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# REGULATION AND STABILITY OF HOST-PARASITE POPULATION INTERACTIONS

## II. DESTABILIZING PROCESSES

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## SUMMARY

(1) Three categories of biological processes are shown to have a destabilizing influence on the dynamical behaviour of model host-parasite associations: parasite induced reduction in host reproductive potential, parasite reproduction within a host which directly increases parasite population size and time delays in parasite reproduction and transmission.

(2) The importance of parasitic species as regulators of host population growth is examined in light of empirical evidence. Data from two particular laboratory studies used to indicate the magnitude of this regulatory influence. Suggestions are made concerning the type of information required from field studies to facilitate critical assessment of theoretical predictions.

## INTRODUCTION

In the preceding paper (Anderson & May 1978; hereafter AM), we discussed some of the features that distinguish host-parasite systems as a special class among prey-predator interactions.

We reviewed empirical evidence pertaining to the distribution of parasites among hosts, and to the dependence of parasite induced host mortality and of natural parasite deaths upon the number of parasites in a given host. The influence of these factors upon the overall dynamical behaviour of host and parasite populations was studied by constructing simple mathematical models; such models give qualitative insight into the circumstances under which parasites are capable of regulating their host population, and of doing so in a stable manner.

The present paper is a sequel to AM, and it does two things.

First, we treat various aspects of host-parasite relations that tend to be destabilising: parasite induced reduction in host reproduction (model D); the effects of parasites reproducing directly inside their host (in addition to the production of transmission stages: model E); and the effects of time delays in parasite reproduction and transmission (model F). All these factors tend to have a destabilizing influence.

Secondly, we pull together the conclusions of AM and of this paper, to give a general discussion (buttressed with empirical observations) of the regulatory influence of parasites in natural populations of animals.

## BASIC MODEL

The Basic model, as defined and discussed more fully in AM(eqns (7) and (8)), is one in

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which the changes in the host population, $dH/dt$, come from host reproduction set against losses from natural and parasite-induced mortality (the latter at a rate linearly proportional to the number of parasites in a host). The corresponding changes in the parasite population, $dP/dt$, come from parasite reproduction and transmission, balanced against losses from natural parasite deaths along with mortality attendant upon host deaths.

We now proceed to modify this Basic model in various ways.

## MODEL D: PARASITE INDUCED REDUCTION IN HOST REPRODUCTION

At the beginning of AM we gave a broad definition, at the population level, of the nature of parasitism: this (*inter alia*) stipulated that a parasite will either increase the death rate

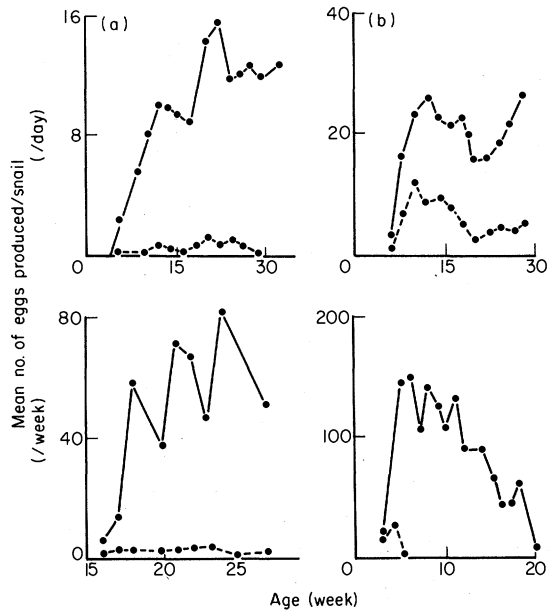


FIG. 1. Some examples of the influence of larval trematode infections on the reproductive potential of their molluscan hosts. The solid lines indicate the rate of production of eggs/snail/unit of time by uninfected snails while the stippled line is the rate for infected snails. (a) *Biomphalaria pfeifferi* (Krauss) infected with *Schistosoma mansoni* (Sambon) (data from Sturrock 1966a); (b) *Bulinus nasutus productus* (Mandahl-Borth) infected with *Schistosoma haematobium* (Weinland) (data from Sturrock 1966b); (c) *Lymnaea stagnalis* (Say) infected with *Trichobilharzia ocellata* (Brumpt) (data from MuClelland & Bourns 1969); (d) *Lymnaea gedrosiana* (Annondale and Prashad) infected with *Ornithobilharzia turkestanicum* (Skrjabin) (data from Massoud 1974).

or/and decrease the reproductive capabilities of its host. In AM, attention was restricted to the former effect.

But parasite induced reduction in host reproduction, and parasitic castration, are well documented phenomena. Such effects are commonly produced by helminth or arthropod parasites, and in particular by larval helminths within invertebrate intermediate hosts. For example, the reduction in fecundity of the mollusc *Biomphalaria truncatus* (Say) infected with the larval stages of the digenean *Schistosoma haematobium* (Sambon) is proportional to the number of larval parasites (miracidia) that have invaded the mollusc

(Chu, Sabbaghian & Massoud 1966). Sometimes the host will resume normal egg laying if the parasitic infection is lost (Berrie 1970). An extreme form of parasite reduced host reproduction occurs when the parasite completely destroys the gonads of its host leading to parasitic castration. Such effects can be caused by protozoa, helminths and arthropods (Bennett 1955; Kurochkini 1961; Noble & Noble 1971).

In general, however, parasites tend to retard but not completely to eliminate host reproduction. Some quantitative examples of the influence of digenean parasites on reproduction by molluscan hosts are illustrated in Fig. 1. Further examples which show the influence of digenean and nematode parasites on the reproductive rates of vertebrate and invertebrate host species are illustrated in Fig. 2.

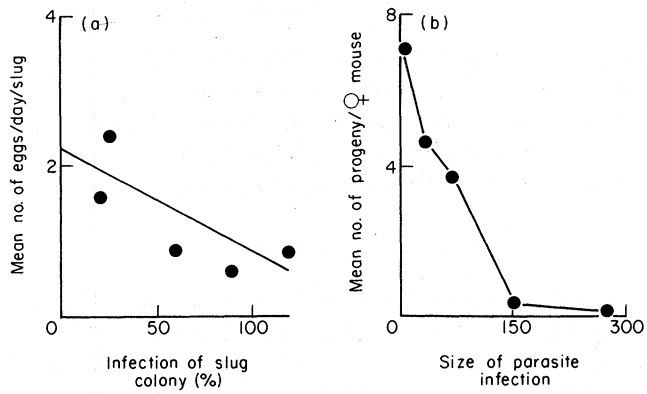


FIG. 2. Some examples of the influence of parasitic infection on the reproductive potential of a host. (a) Colonies of the slug *Agriolimax reticulatus* (Müller) infected with an unidentified Brachylaemid digenean metacercaria (data from Foster 1958); the solid line is the least squares best fit linear model. (b) Laboratory mice infected with the larval stages of the nematode *Trichinella spiralis* (Owen) (data from Weatherly 1971).

