PLANT COMPETITION MEDIATED BY HOST-SPECIFIC PARASITES—A SIMPLE MODEL

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Abstract

It is suggested that competing co-dominant species in natural ecosystems may be maintained in equilibrium by an ecological, negative feedback system involving reciprocal, host-specific parasites. A simple model is derived to describe this relationship and a hypothetical example computed to illustrate its use. The model has important implications for the management of natural forests and for the practice of monoculture. These are briefly discussed.

I. Introduction

There are many instances of natural ecosystems where structurally and physiologically similar plant species appear to exist in stable equilibrium despite strong competition between them for the available resources. This is illustrated very strikingly by the example of co-dominant tree species in temperate mixed forests of the Australian Capital Territory (Pryor 1953). If such species exploit the same physical and chemical resources of the environment, as appears to be the case in the above example, there is a need to explain how the equilibrium is maintained. The slightest inequality in competitive ability for these resources would lead to the eventual extermination of the less fit species. This has been demonstrated by Park (1955) with flour beetles, Utida (1953) with bean weevils, and Slobodkin (1962) with hydras. To explain the apparent stability of the system, it is necessary to postulate the existence of a negative feedback mechanism which would counteract the tendency of one plant to outgrow the other. Pimentel (1963) assigns such a role to genetic feedback mechanisms. The most pertinent of these is where a species, because of its rarity, is exposed to a more acute selection pressure than a more common competitor and consequently evolves towards greater competence in utilization of the environment so that its frequency increases relative to its competitor. Pimentel acknowledges, however, that this mechanism acts slowly and expresses the opinion that other mechanisms like isolation, competition, parasitism, and predation are involved in maintenance of the equilibrium. He does not explain though how these other factors actually operate to achieve this. This paper describes such a system.

It is our contention that two strongly competing plant species can be maintained in stable equilibrium by an ecological feedback mechanism involving two sets

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of host-specific parasites such as fungi, insects, and nematodes. The following conditions need to be satisfied:

(1) an increase in the number of one-host species must cause a disproportionate increase in parasite numbers; and

(2) their enhanced antagonism must be directed back largely or wholly towards that particular host.

The second condition is readily satisfied. Most parasites show some host specificity, often directed towards a very narrow range of hosts. In addition, a parasite will usually inflict different levels of damage upon the various plants constituting its host range. In the local forests referred to above, a preliminary examination suggests that much of the damage caused by leaf spots and insect attack is specific to one or other of the co-dominant eucalypt species present.

The first condition provides an interesting problem. Many pathologists appear to accept as axiomatic the proposition that a dense stand of plants is likely to suffer more severely from infectious disease than would a diffuse stand. Such an effect has often been demonstrated to undergraduate classes by one of us (G.A.C.) using *Pythium* on cress seedlings and is exemplified by the impossibility of culturing *Hevea braziliensis* in dense stands within its native Brazil (Harper 1969). There appears, however, to have been little interest in collecting data to quantify the phenomenon. This may be because plant pathologists generally work with dense agricultural stands where drastic reductions of density as a prophylactic measure would be unacceptable agronomically.

Clearly, the influence of host density upon disease impact must be given special attention during the investigation of the overall hypothesis, but the main difficulty with the hypothesis is to handle, indeed to comprehend, the complex dynamics of the interactions between two host plants and a group of parasites of varying degrees of specificity. For this reason, a simplified model of the system has been devised, and in order to illustrate the main implications of the proposal, computer simulation has been used. This will also serve as a guide to the collection of field data and the design of subsequent experiments.

II. The Model

(*a*) *Simplifying Assumptions*

For initial convenience in modelling, the convention employed by Van der Plank (1963) is followed, whereby the variable components of the system are expressed as proportions. In this case, the environment is imagined to be capable of supporting a limiting quantity of host tissue to which is ascribed the value 1. Two host species (X and Y) compete for space in this environment. All the host-specific parasites are merged into two superparasites x and y which are specific to hosts X and Y respectively. No distinction is made between proportion of parasite and proportion of diseased tissue. There is no provision included for the automatic adjustment of growth rate and death rate constants in response to seasonal changes in the physical environment and the latent period in disease development is ignored. In short, the model deals with a very much simplified ecosystem and describes the density-dependent

interactions of the four biological components, independently of environmental fluctuations.

