (Vinke and Lips) (data from Cox 1966).

Parasite induced host mortalities

In the Basic model we assume that the rate of parasite induced host mortalities is linearly proportional to the number of parasites a host harbours (see Fig. 1(a)–(c)). The number of host deaths in a small interval of time δt, among those with i parasites, may

then be represented as $\alpha i\ \delta t$, where α is a constant determining the pathogenicity of the parasite to the host; the corresponding death rate among hosts with i parasites is αi. The total rate of loss of hosts in a population of size $H(t)$ is therefore

$$\alpha H(t) \sum_{i=0}^{\infty} i \,.\, p(i).$$

Here $p(i)$ is the probability that a given host contains i parasites. Clearly $p(i)$ will depend on i and on various parameters characterizing the parasite distribution within the host population. The above sum is, by definition, the average number of parasites per host at time t:

$$\sum_{i=0}^{\infty} i \,.\, p(i) \equiv E_t(i) = P(t)/H(t).$$

In short , under the above assumptions the net rate of parasite induced host mortality is

$$\alpha P(t). \tag{1}$$

Parasite fecundity and transmission

The rate of production of transmission stages (such as eggs, spores or cysts) per parasite is defined as λ, leading to a net rate for the toal parasite population of

$$\lambda H(t) \sum_{i=0}^{\infty} i \,.\, p(i) = \lambda P(t). \tag{2}$$

In the case of direct life cycle parasites where only a single species of host is utilized, the transmission stages will pass out of the host into the external environment and will survive in this habitat as resistant stages or free-living larvae awaiting contact with or ingestion by a member of the host population. While in the external habitat, they will be subject to natural mortalities due to senescence or predation and thus only a proportion of those released will be successful in gaining entry to a new host. The magnitude of this proportion will depend on the density of the host population in relation to other 'absorbers' of the transmission stages, and the proportion may be characterized by the transmission factor (cf. MacDonald 1961)

$$H(t)/(H_0 + H(t)).$$

Here H_0 is a constant which, when varied, inversely determines the efficiency of transmission. When $H(t)$ is large and H_0 small, the efficiency approaches unity, where all the transmission stages produced gain entry to the host population. Conversely when $H(t)$ is small and H_0 large only a small proportion are successful.

The net rate at which new parasites are aquired within the host population is thus

$$\lambda P(t) \,.\, H(t)/(H_0 + H(t)). \tag{3}$$

This term contains the assumption that transmission is virtually instantaneous, no time delay occurring due to developmental processes between the birth of a transmission stage and successful contact with a new host. In some parasite life cycles, transmission stages are immediately infective to a new host, but in the majority certain developmental processes have to occur before the stage becomes fully infective. The influence of time delays in the transmission term will be examined as a modification to the basic model in the following paper (Model F in May & Anderson 1978).

The assumptions incorporated in the two equation host-parasite model are most closely linked to direct life cycle parasitic species. The population dynamics of indirect life cycle species can also be interpreted in light of the model's predictions if the population processes acting on the intermediate host or hosts and the parasitic larval stages are subsumed into the transmission term (i.e. into the factor H_0 in eqn (3)). This is obviously a major simplifying assumption, particularly in respect to the time delays which will occur during a parasite's development in its intermediate host (for a review of this subject, see May 1977). The dynamical properties of the basic model with time delays will thus be more akin to the population dynamics of indirect life cycle parasites.

Parasite mortalities

The death rate for parasites within the host population has three components.

First, there are losses due to natural host mortalities. With the intrinsic per capita host mortality rate b, such parasite losses are at the net rate

$$b \,.\, H(t) \sum_{i=0}^{\infty} i \,.\, p(i) = b \,.\, P(t). \tag{4}$$

Second , there are losses from parasite induced host deaths, where the per capita host loss rate (discussed above) is taken to be αi. The consequent net mortality rate of parasites from this cause is

$$\alpha H(t) \sum_{i=0}^{\infty} i^2 p(i) \equiv \alpha H(t) E_t(i^2). \tag{5}$$

Here $E(i^2)$ is the mean-square number of parasites per host, the precise value of which depends on the form of the probability distribution of parasite numbers per host, $p(i)$. That is, $E(i^2)$ depends on the mean parasite load, $P(t)/H(t)$, and also on the parameter(s) that specify this distribution. Appendix 1 lists the values of this 'second moment' for some commonly used discrete probability distributions, giving $E(i^2)$ in terms of the mean parasite load and measures of over- and under-dispersion.

Third, there is a component of the parasite death rate generated by natural parasite mortality within the host. Assuming a constant per capita parasite intrinsic mortality rate μ, these losses make a net contribution of

$$\mu P(t) \tag{6}$$

to the overall parasite mortality rate. These 'natural' parasite mortalities include deaths due to host immunological responses, as well as more conventional losses from parasite senescence.

BASIC MODEL: DYNAMICS

The biological ingredients discussed above can now be drawn together to give two differential equations, one describing the rate of change of the host population,

$$dH/dt = (a-b)H - \alpha P \tag{7}$$

and the other describing the parasite population dynamics,

$$dP/dt = (\lambda PH/(H_0+H)) - (b+\mu)P - \alpha H E_t(i^2). \tag{8}$$

If the parasites are distributed independently randomly among hosts, eqn (8) then becomes (see Appendix 1):

$$dP/dt = P(\lambda H/(H_0+H)-(b+\mu+\alpha)-\alpha P/H). \tag{9}$$

Eqns (7) and (9) readily yield the equilibrium ($dH/dt = dP/dt = 0$) host and parasite population values, H^* and P^*. From eqn (7) the equilibrium mean parasite burden is

$$P^*/H^* = (a-b)/\alpha, \tag{10}$$

whence from eqn (9) H^* is

$$H^* = \frac{H_0(\mu+\alpha+a)}{\lambda-(\mu+\alpha+a)}. \tag{11}$$

Provided the host population's intrinsic growth rate is positive ($a-b>0$), eqn (11) reveals that the parasites are capable of regulating the growth of the host population only if

$$\lambda > \mu+\alpha+a. \tag{12}$$

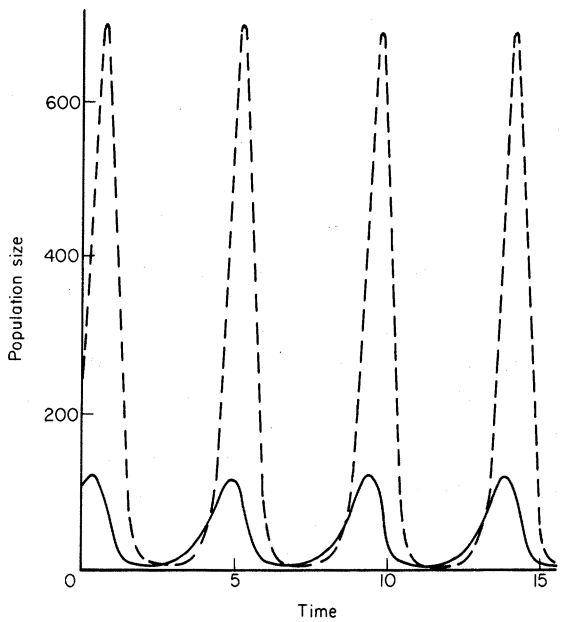

FIG. 2. The trajectories of the host ($H(t)$) and parasite ($P(t)$) populations in time, as predicted by the Basic model (eqns (7) and (9)). The model generates cyclic changes in both $H(t)$ and $P(t)$ and is neutrally stable. Solid line $H(t)$, stippled line $P(t)$. ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\alpha = 0{\cdot}5$, $\lambda = 6{\cdot}0$).

This corresponds to the sensible requirement that the parasite 'birth rate' be in excess of the host birth rate (a) plus parasite death rates (both intrinsic, μ, and due to parasite induced host deaths, α). If this inequality (12) is not satisfied, the host population grows exponentially and will eventually be constrained by other regulatory processes such as finite resources. The parasite will also grow exponentially but at a slower rate than the host population and thus the mean number of parasites per host will tend to zero.