We now proceed to study the effects of such processes upon the overall dynamics of host-parasite associations, by appropriate modifications to the basic population eqns (7) and (8) of the preceding paper. We assume, for simplicity, that a given host's intrinsic birth rate, $a$, is diminished by an amount linearly proportional to the number of parasites in that host. The effect on the average host is then to replace $a$ by

$$a \to a - \beta \sum_{i=0}^{\infty} i \,.\, p(i). \tag{1}$$

Here $p(i)$ is the probability that a host contains $i$ parasites, and the constant $\beta$ determines the severity of the parasite's influence on host reproduction. The form of eqn (1) is rather crude, since the host's reproductive rate cannot meaningfully be negative (as it can, in principle, in eqn (1)); this form nonetheless captures the essentials of the phenomenon whose dynamical consequences we wish to explore, and it enables us to do so without distracting mathematical complications. The sum in eqn (1) is, by definition, the mean parasite burden per host, $P/H$, whence the basic eqn (7) of AM for the host population's growth rate becomes

$$dH/dt = (a-b)H - (\alpha+\beta)P. \tag{2}$$

Notice that, under the assumption that effects are linearly proportional to parasite

density, parasite induced decrease in host reproduction (measured by $\beta$) and parasite induced host mortality (measured by $\alpha$) appear in a similar way.

The equation for the growth rate of the parasite population is as before (eqn (8) in AM),

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

The precise form here depends, as discussed in the preceding paper, upon the distribution of the parasites within the host population.

The analyses of the dynamical behaviour of eqns (2) and (3) is outlined in Appendix 1.

For an overdispersed parasite distribution, characterized by a negative binomial with 'clumping parameter' $k$, equilibrium host and parasite populations are possible only if

$$\lambda > \mu+b+\alpha+[\alpha(a-b)(k+1)]/[k(\alpha+\beta)]. \tag{4}$$

The corresponding condition for the existence of an equilibrium for independently randomly distributed parasites (i.e. a Poisson distribution) follows from eqn (4) with $k\to\infty$, and for an underdispersed parasite distribution (characterized by a positive binomial with parameter $k'$) follows from eqn (4) by putting $k\to -k'$.

For an overdispersed distribution, such an equilibrium point (if it exists) is globally stable if and only if

$$\alpha > \beta k. \tag{5}$$

Equation (5) says that for a host population to be stably regulated by its parasites, it is necessary that the parasites' influence on host reproduction be not too severe relative to the parasite induced mortality (i.e. that $\beta$ be small relative to $\alpha$), and/or that the parasites be highly overdispersed ($k$ small). Too small a $k$ value, however, makes it difficult to satisfy eqn (4), the condition for an equilibrium to be possible in the first place. These remarks are borne out by Fig. 3, which shows slices through the $\alpha-k$ parameter space for various values of $\beta$.

For random (Poisson) or underdispersed parasite distributions, *no* stable equilibrium is possible in eqns (2) and (3).

If the intrinsic death rate of the parasites (the terms involving $\mu$) is density dependent, it has a stabilizing influence, as discussed in model C in the preceding paper. Incorporating a linear dependence of the parasite death rate upon parasite density into eqn (3), we have (see eqn (20) in AM)

$$dP/dt = \lambda PH/(H_0+H)-bP-(\alpha+\mu)HE(i^2). \tag{6}$$

The dynamical properties of eqns (2) and (6) are analysed in Appendix 1.

For an overdispersed parasite distribution, the condition (4) for an equilibrium to be possible is replaced by

$$\lambda > \mu+b+\alpha+[(\alpha+\mu)(a-b)(k+1)]/[k(\alpha+\beta)] \tag{7}$$

and the criterion (5) for this equilibrium to be globally stable is replaced by

$$\alpha+\mu(k+1) > \beta k. \tag{8}$$

This equation makes explicit the interplay among the various stabilizing and destabilizing influences: parasite-induced diminution in host reproduction (increased $\beta$) is destabilizing; overdispersion (small $k$) promotes stability; density dependent parasite death, whether intrinsic ($\mu$) or associated with host deaths ($\alpha$), promotes stability.

Putting $k\to\infty$ in eqns (7) and (8), we see that a random (Poisson) distribution of

parasites can lead to a stable host-parasite equilibrium, provided $\mu > \beta$. The stability character of such systems is illustrated by Fig. 4. For underdispersed parasite distributions, both the underdispersion and the parasite induced reduction in host reproduction

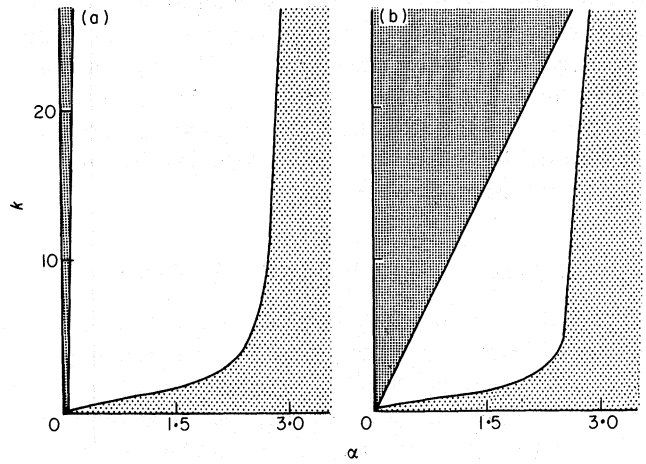


FIG. 3. Model D—Parasite induced reduction in host reproductive potential (parasites overdispersed within the host population). The solid lines enclose unshaded regions in the $k-\alpha$ parameter space in which the model has globally stable equilibria. These boundaries are shown for two different values of $\beta$ ((a) $\beta = 0{\cdot}01$, (b) $\beta = 0{\cdot}1$), which determines the severity of the parasites influence on host reproduction. The lightly shaded regions denote parameter values which lead to unregulated host population growth while the darkly shaded areas indicate parameter values which give rise to globally unstable equilibria ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$).