(*b*) *Equations*

The following four expressions provide descriptions of the rate of change of the four separate components in the system:

Host X:

$$dX/dt = R_X X[1-(X+Y)]-(D_x x+D_n X-D_x x D_n X). \quad (1)$$

Host Y:

$$dY/dt = R_Y Y[1-(X+Y)]-(D_y y+D_n Y-D_y y D_n Y). \quad (2)$$

Parasite x:

$$dx/dt = R_x x(X-x)[1-x(A_X x+A_Y Y)]-(D_x x+D_n x-D_x D_n x^2). \quad (3)$$

Parasite y:

$$dy/dt = R_y y(Y-y)[1-y(A_Y y+A_X X)]-(D_y y+D_n y-D_y D_n y^2). \quad (4)$$

In these equations

X and Y represent the proportions of the two hosts, and $X+Y \leqslant 1$. [Corollary: $1-(X+Y)$ is the proportion of the environment which is not colonized by host plants.]

x and y represent the proportions of two specific parasites in the environment (or the diseased tissue due to them), with $x \leqslant X$ and $y \leqslant Y$. [Corollary: $X-x$ is the proportion of healthy, unparasitized tissue of host X, and $Y-y$ is the proportion of healthy, unparasitized tissue of host Y.]

R_X, R_Y, R_x, R_y are growth rate constants for the four organisms.

D_x and D_y are death rate constants for diseased tissue relating to the specific attacks of parasites x and y.

D_n is a death rate constant applicable to all host tissues which relates to "background" death due to non-specific causes.

A_X and A_Y are absorption constants describing the efficiency with which the two hosts collect inoculum from the environment.

(*c*) *Derivation*

The first term in each of the equations (1) and (2) above describes the rate of increase of one host plant and has the overall form of Van der Plank's (1963) equation number 3.2 which generates a sigmoidal increase in proportion of tissue with time. Thus the two hosts, starting from low proportions, will increase almost exponentially at first, but as the proportion of environment left for them to expand into decreases, their growth rates will fall off and eventually asymptote to zero. Equations (3) and (4) for the two parasites have a similar term, e.g. $R_x x(X-x)$ plus a correction

for absorption of inoculum as the density of absorbing tissue increases, e.g. $1-x(A_Xx+A_YY)$. These latter terms were derived as follows:

During transmission between two host plants, inoculum intensity is known to fall off exponentially with distance from the source due to a combination of dispersal, and absorption by the ground and foliage on the way. Dimond and Horsfall (1960) give an equation (their number 9) describing this phenomenon. In the case of a two-dimensional array of randomly placed plants, where the inoculum may arrive at each plant from any direction over a period of time, it becomes very difficult to predict the actual levels of inoculum arriving at any point. Fortunately for our purposes here, it is only necessary to know how inoculum intensity varies with host density and this relationship resolves itself more simply. Ignoring for the moment the question of absorption of inoculum by foliage, any given point in the array receives doses of inoculum from a range of emission points, where the size of each inoculum dose depends upon the distance travelled and the number of such doses depends upon the number of emission points. If the total number of emission points is increased by random interpolation of diseased plants into the array, then the number of emission points at each distance from the reference point will increase in the same proportion, and so will the number of doses of inoculum received by the reference point. There is, therefore, no need to consider the actual size of each dose of inoculum, merely the number. In general terms then, the amount of inoculum arriving at each point in the random array over a given period of time (inoculum intensity) is directly proportional to the number of emission points (density of diseased plants equals, for example, x).

Similarly the absorption of inoculum will be proportional to the amount of host tissue available to collect it, the constant of proportionality (A_X) being an expression of the efficiency of collection. Thus the amount of inoculum absorbed by plants at any time will be given by:

$$xA_XX.$$

However, only that inoculum which is collected by healthy host tissue can produce disease, so the amount of inoculum absorbed productively is described by:

$$xA_X(X-x).$$

Moreover, the inoculum collected unproductively by tissue which is already diseased, xA_Xx, might otherwise have been available to cause infection. Thus the amount of productive inoculum will be reduced by the interaction factor $(xA_Xx)[xA_X(X-x)]$ giving:

$$xA_X(X-x)-(xA_Xx)[xA_X(X-x)].$$

Since the second host, Y, will also absorb some of this inoculum unproductively, a similar correction must be applied to give:

$$xA_X(X-x)-(xA_Xx)[xA_X(X-x)]-(xA_YY)[xA_X(X-x)],$$

which simplifies to:

$$A_Xx(X-x)[1-x(A_Xx+A_YY)].$$

This expression describes the amount of inoculum which is finally available to cause disease on the appropriate host plant. Not all of it will produce disease, however,

since other factors like inoculum infectivity and viability, host susceptibility, and predisposition all mediate the final response to contact between parasite and host. In Van der Plank's formulae, all these effects are combined into a single infection rate constant typical of the particular host–parasite system operating within a given set of environmental conditions. We achieve the same thing by merging the initial A_X of the previous expression into the growth rate constant R_x.