Equations (7) and (9) may be viewed as a general pair of predator-prey equations, and the following comments made. (i) In the 'prey' equation, (7), the 'predators' have a

constant 'attack rate'; compared with the classic, but unrealistic, Lotka-Volterra model, this is a destabilizing feature, for it prevents the predators being differentially more effective at high prey densities. (ii) In the 'predator' equation, (9), we have a kind of logistic equation, with a predator carrying capacity' proportional to the prey density; this has a stabilizing effect (for a more full discussion and review along these general lines, see May (1976), ch. 4). In order to see how these countervailing tendencies are resolved, we need to make a stability analysis of the system of eqns (7) and (9).

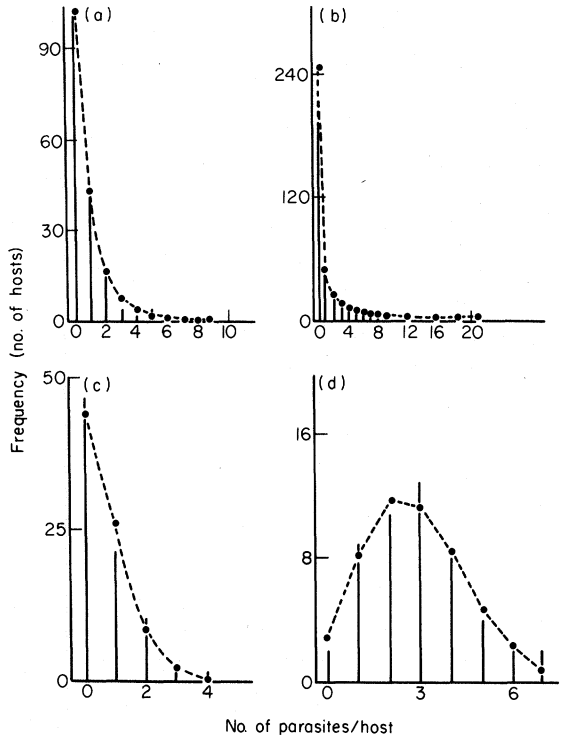

FIG. 3. Some examples of observed frequency distributions of parasite numbers per host which are empirically described by the negative binomial ((a) and (b)) or the Poisson ((c) and (d)) models. (a) The distribution of the tick *Ixodes trianguliceps* (Birula) on a population of the field mouse *Apodemus sylvaticus* (L) (data from Randolph 1975); (b) the distribution of the tapeworm *Caryophyllaeus laticeps* (Pallas) within a population of the bream *Abramis brama* (L.) (data from Anderson 1974a); (c) the distribution of larval stages of the nematode *Cammallanus oxycephalus* (Ward and Magath) in a population of young gizzard shad fish *Dorosoma cepedianum* (Day) sampled in mid summer (data from Stromberg and Crites 1974); (d) the distribution of the nematode *Ascaridia galli* in a population of chickens which had been artificially infected in the laboratory (data from Northam and Rocha 1958).

For this biologically derived pair of equations, a rigorous and fully nonlinear stability analysis may be given elegantly. This is done in Appendix 2. The outcome is surprising: the equilibrium point defined by eqns (10) and (11) is neutrally stable. That is, once perturbed from its equilibrium point the system oscillates with a period determined by the parameters of the model but with an amplitude dictated for ever after by the initial conditions of the displacement. The result is rigorously 'global', which is to say it holds

true for displacements of arbitrary magnitude (provided the initial $H_{(0)}$ and $P_{(0)}$ are positive!). Figure 2 illustrates the dynamical behaviour of such a host-parasite system.

This pathological neutral stability property means that the model defined by eqns (7) and (9) is *structurally unstable*: the slightest alteration in the form of the various underlying biological assumptions will precipitate the system either to stability (disturbances damping back to the equilibrium point) or to instability (oscillations growing

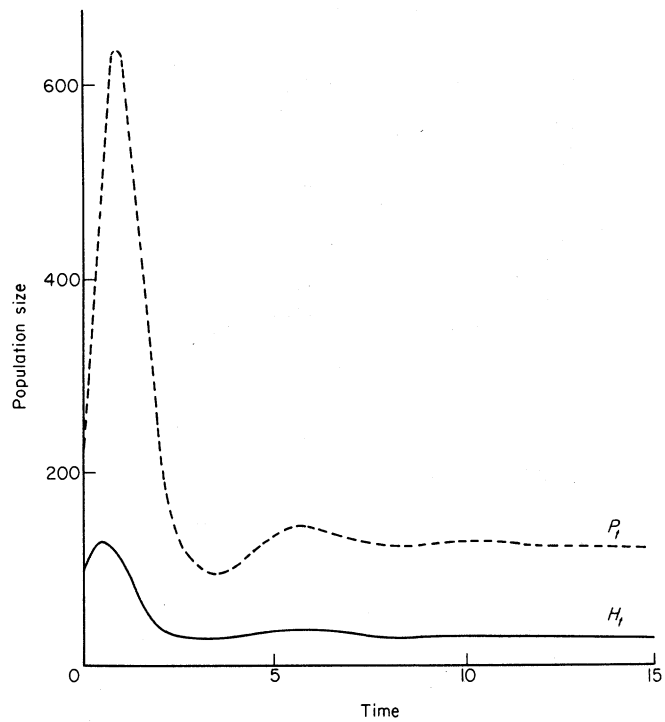

FIG. 4. Overdispersion of parasite numbers per host. The time dependent behaviour of the host ($H(t)$—solid line) and parasite ($P(t)$—stippled line) populations predicted by model A (eqns (7) and (13)). The populations exhibit damped oscillations to globally stable equilibria ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\alpha = 0{\cdot}5$, $\lambda = 6{\cdot}0$, $k = 2{\cdot}0$).

until the host population begins to encounter resource limitation effects as discussed above, whereupon stable limit cycles typically ensue). Such structural instability makes the model biologically unrealistic. The Lotka-Volterra model (although having a different biological and mathematical form from eqns (7) and (9)) also has this pathological and structurally unstable property of neutral stability, for which it has been trenchantly criticized (e.g. May 1975).

The Basic model defined above is nevertheless very useful as a point of departure. We now proceed to modify it by introducing various kinds of density dependences, and of nonrandom parasite distribution among hosts. In each case, the relation between the underlying biology and the overall population dynamics is made clear by comparison with the razor's edge of the Basic model.

DISTRIBUTION OF PARASITES AMONG HOSTS

The parasitological literature contains a great deal of information concerning the distribution of parasite numbers within natural populations of hosts. Almost without exception these observed patterns are over dispersed, where a relatively few members of the host population harbour the majority of the total parasite population. It has become customary for parasitologists to fit the negative binomial model to such data, since this probability distribution has proved to be a good empirical model for a large number of observed parasite distributions. Figure 3 demonstrates the adequacy of this model in describing patterns of dispersion within host populations.

Crofton (1971a) used this observed constancy in the form of parasite distributions as a basis for a quantitative definition of parasitism at the population level. The author based this definition on the large number of parasitological processes which could in theory generate the negative binomial pattern. It is difficult, if not impossible, however, to try to reach conclusions about the biological mechanisms generating a particular distribution pattern by simply examining the resultant observed distribution of parasite numbers per host. A very large number of both biological and physical processes can generate the negative binomial model. Many of these processes have little relevance to the biologies of parasite life cycles while others are important to both parasitic and free-living organisms (Boswell & Patil 1971).

The precise mechanisms giving rise to the patterns shown in Fig. 3(a) and (b) are many and varied. Two major factors are most probably heterogeneity between members of the host population in exposure to infection (Anderson 1976a) and the influence of past experiences of infection on the immunological status of a particular host (Anderson 1976b). Such comments, however, are purely speculative since detailed experimental work is required to suggest generative processes which give rise to observed field patterns.