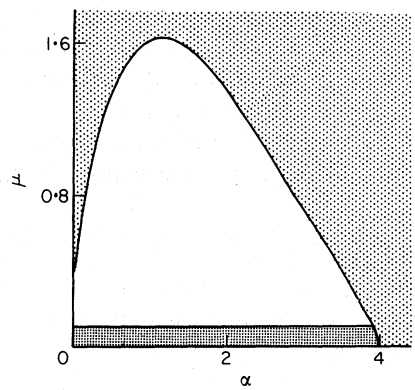


FIG. 4. Model D—Parasite induced reduction in host reproductive potential (parasites randomly (Poisson) distributed within the host population and density dependent parasite mortalities). The solid lines, in the $\mu-\alpha$ parameter space, enclose an unshaded area in which parameter values produce globally stable equilibria. The lightly shaded and darkly shaded regions are as defined for Fig. 3 ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$).

are destabilizing; a density dependent parasite death rate can produce net stability, but only under a very restricted combination of parameters (see Appendix 1).

These models, both with and without density dependent parasite death rates, illustrate that the stability of a host-parasite association can be seriously impaired if the parasite

has a significant influence on the rate of host reproduction. Stability *is* possible (by virtue of effects such as overdispersion of parasites or of density dependent parasite death rates), but only within a narrowly constrained domain of parameter space. On the other hand, many natural host-parasite associations persist, despite the parasite substantially altering the host's reproductive rate; this prompts the observation that the parameters characterizing the interactions between natural populations are a far-from-random set. In particular, associations between larval digeneans and their molluscan hosts, where parasite induced reduction in host reproduction is the rule rather than the exception ($\beta$ large), demonstrate tight density dependent constraints on larval parasite population growth ($\mu$ large) within individual hosts (see Anderson, Whitfield & Mills 1977; Lim & Lie 1969; Lie, Heyneman & Kostanian 1975).

## MODEL E: PARASITES THAT REPRODUCE WITHIN THEIR HOSTS

Many parasitic species (for example, various protozoa) reproduce within their definitive or final host, directly contributing to the growth of their population within that host. This type of reproduction is distinct from the production of transmission stages such as eggs or spores which, as a developmental necessity, pass to the exterior of the host before becoming infective to other members of the host population. The parasitic protozoan *Entamoeba hystolytica* (Schaudinn) exhibits both forms of reproduction. The parasite lives in the alimentary tract of man and multiplies by monotonic fission directly to increase its population size within the gut. Periodically, small precystic individuals are formed which give rise to a resistant cyst containing nutrient reserves; this is discharged with the faeces into the external environment. If such a resistant stage is ingested by an appropriate host, the amoeba excysts and resumes growth and asexual reproduction (Dogiel 1965). Two types of reproduction thus occur: 'normal' births which result directly in population growth, and births which give rise to transmission stages.

The existence of both direct reproduction and transmission stages in the parasite life cycle can make for dynamical properties that are somewhat different from those of models A–D, in which only 'transmission births' occur.

To explore these properties, we modify the basic model A for parasite population growth, eqn (3), to include 'normal' parasite births within the host, at a rate $r$ per parasite:

$$dP/dt = \lambda PH/(H_0+H)+(r-\mu-b)P-\alpha HE(i^2). \tag{9}$$

Here the 'transmission births' are as in model A, and the intrinsic parasite birth and death processes have no density dependence. The corresponding rate of host population growth is as in the Basic model, eqn (7) of AM:

$$dH/dt = (a-b)H-\alpha P. \tag{10}$$

Equations (9) and (10) are those of model A, with the simple modification that the $\mu$ of model A has been replaced by $\mu-r$. Consequently it follows (from eqn (15) of AM, see also Appendix 1) that, for an overdispersed parasite distribution characterized by a negative binomial with parameter $k$, the equilibrium host population is

$$H^* = H_0(d-r)/(\lambda+r-d). \tag{11}$$

Here $d$ is defined as a combination of parasite death rates (due to intrinsic mortality and to parasite induced host deaths) and host population growth rates:

$$d \equiv \mu+\alpha+b+(a-b)(k+1)/k. \tag{12}$$

For an overdispersed parasite distribution, an equilibrium state is therefore possible only if (see eqn (11))

$$\lambda+r > d > r. \tag{13}$$

If $r>d$, the direct reproduction rate of the parasite is so great as to produce excessively severe host mortality: as a result, both host and parasite populations move steadily to extinction. This type of dynamical behaviour has not been encountered in our earlier

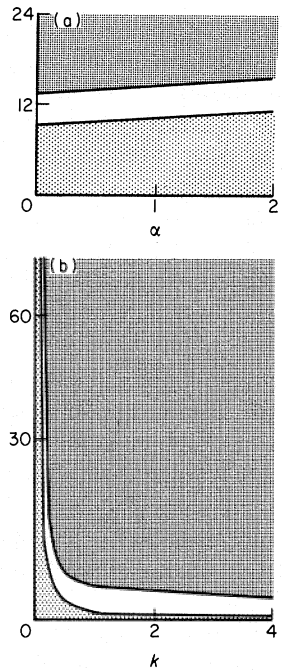


FIG. 5. Model E—Parasites which reproduce directly within their hosts (parasites distributed as the negative binomial model). The unshaded regions in the parameter spaces shown in (a) and (b) denote regions in which parameter values lead to globally stable host and parasite population equilibria. Parameter values in the lightly shaded regions lead to unbounded host population growth while the darkly shaded regions encompass parameter values which lead to the extinction of both host and parasite populations. (a) Shows a slice through the $r-\alpha$ parameter space ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 4{\cdot}0$, $k = 0{\cdot}2$); (b) shows an area in the $r-k$ parameter space. Parameter values as for (a) except $\alpha = 0{\cdot}1$.

models. Conversely, if $\lambda+r<d$, the parasite population cannot increase fast enough to control the host population, and both populations continue to grow exponentially, until resource limitations are encountered by the hosts. This type of behaviour was discussed more fully for model A. If, however, the constraint specified by eqn (13) is satisfied, so that an equilibrium state exists, then it is necessarily globally stable (see Appendix 1).

The corresponding results for a random (Poisson) distribution of parasites are obtained by putting $k\to\infty$; as in model A, this model is globally neutrally stable. For an underdispersed parasite distribution, the equilibrium point (if it exists) is always unstable. These conclusions are substantiated in Appendix 1.

