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Phil. Trans. R. Soc. Lond. B 1981 **291**, 451-524
doi: 10.1098/rstb.1981.0005

THE POPULATION DYNAMICS OF MICROPARASITES AND THEIR INVERTEBRATE HOSTS

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(*Received* 18 *April* 1980)

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 [Published 27 April 1981

We show how directly transmitted microparasites, broadly defined to include viruses, bacteria, protozoans and fungi, may regulate natural populations of invertebrate hosts. The study combines elements of conventional epidemiology (where the host population is assumed constant) with elements of prey–predator studies (which conventionally emphasize how prey and predator populations may be regulated by their interaction).

To this end, we construct simple models embodying the essentials of the dynamical interaction between invertebrate hosts and their directly transmitted microparasites. In successive refinements, these models include the effects of recovery and disease-induced mortality, castration or diminished reproduction of infected hosts, vertical transmission, latent periods of infection, stress-related pathogenicity, the interplay between disease and other density-dependent constraints on host population growth, and free-living infective stages. In analysing the dynamical behaviour of these models, we focus on: the possible regulation of the host population by the parasite; the basic reproductive rate of the parasite, and the way in which it affects the dynamics and the evolution of the host–parasite association; and the threshold host density and its implications for endemic or epidemic maintenance of the infection. These are examined in the light of synoptic compilations of field and laboratory data on: birth rates (and disease-induced reduction thereof), natural death rates and disease-induced death rates of hosts; latent periods and efficiencies of vertical transmission of pathogens; the rate of production and lifetime of free-living infective stages; and some characteristics of long-term cycles and of epidemic outbreaks of disease in forest insects. In particular, our models suggest that the baculovirus and microsporidian infections of many temperate forest insects will tend to produce stable cycles in host abundance and in prevalence of infection, with periods in the range 5–12 years. Enough is known about the European larch budmoth and an associated granulosis virus for us to undertake a detailed comparison between theory and data that strongly suggests that the observed 9–10 year cycles are driven by the host–parasite interaction. We also discuss the possible control of invertebrate pest species by pathogens, showing how our models could guide laboratory or field studies, to help estimate whether a given pathogen is capable of regulating the target pest population, and, if so, roughly what quantity is needed to effect a specific level of (local) control.

Throughout, the emphasis is on the biological ingredients of the models, and on the biological conclusions to be drawn; mathematical details are given in appendixes.

1. Introduction

In this paper we aim at bringing together two separate literatures, one dealing with the ecology of animal populations and the other with invertebrate pathology, that it might be understood how parasitic infections persist within, and may regulate, populations of invertebrates.

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The ecological literature is rich in discussions of the ways in which natural populations may be controlled by the interaction between predators or insect parasitoids and their hosts; such discussions range from the seminal work of Elton and others to contemporary texts (Elton 1927; Slobodkin 1961; Ricklefs 1973; Krebs 1978; Hutchinson 1978). The influence of pathogens upon the dynamics of their host populations tends to have been either ignored (because the pathogens are often difficult to detect), or dismissed as relatively unimportant (possibly because the effects are not easily quantified). We show, however, that pathogens are diverse and abundant in natural communities, and that the parameters characterizing their interactions with their host populations are often such as to make pathogens as important as the more commonly studied predators, parasitoids or resource limitations in constraining the growth of invertebrate populations.

On the other hand, the attention of invertebrate pathologists and virologists has naturally tended to centre on the biology of individual organisms, rather than on overall population behaviour (Brock 1966; Smith 1976; Gibbs 1973; Fenner *et al.* 1974). In the field of invertebrate virology, for example, the basic identification and taxonomy of the pathogen is a major problem (Smith & Wyckoff 1951; Tinsley 1979; Whitcomb & Tully 1979), and only recently have technical advances enabled microbiologists to tackle these tasks successfully. We seek to build on this factual foundation, creating a theoretical framework that elucidates the overall dynamical properties of the host-pathogen system. Such an analytic framework clarifies the factors underlying the maintenance of endemic or epidemic disease in natural populations; as we shall argue below, the absence of such understanding has caused some confusion in the literature on invertebrate pathology (concerning, for example, the relation between epidemic phenomena and host abundance). Moreover, an understanding of the population biology of infectious diseases of invertebrates is relevant to the use of microparasites as agents in the biological control of insect pest species (Burges & Hussey 1971; Huffaker 1974; DeBach 1974; Tinsley 1979).

The present paper focuses on microparasitic infections that are directly transmitted among invertebrate hosts. The paper is organized as follows. A brief discussion of the term 'microparasites', and of the diverse array of life cycles that they can exhibit is given in § 2. The nature of invertebrate responses to parasitic infection is reviewed in § 3. Next, in § 4, we outline the basic components to be assembled into a set of equations for studying the population dynamics of microparasitic infections; each individual component (disease transmission between hosts, disease-induced host mortality, recovery from infection, etc.) of the overall model is based on field and laboratory data, which is surveyed. The notions of the net reproductive rate of the pathogen and of the threshold level of host abundance required for persistence of the disease are also examined in § 4. The dynamical properties of the simplest model (model A) are examined in § 5, with particular attention to the criteria determining whether a pathogen regulates, or merely reduces, the population growth of its host. Data for some invertebrate hosts and associated microparasites are reviewed in this light. In §§ 6–11 we then deal with a series of modifications to this basic model A, incorporating various features that are known to arise in particular host–parasite associations: model B allows for parasite-induced reduction of host reproduction; model C incorporates the effects of vertical transmission; model D includes a latent period, during which hosts are infected but not yet infectious; model E examines the interaction of disease and stress within the host population; model F adds density-dependent constraints on the population growth of the host; and model G extends the range of possible dynamical behaviour by including free-living infective stages of the parasite. In all of these,

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the emphasis is on the biological lessons to be learned from these mathematical metaphors, and on possible tests against empirical evidence.

In § 12, we combine theory and observation to show how pathogens can generate cyclic patterns, with periods in excess of one year, in the abundance of their hosts and in the prevalence of infection. We suggest that the 5–12 year population cycles recorded for many temperate forest insects (and particularly the 9–10 year cycle of the European larch budmoth) arise in this way. In § 13 we deal more generally with the persistence of pathogens within host populations that fluctuate widely with time; we show how vertical transmission, occult or non-apparent infections or free-living infective stages can help solve the problems that such circumstances pose. In § 14 we consider the use of pathogens as agents of biological control of pest species, concentrating on the features likely to lead to successful control. Some general aspects of the coevolution of invertebrate hosts and their pathogens are discussed in § 15. Finally, § 16 summarizes the main conclusions. Lists of the symbols used to denote the variables and parameters in this paper are given in appendix A, and mathematical details and proofs of results given in the main text are outlined in a series of other appendixes.

More generally, we are interested in the population biology of parasitic infections (defined broadly to include viruses, bacteria, protozoans, fungi, helminths and arthropod infections), and in the way that they may regulate the abundance of their host populations. This larger plan embraces both directly and indirectly transmitted infections of both vertebrate and invertebrate host species (for an overview, see Anderson & May (1979*a*) and May & Anderson (1979)). Thus the present study of invertebrate hosts and their directly transmitted microparasites is a piece in a larger picture; it is, however, a most substantial piece, rich in data and in potential practical applications.

2. Microparasites of prokaryotes and invertebrate eukaryotes

We use the term microparasite to describe pathogens, such as viruses, bacteria, protozoans and fungi, that are characterized by small size, short generation time and an extremely high rate of direct reproduction within the host (Anderson & May 1979*a*; May & Anderson 1979).

To counter the onslaught of such rapidly multiplying organisms, the host individual typically mounts some form of response, aimed at reducing the growth rate of the parasite population. A successful response may eliminate the parasite from the body of the host (complete recovery), or may regulate the parasite population at some 'persisting' steady level within the host. Failure of the host to contain the pathogen's population growth will lead to death. Some detailed aspects of these responses are pursued in the next section.

Microparasites may complete their life cycles by passing from one host to the next either *directly*, or *indirectly* via one or more intermediate host species. This paper is restricted to directly transmitted agents, where transmission is achieved by physical contact between hosts or by transmission stages (specialized or unspecialized) of the parasite which pass into the habitat of the host and are picked up by inhalation, ingestion or direct penetration of the host's body. A special case of direct transmission arises when the infection is conveyed by a parent to its unborn offspring (egg, embryo or host chromosomes). This process has been named *vertical transmission* in contrast to the variety of *horizontal transmission* processes just described (Gross 1949, 1951; Fine 1975).

3. Invertebrate responses to parasitic infection

Vertebrate animals appear to differ from other living organisms in their capacity to react to disease agents in a highly specific manner by mounting an immunological response. Vertebrates' immune responses, which are associated with their ability to distinguish between self and non-self, are characterized by specificity, dissemination, amplification and memory (Hobart & McConnell 1978; Mims 1977; Fenner & White 1976). These features of the vertebrate immune response mean that a second or later exposure to a particular infectious agent will evoke an accelerated response, even at a site remote from the primary infection; this ability to mount an enhanced response on second exposure is termed *acquired immunity*. This acquired response can cause second and later infections to be eliminated very rapidly, with no overt signs of disease, so that hosts with acquired immunity in effect join a category that is protected from the infection (analogous to prey species having a refuge that protects them from predators).

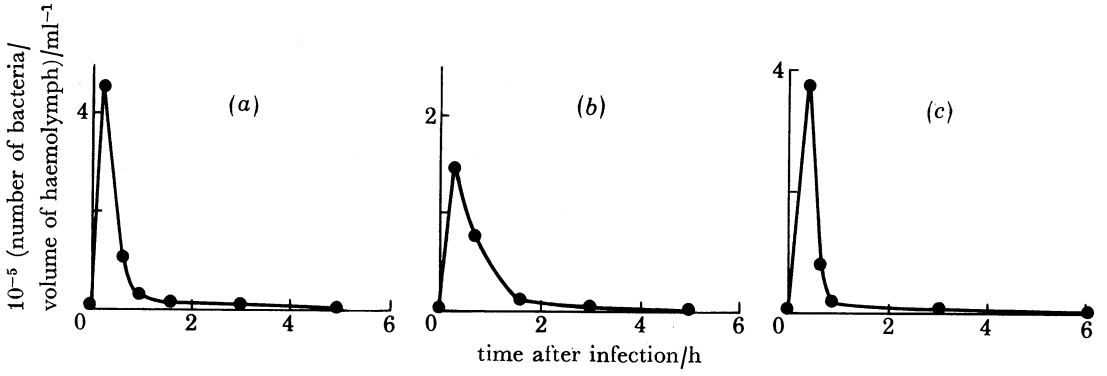

Figure 1. Invertebrate response to microparasitic infection, as illustrated by the population growth of bacteria in the haemolymph of a land snail (*Helix pomatia*) on first and subsequent exposure to infection. The data are from Bayne & Kime (1970), and show: (*a*) first exposure to infection; (*b*) second exposure, 2 weeks after first exposure; (*c*) third exposure, 1 week after second exposure.

Although invertebrate species are usually able to mount cellular or humoral responses to parasitic invasion, these responses do not appear to be enhanced on a second or later exposure to the infectious agent (Bang 1975*a*, *b*). That is, current evidence suggests invertebrate species are unable to develop acquired immunity to agents of infectious disease (Jackson *et al.* 1969; Salt 1970; Bayne 1973; Bang 1973, 1975*a*; Lafferty & Crichton 1973; Maramorosch & Shope 1975; Lackie 1980).

The nature of the initial response to infection appears remarkably uniform among different groups of invertebrates. In general, amoeboid cells phagocytose small pathogens, while larger organisms are encapsulated. Such responses can clearly result in the elimination of the parasite and the recovery of the host; the absence of acquired immunity, however, means that recovered individuals will pass directly back into the pool of hosts susceptible to further infection. There is no immune category. This process of recovery and absence of acquired immunity among invertebrates is nicely illustrated by the experiments of Bayne & Kime (1970) on bacterial infections in land snails, *Helix pomatia*. As shown in figure 1, parasitic bacteria were rapidly eliminated from the snail host by amoebocytic action, but the rate of recovery from infection was not enhanced by repeated exposure to the parasite.

Prokaryotic and protozoan host species do not appear to be able to mount a response to

parasitic invasion (Maramorosch & Shope 1975; Manning & Turner 1976). Recovery from infection is therefore unlikely in such organisms.

In short, there are two main points relevant to our investigation of the overall population dynamics of invertebrate host–parasite associations. First, invertebrate hosts may or may not be able to mount a response to parasitic invasion, but, if they do, recovery can occur. Secondly, responses (whether immunological or non-specific) do not lead to acquired immunity in prokaryote or invertebrate eukaryote organisms; hosts that recover from infection are immediately susceptible to reinfection. This second feature makes the relevant population models simpler than is typical for vertebrate host species.

4. Components of the interaction between invertebrate hosts and microparasitic infections

In the simplest case, we define $X(t)$ to be the number of susceptible hosts, and $Y(t)$ the number of infected hosts, at time t. The total population of invertebrate hosts is thus $H(t) = X(t) + Y(t)$. In this basic two-component model it is assumed that all infected individuals are infectious; at a later stage we consider more detailed subdivisions of the host population (where, for example, there is a latent period in the development of the infection, so that the infected class Y is broken up into infecteds, which are, or are not yet, infectious).

This simple and conventional (Bailey 1975; Dietz 1974) distinction between susceptible (uninfected) and infected hosts makes sense by virtue of the extremely high rates of direct reproduction within the host that pertain for most viral, bacterial, protozoan and fungal infections of invertebrates. More formally, we have defined *microparasites* to be those for which such simple, compartmental models afford a good description, in contrast with *macroparasites* (including essentially all helminth infections), of which there is typically no direct reproduction within the host, and of which the effects of the infection upon the host depend on the number present (rather than simply on presence or absence). These general questions are reviewed elsewhere (Anderson & May 1978; May & Anderson 1978), and the present work is confined to microparasitic infections.

To begin, we make the conventional epidemiological assumption that total host population is held to a constant value, independent of the presence or absence of the infection, by some unspecified mechanism (Bailey 1975; Dietz 1974):

$$H(t) = H = \text{constant}. \tag{1}$$

We now seek to describe the dynamics of the infection within this constant population; that is, we seek a differential equation expressing the change in the number of infected individuals with time, $\mathrm{d}Y/\mathrm{d}t$, as a function of Y and ecological and epidemiological parameters (including H itself). The components of this relation are as follows.

(a) *Transmission of infection among hosts*

In this paper, our attention is restricted to *directly transmitted* infections (as opposed to those with indirect transmission, involving one or more species of intermediate hosts). The simplest assumption is that the rate at which infections are acquired by such direct transmission is proportional to the number of encounters between susceptible and infected hosts. That is, the net rate of transmission of the infection is βXY, where X is the number of uninfected individuals,

Y is the number of infected individuals spreading the infection, and the proportionality constant β is called the 'transmission parameter' (βH has the dimension of 1/time). Of all the parameters in our models, β is by far the hardest to estimate in practical applications. If the age-specific prevalence of the infection in the host population is known and is in equilibrium, β can sometimes be estimated indirectly from the typical age at which the infection is acquired (Dietz 1974); this information is rarely available for invertebrate populations (but see Anderson & May 1979*b*).

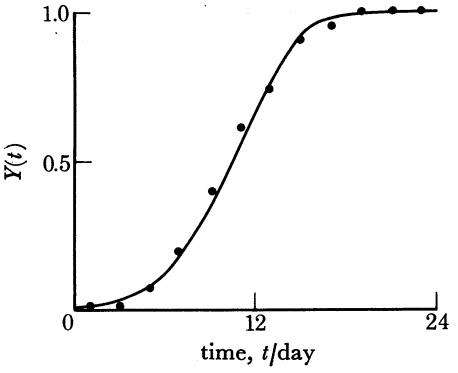

FIGURE 2. The dynamical behaviour of an experimentally induced epidemic of the protozoan *Hydramoeba hydroxena*, within a population of the coelenterate *Chlorohydra viridissima*, is shown. The data points are from Stiven (1967), and show the observed proportion of hosts infected by day t, $Y(t)$. The solid line is given by our simple model for the transmission process (namely, equation (5) with $\beta = 0.0043$).

The assumption that the overall transmission rate is βXY, although crude, can be shown often to give a good description of the observed transmission process. In particular, if no individuals die or recover from infection during the period that a closed population of H hosts is under observation, the rate of change in the number of infected individuals is (rewriting $X = H - Y$)

$$dY/dt = \beta Y(H-Y). \tag{2}$$

Then if Y_0 infecteds are introduced into the population of $H - Y_0$ susceptibles at time $t = 0$, the subsequent course of the infection obeys the familiar sigmoidal solution of the logistic equation (2):

$$Y(t) = \frac{HY_0}{Y_0 + (H-Y_0)\exp(-\beta Ht)}. \tag{3}$$

In figure 2, this theoretical expression is compared with observed data for an epidemic of the protozoan pathogen *Hydramoeba hydroxena* within a population of the coelenterate *Chlorohydra viridissima* (Stiven 1964, 1967). There is only one adjustable parameter (namely β) in equation (3), and the agreement between theory and observation shown in figure 2 is encouraging. Instances where the transmission term βXY describes the dynamics of epidemics and rumours in human populations have been described by many authors (Kermack & McKendrick 1927; Bailey 1975; Dietz 1967). We have recently shown that the βXY term, incorporated in models broadly related to those below, gives an excellent fit to data from laboratory experiments on the regulation of mouse populations by viral and bacterial infections (Anderson & May 1979*a*).

Of course, the simple expression βXY is not always sufficient to describe directly transmitted infection processes. Some general exceptions are discussed by Bailey (1975), Yorke *et al.* (1979) and Anderson (1980*b*), and a refinement of particular relevance to many invertebrate host–parasite systems is pursued below (model G).

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(b) *Host mortality*

Uninfected hosts are assumed to die at a rate b per individual, giving a net death rate bX. The death rate b is assumed to be independent of the age of the individual ('type II survivorship curve'), and corresponds to an expected lifespan of $1/b$ time units.

We define a parasite to be an organism that not only depends on its host to provide habitat and nutrition, but also causes some degree of 'harm' to the host (Anderson & May 1978, 1979*a*; May & Anderson 1979; Price 1980). At the population level, such harm is measured by the parasite's effect upon the vital rates (birth and death rates) of the host population. Here we begin by assuming that infection acts to increase the death rates, such that the death rate per individual infected host is $b+\alpha$, giving a net death rate $(b+\alpha)Y$. The parameter α represents the rate of disease-induced mortality, again assumed to be independent of the host's age; the expected lifespan of an infected host is thus $1/(b+\alpha)$. The effect of parasites on the reproductive rate of infected hosts is considered below (model B).

Values of b and α for a range of invertebrate hosts, infected with various viral, bacterial, protozoan and fungal parasites, are collected in table 1.

(c) *Recovery from infection*

As discussed in §§ 2 and 3, infected hosts are often able to recover from infection (see figure 1). As a rough approximation, we assume an individual recovery rate γ (although a more accurate assumption will often be that there is recovery after some specific interval of time has elapsed); the net rate at which hosts recover and pass back into the pool of susceptible individuals is γY. Under our assumption and in the absence of host mortality, the average duration of infection is $1/\gamma$.

(d) *Number of infected hosts*

Putting all these pieces together, we see the net rate at which infected hosts appear is βXY, and the net rate at which they are lost (by natural or disease-induced death, or by recovery) is $(\alpha+b+\gamma)Y$. The rate of change of Y is therefore

$$\mathrm{d}Y/\mathrm{d}t = \beta XY-(\alpha+b+\gamma)Y. \tag{4}$$

The conventional epidemiological assumption that the total host population H is constant, equation (1), enables us to eliminate $X(t)$ from equation (4) and recast it as an equation for the single dynamical variable $Y(t)$:

$$\mathrm{d}Y/\mathrm{d}t = [(\beta H-\alpha-b-\gamma)-\beta Y]Y. \tag{5}$$

This is a logistic equation for $Y(t)$, and its features have been discussed by many people (e.g.: Kermack & McKendrick 1927; Bailey 1975; Dietz 1974). The discussion can, however, be simplified, and some new insights gained, by rewriting equation (5) in an appropriately dimensionless form. The notion that ecological equations should be brought to dimensionless form, so that attention can be focused on the meaningful combinations of parameters, has been discussed and illustrated elsewhere (May 1976, ch. 2; May *et al.* 1979). More generally, Montroll & Shuler (1979) have discussed the role of rescaling and of dimensionless parameters in the natural sciences and engineering, arguing that many of the observed patterns of success (ship and plane design) and difficulty (controlled nuclear fusion) are attributable to whether

TABLE 1. NATURAL AND PATHOGEN-INDUCED MORTALITY RATES FOR INVERTEBRATE HOSTS OF SOME VIRAL, BACTERIAL, PROTOZOAN AND FUNGAL PARASITES (BASED ON LABORATORY STUDIES)

pathogen	host	natural mortality rate, b / week^{-1}	pathogen-induced mortality rate, α / week^{-1}	reference
viruses				
sack brood virus	*Apis mellifera*	0.17	1.2	Mussen & Furgala (1977)
nuclear-polyhedrosis virus	*Cadra cautella*	0.061	0.54	Hunter *et al.* (1973)
nuclear-polyhedrosis virus	*Hyphantria cunea*	0.003	0.80	Nordin & Maddox (1972)
non-inclusion virus	*Panonychus citri*	0.34	0.91	Gilmore & Tashiro (1966)
A.B.P. virus	*Apis mellifera*	0.25	1.9	Kulincevic *et al.* (1970)
R.O. virus	*Oryctes rhinoceros*	0.10	0.19	Zelazny (1973)
nuclear-polyhedrosis virus	*Porthetria dispar*	0.060	0.63	Doane (1967)
nuclear-polyhedrosis virus	*Malacosoma americanum*	0.070	0.37	Smirnoff (1967)
bacteria				
Bacillus thuringiensis	*Simulium vittatum*	0.035	2.4	Lacey & Mulla (1977)
Bacillus thuringiensis	*Choristoneura fumiferana*	0.001	4.0	Smirnoff (1973)
Aeromonas punctata	*Anopheles annulipes*	0.36	2.9	Kalucy & Daniel (1973)
Erwinia spp.	*Colladonus montanus*	0.031	0.17	Whitcomb *et al.* (1966)
protozoa				
Nosema stegomyiae	*Anopheles albimanus*	0.23	0.41	Anthony *et al.* (1973)
Pleistophora schubergi	*Hyphantria cunea*	0.003	0.036	Nordin & Maddox (1972)
Herpetomonas muscarum	*Hippelates pusio*	0.17	0.43	Bailey & Brooks (1972)
Tetrahymena pyriformis	*Culex tarsalis*	0.26	0.66	Finlayson (1950)
Mattesia dispora	*Laemophloeus minutas*	0.022	0.11	Grassmick & Rowley (1973)
fungi				
Beauveria tenella	*Aedes siemensis*	0.026	0.50	Pinock *et al.* (1973)
Beauveria tenella	*Culex tarsalis*	0.11	0.84	Pinock *et al.* (1973)
Beauveria bassiana	*Musca domestica*	0.27	0.74	Rizzo (1977)
Beauveria bassiana	*Hylemya antiqua*	0.30	0.55	Rizzo (1977)
Beauveria bassiana	*Phormia regina*	0.24	0.56	Rizzo (1977)
Metarrhizium anisopliae	*Musca domestica*	0.27	0.38	Rizzo (1977)
Metarrhizium anisopliae	*Phormia regina*	0.24	0.42	Rizzo (1977)
Metarrhizium anisopliae	*Hylemya antiqua*	0.30	0.48	Rizzo (1977)
Aspergillus flavus	*Culex peus*	0.020	0.17	Toscano & Reeves (1973)
Aspergillus flavus	*Culex tarsalis*	0.061	0.22	Toscano & Reeves (1973)
Fusarium oxysporum	*Culex pipiens*	0.027	0.62	Hasan & Vago (1973)

the characteristic number of dimensionless parameters is small or large. In equation (5) we rescale the variables Y and t, using the new dimensionless variables $y = Y/H$ and $t' = (\alpha+b+\gamma)\,t$; y is simply the infected fraction of the total population (often called the *prevalence* of the infection), and t' is the time measured against the natural time scale $1/(\alpha+b+\gamma)$. Furthermore, we define a dimensionless parameter R:

$$R \equiv \beta H/(\alpha+b+\gamma). \tag{6}$$

Equation (5) now becomes

$$\mathrm{d}y/\mathrm{d}t' = y[(R-1)-Ry]. \tag{7}$$

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It is immediately evident that the dynamics of the infection, the *shape* of the curve $y(t')$, depends only on the quantity R. The scale of the time axis depends on $(\alpha+b+\gamma)$, and the absolute scale of the infected population Y depends on H, but the qualitative nature of the host–parasite interaction here depends only on R.

Two cases may be distinguished.

(i) If $R > 1$ $(\beta H > \alpha+b+\gamma)$, the infection persists within the host population, and the prevalence eventually approaches a steady value (obtained by putting $dy/dt' = 0$ in equation (7)) of

$$y = 1-1/R. \tag{8}$$

The associated value of the susceptible fraction of the population, $x = X/H$, is clearly

$$x = 1/R. \tag{9}$$

(ii) Conversely, if $R < 1$ $(\beta H < \alpha+b+\gamma)$, the right side of equation (7) is necessarily negative for all values of y, and the disease cannot persist within the host population. That is, $y(t)$ always decreases, and the steady solution is $y \to 0$, $x \to 1$.

(*e*) *Basic reproductive rate of the parasite*

These results can be explained in a more directly biological way. Returning to equation (16), we see that R is defined to be the expected number of secondary infections (βH) produced within the infectious period, $1/(\alpha+b+\gamma)$, of one newly introduced host. That is, R is the basic reproductive rate of the parasite (Dietz 1974; Anderson 1980*a*), precisely analogous to the conventional ecologists' and demographers' 'expected number of offspring', R_0 (see, for example, Krebs 1978). Clearly the infection can persist in the host population if, and only if, $R > 1$.

Moreover, equations (9) and (8) for the equilibrium fraction of susceptible and infected hosts can also be obtained by an intuitive argument. At equilibrium, each infected host should produce on average one secondary case. Since a single infection is capable of producing R secondary cases, the susceptible fraction of the host population must be reduced to a value x such that $Rx = 1$; this gives equation (9), and thence (since $x+y = 1$) equation (8).

This concept of a basic reproductive rate for parasitic infections is central to any understanding of the circumstances under which diseases persist within host populations. Environmental factors will often cause the host density H and/or the parameters β, b and α to vary seasonally, or from one year to another, in such a way that the basic reproductive rate R sometimes falls below unity. When this happens, the infection will only persist from year to year if the periods during which R is less than unity are shorter than the maximum lifespan of an infected host or of a free-living infective stage. This point is pursued in § 13.

More generally, expressions for the basic reproductive rate R can be obtained for, and are central to the population biology of, all parasitic infections, whether microparasites or macroparasites and whether transmitted directly or indirectly (Macdonald 1957; Dietz 1974; Bailey 1975; Anderson & May 1978, 1979*a*; May & Anderson 1979; Nold 1979).

(*f*) *Threshold host density*

The condition $R > 1$ for maintenance of the infection may equivalently be expressed as the requirement that the host population exceed a *threshold* density (Kermack & McKendrick 1927; Anderson & May 1978 1979*a*; May & Anderson 1978, 1979).