Consideration of the precise mechanisms which generate a particular pattern is not of overriding importance when examining the qualitative influence of overdispersion on the dynamical properties of a particular host-parasite interaction. We assume that heterogeneity in the distribution of parasite numbers per host is the rule rather than the exception and then proceed to analyse the consequences of such patterns on the population biology of the system.

From the purely phenomenological standpoint, the negative binomial has the virtue of providing a 1-parameter description of the degree of overdispersion, in terms of the parameter k: the smaller k, the greater the degree of parasite clumping. The distribution is discussed more fully in this light by Bradley & May (1977).

It is worth noting, however, that a few reports of random and undispersed distributions of parasites exist in the literature (Fig. 3(c) and (d)). These patterns are often observed either within laboratory populations (Northam & Rocha 1958; Anderson, Whitfield & Mills 1977), or within specific strata of a wild host population such as a particular age class (Anderson 1974b). In other cases random patterns may be observed at a particular point in time where the initial invasion of a host population has been captured by a sampling programme (Stromberg & Crites 1974).

MODEL A: NONRANDOM PARASITE DISTRIBUTIONS

Overdispersed distributions

If the parasites are distributed among the hosts according to a negative binomial, we can use Appendix 1 to get an explicit expression for $E(i^2)$ in eqn (8), which then reads

$$dP/dt = P\{\lambda H/(H_0+H)-(\mu+b+\alpha)-\alpha(k+1)P/(kH)\} \quad (13)$$

As mentioned above, k is the parameter of the negative binomial distribution which gives an inverse measure of the degree of aggregation or contagion of the parasites within the hosts.

The equilibrium host and parasite population values then follow from eqns (7) and (13):

$$H^*/P^* = (a-b)/\alpha \quad (14)$$

$$H^* = \frac{H_0\{\mu+a+\alpha+(a-b)(k+1)/k\}}{\lambda-\{\mu+a+\alpha+(a-b)(k+1)/k\}}. \quad (15)$$

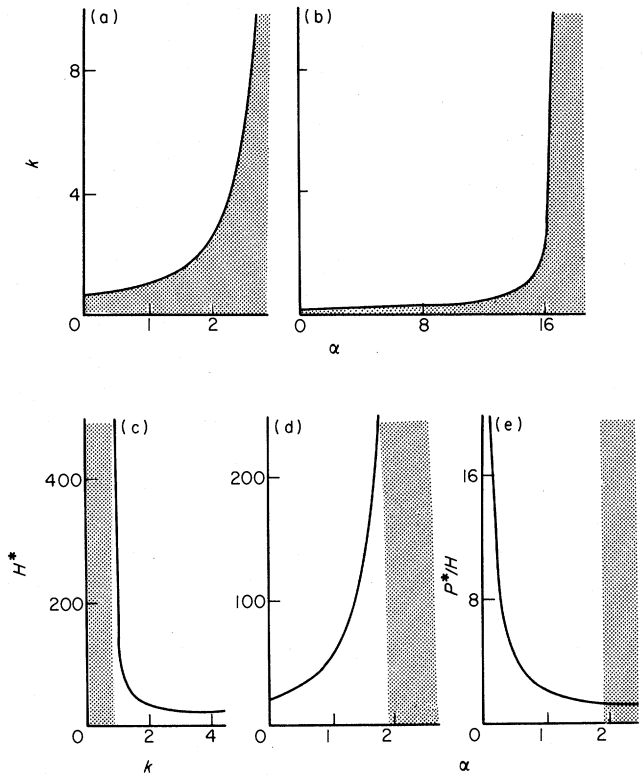

FIG. 5. Model A—overdispersed parasite distributions. Graphs (a) and (b)—the solid lines denote the boundaries in the k and α parameter space which separate regions in which parameter values give rise to globally stable parasite regulated host equilibria (unshaded areas), and unregulated host population growth (shaded areas). These boundaries are shown in terms of k and α for two values of λ. In graph (a) $\lambda = 6{\cdot}0$, and in (b) $\lambda = 20{\cdot}0$. In both (a) and (b), $a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$. In graphs (c), (d) and (e) the solid lines denote the equilibrium size of the host population, H^* (c) and (d)), and the mean parasite burden, P^*/H^* (e) for various values of k and α. The shaded regions denote areas in which the parameter values of the model lead to unregulated host population growth. The parameter values are as indicated for graphs (a) and (b) except that in (c), $\alpha = 0{\cdot}5$, $\lambda = 6{\cdot}0$, and in (d), and (e), $k = 2{\cdot}0$, $\lambda = 6.0$.

This equilibrium point can exist only if

$$\lambda > \mu+a+\alpha+(a-b)(k+1)/k. \quad (16)$$

The stability of the equilibrium is studied in Appendix 3, where it is shown that disturbances undergo damped oscillations back to the equilibrium population values, provided only the aggregation parameter k is finite and positive. (In the limit $k \to \infty$, which corresponds to the Poisson limit, the damping times becomes infinitely large, and we recover the neutral stability of the Basic model.)

Thus if eqn (16) is satisfied, the host population is effectively regulated by the parasites. Figure 4 illustrates the dynamical behaviour of such a system.

If the inequality (16) is not fulfilled, then (as discussed previously) the host population grows exponentially until it encounters environmental carrying capacity limitations. The parasite population will also grow exponentially but at a slower rate than the hosts, and thus the mean parasite burden decreases in size.

The criterion (16) which determines the boundary between host populations that can be regulated by their parasites, and those that cannot, is illustrated in Fig. 5.

Figure 5(a) and (b) are two slices through the k- α parameter space, for two different values of λ (and other quantities held constant); they show that as λ, the production rate of transmission stages, increases a larger range of k and λ values lead to a parasite regulated host equilibrium. When the parasite's distribution is highly overdispersed ($k \to 0$), regulation is difficult to achieve for any value of the rate of parasite induced host mortalities (α). As α increases, the degree of overdispersion must decrease, otherwise too many parasites are lost due to parasite induced host mortalities.

An indication of the influence of α and k on the size of the equilibrium host and parasite populations, H^* and P^*, is given in Figure 5 (c), (d) and (e). Figure 5 (d) appears odd at first glance: an increase in the rate of parasite induced host mortality makes for an increase in H^*. The reason is that those hosts which die from parasite infection harbour above average parasite burdens; such deaths thus have relatively more effect on the parasite population than on the host population. As α increases, this effect becomes increasingly pronounced, leading to a decrease in the mean parasite burden per host (Fig. 5(e)) and an increase in H^* (Fig. 5(d)). Eventually, for α sufficiently large, the parasites can no longer regulate the host population. Notice also that as k increases, the parasites are spread more evenly, and hence the net rate of loss of hosts due to parasite induced mortality rises; i.e. H^* decreases as k increases (Fig. 5(c)).

Quantitative estimates of the parameters in eqn (16) are not easy to come by for natural populations.

Table 2 lists reported values of k for a variety of parasite species. The numbers show that many parasites, particularly helminths, are highly overdispersed within their host populations, the majority of values tending to be less than 1·0. This typically high degree of overdispersion tends to confer stability; it also suggests that net losses from host populations due to parasite infections may be low, since only a few hosts harbour heavy infections. This is a result of the first importance, to which we will return in the concluding discussion.