Thus parasites which reproduce directly within their hosts can exhibit a more complex array of dynamical behaviour than those which only produce transmission stages. The existence of a stable equilibrium state depends on the relation between the parasite's population growth parameters $\lambda$ and $r$ and the composite parameter $d$ of eqn (12): this relation is spelled out in eqn (13), and illustrated in Fig. 5. Too large a value of $r$ produces extinction; too small a value of $r$ can (depending on the other parameter values) lead to unregulated host population growth.

In the real world, observed values of $r$ tend to be high since many parasitic protozoa possess enormous asexual reproductive capabilities in the nutrient rich environments within the hosts. One of the malarial parasites, *Plasmodium falciparum* (Welch), can produce in excess of 30 000 merozoites by mitotic divisions from one schizont during the exoerythrocitic stage of its life cycle, which lasts approximately 5 days (Baker 1969). Assuming that few deaths occur during this period, the value of $r$, for such a parasite, would be approximately 10·0 per 5 day time period. Such a figure is obviously many orders of magnitude greater than the reproductive rate of its human host. Figure 5(b) suggests that for large $r$ the extinction of both host and parasite populations may be avoided if $k$ is small, that is, if the parasites are highly aggregated within the host population. This prediction of the model is, however, difficult to test in practice since frequency distributions for the numbers of parasites per host are virtually impossible to determine for protozoan populations which may number many hundreds of thousands in a single host.

Even when a parasite, such as *Plasmodium falciparum*, is capable of reproducing at a very high rate, population growth cannot persist indefinitely within a host. Sometimes parasitic protozoan populations will grow until the death of the host results. More often, however, either host immunological mechanisms or intra specific competition will reduce the effective growth rate $r$ as the parasite population increases. In the absence of overdispersion of parasite numbers per host, the host population can be regulated provided density dependent constraints operate on individual parasite populations within the members of the host population.

Within the framework of model E, such density dependent constraints could operate or $r$, on $\lambda$, on $\mu$, or on some combination. For simplicity, we take this density dependence to be encapsulated in the parasite's natural death rate, as introduced in model C and further discussed in model D. That is, the parasite's intrinsic death rate is $\mu i^2$ in a host with $i$ parasites, and eqn (9) for the growth rate of the parasite population becomes,

$$dP/dt = \lambda PH/(H_0+H)+(r-b)P-(\alpha+\mu)HE(i^2). \tag{14}$$

Qualitatively similar dynamical properties ensue if, alternatively, $r$ or $\lambda$ is chosen to exhibit density dependence.

The stability analysis of eqns (10) and (14) is similar to that for eqns (9) and (10), and is outlined in Appendix 1. For an overdispersed parasite distribution, the condition for an equilibrium state to exist is again given by eqn (13), except that $d$ is replaced by $d'$, defined as

$$d' \equiv \mu+\alpha+b+(a-b)(\alpha+\mu)(k+1)/(\alpha k). \tag{15}$$

For a Poisson distribution of parasites, we put $k\to\infty$; for an underdispersed distribution, $k\to -k'$. As before, both populations become extinct if $r>d'$, whereas both populations grow exponentially if $\lambda+r<d'$. If an equilibrium state is possible ($\lambda+r>d'>r$), it is necessarily globally stable both for an overdispersed and for a random (Poisson) distri-

bution of parasites among hosts. In addition, the equilibrium state (if it exists) for an underdispersed distribution can be globally stable provided the density dependence is sufficiently large (i.e. relatively large $\mu$) and the under-dispersion is not too severe (i.e. relatively large $k'$); more precisely, stability here requires that

$$\mu(k'-1) > \alpha. \tag{16}$$

Density dependent constraints on $r$, $\lambda$ or $\mu$ therefore remove the necessity for the parasite to be highly overdispersed, but they still do not eliminate the danger of parasite

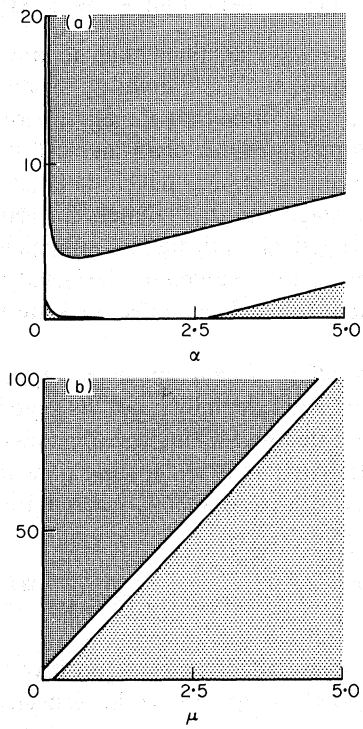


FIG. 6. Model E—Parasites which reproduce directly within their hosts (parasites randomly distributed (Poisson) and with density dependent constraints on the parasite natural death rate); the shaded and unshaded regions are as defined in Fig. 5. (a) Shows an area in the $r-\alpha$ parameter space ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 6{\cdot}0$); (b) illustrates a slice through the $r-\mu$ parameter space (parameter values as for (a), except $\alpha = 0{\cdot}1$).

induced host mortalities causing the extinction of both host and parasite populations if $r$ is too large. These remarks about the way the stability character depends on $r$, $\mu$, $k$ and other parameters are illustrated by Fig. 6.

The general conclusion emerging from model E is that direct reproduction by the parasites within their hosts leads to dynamical behaviour which is, in many respects, similar to that exhibited by models A–D. Overdispersion or density dependent processes are required if the parasites are to be capable of stably regulating the host population.

Model E has, however, one qualitatively new feature: too high a rate of direct parasite reproduction can lead to extinction of both host and parasite populations. The fact that many natural host-parasite associations persist despite the parasites' having a high rate of direct reproduction suggests that other interaction parameters have

coevolved in such a way as to maintain the continued existence of both populations. In particular, the evolution by the host of a highly efficient immune response to the presence of the parasite (high values of $\mu$, see Fig. 6(b)) will tend to prevent extinction of the host population, and hence also ensure the survival of the parasite. For a more general exposition of this point, see Bradley (1974). Quantitative evaluation of these predictions is not at present possible; numerical estimates of the relevant parameters for host-protozoan parasite associations are greatly to be desired.