For the system of equations discussed above, the threshold host density is defined as

$$H_T = (\alpha + b + \gamma)/\beta. \tag{10}$$

Equation (6) for R can then be rewritten as

$$R = H/H_T. \tag{11}$$

The equivalence between the requirement $R > 1$ and $H > H_T$, for maintenance of the infection, is thus clear. The requirement $H > H_T$ could, of course, have been obtained directly from the requirement that the right side of the original equation (5) be positive for small Y; the above discussion, however, makes the underlying biological reason more explicit.

Several biological conclusions about the persistence of parasitic infections within host populations may be drawn from equation (10). Microparasites with low transmission efficiency (β small) will in general only persist within high density populations of hosts; conversely, microparasites with high transmission efficiency (β large) can persist in low density host populations. Diseases with low transmission efficiency may, however, be able to persist in relatively low density host populations, provided that the expected lifespan of infected hosts is long (that is, $1/(\alpha + b + \gamma)$ large). This can happen if natural and parasite-induced mortality are both low ($\alpha + b$ small) and the average duration of infection is long (γ small). Highly pathogenic parasites (α large) will only survive in relatively high-density host populations.

5. Basic dynamics of host–parasite associations: model A

The discussion in § 4 is mainly review of conventional epidemiological ideas, albeit with more emphasis on the overall population aspects than is common. Our purpose was to create a framework, and to develop some basic notions, in a relatively simple context. We now break new ground by treating the total host population H as a dynamical variable, and explore the circumstances under which parasitic infections will actually regulate their host populations.

That microparasites can have pronounced effects on the growth characteristics of their host populations has been clearly demonstrated in laboratory studies. For example, Park (1948) and Finlayson (1950) have shown that arthropod populations infected with protozoan parasites are depressed to densities significantly below the disease-free levels (see figure 3*a*, *b*). In some cases, pathogens may regulate the growth of host populations, in the absence of any other constraints (such as resource limitation). Doerman (1948), for example, demonstrated how the introduction of a phage virus, T4 bacteriophage, into populations of the bacterium *Escherichia coli* converted patterns of exponential growth into regulated growth toward an equilibrium bacterial density (see figure 4*a*). Anderson's (1957) work on the influence of the bacteriophage Vi-type A on the growth of populations of the bacterium *Salmonella typhi*, illustrated in figure 4*b*, provides another example. Anderson's study, moreover, gives a good example of the threshold phenomenon: as shown in figure 4*b*, the population of phage virus did not increase above its introduction level until its host bacterial population exceeded a threshold density; once this threshold was exceeded, the virus population increased and, by its effects on bacterial survival and reproduction, transformed the exponential growth of the host population into decline towards an equilibrium density.

To arrive at a fully dynamic model for the host–parasite system, we need to add one more ingredient to the recipe discussed in § 4, namely a description of the birth process whereby new susceptibles are recruited into the host population. We assume that the individual host

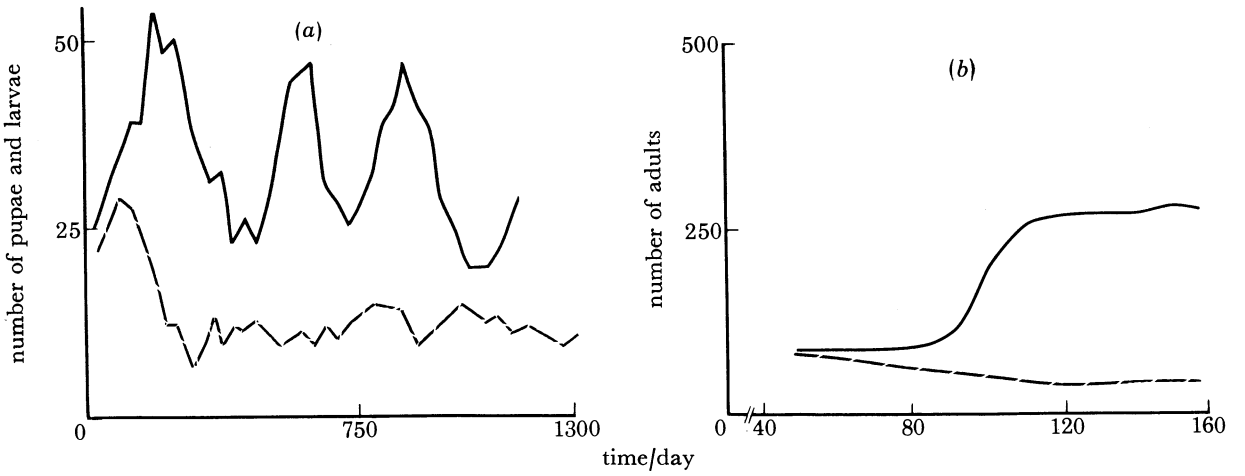

FIGURE 3. Two laboratory examples of the impact of parasitic infections on the population growth of their host: (*a*) *Tribolium casteneum* infected with the protozoan parasite *Adelina triboli* (data from Park (1948)); (*b*) *Laemophloeus minutus* infected with the protozoan *Mattesia dispora* (data from Finlayson (1950)). In both cases, the solid lines depict the uninfected population, and the dashed lines depict the infected population.

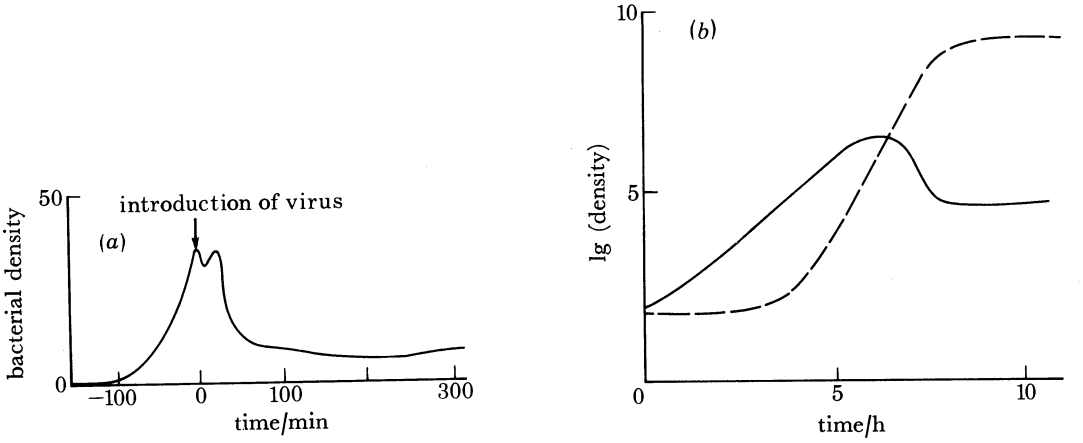

FIGURE 4. Two figures illustrating the impact that bacteriophages can have on their bacterial hosts: (*a*) a population of *Escherichia coli* infected with T4 phage virus (data from Doermann (1948); the arrow shows when the virus was introduced into the bacterial population); (*b*) a population of *Salmonella typhi* (Vi-type A) infected with the phage virus Vi-phage A (data from Anderson (1957); the solid line depicts the density of bacteria, and the dashed line the density of phage particles). Notice that in (*b*) the phage population does not 'take off' until the bacterial host population exceeds some threshold value.

birth rate, a, is independent of whether or not the host is infected; thus the net birth rate is $a(X+Y)$. The way in which the populations of susceptibles and infecteds change as a result of various kinds of gain and loss terms is depicted schematically in figure 5. Susceptible individuals are gained by birth ($a(X+Y)$) and by recovery of infecteds (γY), and are lost by natural death (bX) or by acquisition of infection (βXY). The rate of change of $X(t)$ is thus

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - bX - \beta XY + \gamma Y. \tag{12}$$

The rate of change of $Y(t)$ is, as above, equation (4), but repeated here for clarity,

$$\mathrm{d}Y/\mathrm{d}t = \beta XY - (\alpha + b + \gamma)\, Y. \tag{13}$$

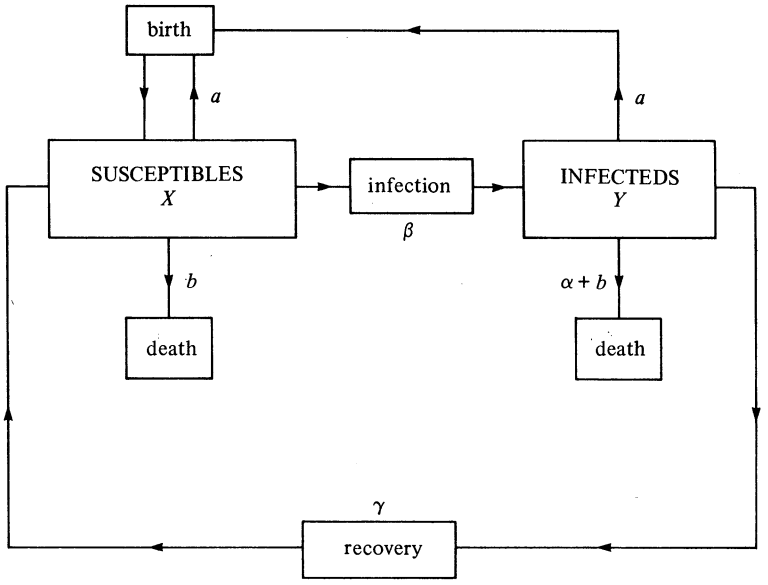

FIGURE 5. Schematic representation of the assumptions embodied in model A. The model is defined more explicitly by equations (12)–(14), and the various rate parameters are as listed in appendix A.

The rate of change of the total population of hosts, H, is obtained by adding equations (12) and (13) to give

$$dH/dt = rH - \alpha Y. \tag{14}$$

Here we have defined $r = a - b$; r is the intrinsic growth rate of the disease-free host population, and we assume $r > 0$. Any two of these three equations (in conjunction with the identity $H = X + Y$) constitute a complete description of the dynamical behaviour of the system. We usually choose to work with equations (13) and (14).

In equations (12)–(14), the description of the transmission of parasitic infections remains as in § 4, and the parasite's basic reproductive rate, R, is still defined by equation (6). Likewise the threshold desnity, H_T, of the host population for maintenance of the infection remains as defined by equation (10); the criterion $H > H_T$ (with H_T given by equation (10)) equivalently follows directly from the requirement that the right side of the equation (13) be positive for small Y. In contrast with the situation in §4, however, the host population, H, is not a predetermined constant, but is a dynamic variable; in the absence of the disease, $H(t)$ increases exponentially at the rate r (as can be seen directly from equation (14) with $Y = 0$). Thus, if the host population is initially below the threshold value ($H < H_T$ and $R < 1$), it grows exponentially until it eventually does attain a density high enough to sustain the infection. When this happens ($H > H_T$ and $R > 1$), one of two things follows.

(i) If the parasite is sufficiently pathogenic,

$$\alpha > r, \tag{15}$$

it regulates the host population at a stable equilibrium level, H^* (see appendix B). This disease-controlled equilibrium population is

$$H^* = \frac{\alpha(\alpha + b + \gamma)}{\beta(\alpha - r)}, \tag{16}$$

or, equivalently (from equation (10)),

$$H^* = \left(\frac{\alpha}{\alpha - r}\right) H_T. \tag{17}$$

Equation (17) makes it explicit that H^* must exceed H_T, although $H^* \approx H_T$ if $\alpha \gg r$. Of this equilibrium population of hosts, the fraction infected (that is, the prevalence, $y = Y/H$) follows directly from equation (14):

$$y^* = r/\alpha. \tag{18}$$

Notice that, if α is very much larger than r, the prevalence of the infection will be very low, and stochastic effects may extinguish the disease if the equilibrium host density, H^*, is low.

(ii) Conversely, if equation (15) is not satisfied,

$$\alpha < r, \tag{19}$$

the disease is not able to regulate the host population to a stable level. This is biologically obvious; if births exceed deaths, even for infected hosts ($a > b + \alpha$ and $r > \alpha$), the disease cannot halt population growth. In this case, the system eventually settles to a state in which the total population grows exponentially, at a rate ρ, which is less than r:

$$\rho = r - \alpha. \tag{20}$$

Asymptotically, the host population becomes very large ($H \gg H_T$), and essentially all hosts are infected ($Y^* \to H^*$; $y^* \to 1$). The number of susceptibles remains roughly constant at $X^* \approx (a+\gamma)/\beta$, thus constituting an ever diminishing fraction of the exponentially growing total population (see appendix B). This 'run away' of the host population for $r > \alpha$ is a consequence of the omission of any density-dependent effects, other than the disease itself, in our model. In reality, other factors, such as resource limitation, will limit population growth, and the prevalence of infection will rarely, if ever, approach unity.

The relation between the various ecological and epidemiological parameters and the overall behaviour of the host–parasite system is explored in figure 6, which shows how the equilibrium host population, H^*, and prevalence of infection, y^*, depend on the parameters α, b, γ and β.

Figure 6*a* shows H^* as a function of the pathogenicity parameter α (equation (16)). As α first exceeds the intrinsic growth rate, r, of the host population (equation (15)), increasing pathogenicity results in a greater degree of depression of the host population. Eventually, however, there comes a point when the rate of loss of infected hosts begins to have a detrimental effect on disease transmission, and beyond this point increased values of α make for less depression of the host population. This suggests that, in selecting a pathogen for biological control of an invertebrate pest species, we should not simply seek the most pathogenic (largest α) agent available, but instead try to estimate the optimal pathogenicity (corresponding to the trough in figure 6*a*) for sustained control. This point is elaborated elsewhere (Anderson 1979*b*).

Figure 6*b* illustrates the corresponding relation between prevalence, y^*, and pathogenicity, α, equation (18). Here y^* decreases steadily with increasing α, suggesting that low levels of prevalence of infection in natural populations may not simply reflect poor transmission, but may betoken relatively high pathogenicity. Low prevalence levels are not inconsistent with the disease actually regulating the host population.

Figure 6*c* depicts H^* as a function of the recovery rate γ, showing that high recovery rates result in relatively large host populations. Conversely, high transmission efficiencies (large

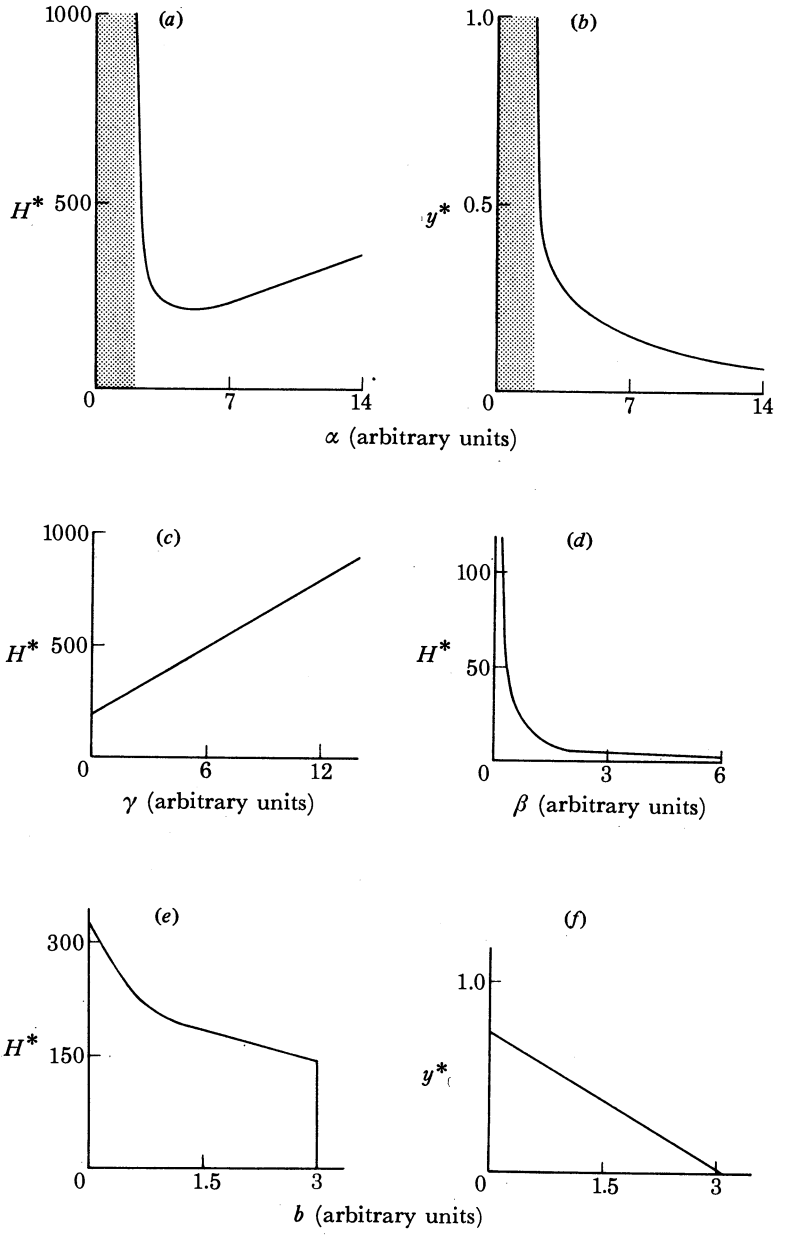

FIGURE 6. These figures illustrate the relation between the equilibrium density of hosts H^* or the equilibrium prevalence of infection y^* and various of the rate parameters in model A: (*a*) H^* as a function of parasite pathogenicity, α (with $a = 2$, $b = 1$, $\gamma = 0.1$ and $\beta = 0.05$); (*b*) the corresponding plot of y^* against α; (*c*) H^* as a function of the recovery rate, γ (with $\alpha = 3$ and the other parameters as in (*a*)); (*d*) H^* plotted against the transmission coefficient, β (with again $a = 2$, $b = 1$, $\gamma = 0.1$ and $\alpha = 3$); (*e*) H^* plotted against the natural mortality rate, b, of the hosts (with $a = 2$, $\gamma = 0.1$, $\beta = 0.05$ and $\alpha = 4$); (*f*) y^* as a function of b (with the other parameter values as in (*e*)).

values of β) lead to relatively low host populations, as demonstrated in figure 6*d*. The natural death rate, b, of hosts also influences H^* and y^*, as shown in figure 6*e*,*f* respectively, with higher death rates understandably resulting in lower values of the equilibrium host population and the prevalence of infection.

More broadly, rapid turnover in the host population, whether produced by the disease (α)

or by natural causes (b), tends to reduce the equilibrium abundance of a disease organism (see figure 6b,f). Just as high rates of reproduction are characteristic of free-living organisms that persist in unstable habitats ('r strategists'), so, in an analogous manner, relatively high transmission efficiencies (large β) are required for the persistence of parasites whose host populations turn over rapidly (unstable parasite habitat).

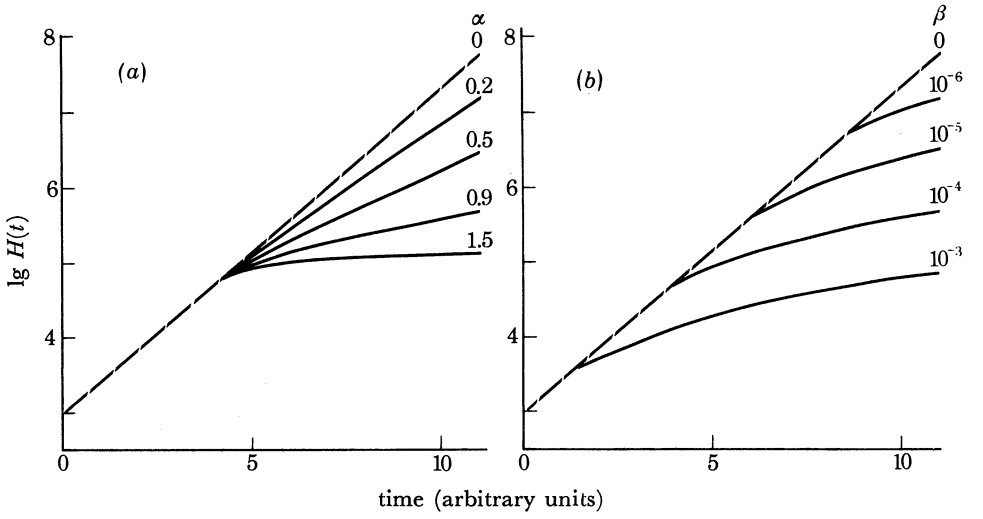

FIGURE 7. The dynamical behaviour of the host population H is shown as a function of time t, for (a) various values of the parasite pathogenicity α, and (b) various values of the transmission coefficient β. In both figures, the dashed line is for the purely exponential growth of the host population occurring in the absence of infection ($\alpha = 0$ or $\beta = 0$); since the host population is plotted logarithmically, such exponential growth shows up as a straight line. The rate parameters have the values $r = 1.0$, $b = 3.0$, $\gamma = 0.01$, $\beta = 0.001$ (in (a)), and $\alpha = 0.9$ (in (b)). The host population is initially $H(0) = 1000$.

The threshold density for disease maintenance also depends on the parameters α, b, γ and β, as described by equation (10). Some of the dynamical properties discussed above are illustrated in figure 7, which shows the host population, H, as a function of time, t, for various values of α (figure 7a) and for various values of β (figure 7b). In all cases, the initial host population is below the threshold value, H_T, and grows exponentially until it exceeds H_T; the subsequent dynamical fate of the population (regulation to a constant value, or exponential growth at a diminished rate) depends on the interplay among the parameters, as shown.

There remains the central biological question, do natural populations of invertebrates typically have microparasitic infections capable of regulating them ($\alpha > r$), or not?

Viruses, bacteria, protozoans and fungi certainly are often observed in natural communities, particularly within arthropod species (Tinsley 1979; Overstreet 1978; Crawford & Kalmakoff 1977; Breed & Olson 1977; Beesley 1977; Smith 1976; Tinsley & Entwistle 1974; Andrews & Castagna 1978; Ignoffo *et al.* 1976; Zacharuk & Tinline 1968; Whitcomb *et al.* 1966; David 1965; Tanada 1964; Aizawa 1963; Bergold 1953, 1958; Steinhaus 1958, 1963; Bird 1955; Whitcomb & Tully 1979). Where long-term studies exist, such diseases are seen to persist, although their prevalence may vary seasonally and may be characterized by sudden changes or epidemics (Beesley 1977, 1978; Andrews & Castagna 1978; Zelazny 1977; Tanada & Omi 1974; Pramer & Al-Rabiai 1973; Stairs 1972; Putman 1970; Bird & Elgee 1957; Bird & Whalen 1953).

Unfortunately, very few field studies have yielded estimates both of the rate of disease-induced mortality and of the natural rate of increase of the host population, so that it is hard to say how frequently α exceeds r. Laboratory studies, however, clearly indicate that many of these disease agents are highly pathogenic. The data compiled in table 1 testify to this point, and, furthermore, suggest that viral and bacterial agents tend to be the most pathogenic. The disease-induced death rates, α, in table 1 are typically about an order of magnitude greater than the corresponding natural death rates, b; the effective birth rates a for these invertebrates are harder to determine. The incompleteness of the information is compounded by the fact that laboratory studies tend to underestimate natural mortality rates (as many natural causes of death are not present in the laboratory), and by the biases introduced by invertebrate pathologists' interest in the more pathogenic disease agents by virtue of their possible use in biological control programmes. Some of these problems are discussed more fully by Hassell *et al.* (1976) and by Tinsley (1979).

All these caveats having been issued, it remains plausible that many of the infections catalogued in table 1 may contribute, wholly or in part, to the regulation of their invertebrate host populations.

We now proceed to add a variety of realistic refinements to our basic model A.

TABLE 2. PARASITE-INDUCED REDUCTION OF HOST REPRODUCTIVE POTENTIAL

		rate of reproduction† (per host)		
pathogen	host	uninfected, a	infected, $a(1-f)$	reference
Nosema whitei	*Tribolium castaneum*	1.9 per week	1.7 per week	Milner (1972*a*)
Amblyospora sp.	*Culex salinarius*	2.8 per lifespan	2.4 per lifespan‡	Andreadis & Hall (1979)
non-inclusion virus	*Panonychus citri*	1.8 per week	0.93 per week	Gilmore & Tashiro (1966)
Mattesia grandis	*Anthonomus grandis*	1.7 per week	1.2 per week	McLaughlin (1965)
Nosema stegomyiae	*Anopheles albimanus*	2.4 per first gonotrophic cycle	2.3 per first gonotrophic cycle	Anthony *et al.* (1973)
rhabion virus (R.O.V.)	*Oryctes rhinoceros*	1.9 per week	0.45 per week	Zelazny (1973)

† Assumes 1:1 sex ratio. ‡ No difference between infected and uninfected lifespans.

6. PARASITE-INDUCED REDUCTION OF HOST REPRODUCTION: MODEL B

Many microparasites of invertebrates not only increase the death rate but also decrease the reproductive rate of infected hosts. For example, as shown in table 2, many protozoan infections substantially reduce the reproductive capabilities of the host (McLaughlin 1965, 1971).

The basic model A may be modified to incorporate this effect by taking the birth rate of infected hosts to be $a(1-f)$. Here $f\,(1 \geqslant f \geqslant 0)$ measures the severity of the parasite's effect on host reproduction: $f = 1$ corresponds to elimination of all reproduction by infected hosts (as happens, for example, when the parasite castrates the host); $f = 0$ is the opposite extreme where there is no effect. In this way, we obtain model B:

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - bX - \beta XY + (\gamma - fa)\,Y; \tag{21}$$

$$\mathrm{d}Y/\mathrm{d}t = \beta XY - (\alpha + b + \gamma)\,Y; \tag{22}$$

$$\mathrm{d}H/\mathrm{d}t = rH - (\alpha + fa)\,Y. \tag{23}$$

The dynamics are analysed in appendix B. The basic reproductive rate, R, of the parasite

and the threshold host density, H_T, for maintenance of the infection remain as in model A. The criterion for the parasite to be able to regulate its host population (after the threshold is exceeded) is, however, modified from equation (15) to the less stringent requirement

$$\alpha > a(1-f) - b. \tag{24}$$

We recall the underlying biological reason for this condition, namely that deaths should outrun births for infected hosts, which makes it clear why such depression of the birth rate should facilitate regulation.

If equation (24) is satisfied, the host population settles to the stable equilibrium value

$$H^* = \left(\frac{\alpha+fa}{\alpha+fa-r}\right) H_T, \tag{25}$$

with H_T defined by equation (10). The equilibrium prevalence of infection is

$$y^* = r/(\alpha+fa). \tag{26}$$

Other things being equal, both H^* and y^* are lower than when parasites have no effect on host reproduction ($f = 0$).

Two special instances of equation (24) are of interest.

First, if infected hosts are unable to reproduce ($f = 1$), equation (24) is automatically satisfied (even if $\alpha = 0$), and the disease is always able to regulate the host population. This applies to the many pathogens that castrate infected hosts; one example is the protozoan *Pleistophora cragoni*, which castrates the sand shrimp *Cragon nignianda* (Breed & Olson 1977).

Secondly, a parasite that reduces host reproduction without affecting host survival ($\alpha = 0$) can regulate its host population provided that $b > a(1-f)$. This general circumstance is illustrated by species of the protozoan parasite *Amblyospora*, which reduce the rate of reproduction of the mosquito host *Culex salinarius*, but have no apparent effect on survival.