The rate of production of transmission stages by the parasite, λ, must exceed the sum of the hosts reproductive rate, a, plus the host intrinsic growth rate multiplied by $(k+1)/k$, plus μ and α. Particularly if k is small, this requires that the parasite must have a much higher reproductive rate than the host, which accords with traditional beliefs. Reproductive rates of parasitic species, particularly protozoa and helminths, are invariably high and almost without exception very much greater than the host's potential for reproduction. For example, at the top end of the spectrum lie certain namatode species

TABLE 2. Values of the negative binomal parameter k observed in natural populations of hosts and parasites

Taxonomic group of parasite		Parasite species	Host species	Range of k values	Author(s)
Platyhelminthes, Monogenea		*Diclidophora denticulata* (Olsson)	*Gadus virens* (L.)	0·7	Frankland (1954)
	Digenea	*Diplostomum gasterostei* (Williams)	*Gasterosteus aculeatus* (L.)	0·1–0·7	Pennycuick (1971)
	Cestoda	*Caryophyllaeus laticeps* (Pallas)	*Abramis brama* (L.)	0·1–0·5	Anderson (1974a)
		Schistocephalus solidus (Muller)	*Gasterosteus aculeatus*	0·7–2·4	Pennycuick (1971)
Nematoda		*Chandlerella quiscoli* (von Linstow)	*Culicoides crepuscularis* (Malloch)	0·5	Schmid & Robinson (1972)
		Toxocara canis (Werner)	*Vulpes vulpes* (L.)	0·5	Watkins & Harvey (1942)
		Ascaridia galli (Schrank)	*Gallus gallus* (L.)	0·7	Northam & Rocha (1958)
Acanthocephala		*Polymorphus minutus* (Groeze)	*Gammarus pulex* (L.)	0·6–3·1	Crofton (1971a)
		Echinorhynchus clavula (Dujardin)	*Gasterosteus aculeatus* (L.)	0·07–0·5	Pennycuick 1971)
Arthropoda	Copeopoda	*Lepeophtheirus pectoralis* (Muller)	*Pleuronectes platessa* (L.)	0·3–10·0	Boxhall (1974)
		Chondracanthopsis nodosus (Muller)	*Sebastes marinus* (L.)	0·6	Williams (1963)
		Chondracanthopsis nodosus	*Sebastes mentella* (L.)	0·2	Williams (1963)
	Arachnida	*Ixodes trianguliceps* (Birula)	*Apodemus sylvaticus* (L.)	0·04–0·4	Randolph (1975)
		Liponysue bacoti (Hirst)	*Rattus rattus* (L.)	0·2	Cole (1949)
	Insecta	*Pediculus humanus capitis* (L)	*Homo sapiens*	0·14	Buxton (1940)

such as *Haemonchus contortus* (Rudolphi), a parasite of sheep, which is capable of producing 10 000 eggs per day (Crofton 1966).

Estimates of α, the rate of parasite induced host mortalities, are more difficult to extract from the literature. As a rather broad generalization is appears to be widely accepted in the parasitological literature that the majority of protozoan and helminth parasites do not cause the death of many hosts in natural populations. However, where quantitative rate estimates are available from laboratory experiments (Fig. 1), it appears as though many parasite species have the capability of causing high host death rates. This is particularly apparent in parasite life cycles which utilise invertebrate hosts. Infected individuals within a host population have extremely poor survival characteristics when compared with uninfected hosts (Fig. 6). In natural populations, counter to popular opinion, parasites may play a crucial role in regulating the size of their host populations due to high parasite induced host mortality rates, coupled with small k values.

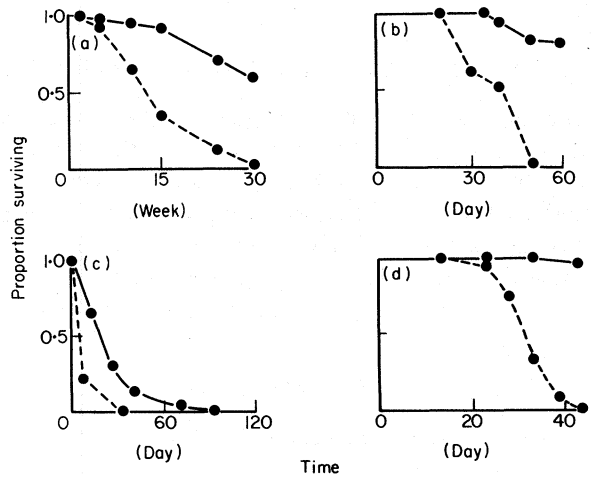

FIG. 6. Some examples of the survival characteristics of infected hosts as compared with uninfected hosts. (a) Snail host *Australorbis glabratus* (Say) infected with larval stages of the digenean *Schistosoma mansoni* (Sambon) (data from Pan 1965); (b) snail host *Lymnaea gedrosina* infected with larval stages of the digenean *Ornithobilharzia turkestanicum* (data from Massoud 1974); (c) mosquito *Aedes aegypti* (L.) infected with microfilariae of the nematode *Dirofilaria immitis* (Leidy) (data from Kershaw, Lavioipierre & Chalmers 1953); (*d*) snail host *Australorbis glabratus* infected with the nematode *Daubaylia potimaca* (Chitwood and Chitwood) (data from Chernin 1962) (solid lines—uninfected hosts, stippled lines —infected hosts).

A present, however, it is difficult to substantiate this conjecture, because of the difficulty in assessing α in natural populations. Our model suggests that a rough, indirect estimate of α may be obtained from eqn (14):

$$\alpha = (a-b)(H^*/P^*). \tag{17}$$

That is, the rate α is equal to the natural intrinsic growth rate of the host population divided by the mean parasite burden P^*/H^*. This is a useful result in itself. It suggests that when overdispersed burdens of parasites per host, with small mean, are observed in the field, then α is likely to be relatively high.

Underdispersed distributions

As mentioned earlier, parasites may occasionally be under dispersed, i.e. more evenly distributed than if they were independently randomly distributed among hosts. Such underdispersed distributions may be described by a positive binomial, with a parameter k' that is inversely proportional to the degree of under-dispersion: in the limit $k' \to 0$, all hosts carry identical parasite burdens.

As shown in Appendix 1, the appropriate version of eqn (8) is obtained by simply replacing k with $-k'$ in eqn (14). It is easily shown (see Appendix 3) that such a system, which amounts to model A with a negative value of k, can have *no* stable equilibrium.

In short, an overdispersed pattern of parasite numbers per host can enable the parasites stably to regulate the host population. The exact form of the pattern of overdispersion is immaterial; the above analysis based on the negative binomial can be repeated for other qualitatively similar distributions such as the Neyman type A, with the same conclusion.

MODEL B: NONLINEAR PARASITE INDUCED MORTALITIES

So far, the parasite induced host mortality rate has been taken to be linearly proportional to the parasite burden (cf. Figs 1(a)–(c)). Often, however, the relationship between host death rate and parasite burden is of a more severe form, as indicated in Fig. 1(d)–(f).

In model B, we examine the dynamical consequences of a more steeply density dependent parasite induced death rate. For specificity, we typify such steeper density dependence by assuming that the parasite induced mortality rate varies as the square of the number of parasites in a host, i.e. as αi^2. By recapitulating the arguments that led to eqns (7) and (8), we have now

$$dH/dt = (a-b)H - \alpha HE(i^2) \tag{18}$$

$$dP/dt = \lambda PH/(H_0+H) - (\mu+b)P - \alpha HE(i^3). \tag{19}$$

The exact form of the average values of i^2 and i^3 depends on the parasite distribution among hosts. In Appendix 3, we indicate the equilibrium population values, and their stability character, under the various assumptions that the parasites are overdispersed (negative binomial), independently randomly distributed (Poisson), and underdispersed (positive binomial).

The nonlinearly severe parasite induced host mortality has two consequences, both of which are illustrated by Fig. 7.

On the one hand, the effect enhances the dynamic stability of an equilibrium point, provided one exists. Thus if the parameters are such that equilibrium populations H^* and P^* exist, then this point is stable for overdispersed or Poisson parasite distributions (the latter in contrast to the Basic model), and also for underdispersed distributions so long as k' is not too small (in contrast to model A, where all such underdispersed cases are unstable).

On the other hand, the domain of parameter space for which the equilibrium exists (i.e. satisfying the analogue of eqns (12) and (16)) is always smaller for model B than for the corresponding model A. This can be seen by comparing Figs 5 and 7.

All this can be explained intuitively. Compared with model A, the more steeply severe parasite induced host mortality makes it relatively harder for the parasite to check the

host population's intrinsic propensity to growth. But if it can do so, this density dependence results in relatively faster damping of perturbations.