## MODEL F: THE INFLUENCE OF TIME DELAYS

In all the preceding models we have assumed that no time delays occur between the production of a transmission stage and its availability for reinfection of a new member of the host population. That is, we assume that the eggs, spores or cysts produced by the parasite are sufficiently developed to enable them successfully to infect a host if contact is made. Many parasites, however, do exhibit developmental delays between the point of production of a transmission stage and its readiness for reinfection. The length of such delays will often be associated with climatic factors influencing the habitat. For example, Rose (1956) demonstrated that the time taken by the larval stages of the cattle nematode *Dictyocaulus viviparous* (Bloch) to reach the infective state was very dependent on temperature.

A qualitative account of the effect of such time delays upon the dynamical behaviour of host-parasite associations can be obtained by modifying model A. A developmental time lag of length $T$ can be incorporated by making the transmission term depend on $P(t-T)$ instead of $P(t)$. To be explicit, we use an overdispersed parasite distribution, characterized by a negative binomial model with parameter $k$, whereupon the time delayed version of eqn (3) is

$$dP/dt = \frac{\lambda P(t-T)H}{(H_0+H)} - (\mu+\alpha+b)P(t) - \frac{\alpha(k+1)P(t)^2}{kH}. \tag{17}$$

As always, random (Poisson) or underdispersed (positive binomial with parameter $k'$) parasite distributions correspond to eqn (17) with, respectively, $k\to\infty$ or $k\to -k'$. For the host population, the growth equation remains eqn (10).

The equilibrium host and parasite populations, $H^*$ and $P^*$, are exactly as for model A, as is the condition for this equilibrium state to be possible (namely $\lambda > d$, with $d$ defined by eqn (12), see eqn (16) of AM). The analysis of the stability of this equilibrium state is now, by virtue of the time delay, much more complicated. The analysis is outlined in Appendix 2. If the developmental time lag, $T$ is small compared with other relevant time scales (in a sense defined more precisely in Appendix 2), we have the approximate stability criterion

$$T[\mu+\alpha+a+(a-b/k)] \lesssim 1/k. \tag{18}$$

Equivalently, using the definition of $d$ given by eqn (12), this requires $Td \leqslant 1/k$. In model A, with no time lag, the equilibrium point is necessarily stable for an overdispersed parasite distribution. If the stability criterion (18) is fulfilled, the equilibrium point remains stable. But if the criterion (18) is not fulfilled, the host and parasite populations oscillate in stable limit cycles, the severity of which can easily produce extinction. Notice that if $T$ is significantly in excess of the intrinsic growth time for the host population (i.e. if $T$ significantly exceeds $1/(a-b)$), then eqn (18) cannot be fulfilled for any $k$, and stable limit cycles must ensue.

An alternative approach is to introduce a third differential equation, giving an explicit account of the population dynamics of the free-living infective stages (see Anderson 1976). If $W(t)$ is the number of infective stages at time $t$, then a simple three-equation generalization of model A is

$$dH/dt = (a-b)H - \alpha P \tag{19}$$

$$dP/dt = \beta WH - (\mu+\alpha+b)P - \alpha(k+1)P^2/(kH) \tag{20}$$

$$dW/dt = \lambda P - \gamma W - \beta WH. \tag{21}$$

Here $\gamma$ is the rate of loss of infective stages (due to death or other processes that foreclose the possibility of their infecting a host), and $\beta$ determines the rate at which hosts pick up infective stages. The net transmission rate is assumed proportional to the density of both host and infective stages ($\beta WH$, so that $\beta$ is the transmission rate per host). The other assumptions are as for model A, and in particular the rate of production of infective stages is $\lambda P$; the parasites are taken to be overdispersed.

The dynamical properties of this three-equation model are elucidated in Appendix 2. The condition for an equilibrium state to be possible is again identical with that for model A (eqn (16) of AM), namely that $\lambda > d$ with $d$ as usual defined by eqn (12). The criterion for this equilibrium (which is necessarily stable in model A) to be stable is complicated, and is given exactly in Appendix 2. If the birth, death and transmission rates ($\lambda$, $\gamma$ and $\beta$) of the infective stages are high relative to other rates (as is often the case in natural host-parasite associations), we have the approximate stability condition

$$d/\gamma \lesssim 1/k. \tag{22}$$

If this condition is significantly violated, the system is unstable, with host and parasite populations oscillating in stable limit cycles. Note that eqns (18) and (22) are equivalent if we make the plausible identification that $1/\gamma$ (the characteristic time scale for changes in $W$, eqn (21)) is equal to $T$ (the developmental time for infective stages, eqn (17)).

These remarks about the dynamical properties of host populations described by eqns (19), (20) and (21) are illustrated by Fig. 7.

The limit $\gamma \to \infty$ and $\beta \to \infty$ (with $\gamma/\beta$ finite) is of special interest, because it illuminates the relationship between the three-equation system (19), (20) and (21) with a transmission rate of infective stages linearly proportional to host density ($\beta WH$), and the two-equation model A, eqns (3) and (10), with its saturated transmission term ($\lambda PH/[H_0+H]$). This limit clearly represents the case where there are no developmental lags; the time scale for infective stages to be transmitted is shorter than any other relevant time scale in the model. As a consequence, $W(t)$ always has its equilibrium value, obtained for given H and P by putting $dW/dt = 0$ in eqn (21). This value, $W = \lambda P/(\gamma+\beta H)$, can be substituted into eqn (20), to reduce the three dynamical equations (19), (20) and (21) to the two equations (10) and

$$dP/dt = \lambda PH/(\hat{H}_0+H) - (\mu+\alpha+b)P - \alpha(k+1)P^2/(kH). \tag{23}$$

Equations (10) and (23) are identically model A. Here $\hat{H}_0$ has been defined as

$$\hat{H}_0 = \gamma/\beta. \tag{24}$$

Not only does this discussion shed light on the relationships among the various models, and on the influence of the time scale for the development or transmission of infective stages, but it also gives a biologically meaningful way of interpreting the quantity $H_0$.

For a naturally occurring host-parasite association, eqn (24) suggests that the constant $H_0$ of the earlier models may be estimated as the ratio of the death rate of the infective stages ($\gamma$) to the rate at which infective stages are picked up by a single host ($\beta$).

In all, we see that time delays in the development or transmission of infective stages of the parasite have a destabilizing influence. Such time lags can be described either by introducing them explicitly in the familiar two-equation models, or by introducing a third equation for the dynamics of the free-living transmission stages: the conclusions are broadly similar for the two cases. If the time delay is relatively small ($T$ small compared with $1/d$), the system can be stabilized by overdispersion of the parasites (small $k$).