In short, parasites that reduce reproduction in infected hosts have no effect on threshold densities, but they can more easily regulate host populations, and the equilibrium host densities are lower.

7. Vertical transmission: model C

So far, we have considered pathogens that are transmitted horizontally between hosts. But many microparasites of invertebrates are transmitted vertically from parent to unborn offspring.

Two basic types of vertical transmission are conventionally distinguished. In *transovarial* transmission the pathogen gains entry to the egg or embryo within the host, via infection of the reproductive organs of the parent. For example, the eggs of the insect *Plodia interpunctella* often contain spores of the protozoan *Nosema plodiae*, with the infection being acquired during egg formation (Kellen & Lindegren 1973). In certain genera of the microsporidian protozoan parasites, transovarial transmission appears to be the principal mechanism by which infection is passed from host to host (Chapman *et al.* 1966; Kellen *et al.* 1966; Andreadis & Hall 1979). Similarly, many virus pathogens of insects rely on this form of transmission (Smith 1967, 1976; Hukuhara 1962). In *transovum* transmission the pathogen is adsorbed on, or contaminates, the exterior of the egg during the birth process; infection results when the newly hatched host contacts or eats its own egg case. Transovum transmission is common among pathogens of arthropods (Neelgund & Mathad 1978; Smith 1976; Doane 1969; Steinhaus 1963).

Transovarial and transovum transmission both have the same effect on the dynamics of the host–parasite association, in that offspring of infected parents are more likely to become infected than are offspring of uninfected parents. The efficiency of vertical transmission, however, varies widely among host–parasite associations. Thus, only 23% of the progeny of the silkworm *Bombyx mori* infected with a cytoplasmic polyhedrosis virus acquired infection (Hukuhara 1962). In contrast, 90% of the progeny of the mosquito *Culex salinarius* infected with the protozoan *Amblyospora* developed the disease (Andreadis & Hall 1979).

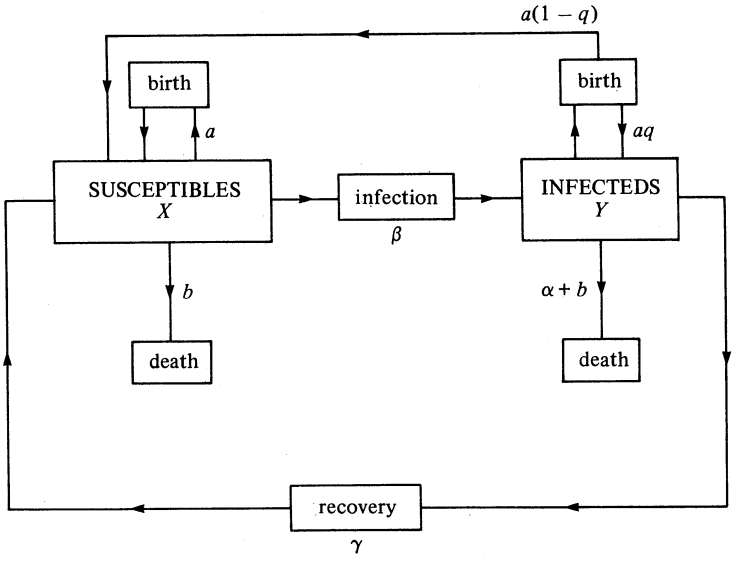

FIGURE 8. Schematic representation of the assumptions embodied in model C. The model is defined more explicitly by equations (27)–(29), and the various rate parameters are as listed in appendix A.

To incorporate vertical transmission in the basic model A, we assume that a fraction $q(1 \geqslant q \geqslant 0)$ of the offspring of infected hosts pass directly into the infected class. Thus the net birth rate from infecteds, aY, is apportioned between $a(1-q)\,Y$ appearing as new susceptibles, and aqY appearing as new infecteds. In addition, horizontal transmission is included as before. The resulting model C is depicted schematically in figure 8, and can be written

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - bX - \beta XY + (\gamma - aq)\,Y, \tag{27}$$

$$\mathrm{d}Y/\mathrm{d}t = \beta XY - (\alpha + b + \gamma - aq)\,Y, \tag{28}$$

$$\mathrm{d}H/\mathrm{d}t = rH - \alpha Y. \tag{29}$$

The dynamical behaviour of this set of equations is analysed in appendix B. The basic biological considerations of the parasite's reproductive rate, R, and the host threshold density, H_T, are now modified by the ability of a single infected host to produce secondary cases by vertical as well as by horizontal transmission. The threshold host density, H_T, above which Y will increase from small initial values, is (as can be seen from equation (28))

$$H_T = (\alpha + b + \gamma - aq)/\beta. \tag{30}$$

Thus the presence of vertical transmission ($q \neq 0$) tends to lower H_T, making it easier for the parasite to persist at relatively low host densities. We might therefore expect vertical

transmission to be characteristic of microparasitic diseases that are endemic in low-density invertebrate populations, or in seasonal environments where host densities are low during certain months of the year (Tinsley 1979; Smith 1976).

The criterion for the disease to be able to regulate its host population is again equation (15), $\alpha > r$, as in the basic model A. The stable equilibrium host population, H^*, is again given by equation (17), but with equation (30) replacing equation (10) for H_T, so that

$$H^* = \frac{\alpha(\alpha+b+\gamma-aq)}{\beta(\alpha-r)}. \tag{31}$$

Thus H^* is lower than it is without vertical transmission ($q = 0$). The prevalence, y^*, is again given by equation (18), $y^* = r/\alpha$.

Conversely, if $\alpha < r$ (equation (19)), the disease does not regulate the host population, which asymptotically grows exponentially at the diminished rate, $\rho = r-\alpha$, of equation (20). Notice that it can, in principle, be that the threshold density, H_T, of equation (30) is negative (if $aq > \alpha+b+\gamma$). If this happens, it necessarily follows that $r > \alpha$, and the disease will become established at arbitrarily low host levels but is not able to regulate the host population.

Some people have suggested that certain species of microparasites rely entirely on vertical transmission, with horizontal transmission either absent or negligible (Andreadis & Hall 1979; Kellen *et al.* 1966; Chapman *et al.* 1966). We show in appendix B, however, that, in the absence of horizontal transmission ($\beta = 0$), the prevalence of infection asymptotically tends to zero under all circumstances. Some degree of horizontal transmission is therefore essential for a parasitic infection to persist.

In short, vertical transmission lowers threshold host densities (possibly to zero) and equilibrium host levels (if they exist), but does not directly affect the ability of the parasite to regulate its host population. Microparasitic infections cannot be maintained purely by vertical transmission.

8. Latent periods of infection: model D

Many pathogens undergo an incubation or latent period within the host before beginning to produce transmission stages (for horizontal transmission) or to contaminate or infect unborn progeny of the host (for vertical transmission). Some examples of such incubation periods are listed in table 3.

The basic model A can be modified to incorporate this feature, by separating the infecteds into two classes: the latent class (infected but not yet infectious), comprising $M(t)$ individuals at time t; and the infectious class, comprising $Y(t)$ individuals. Hosts are assumed to pass from latent into infectious at a constant individual rate, v, so that the typical duration of the latent period is $1/v$ (in reality, the latent period is more often a fixed time interval, but our rough treatment captures the essentials). This model D is represented schematically in figure 9, and obeys the following equations:

$$\mathrm{d}X/\mathrm{d}t = a(X+M+Y) - bX - \beta XY + \gamma Y, \tag{32}$$

$$\mathrm{d}M/\mathrm{d}t = \beta XY - (b+v)M, \tag{33}$$

$$\mathrm{d}Y/\mathrm{d}t = vM - (\alpha+b+\gamma)Y, \tag{34}$$

$$\mathrm{d}H/\mathrm{d}t = rH - \alpha Y. \tag{35}$$

We need only three of these four equations, along with the identity $H = X+M+Y$.

TABLE 3. INCUBATION PERIODS $(1/v)$ OF SOME VIRAL AND PROTOZOAN PATHOGENS

parasite	host	incubation period/day	reference
viruses			
granulosis virus	*Heliothis ormigera*	2–5	Whitlock (1977)
nuclear-polyhedrosis virus	*Heliothis ormigera*	3–5	Whitlock (1977)
non-inclusion virus	*Panonychus citri*	2–3	Gilmore & Tashiro (1966)
non-inclusion virus	*Panonychus ulmi*	3	Putman (1970)
nuclear-polyhedrosis virus	*Telea polyphemus*	5–10	Smith & Wyckoff (1951)
protozoa			
Vairimorpha necatrix	*Heliothis zea*	7	Fuxa & Brooks (1979)
Mattesia grandis	*Anthonomus grandis*	7	McLaughlin (1965)

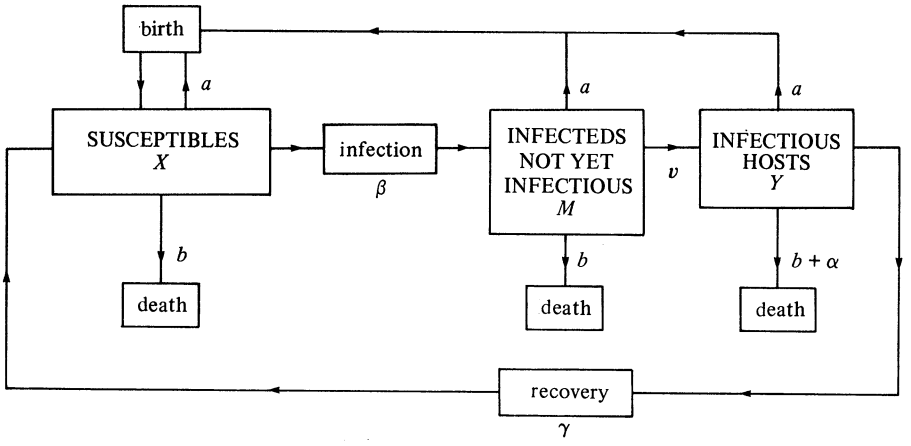

FIGURE 9. Schematic representation of the assumptions embodied in model D. The model is defined more explicitly by equations (32)–(35), and the various rate parameters are as listed in appendix A.

The analysis of this system is outlined in appendix C. Beginning with the basic population biology of the situation, we note that the parasite's reproductive rate R, as defined and discussed in § 4, is here

$$R = \left(\frac{\beta H}{\alpha+b+\gamma}\right)\left(\frac{v}{b+v}\right). \tag{36}$$

The threshold host density for persistence of the disease, given by the requirement $R > 1$, is now

$$H_T = \left(\frac{\alpha+b+\gamma}{\beta}\right)\left(\frac{b+v}{b}\right). \tag{37}$$

Thus the threshold exceeds that in the absence of latency, equation (10), by the factor $(b+v)/v$. Biologically, this factor arises from the possibility that infected individuals will die before becoming infectious; the factor is large if the latent period is relatively long (v small in relation to b), and is essentially unity if the latent period is relatively short (v large in relation to b).

The condition for the disease to be able to regulate the host population is

$$\alpha > [1+(\alpha+b+\gamma)/v]. \tag{38}$$

If equation (38) is satisfied, the host population may settle to a stable equilibrium value

$$H^* = \frac{\alpha}{\alpha - r\,[1+(\alpha+b+\gamma)/v]}\,H_T. \tag{39}$$

The prevalence, as before, is given by equation (18). Alternatively, the system may exhibit stable cyclic oscillations, centred around the equilibrium values. Broadly, there will tend to be an equilibrium point if the latent period is either very short (large v) or very long (small v, but with equation (38) satisfied), or if the pathogenicity, α, is large compared to other rate parameters; stable cyclic behaviour can ensue if α and v are comparable in magnitude, and both significantly larger than the vital rates (a, b, $r = a - b$) of the host population. In the absence of data to guide the discussion, further consideration of the circumstances under which the disease-regulated host population exhibits a stable point or stable cyclic behaviour is relegated to appendix C.

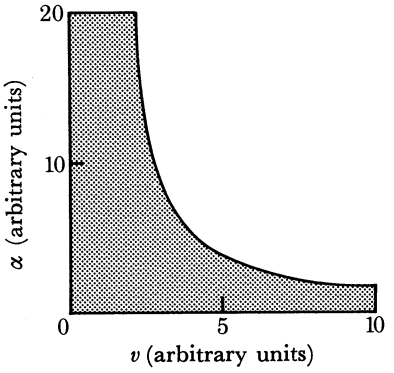

FIGURE 10. For model D, this figure shows the domain of α–v parameter space in which the disease regulates the host population in a stable or a cyclic equilibrium state (the unshaded region), and the domain in which the host population grows exponentially, with the disease unable to regulate it (the shaded region). As always, α represents the parasite pathogenicity and v the rate at which infected hosts become infectious ($1/v$ measures the duration of the latent period); the other rate parameters are taken here to be $a = 3$, $b = 1$, $\gamma = 0.1$.

On the other hand, if equation (38) is not satisfied, the host population continues to grow exponentially, albeit at a rate less than the disease-free rate, r, until limited by other ecological factors.

The condition, equation (38), for existence of disease-controlled equilibrium is more restrictive than the simple condition $\alpha > r$ of model A. Moreover, the equilibrium host population is larger than for model A. Both these effects become increasingly pronounced as the latent period lengthens (that is, as $1/v$ increases). As illustrated in figure 10, pathogens with long incubation periods ($1/v$ large) are unlikely to regulate host population growth unless they are highly pathogenic (α large).

9. Disease and stress: model E

Various people have argued that the pathogenicity of many parasites of invertebrates depends on the degree to which the host population is under 'stress' from prevailing environmental conditions, such as food shortages, overcrowding, or extremes of temperature or humidity (David & Gardiner 1965; Hurpin 1968; Bucher & Harris 1968; Breed & Olson 1977). More specifically, the observation that epidemic outbreaks of disease typically occur when host density is high has led to the suggestion that stress produced by overcrowding results in increased pathogenicity (Steinhaus 1958; Tanada 1964).

But we have seen that there are threshold host densities, below which a pathogen cannot maintain itself. Thus, on simple dynamical grounds, epidemic outbreaks of disease will more commonly be observed in high-density host populations. No change in pathogenicity need be invoked to explain such patterns (Bailey 1973).

On the other hand, the immunological competence of vertebrate hosts is indeed closely correlated with their nutritional state (Scrimshaw *et al.* 1968; Mims 1977). It is thus plausible that malnourished invertebrates are less able to mount a response (whether immunological or non-specific) to infection than are their well nourished counterparts. For example, Bucher & Harris (1968) demonstrated experimentally that a cytoplasmic virus of the insect *Calophasia lunula* was more pathogenic to malnourished individuals than to well fed controls; conversely, the same authors showed that the nutritional state of this same species of insect host had no effect on the pathogenicity of a highly pathogenic nuclear-polyhedrosis virus.

Thus we have at least two independent explanations for the observed association between epidemics and high host density, one based on population dynamics and transmission thresholds and the other on density-dependent pathogenicity. As discussed below, we believe that most of the observed phenomena can be explained by dynamical considerations. Even so, the consequences of density-related pathogenicity deserve exploration, and this we now do.

For simplicity, we assume that the pathogenicity of the parasite is linearly related to host density:

$$\alpha(H) = \hat{\alpha}H. \tag{40}$$

This assumes that the average level of nutrition declines in direct proportion to the host density, and that this decline in nutritional state is linked with pathogenicity. Replacing α by $\hat{\alpha}H$ in model A, we arrive at model E:

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - bX - \beta XY + \gamma Y; \tag{41}$$

$$\mathrm{d}Y/\mathrm{d}t = \beta XY - (b+\gamma+\hat{\alpha}H)\,Y; \tag{42}$$

$$\mathrm{d}H/\mathrm{d}t = rH - \hat{\alpha}HY. \tag{43}$$

The analysis of this set of equations is sketched in appendix D. Following the lines laid down in § 4, we can see that the basic reproductive rate R of the parasite is now

$$R = \beta H/(b+\gamma+\hat{\alpha}H). \tag{44}$$

It immediately follows that R is always less than unity if $\hat{\alpha} > \beta$; that is, the disease can only be maintained ($R > 1$) if

$$\beta > \hat{\alpha}. \tag{45}$$

If this condition is met, the threshold host density (the value of H for which $R = 1$) is

$$H_{\mathrm{T}} = (b+\gamma)/(\beta-\hat{\alpha}). \tag{46}$$

Provided that equation (45) is satisfied, the disease will always regulate the host population to a stable equilibrium value, which can be written (with use of equation (46)) as

$$H^* = H_{\mathrm{T}}\left[1+\frac{\beta r}{\hat{\alpha}(b+\gamma)}\right]. \tag{47}$$

This relation between H^* and the parameter $\hat{\alpha}$ (which defines the severity of the association between pathogenicity and host density) is illustrated in figure 11. As $\hat{\alpha}$ increases, H^* falls

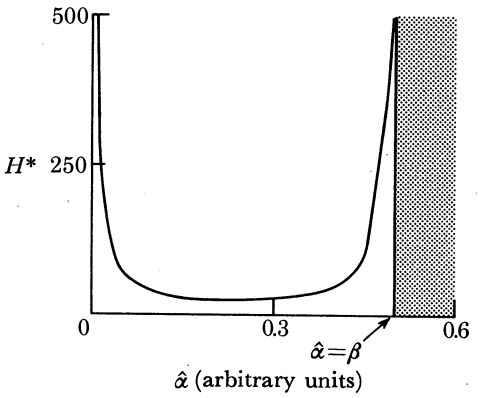

FIGURE 11. For model E, this figure illustrates the relation between the equilibrium density of the host population, H^*, and the parameter $\hat{\alpha}$, which measures the severity of the association between the rate of disease-induced host mortality and host density (see equation (40)). The other rate parameters have the values $a = 4$, $b = 1$, $\gamma = 0.1$ and $\beta = 0.5$. The general features illustrated here are discussed more fully in the text.

until a point is reached where the high death rate of infected hosts decreases the transmission efficiency of the infection. Beyond this point, H^* rises until the expected lifespan of an infected host becomes too short for effective transmission to occur; for larger $\hat{\alpha}$ ($\hat{\alpha} > \beta$) the parasite cannot persist ($R < 1$).

In contrast to the earlier models (where ability of the parasite to regulate its host population depended on the mortality rates), the fate of this system depends wholly on equation (45). If the transmission efficiency is sufficiently high ($\beta > \hat{\alpha}$), the disease persists and regulates the host population; if not ($\beta < \hat{\alpha}$), the disease dies out. The underlying biological reason is simple: when pathogenicity is related to host abundance, the parasite will always be capable of regulating population growth, and its main problem is to transmit itself fast enough to counter-balance the rapid death of infected hosts.

10. DENSITY-DEPENDENT CONSTRAINTS: MODEL F

Until now, the only density-dependent effects acting to regulate the host population have come from the parasitic infection. When these effects were too weak, our host populations manifested unbounded exponential growth. In reality, of course, other density-dependent constraints, such as resource limitation or the action of predators, will sooner or later limit population growth. Indeed, in natural situations pathogens will often act in conjunction with other regulatory factors (Lack 1954; Anderson 1980*b*; Park 1948; Finlayson 1950); two examples were given in figure 3.

We now extend the basic model A, to include other density-dependent effects. These density-dependent constraints (induced by resource limitation, predation, or whatever) are assumed to act on the natural death rate, b, of the host, in a manner linearly proportional to host density, such that

$$b(H) = b_0 + sH. \tag{48}$$

The parameter s measures the severity of the density-dependent constraints on host population growth. We thus arrive at model F, described by the set of equations

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - (b_0+sH)X - \beta XY + \gamma Y, \tag{49}$$

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$$dY/dt = \beta XY - (\alpha + b_0 + sH + \gamma)\, Y, \tag{50}$$

$$dH/dt = (a - b_0 - sH)\, H - \alpha Y. \tag{51}$$

In the absence of disease ($Y = 0$), the host population obeys a logistic equation, having a stable equilibrium value

$$H^* = K \equiv (a - b_0)/s. \tag{52}$$

Here K is the usual 'carrying capacity'.

The dynamical behaviour of equations (49)–(51) is outlined in appendix E. As always, the essentials can be understood by considering the basic reproductive rate R of the parasite, which here is

$$R = \beta H/(\alpha + b_0 + \gamma + sH). \tag{53}$$

If $\beta < s$, then R is ineluctably less than unity, and the disease can never be maintained; the host population will settle to the disease-free equilibrium value of equation (52). Conversely, if

$$\beta > s, \tag{54}$$

the disease can be maintained ($R > 1$) for sufficiently high values of H, $H > H_T$. This threshold host density can be obtained by putting $R = 1$ in equation (53), and is

$$H_T = (\alpha + b_0 + \gamma)/(\beta - s). \tag{55}$$

We now encounter a second, and new, constraint, associated with the other density-dependent effects; the infection can only be maintained if

$$H_T < K. \tag{56}$$

Here H_T is given explicitly by equation (55), and K by equation (52). If this inequality, equation (56), is violated (that is, if $H_T > K$), the threshold host density for maintenance of the parasitic infection cannot be attained, because carrying capacity constraints prevent the host population from exceeding the level K.

If both the criteria of equations (54) and (56) are satisfied, the parasite will become established within the host population, and will regulate it to a stable equilibrium value, H^*, which is less than the disease-free level, $H^* < K$ (the explicit expressions for H^* and for the prevalence of infection are given in appendix E). We define a quantity d ($1 \geqslant d \geqslant 0$) to measure the degree to which the host population is depressed below the disease-free level,

$$d \equiv 1 - H^*/K. \tag{57}$$

Figure 12 illustrates these different dynamical possibilities, showing dynamical trajectories of the system in the 'phase plane' of $Y(t)$ and $X(t)$ values. In figure 12*a* H_T lies below K, and the system settles to a stable equilibrium with the disease maintained in a total host population less than K. In figure 12*b*, H_T lies above K, and the system settles to the disease-free equilibrium state at $H^* = K$, independent of the initial conditions.

Equation (54) makes it clear that the ability of the parasite to persist within the host population, depressing it below its disease-free level, depends on the transmission efficiency, β, being large enough. Similarly, equation (56) in conjunction with equations (52) and (55) requires that α be not too large. These considerations are illustrated explicitly in figures 13 and 14.

Figure 13 shows the domain wherein the parasite and host coexist, as a function of the

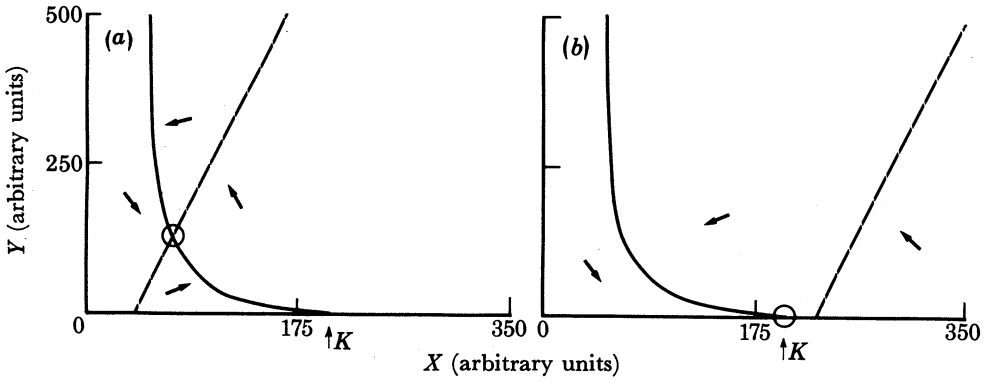

FIGURE 12. These are phase–space plots, illustrating the dynamical behaviour of model F, in which there are density-dependent constraints on the growth of the host population (in addition to the effects of disease-induced host mortality). In both (*a*) and (*b*), the dashed line is the isocline along which $dY/dt = 0$, and the solid line is the isocline along which $dX/dt = 0$; the arrows indicate the directions in which trajectories must therefore move in the various regions of this X–Y phase plane. In (*a*), the parameters have the values $a = 3$, $b_0 = 1$, $\alpha = 0.5$, $\gamma = 0.1$, $\beta = 0.05$ and $s = 0.01$ (whence $K = 200$ and $H_T = 40$); in this case, equation (56) is satisfied, and host and parasite stably coexist at the equilibrium point where the isoclines intersect. In (*b*), the parameter values are as in (*a*) except that now $\beta = 0.017$; equation (56) is no longer satisfied (corresponding to the isoclines no longer intersecting), and the system settles to the disease-free state with $H^* = K$ and $Y^* = 0$.

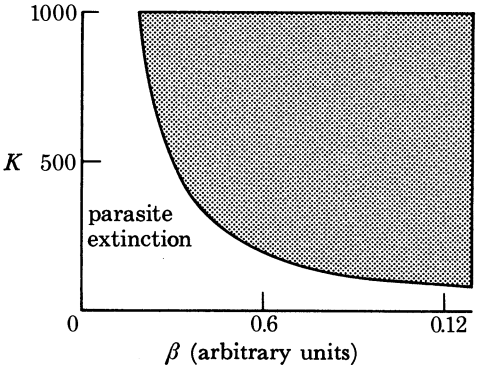

FIGURE 13. For model F, this figure displays the domain of K–β parameter space, where host and parasite coexist at a stable equilibrium point with $H^* < K$ and $Y^* > 0$ (the hatched region), and the domain of parameter space corresponding to extinction of the parasite ($H^* = K$, $Y^* = 0$; the unhatched region). The other parameter values are here taken to be $\alpha + b + \gamma = 10$ (these parameters enter only in this combination) and $s = 0.01$.

parameters β and K (from equation (52), K is an inverse measure of s). Coexistence is most likely when β and K are both relatively large (s relatively small).