Actually the problem of absorption is more complex than the above expression allows because inoculum during transmission is subjected to a sequential absorption. The above correction is adequate, however, unless A_X becomes extremely large or the rate of increase of disease at very high values of x is required to be computed with great accuracy.

The second major term in each equation of the model describes the decay rate of the component in terms of the death rate due to the specific parasite plus the death rate due to non-specific causes, less a correction factor for interaction between the two (the same piece of tissue cannot be killed twice). Biotrophic parasites would of course exert their major effect upon the host by reducing growth and reproductive rates, but again for simplicity in this initial model the parasites are imagined to exert all their effect upon the hosts through the death rates.

Equations (1) and (2) describing the behaviour of the two host species, are linked only by the condition that X and Y compete for space in the same environment. This competition is provided by the term $[1-(X+Y)]$ appearing in both equations. Equations (1) and (3), and also (2) and (4) are intimately linked at several points befitting the host–parasite relationships which they represent. However, equations (3) and (4) are essentially distinct from each other in that they contain no common terms except those required by the mutual interception of inoculum by their respective hosts. The model thus consists of two symmetrical halves which are linked only through the fact of competition between the two hosts. This needs to be borne clearly in mind when the model is operated and interactions appear.

III. Computed Illustration

To illustrate the working of the model and the hypothesis which underlies it, an example was computed employing the digital analog simulator program CSMP modified for use with teletypes and a CDC3600 computer. A set of parameters were allotted to the four components of the model and the equations integrated through time to provide graphical descriptions (Fig. 1) of the growth of hosts and parasites in different combinations. Figure 1(*a*) shows the increase of host X when grown alone, from a very low initial value up to an equilibrium level determined by the balance of growth rate and non-specific death rate. At point p, the specific parasite x was introduced at a low level. The parasite increased rapidly until its depredations reduced the level of host X. Both host and parasite then equilibrated to levels determined by their interaction. Figure 1(*b*) shows the same events for host Y and parasite y. Host Y had been given a growth rate somewhat less than host X which accounts for its lower equilibrium levels with and without the parasite. Figure 1(*c*) illustrates the competition between the two hosts when low initial numbers were permitted to multiply in an environment containing no parasites. No equilibrium was reached and it is clear that the faster growing X would eventually eliminate Y from the system.

Figure 1(*d*) shows what happened when all four components of the ecosystem were present. The curves show many features which are common to the other graphs. Host *X* initially outgrew *Y* as in Figure 1(*c*), but then its specific parasite increased and the consequent reduction of *X* provided *Y* with a temporary advantage. Host *Y* was then subjected in its turn to an epidemic of parasite *y* which redressed the balance.