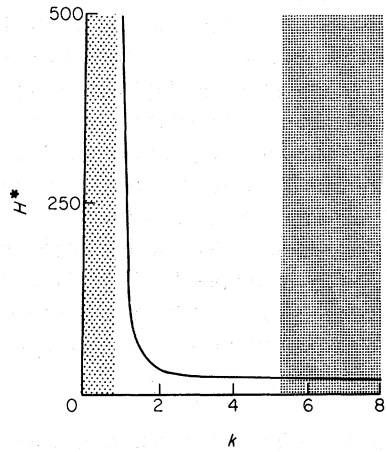


FIG. 7. Model F—The influence of time delays on the dynamics of host-parasite associations (parasites over-dispersed (negative binomial) within the host population). The influence of the negative binomial parameter $k$ on the size of the host population equilibrium H*; equilibria in the unshaded region are globally stable, while in the lightly shaded area the parasite fails to regulate the growth of the host population. In the darkly shaded region, the equilibria are unstable due to the influence of the time delay $T$. ($a = 3{\cdot}0$, $b = 1{\cdot}0$, $\mu = 0{\cdot}1$, $H_0 = 10{\cdot}0$, $\lambda = 20{\cdot}0$, $\alpha = 0{\cdot}5$, $T = 0{\cdot}1$).

Figure 7 bears out this remark. As $T$ becomes larger, more extreme overdispersion is required, and if $(a-b)T$ significantly exceeds unity stability is impossible; here the host and parasite populations exhibit stable limit cycles.

## CONCLUSIONS

In the preceding paper, we distinguished the phenomenon of parasitism among the general class of prey-predator interactions. We then discussed various features that tend to stabilize host-parasite associations: overdispersion of the parasites among the hosts (for which there is considerable empirical evidence, see Table 2 of AM); density dependence in parasitic mortality factors (see Fig. 12 of AM); parasite induced host mortality that increases faster than linearly with the parasite burden (see Fig. 1 of AM).

In the present paper, we have concentrated on other biologically realistic features that make for instability: parasite induced reduction in host reproductive potential (model D); a degree of direct reproduction of parasites within their hosts (model E); time lags in the development of transmission stages of the parasite (model F).

Real host-parasite associations exhibit all the above effects, to a greater or lesser extent. As such, they are in tension between the stabilizing and destabilizing elements of the interaction. Overall, our investigations make it clear that parasitic species are capable of regulating the growth of host populations, even in the complete absence of other influences such as predation or intraspecific competition.

An interesting question raised by this insight concerns the importance of parasites in the real world in constraining the growth of animal populations. In theory, the difference in size between a host population equilibrium generated by parasitic regulation, and the conceptual carrying capacity of the host's environment (which would be attained in the absence of the parasite) will indicate the magnitude of the regulatory role played by a parasite. In our models this difference is determined by the value of the parameter $\alpha$, the rate of parasite induced host mortalities. The equations underlying our models suggest that, in natural populations of hosts, an indication of the severity of the influence of a parasite may be gained by the measurement of the mean parasite load per host. The smaller this mean, the more severe the influence of the parasite (eqn (17) of AM), assuming of course that the populations are in some sort of balance or equilibrium. This indicator, however, is rather crude and should be accepted with a degree of caution since more complex models suggest that the mean parasite load may also, under certain conditions, be weakly dependent on the transmission rate (the term $\lambda PH/(H_0+H)$).

Precisely to examine the importance of parasites in determining the growth characteristics of their host population, observations are ideally required for populations of hosts with and without parasitic infection. Two particular laboratory studies throw some light on this question.

In a classical study of interspecific competition between two species of flour beetles, *Tribolium confusum* (Duval) and *Tribolium casteneum* (Herbst), Park (1948) found that his laboratory cultures were infected with species of the sporozoan parasite *Adelina*. Species of this genus of parasitic protozoa inhabit the intestinal epithelial cells of invertebrates and cause severe damage in certain host species. Park (1948) found that the growth of populations of *T. casteneum* was drastically curtailed by the protozoan. The mean density of *T. casteneum* in cultures with the parasite was approximately 50% less than the density in cultures without the parasite. Park also observed that the parasite, under certain environmental conditions, reversed the competitive advantage between the two beetle species.

A slightly less dramatic demonstration of the influence of a parasite on host population growth is described in an excellent experimental study of the water mite *Hydryphantes tenuabilis* (Bueno) parasitic on the aquatic insect *Hydrometra myrae* (Marshall) (Lancinani 1975). This author demonstrated that the instantaneous rate of increase of uninfected hosts was approximately double that of hosts harbouring an average of ten parasites. Some results from this study are shown in Fig. 8, where the influence of parasite burden on host population growth are recorded.

The studies of Park (1948) and Lancinani (1975) elegantly demonstrate the potential of parasitic species as regulatory agents of host population growth. Is this suggested potential, however, realised in natural populations of hosts and parasites? Unfortunately, due to the immense difficulties in measurement in field conditions, it is not possible at present to answer this question. Autopsy reports of human deaths sometimes provide information concerning the influence of parasitic infections in mortality statistics of underdeveloped regions of the world. Establishing the cause of death, however, is often extremely difficult. Those studies which have been carried out generally suggest that, although the incidence

of a particular parasitic disease may be high in a given human population, the number of deaths ascribed to the influence of the parasite is usually low (Smith, Dyck & Connor 1976; Cohen 1974). Some reports, however, yield a different picture. Bruce-Chwatt & Bruce-Chwatt (1974), for example, reported that infections of malarial parasites caused 40 000 deaths in a human population of 330 000 on the island of Mauritius during the year 1867. Today, for all but a few primitive tribes, it is unlikely that parasites play a major role in determining the growth of human populations. In particular, in the developed world, potential infections are often successfully prevented or terminated by chemotherapeutic treatment.