MODEL C: DENSITY DEPENDENCE IN PARASITE POPULATION GROWTH

The growth of a parasite population within a single host is often constrained by the influence of density dependent death or reproductive processes. (see Anderson 1976c). Such regulatory mechanisms may be due to either parasite induced host immunological responses or intraspecific competition for finite resources such as space or nutrients

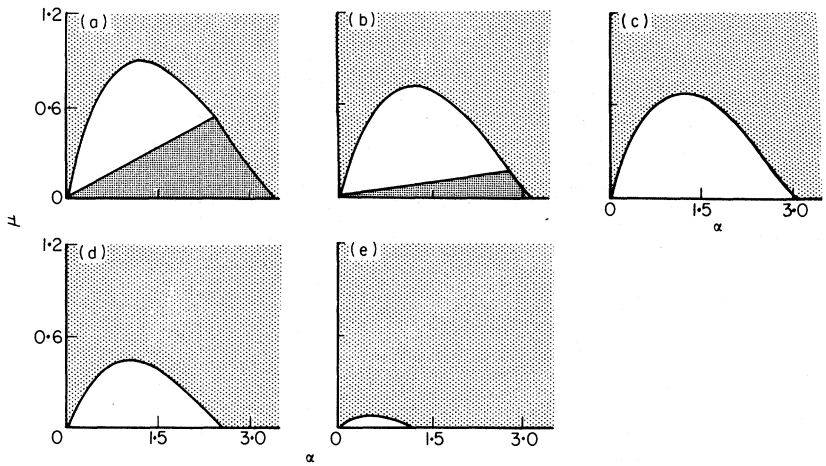

FIG. 7. Model B—non-linear parasite induced mortality rates. The influence of the distribution pattern of the numbers of parasites per host, and the rate of parasite induced host mortalities (α) on the numerical size of the host equilibrium population and its global stability properties. The solid line denotes the values of H^*, while the unshaded regions denote areas of parameter values in which the host population growth is regulated by the parasite and globally stable equilibria arise. The lightly shaded areas define regions where the parasite fails to regulate the growth of the host population; the darkly shaded areas define regions where the parasite regulates host population growth but the equilibria generated are unstable. In (a) and (b), the parasites are underdispersed following the positive binomial model. The value of k' is 6·00 in (a) and 20·0 in (b); in graphs (c) the parasites are randomly distributed (Poisson model); in (d) and (e) the parasites are overdispersed following the negative binomial model where in (d) $k = 8{\cdot}0$ and in (e) $k = 2{\cdot}0$ ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$).

within or on the host. The severity of an immunological response by the host is usually functionally related to the degree of antigenic stimulation received (number of parasites). Such responses, whether humoral or cell mediated, tend to increase the death rate of a parasite population and/or reduce its reproductive potential (Anderson & Michel 1977; Bradley 1971). Some examples of density dependent parasite death rates for helminth species in vertebrate hosts are illustrated in Fig. 8.

The influence of such processes on the dynamical properties of host-parasite interactions may be examined by appropriate modification of the basic eqns (7) and (8). For specificity, we assume the density dependence to be such that natural parasite mortality

occurs at a rate μi^2, in hosts harbouring i parasites. The model is then eqn (7) for dH/dt, together with

$$dP/dt = \lambda PH/(H_0+H) - bP - (\mu+\alpha)HE(i^2). \tag{20}$$

Apart from this modification, the model is exactly as for model A: again the detailed form of eqn (20) will depend on the pattern of parasite distribution.

The dynamical behaviour of model C is elucidated in Appendix 3. For a negative binomial distribution (with parameter k), equilibrium host and parasite populations exist only if

$$\lambda > b+\mu+\alpha+(\alpha+\mu)(a-b)(1+k)/(\alpha k). \tag{21}$$

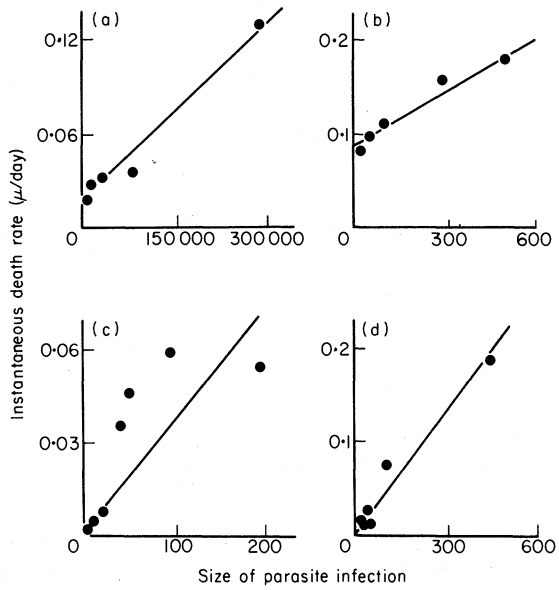

FIG. 8. Some examples of the relationship between the instantaneous parasite 'natural' death rate (μ) and parasite density (i) within individual hosts. The solid lines are the least squares best fit linear models of the form $\mu(i) = \hat{a}+\hat{b}i$ (a) Calves infected with the gut nematode *Ostertagia ostertagi* (Stiles); $\hat{a} = 0{\cdot}0171$, $\hat{b} = 0{\cdot}0308\times10^{-5}$ (rate/day) (data from Anderson & Michel 1977). (b) Chickens infected with the fowl nematode *Ascaridia lineata* (Schneid); $\hat{a} = 0{\cdot}0856$, $\hat{b} = 0{\cdot}00019$ (rate/day) (data from Ackert, Graham, Xolf & Porter 1931). (c) Rats infected with the tapeworm *Hymenolepis diminuta* (Rud); $\hat{a} = 0.00636$, $\hat{b} = 0{\cdot}00032$ (rate/day) (data from Hesselberg & Andreassen 1975). (d) Rats infected with the nematode *Heterakis spumosa* (Schneider); $\hat{a} = 0{\cdot}0265$, $\hat{b} = 0{\cdot}000037$ (rate/day) (data from Winfield 1932).

For independently randomly distributed parasites, the Poisson result follows as the limit $k\to\infty$ in eqn (21); for underdispersion (positive binomial with parameter k') one simply replaces k with $-k'$ in eqn (21). If the equilibrium exists, it is stable for overdispersed or Poisson distributions, and also for underdispersion provided the parameter k' is not too small (explicitly, provided $k' > (\alpha+\mu)/\mu$).

The general pattern of relationship between model C and model A is similar to that between model B and model A. As is made clear by the comparison between eqns (16) and (21), λ must be relatively larger if an equilibrium is to exist (i.e. if parasites are to be able to check host population growth) in model C; this is particularly marked if over-

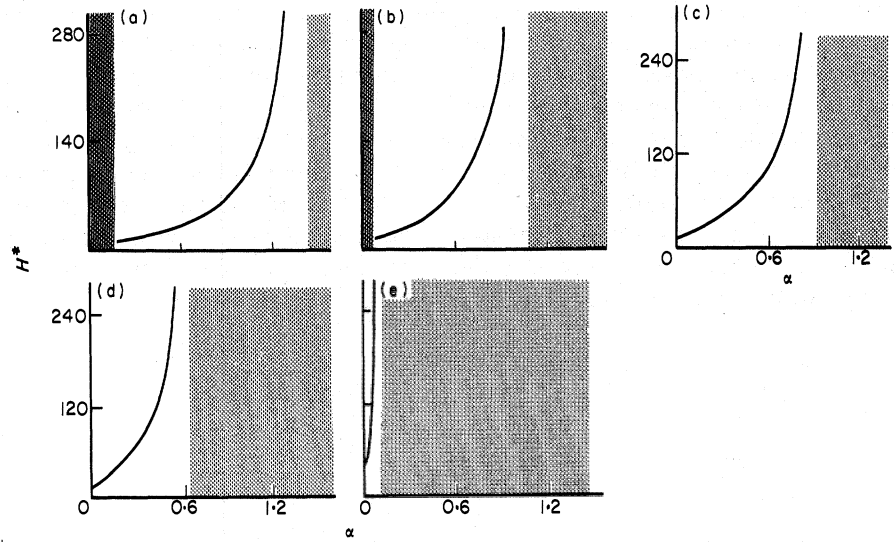

FIG. 9. Model C—density dependent constraints on parasite population growth; The solid lines enclose unshaded regions in which the parameter values lead to parasite regulated host population equilibria which are globally stable. These boundaries are shown in terms of μ and α for various patterns of dispersion of parasite numbers per host; the lightly shaded regions indicate the parameter values which give rise to unregulated host population growth. In the darkly shaded areas the parasite regulates host population growth but the equilibria produced are unstable. (a) Positive binomial, $k' = 6{\cdot}0$; (b) positive binomial, $k = 20{\cdot}0$; (c) Poisson; (d) negative binomial, $k = 4{\cdot}0$; (e) negative binomial, $k = 1.0$; ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$).