The degree of depression of host density, d, and the prevalence of infection, y^*, are shown as functions of pathogenicity, α, in figure 14*a*, *b*, respectively. Maximum depression is attained for some intermediate pathogenicity, whereas y^* decreases steadily with increasing α. Once α becomes too large, infected hosts die before effective transmission is achieved, and the disease is unable to persist in this host population (constrained not to exceed K). Highly pathogenic organisms are likely to cause their own extinction, but not that of their hosts. Figure 14*a* evokes a theme of practical importance that has been sounded earlier in this paper and else-

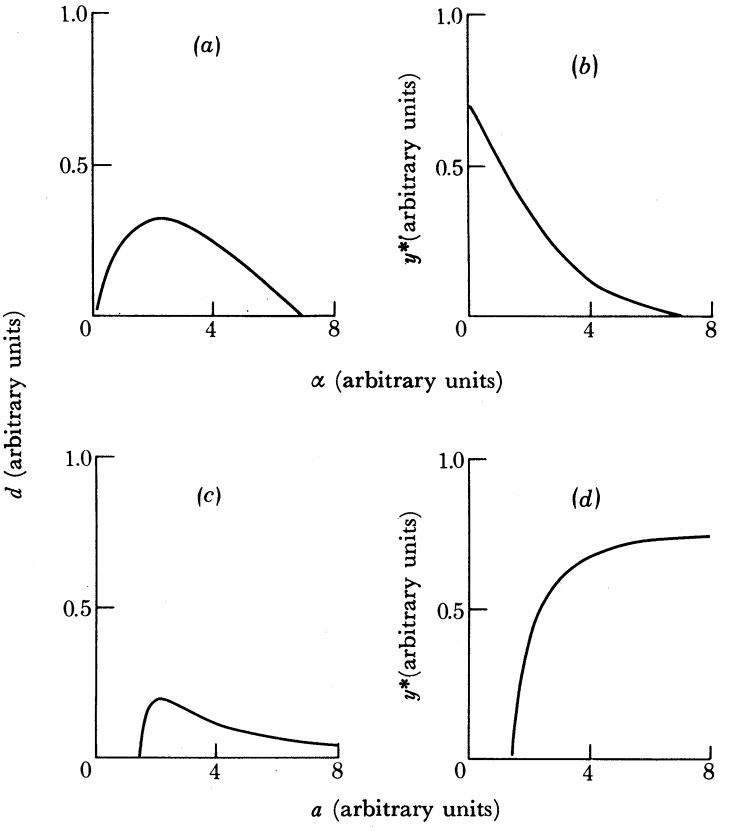

FIGURE 14. For model F, in which there are density-dependent constraints on host population growth, (*a*) shows how the degree of depression of the host population ($d = 1 - H^*/K$) varies with parasite pathogenicity, α; the other parameters have the values $a = 3$, $b = 1$, $\beta = 0.05$, $s = 0.01$ and $\gamma = 0.1$. Figure (*b*) shows the equilibrium prevalence of infection y^* as a function of α, for the same set of other parameter values. Similarly, (*c*) and (*d*) display d and y^*, respectively, as functions of the host birth rate a; the parameter values are again as in (*a*) and (*b*), and $\alpha = 0.5$. The features of these figures are discussed in the text.

where (Anderson 1979*b*; Anderson & May 1979*a*); for sustainable, equilibrium control of invertebrate populations, the optimal pathogen is one with some intermediate pathogenicity.

Figure 14*c*, *d* plots d and y^*, respectively, as functions of the host birth rate, a. The figures show that the persistence of microparasitic infections depends importantly on the rate at which susceptible hosts enter the population. As seen in figure 14*c*, high birth rates in the host population tend to offset disease-induced deaths, and thus the degree of depression, d, decreases as a increases. High birth rates also have the effect of enhancing transmission efficiency, and thus lead to high prevalence of infection, as shown in figure 14*d*.

11. FREE-LIVING INFECTIVE STAGES: MODEL G

We now extend our series of models of the host–parasite system to include a dynamical description of the populations of free-living infective stages of microparasites. These infective stages are important in the life cycle of the parasite, carrying the infection from one host to the next by horizontal, or transovum vertical, transmission. Although it is often a good approximation to assume, as we have up to now, that the net rate of gain of infected hosts is simply

proportional to the rate of encounters between susceptible and infected individuals, βXY (Anderson & May 1979*a*; Anderson & Finlay 1981), this assumption is not always adequate.

There follows a survey of salient aspects of the natural history of such free-living infective stages, with particular attention to their lifespan, rate of production and method of transmission. The dynamical consequences of this refinement are then explored.

TABLE 4. ESTIMATED LIFESPANS OF FREE-LIVING INFECTIVE STAGES OF VARIOUS PATHOGENS

(These are estimated *maximum* lifespans, which can be significantly longer than *average* lifespans.)

pathogen	type of infective stage	maximum lifespan / year	reference
viruses			
nuclear-polyhedrosis virus of *Trichoplusia ni*	polyhedra	> 6	Jacques (1969)
nuclear-polyhedrosis virus of *Gilpinia hercyniae*	polyhedra	4–9	Thomas *et al.* (1972)
nuclear-polyhedrosis virus of *Kotachalia junodi*	polyhedra	> 1	Ossowski (1959)
nuclear-polyhedrosis virus of *Orgyia pseudotsugata*	polyhedra	11	Thompson & Scott (1979)
granulosis virus of *Pieris brassicae*	capsule	2–3	David (1965)
virus of *Bombyx mori*	capsule	21	Steinhaus (1960)
non-inclusion virus of *Panonychus ulmi*	viral particle	0.02	Putman (1970)
protozoa			
Vairimorpha necatrix	spore	2	Fuxa & Brooks (1979)
Nosema whitei	spore	1–2	Milner (1972*b*)
Nosema oryzaephili	spore	0.8	Burges *et al.* (1971)
Nosema melolantha	spore	1	Hurpin (1968)
Nosema locustae	spore	5	Henry & Oma (1974)
bacteria			
Bacillus larvae	spore	0.014	Wilson (1972)
Bacillus noctuarum	spore	> 1	White (1923)
Coccobacillus acridianum	spore	2	d'Herelle (1915)
Streptococcus pluton	spore	1.5	Stephens (1957)
Bacillus thuringiensis	spore	0.8	Raun *et al.* (1966)
fungi			
Nomuraea rileyi	conidia	0.008	Ignoffo *et al.* (1976)

(*a*) *Lifespan of infective stages*

The infective stages of some microparasites are morphologically well defined. This is so for the spores of bacteria, protozoans and fungi, which often possess tough proteinaceous coats which enhance the ability of the infective agent to survive outside the host. Alternatively, rather unspecialized stages, such as the capsules, polyhedra or free particles of the viruses, may be produced (Smith 1976). The more specialized of these viral stages can be very resistant to fluctuations in physical parameters such as temperature and humidity.

As indicated by some of the entries in table 4, the tough, resistant outer coverings of many infective stages enable them to persist in the external environment for long periods of time. Certain microsporidian protozoan spores and viral polyhedra can survive for many years in the soil or litter of grassland and forest habitats (Tinsley & Entwhistle 1974; Thompson & Scott 1979; Henry & Oma 1974; Thomas *et al.* 1972). For example, David & Gardiner (1967*a*, *b*) showed that a granulosis virus of *Pieris brassicae* larvae was very stable in soil and sand, and manifested little deterioration after two years. These authors also demonstrated that the virus could not readily be washed out of the soil by heavy rainfall. Similar results were

obtained by Jaques (1969) for a nuclear polyhedrosis virus of the cabbage looper, *Trichoplusia ni*, which retained its infectivity after four years in field plots. Jaques (1964, 1967*a*, *b*) showed in general that insect baculoviruses persist in soil in inclusion bodies, which are crystalline, proteinaceous and polyhedra-shaped; these appear to adhere to the surface of soil particles, and so are prevented from being washed from the surface layers of the soil. One of the most remarkable cases of virus persistence is reported by Thompson & Scott (1979), who suggest that a nuclear polyhedrosis virus of the Douglas fir tussock moth, *Orgyia pseudotsugata*, once incorporated in the surface soil of the forest, is subject to little vertical movement in the soil and may remain active for up to 11 years.

TABLE 5. PRODUCTION OF INFECTIVE STAGES OF PATHOGENS

parasite	host	number of infective stages per host, Λ	reference
viruses			
nuclear-polyhedrosis virus	*Malacosoma* spp.	1×10^9	Stairs (1972)
nuclear-polyhedrosis virus	*Orgyia pseudotsugata*	1×10^7	Thompson & Scott (1979)
protozoa		(spores)	
Vairimorpha necatrix	*Heliothis zea*	1.7×10^{10}	Fuxa & Brooks (1979)
Nosema heliothidis	*Heliothis zea*	2×10^9	Cole (1970)
Nosema locustae	*Melanoplus bivittatus*	1×10^9	Henry (1971)
Mattesia grandis	*Anthonomus grandis*	1.7×10^6	McLaughlin & Bell (1970)
Glugea gasti	*Anthonumus grandis*	6.8×10^7	McLaughlin & Bell (1970)
Nosema pyrausta	*Ostrinia nubilalis*	9×10^7	Raun *et al.* (1960)

It is important to note, however, that, although the reported maximum lifespan of these infective stages may be long, the *expected* lifespan of an individual stage will often be many orders of magnitude less. Thus data presented by Thompson & Scott indicate an expected lifespan for the nuclear-polyhedrosis virus of *O. pseudotsugata* of approximately 2–3 months in the duff layer of soil of forests in the Cascade Mountains of North America (table 2 of Thompson & Scott 1979), which is to be compared with the estimated maximum lifespan of 11 years.

In contrast to these comparatively long-lived infective agents of the insect baculoviruses and microsporidians, many pathogens produce stages that are very short-lived in the external habitat. For example, a non-inclusion virus of the European red mite, *Panonychus ulmi*, is very unstable outside the host, and only remains infective for about one week (Putman 1970). Similarly, as seen in table 4, some bacterial and fungal spores have short lifespans (Wilson 1971, 1972; Ignoffo *et al.* 1976).

(*b*) *Rate of production of infective stages*

We define λ to be the rate at which an infected host produces infective stages of the parasite. For many invertebrate species, an infected host does not release infective stages at a steady rate, but rather releases Λ infective particles into the environment when it dies; this is essentially equivalent to a host producing infective stages at a steady rate

$$\lambda = \Lambda(\alpha + b + \gamma) \tag{58}$$

throughout the expected lifespan, $1/(\alpha + b + \gamma)$, of the infection. In the absence of recoveries, $\gamma = 0$ (which commonly is the case for microparasitic infections of invertebrates), this equivalence formula reduces to $\lambda = \Lambda(\alpha + b)$. The production rate, λ, varies widely among species of pathogens. As documented in table 5, the nuclear-polyhedrosis viruses and microsporidians of insects

often produce very large numbers of infective agents per infected host. The millions of baculovirus inclusion bodies (polyhedra or granules) liberated on the death of an infected host may each contain many virus particles (as for nuclear-polyhedrosis viruses) or a single particle (as for granulosis viruses) (Tinsley 1979). The polyhedra of the nuclear-polyhedrosis viruses thus constitute 'packets' of infective particles. The inclusion body, or polyhedron, serves to preserve the infectivity of the virus in the external habitat.

(*c*) *Natural history of transmission*

The free-living infective stages of microparasites employ a variety of pathways for their passage from one host to the next. The infective stages may exit from an infected host through the digestive tract (in the faeces), or through respiratory pores or reproductive canals (particularly important in transovum transmission). Pathogens that live in the body cavity of the host often rely on the death and dissolution of the host for the release of the infective stages (cf. equation (58)). Consumption of dead infected hosts by individuals of the same species obviously constitutes an effective method of transmission.

The consumption of infected hosts by scavengers and predators, particularly birds and small mammals, can lead to dissemination of the infective stages over a wide area, provided they are able to survive passage through the dispersing animal's gut (as the infective stages of many virus and protozoan infections of invertebrates can) (Tinsley 1979; Stairs 1972). For example, the spores of the protozoans *Nosema polyvora* and *Pleistophora schubergi* pass through the alimentary tracts of birds, *Parus major*, with little or no loss in virulence (Gunther 1959; Tanada 1964). Entwistle *et al.* (1977) suggest that birds play a major role in the dispersal, and hence persistence, of an endemic nuclear-polyhedrosis virus of the sawfly, *Gilpinia hercyniae*, in forest ecosystems.

(*d*) *Host–parasite dynamics with free-living infective stages*

We define the population of free-living infective stages of the parasite to be $W(t)$ at time t. The rate at which uninfected hosts acquire infection is assumed to be proportional to the number of susceptible hosts, X, and to the number of infective stages, W; that is, the transmission term βXY of the basic model A is replaced by a term νWX, where ν is a proportionality constant measuring the transmission efficiency (νW has the dimension 1/time). Thus equations (12)–(14) are replaced by

$$\mathrm{d}X/\mathrm{d}t = a(X+Y) - bX - \nu WX + \gamma Y, \tag{59}$$

$$\mathrm{d}Y/\mathrm{d}t = \nu WX - (\alpha + b + \gamma)\, Y, \tag{60}$$

$$\mathrm{d}H/\mathrm{d}t = rH - \alpha Y. \tag{61}$$

As before, we may use any two of these three equations, along with the identity $H = X + Y$. It remains to close the system of equations with a description of the dynamical behaviour of W. By our earlier definition, infective stages are produced from infected hosts at a net rate λY. Free-living infective stages are lost upon being picked up by hosts (either uninfected or infected) at a net rate νWH, or by mortality at an individual rate μ (corresponding to a net death rate μW). Thus the rate of change of the population of free-living infective stages is

$$\mathrm{d}W/\mathrm{d}t = \lambda Y - (\mu + \nu H)\, W. \tag{62}$$

This set of equations constitutes model G, which is depicted schematically in figure 15. The dynamics of this system is analysed in appendix F.

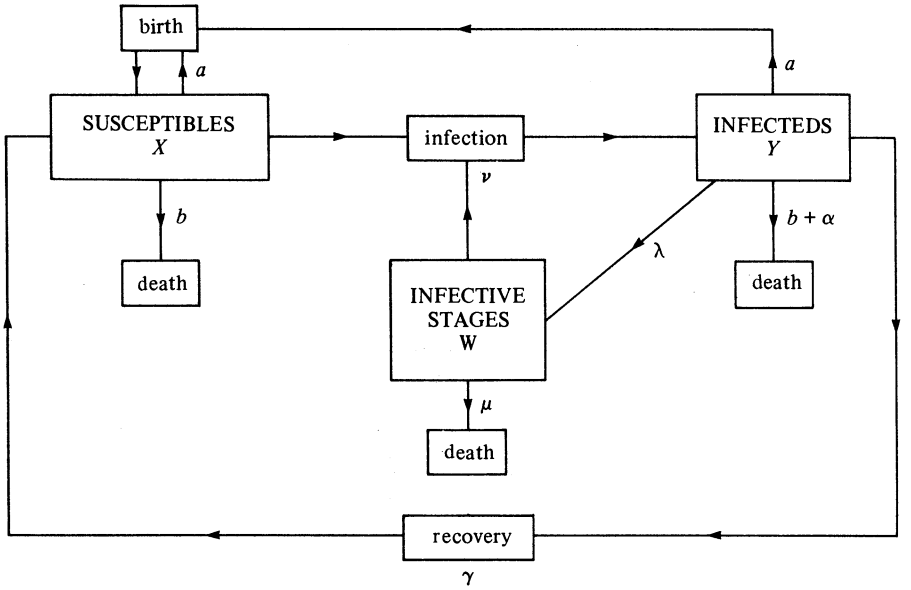

FIGURE 15. Schematic representation of the assumptions embodied in model G, where we take explicit account of the free-living infective stages of the parasite. The model is defined more explicitly by equations (59)–(62), and the various rate parameters are as listed in appendix A.

As usual, we begin by looking at the basic reproductive rate, R, of the parasite, which here is

$$R = \frac{\lambda}{(\alpha+b+\gamma)}\left(\frac{\nu H}{\mu+\nu H}\right). \tag{63}$$

This relation may be understood in biological terms: throughout the lifespan of an infected host individual (of average duration $1/(\alpha+b+\gamma)$), infective stages are produced at a rate λ; of these infective stages, a fraction $\nu H/(\mu+\nu H)$ are successfully transmitted to new hosts (νH), as opposed to dying (μ). It is immediately apparent that R is always less than unity, and the infection cannot persist, unless

$$\lambda > (\alpha+b+\gamma). \tag{64}$$

Equivalently, for the special case when a total of Λ infective stages are released on the death of the host, equation (58) can be used to rewrite equation (64) as

$$\Lambda > 1. \tag{65}$$

The general equation (64) or the special equation (65) is simply the trivial requirement that each infected host must produce, on average, more than one infective stage. Once equation (64) is satisfied, the threshold host density, H_T, follows by putting $R = 1$ in equation (63), to give

$$H_T = \left(\frac{\mu}{\nu\lambda}\right)\frac{\alpha+b+\gamma}{[1-(\alpha+b+\gamma)/\lambda]}. \tag{66}$$

A glance at tables 1 and 5 suggests that λ is essentially always vastly greater than $\alpha+b+\gamma$ for microparasitic infections of invertebrates; indeed, for the cases in table 5, $\Lambda = \lambda/(\alpha+b+\gamma)$ is typically of the order of 10^6 or more. Thus, not only are the inequalities of equations (64) and (65) strongly fulfilled, but the term in square brackets in the denominator in equation (66) is, to an excellent approximation, unity. We shall henceforward simplify the presentation

by neglecting all terms of order $(\alpha+b+\gamma)/\lambda$, relative to unity; the exact results are given in appendix F. Furthermore, we may then define

$$\beta = \nu\lambda/\mu, \tag{67}$$

and express the threshold host density as

$$H_T = (\alpha+b+\gamma)/\beta. \tag{68}$$

This is identical with equation (10), except that the transmission parameter β is expressed in more basic terms, involving the rates of production (λ), death (μ) and transmission (ν) of free-living infective stages. Note that large λ and small μ (long-lived infective stages) help to keep the threshold host density relatively low.

As in model A, the host population will grow exponentially (at the rate r) until it exceeds H_T, whereupon the disease is able to persist and will regulate the population provided that

$$\lambda > \alpha(\alpha+b+\gamma)/(\alpha-r) > 0. \tag{69}$$

If

$$\alpha > r, \tag{70}$$

equation (68) is essentially always fulfilled (unless α is improbably close to r) for the effectively infinite values of λ characteristic of microparasitic infections of invertebrates.

Unlike models A–F, the regulated state is not necessarily a stable equilibrium host population. Two cases can be distinguished.

(i) There is a stable equilibrium if

$$(\mu+\alpha D-r)(D-1)-(\alpha+b+\gamma) > 0, \tag{71}$$

where D is defined for notational convenience as $D = (\alpha+b+\gamma)/(\alpha-r)$. This stable equilibrium value H^* of the host population is

$$H^* = \left(\frac{\alpha}{\alpha-r}\right)H_T, \tag{72}$$

with H_T defined by equation (68). The equilibrium prevalence is $y^* = r/\alpha$. Thus the simple expressions derived for model A, equations (17) and (18), remain true in this circumstance. Equations (71) and (72) are for λ effectively infinite, in the sense discussed above; exact results are given in appendix F.

In short, a stable equilibrium is achieved if: the parasite pathogenicity, α, exceeds the host population's intrinsic growth rate, r (equation (69)); the rate of production of infective stages, λ, is large (equation (69)); and equation (71) is satisfied. The last condition is not particularly transparent, but equation (71) tends to be fulfilled provided that the infective stage is short-lived (μ large), and the pathogenicity, α, is not too much larger than r. These points are summarized in figure 16, which shows the way in which dynamical behaviour depends on α and λ, for various values of μ and r (and $\gamma = 0$).

(ii) Conversely, if equation (68) is satisfied, but equation (71) is not, the regulated state is a stable limit cycle. The variables $X(t)$, $Y(t)$, $H(t)$ and $W(t)$ oscillate in stable cycles, the period, amplitude and overall shape of which are uniquely determined by the parameters of the model. Equation (71) suggests that this circumstance is likely to arise when the infective stage is long-lived (μ small) and the pathogenicity very high (α large).

The important conclusion is that highly pathogenic microparasites producing very large numbers of long-lived infective stages are likely to lead to non-seasonal cyclic changes in the abundance of their invertebrate

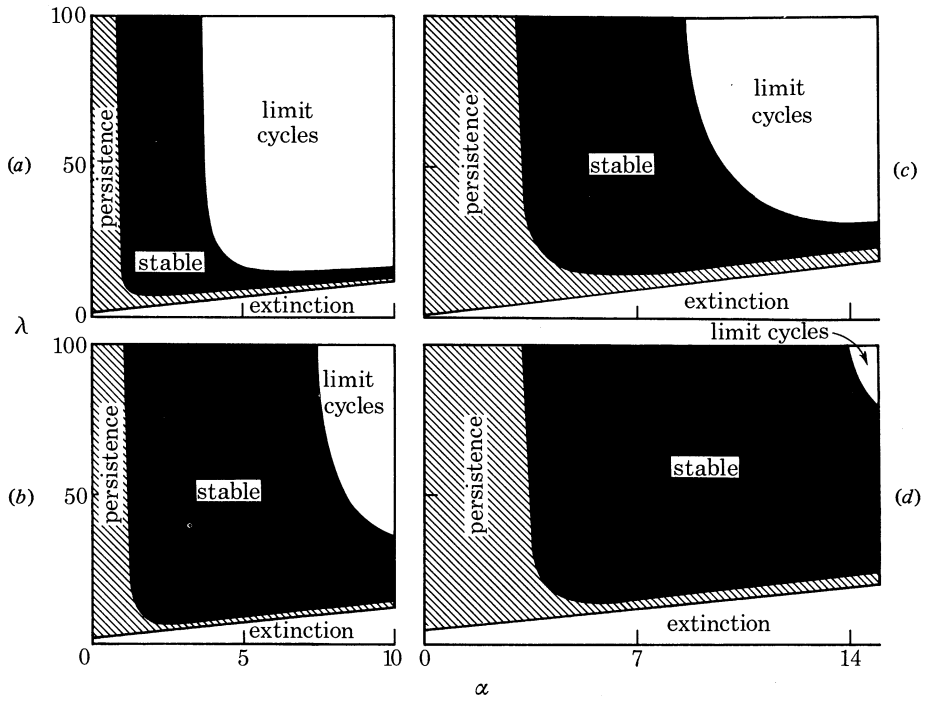

FIGURE 16. The four regimes of dynamical behaviour that can be exhibited by model G are here illustrated in the λ–α parameter space (λ is the rate of production of infective stages per infected host, and α measures the parasite pathogenicity). As discussed in the text, these four régimes are: the pathogen regulates its host population to a stable equilibrium value ('stable'); the pathogen regulates its host population to stable cyclic oscillations ('limit cycles'); the pathogen fails to regulate the host population, but persists within it as it undergoes exponential growth ('persistence'); and the pathogen cannot maintain itself within the unregulated host population ('extinction'). In (*a*) the other parameter values are $r = 1.0$ week^{-1}, $b = 1.0$ week^{-1}, $\gamma = 0$ week^{-1} and $\mu = 0.02$ week^{-1}; the infective stages are long-lived. Figure (*b*) has the same parameter values, except that the infective stages are short-lived, with now $\mu = 14$; (*c*) and (*d*) are similar to (*a*) and (*b*), respectively, except that here $r = 3.0$ week^{-1}; this faster growth rate for the host population diminishes the domain of λ–α parameter space where limit cycles arise.

hosts and in the prevalence of infection. In the next section this conclusion is tested against available data.

(iii) If equation (69) is violated (which, for large λ, essentially means $r > \alpha$), the parasite is unable to regulate its host population, which grows exponentially. The parasite will, however, persist within the host population, asymptotically slowing its rate of exponential growth, provided that

$$\lambda - (\alpha + b + \gamma) > r. \tag{73}$$

Equation (73) (which is necessarily a weaker condition than equation (69)) differs from the simpler equation (64) because the asymptotic rate of reproduction of the parasite must not merely be positive but must exceed that of its host if the parasite is to persist within the exponentially expanding host population. The condition equation (73) is, interestingly, identical with that derived from models describing the dynamics of metazoan macroparasites with direct life cycles, such as many nematodes, tapeworms and flukes (Anderson & May 1978; May & Anderson 1978). Such inequalities accord with the observed fact that most microparasites and macroparasites possess reproductive potentials much greater than those of their hosts.

(iv) Finally, if equation (73) is not satisfied the parasite cannot persist and the host population grows exponentially at the disease-free rate r.

These four basic regimes of dynamical behaviour depend on the interplay among the ecological and epidemiological parameters α, r, μ, λ, b and γ. Figure 16 illustrates some of these relations, as do figures 18 and 19.

12. Insect population cycles induced by microparasitic infections

Many viral and microsporidian pathogens of insects possess the characteristics of high pathogenicity and very high rates of production of long-lived infective stages required to generate the stable cycles discussed above.

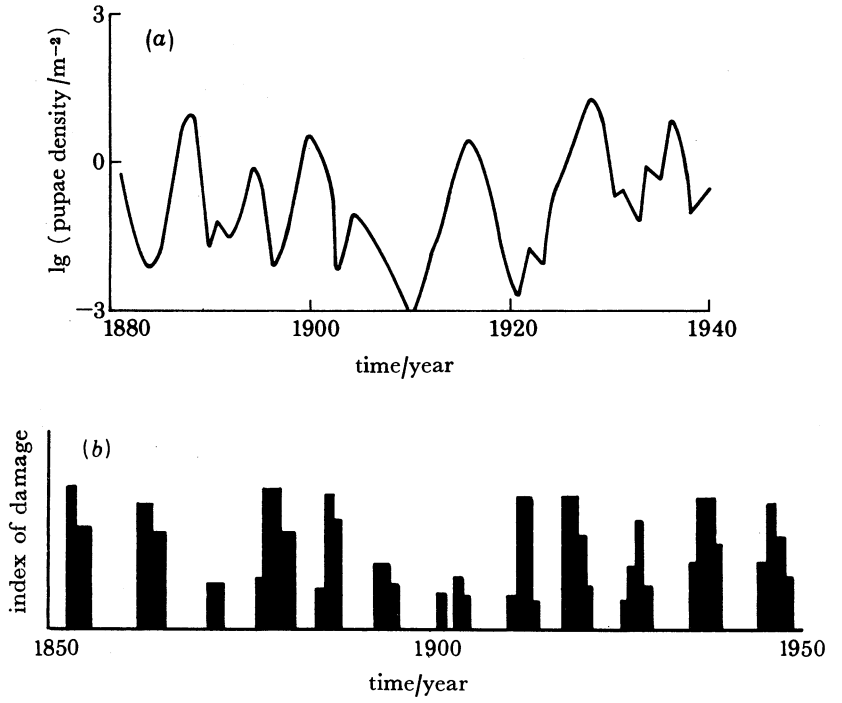

Figure 17. Two examples of long-term periodic fluctuations of insect abundance: (*a*) population fluctuations of the pine looper moth, *Bupalis piniarus*, in Germany (data from Varley (1949)); (*b*) population fluctuations of the larch budmoth, *Zeiraphera diniana*, in the European Alps, as reflected in an index of the intensity of damage to the larch stands (data from Baltensweiler (1964)).

Cyclic changes in the abundance of insect species have been reported in many temperate forest regions in Europe and North America (Varley *et al.* 1973). Many of these forest insect species are pests, causing economic damage to the standing crop of trees in years of high pest abundance. Such insects have therefore been much studied by forest entomologists, and the long-term records of population data are among the best that exist for any animal populations. The striking feature of many of these studies is the regularity of cyclic changes in insect abundance; some species have predictable population peaks every eight to ten years, while others appear to cycle on shorter or longer time scales (Klomp 1966; Baltensweiler 1964; Auer 1968; Baltensweiler *et al.* 1977; Schwerdtfeger 1941; Varley 1949). Two examples of long-term cycles are displayed in figure 17 (with the data extending in one over 60 years, and in the other over 100 years); see also figure 21.

There has been much unresolved speculation about the mechanisms responsible for these regular peaks in insect abundance (Varley *et al.* 1973). Some authors have suggested that the cycles are driven by specific parasitoids, or by time lags in the action of density-dependent effects (such as regeneration of food resources, or the abundance of predators) (Klomp 1966; Varley 1949; Ludwig *et al.* 1978; May 1974). Others have noted that epidemic outbreaks of viral or protozoan parasites are often observed in years of peak pest abundance (see table 6), and have argued that such pathogens are important in causing dramatic reductions in host abundance (Bird & Whalen 1954; Prebble & Graham 1945; Morris 1963; Thompson & Scott 1979; Entwistle *et al.* 1977; Stairs 1972; Lloyd & Dybas 1966*a*, *b*).