The frequency of parasite regulated host populations in the real world is more likely to be measured by reference to non-human populations. Unfortunately, however,

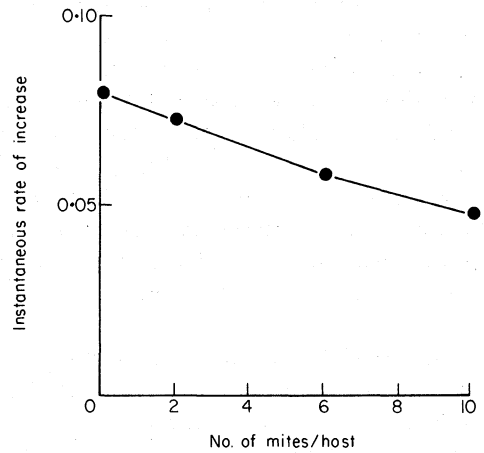


FIG. 8. The influence of parasite burden of the water mite *Hydryphantes tenuabilis* (Marshall) on the instantaneous rate of population increase of its host *Hydrometra myrae* (Bueno). The hosts were fed on a standardized diet of ½ fruit fly/day (data from Lanciani 1975).

establishing the cause of death of animals is perhaps even more difficult in many cases than for humans.

Our knowledge of the role played by parasites in determining the growth of animal populations could be greatly improved by experimental studies of the components of such associations under laboratory conditions. The literature contains relatively few studies along these lines (i.e. Park 1948; Lancinani 1975; Anderson, Whitfield & Mills 1977). Such work is more likely to provide indications of the range of population parameter values that can occur in natural host-parasite associations and hence provide the means of falsifying or supporting theoretical predictions. Although ecological studies have been carried out on a variety of host-parasite associations (see Fig. 1 of AM), concentration on one specific interaction and all its population components has not occurred. As pointed out by Beddington, Hassell & Lawton (1976) with respect to predator-prey interactions, the acquisition of such sets of data is vitally important.

This point is particularly relevant in the case of parasites of economic importance. The parameter boundaries predicted by our models, between a parasite regulated state and 'exponential explosion' of the host population, suggest that control measures designed to eradicate a particular parasite may alter the dynamics of its host population, releasing the

latter from the regulatory constraints of the parasite. For example, in model A, the inequality represented in eqn (16) of AM may not be satisfied if the death rate $\mu$ of the parasite is suddenly increased by the application of chemotherapeutic agents within the host population. Such a change would result in exponential growth of both host and parasite populations until other constraints limit population growth. The control measures would therefore have resulted in an increase in both host and parasite populations and may not even have reduced the mean parasite burden per host. Control measures designed to eradicate a parasite may thus result in the further protection of the parasite population from extinction due to its increased population size.

Such considerations suggest that the acquisition of quantitative estimates of the population rates involved in host-parasite associations of economic significance is a necessity and not an academic luxury.

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## APPENDIX 1

In this appendix, we analyse the stability properties of models D and E. These models are of the general class discussed in Appendix 3 of the preceding paper (Anderson & May 1978), namely

$$dH/dt = c_1 H - c_2 P \tag{1.1}$$

$$dP/dt = P[\lambda H/(H_0+H) - c_3 - c_4 P/H]. \tag{1.2}$$

Here $c_2$ is necessarily positive. As shown previously, such a system can have an equilibrium state only if

$$\lambda > c_3 + c_1 c_4/c_2 > 0. \tag{1.3}$$

Furthermore, this equilibrium (if it exists) is *globally* stable if

$$c_4 - c_2 > 0 \tag{1.4}$$

and globally unstable otherwise (unless $c_4 = c_2$, corresponding to the knife-edge of neutral stability).

Two variants of model D were discussed. In the first, which will henceforth be referred to as model D(a), the *per capita* intrinsic death rate of the parasites was density independent; in the second, henceforth model D(b), this death rate was density dependent. Likewise models E(a) and E(b) have density independent and density dependent parasite death rates, respectively. Specifically, model D(a) is defined by eqns (2) and (3), model D(b) by eqns (2) and (6), model E(a) by eqns (9) and (10), and model E(b) by eqns (10) and (14). Assuming a negative binomial distribution of parasites, we may tabulate the general coefficients $c_i$ ($i = 1,2,3,4$) in eqns (1.1 and (1.2) for these various specific models:

| General form | Model D(a) | Model D(b) | Model E(a) | Model E(b) | |
|---|---|---|---|---|---|
| $c_1$ | $a-b$ | $a-b$ | $a-b$ | $a-b$ | (1.5) |
| $c_2$ | $\alpha+\beta$ | $\alpha+\beta$ | $\alpha$ | $\alpha$ | (1.6) |
| $c_3$ | $\mu+\alpha+b$ | $\mu+\alpha+b$ | $\mu+\alpha+b-r$ | $\mu+\alpha+b-r$ | (1.7) |
| $c_4$ | $\alpha(k+1)/k$ | $(\mu+\alpha)(k+1)/k$ | $\alpha(k+1)/k$ | $(\alpha+\mu)(k+1)k$ | (1.8) |

As ever, the corresponding forms for a random (Poisson) distribution of parasites are obtained by putting $k \to \infty$, and for a positive binomial distribution by putting $k \to -k'$. These expressions may be compared with the somewhat simpler ones for the models A and C, eqns (3.3)–(3.6) in AM.

Use of the general criteria (1.3) and (1.4) in conjunction with the specific forms (1.5)–(1.8) now enables the dynamical behaviour of models D and E to be examined.

*Model D(a)*. Substituting from the column of $c_i$ values for this model into eqn (1.3), eqn (4) of the main text is obtained as the condition for an equilibrium state to be possible. Equation (1.4) shows this equilibrium to be globally stable if

$$\alpha(k+1)\ k-(\alpha+\beta) > 0 \tag{1.9}$$

which gives eqn (5) above for $k>0$. This stability condition can never be satisfied for a Poisson ($k \to \infty$) or positive binomial ($k$ negative) distribution.

*Model D(b)*. The eqn (7) for an equilibrium state to be possible follows from eqn (1.3). The stability criterion equation (1.4) reads

$$(\alpha+\mu)(k+1)/k-(\alpha+\beta) > 0 \tag{1.10}$$

which for $k>0$ leads to eqn (8) above. For a random (Poisson) distribution, this stability condition becomes simply ($k \to \infty$)

$$\mu > \beta \tag{1.11}$$

and for an underdispersed distribution ($k \to -k'$) it is

$$\mu(k-1) > \beta k' + \alpha. \tag{1.12}$$

These stability criteria bear out the remarks made in the main text and illustrated in Fig. 4.