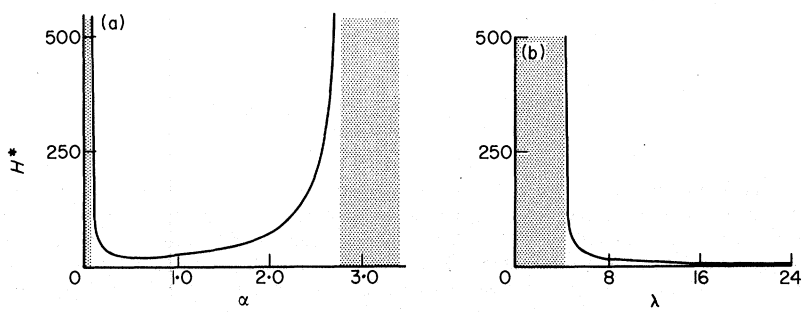

FIG. 10. Model C—density dependent constraints on parasite population growth. (a) The influence of the parameter α on the numerical size of the host population equilibrium H^* (parasites randomly distributed—Poisson model); the shaded region defines parameter values which lead to unregulated host population growth, while in the unshaded regions the parasite regulated host equilibria are globally stable. (b) The influence of the rate of parasite reproduction, λ, on H*; the shaded and unshaded regions are as defined for (a); ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$, $\alpha = 0{\cdot}5$).

dispersion is high (small k). But if the equilibrium can exist, for given k, it is more stable in model C than in model A.

These remarks are further bourne out by Figs 9 and 10. The stable domain in the μ,α parameter space increases in size when the pattern of dispersion of the parasites within the host population moves from regular to random (Fig. 9(a)–(c)), and then decreases as the pattern changes from random to aggregated (Fig. 9 (c)–(e)). When overdispersion is marked (k small), as is the case for many parasitic species (Table 2), the region of parameter space in which the parasite regulates the host population is rather small (Fig. 9(e)). The model's predictions therefore suggest that when the parasite population is tightly controlled by density dependent constraints (i.e. when μ is large) and parasites are overdispersed within the host population, other factors than parasite induced host mortalities will tend to stabilise and regulate host population growth.

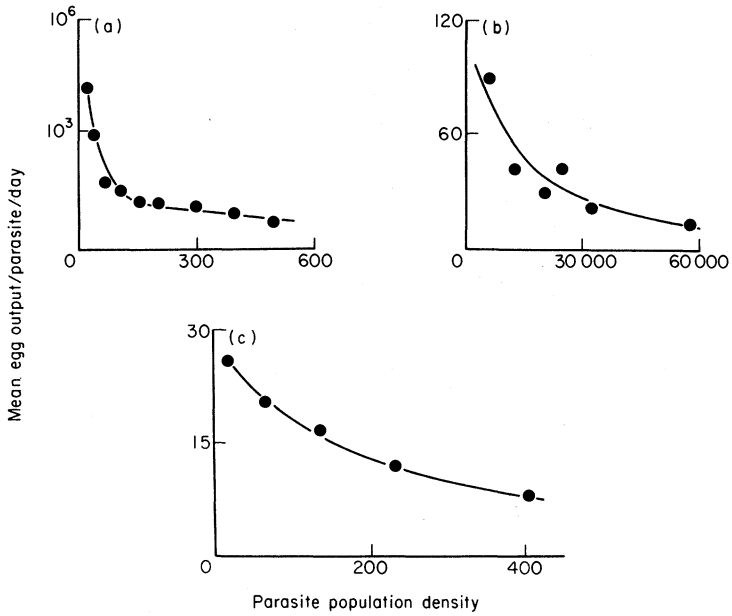

FIG. 11. Some examples of functional relationships between the rate of production of parasite transmission stages (λ) and the parasite population density (i) within individual hosts; the dots are observed points while the solid lines are fitted by eye. (a) Laboratory mice infected with the tapeworm *Hymenolepis nana* (Siebold) (data from Ghazal & Avery 1974); (b) calves infected with *Ostertagia ostertagi* (data from Michel 1969); (c) sheep infected with the fluke *Fasciola hepatica* (data from Boray 1969).

Mammalian hosts which possess well developed immunological responses to parasitic invasion, creating tight density dependent controls on parasite population growth, may well fall into this category.

When parasites are independently randomly distributed, however, the main effect of the density dependence of model C is to help stabilize the system. In the case of Poisson distributed parasites, a further insight, concerning the influence of α upon H^*, is illustrated in Fig. 10(a): too high or too low values of α lead to exponential runaway of the host population.

The above discussion has been for density dependence in the natural parasite mortality

rate. For many host-parasite associations, the 'fecundity' λ is also known to be a function of the parasite population density due, as in the case of the death rate, to either immunological processes or intraspecific competition. Some quantitative examples of such responses are shown in Fig. 11 for helminth parasites of vertebrates. The inclusion of such processes in the basic model leads to qualitatively similar dynamical properties to those outlined above for density dependent parasite death rates.

CONCLUSIONS

Our population models of host-parasite interactions are all characterized by the central assumption that parasites cause host mortalities. In particular, we have assumed that the net rate of such mortalities is related to the average parasite burden of the members of a host population, and therefore to the statistical distribution of the parasites within a host population. We regard the inducement of host mortalities and/or reduction in host reproductive potential as a necessary condition for the classification of an organism as parasitic.

Species which exhibit such characteristics may in certain circumstances play an important role in regulating or controlling the growth of their host population. In such cases, the parasite will play an analogous role to a predator which suppresses the growth of its prey population. We have demonstrated that three specific categories of population processes are of particular significance in stabilizing the dynamical behaviour of a host-parasite interaction and enhancing the regulatory influence of the parasite.

The *first* of these categories of population processes concerns the functional relationships between the rate of parasite induced host deaths and the parasite burden of individual hosts. It is interesting to compare the significance of these relationships on the dynamical properties of a given host-parasite association, with the role played by functional responses in predator-prey, and host-insect parasitoid interactions (Solomon 1949; Holling 1959; Hassell & May 1973). The presence of a complex sigmoid function response between the attack rate of a predator and the density of prey (type III response in the terminology of predator-prey interactions as recently reviewed by Murdoch & Oaten 1975) may, or may not, have a stabilizing influence on the dynamics of such interactions. There will be a range of prey densities over which the death rate of the prey imposed by the predator is an increasing function of prey density and hence such a response will be stabilizing since it acts in a density dependent manner. However, in contrast, if the prey death rate is constant over a wide range of prey densities (as in type II responses), then the predator will not play a major stabilizing role in the dynamics of the prey. In such cases other constraints, such as intraspecific competition for finite resources amongst the prey population, will stabilise the interaction.

We have demonstrated, in the case of host-parasite associations, that when the parasites are randomly distributed within the host population, a linear relationship between host death rate and parasite burden gives rise to the pathological condition of neutral stability (Basic model). If however, the rate of parasite induced host mortalities is an increasing function of parasite burden, perhaps following a power law or exponential form, random distributions of parasites may lead to globally stable equilibria where the parasite is effectively regulating the growth of its host population (model B). Thus, for a given distribution pattern of parasites, certain types of functional response may stabilize the dynamics, while others lead to instabilities as in the case of predator-prey associations.