TABLE 6. EPIDEMICS OF VIRUS DISEASE IN HIGH DENSITY POPULATION OF ARTHROPODS

host	locality	type of virus	percentage infection	reference
Panonychus citri (citrus red mite)	California	non-occluded	25	Shaw & Beavers (1970)
Chironomus plamous (midge)	Wisconsin	iridescent	40	Stolz *et al.* (1968)
Chironomous luridus (midge)	Germany	insect-pox	20–40	Huger *et al.* (1970)
Trichoplusia ni (cabbage looper)	California	cytoplasmic polyhedrosis	40	Bailey (1973)
Malacosoma alpicda (tent caterpillar)	Switzerland	granulosis	53	Benz (1962)
Orgyia pseudotsugata (tussock moth)	Oregon	nuclear-polyhedrosis	50	Thompson & Scott (1979)
Porthetria dispar (gypsy moth)	Connecticut	nuclear-polyhedrosis	80	Doane (1969)
Neodiprion sertifer (sawfly)	Canada	nuclear-polyhedrosis	20–90	Bird (1961)
Diprion hercyniae (sawfly)	Canada	nuclear-polyhedrosis	60	Bird & Elgee (1957)
Wiseana spp. (Lepidoptera)	New Zealand	nuclear-polyhedrosis	80	Crawford & Kalmakoff (1977)
Orcyctes rhinoceros (rhinoceros beetle)	western Samoa	baculovirus	60	Zelazny (1977)

Table 7 summarizes the average periods of cycles in the abundance of particular species of forest insect pests. The table also lists some pathogens that have been observed in these insect populations. We now argue, in detail, that these cycles (with periods of the observed magnitudes) are produced by host–parasite interactions of the kind studied in model G. To this end, we give a seriatim discussion of the four main parameters, μ, α, λ and r.

First, the longevity, $1/\mu$, of viral infective stages, be they granules, particles or polyhedra, depends on the physical environment. Jaques (1977) and others found that sunlight, most probably the ultraviolet part of the spectrum, is the most important factor determining loss of infectivity of insect baculoviruses in the field; baculoviruses, of either the nuclear-polyhedrosis or granulosis type, are principally parasites of lepidoptera, hymenoptera and diptera (Tinsley 1979). Temperature and humidity also are important, with baculoviruses retaining their infectivity at moderate to low temperatures, provided that the humidity is not too low (Tinsley 1979; Jaques 1975). Thus the soil environment of temperate forest habitats appears ideally suited for the long-term survival of viral infective agents. As indicated in table 4, maximum lifespans of nuclear-polyhedrosis viral particles range up to 11 years in natural

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forest environments (Thompson & Scott 1979; Thomas *et al.* 1972), although, as discussed in § 11, expected lifespans are usually much shorter.

Secondly, as shown in table 1 (and discussed by Stairs 1972; Tinsley 1979; Thompson & Scott 1979; Morris 1963), baculoviruses and microsporidians of insects are in general extremely pathogenic, and cause very high rates of host mortality (α large). Infected insects have little or no chance of recovery from infection ($\gamma = 0$). Moreover, as can be seen from table 2, many of these pathogens also reduce the fecundity of infected hosts, which further increases their effective pathogenicity in the manner discussed in § 6 (model B).

TABLE 7. CYCLIC VARIATIONS IN THE ABUNDANCE OF FOREST INSECT SPECIES

host insect species	locality	period of cycles in population abundance / year	pathogen	reference
Orgyia pseudotsugata (Douglas-fir tossuck moth)	North America	7–10	nuclear-polyhedrosis virus	Thompson & Scott (1979)
Acleris variana (black-headed budworm)	eastern Canada	10–15	nuclear-polyhedrosis virus	Prebble & Graham (1945); Miller (1966)
Bupalus piniarius (pine looper)	Europe	5–8	nuclear-polyhedrosis virus	Klomp (1966)
Zeiraphera diniana (larch budmoth)	Europe	9–10	granulosis virus	Auer (1968); Baltensweiler (1964)
Diprion hercyniae (spruce sawfly)	North America	8	nuclear-polyhedrosis virus	Bird & Elgee (1957)
Malacosoma disstria (tent caterpillar)	North America	8–12	nuclear-polyhedrosis virus; microsporidian protozoan	Hodson (1941); Thomson (1960)

TABLE 8. ESTIMATES OF THE NATURAL INTRINSIC GROWTH RATE, r, OF SOME FOREST INSECT SPECIES

host	approximate r (per capita) / year^{-1}	reference
Bupalus piniarius (pine looper)	0.8–1.2	Klomp (1966)
Zeiraphera diniana (larch budmoth)	1.0	Auer (1968)
Acleris variana (black-headed budworm)	1.5	Morris (1959)
Zeiraphera griseana (grey larch budmoth)	1.0–1.2	Baltensweiler (1964)
Choristoneura fumiferana (spruce budworm)	1.0	Morris (1963)
Diprion hercyniae (spruce sawfly)	1.0	Bird & Elgee (1957)

Thirdly, we see from table 5 that the number, A, of infective stages released into the environment upon the death of an infected host is typically many millions for both microsporidians and viruses. As discussed in § 11, this means that the effective rate of production, λ, of infective stages is not merely very large in relation to other rate parameters, but is for many purposes effectively infinite.

Fourthly, many of the forest insect pests under consideration are univoltine (one generation per year), and characteristically exhibit relatively low rates of annual population growth. The rough estimates summarized in table 8 suggest the intrinsic growth rate, r, of individuals of such insect species is typically around unity (in units of reciprocal years (year^{-1})).

In short, for many insect–host pathogen associations in temperate forests we have the con-

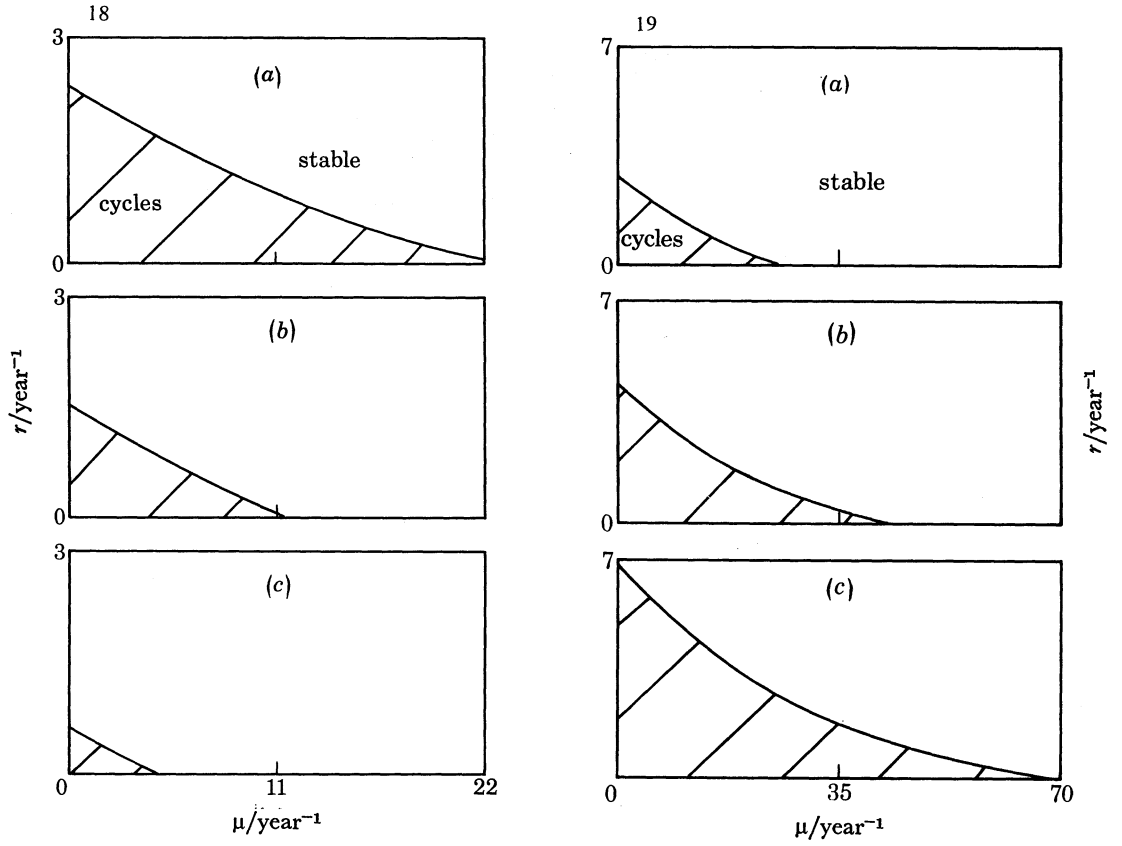

FIGURE 18. For model G, these figures illustrate the domain of r–μ parameter space in which the parasite regulates its host population at a stable equilibrium value (unhatched region), and in which host and parasite populations exhibit stable limit cycle behaviour (hatched region); r is the intrinsic growth rate of the host and μ the death rate of infective stages ($1/\mu$ is thus the expected lifespan of infective stages). The other relevant rate parameters have the values $b = 3.3$ year^{-1}, $\gamma = 0$ year^{-1}, $\lambda = 10^6$ year^{-1} (which is to say Λ is effectively infinite). The parasite pathogenicity α takes the values: (*a*) 9.0 year^{-1}; (*b*) 7.0 year^{-1}; and (*c*) 5.0 year^{-1}. A discussed in the text, cycles are more likely to arise for relatively small values of r and μ, and relatively large values of α.

FIGURE 19. These figures are similar to those in figure 18, except that now we have extended model G to include the effects of parasite-induced decrease in the reproductive rate of infected hosts. Thus we show the domain of r–μ parameter space corresponding to a stable equilibrium and to stable limit cycles for a host–parasite system described by a combination of models G and B (the boundary between the two domains in figure (19) is now given by equation (34) of appendix F; the boundary in figure (18) is given by the simpler equation (71) of § 11). In (*a*) the reproductive rate of infected hosts is depressed by 10% relative to that of uninfected ones, $f = 0.1$; in (*b*) $f = 0.3$; and in (*c*) $f = 0.5$. The other relevant rate parameters have the values: $\alpha = 9.0$ year^{-1}; $b = 3.3$ year^{-1}; $\gamma = 0$ year^{-1}; $\lambda = 10^6$ year^{-1}. As discussed in the text, figure (19) shows that depression of the birth rate of infected hosts (increasing f) makes stable limit cycle behaviour more likely.

catenation of large α, small μ, relatively small r and very large λ (tables 1, 4, 8, 5) that produces stable limit cycles. This point is illustrated explicitly in figure 18, which shows the domain of cyclic (rather than stable equilibrium) behaviour as a function of μ and r, for various α, with all parameters having values in the range appropriate to forest insects. Figure 19 shows that the domain of stable cyclic behaviour is further enlarged if the pathogen also reduces the fecundity of infected hosts (the fusion of models B and G underlying figure 19 is outlined in appendix F). The period of the stable limit cycles (obtained by numerical studies of the

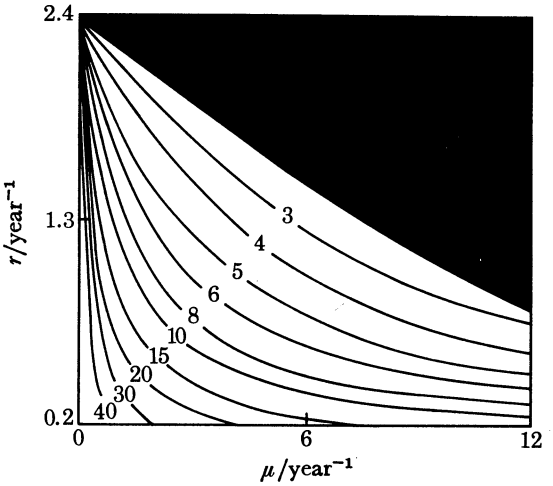

FIGURE 20. As discussed more fully in the text, this figure shows the period of the cycles in host abundance and prevalence of infection, as a function of the parameters r and μ of model G; the contour lines are for particular periods (labelled according to the period, in years) from 3 to 40 years. In the shaded region, there is a stable equilibrium point. Rate parameters: $\alpha = 9.0$ year^{-1}; $b = 3.3$ year^{-1}; $\gamma = 0$ year^{-1}; $\lambda = 10^6$ year^{-1} (i.e. Λ is effectively infinite).

nonlinear set of differential equations) is shown as a function of μ and r, for a typical value of α, in figure 20.

We now analyse one example, namely the European larch budmoth, in detail. First, however, we bring the equations (59)–(62) into dimensionless form, to make quite clear which parameters are, and which are not, involved in fitting our theory to the data. As has been discussed above, in connection with table 5, and is justified more formally in appendix F, we take the rate of production of infective stages λ to be much larger than other rate processes ($\Lambda \gg 1$ in equations (58), (64), (65)). We then define the dimensionless variables $X' = X/H_T$, $Y' = Y/H_T$, $H' = H/H_T$ and $W' = \mu W/\lambda H_T$, with H_T itself defined by equations (67) and (68) as $H_T = \mu(\alpha+b+\gamma)/\lambda\nu$. The closed system of equations (60)–(62) then becomes

$$dY'/dt = (\alpha+b+\gamma)[W'(H'-Y')-Y'], \tag{74}$$

$$dH'/dt = rH'-\alpha Y', \tag{75}$$

$$dW'/dt = \mu(Y'-W'). \tag{76}$$

It is now clear that the dynamical behaviour of these equations (the shape and period of the cycles) depends only on the rate parameters μ, α, r and the combination $\alpha+b+\gamma$. The prevalence ($y = Y/H$), being intrinsically dimensionless, also depends only on these parameters. On the other hand, the absolute scale of the population variables X, Y and H involves H_T, and thence the additional parameter combination $\nu\lambda$. The parameters μ, α, r, b and γ are more or less determinable, but estimation of λ (beyond the fact it is very large) is difficult, and of ν hopeless.

Figure 21 shows data for the abundance of the larch budmoth, *Zeiraphera diniana*, in the European Alps, and for the prevalence of infection with a granulosis virus, over an interval of 20 years (Auer 1968).

Figure 22 shows the same quantities, namely budmoth abundance and prevalence of infection, as calculated from equations (74)–(76) with values of the parameters α, μ, r, b and γ

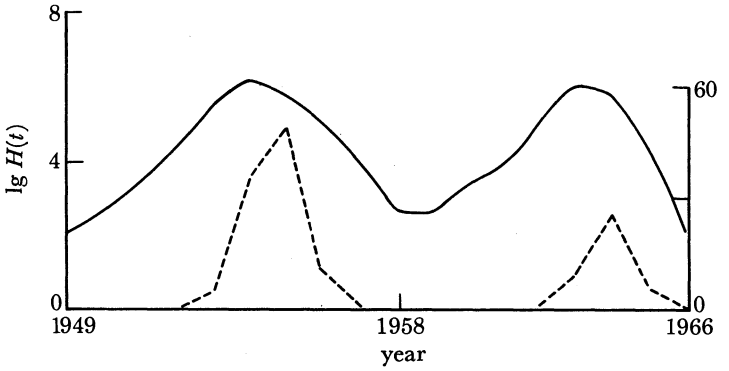

FIGURE 21. The solid line shows observed changes in the abundance of the larch budmoth, *Zeiraphera diniana*, in the European Alps, and the dashed line shows the percentage prevalence of infection with a granulosis virus in this population (data from Auer (1968)). This figure and figure 17*b* show that cycles in the abundance of this forest insect occur approximately every 9–10 years.

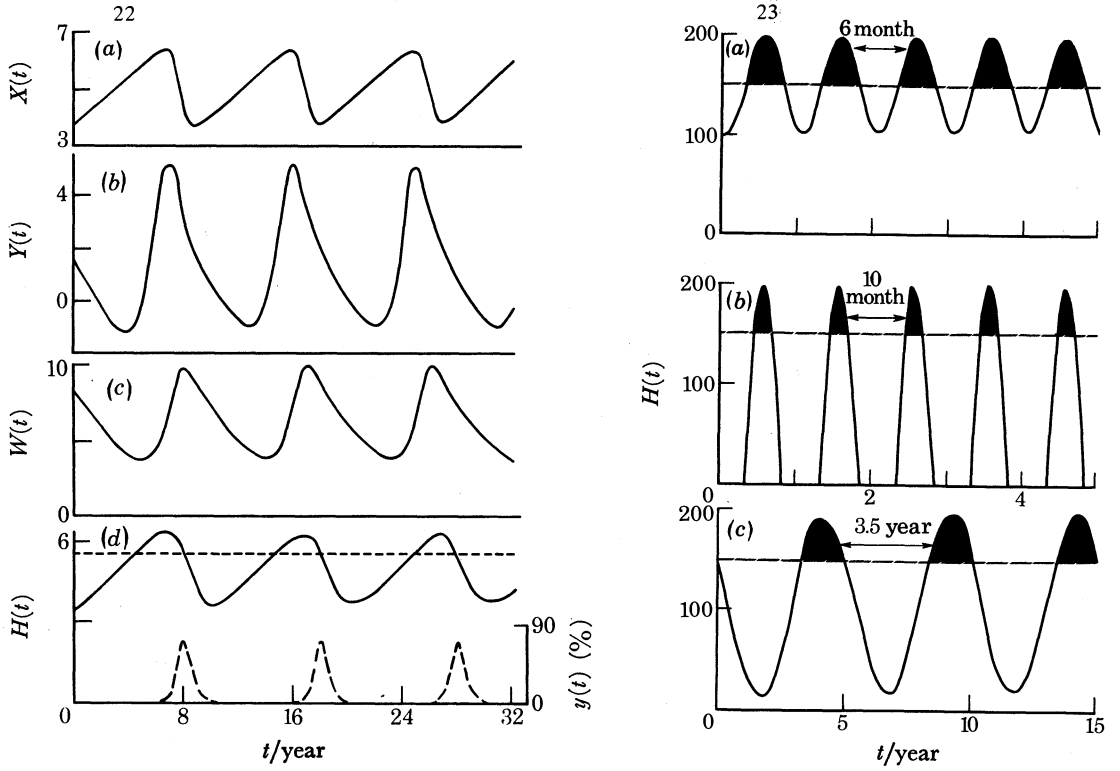

FIGURE 22. This figure depicts numerical solution of model G, equations (59)–(62), using parameter values that crudely approximate those for the larch budmoth–granulosis virus interaction illustrated in figure 21. We specifically choose $r \approx 1.0$ year^{-1}, $b = 3.3$ year^{-1}, $\gamma = 0$ year^{-1}, $\mu \approx 3.0$ year^{-1}, $\alpha \approx 14$ year^{-1}, $\lambda = 10^6$ year^{-1} (Λ effectively infinite); the transmission efficiency ν is arbitrarily set at 10^{-10} year^{-1}. These choices, and the associated references, are discussed in the text. (*a*)–(*c*) The change in abundance of susceptible hosts, $X(t)$, infected hosts, $Y(t)$, and free-living infective stages of the parasite, $W(t)$, respectively (all plotted as logarithms to base 10), as functions of time t (in years). Figure (*d*) likewise shows total host abundance, $H(t)$ (plotted logarithmically) as a function of time; the dashed horizontal line indicates the threshold host density for maintenance of the parasite within the host population. The prevalence of infection, $y(t)$ (as a percentage), within the host population is also shown by dashed lines in (*d*).

FIGURE 23. Idealized representation of the relation between the threshold host density, H_T, for maintenance of the parasite (the dashed horizontal lines) and cyclic changes in host abundance, $H(t)$ (the vertical axis) As discussed in the text, (*a*) illustrates the kind of pattern likely to arise from seasonal effects (with host abundance below the threshold level for around 6 months or so); (*b*) illustrates the situation likely to arise when there are discrete non-overlapping generations of the host population (with host abundance typically below threshold for the greater part of one year); and (*c*) illustrates the kind of pattern generated by long-term limit cycles in host abundance (where host abundance can be below threshold levels for several years.)

estimated *independently* of the population data of figure 21. Specifically, we use information given by Baltensweiler *et al.* (1977), Baltensweiler (1964), Benz (1962) and Auer (1968) (see tables 1, 4, 8) to assign the values: $r \approx 1$ year^{-1} (a necessarily rough estimate); $b \approx 3.3$ year^{-1} ($1/b$ *ca.* $3\frac{1}{2}$ months); $\gamma = 0$ year^{-1} (negligible recovery); $\alpha \approx 14$ year^{-1}; $\mu \approx 3.0$ year^{-1} ($1/\mu$ around 3–4 months). The cycles in abundance of susceptibles X, infecteds Y, and infective stages of the parasite W are also shown in figure 22. The host population H is plotted on a logarithmic scale; thus the undetermined scaling parameter H_T only enters in setting the absolute level of H, and does not enter into the logarithmically plotted amplitude (we set the absolute scale by putting $\lambda = 10^6$ and arbitrarily choosing $\nu = 10^{-10}$).

The agreement between the data in figure 21 and theoretical results in figure 22, with respect to the period and to the shape and magnitude of the oscillations in budmoth population and prevalence of virus infection, is encouraging. It is to be emphasized that no adjustable parameters are involved in this fit.

Unfortunately, we know of no other examples where all the important parameters in our host–parasite model can be estimated independently of the data on the population cycle itself. Figure 24*a* gives data for the spruce sawfly, *Diprion hercyniae*, and a virus disease that has been argued to be regulating it (Bird & Elgee 1957; Southwood 1977); we can fit this data with our model, but not with parameters estimated independently from the data being fitted.

Several general features of population cycles driven by host–parasite associations are illustrated by figure 22. First, the peak in prevalence of the infection within the host population occurs shortly after the peak in host abundance. Secondly, the host population falls below the threshold value, H_T, during part of the cycle; the infection survives primarily by virtue of its relatively long-lived transmission stages. Thirdly, when H is below H_T the prevalence declines effectively to zero, so that the disease seems to have disappeared from its host population; *it is a mistake to think that disappearance of the disease, or epidemic reappearance, is inconsistent with the pathogen driving the host population cycle*. Fourthly, the cyclic patterns of host abundance (figure 22*d*) tend to be characterized by a slow rise and a rapid fall, whereas the cycles in the number of infected hosts (figure 22*b*) and in the population of free-living infective stages (figure 22*c*) tend to have a quick rise and a slow decline.

If the fluctuations in host and parasite abundance are very severe, stochastic effects can produce extinction of the host of or the pathogen, complicating our story. This is especially liable to happen when r is relatively small, so that the system cycles with a long period and the density of free-living infective stages can fall to low levels.

In general, figures 18–20 show that interactions between invertebrate hosts and their microparasites are likely to result in population cycles. Such cycles may have periods shorter than one year if the host annually produces many generations (relatively large r), or the periods may be several decades if r and μ are both small; typical vital rates, however, suggest periods in the range 5–12 years. Of course, there are other factors, some of which are discussed in the next section, that can lead to cycles in host–parasite associations. We suggest, however, that the simple mechanism discussed above is sufficient to account at least for most long-term population cycles in forest insects.

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13. Persistence of microparasites in fluctuating host populations

Cyclic or erratic variation in the abundance of invertebrate hosts can arise in many ways. Seasonal changes in temperature, humidity or other environmental factors may cause changes in birth rates, natural death rates, or in the transmission efficiency of a pathogen that influences host dynamics. The life cycle of many arthropods and other invertebrates consists of discrete, non-overlapping generations, and larval or adult individuals may only appear during a brief part of the year (the adults of cicadas with periods 13 and 17 years are in extreme case; Lloyd & Dybas 1966*a*, *b*). Alternatively, limit cycles with long or short periods may be produced by the interaction between the host population and a microparasite (as discussed above), a predator (May 1972, 1974, 1977; Hassell 1976, 1979; Gilpin 1975), food supplies (Caughley 1976), or, more generally, by time lags in regulatory mechanisms (May 1974).

Regardless of the mechanism, such changes in host abundance can create problems for the pathogen if the host density H fluctuates below the threshold level H_T necessary for pathogen persistence. As inverbetrate populations are characterized by a level of fluctuation higher than that for vertebrates (Southwood 1976), these problems are more acute for directly transmitted parasites with invertebrate hosts. Figure 23 gives an idealized representation of the threshold density, H_T, in host populations undergoing cyclic variations caused by: (*a*) seasonal effects; (*b*) discrete, non-overlapping generations; and (*c*) limit cycles with long periods.

Microparasites of invertebrates appear to have evolved three basic mechanisms to cope with the problems created by wide fluctuations in host abundance. Each of these mechanisms – vertical transmission, occult infection, free-living infective stages – is now discussed in the light of the analysis in §§ 4–12.

(*a*) *Vertical transmission*

As was seen in model C, vertical transmission lowers the threshold host density H_T (equation (31)), facilitating the persistence of pathogens in relatively low density host populations. Both transovarial and transovum forms of vertical transmission are widely observed among the pathogens of invertebrate species, and they undoubtedly help pathogens to persist with host populations that undergo large seasonal fluctuations.

In particular, such transmission mechanisms help to prevent the extinction of the parasite in host populations with discrete, non-overlapping generations, where replication and population growth of the pathogen within an individual host is restricted to specific developmental stages in the host's life cycle (e.g. specific instars or solely the adult stage) (see Tanada 1964; Stairs 1972; Smith 1976; Tinsley 1979). For instance, Clark (1956) suggests that transovarial transmission is important in enabling virus infections of tent caterpillars, *Malacosoma fragile*, to persist through the period of 9–10 months during which no susceptible stages of the insect are present.

(*b*) *Occult or non-apparent infections*

The occurrence of occult, latent or non-apparent infection within invertebrate populations has been much discussed in recent years. The phenomenon has been variously defined in different areas of invertebrate pathology; one of the simplest definitions is by Tanada (1964), who argues that the presence of an occult infection can only be unequivocally demonstrated by placing apparently healthy hosts under stress, and observing the increase in mortality or decrease in reproduction resulting from the activation of a previously quiescient microparasitic infection. Such occult or non-apparent infections are common among insect–virus associations.

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For example, latent infection with cytoplasmic polyhedrosis viruses occur in bees, *Bombus mori*, in tent caterpillars, *Malacosoma* sp., in winter moths, *Operophtera brumata*, in pine loopers, *Bupalis piniarius*, and in the small white butterfly, *Pieris rapae* (Smith 1976). Non-inclusion baculoviruses, such as those causing acute or chronic bee paralysis, occur commonly in apparently healthy honey bees and bumble bees (Bailey *et al.* 1963, 1964).

These occult viral infections of insects can be activated by various stresses, including those associated with physical conditions (such as temperature or humidity), food quantity and quality, and infection of the host by more than one species of parasite or superinfection by one species of parasite (Bergold 1958; Steinhaus 1958; Grace 1962; Tanada 1964; Smith 1976). The underlying mechanisms are, in general, poorly understood. Viral replication within poikilothermic hosts is undoubtedly affected by temperature and by the associated metabolic rate of the host; the population growth of microparasites within the host body will be markedly slower during periods of aestivation or hibernation. Genetically determined variability in the susceptibility of hosts to infection may also be important, with some host individuals entirely overcoming the invading pathogens, while others constrain population growth to such a low level that the infection seems to be latent or non-apparent.