*Model E(a)*. Equation (1.3) in conjunction with the appropriate column of $c_i$ values gives

$$\lambda > \mu+\alpha+b-r+(a-b)(k+1)/k > 0. \tag{1.13}$$

Using the definition (12) for $d$, this gives eqn (13) in the main text. A phase-plane argument readily shows that $r>d$ leads to extinction of host and parasite populations, whereas $\lambda+r<d$ leads to unbounded exponential growth. As for model A, $c_4-c_2$ is simply $\alpha/k$, so that the equilibrium state is globally stable for $k>0$ (negative binomial), neutrally stable for $k \to \infty$ (Poisson), and unstable when $k \to -k'$ (positive binomial).

*Model E(b)*. Again eqn (1.3), in conjunction with the definition (15) for $d'$, leads to the condition

$$\lambda > d'-r > 0 \tag{1.14}$$

for an equilibrium state to be possible. The global stability condition (1.4) is here

$$(\alpha+\mu)(k+1)/k-\alpha > 0. \tag{1.15}$$

This is automatically satisfied for overdispersed ($k>0$) or Poisson ($k \to \infty$) distributions. An underdispersed ($k \to -k'$) distribution can be stable, provided eqn (16) above is satisfied.

## APPENDIX 2

If a developmental time lag is introduced into the Basic model A via an explicit time delay in the transmission term, we have the pair of differential eqns (10) and (17).

The equilibrium host and parasite populations are found by putting $H(t)$ and $P(t)$ equal to the constant values $H^*$ and $P^*$, respectively. The time delay is then irrelevant, and $H^*$ and $P^*$ are exactly as given previously for model A, namely $H^* = H_0\, d/(\lambda-d)$ and $P^* = H^*\,(a-b)/\alpha$ (with $d$ defined by eqn (12), $d = \mu+\alpha+a+(a-b)(k+1)/k$).

A linearized stability analysis of this equilibrium point proceeds along the lines outlined in the preceding paper. We put $H(t) = H^*+x(t)$ and $P(t) = P^*+y(t)$, and keep only terms linear in $x$ or $y$. The damping rates (or eigenvalues) $\Lambda$ that determine the stability character of the system may then be obtained by noting that the time-dependence in $x(t)$ and $y(t)$ goes as $\exp(\Lambda t)$. For eqns (10) and (17) in the neighbourhood of the equilibrium point, this leads to

$$\Lambda x = (a-b)x-\alpha y \tag{2.1}$$

$$\Lambda y = [d(a-b)(\lambda-d)/(\alpha\lambda)+(a-b)^2(k+1)/(\alpha k)]x- [(a-b)(k+1)/k+d(1-\exp\{-\Lambda T\})]y. \tag{2.2}$$

The quantity $d$ is as defined above, eqn (12).

In place of the straightforward quadratic equation for $\Lambda$ that we encountered heretofor, we now have a nasty transcendental equation:

$$\Lambda^2+\Lambda[(a-b)/k]+[d(a-b)(\lambda-d)/\lambda]+d[\Lambda-(a-b)][1-\exp(-\Lambda T)] = 0. \tag{2.3}$$

Methods for analysing such stability-determining equations are given, along with further references to the pertinent literature, in May (1973). If $T$ is small, in the sense that $\Lambda T$ is significantly less than unity, the exponential may be expanded to get the quadratic equation,

$$a_1\Lambda^2+a_2\Lambda+a_3 \simeq 0. \tag{2.4}$$

For stability, it is required that $a_1$, $a_2$, $a_3$ all be positive. Here $a_1 = 1+dT>0$, $a_3 = d\,(a-b)\,(\lambda-d)/\lambda>0$ provided the equilibrium point exists, and $a_2 = (a-b)/k-(a-b)dT$. The approximate stability criterion is therefore that

$$1/k-dT \gtrsim 0 \tag{2.5}$$

which is the result (18) quoted above.

An interesting exact result may be obtained from (2.3) in the limit of very large $\lambda$ and small $k$; i.e. for $\lambda>>d$ and $k<<1$ so that $d\simeq(a-b)/k$. Then, neglecting terms of relative order $k$ or $d/\lambda$, eqn (2.3) reduces to

$$\Lambda+(a-b)+[\Lambda-(a-b)][1-\exp(-\Lambda T)] = p. \tag{2.6}$$

It can be shown exactly that the equilibrium state is stable if $(a-b)T<\Pi/\sqrt{3}$, and unstable (stable limit cycles) otherwise. This exact results accords (to within the numerical factor $\Pi/\sqrt{3}$) with the appropriate limit of the simple approximation (2.5).

If the stability condition derived from eqn (2.3), or approximated by eqn (2.5), is not satisfied, numerical simulations and analogy with other prey-predator studies suggest that stable limit cycles ensue: this is a plausible conjecture, not (as yet) a theorem.

The developmental time lags may alternatively be handled by introducing a third differential equation, to get the three-equation system (19), (20) and (21). The equilibrium populations are obviously

$$H^* = \gamma d/[\beta(\lambda-d)] \tag{2.7}$$

$$W^* = (a-b)d/(\alpha\beta) \tag{2.8}$$

$$P^* = H^*(a-b)/\alpha. \tag{2.9}$$

Here, as ever, $d$ is defined by eqn (12). The linearized stability analysis is routine, and follows the lines outlined above and elaborated elsewhere (e.g. May 1975). Since there are now three first order differential equations, the equation for the stability-determining damping rates $\Lambda$ is a cubic:

$$\Lambda^3+A\Lambda^2+B\Lambda+C=0. \tag{2.10}$$

The coefficients A, B, C can be calculated to be

$$A=\gamma\lambda/(\lambda-d)+d+(a-b)/k \tag{2.11}$$

$$B=(a-b)\gamma\lambda/[k(\lambda-d)] \tag{2.12}$$

$$C=(a-b)\gamma d. \tag{2.13}$$

The Routh-Hurwitz criteria for this system to be stable is that all three coefficients be positive (which they are), and that $AB>C$. The exact condition for the equilibrium populations in this system (if they exist) to be stable is, therefore

$$\frac{\lambda}{k(\lambda-d)}\left[\frac{\lambda}{(\lambda-d)}+d+\frac{(a-b)}{k}\right]> \ . \tag{2.14}$$

This stability condition is independent of $\beta$.

If $\gamma$ is large compared to other relevant rates, the first term in the brackets predominates, and the criterion (2.14) becomes approximately

$$\gamma > kd[(\lambda-d)/\lambda]^2. \tag{2.15}$$

If, furthermore, $\lambda$ is significantly larger than $d$, eqn (2.15) gives the approximate result (22) discussed in the main text.

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