The *second* category of population processes, which influence the regulatory role of parasites, concerns the statistical distribution of parasite numbers per host. When the rate of parasite induced host mortalities is linearly dependent on the number of parasites harboured, we have demonstrated that parasites may regulate host population growth provided they exhibit an overdispersed pattern of distribution within the host population (model A). The accumulating information in the literature on the overdispersed character of parasite distributions in natural populations of hosts (*vide* Table 2) lends support to the importance of this insight. The stabilizing influence of parasite contagion is somewhat analogous to the influence of aggregated distributions of insect parasitoids on the stability of host-parasitoid associations (Hassell & May 1974). Parsitoid aggregation was shown, in certain circumstances, to stabilize models which otherwise would have been quite unstable.

The *third* category of processes which enhance the regulatory role played by a parasite act within each individual parasite population (or subpopulation) harboured in each member of the host population. Model C demonstrated that density dependent constraints on parasite population growth, caused either by intraspecific competition for finite resources or immunological attack by the host, can in the absence of overdispersion or non-linear functional responses stabilise the dynamics of an interaction.

On the other hand, there are features of host-parasite interactions which tend to destabilize the system. These are discussed in the following paper (May & Anderson 1978), and a general review of pertinent field and laboratory data on host parasite associations is then presented.

ACKNOWLEDGMENTS

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APPENDIX 1

This appendix lists the probability generating functions (p.g.f.'s), $\Pi(Z)$, of the probability $p(i)$ of observing (i) parasites in a host, for three discrete probability distributions. In addition, the expectations of i^2 and i^3 are outlined for each of the distributions in terms of a few parameters such as the mean number of parasites per host, m, and measures of overdispersion k and underdispersion k'.

(a) *Positive binomial*

$$\Pi(Z) = (q - pZ)^{k'} \tag{1.1}$$

where p is the probability of a successful infection occurring, and $q = 1-p$. The parameter k' represents the maximum number of parasites a host can harbour (see Anderson 1974b).

The mean and variance of this distribution are

$$m \equiv E(i) = k'p \tag{1.2}$$

$$\text{var}(i) = k'pq. \tag{1.3}$$

The expectations of i^2 and i^3 are

$$E(i^2) = (k'p)^2 + k'pq. \tag{1.4}$$

that is,

$$E(i^2) = m^2(k'-1)/k' + m. \tag{1.4a}$$

$$E(i^3) = (k'p)^3(k'-1)(k'-2)/k'^2 + 3(k'p)^2(k'-1)/k' + k'p. \tag{1.5}$$

(b) *Poisson*

$$\Pi(Z) = \exp\{m(Z-1)\} \tag{1.6}$$

where $\boldsymbol{m}$ is the sole parameter of the distribution and

$$E(i) = m \tag{1.7}$$

$$\text{var}(i) = m. \tag{1.8}$$

The expectations of i^2 and i^3 are

$$E(i^2) = m^2 + m \tag{1.9}$$

$$E(i^3) = m^3 + 3m^2 + m. \tag{1.10}$$

The Poisson distribution corresponds to the limit $k' \to \infty$(positive binomial), or $k \to \infty$ (negative binomial).

(c) *Negative binomial*

$$\Pi(Z) = (q - pZ)^{-k} \tag{1.11}$$

where p is as defined for the positive binomial, $q = 1 + p$, and the parameter k is a measure which varies inversely with the degree of aggregation of the parasites within the host population.

The mean and variance are given by

$$m \equiv E(i) = kp \tag{1.12}$$

$$\text{var}(i) = kpq. \tag{1.13}$$

The expectations of i^2 and i^3 are

$$E(i^2) = (kp)^2(k+1)/k + kp. \tag{1.14}$$

That is,

$$E(i^2) = m^2(k+1)/k + m \tag{1.14a}$$

$$E(i^3) = (kp)^3(k+1)(k+2)/k^2 + 3(kp)^2(k+1)/k + kp. \tag{1.15}$$

Notice that (as follows from comparison of the defining functions (1.1) and (1.11)) the negative binomial results can all be obtained from the positive binomial ones by the simple transformation $p \to -p$ and $k' \to -\text{k}$, or equivalently, $m \to m$ and $k' \to -\text{k}$. In particular, positive and negative binomial expressions for $E(i^2)$ are interrelated by $k' \to -k$.

APPENDIX 2

This appendix gives a global account of the stability properties of the Basic model defined by eqns (7) and (9).

The expressions for the equilibrium populations H^* and P^* are given by eqns (10) and (11), and the criterion that this point be biologically sensible ($H^* > 0$) is expressed by eqn (12).

For a fully nonlinear discussion of the stability properties of this model, it is sufficient to notice that the function

$$V(H, P) \equiv e^{\alpha P/H}(H_0 + H)^{\lambda} H^{-(b+\mu+\alpha)} P^{(b-a)} > 0 \tag{2.1}$$

is a Lyapunov potential for this system (Minorsky 1962), and furthermore that

$$dV/dt = 0 \tag{2.2}$$

for all t. Thus V is a positive constant, determined by the initial values of H and P; the population values $H(t)$ and $P(t)$ will endlessly cycle around some closed trajectory. In other words, the Basic model corresponds to a neutrally stable or conservative dynamical system, with the consequences discussed in the main text.

Although the potential $V(t)$ of eqn (2.1) was found by analytical trickery, rather than being revealed in a vision, simply writing it down saves space while preserving rigour. Its properties may be verified by differentiating to get dV/dt in terms of dH/dt and dP/dt, whereupon use of eqns (7) and (9) lead to the central result (2.2).

APPENDIX 3

This appendix outlines the analysis of the stability properties of the various host-parasite models presented in this paper; similar analysis pertains to those in the following paper (May & Anderson 1978).

With the exception of model B (which involves $E(i^2)$ in the equation for dH/dt, and $E(i^3)$ in that for dP/dt), and model F in the following paper (which involves time delays), the models under consideration all have the mathematical form

$$dH/dt = c_1 H - c_2 P \tag{3.1}$$

$$dP/dt = P\{\lambda H/(H_0+H) - c_3 - c_4 P/H\}. \tag{3.2}$$

Here c_i (i = 1,2,3,4,) are constants which depend upon the particular biological assumptions in a given model.

Specifically, using eqn (1.14a) to express $E(i^2)$ in eqns (8) and (20) in terms of P/H and k, for a negative binomial distribution of parasites we may tabulate c_i for models A and C as follows:

Model A	Model C	
$c_1 = a-b$	$c_1 = a-b$	(3.3)
$c_2 = \alpha$	$c_2 = \alpha$	(3.4)
$c_3 = b+\mu+\alpha$	$c_3 = b+\mu+\alpha$	(3.5)
$c_4 = \alpha\{k+1\}/k$	$c_4 = \{\alpha+\mu\}\{k+1\}/k.$	(3.6)

The corresponding expressions for a Poisson distribution of parasites are obtained by putting $k \to \infty$, and for a positive binomial by putting $k \to -k'$.