In terms of the overall population biology of the host–parasite association, quiescent or occult infections that induce no change in the mortality or reproduction of infected hosts may enable the parasite to persist during periods of low host density. In particular, in seasonal environments, low temperatures during the winter months may inhibit viral replication within a host, thus enabling the overwintering host and the pathogen to survive. In general, however, little is yet understood about the population consequences of occult or non-apparent infections.

(*c*) *Long-lived infective stages: general*

Free-living infective stages, with long lifespans, clearly represent a third method whereby a pathogen can remain endemic in a host population that fluctuates widely on either a seasonal or a longer-term basis. In particular, as we saw in model G (equation (66) or (67)), the threshold host density for maintenance of the infection decreases as the average lifespan of infective stages lengthens (i.e. as μ decreases). More generally, the free-living infective stages enable the parasite population to persist *outside* the host during the troughs, when host abundance is below the threshold density required for maintenance of the infection *within* the host population; the overall parasite population is restocked during the episodes of host abundance.

This strategy for parasite survival in a widely fluctuating host population requires that the maximum lifespan of the free-living infective stages exceed the maximum length of the intervals during which the host density is below threshold. As we have seen, in table 4, the maximum lifespans of the infective stages can indeed be very long.

At least three circumstances can be distinguished in discussing the role of free-living infective stages in maintaining infections within fluctuating host populations. The first (where the host–parasite interaction itself produces population cycles of periods of 5–12 years or longer) has already been discussed in detail in §§ 11 and 12. The second (where seasonal changes in rate processes drive host population cycles) and the third (host population cycles driven by prey–predator interactions) are discussed below.

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(*d*) *Long-lived infective stages: seasonal changes in rate processes*

In temperate habitats, most invertebrate species exhibit seasonal changes in population density (Fretwell 1972; Krebs 1978). Often the prevalence of microparasitic infection also changes seasonally, presumably in response to the varying host abundance. For example, Beesley (1977) reported seasonal changes in the abundance of larval leatherjackets, *Tipula paludosa*, and in the prevalence of infection with a coccidian pathogen, *Raajeyna nannyla* (figure 24*b*); the peak in pathogen prevalence appears to occur one to two months after the peak in host abundance. In this example, the protozoan parasite produces infective stages (oocysts) that are relatively long-lived in the damp regions that the hosts inhabit (Beesley 1977, 1978). These stages are thus well suited to surviving through the winter months, when host density is very low.

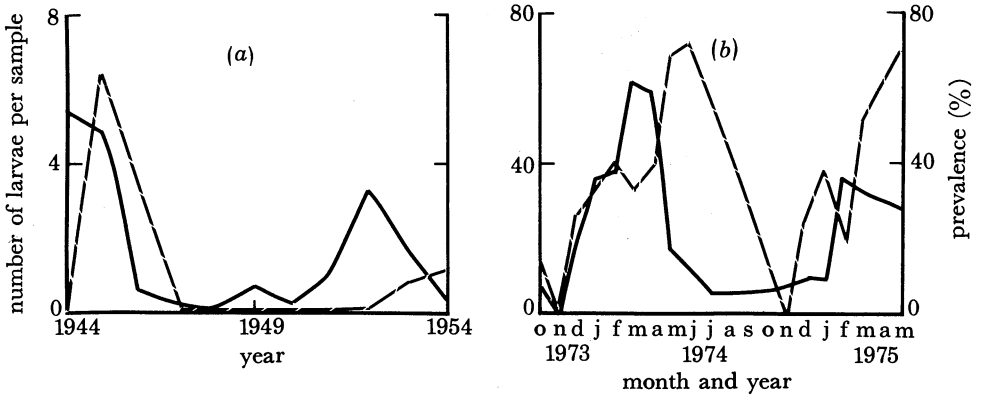

FIGURE 24. Two examples of observed fluctuations in host abundance and in the prevalence of infection. (*a*) Long-term fluctuations in the abundance of the spruce sawfly, *Diprion hercyniae*, in Canada and in the prevalence of infection with a virus (data from Bird & Elgee (1957)). (*b*) Seasonal changes in the abundance of an arthropod, *Tipula paludosa*, in England, and in the prevalence of infection with a protozoan parasite, *Rasajeyna nannyla* (data from Beesley (1977)). The features of these figures are discussed in the text. In both graphs the solid line denotes insect abundance and the dashed line disease prevalence.

A more detailed understanding of the interplay between seasonal changes in host abundance and parasite persistence can be gained by modifying model G to incorporate an annual periodicity in the reproduction rate of the host. That is, we keep equations (60), (61) and (62) for $Y(t)$, $H(t)$ and $W(t)$, respectively, but we replace the constant *per capita* growth rate r of the host population in equation (61) by the time-dependent function $r(t)$:

$$r(t) = [\![A + B\{\sin[2\pi(t-\tau)] + 1\}]\!] - b. \qquad (77)$$

Here t is measured in years (corresponding to $r(t)$ having a one-year period); τ determines the phase of the seasonal changes in r (r has its midpoint value in the months corresponding to $\tau = 0$); and A and B are coefficients defininining the amplitude of the oscillations in the birth rate. This periodic behaviour of $r(t)$ is illustrated in figure 25*a*. All the other rate parameters in equations (60)–(62) remain constant.

A numerical solution of this system of differential equations, for a particular set of parameter values, gives the stably periodic patterns of dynamical behaviour depicted in figure 25*b*. (If $r(t)$ in equation (77) is replaced by its average value, then this particular set of parameter values corresponds to the infection regulating its host population to a stable equilibrium value; the cycles in figure 25*b* are driven by seasonality in equation (77), not by host–parasite

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interaction.) If the pathogen were not present, the host population would undergo exponential growth.

Several general points are illustrated by figure 25*b*, and deserve emphasis. First, seasonal cycles in host abundance generate seasonal cycles in the prevalence of infection. Secondly, the peak prevalence is attained somewhat after the peak in host abundance. This feature of the abstract figure 25*b* is in accord with the data exhibited in figure 24*a*, *b*. Thirdly, the pathogen is able to regulate host population growth, even though r exceeds α (violating the requirement $\alpha > r$ (equation (70)) of model G) during certain months of the year (see figure 25*a*). Fourthly, the pathogen is able to persist despite the host density falling below the threshold value, H_T, for more than half the year; see figure 25*b*. In this example, the infective stages have a constant death rate corresponding to an average lifespan of about two weeks, yet the infection is maintained over the eight-month interval when $H < H_T$ by virtue of the very large numbers of infective stages prodcued at the peak times (a few of which will, by chance, survive as long as the maximum possible lifespan exceeds eight months). The example illustrates the importance of free-living infective stages in the maintenance of disease in seasonal environments, even when the average lifespan of the infective stages is relatively short.

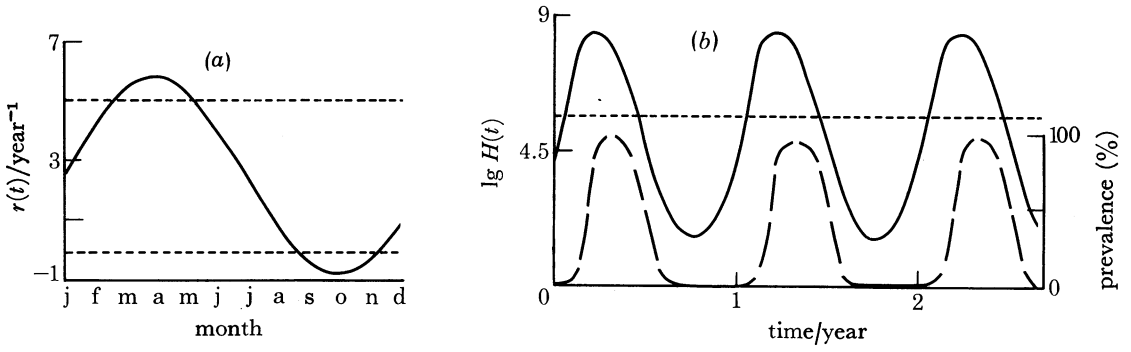

FIGURE 25. This figure illustrates effects that can arise from seasonal changes in host abundance. (*a*) A host population in which the intrinsic growth rate per individual, $r(t)$, changes seasonally, as described by equation (77), with the explicit parameter values $A = 0.1$ year^{-1}, $B = 3.4$ year^{-1}, $b = 1.0$ year^{-1}, and $\tau = 1$ month. As discussed in the text, the upper dashed line denotes $r = \alpha$ (above which level the disease cannot regulate the host population), and the lower dashed line denotes $r = 0$ (below which the disease-free population decreases exponentially). (*b*) The consequent dynamical behaviour of the host and pathogen populations, as described by model G with a seasonally varying $r(t)$, and with $\alpha = 5.0$ year^{-1}, $\mu = 2.0$ year^{-1}, $\gamma = 0$ year^{-1}, $\lambda = 200$ year^{-1} and $\nu = 10^{-6}$ year^{-1}. The solid curve represents the host abundance, $H(t)$, which is below the threshold value, H_T (denoted by the dashed horizontal line), for part of each year; the dashed curve represents the prevalence of infection (expressed as a percentage).

The cycles shown in figure 25*b* are relatively simple. Seasonal changes in the population growth rates and other parameters in equations (60)–(62) are capable of producing much more complex dynamical behaviour.

For instance, if the average rate of host reproduction is relatively low and the infective stages are relatively long-lived and produced in large numbers, seasonality can induce non-seasonal cycles in the prevalence of infection, with periods of two or more years. An example of such a stable cycle is given in figure 26. The parameters here are such that, if $r(t)$ is replaced by its average value, the infection regulates the host population to a stable equilibrium point. The biological explanation of the dynamical behaviour illustrated in figure 26 lies in the inter-

play between the (constant) threshold host density, H_T, for the disease to take hold ($R > 1$), and the tendency toward seasonal variation in $H(t)$. In this figure, we see an epidemic occurring after $H(t)$ rises above H_T. This outbreak of disease causes a dramatic crash in the host population, which carries the prevalence of the infection back to low levels. The host population now grows relatively slowly, with annual oscillations imposed on exponential growth, and it takes two years before $H(t)$ again exceeds H_T, precipitating the next epidemic. The rate constants can clearly be adjusted to produce stable cycles in the outbreak of disease with periods of three or more years.

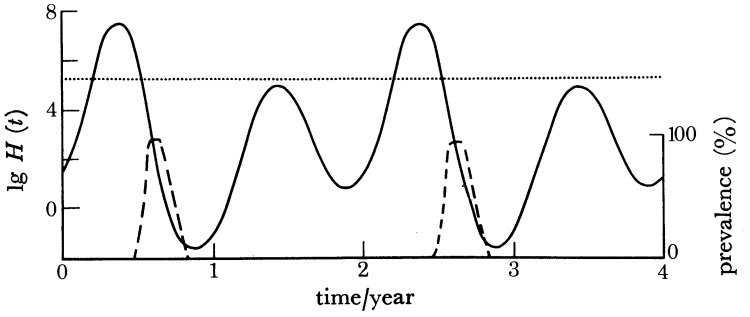

FIGURE 26. This figure is similar to figure 25, showing the dynamical behaviour of host and pathogen populations when there is seasonal variation in the intrinsic growth rate of the host population. The parameters here are $b = 3.0$ year^{-1}, $\alpha = 4.0$ year^{-1}, $\mu = 3.0$ year^{-1}, $\gamma = 0$ year^{-1}, $\lambda = 10^6$ year^{-1}, $\nu = 10^{-10}$ year^{-1}; A, B and τ are as for figure 25. The 'outbreaks' of disease, as measured by prevalence within the host population, here have a 2 year period, as discussed further in the text.

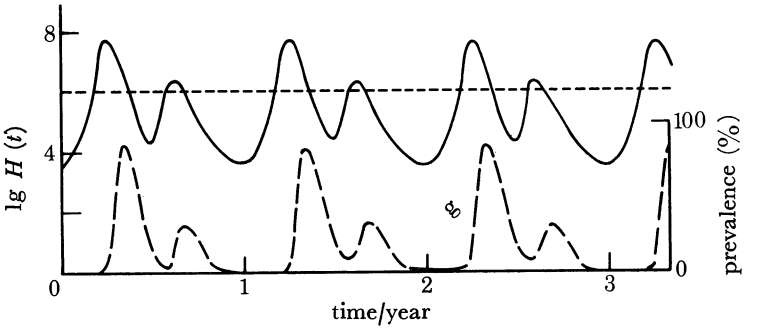

FIGURE 27. This figure is also similar to figure 25, with all parameter values the same as in figure 25 except that here $\alpha = 12.0$ year^{-1}. This relatively high pathogenicity, coupled with the relatively high growth rate of the host population, results in host abundance falling quickly once H_T (horizontal dotted line) is exceeded, yet subsequently rising again quickly, to give two 'outbreaks' of disease in each year. For a fuller discussion, see the text.

Alternatively, the combination of seasonality with relatively high values of the average value of r and very high pathogenicity (α very large) can lead to cycles with periods of less than one year. Figure 27 gives a numerical example. Here, there is an epidemic early in the year, once seasonal increase in host reproduction takes the population above H_T. The resultant effects of disease cause a rapid fall in host density, and subsequently in prevalence. But this fast-growing population recovers quickly enough to climb back above H_T for a second time in the same year, thus triggering a second epidemic.

In short, annual periodicity in the growth rate of the host population can interact with the

effects of a pathogen, to produce stable cycles in the prevalence of infection, with periods greater than, equal to, or less than one year. All this is in the circumstance when the parasite would regulate its host population to a stable equilibrium point if $r(t)$ were replaced by its average value (i.e. in the absence of seasonality). If, however, the rate parameters have values such that the host–parasite association is intrinsically cyclic, as discussed in §§ 11 and 12, the additional complications attendant upon seasonality can lead to extraordinarily complex dynamical behaviour.

Motivated by evidence for epidemics of measles in New York city with a two-year period, Dietz, Yorke and others (London & Yorke 1973; Yorke & London 1973; Dietz 1976; Grossman *et al.* 1977; Yorke *et al.* 1979) have recently shown that annual periodicity in transmission rates can produce non-seasonal cycles in prevalence. Their work employs conventional epidemiological models (simpler than ours in having the total host population assumed constant; more complicated in having a class of immune hosts). The non-seasonal patterns discussed above are broadly related to those found by the above workers, and this area is evidently a rich one, deserving further exploration.

(*e*) *Long-lived infective stages: extrinsic cycles*

Interactions with long-lived predators, or with food supplies that regenerate slowly, can cause host populations to exhibit stable cycles with long periods (May 1974, 1976). The cycles of 30–40 years in the Canadian spruce budworm have, for example, been analysed in this light (Ludwig *et al.* 1978). Such intrinsic cycles in host abundance can lead to long-period cycling in the prevalence of parasitic infections, even if the parasites have little or no effect on the population dynamics of the host.

Such host populations are likely to spend long times at densities below the threshold necessary for disease maintenance. Thus, again, the parasite will need to produce large numbers of long-lived infective stages while its prevalence is high, if it is to persist in the system (but necessarily *outside* the host population for significant periods of time). Many parasites of invertebrates do this. Stairs (1972), for example, reports that an epidemic of a nuclear-polyhedrosis virus in North American tent caterpillars, *Malacosoma* sp., can result in an increase by a factor of 10^{10} in virus polyhedra in forest environments, within a span of 20 days.

For many species of arthropods inhabiting forests or pastures, populations of viral and protozoan parasites tend to exhibit epidemic outbreaks when host density is high (table 6; see also: Stairs 1972; Tanada & Omi 1974; Thompson & Scott 1979; Tinsley 1979). This observation has occasionally prompted the suggestion that transmission efficiency is enhanced by high density (Bailey 1973; Stairs 1972). We wish to stress that such a correlation between high abundance of hosts and high prevalence of disease is exactly as expected on threshold considerations; there is no dynamical reason (nor, to our knowledge, any experimental evidence) to suggest that the individual rate of transmission, the β or ν of our models, is density-dependent.

14. Microparasites in the biological control of pests

Much interest currently centres on the use of viruses, bacteria and protozoans as agents in the control of invertebrate pest species (Tinsley 1979; Anderson 1979*a*, *b*; Bailey 1973; Burges & Hussey 1971; Henry 1971; Huffaker 1974; DeBach 1974; Bucher 1961; Bird 1953). Baculoviruses, in particular, appear to have great promise in the control of certain insect species, such as the forest pests listed in table 7. For example, a nuclear-polyhedrosis virus, sprayed

from the air in an oil–bentonite suspension, has proved very effective in controlling sawfly outbreaks in forest habitats, both in Europe and in North America (McIntyre & Dutky 1961; Smirnoff *et al.* 1962; Bird 1953, 1961; Bird & Whalen 1954; Gershenzon 1969).

What are the main characteristics required of a pathogen if it is to be able to control a given pest species? In what quantities need the pathogen be introduced? Our models for the dynamics of host–parasite associations give some insight into these questions.

Setting aside such complications as vertical transmission, reduction of reproduction in infected hosts and latency (all of which can easily be added if needed, but which complicate the discussion), we modify model G by having the biological control program introduce infective stages of the pathogen into the habitat, at a constant rate, A. Equations (60) and (61) for the dynamics of $Y(t)$ and $H(t)$ remain as before, and equation (62) for $W(t)$ becomes

$$\mathrm{d}W/\mathrm{d}t = A + \lambda Y - (\mu + \tau H)\, W. \tag{78}$$

The condition that must be satisfied if the disease is to regulate host population growth is, as before, equation (69). As discussed earlier, for the large values of λ that are essentially always found (table 5), this control condition boils down to $\alpha > r$; the disease-induced mortality rate must exceed the disease-free growth rate of the host population. Given that this is so, the pathogen will eradicate the host species provided that it is introduced at a rate, A, in excess of a critical value A_c:

$$A > A_c \equiv \frac{\mu r(\alpha + b + \gamma)}{\nu(\alpha - r)}. \tag{79}$$

That is, if $A > A_c$ the system settles to a stable equilibrium with $H^* = 0$, $Y^* = 0$ (host population extinguished) and $W^* = A/\mu$ (population of infective stages in equilibrium between introductions and deaths). If the introduction rate A is not large enough to satisfy equation (79), the pathogen regulates the host population, either to a stable equilibrium or in stable cycles, but does not eliminate it. These results are established in appendix G.

Such control efforts are more likely to be successful if they require relatively low rates of introduction of the pathogen. From equation (79), we see that this means parasites that are highly pathogenic (α large), are long-lived (μ small), and have high transmission efficiency (ν large). In accord with commonsense, both equation (79) and the overriding constraint $\alpha > r$ state that pest species with high population growth rates (large r) are relatively difficult to control.

A quantitative estimate of the critical introduction rate, A_c, is made hard by the difficulty in estimating ν. The quantity A_c can, however, be re-expressed as

$$A_c = \lambda Y_0^*. \tag{80}$$

Here Y_0^* is the equilibrium population of infected hosts, and λY_0^* the equilibrium net rate of production of infective stages by infected hosts, in a natural system where $A = 0$. Thus, if we are using a pathogen found in natural systems and believed to have $\alpha > r$, we have only to introduce infective stages at a net rate in excess of the rate at which they are produced by the pristine host–parasite association, to eradicate the host population. It is plausible that this critical rate can be estimated, and attained, for some of the baculoviruses listed in table 7.

Our simple dynamical models thus suggest that pathogens can be used to control certain pest species having low to moderate r values. One interesting set of possibilities are the

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baculoviruses that already infect many forest pest populations (table 7); their regulatory impact appears to be such that it only requires a boost for eradication to be achieved.

Some cautionary notes must, however, be sounded.

First, our models make no mention of spatial heterogeneity, which is crucial in many multi-species situations. In particular, our analysis of the possibility of eradication has ignored immigration of pests into the population from other areas.

Secondly, numerical studies show that the stable equilibrium state with hosts eliminated is only attained by keeping $A > A_c$ for many years. Even then, constant surveillance and reintroduction of infective stages are needed to prevent resurgence of the pest species; the $H^* = 0$ state is only stable as long as $A > A_c$.

Finally, population dynamics is always confounded by population genetics. Clearly, the pathogen will exert a strong selective pressure on the pest population, selecting for individuals with reduced susceptibility to infection. These problems are examined in the next section.

15. Evolutionary trends

Any study of the relations among the population parameters determining the dynamics of host–parasite associations must ultimately take account of the evolutionary pressures on both host and parasite.

Parasites, by definition, reduce host survival and/or reproduction. Thus, by their nature, parasites tend to select for host individuals with reduced susceptibility to the disease. For this reason alone, we might expect the pathogenicity of the parasite to decrease through evolutionary time. Many examples of this phenomenon can be culled from the literature, for both vertebrate and invertebrate hosts. Among vertebrates, conspicuous examples are the blood-groups conferring a degree of resistance to malaria in regions where the disease is endemic (e.g. the sickle-cell phenomenon in human populations), and the history of myxomatosis introduced into rabbit populations in Australia and Europe (Bradley 1977; Fenner & Ratcliffe 1965). For pathogens of invertebrates, Martignoni (1957) found that the pathogenicity of a granulosis virus of the European larch budmoth, *Eucosoma griseana*, declined during an epidemic outbreak of infection in Switzerland, with larvae collected after the epidemic being much more tolerant to infection than those collected at the onset of the outbreak. Similar changes were observed by Martignoni & Schmid (1961) in populations of the California oakworm, *Phryganidia californica*, infected with a nuclear-polyhedrosis virus. Several laboratory studies have demonstrated reduced susceptibility to microparasitic infection among the survivors of experimental infections (Harvey & Howell 1965; David & Gardiner 1960, 1965; Watanabe 1967).

The intensity of the selective forces exerted by a pathogen on its host are obviously related to the prevalence of the infection within the population. In the cyclic epidemics of viral infections in forest insects, the pathogen will exert strong selective pressure during the outbreaks of disease, but will have little, if any, effect during the interregnum between epidemics. In natural systems, therefore, selection for reduced susceptibility may often fluctuate. Moreover, in many circumstances reduced susceptibility to infection is linked with deleterious traits, such as reduced reproduction. Thus, over a period of years, the average susceptibility can depend on a complex of evolutionary pressures, of which the pathogen is only one along with predation, competition and climatic factors.

Turning from the host population, we note that selection also acts on the parasite itself.

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On the one hand, there can be pressures for high pathogenicity, in that α is often correlated with the ability of the microparasite to reproduce rapidly within the invertebrate host before the host mounts a cellular or humoral response (Maramorosch & Shope 1975). Fast reproduction within the host before such responses become effective can be advantageous, even though the consequence is eventually to destroy the parasite's habitat by killing the host. On the other hand, as is discussed in detail below, there are countervailing aspects of the population dynamics of the parasite that tend to favour reduced pathogenicity.

To explore these evolutionary aspects of the parasite's pathogenicity, we focus on the factors determining the reproductive success of an individual parasite. Within an individual invertebrate host, populations of microparasites often arise by asexual replication processes (not involving exchange of genetic material) originating from an infection created by a single infective stage; hence the microparasitic populations are often genetically homogeneous within a given host. In contrast, the production of transmission stages typically entails some sort of sexual process, where genetic exchange occurs. The reproductive success of a specific genetic strain of pathogen will therefore tend to depend on the number of hosts infected by the transmission stages produced by the host with the primary infection. This quantity, which effectively measures the Darwinian 'fitness' of the parasite, is simply the basic reproductive rate R defined and discussed in § 4.

In model G, which incorporates a description of the infective stages, the basic reproductive rate of the parasite in the case when it regulates the population growth of the host (essentially when $\alpha > r$) is

$$R = \alpha/(\alpha - r). \tag{81}$$

This result is established in appendix F. In these circumstances R is, of course, greater than unity. We see that increasing pathogenicity (large α) will decrease the reproductive success of the pathogen, and that maximum values of R are attained for α lying just above r.

More generally, if the host population is controlled to an equilibrium level, H^*, by factors other than the pathogen, the basic reproductive rate of the parasite is, from equation (63),

$$R = \frac{\lambda \nu H^*}{(\alpha + b + \gamma)(\mu + \nu H^*)}. \tag{82}$$

Selection can act to increase the value of R for the parasite by increasing the rate of production of infective stages (increasing λ), by increasing the longevity of the infective stages (decreasing μ), or, as before, by reducing the pathogenicity (decreasing α).

Equations (81) and (82) suggest that evolutionary pressures on the parasite act to reduce its pathogenicity, independently of whether the parasite actually regulates its host population. This tendency is also advantageous from the point of view of the host.

The fault in the above analysis is that it assumes that evolutionary changes in the various ecological and epidemiological parameters can take place independently of each other. This will rarely be true. In particular, as discussed immediately above, high rate of production of infective stages, λ, is likely to be associated with high pathogenicity, α.

As a crude caricature of this interrelation among the parameters, we may assume in model G that the rate of production of infective stages is directly proportional to the pathogenicity:

$$\lambda(\alpha) = \hat{\lambda}\alpha. \tag{83}$$

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The general dynamical properties remain unchanged, except that $\hat{\lambda}\alpha$ everywhere replaces λ. The basic reproductive rate of the parasite (equation (63) or (82)), now becomes

$$R = \frac{\hat{\lambda}\alpha\nu H^*}{(\alpha+b+\gamma)(\mu+\nu H^*)}. \tag{84}$$

Here, selective pressures acting on the parasite to increase its R will have the effect of increasing the pathogenicity, α. Indeed, a certain minimum value of α is now required if the parasite is to be able to persist ($R > 1$):

$$\alpha > \frac{(b+\gamma)(\mu+\nu H^*)}{\nu H^*(\hat{\lambda}-1)-\mu}. \tag{85}$$

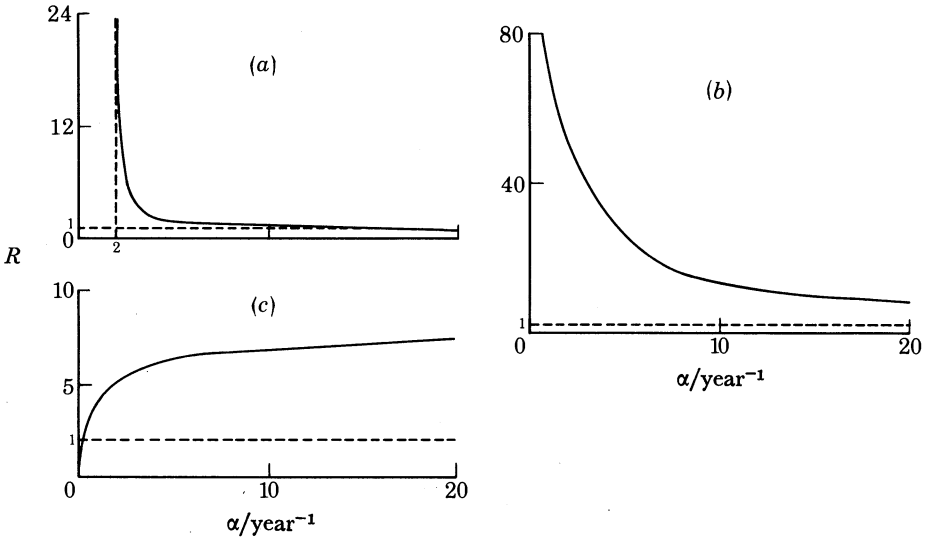

FIGURE 28. The relation between the basic reproductive rate, R, of the parasite and the rate of disease-induced host mortality, α, is illustrated under various assumptions. (*a*) The relation, equation (81), derived from the basic model A with λ independent of α; here R is a dimensionless number, α is in units of year^{-1}, and $r = 2.0$ year^{-1}. (*b*) The more general relation, equation (82), where the equilibrium host population H^* is not necessarily regulated by the pathogen; for illustrative purposes, we take $\nu H^* = 10$, $\lambda = 200$, $\mu = 3$, $b = 1$ and $\gamma = 0$. (*c*) The relation, equation (84), arising when the rate of production of free-living infective stages is dependent on parasite pathogenicity (explicitly, $\lambda = \hat{\lambda}\alpha$ (equation (83)); the parameter values are as for (*b*) except that $\hat{\lambda} = 10$ (all units in year^{-1}).