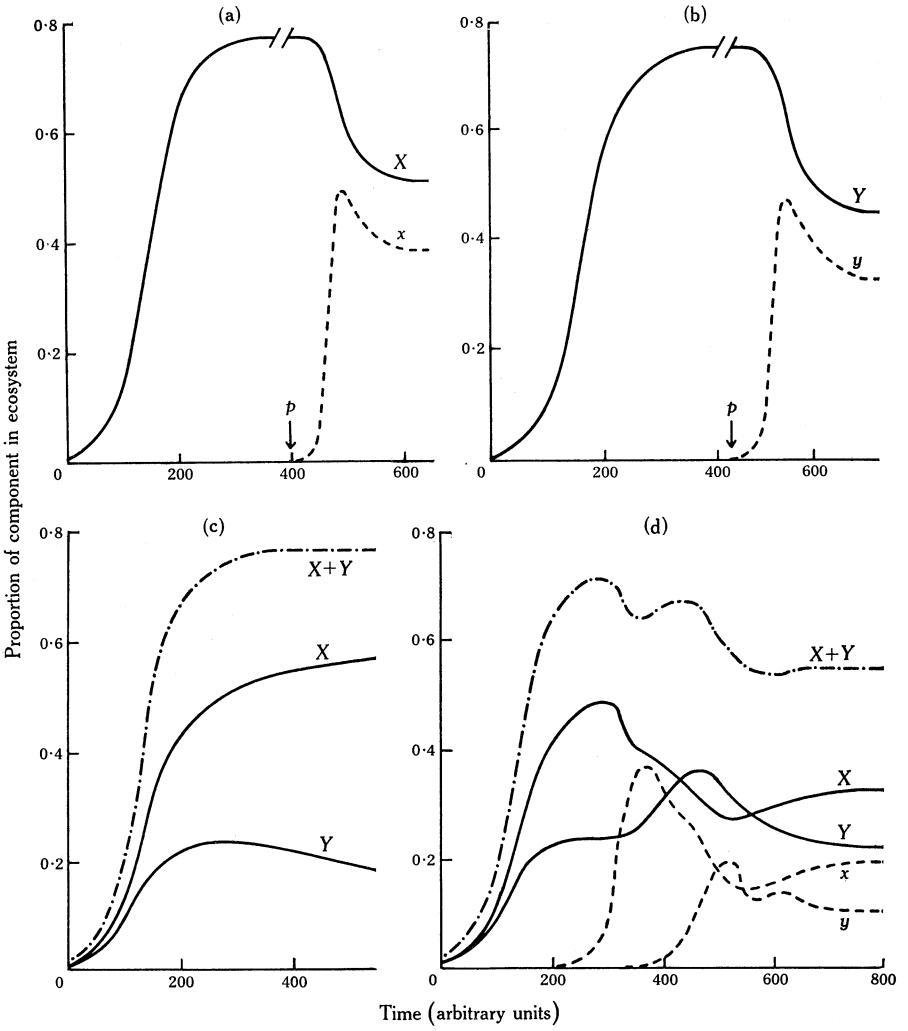

Fig. 1.—Operation of the model. *X* and *Y* are the proportions of two host species; *x* and *y* the proportions of their respective host-specific parasites. In all systems where they appear, each component has been assigned the same rate constants and initial values. (*a*) Host *X* alone, parasite *x* added at *p*; (*b*) host *Y* alone, parasite *y* added at *p*; (*c*) host *X* and *Y* together in the absence of parasites; (*d*) hosts *X* and *Y* with parasites *x* and *y*.

After a small number of damping oscillations, all four components came into equilibrium with one another. Competition between the hosts was responsible for the

failure of the four components to find stable levels immediately. Such interactions are common features of biological models.

Particular attention is drawn to the comparison between the equilibria levels of X in Figure 1(a), Y in Figure 1(b), and $X+Y$ in Figure 1(d). The system containing all four organisms produced significantly more host tissue ($X+Y$) than either of the systems containing only one host and its parasite. This is because the four-component system has less total parasite present due to their reduced rate of increase at lower host densities.

A number of other examples have been computed, the results of which show that the equilibria eventually arrived at are typical of the rate constants used in the model but are quite independent of the starting proportions of X, Y, x, and y. The system is thus stable in the sense that for a given set of parameters it will return the proportions of the four components to the same level from any starting point. An equilibrium, like the one illustrated, of all four components is possible over a reasonably wide range of values of the constants, but if the growth rates of the two hosts are too discrepant, then a three-component equilibrium is achieved between the two hosts and the parasite of the fastest-growing host, the other parasite being reduced to negligible proportions.

Several variations of the equations have been tried with similar results, demonstrating that the model is quite robust. For instance, the functions correcting for interactions, $(1-x(A_X x+A_Y Y)]$ and $(D_x D_n x^2)$, can be left out of the model without altering the general shape of the growth curves or the positions of the equilibria to any great degree.

IV. Implications

Computer modelling is a powerful tool for the initial investigation of hypotheses that deal with complex systems. For instance, the above model has already served a useful purpose by demonstrating that the main hypothesis is reasonable and worth pursuing. It has also drawn attention to the main parameters which need to be measured in the field. In its present simplified form, the model should have prediction value when applied to experiments with selected host–parasite pairs maintained under controlled conditions. On the other hand, considerable elaboration will be necessary before the model can be used to make predictions about changes occurring in complex natural ecosystems. Further development, however, requires a feedback of empirical data. At present, very little information about parasitism in balanced communities is available.

There are practical advantages to be gained from an increased understanding of the host–parasite balance in natural communities. For instance, it is not uncommon practice during the management of mixed natural forests, such as the one which provides our starting point, for one tree species to be removed selectively in the simple expectation that it will be replaced by a worth-while quantity of the other, more commercially desirable species. It would be useful to have a model capable of predicting the reduction in total host tissue, or "monoculture deficit", which would result from such a change. Similarly in the present climate of growing concern about the dangerous consequences of the use of pesticides and fungicides, it might be worth while

investigating the possibility of growing some agricultural and horticultural plants in mixed stands, as an alternative to monoculture, with the object of reducing the overall disease impact. It seems probable that some of the success of the mixed pasture evolved by northern European ley farmers is attributable to disease control effected by this mechanism.

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VI. References

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