From eqns (3.1) and (3.2), the equilibrium populations H^* and P^* are

$$P^*/H^* = c_1/c_2 \tag{3.7}$$

$$H^* = H_0(c_3 + c_1 c_4/c_2)/(\lambda - c_3 - c_1 c_4/c_2). \tag{3.8}$$

This provides the first important constraint, namely that for a biologically sensible equilibrium to be possible it is required that

$$\lambda > c_3 + c_1 c_4/c_2. \tag{3.9}$$

A linearized stability analysis of this equilibrium is carried out along standard lines (see, e.g. May 1975). Writing $H(t) = H^* + x(t)$ and $P(t) = P^* + y(t)$, and linearizing by neglecting terms of order x^2, xy and y^2, we get from eqns (3.1) and (3.2)

$$dx/dt = c_1 x - c_2 y_1 \tag{3.10}$$

$$dy/dt = c_5 x - (c_1 c_4/c_2) y. \tag{3.11}$$

Here for notational convenience, c_5 is defined as

$$c_5 = (\lambda H_0 H^*/(H_0+H^*)^2 + c_1 c_4/c_2)(c_1/c_2). \tag{3.12}$$

The temporal behaviour of $x(t)$ and $y(t)$ then goes as exp (Λt), where the stability—determining damping rates (or eigen values) Λ are given from eqns (3.10) and 3.11) by the quadratic equation

$$\Lambda^2 + A\Lambda + B = 0. \tag{3.13}$$

Here

$$A \equiv (c_1/c_2)(c_4 - c_2) \tag{3.14}$$

$$B \equiv c_2 c_5 - c_1^2 c_4/c_2 \equiv c_1 \lambda H_0 H^*/(H_0+H^*)^2. \tag{3.15}$$

The requirement for neighbourhood stability is that the real part of both eigen values Λ be negative; the necessary and sufficient condition for this to be so is given by the Routh-Hurwitz criterion $A>0$ and $B>0$. From eqn (3.15) we see that B is always positive. Therefore the equilibrium point will be locally stable if $A>0$, that is if

$$c_4 - c_2 > 0. \tag{3.16}$$

This provides the second important constraint, determining the stability character of the equilibrium point (if it exists).

Since the above models are modifications of the globally neutrally stable Basic model, it is reasonable to assume that if they are locally stable, they are globally stable; and conversely that if they are locally unstable, they are globally unstable. A rigorous proof follows from the observation that, for the general pair of eqns (3.1) and (3.2), the function

$$V = \{\exp(c_2 P/H)\}(H_0+H)^{\lambda} P^{-c_1} H^{\{c_1 - c_3 - c_1 c_4/c_2\}}$$

is a global Lyapunov function. That is, $V>0$ (provided the initial host and parasite populations are positive), and

$$dV/dt = (c_2 - c_4)(c_1 - c_2 P/H)^2 c_2^{-1},$$

as may be verified by differentiating $V(H,P)$, and using eqns (3.1) and (3.2) to express dH/dt and dP/dt. Local stability requires $c_4 > c_2$ (see eqn (3.16)), whence $dV/dt<0$, connoting global stability. Conversely local instability requires $c_4 < c_2$, whence $dV/dt>0$, connoting global instability. The structurally unstable razor's edge of $c_4 = c_2$ gives both local (eqn (3.16)) and global ($dV/dt = 0$) neutral stability, as discussed more fully in Appendix 2.

In brief, local and global stability properties march together in these models.

We now proceed to indicate how the basic criteria (3.9) and (3.16) may be applied to the various models to derive the dynamical properties discussed in the main text, and illustrated in Figs 5, 6, 7, 9 and 10.

Model A

For a negative binomial distribution of parasites, the expressions (3.3) through (3.6) may be substituted in eqn (3.9) to arrive at the criterion for an equilibrium solution to be possible. This expression is given and discussed as eqn (16) in the main text. The stability criterion (3.16) here reads.

$$\alpha/k > 0 \tag{3.17}$$

which is always satisfied; the equilibrium point is necessarily stable.

A Poisson distribution of parasites (as in the Basic model) is obtained as the limit $k\infty \rightarrow$, thus producing eqn (12) as the criterion for an equilibrium point to exist. In this case, $c_4 = c_2 = \alpha$, so that $A=0$ and the eigen value equation (3.13) becomes $\Lambda^2+B = 0$, or

$$\Lambda = \sqrt{-B}. \tag{3.18}$$

That is, both eigen values are purely imaginary, leading to neutral stability in the linearised analysis; this, of course, is the local version of the global result established in Appendix 3.

For a positive binomial distribution, the stability criterion (3.16) becomes

$$-\alpha/k' > 0 \tag{3.19}$$

which cannot be satisfied. This model is ineluctably unstable.

Model C

For an overdispersed distribution, we substitute the appropriate expressions (3.3) through (3.6) into eqn (3.9) to arrive at eqn (21) for the criterion for an equilibrium point to be possible. The stability criterion (3.16) is here

$$\{\alpha+\mu(k+1)\}/k > 0 \tag{3.20}$$

which is always satisfied.

For a Poisson distribution, putting $k\rightarrow\infty$ in eqn (21) produces the criterion for an equilibrium to exist. Similarly, putting $k\rightarrow\infty$ in eqn (3.20) gives the stability condition $\mu>0$, which always holds.

For an underdispersed distributions, we put $k\rightarrow -k'$ in eqn (21) to get the condition for equilibrium to exist; see Fig. 10. By likewise putting $k\rightarrow -k'$ in the stability criterion (3.20), we see that such an equilibrium will be stable if

$$\mu k'-\mu-\alpha > 0. \tag{3.21}$$

Stability is helped by high natural parasite mortality compared with parasite induced host mortality (i.e. by μ large compared with α), and hindered by high underdispersion (i.e. by small k').

Model B

In this model, the equation for dH/dt involves $E(i^2)$ and thence a term in P^2/H, and the equation for dP/dt involves $E(i^3)$ and thence a term in P^3/H^2. Consequently these host-parasite equations are not exactly of the form of eqns (3·1) and (3·2), and require separate treatment.

For a negative binomial distribution of parasites, use of the expressions (1.14) and (1.15) for $E(i^3)$ and $E(i^2)$ in eqns (18) and (19), respectively, leads to

$$dH/dt = (a-b)H-\alpha P-\alpha\{(k+1)/k\}P^2/H, \tag{3.22}$$

$$dP/dt = P\{\lambda H/(H_0+H)-(\mu+b+\alpha)-3\alpha\{k(+1)/k\}P/H-\alpha\{(k+1)(k+2)/k^2\}P^2/H^2\}. \tag{3.23}$$

As ever, the corresponding equations for a Poisson distribution are obtained as the limit $k\rightarrow\infty$, and for a positive binomial by the substitution $k\rightarrow -k'$.

The equilibrium mean parasite burden per host, $m^* = P^*/H^*$, follows from eqn (3.22):

$$m^* = \tfrac{1}{2}\{k/(k+1)\}[\{1+4(a-b)(k+1)/(k\alpha)\}^{\frac{1}{2}}-1]. \tag{3.24}$$

It then follows from eqn (3.23), after some tedious algebraic manipulations, that the equilibrium host population is

$$H^* = H_0\zeta/(\lambda-\zeta), \tag{3.25}$$

with ζ defined for convenience as

$$\zeta = \mu+a+\alpha+2(a-b)/k+\{(2k+1)/k\}\alpha m^* \tag{3.26}$$

A biologically sensible equilibrium is possible only if

$$\lambda > \zeta. \tag{3.27}$$

This relation among the parameters is illustrated in Fig. 7; remember that for a Poisson distribution the above equations are to be read with $k\to\infty$, and for a positive binomial with $k\to -k'$.

The stability of such an equilibrium point is determined by a linearized stability analysis of the kind described above (and, e.g. in May 1975). Linearizing eqns (3.22) and (3.23) about the equilibrium point defined by eqns (3.24) and (3.25), we eventually obtain the quadratic eigen value equation (3.13), where the coefficients A and B are

$$A = \alpha m^*\{k(2k+3)+4(k+1)m^*\}/k^2, \tag{3.28}$$

$$B = \alpha\{1+2m^*(k+1)/k\}\{\lambda H_0 P^*/(H_0+H^*)^2\}. \tag{3.29}$$

From eqn (3.24) it follows that $B>0$ for all positive m^*.

Clearly $A>0$ for overdispersed or Poisson ($k\to\infty$) distributions, so their equilibrium points are stable. For underdispersed distributions (positive binomial, $k\to -k'$), the stability condition $A>0$ leads to the requirement that $k'>1{\cdot}5$ and

$$4m^* < k'(2k'-3)/(k'-1). \tag{3.30}$$

Thus underdispersed distributions can have stable equilibria, provided the mean parasite burden is relatively low (small m^*) and the underdispersion is not pronounced (large k'): Fig. 7 bears out these remarks.

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