These points are illustrated in figure 28. It is also worth noting that when the value of λ is associated with α (equation (83)) and when the parasite regulates host population growth ($\alpha > r$), then the equilibrium density of the host population falls steadily with increasing pathogenicity; see appendix F. This contrasts with the simpler, earlier models, where the values of λ and α are independent, and where the equilibrium host density attains its minimum value for intermediate values of α (as shown in figure 6).

More generally yet, we can recall the quantity Λ (equation (58)), which measures the total number of infective stages produced over the lifespan of an infected host. Equation (82) for the basic reproductive rate of the parasite then becomes

$$R = \Lambda\nu H^*/(\mu+\nu H^*). \tag{86}$$

This general expression makes it clear that the quantity of evolutionary significance is the total number of infective stages Λ produced in an infected host; the quantity Λ may be made

large by a rate of production of infective stages so large as to kill the host quickly (large λ and α), or by a relatively slow rate of production extending throughout a long-lived infection (small λ and α). This touches on a broader biological theme. Plants and animals exhibit a spectrum of reproductive strategies, of which the simplistic extremes are the production of a very large number of offspring, few of which survive (the so-called r strategy), and the production of relatively few offspring, with significant parental investment enhancing their probability of reaching maturity (the so-called K strategy). Parasites clearly tend to lie at the r strategy end of this spectrum, since in general they produce enormous numbers of offspring in the form of transmission stages, few of which survive to infect and reproduce in a new host. Even at this extreme of the continuum, however, great variation can exist, with some pathogens producing very large numbers of infective stages with low survival probabilities (large λ or Λ offset by large μ), while other species produce fewer offspring, whose chances of survival in the external habitat are greater (lower λ or Λ offset by lower μ); see equation (86) and the discussion in Bradley (1977).

The essential point is that natural selection will act concomitantly on both host and parasite, with the selective pressures on the host often opposed to those on the parasite. Specifically, selection on the host will tend to decrease α, while selection on the parasite will often tend to increase α. The point where a dynamic balance is struck between forces acting on the host and forces acting on the parasite will generally depend on the detailed natural history of the association, in a way that lies outside the scope of our study (which is orientated to population dynamics rather than evolutionary questions). In some instances, stable oscillations in the abundance of hosts and parasites (as seen in §§ 11, 12, 13) may lead selective pressures to rise and fall rhythmically, resulting in cyclic changes in the genetic constitution of the population (see Pimentel 1968).

Because the generation times of most hosts are very much longer than those of their microparasites, it is often concluded that selection acts more rapidly on the pathogen. However, as we have discussed elsewhere (May & Anderson 1979), when parasites act severely to reduce the survival or reproduction of their hosts, the pace of host evolution tends to be kept in step with that of the parasites.

16. Conclusions

Our theoretical studies and survey of field and laboratory data indicate that the population density of invertebrate species may often be regulated by microparasites, acting alone or in conjunction with other regulatory influences such as parasitoids, predators or resource limitations. This supports the earlier, but less analytical, suggestions of Lack (1954) and other authors (reviewed in Southwood & Comins 1976; Anderson & May 1979*a*; May & Anderson 1979).

Regulation

The criterion for a microparasite to be able to regulate its host population, in our various mathematical models, is essentially that the net death rate of infected hosts exceed their net birth rate. This criterion, which accords with biological intuition, may be met by the pathogen either raising the death rate or depressing the birth rate of infected hosts, or by a combination of both effects. The precise criteria that pertain in the specific biological situations detailed in models A–G are summarized in table 9. The regulated state of the host population may be a

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stable constant value, or a stable cyclic oscillation. In particular, highly pathogenic microparasites that produce large numbers of relatively long-lived infective stages are likely to give rise to population cycles in host species with relatively low intrinsic growth rates. Such circumstances arise for baculovirus and microsporidian protozoan infections of many temperate forest insects, where rough estimates of the parameters characterizing the host–parasite association suggest cycles in host abundance and prevalence of infection with periods in the range 5–12 years; such cycles are indeed observed in many forest insects, and this may be the driving mechanism.

TABLE 9. FACTORS THAT DETERMINE THE REGULATORY POTENTIAL OF A PARASITE

factor	condition that must be satisfied if parasite is to regulate the growth of its host population
high pathogenicity (α) relative to the intrinsic growth rate (r) of the host population: model A	$\alpha > r$
reduced reproduction (by factor $1-f$) in infected hosts: model B	$\alpha > a(1-f)-b$
relatively short latent period of infection ($1/v$): model D	$\alpha > r[1+(\alpha+b+\gamma)/v]$
pathogenicity an increasing function of host density ($\alpha = \hat{\alpha}H$): model E	$\beta > \hat{\alpha}$
production of large numbers of infective stages: model G	$\lambda > \alpha(\alpha+b+\gamma)/(\alpha-r) > 0$

Prevalence

Broadly, the prevalence of infection within the host population is inversely related to the pathogenicity, α. Thus the more pathogenic the parasitic infection, the more likely it is to regulate its host population, yet, paradoxically, the lower will be its equilibrium prevalence. It is, therefore, incorrect to conclude (as ecologists have often done) that a disease of low prevalence is unlikely to contribute to the regulation of host abundance. On the contrary, parasites of low pathogenicity are likely to be very prevalent but to have little effect on the population dynamics of their host, whereas persisting parasites of high pathogenicity that contribute significantly to host population regulation will in general be characterized by low prevalence. These remarks are subject to the reservation that excessively pathogenic parasites are unable to persist within the host populations; i.e. they cause their own extinction (see model F and figure 14). The inverse relation between pathogenicity or regulatory potential and prevalence of the infection holds generally for microparasitic and macroparasitic infections of vertebrates and invertebrates (Anderson & May 1979*a*; May & Anderson 1979); it is basically an example of the inverse relation between standing crop and turnover rate that arises in many biological systems (May 1977).

This observation argues for a reappraisal of some field data, where infectious diseases have been dismissed as inconsequential to the dynamics of invertebrate populations, on the grounds that the prevalence of infection is low.

Thresholds

The interaction between host and parasite is a nonlinear or density-dependent one. In particular, the parasite can only be maintained within the host population if host density exceeds a threshold value, H_T (in this sense, parasitism is an 'all-or-nothing' phenomenon, in contrast with simple models for predation or resource limitation). As shown in table 10,

H_T is broadly dependent on pathogenicity α. Thus parasites of low pathogenicity typically have relatively low threshold host densities and are likely to exhibit patterns of steady, endemic prevalence. Conversely, highly pathogenic parasites typically are associated with high threshold density, which the host population may only attain intermittently; highly pathogenic diseases will often be characterized by patterns of epidemic outbreak. In this latter case, when host abundance exceeds threshold levels periodically or episodically, the parasite may maintain itself during the subthreshold intervals by infective stages that are free-living (outside the host), or by vertical transmission, or by occult infection. In the intervals between outbreaks, other factors, such as parasitoids, predators or food supplies, are likely to be the major determinants of host mortality (Southwood & Comins 1976).

TABLE 10. FACTORS THAT ENHANCE THE ABILITY OF A PATHOGEN TO PERSIST IN A POPULATION OF HOSTS THAT EITHER IS OF LOW DENSITY OR FLUCTUATES WIDELY IN ABUNDANCE

factor	threshold host density, H_T, for maintenance of disease
low pathogenicity (α); low rate of natural host mortality (b); low rate of host recovery from infection (γ); high transmission efficiency (β): model A	$H_T = (\alpha+b+\gamma)/\beta$
vertical transmission: model C	$H_T = (\alpha+b+\gamma-aq)/\beta$
short latent or incubation period: model D	$H_T = [(b+v)/v]\,[(\alpha+b+\gamma)/\beta]$
pathogenicity an increasing function of host density: model E	$H_T = (a+\gamma)/(\beta-\hat{\alpha})$
other 'carrying capacity' constraints on host density corresponding to densities in excess of H_T: model F	$H_T < K$
production of large numbers of infective stages (λ); long-lived infective stages (γ): model G	$H_T = \dfrac{\mu(\alpha+b+\gamma)}{\nu[\lambda-(\alpha+b+\gamma)]}$

In general, as can be seen from the expressions for H_T in table 10, vertical transmission is a mechanism serving to reduce the effective value of the threshold host density, and thus facilitating the persistence of parasites in relatively low-density populations of hosts. To the contrary of some earlier suggestions, we have, however, shown that it is not usually possible for a parasite to be maintained by vertical transmission alone.

The threshold phenomenon, which arises in all our models independent of the nature of the pathogen and the host (just as it does in conventional epidemiological models where host populations are assumed constant), is sufficient to explain the observed correlation between high density of invertebrate hosts and epidemic outbreak of disease. There is no need to invoke (as some authors have done) any association between pathogenicity and stress on the host population, induced by overcrowding, to explain the observed correlations.

Future research

A brief survey of, e.g., the *Journal of Invertebrate Pathology* shows that the rate at which new species or types of viruses and bacteria have been discovered within invertebrate populations has been accelerating in recent years. This is a consequence partly of new techniques (Gibbs 1973; Smith 1976; Tinsley 1979; Whitcomb & Tully 1979) and partly of increasing attention. It suggests that ecologists and parasitologists have identified only a small fraction of the pathogens of invertebrates occurring in natural habitats. This observation, taken in conjunction with our demonstration of the role that pathogens are capable of playing in the natural (or artificial) regulation of invertebrate populations, strongly suggests that considerably more

attention should be paid to the microparasitic organisms associated with natural populations of invertebrates.

The first step in such an enterprise is clearly to identify the microparasitic organisms and to record their prevalence within the host population at different times and places. For a more quantitative understanding of the host–parasite dynamics, however, information about rate processes (such as pathogenicity, α, and transmission efficiency, β or ν) is necessary. Here laboratory studies comparing the dynamics of infected and non-infected populations of hosts can be valuable (cf. figures 2, 3, 4).

Applications

Such theoretical and empirical studies can do more than help remedy the conspicuous absence of discussion of host–parasite associations in contemporary ecology texts. With the increasing incidence of invertebrates evolving resistance to chemical pesticides, pathogens are more and more being used in efforts to control pest species. Insights into the dynamics of the interaction between a pathogen and its invertebrate host can guide the design of laboratory or field experiments, to estimate whether the pathogen is capable of regulating the target population, and, if so, in what quantity it must be introduced to effect a specified level of control or even local eradication (§ 14). The use of pathogens as control agents will, of course, also tend to be beset by problems, arising from evolutionary changes in the genetic make-up of host and pathogen populations. Although we have touched on evolutionary questions briefly (§ 15), our attention has centred on population dynamics; the population genetics of host–parasite associations deserves further attention.

We are indebted to many people, and particularly to M. P. Hassell and T. R. E. Southwood, F.R.S., for helpful comments and advice. This work was supported in part by the N.S.F. under grant DEB79-03290.

Appendix A

Glossary of symbols used in this paper

Population variables

H	total number of hosts
X	number of susceptible hosts
Y	number of infectious hosts
x	susceptible fraction of host population, X/H
y	prevalence of infection, Y/H
H^*	equilibrium number of hosts
y^*	equilibrium value of prevalence
M	number of infected, but not yet infectious, (latent) hosts
W	number of free-living infective stages of parasite
H_T	threshold host density (see table 10)
K	carrying capacity for host population

Rate and other parameters

a	host birth rate (*per individual*)
b	natural mortality rate of hosts

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r intrinsic growth rate of host population, $a-b$

α disease-induced mortality rate

γ rate of host recovery from infection

β transmission coefficient (in models where infection is transmitted directly from infecteds to susceptibles)

R basic reproductive rate of the parasite

ρ exponential growth rate of host population when the disease is maintained, but does not regulate the population to a steady value

f fractional decrease in birth rate of infected hosts; birth rate of infected hosts is $a(1-f)$

q proportion of offspring of infected hosts acquiring infection by vertical transmission

v rate at which hosts pass from infected to infectious class; $1/v$ is length of incubation or latent period

$\hat{\alpha}$ proportionality constant when pathogenicity is proportional to host density, $\alpha = \hat{\alpha}H$.

s parameter determining the severity of density-dependent effects on host mortality; $K = r/s$

d degree of disease-induced depression of host population below disease-free carrying capacity value; $d = 1-H^*/K$

μ mortality rate of free-living infective stages

λ rate of production of infective stages, per infected host

Λ total number of infective stages produced per infected host, on average; $\Lambda = \lambda/(\alpha+b+\gamma)$

ν rate at which infective stages successfully infect hosts (transmission coefficient of free-living infective stages)

A rate of introduction of infective stages (in a biological control program)

Appendix B: models A, B, C

The dynamical properties of model A are outlined in § 5. This appendix presents the underlying mathematical analysis. The analysis is set out in some detail for this basic model A; for the subsequent models B–G the mathematical essentials are similar, and the analysis is presented more sketchily.

Model A

Given the identity $H = X+Y$, we can (as explained in § 5) choose to work with any two of the three equations (12)–(14). Throughout these appendixes, we arbitrarily choose $H(t)$ and $Y(t)$ as the dynamical variables. For notational convenience, we define

$$\Gamma = \alpha+b+\gamma. \tag{B 1}$$

The quantity Γ is the overall rate at which hosts are lost from the infected class, by natural death, disease-induced mortality, or recovery. It is also useful to recall equation (10) for the threshold host density, $\bar{H}_T$, in this simplest, basic model:

$$\bar{H}_T = \Gamma/\beta. \tag{B 2}$$

Model A now consists of the equations (13) and (14); rewritten here for convenience, these are

$$dY/dt = \beta Y(H-Y-\bar{H}_T), \tag{B 3}$$

$$dH/dt = rH-\alpha Y. \tag{B 4}$$

Alternatively, as explained in § 4, we can define the dimensionless variables $H' = H/\bar{H}_T$ and $Y' = Y/\bar{H}_T$, to re-express equations (B 3) and (B 4) as

$$dY'/dt = \Gamma Y'(H' - 1 - Y'), \tag{B 5}$$

$$dH'/dt = rH' - \alpha Y'. \tag{B 6}$$

This makes it clear that the *dynamical* behaviour of the system depends only on the rate parameters r, α and the combination Γ; β or $\bar{H}_T$ enter only in setting the absolute magnitude of the host population.

The analysis of equations (B 3) and (B 4), or (B 5) and (B 6), follows along routine lines (see, for example, May 1974, ch. 2).

First, equilibrium solutions, H^*, Y^*, are found by solving the algebraic equations obtained by setting $dH/dt = 0$ and $dY/dt = 0$:

$$H^* - Y^* = \bar{H}_T, \tag{B 7}$$

$$rH^* - \alpha Y^* = 0. \tag{B 8}$$

Since $H > Y$, it is clear that no meaningful such solutions can be found for equation (B 8) unless $\alpha > r$ (equation (15)). If equation (15) is satisfied, equations (B 7) and (B 8) lead directly to the equilibrium expressions (16)–(18) given in the main text.

Secondly, the stability of this equilibrium in response to small disturbances is determined by the standard linearized analysis. We write $H(t) = H^* + \hat{H}(t)$, $Y(t) = Y^* + \hat{Y}(t)$ in equations (B 3) and (B 4), and discard all terms of second order or higher in $\hat{H}$ and $\hat{Y}$, to arrive at a pair of equations linear in $\hat{H}$ and $\hat{Y}$. The time dependence of the dynamical variables in these linear, first-order differential equations can then be expressed as $\hat{H}(t) = \hat{H}\exp(\sigma t)$, $\hat{Y}(t) = \hat{Y}\exp(\sigma t)$. Thus we arrive at the pair of linear algebraic equations

$$\sigma\hat{Y} = \beta Y^*(\hat{H} - \hat{Y}), \tag{B 9}$$

$$\sigma\hat{H} = r\hat{H} - \alpha\hat{Y}. \tag{B 10}$$

The dynamical response of the system to small disturbances is now characterized by the eigenvalues σ. Using the result $\beta Y^* = r\Gamma/(\alpha - r)$, we see that these are the solutions of the quadratic equation

$$\sigma^2 + [r(a+\gamma)/(\alpha - r)]\sigma + r\Gamma = 0. \tag{B 11}$$

According to the Routh–Hurwitz conditions, both eigenvalues will have negative real parts (corresponding to a locally stable equilibrium point) if, and only if (iff), both coefficients in this quadratic equation are positive, which they obviously are for $\alpha > r$. Thus iff $\alpha > r$ there is a locally stable equilibrium point; small disturbances will die away, either exponentially or by damped oscillations. For a more detailed exposition of the techniques employed here, see, for example, May (1974, ch. 2 and app. I, II).

The dynamical behaviour of the system in the case $\alpha > r$ is illustrated in figure 29*a*, which depicts the 'phase plane' of $H(t)$–$Y(t)$ values (note that the shaded region $Y > H$ is not accessible). The isoclines along which $dY/dt = 0$ ($Y = H - \bar{H}_T$) and $dH/dt = 0$ ($Y = (r/\alpha)H$) are as shown; these isoclines intersect iff $\alpha > r$. The arrows show the directions of the dynamical trajectories in the various regions of this phase plane, and one typical trajectory is illustrated. It can be shown that equations (B 3) and (B 4) satisfy the Kolmogorov conditions (see, for example, May 1974, ch. 4), so that if a locally stable equilibrium point exists, it is globally stable.

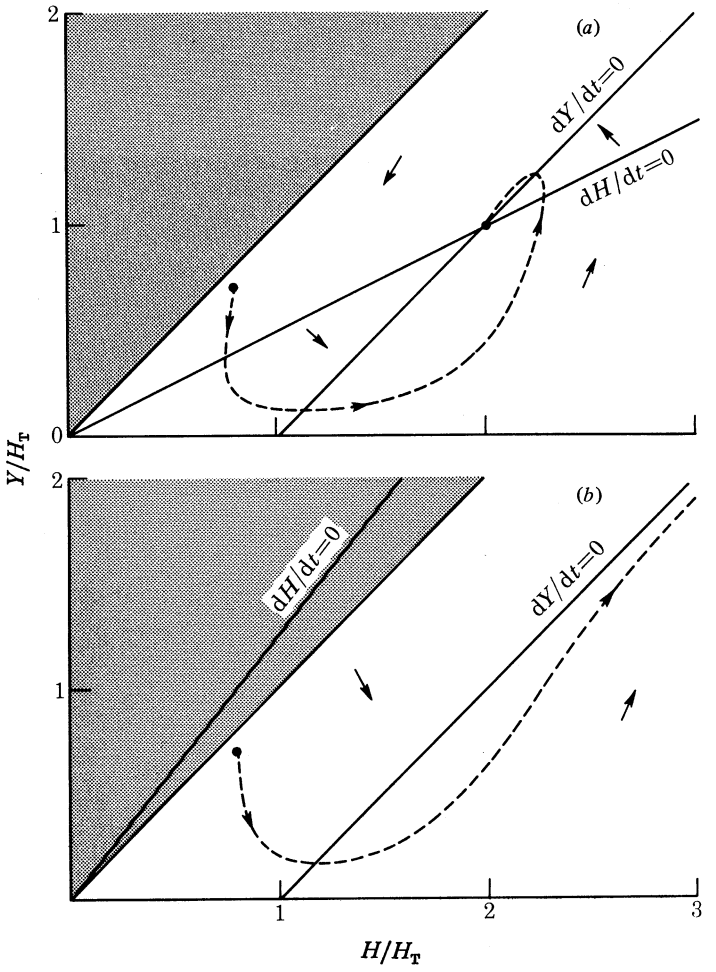

FIGURE 29. (*a*) This illustrates the *H*–*Y* phase plane for the basic model A. The total of number of hosts H and the number of susceptible hosts Y are plotted in dimensionless form as H/H_T and Y/H_T; note that the shaded region $Y > H$ is inaccessible. The figure shows the isoclines along which $dY/dt = 0$ and $dH/dt = 0$; these isoclines intersect, giving a stable equilibrium point, if $\alpha > r$. The arrows show the general direction in which dynamical trajectories must move in the various parts of the phase plane, and one typical trajectory is illustrated. (*b*) The *H*–*Y* phase plane is again depicted for host–parasite associations obeying the basic model A. Here, in contrast to in (*a*), $r > \alpha$ and the isoclines along which $dY/dt = 0$ and $dH/dt = 0$ cannot intersect (indeed, dH/dt is necessarily positive for all accessible values of H and Y). Host and parasite populations thus both asymptotically undergo unbounded exponential growth, as discussed in the text; a typical trajectory is illustrated.

In short, either analytic or geometric methods can be used to show that the basic model A possesses a globally stable equilibrium point iff $\alpha > r$.

Conversely, if $r > \alpha$ there is no equilibrium point, and $H(t)$ and $Y(t)$ undergo unbounded exponential increase. This circumstance is illustrated in figure 29*b*.

To study the asymptotic behaviour of model A in the case $r > \alpha$, we first combine equations (B 3) and (B 4) to get a differential equation for the prevalence of infection, $y \equiv Y/H$:

$$dy/dt = (dY/dt - y\, dH/dt)/H, \qquad \text{(B 12)}$$

$$= y[\beta H(1-y) - (\Gamma + r - \alpha y)]. \qquad \text{(B 13)}$$

Now suppose that $H(t)$ grows asymptotically at some exponential rate ρ:

$$H(t) \to (\text{constant}) \exp \rho t. \tag{B 14}$$

Since $y(t)$ cannot continue to grow exponentially, equation (B 13) implies that, asymptotically,

$$y \to 1. \tag{B 15}$$

Thence, from equation (B 4),

$$\rho \to r - \alpha. \tag{B 16}$$

This is the result, equation (20), discussed in the main text. Returning to equation (B 3), and remembering that $X = H - Y$, we obtain an expression for the asymptotically constant number of susceptibles, X^*, for when case $r > \alpha$:

$$\rho = \beta(X^* - \bar{H}_T). \tag{B 17}$$

In conjunction with equation (B 16) for ρ, and equation (B 2) for $\bar{H}_T$, this gives the result quoted in the main text:

$$X^* = (a+\gamma)/\beta. \tag{B 18}$$

It is clear from the above analysis (see, for example, equation (B 3)), or from the more intuitively based discussion in the text, or from figure 29, that $Y(t)$ can increase from small initial values only if $H > \bar{H}_T$. This makes plain the role of the threshold host density. Similarly, the dimensionless equation (B 5) shows that the infection can maintain itself only if the dimensionless quantity $H' - 1$ is positive, which leads to the definition of the basic reproductive rate of the parasite,

$$R = H' = H/\bar{H}_T = \beta H/\Gamma. \tag{B 19}$$

At equilibrium, the parasite's basic reproductive rate (obtained by substituting from equation (17) for H^*) is

$$R^* = \alpha/(\alpha - r). \tag{B 20}$$

These expressions for the threshold host density and the basic reproductive rate of the parasite are discussed more fully in §§ 4, 5 and 15.

Model B

The effects of parasite-induced reduction of host reproduction are incorporated in the set of differential equations (21)–(23). These equations may be obtained directly from model A by formally rewriting the parameters α and γ in the model as:

$$\alpha \to \alpha + fa, \tag{B 21}$$

$$\gamma \to \gamma - fa. \tag{B 22}$$

Note that $\Gamma \equiv \alpha + b + \gamma$ remains unaltered under this transformation. Hence, *mutatis mutandis*, all the results for model B follow immediately from those for model A. In particular, the expressions for R and H_T involve the combination of rate parameters Γ, and thus do not change. But the criterion for the parasite to regulate its host population involves α, and hence does change (equation (24)).

Model C

Similarly, the effects of vertical transmission are incorporated in the set of differential equations (27)–(29), which are formally identical to model A, with the redefinition

$$\gamma \to \gamma - aq. \tag{B 23}$$

Note that this implies $\Gamma \to \Gamma - aq$. Again, *mutatis mutandis*, all the results for model C follow from those for model A. The condition for the parasite to regulate its host population does not change (because α does not change), but the expressions for R and H_T do change (because the expression for Γ changes.)

At the end of § 7 we asserted that a parasite cannot be maintained by vertical transmission alone. To prove this, we put $\beta = 0$ (no horizontal transmission) in the appropriately altered (that is, $\gamma \to \gamma - aq$) equation (B 13) for the dynamics of the prevalence of infection:

$$dy/dt = -y(\alpha + a + \gamma - aq - \alpha y). \tag{B 24}$$

Rearranging this equation, we have

$$dy/dt = -y[\alpha(1-y) + a(1-q) + \gamma]. \tag{B 25}$$

The expression inside the square brackets is necessarily positive (except in the extreme circumstance of zero pathogenicity, perfect vertical transmission and no recovery: $\alpha = 0$, $q = 1$, $\gamma = 0$). Therefore $dy/dt < 0$, and the prevalence must decline toward zero.

Appendix C: model D

If a class of latent (infected but not infectious) individuals M is added to model A, we have model D. Expressed in terms of the dynamical variables $H(t)$, $M(t)$ and $Y(t)$, these equations (33)–(35) are

$$dH/dt = rH - \alpha Y, \tag{C 1}$$

$$dM/dt = \beta Y(H - M - Y) - (b+v)M, \tag{C 2}$$

$$dY/dt = vM - \Gamma Y. \tag{C 3}$$

The number of infectious individuals will increase from an initially small value provided that $\beta HY > (b+v)M$ and $vM > \Gamma Y$; that is, if

$$\beta v H/\Gamma(v+b) > 1. \tag{C 4}$$

This underlies the definition of R given in equation (36), and leads to equation (37) for the threshold host density, H_T.

Putting all time derivatives equal to zero in equations (C 1)–(C 3), and solving the resulting set of three simultaneous algebraic equations, we get the equilibrium expressions

$$H^* = \frac{\Gamma}{\beta}\frac{(b+v)}{v}\frac{\alpha}{[\alpha - r(1+\Gamma/v)]}, \tag{C 5}$$

$$M^* = (r\Gamma/\alpha v)H^*, \tag{C 6}$$

$$Y^* = (r/\alpha)H^*. \tag{C 7}$$

The criterion $\alpha > r(1+\Gamma/v)$ of equation (38) follows immediately if a biologically sensible equilibrium solution, $H^* > 0$, is to be possible.

The stability of this equilibrium point is studied by a straightforward extension of the linearized analysis outlined in appendix B for model A. Writing $H(t) = H^* + \hat{H}(t)$ $M(t) = M^* + \hat{M}(t)$, $Y(t) = Y^* + \hat{Y}(t)$, expanding in Taylor series and extracting the time-dependence of the dynamical variables in the consequent set of three linear differential equations as $\exp(\sigma t)$, we arrive at

$$\sigma\hat{H} = r\hat{H} - \alpha\hat{Y}, \tag{C 8}$$

$$\sigma\hat{M} = (\beta Y^*)\hat{H} + \beta(H^* - M^* - 2Y^*)\hat{Y} - (\beta Y^* + b + v)\hat{M}, \tag{C 9}$$

$$\sigma\hat{Y} = -\Gamma\hat{Y} + v\hat{M}. \tag{C 10}$$

The stability-determining eigenvalues σ thus obey the cubic equation

$$\sigma^3 + A\sigma^2 + B\sigma + C = 0. \tag{C 11}$$

The coefficients A, B, C are defined as

$$A = \Gamma + b + v - r + [r\Gamma/(\alpha - \hat{r})][(b+v)/v], \tag{C 12}$$

$$B = [r\Gamma/(\alpha - \hat{r})][(b+v)/v](\Gamma + v - r) - r(\Gamma + b + v), \tag{C 13}$$

$$C = r\Gamma(b+v). \tag{C 14}$$

Here

$$\hat{r} \equiv r(1+\Gamma/v). \tag{C 15}$$

The condition for an equilibrium point to be possible (equation (38)) is thus $\alpha > \hat{r}$. The Routh–Hurwitz criteria for all three roots to have negative real parts (corresponding to a locally stable equilibrium) are $A > 0$, $B > 0$, $C > 0$ and

$$AB > C. \tag{C 16}$$

For a more full exposition, see May (1974, app. II). It is relatively easy to show that A, B and C are indeed positive quantities, provided that $\alpha > \hat{r}$. The remaining stability condition, $AB > C$, is, however, not always fulfilled. Without becoming enmeshed in too much detail, we now indicate some limiting cases.

First, if the latent period is short compared with all other relevant time scales (i.e. v greater than all other relevant rate parameters), we have the limiting expressions $A \to v$, $B \to vr(a+\gamma)/(\alpha - r)$, $C \to vr\Gamma$. In this limit, $AB > C$, and the equilibrium point is stable (as we expect, because in the formal limit as $v \to \infty$ we recover model A).

Secondly, if the pathogenicity is very high, so that the lifespan of infectious individuals is shorter than any other relevant time scale in the system (i.e. α significantly greater than all other rate parameters), we have $A \to \alpha$, $B \to \alpha rb/v$, $C \to \alpha r(b+v)$. Again $AB > C$ in this limit, and again the equilibrium point is stable. This result is less intuitively obvious than the preceding limit of a very short latent period.

Thirdly, suppose that the latent period is very long (v very small), so that $\hat{r} = r(1+\Gamma/v)$ increases toward the value α (remember that for $\hat{r} > \alpha$ there is no disease-regulated equilibrium; see equation (38)). As $\alpha - \hat{r}$ becomes small, its reciprocal becomes large, and hence so do A and B. That is, in the limit of a long latent period (but still $\alpha \to r(1+\Gamma/v)$), $AB > C$, and there is a stable point. Again, the result is not intuitively obvious.

Fourthly, and finally, consider the case when the host population's vital rates a, b and r ($r = a-b$) are all much smaller than both the rate parameters α and v. Further, for simplicity, let the recovery rate be zero: $\gamma = 0$. In this limit, $A \to (\alpha+v)$, $B \to ra[(\alpha+v)^2-\alpha v]/\alpha v$, and $C \to r\alpha v$. The stability criterion, equation (C 16), now becomes

$$a > \frac{\alpha^2 v^2}{(\alpha+v)\,[(\alpha+v)^2-\alpha v]}. \tag{C 17}$$

We see that this condition may well be satisfied if either one of α or v is much larger than the other (as discussed in the first and second cases above). But if α and v are of comparable magnitude and both significantly larger than the individual host birth rate, a, equation (C 17) will not be fulfilled; in this event, it appears that the system will exhibit stable limit cycle behaviour.

In most of the discussion in the main text, the latent period is assumed to be short (very large v), and these complications are effectively ignored.

Appendix D: model E

Model E, which allows for the effects of stress-dependent pathogenicity, is a straightforward extension of the basic model A. The expressions for the parasite's basic reproductive rate and for the threshold host density need no discussion.

The equilibrium values of H^* and Y^* are found by putting $\mathrm{d}H/\mathrm{d}t = 0$ and $\mathrm{d}Y/\mathrm{d}t = 0$ in equations (43) and (42), respectively:

$$\beta(H^*-Y^*)-(b+\gamma)-\hat{\alpha}H^* = 0, \tag{D 1}$$

$$r-\hat{\alpha}Y^* = 0. \tag{D 2}$$

These equations lead to equation (47) for H^*, provided that $\beta > \hat{\alpha}$. As outlined in appendix B, the local stability of this equilibrium point is characterized by the eigenvalues σ, which obey the linearized equations

$$\sigma\hat{Y} = Y^*[(\beta-\hat{\alpha})\hat{H}-\beta\hat{Y}], \tag{D 3}$$

$$\sigma\hat{H} = -\hat{\alpha}H^*\hat{Y}. \tag{D 4}$$

It follows that the eigenvalues are the solutions of the quadratic equation

$$\sigma^2+(\beta Y^*)\,\sigma+\hat{\alpha}(\beta-\hat{\alpha})\,H^*Y^* = 0. \tag{D 5}$$

Both coefficients in this quadratic equation are positive if $\beta > \hat{\alpha}$, and hence if the equilibrium point exists it is locally stable.

Appendix E: model F

The introduction of density dependent constraints, additional to those associated with the host–parasite interaction itself, does not affect the basic analysis set out in appendix B. It does, however, substantially add to the algebraic complications in carrying out the analysis.

Expressed in terms of the dynamical variables $H(t)$ and $Y(t)$, equations (50) and (51) for model F become:

$$\mathrm{d}Y/\mathrm{d}t = Y[(\beta-s)\,H-\beta Y-\Gamma], \tag{E 1}$$

$$\mathrm{d}H/\mathrm{d}t = rH-sH^2-\alpha Y. \tag{E 2}$$

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Here $r = a - b_0$; Γ is defined as in equation (B 1),

$$\Gamma = \alpha + b_0 + \gamma; \tag{E 3}$$

and H_T is as in equation (55), namely $H_T = \Gamma/(\beta - s)$. In the absence of the disease, the host population has a stable equilibrium at the carrying capacity, $K = r/s$ (see equation (52)).

The isoclines for this system are illustrated in figure 12 (albeit in the X–Y phase plane). The equilibrium values H^* and Y^* are, as ever, obtained by setting $dY/dt = 0$ and $dH/dt = 0$, and solving the resulting pair of simultaneous algebraic equations. The outcome is a quadratic equation for H^*, which has the positive solution

$$H^*/K = \tfrac{1}{2}\{1 - \zeta + [(1-\zeta)^2 + 4\zeta\xi]^{\frac{1}{2}}\}. \tag{E 4}$$

Here ζ and ξ have been defined for notational convenience:

$$\zeta = \frac{\alpha}{r}\left(1 - \frac{s}{\beta}\right), \tag{E 5}$$

$$\xi = H_T/K = \frac{\Gamma}{r}\left(\frac{s}{\beta - s}\right). \tag{E 6}$$

The quantity ξ is the ratio between the threshold host density for maintenance of the parasite, and the disease-free carrying capacity. Notice also the requirement that $\beta > s$, which is discussed following equation (53) in the main text. The expression for the equilibrium population of infected hosts is

$$Y^*/K = \tfrac{1}{2}(1 - s/\beta)\{1 - \zeta - 2\xi + [(1-\zeta)^2 + 4\zeta\xi]^{\frac{1}{2}}\}. \tag{E 7}$$

For the disease to be maintained at equilibrium, we obviously require that $Y^* > 0$, whence (for $\beta > s$, equation (54)) we require

$$1 - \zeta - 2\xi + [(1-\zeta)^2 + 4\zeta\xi]^{\frac{1}{2}} > 0. \tag{E 8}$$

Some routine algebraic manipulation shows the inequality (E 8) to be fulfilled iff

$$\xi < 1. \tag{E 9}$$

This is equation (56) of the main text; there is an equilibrium solution in which the parasite is maintained iff $H_T < K$. More explicitly, it can be shown from equations (E 4) and (E 7) that if $H_T < K$ ($\xi < 1$), then $Y^* > 0$ and $K > H^* > H_T$. On the other hand, if $H_T > K$ ($\xi > 1$), then $Y^* < 0$ and $K < H_T$.

It remains to prove this equilibrium point is locally stable. Following the standard procedure, we linearize equations (E 1) and (E 2) around the equilibrium point H^*, Y^*, to obtain the pair of equations

$$\sigma\hat{Y} = Y^*[(\beta - s)\hat{H} - \beta\hat{Y}], \tag{E 10}$$

$$\sigma\hat{H} = r(1 - 2H^*/K)\hat{H} - \alpha\hat{Y}. \tag{E 11}$$

The eigenvalues σ are thus the solutions of a quadratic equation

$$\sigma^2 + A\sigma + B = 0, \tag{E 12}$$

with the definitions

$$A = r[(\beta/s - 1)(H^*/K - \xi) + 2H^*/K - 1], \tag{E 13}$$

$$B = r\beta Y^*[2H^*/K - 1 + \zeta]. \tag{E 14}$$

According to the standard Routh–Hurwitz conditions, the equilibrium point is locally stable iff $A > 0$ and $B > 0$. From equation (E 14) in conjunction with equation (E 4), it is clear that $B > 0$. The proof that $A > 0$ is trickier. First notice the definitions in equations (E 3), (E 5) and (E 6) imply that

$$\beta/s - 1 = \Gamma/r\xi > \zeta/\xi. \tag{E 15}$$

Thus in equation (E 13) we have

$$\xi A/r > \zeta(H^*/K - \xi) + \xi(2H^*/K - 1). \tag{E 16}$$

Substituting from equation (E 4) for H^*/K, we can now show that the expression on the right side of equation (E 16) is positive when $\xi < 1$, whence $A > 0$ for $\xi < 1$. This completes the proof that the disease-regulated equilibrium point (which only exists if $\xi < 1$) is stable. The phase-plane portrait of figure 12 suggests that local and global stability go together here.

Finally, the degree to which the disease depresses the host population below its disease-free level K (see equation (57)) is

$$d = \tfrac{1}{2}\{1 + \zeta - [(1-\zeta)^2 + 4\zeta\xi]^{\frac{1}{2}}\}. \tag{E 17}$$

As expected (and as illustrated in figure 14), $d \to 0$ both when $\alpha \to 0$ (i.e. $\zeta \to 0$) and when $H_T \to K$ (i.e. when $\xi \to 1$).

Appendix F: model G

The system with free-living infective stages, model G, is defined by the set of differential equations (60)–(62). Following the scheme outlined in § 12 (preceding the set of approximate equations (74)–(76)), we introduce the dimensionless variables $H' = H/\bar{H}_T$, $Y' = Y/\bar{H}_T$ and $W' = \mu W/\lambda\bar{H}_T$ (with $\bar{H}_T$ defined by equation (B 2) in conjunction with equation (67) for β; i.e. $\bar{H}_T = \mu\Gamma/\nu\lambda$):

$$\mathrm{d}Y'/\mathrm{d}t = \Gamma W'(H' - Y') - \Gamma Y', \tag{F 1}$$

$$\mathrm{d}H'/\mathrm{d}t = rH' - \alpha Y', \tag{F 2}$$

$$\mathrm{d}W'/\mathrm{d}t = \mu Y' - \mu[1 + (H'/\Lambda)]\,W'. \tag{F 3}$$

Here Γ is defined, as before, by equation (B 1), and Λ is the total number of free-living infective stages produced in the lifespan of the average infected host (equation (58)). As discussed in § 11, it will usually be an excellent approximation to put $\Lambda \to \infty$, whereupon the approximate set of (dimensionless) equations (74)–(76) is obtained.

As always, the equilibrium solution H^*, Y^*, W^* is found by putting all time derivatives equal to zero, and solving the ensuing set of algebraic equations. The result, expressed in terms of the absolute population variables H^* and W^*, is

$$H^* = \frac{\Gamma}{\beta(1 - r/\alpha - 1/\Lambda)}, \tag{F 4}$$

$$y^* = r/\alpha, \tag{F 5}$$

$$W^* = r\Gamma/\nu(\alpha - r). \tag{F 6}$$

Notice that we require the denominator in equation (F 4) to be positive. Thus a disease-regulated equilibrium is only possible if

$$\Lambda > \alpha/(\alpha - r) > 0. \tag{F 7}$$

Substituting from the definition $\Lambda = \lambda/\Gamma$ (equation (58)) we obtain the condition, equation (69), discussed in the main text. In the usual limit $\Lambda \to \infty$, equation (F 4) reduces to the approximate result given in the main text, equation (72), and the condition for the parasite to be capable of regulating its host population (equation (F 7) or (69)) becomes simply $\alpha > r$ (equation (70)).

For the stability analysis, we expand around the equilibrium point, $H'(t) = H'^* + \hat{H}(t)$, $Y'(t) = Y'^* + \hat{Y}(t)$, $W'(t) = W'^* + \hat{W}(t)$, and discard terms of second or higher order in the quantities $\hat{H}$, $\hat{Y}$, $\hat{W}$. Extracting the time-dependence of these dynamical variables in the subsequent linear differential equations, in the factor $\exp(\sigma t)$, we arrive via the canonical route at the set of equations

$$\sigma\hat{Y} = \Gamma W'^*\hat{H} - \Gamma(W'^* - 1)\hat{Y} + \Gamma(H'^* - Y'^*)\hat{W} \tag{F 8}$$

$$\sigma\hat{H} = r\hat{H} - \alpha\hat{Y}, \tag{F 9}$$

$$\sigma\hat{W} = -(\mu W'^*/\Lambda)\hat{H} + \mu\hat{Y} - \mu(1 + H'^*/\Lambda)\hat{W}. \tag{F 10}$$

This set of simultaneous linear equations gives a cubic equation for the eigenvalues σ:

$$\sigma^3 + A\sigma^2 + B\sigma + C = 0. \tag{F 11}$$

Here the coefficients A, B, C are defined as

$$A = \mu\Delta + \alpha\Gamma/(\alpha - r) - r, \tag{F 12}$$

$$B = \mu r\Delta(\Gamma - \alpha + r)/(\alpha - r), \tag{F 13}$$

$$C = r\mu\Gamma, \tag{F 14}$$

and the quantity Δ is

$$\Delta = (\alpha - r)/(\alpha - r - \alpha/\Lambda). \tag{F 15}$$

The Routh–Hurwitz criteria for all three roots of this cubic to have negative real parts (corresponding to the equilibrium point being locally stable) are, as before, $A > 0$, $B > 0$, $C > 0$, and $AB > C$. As long as the basic condition of equation (F 7) is satisfied, A, B and C are all positive; the remaining condition,

$$AB > C, \tag{F 16}$$

may, or may not, be satisfied, depending on the values of the rate parameters. If the values of A, B and C are such that equation (F 16) is satisfied, then there is a stable equilibrium point with the host population having the value H^* of equation (F 4) and the population of free-living infective stages having the value W^* of equation (F 6). Conversely, if equation (F 16) is not satisfied (but equation (F 7) is), numerical investigations show that the system settles to some unique stable limit cycle. Results from numerical studies of the period of these stable limit cycles are shown in figure 20. In the limit $\Lambda \to \infty$, we have $\Delta \to 1$; substitution of the resulting relatively simple expressions for A, B and C into equation (F 16) leads to equation (71), which describes this approximate boundary between stable point and stable limit cycle behaviour (when the parasite is capable of regulating its host population).

The types of parameter combinations likely to produce stable limit cycles are discussed in the main text, in §§ 11 and 12 and in figures 18 and 19. In particular, in the limit when $\alpha \gg \mu$ and $\alpha \gg r$ (and $\Lambda \to \infty$), we have $A \approx \alpha$, $B \approx r\mu(a + \gamma)/\alpha$, and $C \approx r\mu\alpha$. Under these circumstances, $C > AB$, and the roots of the cubic equation (F 11) are approximately

$\sigma \approx -\alpha$ and $\sigma \approx \pm i(\mu r)^{\frac{1}{2}}$ (with correction terms of relative order $(\mu r)^{\frac{1}{2}}/\alpha$). That is, the system oscillates in a stable limit cycle, the approximate period of which is

$$T \approx 2\pi(\mu r)^{-\frac{1}{2}}. \tag{F 17}$$

The numerical studies summarized in figure 20 show that this approximation tends to underestimate the period of the cycles, unless α is much greater than μ and r.

If the condition (F 7) is not satisfied (which for $\Lambda \to \infty$ means when $r > \alpha$), the disease cannot regulate the host population to a steady value or in a stable limit cycle. In this event, $H(t)$ will undergo unbounded exponential growth, at some growth rate ρ;

$$H'(t) \to (\text{constant}) \exp(\rho t). \tag{F 18}$$

The analysis of this case proceeds along the line laid down more fully in appendix B for model A. For equations (F 1) and (F 2) to be satisfied, $Y'(t)$ must increase at the same rate as $H'(t)$:

$$Y'(t) \to yH'(t), \tag{F 19}$$

with the prevalence y tending asymptotically to a constant value. For equation (F 3) to be satisfied, it can be shown that $W'(t)$ takes the asymptotic form

$$W'(t) \to \Lambda y(1-\Lambda/H' + \ldots). \tag{F 20}$$

Here the correction terms are of order $\exp(-2\rho t)$. The asymptotically constant quantities ρ and y are now found by substituting the formulae (F 18)–(F 20) into equations (F 1) and (F 2), to get

$$\rho y = \Gamma y[\Lambda(1-y) - 1], \tag{F 21}$$

$$\rho = r - \alpha y. \tag{F 22}$$

Thus the prevalence settles to the asymptotic value

$$y \to \frac{\Gamma\Lambda - \Gamma - r}{\Gamma\Lambda - \alpha}. \tag{F 23}$$

Equation (F 23) in conjunction with equation (F 22) gives the growth rate ρ of the host population at large times. Notice that the expression (F 23) for y is necessarily less than unity, but is positive only if

$$\Gamma\Lambda - \Gamma > r. \tag{F 24}$$

Recalling the definition $\Gamma\Lambda = \lambda$, we arrive at equation (73) in the main text as the condition for the disease to be maintained in the 'run-away' host population. In the usual limit $\Lambda \to \infty$, equation (F 24) or (73) is automatically satisfied, and $y \to 1$ and $\rho \to r - \alpha$ (as in model A) after sufficient time has elapsed.

Conversely, if equation (F 24) or (73) is not fulfilled, asymptotically $y \to 0$ and the disease is *not* maintained in the host population, which grows exponentially at the disease-free population growth rate, r.

The above analysis lays bare the four qualitatively distinct regimes of dynamical behaviour catalogued and discussed in § 11.

It remains to establish the results for R and H_T that are discussed in § 15. Using the original,

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unscaled form of equations (60)–(62), we see that a small initial population of infected host individuals will tend to increase provided that $\nu HW > \Gamma Y$ and $\lambda Y > (\mu+\nu H)W$; that is, if

$$\frac{\lambda\nu H}{\Gamma(\mu+\nu H)} > 1. \tag{F 25}$$

In the usual way (discussed more fully in § 4 and appendix B), we identify the left side of equation (F 25) with the basic reproductive rate, R, of the parasite. This gives the general equations (63) and (82). Substituting from equation (F 4) for H^*, the equilibrium value of R is seen to be simply

$$R^* = \alpha/(\alpha-r), \tag{F 26}$$

as stated in equation (81); this result could alternatively be obtained more directly by the general biological argument outlined in § 4. The above results do not require λ to be independent of α, and therefore remain valid if λ is some arbitrary function of α. Hence we obtain equation (84) for R when $\lambda = \hat{\lambda}\alpha$, and equation (86) for R in general.

As explained in the main text, the threshold host density H_T is that density for which the disease only just maintains itself, $R = 1$. From equation (63) or (F 25), or directly from the basic equations (60) and (62), we get

$$H_T = \frac{\mu\Gamma}{\nu\lambda(1-1/\Lambda)}. \tag{F 27}$$

This is equation (66) of the main text. Under the usual approximation $\Lambda \to \infty$, and with equation (67) defining $\beta = \nu\lambda/\mu$, we recover the approximate result $H_T = \Gamma/\beta$ of equation (68). Again, the formulae for H_T all remain valid in the event that λ is some arbitrary function of α.

Model G *combined with model* B

Figure 19 shows the various domains of dynamical behaviour (stable point, stable limit cycle, exponential growth with or without the parasite maintained) in μ–r parameter space, in the case where the parasite diminishes the reproduction of infected hosts. The underlying equations here are a combination of models G and B. That is, they are equations (60)–(62) in conjunction with the formal replacements

$$\alpha \to \alpha + fa, \tag{F 28}$$

$$\gamma \to \gamma - fa, \tag{F 29}$$

and thus $\Gamma \to \Gamma$ (see the discussion of model B in appendix B). All the formulae and discussion in the present appendix, and in § 11, now apply, subject only to the reinterpretation of the parameters α and γ indicated by equations (F 28) and (F 29). In particular, the quantities A, B, C and Δ of equations (F 12)–(F 15), which are involved in determining the boundary between stable point and stable limit cycle behaviour when the disease does regulate its host population, are now:

$$A' = \mu\Delta' + \Gamma(\alpha+fa)/(\alpha+fa-r) - r, \tag{F 30}$$

$$B' = \mu r\Delta'(\Gamma-\alpha-fa+r)/(\alpha+fa-r), \tag{F 31}$$

$$C' = r\mu\Gamma, \tag{F 32}$$

$$\Delta' = (\alpha+fa-r)/[\alpha+fa-r-(\alpha-fa)\Lambda]. \tag{F 33}$$

In the usual limit $A \to \infty$, $A' \to 1$ as before, and the condition $A'B' > C'$ for stable point (rather than stable limit cycle) behaviour becomes

$$[\mu + (\alpha + fa) D' - r](D' - 1) - \Gamma > 0. \tag{F 34}$$

Here D' is defined for notational convenience as

$$D' = \Gamma/(\alpha + fa - r). \tag{F 35}$$

It is interesting to compare equation (F 34) with the corresponding stability criterion, equation (71), in the absence of any decrease in the reproductive capabilities of infected hosts. As f increases toward unity, both D' and $(\alpha + fa) D'$ are smaller than the corresponding factors D and αD in equation (71). Thus, other things being equal, equation (F 34) for finite f is harder to satisfy than is equation (71) for $f = 0$, and stable limit cycle behaviour is more easily exhibited by host–parasite systems in which the parasite diminishes host reproduction. These trends are illustrated in figure 19.

Appendix G

When free-living infective stages of the parasite are artificially introduced at a rate A, as discussed in § 14, the model G system obeys equations (60), (61) and (72), namely

$$\mathrm{d}Y/\mathrm{d}t = \nu W(H - Y) - \Gamma Y, \tag{G 1}$$

$$\mathrm{d}H/\mathrm{d}t = rH - \alpha Y, \tag{G 2}$$

$$\mathrm{d}W/\mathrm{d}t = A + \lambda Y - (\mu + \nu H) W. \tag{G 3}$$

Possible equilibrium points are, as ever, obtained by putting all time derivatives equal to zero and solving the ensuing set of three simultaneous algebraic equations. Two biologically meaningful solutions are possible.

First, there is a solution with

$$H^* = Y^* = 0, \tag{G 4}$$

$$W^* = A/\mu. \tag{G 5}$$

This corresponds to the parasite being introduced at a rate sufficiently high to extinguish the host population; the population of free-living infective stages of the parasite is maintained by a balance between immigration (at the net rate A) and death (at the individual rate μ).

The stability of this equilibrium is studied in the standard way by writing $H(t) = H^* + \hat{H}(t)$, $Y(t) = Y^* + \hat{Y}(t)$, $W(t) = W^* + \hat{W}(t)$, and expanding in Taylor series about the equilibrium point. In the resulting set of linear, first-order differential equations the time-dependence of the dynamical variables may be extracted as a factor $\exp(\sigma t)$, to get

$$\sigma \hat{Y} = -(\nu A/\mu + \Gamma) \hat{Y} + (\nu A/\mu) \hat{H}, \tag{G 6}$$

$$\sigma \hat{H} = r\hat{H} - \alpha \hat{Y}, \tag{G 7}$$

$$\sigma \hat{W} = -(\nu A/\mu) \hat{H} + \lambda \hat{Y} - \mu \hat{W}. \tag{G 8}$$

The eigenvalues σ thus obey the cubic equation

$$(\sigma + \mu)(\sigma^2 + B\sigma + C) = 0, \tag{G 9}$$

with the definitions

$$B = \nu A/\mu + \Gamma - r, \tag{G 10}$$

$$C = (\alpha - r)\nu A/\mu - r\Gamma. \tag{G 11}$$

The equilibrium is thus locally stable iff both B and C are positive. The condition $C > 0$ is fulfilled iff

$$\alpha > r \tag{G 12}$$

and

$$A > \mu r\Gamma/\nu(\alpha - r). \tag{G 13}$$

If these conditions are satisfied, it automatically follows that $B > 0$. As discussed in the main text, equation (G 12) is the familiar requirement that the parasite be sufficiently pathogenic to be capable of regulating the host population, and equation (G 13) then gives the threshold rate of introduction of free-living infective stages needed to eradicate the host population (equation (79)). Looking back at equations (F 4) and (F 5) for H^* and Y^* when $A = 0$, and taking the usual limit $\Lambda \to \infty$, we see that equation (G 13) can be rewritten as

$$A > \lambda Y_0^*. \tag{G 14}$$

Here Y_0^* is the equilibrium population of infected hosts in the absence of any artificial introduction of free-living infective stages, $A = 0$; this is equation (80) of the main text.

Secondly, equations (G 1)–(G 3) have another equilibrium solution:

$$H^* = \frac{\alpha[\mu r\Gamma/\nu - A(\alpha - r)]}{r[\lambda(\alpha - r) - \alpha\Gamma]}, \tag{G 15}$$

$$Y^* = (r/\alpha)H^*, \tag{G 16}$$

$$W^* = r\Gamma/\nu(\alpha - r). \tag{G 17}$$

For a biologically meaningful solution $H^* > 0$, we require both that equation (69) be satisfied (this being the familiar condition of § 11, expressing the criterion for the parasite to be capable of regulating its host population), and that

$$\mu r\Gamma/\nu(\alpha - r) > A. \tag{G 18}$$

Equation (G 18) is the opposite of equation (G 13), and requires the introduction rate A not to exceed the threshold value of equation (79) or (G 13) if the host population is to persist at a finite value of H^*.

The stability analysis for this second equilibrium, equations (G 15)–(G 17), proceeds along the lines laid down above and in appendix F. Provided that equations (G 18) and (69) are satisfied, there can be a stable point or a stable limit cycle (with the two regimes divided by a criterion that is the appropriate generalization of equation (71)); we omit the details.

In brief, the host population is extinguished (i.e. the equilibrium solution of equations (G 4) and (G 5) is stable) if $A > A_c$. The host population persists, albeit at a level below that for $A = 0$ (i.e. the equilibrium solution of equations (G 15)–(G 17) is stable, or is the centre of a stable limit cycle) if $A < A_c$. The critical introduction rate A_c is defined by equations (79), (G 13) or by equations (80), (G 14).

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